Diving and Subaquatic Medicine

FOURTH EDITION

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JOHN PENNEFATHER & ROBYN WALKER

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Fourth edition

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Preface

I take full responsibility for the decision to produce a fourth edition of this text. There have been many changes in diving medicine over the past decade, and some of these have been worthy of inclusion.

The problems of diver safety, and the causes of deaths while diving, have not altered appreciably over the past thirty-five years. The lessons from the past have not been well learnt.

Nevertheless, the scope of diving medicine has increased, partly due to the wider range of divers – both psychologically and medically. It is also due to the extension of the diving envelope, including the diving exposures and equipment available.

The final reason for producing a new edition is the admission that the previous texts were incomplete in that they did not pay adequate attention to the problems of free divers, divers with disabilities, and technical divers.

I no longer promise that this will be the last edition. What I do promise is that it will be the last edition for the original three authors. Already we have had to enlist the assistance of a younger, but very experienced and skilled co-author, Dr Robyn Walker.

Hopefully, and because of the enthusiasm, expertise and skills of our younger colleagues, we can with confidence pass the baton to them. Our legacy and intent is that they will experience as much excitement, achievement, camaraderie and fun from diving as we have.

Carl Edmonds

Preface (Excerpts from earlier Editions)

This book is written for doctors and paramedics who are called upon to minister to the medical needs of those divers who venture on or under the sea.

The very generous praise given by reviewers to the first edition of *Diving and Subaquatic Medicine*, and its surprising acceptance outside the Australasian region, inspired us to prepare further editions of this text.

Diving accidents are now better defined, investigated and treated, than when we commenced writing on this subject, some 30 years ago. It is our intent to present, as completely as possible, an advanced and informative book on clinical diving medicine. We have avoided the temptation to write either a simplistic text or a research tome.

Our primary audience is the diving clinician, the physician responsible for scuba divers, the diving paramedic and the exceptional diving instructor who needs a factual reference text. We have not encompassed the needs of specialized deep or saturation diving units, submarine and military operations. But because good clinicians have a renaissance quality, refusing to accept the limitations of any speciality, we have included some information on these subjects.

The recent extension of diving as a recreational and as a commercial activity has led to the bewildered medical practitioner being confronted with diving problems about which he or she has received little or no formal training. Doctors experienced in diving had previously found themselves in a similar situation – without a comprehensive clinical text.

This book encompasses the range of diving disorders experienced by both the amateur scuba diver and the professional deep-sea diver. It presents all aspects of diving medicine from ancient history to the latest research, in a concise and authoritative manner. Each disorder is dealt with from an historical, aetiological, clinical, pathological, preventative and therapeutic perspective. Summaries, case histories and revision aids are interspersed throughout. For the doctor who is not familiar with the world of diving, introductory chapters on physics and physiology, equipment and the diving environments have been included.

In the later editions, we attempted to be less insular. Instead of an Australian book for Australians, we sought the advice and guidance of respected friends and colleagues from other countries, and from other disciplines, especially in the UK, USA, Canada, Japan and mainland Europe. This has not prevented us from being judgemental and selective when we deemed it fit. The inclusion of anecdotes and occasional humour may lessen the load on the reader – as it does on the authors.

This Third edition was to be the last of this text, at least in its current format. One of the physicians is more interested in diving the remote areas, than in writing about them. Another has moved into a specialist anaesthetic practice, and the scientist author is the sole survivor at the Royal Australian Navy Submarine and Underwater Medicine Unit.

To the speciality of diving medicine, to its courageous pioneers (many of whom are still bubbling and finning), and for the comradeship that diving has engendered, we shall be forever grateful.

Carl Edmonds
Christopher Lowry
John Pennefather

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We wish to acknowledge the assistance given by the Royal Australian Navy, the Royal Navy and the United States Navy for permission to reproduce excerpts from the *Diving Manuals*, and to the many authors upon whose work we have so heavily drawn, our families who have suffered unfairly, and our clinical tutors – the divers.

Special appreciation is given to the Medical Services of the Royal Australian Navy. Approval and support from this department, and its respective directors, has allowed the development of a diver's sick bay into the Submarine and Underwater Medicine Unit, where we met and worked together.

A number of experts have been consulted, to review and advise on specific chapters of this and previous editions. Our gratitude is extended to these valued colleagues, but they are not to blame for the final text. They include:

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- Peter Sullivan
- Ed Thalmann
- John Tonkin
- John Williamson
- David Yount

Dedication

This book is dedicated to the memory of Pluto who died, even though he never left dry land.

1

History of diving

JOHN PENNEFATHER

BREATH-HOLD DIVING

The origins of breath-hold diving are lost in time. Archaeologists claim that Neanderthal man, an extinct primitive human, dived for food, but we cannot be sure if they gathered shell fish by wading at low tide. By 4500 BC, underwater exploration had advanced from the first timid dive to an industry that supplied the community with shells, food and pearls.

From the ancient Greek civilization until today fishermen have dived for sponges. In earlier days, sponges were used by soldiers as water canteens and wound dressings, as well as for washing. Breath-hold diving for sponges continued until the nineteenth century when helmet diving equipment was introduced, allowing the intrepid to gamble their lives in order to reach the deeper sponge beds. Greek divers still search the waters off northern Africa for sponges.

The ancient Greeks laid down the first rules on the legal rights of divers in relation to salvaged goods. The diver's share of the cargo was increased with depth. Many divers would prefer this arrangement to that offered by modern governments and diving companies. One Greek fisherman and diver named Glaucus plunged into the ocean never to return, and was raised to the status of a God.

In other parts of the world, industries involving breath-hold diving persist to this time. Notable examples include the **Ama**, or diving women of Japan and Korea, and the pearl divers of the Tuamoto Archipelago.

The Ama have existed as a group for over 2000 years. Originally, the male divers were fishermen and the women collected shells and seaweeds that are a prized part of Korean and Japanese cuisine. In more recent times diving has been restricted to the women, with the men serving as tenders. Some attribute the change in pattern to better endurance of the women in cold water; others pay homage to the folklore that diving reduces the virility of males – a point many divers seem keen to disprove.

There is a long history of the use of divers for strategic purposes. Divers were involved in operations during the Trojan Wars from 1194 to 1184 BC. They sabotaged enemy ships by boring holes in the hull or cutting the anchor ropes. Divers were also used to construct underwater defences designed to protect ports from the attacking fleets. The attackers in their turn used divers to remove the obstructions.

By Roman times precautions were being taken against divers. Anchor cables were made of iron chain to make them difficult to cut, and special guards with diving experience were used to protect the fleet against underwater attackers.

Some Roman divers were also involved in a rather different campaign, for example Mark Anthony's attempt to capture the heart of Cleopatra. Mark

Anthony participated in a fishing contest held in Cleopatra's presence and attempted to improve his standing by having his divers ensure a constant supply of fish on his line. The Queen showed her displeasure by having one of her divers fasten a salted fish to his hook.

Marco Polo and other travellers to India and Sri Lanka saw the pearl diving on the Coromandel coast. They report that most diving was to depths of 10–15 metres but that the divers could reach 27 metres. (*Note:* The conversion 10 metres = 32.8 feet may assist readers whose concept of depth is derived from the anatomy of King Henry I.) These divers used a weight on a rope to assist descent and a net in which to put the oysters. When they wished to surface, they were assisted by an attendant who hauled on a rope attached to the net. The divers were noted to hold the nose during descent.

The most skilled of the American native divers came from Margarita Island. Travellers who observed them during the sixteenth, seventeenth and eighteenth centuries reported that the natives could descend to 30 metres and remain submerged for 15 minutes. They could dive from sunrise to sunset seven days a week. The divers attributed their endurance to tobacco! They also claimed to possess a secret chemical which they rubbed over their bodies to repel sharks. The Spaniards exploited the native divers for pearling, salvage and smuggling goods past customs. The demand for divers was indicated by their value on the slave market; prices up to 150 gold pieces were paid.

EARLY EQUIPMENT

The history of diving with equipment is long and complex, and in the early stages it is mixed with legend. The exploits of Jonah are described with conviction in one text, but there is a shortage of supporting evidence. Further reference is made to him later, on the technicality that he was more a submariner than a diver. As his descent was involuntary he was at best a reluctant pioneer diver. The history of submarine escape, when the submariner may become a diver, is discussed in Chapter 70.

Some claim that Alexander the Great descended in a diving bell during the third century BC. Details of the event are vague, and some of the fish stories attributed to him were spectacular – one fish was said to have taken three days to swim past! It is most unlikely that the artisans of the time could make glass as depicted in most of the illustrations of the 'event'. This may have been a product of artistic licence, or evidence that the incident is based more on fable than fact.

Snorkels – breathing tubes made from reeds and bamboos (now plastic and rubber) – were developed in many parts of the world. They allow a diver to breathe with his/her head under water. Aristotle infers that the Greeks used them, while Columbus reported that the North American Indians would swim towards wild fowl, breathing through a reed and keeping their bodies submerged. They were able to capture the birds with nets, spears or even with their bare hands. The Australian aborigine used a similar approach to hunt wild duck. Various people have 'invented' long hose snorkels; the one designed by Vegetius, dated 1511, blocked the diver's vision and imposed impossible loads on his breathing muscles.

Some have interpreted an Assyrian drawing dated 900 BC as an early diving set. The drawing shows a man with a tube in his mouth, the tube being connected to some sort of bladder or bag. However, this arrangement is more probably a float or life jacket.

Leonardo da Vinci sketched diving sets and fins. One set was really a snorkel that had the disadvantage of a large dead space. Another of his ideas was for the diver to have a 'wine skin to contain the breath', and this was probably the first recorded design of a self-contained breathing apparatus. Da Vinci's drawings appear tentative, so it is probably safe to assume that there was no practical diving equipment in Europe at that time.

Another Italian, Borelli, in 1680, realized that Leonardo was in error and that the diver's air would have to be purified before he breathed it again. Borelli suggested that the air could be purified and breathed again by passing it through a copper tube cooled by sea water. With this concept he had the basic idea of a rebreathing set. It might also be claimed that he had the basis of the experimental cryogenic diving set in which gas is carried in liquid form and purified by freezing out carbon dioxide.

Diving bells were the first successful method of increasing endurance underwater, apart from

snorkels. These consist of a weighted chamber, open at the bottom, in which one or more people could be lowered under water. The early use of bells was limited to short periods in shallow water. Later a method of supplying fresh air was developed. The first fully documented use of diving bells dates from the sixteenth century.

In 1691 Edmund Halley, the English astronomer who predicted the orbit of the comet that bears his name, patented a diving bell which was supplied with air in barrels. With this development diving bells became more widespread, and, they were used for salvage, treasure recovery and general construction work. Halley's bell was supplied with air from weighted barrels, which were hauled from the surface. Dives to 20 metres for up to 90 minutes were recorded. Halley also devised a method of supplying air to a diver from a hose connected to the bell, though the length of hose restricted the diver to the area close to the bell. It is not known if this was successful, but Halley was one of the earliest recorded sufferers of middle ear barotrauma.

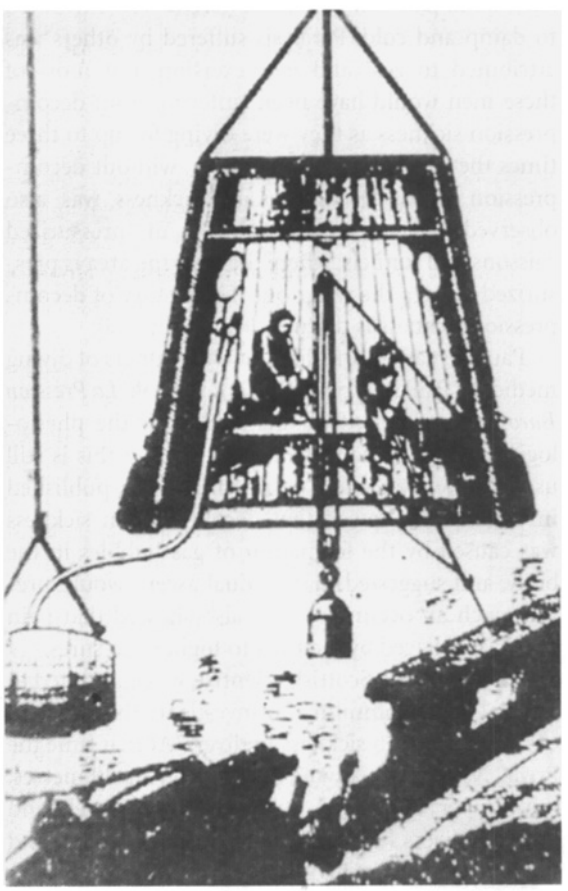

Figure 1.1 *Edmund Halley's diving bell (1691); the weighted barrels of air which were used to replenish the air inside the bell can be clearly seen.*

It is probable that Halley was not entitled to his patent. Swedish divers had devised a small bell, occupied by one man, with a gas supply similar to that later patented by Halley. Between 1659 and 1665, fifty bronze cannon, each weighing over 1000 kg, were salvaged from the *Vasa*, a Swedish warship which had sunk in 30 metres of water in Stockholm harbour. The guns were recovered by divers working from a bell, assisted by ropes from the surface. This task would not be easy for divers, even with the best of modern equipment.

MODERN DIVING EQUIPMENT

The first people to be exposed to a pressure change in a vessel on the surface were patients exposed to higher or lower pressure as a therapy for various conditions. This was the start of hyperbaric medicine, and the origins of diving medical research can also be traced to these experiments.

During the second half of the eighteenth century reliable air pumps were developed that were able to supply air against the pressures experienced by divers. Several people had the idea of using these pumps for diving, and this in turn led to the development of what are now called 'open helmets'. These covered the head and shoulders, and air was pumped down to the diver, the excess air escaping from the bottom of the helmet. The diver could breath because his head and neck were in air, or at least they were until he bent over or fell. If this happened, or if the hose or pump leaked, the helmet flooded and the diver was likely to drown. The Deane family were among the major users of this equipment, and continued to use it up to the time of the Crimean War.

Standard rig or **standard diving dress** was first produced in 1837 by Augustus Siebe, a naturalized Englishman. This equipment consisted of a rigid helmet sealed to a flexible waterproof suit. Air was pumped down from the surface into the helmet, and

Figure 1.2 *Augustus Siebe's first helmet.*

excess air bled off through an outlet valve. The diver could control his buoyancy by adjusting the flow through his outlet valve, and thus the volume of air in his suit. This type of equipment, with a few refinements, is still in use.

Siebe's firm came to be the major constructor, but his role in the design may have been overstated – possibly for the marketing advantages gained by his firm, which marketed the first acceptable equipment of this type. The origins and evolution from open helmet and standard dress are the subject of a recent study by Bevan, who discusses several designs that were developed at the same time with borrowing and stealing of ideas from each other.

Several types of diving suits and a bell were used by the Royal Engineers on dives on the wreck of the *Royal George*, which was a danger to navigation in Spithead anchorage. Indeed, Corporal Jones, who had gained his experience on the wreck of the *Royal George*, was the instructor of the first diving school, set up by the Royal Navy, in 1843. The Siebe suit was found to be greatly superior to the other designs. Siebe's apparatus allowed the diver to bend over, or even lie down without the risk of flooding the helmet. Also, the diver could control his depth easily. A diver in an open helmet had to climb a ladder or rely on his tenders to do this.

Improved versions of the Siebe suit are still in use, some of them made by Siebe Gorman Ltd, the continuation of the firm started by Siebe. In more modern versions the helmet is fitted with communications to allow the diver to confer with another diver or the surface. One of the developments from the Siebe closed helmet was the United States Navy Mark 5 helmet, which probably set a record by being in service for 75 years.

Following the development of these diving suits, decompression sickness was noted in divers. Divers were given fresh dry under-garments because the rheumatic pains that some suffered were attributed to damp and cold. Paralysis suffered by others was attributed to zeal and over-exertion, but most of these men would have been suffering from decompression sickness as they were diving for up to three times the accepted limits for dives, without decompression stops. Decompression sickness was also observed in workers employed in pressurized caissons and tunnels, where the working area is pressurized to keep the water out. The history of decompression sickness is discussed in Chapter 10.

Paul Bert and J.S. Haldane are the fathers of diving medicine. Paul Bert published a text book, *La Pression Barometrique*, based on his studies of the physiological effect of changes in pressure, and this is still used as a reference text despite first being published in 1878. Bert showed that decompression sickness was caused by the formation of gas bubbles in the body, and suggested that gradual ascent would prevent such an occurrence. He also showed that pain could be relieved by a return to higher pressures.

J.S. Haldane, a Scottish scientist, was appointed to a Royal Navy committee to investigate the problem of decompression sickness in divers. At that time the Royal Navy had a diving depth limit of 30 metres, but deeper dives had been recorded. Greek and Swedish divers had reached 58 metres in 1904, and Alexander Lambert had recovered gold bullion from a wreck in 50 metres of water in 1885, but had developed partial paralysis as a result of decompression sickness.

Haldane concluded from Paul Bert's results that a diver could be hauled safely to the surface from 10 metres with no evidence of decompression sickness. He deduced from this that a diver could be surfaced from greater than 10 metres in stages, provided that time was spent at each stage to allow absorbed nitrogen to pass out of the body in a controlled manner. This theory was tested on goats, and then men in chambers. His work culminated in an open-water dive to 64 metres in 1906, and the publication of the first acceptable set of decompression tables. Haldane also developed several improvements to the diving equipment used.

In 1914, US Navy divers reached 84 metres, and the following year they raised a submarine near Hawaii from a depth of 93 metres. This was a remarkable feat considering that the salvage techniques had to be evolved by trial and error. The divers used air, so they were exposed to a dangerous degree of nitrogen narcosis, as well as decompression sickness.

SELF-CONTAINED EQUIPMENT

Scuba (self-contained underwater breathing apparatus) is used to describe any diving set which allows the diver to carry his/her air supply with them. There are several claims to its invention, based on old drawings, the first workable form probably dating from the early nineteenth century. There is a brief report of an American engineer, Charles Condert, who made a scuba in which the air was stored in a copper pipe worn around his body. The gas was released into a hood that covered the upper half of his body. Accumulation of carbon dioxide was controlled by allowing the respired gas to escape through a small hole, whereupon it was replaced by fresh gas from the storage pipe. Unfortunately, Condert died while diving with his equipment in the East River in 1831.

Another early development was the Rouquayrol and Denayrouze device of 1865. This set was supplied with air from the surface in the same manner as the Siebe closed helmet suit, and was fitted with an air reservoir so that the diver could detach himself from the air hose for a few minutes. The endurance, as a scuba, was limited by the amount of air in the reservoir.

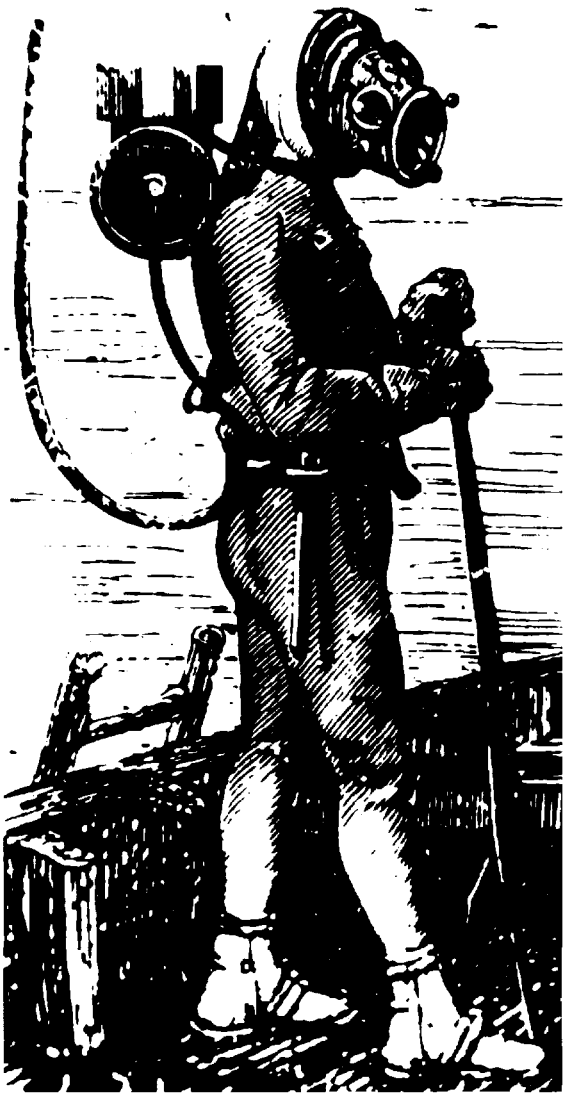

Figure 1.3 *The aerophore, devised by Rouquayrol and Denayrouze (1865): this device was widely used and was an important milestone in the development of the modern scuba.*

The first successful scuba equipment with an air supply appears to have been developed and patented in 1918 by Ohgushi, a Japanese. His system could be operated with a supply of air from the surface, or as a scuba with an air supply cylinder carried on the back. The diver controlled his air supply

by triggering air flow into his mask with his teeth. Another scuba was devised by Le Prieur in 1933; in this set the diver carried a compressed air bottle on his chest and released air into his face mask by opening a tap.

In 1943, Cousteau and Gagnan developed the first scuba incorporating an automatic **demand valve** to release air as the diver inhaled. This valve was triggered by the diver's breathing, so the diver no longer had to operate a tap to obtain each breath of air. In developing this valve, which was pressure-compensated so that changes in depth did not affect its function, Cousteau and Gagnan invented the scuba as we know it today. It was an adaptation of a reducing valve Gagnan had evaluated for use in gas-powered cars.

Closed-circuit oxygen sets were developed during the same period as the modern scuba. In these rebreathing sets the diver is supplied with oxygen, and the carbon dioxide is removed by absorbent. These sets are often called scuba, but they may be considered separately because of the difference in principles involved. The first successful rebreathing set was designed by an Englishman, H.A. Fleuss, in 1878. This was an oxygen set in which carbon dioxide was absorbed by rope soaked in caustic potash.

Because of the absence of lines and hoses from the diver to the surface the set was used in flooded mines and tunnels where the extra mobility, compared to the standard rig, was needed. Great risks were taken with this set and its successors, because the work of Paul Bert on oxygen toxicity was not widely known. This equipment was the precursor of oxygen sets used in clandestine operations in both world wars, and of other sets used in submarine escape, fire-fighting and mine rescue.

MODERN MILITARY DIVING

The military use of divers in modern warfare was, until 1918, largely restricted to the salvage of damaged ships, clearing of channels blocked by wrecks, and assorted ships' husbandry duties. One significant clandestine operation conducted during the First World War was the recovery of code books and minefield charts from a sunken German submarine. This was of more significance as an intelligence operation, although the diving activity was also kept secret.

During the First World War, Italy developed a human torpedo or chariot that was used in 1918 to attack an Austrian battleship in Pola Harbour. The attack was a success in that the ship was sunk, but unfortunately it coincided with the fall of the Austro-Hungarian Empire and the ship was already in friendly hands! The potential of this method of attack was noted by the Italian Navy. They put it to use in the Second World War with divers wearing oxygen rebreathing sets as underwater pilots. In passing it is interesting to note that the idea of the chariot was suggested to the British Admiralty in 1909, and Davis took out patents on a small submarine and human torpedo controlled by divers in 1914. This was pre-dated by a one-man submarine designed by J.P. Holland in 1875.

Diving played a greater part in offensive operations during the Second World War. Exploits of note include those of the Italian Navy, which used divers riding modified torpedos to attack ships in Gibraltar and Alexandria. After a series of unsuccessful attempts, with loss of life, they succeeded in sinking several ships in Gibraltar harbour in mid-1941. Later that year three teams managed to enter Alexandria harbour and damage two battleships and a tanker. Even Sir Winston Churchill, who did not often praise his enemies, said they showed 'extraordinary courage and ingenuity'. Churchill had previously been responsible for rejecting suggestions that the Royal Navy use similar weapons.

In Gibraltar a special type of underwater war evolved. The Italians had a secret base in Spain, only six miles away, and launched several attacks that were opposed by British divers who tried to remove the Italian mines before they exploded.

Divers from the allied nations made several successful attacks on enemy ships, but their most important offensive role was in the field of reconnaissance and beach clearance. In most operations the divers worked from submarines or small boats. They first surveyed the approaches to several potential landing sites. After a choice had been made they cleared the obstructions that could impede the landing craft. One of the more famous

exploits of an American diving group was to land unofficially and leave a 'Welcome' sign on the beach to greet the US Marines spearheading the invasion of Guam. The British clearance diver and the American SEALs evolved from these groups. The clearance divers get their name from their work in clearing mines and other obstructions from recaptured ports, a role they repeated during and after the Gulf War.

The research back-up to these exploits was largely devoted to improvement of equipment and the investigation of the nature and onset of oxygen toxicity (see Chapter 17). This work was important because most of these offensive operations were conducted by divers wearing oxygen breathing apparatus. The divers who acted as subjects were the unsung heroes of the work. This group of scientists, sailors and conscientious objectors deliberately and repeatedly suffered oxygen toxicity in attempts to understand the condition.

Oxygen/nitrogen mixtures were first used for diving by the Royal Navy in conjunction with standard diving rig. This was based on an idea proposed by Sir Leonard Hill and developed by Siebe, Gorman and Co. Ltd. The advantage of this equipment was that by increasing the ratio of oxygen to nitrogen in the breathing gas one can reduce or eliminate decompression requirements. It is normally used with equipment in which most of the gas is breathed again after the carbon dioxide has been removed. This allows reduction of the total gas volume required by the diver.

During the Second World War this idea was adapted to a self-contained semi-closed rebreathing apparatus which was first used extensively by divers clearing mines. This development was conducted by the British Admiralty Experimental Diving Unit in conjunction with Siebe Gorman & Co. Ltd. The change to a self-contained set was needed to reduce the number of people at risk from accidental explosions in mine-clearing operations. The reduction, or elimination of decompression time was desirable in increasing the diver's chances of survival if something went wrong. The equipment was constructed from non-magnetic materials to reduce the likelihood of activating magnetic mines and was silent during operation, for work on acoustically triggered mines.

DEEP DIVING

The search for means to allow man to descend deeper has been a continuing process. By the early twentieth century deep diving research had enabled divers to reach depths in excess of 90 metres; at this depth the narcosis induced by nitrogen incapacitated most men.

After the First World War the Royal Navy diving research tried to extend their depth capability beyond 60 metres. Equipment was improved, the submersible decompression chamber was introduced, and new decompression schedules were developed. These used periods of oxygen breathing to reduce decompression time. Dives were made to 107 metres, but nitrogen narcosis at these depths made such dives unrewarding and dangerous.

Helium diving resulted from a series of American developments. In 1919 a scientist, Professor Elihu Thompson, suggested that nitrogen narcosis could be avoided by replacing the nitrogen in the diver's gas supply with helium. At that stage, the idea was not practical because helium cost over $US 2000 per cubic foot. Later the price dropped to about 3 cents per cubic foot, following the exploitation of natural gas supplies which contained helium.

Research into the use of helium was conducted during the 1920s and 1930s, and by the end of the 1930s divers in a compression chamber had reached a pressure equal to a depth of 150 metres, while a dive to 128 metres was made in Lake Michigan. Between the two world wars the USA had a virtual monopoly on the supply of helium, and so dominated research into deep diving.

Hydrogen diving, using hydrogen in gas mixtures for deep diving, was first tried by Arne Zetterstrom, a Swedish engineer. His pioneering work on the use of hydrogen in a diver's gas mixture is still being developed. He demonstrated that hypoxia and risks of explosion could be avoided if the diver used air from the surface to 30 metres, changed to 4% oxygen in nitrogen, and then changed to 4% or less oxygen in hydrogen. In this manner the diver received adequate oxygen and the formation of an explosive mixture of oxygen and hydrogen was prevented.

In 1945, Zetterstrom dived to 160 metres in open water, but unfortunately an error was made

by the operators controlling his ascent. They hauled him up too fast and he died from hypoxia and decompression sickness. The error was accidental and was not related to his planned decompression schedule.

The cheapness of hydrogen compared to helium, and the probability of a helium shortage in the future, may mean that hydrogen will be more widely used in deep dives. French workers have reported good results using a mixture of hydrogen and helium as diluting gas.

Other European workers have followed Zetterstrom with radical approaches to deep diving. The Swiss worker, Keller performed an incredible 305 metres (1000 ft) dive in the open sea in December 1962. He was assisted by Buhlmann who has developed and tested several decompression tables.

Modern gas mixture sets have evolved as the result of several forces. The price of helium has become a significant cost, and this – combined with a desire to increase the diver's mobility – has encouraged the development of more sophisticated mixed gas sets. The most complex of these have separate cylinders of oxygen and diluting gas. The composition of the diver's inspired gas is maintained by the action of electronic control systems which regulate the release of gas from each cylinder. The first of these sets was developed in the 1950s, but they are still being refined and improved.

Modern air or gas mixture helmets have several advantages compared to the older equipment. A demand system reduces the amount of gas used, compared to the standard rig. The gas-tight sealing system reduces the chance of a diver drowning by preventing water inhalation. The primary gas supply normally comes to the diver from the surface or a diving bell, and may be combined with heating and communications. A second gas supply is available from a cylinder on the diver's back. The Americans Bob Kirby and Bev Morgan have led the way with a series of helmet systems, their Superlite 17 being used with either compressed air or gas mixtures. When used with gas mixtures it can also be modified to use gas supplied from a diving bell and return the exhaled gas to the bell. This requires two pumps (called a push–pull system) to move the gas to and from the diver. These helmets can operate to depths over 450 metres.

Saturation diving is probably the most important development in commercial diving since the Second World War. Behnke, an American diving researcher, suggested that caisson workers could be kept under pressure for long periods and decompressed slowly at the end of their job rather than undertake a series of compressions, and risk decompression sickness after each.

A US Navy Medical Officer, George Bond and others adopted this idea for diving, the first of these dives involving tests on animals and men in chambers. In 1962 Robert Stenuit spent 24 hours at 60 metres in the Mediterranean Sea off the coast of France.

Despite the credit given to Behnke and Bond it might be noted that the first people to spend long periods in an elevated pressure environment were patients treated in a hyperbaric chamber. Between 1921 and 1934 an American, Dr Orval Cunningham, pressurized people to a pressure equal to a dive to 20 metres for up to five days and decompressed them in two days.

Progress in saturation diving was rapid with the French-inspired 'Conshelf' experiments and the American 'Sealab' experiments seeking greater depths and durations of exposure. In 1965, the former astronaut Scott Carpenter spent a month at 60 metres, while two divers spent two days at a depth equivalent to almost 200 metres. Unfortunately, people paid for this progress, as lives were lost and there has been a significant incidence of bone necrosis induced by these experiments.

In saturation diving systems, the divers either live in an underwater habitat or in a chamber on the surface. In the latter case, another chamber is used to transfer them under pressure to and from their work site. Operations can also be conducted from small submarines or submersibles, with the divers operating from a compartment that can be opened to the sea. They can either move to a separate chamber on the submarine's tender or remain in the submarine for their period of decompression. The uses of this equipment offers several advantages. The submarine speeds the diver's movement around the work site, provides better lighting and carries extra equipment. Also, a technical expert who is not a diver can observe and control the operation from within the submarine.

Operations involving saturation dives have

become routine for work in deep water, the stimulus for this work being partly military and partly commercial. Divers work on the rigs and pipelines needed to exploit oil and natural gas fields. The needs of the oil companies have resulted in strenuous efforts to extend the depth and efficiency of the associated diving activities. Diving firms are now prepared to sign contracts that may require them to work at over 500 metres.

Man is pursuing other avenues in his efforts to exploit the sea. **Armoured diving suits** withstand the pressure exerted by the water and allow the diver to avoid the hazards of increased and changing pressures. In effect, the diver becomes a small submarine. The mobility and dexterity of divers wearing earlier armoured suits were limited and they were not widely used. The newer suits such as the British 'JIM' and the Canadian suit made by Hard Suits have become accepted pieces of diving equipment. They can be fitted with claws for manipulating equipment. The Hard Suit product is of interest because its joint system is more mobile than that of JIM, reducing the work input needed for movement. The Hard Suit and the WASP, a compromise between a diver and a one-man submarine, have propellers to aid movement, and the designers are now aiming for depths beyond 660 metres (2000 ft).

Liquid breathing trials, in which the lungs are flooded and the body supplied with oxygen in solution have only been conducted in laboratories and hospitals. The potential advantages of breathing liquids are the elimination of decompression sickness as a problem, freedom to descend to virtually any depth, and the possibility of the diver extracting the oxygen dissolved in the water.

RECREATIONAL DIVING

Amateur diving started with breath-hold diving, mainly by enthusiasts in Italy and the south coast of France, who were keen spear fishermen. This was also the area where compressed air scuba diving

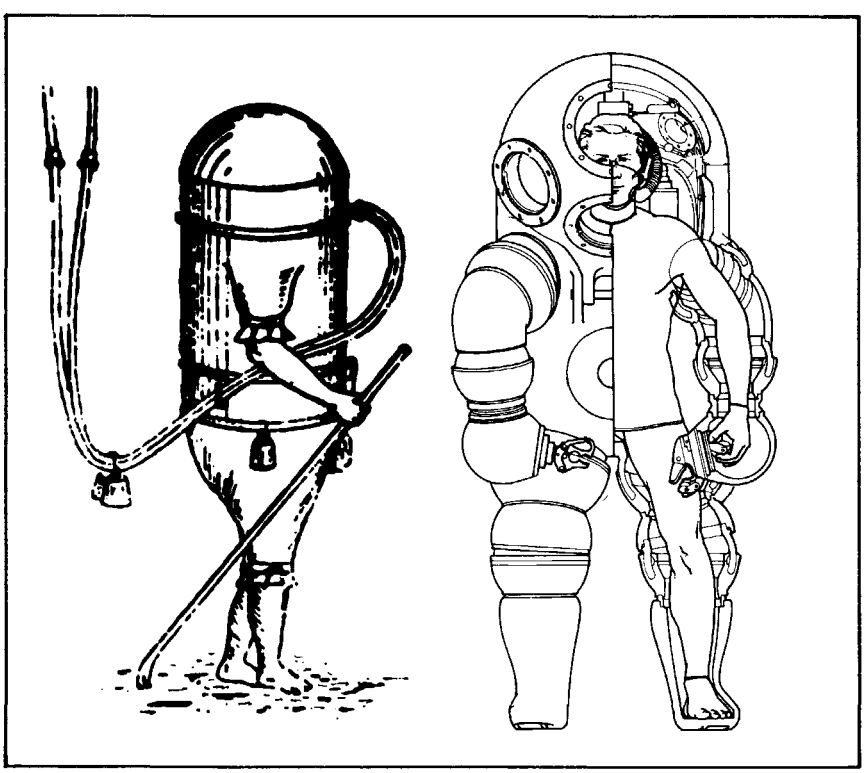

Figure 1.4 *Armoured diving suits, past and present (JIM).*

developed as a result of the work of Cousteau and co-workers. As a sport, diving rapidly spread to Britain and the USA and the rest of the world.

From this beginning diving has become a sport that is often combined with tourism and photography. Others explore caves and wrecks and seek the excitement that deeper and further penetrations provide. Special interest groups such as cave and technical divers have developed, and in some areas are the modern pathfinders. These groups and their problems are discussed in greater detail later.

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2

Physics and physiology

JOHN PENNEFATHER

INTRODUCTION

A basic knowledge of the physics and physiology of diving is essential to understand most of the medical problems encountered. Aspects of physics and physiology that have a wide application to diving are discussed in this chapter.

Some of the basic physiological implications are also mentioned, but most aspects of diving physiology and pathophysiology are relegated to the relevant chapters on specific diving disorders.

PRESSURE, GASES AND DIVING

On the surface of the earth, we are exposed to the pressure exerted by the atmosphere; this is called the atmospheric or barometric pressure. Most people regard this pressure as being due to the mass of the atmosphere pressing down on them. A flaw in this argument is that the pressure remains in a bottle after it is sealed, although its contents are contained and are no longer exposed to the column of air above. The physically correct explanation is that atmospheric pressure is generated by collisions of the molecules of gas in accordance with the kinetic theory of gases. Either explanation is acceptable for the following discussion.

An observer finds that the pressure decreases as he moves upward through the atmosphere, and increases as he moves down into a mine, or into the sea. At the top of Mount Everest the atmospheric pressure is about 40 per cent of that at sea level. Because water is much heavier than air, the pressure changes experienced by divers are much greater than those encountered by climbers or aviators.

Pressure is measured in a variety of units from either of two reference points. It can be expressed with respect to a vacuum, i.e. zero pressure – this reading is called an **absolute pressure**. The second method measures pressures above or below local pressure – these readings are called **gauge pressures**. At sea level the absolute pressure is 1 atmosphere

(1 ATA), and the gauge pressure is 0. These units are commonly abbreviated to ATA and ATG. A common example of an absolute pressure is the barometric pressure used by weather forecasters, while an example of a gauge pressure reading is that of blood pressure.

With descent in water, pressure increases. For each 10 metres depth in sea water, the pressure increases by 1 atmosphere, starting from 1 ATA or 0 ATG at the surface. The gauge pressure remains 1 atmosphere less than the absolute pressure, e.g. at 10 metres, the pressure is 2 ATA and 1 ATG. At 90 metres, the pressure is 10 ATA and 9 ATG.

Pressure units

Because diving involves facets of engineering and science it is plagued with many units of pressure. These include absolute and gauge atmospheres, Pascals and multiples such as the kilopascal, feet or metres of seawater, pounds per square inch, bars and torrs, and several other rarer units. Conversions for the more commonly used units are listed in Table 2.1.

Pressure and the diver's body

Many people have difficulty in understanding why the pressure of the water does not crush the diver. The answer to this problem may be considered in two parts.

Table 2.1 *Pressure conversion factors (commonly used approximations shown in brackets)*

1 ATMOSPHERE
- = 10.08 (10) metres sea water (msw)
- = 33.07 (33) feet sea water
- = 33.90 (34) feet fresh water
- = 101.3 kilopascals (kPa) = 0.1013 mega pascals (MPa)
- = 1.033 kg/cm^2
- = 14.696 (14.7) lbs/in^2
- = 1.013 bars
- = 760 millimetres mercury (mmHg) = 760 Torr
- = 1 ATA

Actual conversions from sea water depth to ATA depends on salinity and temperature.) A complete conversion metrix is provided at the end of the chapter.

The solid and liquid parts of the body are virtually incompressible, so a pressure applied to them does not cause any change in volume: the pressure is transmitted through them. After immersion, the increased pressure pushes on the skin, which in turn pushes on the tissues underneath and so on through the body until the skin on the other side is pushed back against the water pressure. So the system remains in balance. Readers with a background in physics will recognize the application of **Pascal's principle**.

The effect of pressure on the gas spaces in the diver's body is more complex. The applied pressure does not cause any problems if the pressure in the gas space is close to that of the surrounding water. There is, for example, no physical damage to diver's lungs if the air space was exposed to a pressure of a hundred metres of water pressure – provided that this pressure was balanced by the pressure exerted by surrounding water acting on the walls of the lung to balance any tendency of the lungs to expand. If the lungs were exposed to a pressure of a few metres water more than the surrounding atmospheric tissue, they would over-expand and burst.

Water pressure and lung inflation

Immersion up to the neck in water reduces vital capacity by about 10 per cent (see Fig. 2.1 for lung volumes). This is caused in part by the hydrostatic pressure of the water compressing the thorax. With immersion, there is also a loss of gravitational effects, which reduces the volume of blood in lower (mainly leg) veins and increases thoracic blood volume. This in turn reduces the compliance of the lungs.

When using breathing equipment, pressure at the point from which the gas is inhaled can be different to the pressure at the chest. For example, if upright in the water, a scuba diver is inhaling air released at mouth pressure, but if a snorkel diver is inhaling air from the surface this is at surface pressure. In both these cases the air is at a lower pressure than the diver's lungs, and this reduces the amount of air that can be inhaled because part of the inhalation force is used in overcoming this pressure. Conversely, when swimming down, a diver whose air is released at mouth pressure can inhale to greater than normal

Figure 2.1 *Lung volumes and intrapulmonary pressure. The various components of lung volumes are labelled on the left. On the right, the relationship between lung volume, airway pressure and the maximum effort that can be made for inhalation and exhalation of air are plotted. Curve 1 is the volume change during quiet breathing, and curve 2 is the volume change during a maximum inhalation starting at the residual volume. IRV = inspiratory reserve volume; TV = tidal volume; ERV = expiratory reserve volume; RV = residual volume; VC = vital capacity. (Redrawn from Lanphier and Camporesi (1982) with permission.)*

vital capacity, but could not exhale to the normal residual volume. This is because, in this orientation, the water pressure is helping to inflate the lungs.

Pressure and volume changes

When a diver descends, the increased pressure of the water compresses air in his gas spaces. This is one of the many aspects of diving medicine that is concerned with the relationship between pressure change and change of gas volume. The relationship between changes in volume of a gas and the pressure applied to it are described by **Boyle's Law**. This states that, *if the temperature remains constant, the volume of a given mass of gas is inversely proportional to the absolute pressure.* This means that the absolute pressure multiplied by volume has a constant answer, the value of the constant changing with the mass of gas considered. To a mathematician, this means that $P \times V = K$ or $P_1 \times V_1 = P_2 \times V_2$ where P and V are pressure and volume. For example, 10 litres of gas at sea level pressure (1 ATA) will be compressed to:

- 5 litres at 2 ATA (10 metres);
- 2 litres at 5 ATA (40 metres); and
- 1 litres at 10 ATA (90 metres).

During ascent into the atmosphere the reverse happens and the gas expands – the 10 litres of air would expand to 20 litres at 0.5 ATA (an altitude of about 5000 metres or 18 000 feet) to 40 litres at 0.25 ATA (an altitude of about 10 300 metres or 33 400 feet).

> *Gases volumes expand when pressure decreases, and contract when pressure increases.*

The volume of a mass of gas in a flexible container decreases with pressure or depth increase and expands during ascent or pressure reduction (Fig. 2.2). It should be noted that volume changes are greatest near the surface. Conversely, gas has to be added if the volume of a container or gas space is to remain constant as the pressure is increased. The effects of this law are important in many aspects of diving medicine.

During descent, the increasing pressure in the water is transmitted through the body fluids to the tissue surrounding the gas spaces and to the gas spaces. The pressure in any gas space in the body should increase to equal the surrounding pressure. In the lungs, during breath-hold dives, this is accompanied by a decrease in lung volume. Air should enter cavities with rigid walls, such as the sinuses or

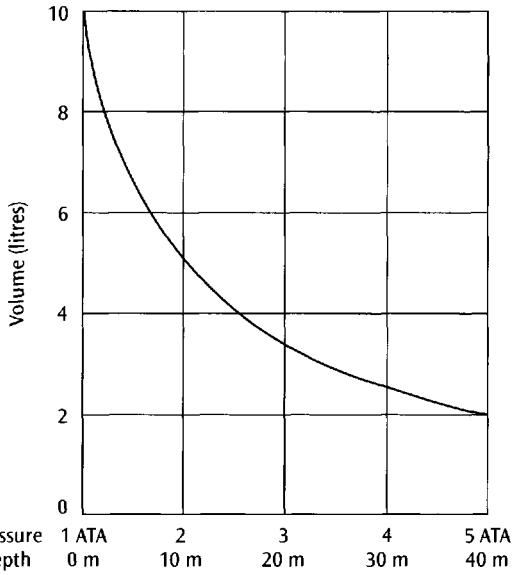

Figure 2.2 *Boyle's law: while breathing under water, the diver's respiratory volume is about the same as it would be if he worked at the same rate on the surface. Due to the increase in density of this breathing gas under increased pressure, he must move a greater mass of gas with each breath. In some situations, this physical effect can limit the diver's capacity to do work.*

the middle ear. If air entry does not take place to equalize pressures, then a pressure difference between the space and the surrounding tissue will develop, with the pressure in the gas space being less than the surrounding tissue. The result is **tissue distortion** and damage such as congestion, oedema or haemorrhage. During ascent, gas spaces expand and can rupture the tissue containing them unless the gas can escape. Pressure changes in the middle ear can also result in rupture of the tympanic membrane.

During ascent, as the pressure decreases, gas within body spaces will expand. Unless gas is vented from the space, the expanding gas will exert pressure on the surrounding tissue and will eventually damage it.

The same volume changes with pressure occur in bubbles in tissue or blood. Again, the volume changes are greatest close to the surface. An injury caused by pressure change is called **barotrauma**.

> *Barotrauma is the general name for an injury caused by pressure change.*

Respiration in water and under pressure

While breathing air underwater, the diver's respiratory volume is about the same as it would be if he worked at the same rate on the surface. A consequence of this is that a cylinder that contains enough air for 100 minutes at 1 ATA would last about 50 minutes at 2 ATA (10 metres), or 20 minutes at 5 ATA (40 metres) for dives with the same energy expenditure. This is because the gas in the cylinder expands to a smaller volume when it is released at depth than it would if used at the surface. A cylinder that contains 5000 litres of gas if it is released at the sea surface contains only 1000 litres of gas if it is released at 5 ATA, or 40 metres. A diving medical officer needs to keep this in mind when estimating the amount of gas needed for any task or therapy.

With depth, gas is compressed and there is an increase in density of the gas because there are more molecules in a given space. So, at depth, a diver must move a greater mass of gas with each breath. This requires greater effort and hence an increase in the work of breathing. In some situations, this can limit the capacity of the diver to do work.

The density can be reduced by replacing nitrogen with a lighter gas such as helium. For example, the density of air at 1 ATA is about 1.3 kg/cubic metre. At 10 ATA the density of air would be about 13 kg/cubic metre. The use of lighter gas helps to reduce density. For example at 40 ATA the density of a 1 per cent oxygen + helium mixture is 6.7 kg/cubic metre.

As the density of a gas increases there is an increased tendency for the flow to become turbulent. This causes a further increase in the energy used in breathing.

These factors can lead to fatigue of the inspiratory muscles and a reduction in maximum breathing capacity and work output. To minimize this load, the body responds by using less gas for a given work load, but this can result in the development of hypercapnia. Continued exposure to dense gas, as is encountered in deep dives, may cause an adaptive response.

Temperature and volume changes

Charles' law states: *If the pressure is constant, the volume of a mass of gas is proportional to the absolute temperature.*

The absolute temperature (A°) is always 273° more than the centigrade temperature. A more useful expression of the law is:

$$\frac{V_1}{T_1} = \frac{V_2}{T_2} \text{ or } \frac{V}{T} = K$$

where V_1 is the volume of a mass of gas at temperature T_1°A and V_2 is its volume after the temperature has changed to T_2°A. This law has much less relevance to diving medicine than Boyle's law; however, it should be remembered when considering gas volumes and how they may change.

Boyle's and Charles' laws may be combined and used if temperature and pressure both change – from P_1 and T_1 to P_2 and T_2 with a volume change from V_1 to V_2. The combined laws can be expressed as the **universal gas equation:**

$$\frac{P_1 \times V_1}{T_1} = \frac{P_2 \times V_2}{T_2}$$

A temperature–pressure problem that often causes discord can be used to illustrate the use of this equation. This is the effect of temperature on the pressure in a gas cylinder. A diver might ask to have his compressed air cylinder filled to 200 ATA. The gas compressor heats the gas so that the cylinder may be charged with gas at 47°C. However, when the diver enters the water at 7°C he is incensed to find he only has 175 ATA in his cylinder. In this case, $V_1 = V_2$ because the cylinder is rigid, and the pressure falls as the gas cools.

$$47°C = 320°A, 7°C = 280°A, V_1 = V_2$$

$$\frac{200 \times V_1}{320} = \frac{P_2 \times V_2}{280}$$

$$P_2 = 175 \text{ ATA}$$

So, the reduced pressure is a result of temperature change, and not due to a leaking valve or fraud by the air supplier.

Partial pressures in gas mixtures

Dalton's law states that, *the total pressure exerted by a mixture of gases is the sum of the pressures that would be exerted by each of the gases if it alone occupied the total volume.* The pressure of each constituent in a mixture is called the **partial pressure.** In air, which is approximately 80 per cent nitrogen and 20 per cent oxygen, the total pressure at sea level (1 ATA) is the sum of the partial pressures of nitrogen, 0.8 ATA, and oxygen, 0.2 ATA. At 2 ATA (10 metres) these partial pressures will rise to 1.6 and 0.4 ATA, respectively.

The partial pressures of breathing gases can be manipulated to the diver's advantage. For example, the composition of the gas breathed may be modified to reduce the chance of decompression sickness by decreasing the percentage of inert gas in the mixture.

Undesirable effects can also occur. Air from an industrial area may contain over 0.3 per cent carbon dioxide and 0.002 per cent carbon monoxide. At high pressures, both contaminants could be toxic unless measures were taken to remove them before use.

It may be necessary to combine Boyle's and Dalton's laws in calculations. For example, it may be decided that a diver should be given a mixture with a partial pressure of 0.8 ATA oxygen and 1.2 ATA nitrogen in a recompression chamber pressurized to 2 ATA. If oxygen and air are the only gases available, the gas laws can be used to calculate how to prepare a cylinder charged with the correct gas mixture. The mixture will need to be 40 per cent oxygen and 60 per cent nitrogen (Dalton's law).

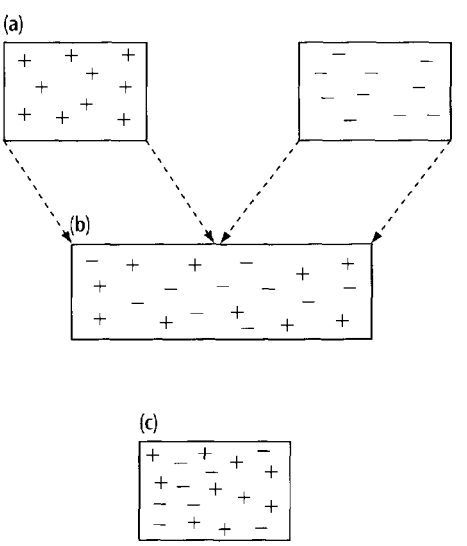

Figure 2.3 *Dalton's law: (a) two spaces each at 1 ATA; (b) total pressure 1 ATA, 0.5 ATA of each component of the mixture; (c) total pressure 2 ATA, 1 ATA of each component of the mixture.*

If the gas is to be prepared in a cylinder charged to 200 ATA it should contain 120 ATA of nitrogen (60 per cent of 200). If this is to be obtained from compressed air (assumed to be 80 per cent nitrogen in this exercise) it will be necessary to put 150 ATA of compressed air into the cylinder (30 ATA of oxygen + 120 ATA of nitrogen) with 50 ATA of oxygen.

This simple mixing process cannot be used as successfully with helium mixtures. At high pressures, helium does not follow the predictions of Boyle's law accurately as it is less compressible than the ideal gas described by Boyle's law. Mixing can be conducted by making an allowance for this, or by putting a calculated *weight* of each gas in the cylinder.

Solution of gases in liquids

Henry's law states that *at a constant temperature the amount of a gas that will dissolve in a liquid is proportional to the partial pressure of the gas over the liquid.* This law implies that an equilibrium is established with each gas passing into and out of any solution in contact with it. At sea level (1 ATA), a man's body tissues contain about 1 litre of gaseous nitrogen in solution. If he dived to 10 metres and breathed air at 2 ATA, more gas would dissolve and he would eventually reach equilibrium again, and have twice as much nitrogen in solution in his body. The time taken for any inert gas to reach a new equilibrium depends on the solubility of the gas in the tissues and the rate of gas supplied to each tissue.

When the total pressure (or the partial pressure of a particular gas) is reduced, gas must pass out of solution. If a rapid total pressure drop occurs, a tissue may contain more gas than it can hold in solution. In this situation bubbles may form and cause decompression sickness.

The physiological effects of the solubility of gases are also relevant in nitrogen narcosis and oxygen toxicity.

It should be noted that each gas has a different solubility, and the amount of any gas that will dissolve in a liquid depends on the liquid. For example, carbon dioxide is very soluble in water compared to other common gases. Beer aerated with compressed air instead of carbon dioxide would have far fewer bubbles. Nitrogen is more soluble in fats and oils than in aqueous solutions.

Henry's law is also time-dependent – it takes time for gases to enter and leave solution or form bubbles. If this was not so, champagne would go flat as soon as the cork was popped.

At depth, a diver breathing air absorbs nitrogen in accord with Henry's law, the amount depending on depth and time of exposure. When he surfaces, the excess nitrogen must pass from the body. If it is eliminated from solution through the lungs there will not be any complications. In some cases the nitrogen comes out of solution in the blood or tissues forming bubbles and may cause **decompression sickness** (DCS).

Gas movement in body tissues

Gas transfer from the lungs to the tissues is dependent on the cardiovascular circulation and the gas supplied to a portion of tissue depends on the blood perfusing it. In a permeable substance, such as body

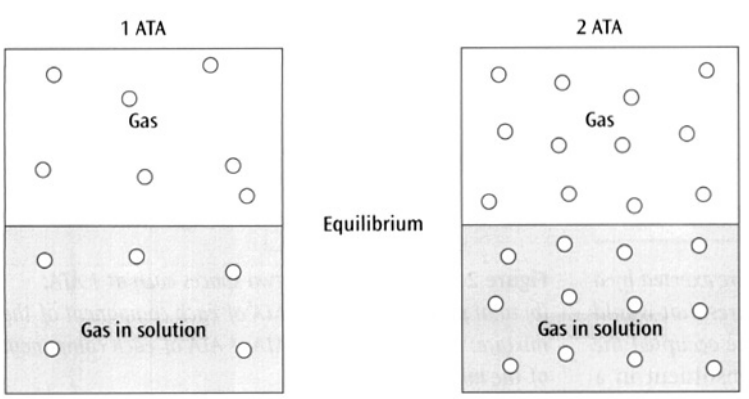

Figure 2.4 *Henry's law.*

tissues, gas molecules can migrate by diffusion. That is, gas molecules dissolve in the tissue fluids and tend to move from areas of high to low partial pressure until the partial pressure of the dissolved gas is uniform, a process which can take hours. It is the dissolved gas pressures that tend to equilibrate, not the number of gas molecules. If a gas is twice as soluble in one tissue compared to another, then twice as many molecules will be in the first tissue to produce the same partial pressure in the tissue. This information can be estimated from the solubility coefficients of the gas in the components of the tissue.

The rate of gas movement between the two points depends on several factors. The difference in partial pressure and the distance between the two points may be combined into a **concentration gradient**. The other major factor is the permeability of the tissue, an expression of the ease of gas movement. A large partial pressure between two points that are close together (a steep gradient) and a greater permeability both increase the rate of gas transfer.

Metabolic gas exchange

In divers, gas exchange mechanisms are basically the same as at normal pressure. Oxygen diffuses down a concentration gradient from the lungs to the tissues, and the carbon dioxide gradient is normally in the opposite direction. The exchange of inert gases becomes important, and there are changes in the finer details of metabolic gas exchange.

With increasing depth there is an increase in the partial pressures of the constituents of the breathing mixture in accordance with Dalton's law. This causes higher alveolar pressures and arterial pressures of the inhaled gases.

Elevated pressures of oxygen facilitates oxygen transport, but it may interfere with the elimination of carbon dioxide in two ways: First, by the depression of respiration induced by high arterial oxygen tensions; and second, by direct interference with the transport of carbon dioxide. When the inspired oxygen partial pressure is elevated, there is an increase in oxygen transport in solution in the plasma (Henry's law). When inhaling oxygen at a partial pressure above 3 ATA all the oxygen requirement may be carried in solution. If this happens, the haemoglobin may be still saturated with oxygen in the venous blood. This stops the transport of carbon dioxide in the form of carbaminohaemoglobin.

The result is an increased tissue carbon dioxide level, and in some situations there may also be an increase in the inspired carbon dioxide pressure. Causes include the external deadspace of the equipment, inadequate ventilation or failure of the absorbent system.

There is a tendency for experienced divers to be less sensitive to elevated carbon dioxide partial pressures. This reduces the total ventilation requirement during working dives. Elevated arterial carbon dioxide levels increase susceptibility to oxygen toxicity, decompression sickness and nitrogen narcosis. For these reasons it is desirable to control the factors that cause carbon dioxide retention.

> *Diving is associated with a tendency to retain carbon dioxide.*

Inert gas exchange

The topic of inert gas exchange is considered in the chapters on DCS, and so to avoid duplication the topic is not considered in detail here. As indicated above, increased total pressure is usually accompanied by an increase in nitrogen pressure (Dalton's law), causing gas transfer to the body tissues. When pressure is reduced at the end of the dive the transfer is reversed. If there is an excess of gas then it can come out of solution as bubbles, and these are the cause of DCS. If bubbles do occur they are also subject to the same physical laws – their size decreases if the pressure is increased and gas enters or leaves them depending on the concentration gradients of gases.

BUOYANCY

Archimedes' principle states that *any object, wholly or partially immersed in liquid, is buoyed up by a force equal to the weight of liquid displaced.* A diver is an

object immersed in water, and so is affected by this principle as it determines the effort he must employ in order to dive. If a diver weighs less than the weight of water he displaces, he will tend to float to the surface – he has **positive buoyancy**, which makes descent difficult. However, if he weighs more than the weight of water he displaces he has negative buoyancy, which will assist descent and make ascent harder.

A diver can change buoyancy in several ways. If he wears a weight belt he increases weight by a significant amount and displaces a little more water, so he will decrease his buoyancy. If the diver displaces more water he will increase buoyancy. This can be done by retaining more air in the lungs at the end of each breath. Suits worn by divers increase buoyancy because they trap air. A buoyancy compensator (BC) is a device used to control buoyancy; it has an air space that the diver can inflate or deflate to make him positive, negative or neutrally buoyant as needed.

An interesting combination of the effects of Boyle's law and Archimedes' principle is shown by the changes in buoyancy experienced by a diver wearing a BC or a compressible suit. If slightly positively buoyant at the surface with air in the BC, the diver will experience some difficulty in descending. As he descends he will pass through a zone where he is neutrally buoyant, and if he descends further he becomes negatively buoyant. The increased pressure reduces the volume of gas in the vest or suit, the volume of fluid displaced, and hence buoyancy.

The weight of scuba cylinders decrease as gas is consumed from them, and this will lead to an increase in buoyancy. An empty cylinder weighs 1–2 kg less than a full one.

Immersion creates a condition resembling the gravity-free state experienced by astronauts. In air, a standing person has a pressure gradient in the circulation, where the hydrostatic pressure is greatest at the feet and least at the head. For an immersed diver, the hydrostatic gradients in the circulatory system are almost exactly counterbalanced by the ambient water pressure. This reduces the volume of pooled blood in the leg veins. Also, peripheral vasoconstriction will occur in response to any cold stress. These changes result in an increase in central blood volume, leading to water diuresis and subsequent haemoconcentration and decreased plasma volume.

The effect of haemoconcentration on normal dives is not major except that it gives divers a physiological excuse for a well-developed thirst. Urine production rates of over 300 ml/hour cause problems for divers trying to keep their dry suit dry!

The other effect of increased central blood volume is on cardiac performance, there being an increase in cardiac output as a result of increased stroke volume.

ENERGY EXPENDITURE

Measurements of energy expenditure, while swimming on the surface and underwater, have been made using indirect calorimetry, and by prediction from heart rate. These results show that oxygen consumption underwater of over 3 l/min is possible, and values over 2 l/min are quite common. The diver's energy expenditure when inactive may be lower than found on land, presumably because the absence of gravitational effects reduce the energy required to maintain posture underwater.

Typical gas consumption and energy expenditure levels are: for a slow swim (0.5 knots) the diver would have an air consumption of 20 l/min and an oxygen consumption of 0.8 l/min; a swim of 0.8 knots would cause an air consumption of 30 l/min and an oxygen consumption of 1.4 l/min; a fast swim (1.2 knots) would cause an oxygen consumption of about 3 l/min and an air consumption of 50 l/min (air consumption measured at the depth the diver was swimming and oxygen consumption at 1 ATA).

Although increased gas density increases the work of breathing, investigations have shown that despite this divers can perform useful work at depth. The limit to diving depth imposed by the respiratory effects of increased gas density is probably about 1500 metres (unless the divers are prepared to wear ventilators).

Gas density may prove to be the limiting factor for deep diving.

It might be expected that the higher oxygen partial pressures in hyperbaric environments could improve physical performance. Chamber experiments, where the subjects exercised while breathing oxygen at 3 ATA, showed that the maximum aerobic work performance was not significantly increased.

Physically fit swimmers can swim for an hour at 65 per cent of their maximum speed, while runners can maintain only 55 per cent of their maximum speed for 1 hour. This difference has been attributed to the ease of heat dissipation in water.

ALTITUDE AND SATURATION DIVING

Our normal idea of diving is that a diver descends from sea level (1 ATA) and returns when the dive has finished but there are a series of variations from this. A diver might have to dive in a mountain lake where the pressure on the surface is less than 1 ATA, or he may start from an environment where the pressure is greater than 1 ATA. This happens when divers operate from a pressurized compartment or underwater house. These conditions introduce complexities that require understanding of the physics involved.

A diver operating in a high mountain lake is returning to a lower surface pressure than a diver at sea level. This decreases the pressure at which he is at while releasing inert gas after a dive, and so increases the tendency to form bubbles. Hence, he may need to modify his decompression plan. Another minor correction will be required if it is a fresh water lake. Because fresh water is less dense than salt water, the diver is exposed to a slightly smaller pressure change per unit depth.

This diver will also have to exhale faster during ascent. A diver who ascends from 10 metres (2 ATA) to the surface (1 ATA) would find that the volume of gas in his lungs has doubled. Most divers realize this and exhale at a controlled rate during ascent. They may not realize that a similar doubling in gas volume occurs during the last 5 metres of ascent to the surface, if the pressure at the surface was 0.5 ATA.

High-altitude diving may require that the depth or duration of dive and the rate of ascent be reduced to allow for the reduction in surface pressure at the end of the dive.

A diver living in a man-made environment where the pressure is high can operate to deeper than normal depths. This system is used in saturation diving, where the diver operates from a base at increased pressure and becomes equilibrated with it. Although the eventual return to the surface can take many days, the use of such environments has proved to be invaluable where deep or long dives are required (see Chapter 68).

Another pressure problem occurs when a diver dives and then either travels by air or ascends into mountains. Some dives and ascents may require the diver to pause at the surface if he is to avoid DCS. This problem is encountered especially by a diver tourist who wants to fly home after his last dive, and is also encountered when a diver with DCS has to be transported. There may be an increase in manifestations of DCS when the pressure is decreased, even by a small amount.

PHYSICAL ASPECTS OF THE MARINE ENVIRONMENT

Heat

Diving and exposure to high pressures changes the heat transfer from a diver's body. In air, there is some insulation from the air trapped near the body, either by the clothes or the hair and the boundary layer, but in water this is lost. The water adjacent to the skin is heated, expands slightly so as to cause a convection current, and this tends to remove the layer of warmed water. The process is accelerated by movement of either the diver or the water. The net result is that a diver cools or heats up much more quickly than he would in air of the same temperature.

Heat loss is also increased in warming the inhaled air or gas. For a diver breathing air, most of this heat is used to humidify the dry air used for diving, and it is not sufficient to cause concern in most circumstances. However, the heat lost in a helium dive is more significant, as helium has a greater specific heat than nitrogen. The problem is compounded because, at depth, the mass of gas inhaled is increased and so the heat loss is increased.

The heat transfer by conduction is also increased in a helium environment, the result being that a helium diver may need external heating to maintain body warmth at a water, or gas, temperature where external warming would not be required if he was in an air environment.

In warm environments it is possible for a diver to suffer heat stress. If he is wearing a protective suit he cannot lose heat by sweating because the sweat cannot evaporate. In a pressure chamber the atmosphere can become saturated with water and evaporative cooling is prevented. The heat stress for a given temperature is also increased if there is helium in the mixture.

A diver in water or a helium-rich environment can cool or heat up at a temperature that would be comfortable in an air environment.

Light

Even in the cleanest ocean water, only about 20 per cent of the incident light reaches a depth of 10 metres, and only 1 per cent reaches 85 metres. Clean water has a maximum transparency to light with a wavelength of 480 millimicrons (blue). This variation of absorption with wavelength causes distortion of colours, and is responsible for the blue-green hues seen at depth. Red and orange light is absorbed most. Because of the absorption of light the deep ocean appears black, and lights are needed for observation or photography. Because of the greater absorption of reds by water some illumination is needed to see the true colours, even at shallow depths. Part of the appeal of diving at night is that objects that have a blue-green colour in natural light have a new brightness when illuminated with a torch.

Coastal water, with more suspended material, has a maximum transparency in the yellow-green band, about 530 millimicrons. Absorption and scattering of light by suspended particles restricts vision and can tend to even out illumination. This can make the light intensity the same in all directions, and is an important factor in causing loss of orientation.

When the eye focuses on an object in air, most of the refraction of light rays occurs at the air–cornea interface. In water, this refractive power is lost and the eye is incapable of focusing. A facemask provides an air–cornea boundary, which restores refraction at the cornea surface to normal. Refraction also occurs at the facemask surface, mainly at the glass–air boundary, and this results in an apparent size increase of about 30 per cent, making objects appear closer than they are. Practice and adaption of the hand–eye coordination system allows the diver to compensate for this distortion, except when describing the size of fish (Fig. 2.5).

Masks also restrict vision by narrowing the peripheral fields, and distort objects that subtend large visual angles. Both absorption of light by water, which reduces apparent contrast, and scattering by suspended particles, reduce visual acuity. Attempts have been made to improve the diver's vision by modification of the face mask, the use of coloured filters, and contact lenses. However, these have been only partly successful and impose their own problems.

Sound

Sound in water is transmitted as waves with a longitudinal mode of vibration. The speed of sound in water is about 1550 metres/second in sea water and

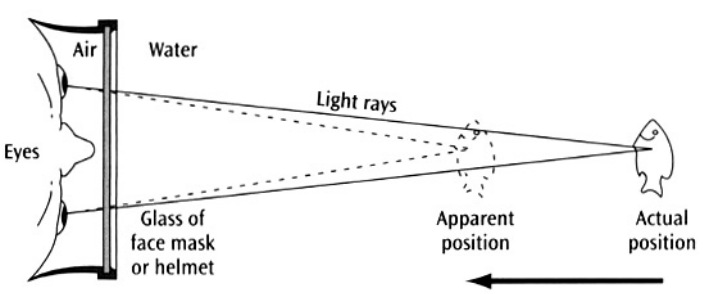

Figure 2.5 *Displacement of image in water.*

1410 metres/second in fresh water at 15°C. Water is a better transmitter of sound than air, so sounds travel greater distances under water. Low-pitched sounds travel further than higher-pitched sounds. Transmission of sound is enhanced by reflection from the surface; this reflection also enhances the transmission of sound in air over water, but reduces the transmission of sounds from air to water and from water to air.

Both high-pressure air and helium/oxygen mixtures cause speech distortion. This is greater when breathing helium mixtures, and can render speech unintelligible. Distortion in air results in the voice becoming more nasal and crisp as the pressure increases.

It is often thought that divers cannot talk underwater, but this is not so if the diver has an air space to speak into. Helmet divers can communicate easily by touching their helmets together, and using the air/copper/air pathway. Some scuba divers have mastered the art of talking by taking their demand valve from their mouth and speaking into an air space created by cupping their hands.

DIVING GASES

Most diving is based on the use of compressed air and other oxygen/nitrogen mixtures as a breathing gas. However, commercial, military, technical and experimental diving may involve the use of other gas mixtures, and for this reason it is desirable to provide the reader with some salient points on the gases mentioned in this text and related literature.

Oxygen (atomic weight 16, molecular weight 32) is the essential constituent of all breathing mixtures. At high altitude, people survive with less than 0.1 ATA in their inspired air; for diving, oxygen should be present at a partial pressure of at least 0.2 ATA to avoid hypoxia. At higher partial pressures oxygen causes oxygen toxicity. Prolonged exposure to over 0.5 ATA causes pulmonary oxygen toxicity, while shorter exposures to over about 1.8 ATA results in central nervous system effects. The risk of these problems may be acceptable in a recompression chamber where oxygen may be used at partial pressures of up to 2.8 ATA.

In the range 0.2–2.8 ATA oxygen has little effect on the respiratory centre, and minute volume will remain close to normal. Oxygen is vasoactive; high oxygen tensions cause vasoconstriction.

Nitrogen (atomic weight 14, molecular weight 28) is the major component of air, about 79 per cent. Nitrogen is often considered to be physiologically inert. Bubbles, composed mainly of nitrogen, can cause decompression sickness if a diver who has been breathing air or an oxygen/nitrogen mixture ascends too rapidly. In solution, it may cause nitrogen narcosis at depth. If the partial pressures of nitrogen are greater than about 3 ATA, there is a demonstrable fall-off in the diver's performance. At higher partial pressures the effect is likely to cause the diver to make mistakes. The other problem that restricts the use of nitrogen is its density at increased pressure increases the work of breathing.

Despite these disadvantages, nitrogen is of major importance in diving, at depths less than 50 metres, and as a part of more complex mixtures at greater depths.

Helium (atomic weight 4) is a light, inert gas. It is found in natural gas wells in several countries. Helium is used to dilute oxygen for dives to depths greater than 50 metres, where nitrogen should not be used alone. The two major advantages of helium are that it does not cause narcosis and, because of its lightness, helium/oxygen mixtures are easier to breathe than most alternatives. Helium/oxygen mixtures can allow a shorter decompression time than an equivalent saturation dive with the diver breathing air, because helium diffuses more rapidly than nitrogen.

The use of helium can cause several problems. The speech of a diver at depth may need electronic processing to make it understandable, because of the distortion. A diver in a helium atmosphere is more susceptible to heat and cold because the high thermal conductivity speeds the transfer of heat to and from the diver. The other problem with the use of helium is that it is associated with a disorder called the high-pressure neurological syndrome (see Chapter 20).

Hydrogen (atomic weight 1, molecular weight 2) has the advantage of being readily available at low cost. Because of its lightness it is the easiest gas to breathe. These factors may lead to its use as a

Table 2.2 *Pressure conversions*

	atm	n/m^2 or Pa	bars	mb	kg/cm^2	gm/cm2 (cm H_2O)	mm Hg	lb/in^2 (psi)
1 atmosphere	1	1.013×10^5	1.013	1013	1.033	1033	760	14.70
1 Newton (N)/m^2 or Pascal (Pa)	0.9869×10^{-5}	1	10^{-5}	0.01	$1.02X10^{-5}$	0.0102	0.0075	0.1451×10^{-3}
1 bar	0.987	10^5	1	1000	1.02	1020	750.2	14.51
1 millibar (mb)	0.9869×10^{-3}	100	0.001	1	0.00102	1.02	0.7502	0.01451
1 kg/cm^2	0.9681	0.9806×10^5	0.9806	980.6	1	1000	736	14.22
1 gm/cm^2 (1 cm H_2O)	968.1	98.06	0.9806×10^{-3}	0.9806	0.001	1	0.736	0.01422
1 mmHg	0.001316	133.3	0.001333	1.333	0.00136	1.36	1	0.01934
1 lb/in^2 (psi)	0.06804	6895	0.06895	68.95	0.0703	70.3	51.70	1

replacement for helium. The reluctance to use it is due to fears of explosion, but this can be prevented if the oxygen level does not exceed 4 per cent; such a mixture is breathable at depths in excess of 30 metres. Hypoxia can be prevented by changing to another gas near the surface. Hydrogen causes similar thermal and speech distortion problems to helium.

RECOMMENDED READING

Bennett, P.B. and Elliott, D.H. (eds) (1993) *The Physiology and Medicine of Diving.* 4th edn. Saunders, London.

Schilling, C.W., Werts, M.F. and Schandelmeier, N.R. (eds) (1976) *The Underwater Handbook. A Guide to Physiology and Performance for the Engineer.* Plenum Press, New York.

US Navy Diving Manual (1996) Vol. 1. Chapters 2 and 3 deal with these topics in a manner that assumes no previous knowledge.

3

Free diving

CHRIS LOWRY

INTRODUCTION

Free diving (also known as breath-hold diving) is regarded as the purest and most natural form of diving. Unencumbered by bulky equipment, one is free to move weightlessly and silently in the underwater world. Practised in some societies for thousands of years, free diving in its simplest form requires no equipment at all. The introduction of various performance-enhancing apparatus such as facemasks, fins, weight belts, buoyancy vests and thermal protection suits may present new problems. The addition of goggles or facemasks allows for clearer vision but introduces the risk of barotrauma. Wetsuits, weight belts and vests alter buoyancy and restrict mobility. Recreational snorkellers and spearfishermen may wear a mask, snorkel, fins, wet suit and weights and carry a spear gun, knife and bag. The free diving record-seeker may also employ specialized devices such as sleds, weights and balloons to achieve remarkable depths. Nonetheless, even with modern specialized equipment, human diving capabilities are paltry in comparison with other diving air-breathing animals such as reptiles, birds and mammals.

The study of diving animals offers the scientist an ideal opportunity to study the physiological consequences and defence mechanisms required in order to survive extended breath-holding. It is also of great interest to diving physicians to see how diving animals avoid the perils induced by exposure to pressure and hypothermia. Adaptations made by marine mammals in order to cope with the peculiar problems of an aquatic environment are briefly outlined, as their anatomy and physiology is closer to humans than birds and reptiles.

MAN AS A FREE DIVER

'You're running on reserve tank and there's no warning before you hit empty!'

Record-holding free diver

In Mediterranean countries where free diving is popular, approximately 55 divers die each year – many near the surface after deep dives. For humans, free diving is dangerous – and the deeper the dive the greater the risk.

Between underwater excursions the breath-hold diver is either: floating vertically in the water breathing atmospheric air against the pressure of the surrounding column of water; or swimming horizontally and breathing through a snorkel tube. Both of these circumstances have physiological consequences. Snorkel breathing is discussed in Chapter 4.

Head-out immersion

Because a swimmer breaths air at 1 ATA, the pressure in the lungs must be also 1 ATA. However, the surrounding water pressure increases with depth so the chest is subject to a pressure greater than 1 ATA and this pressure is greater in the lower parts of the vertical lung. The swimmer must therefore produce a negative of about 20 cm intrathoracic pressure to inhale. This reduces the functional residual capacity, the expiratory reserve volume and, to a lesser extent, the vital capacity. The work of breathing is increased. Another outcome of this increased negative intrathoracic pressure is increased venous return to the heart. There is also a loss of gravitational effect, which reduces the volume of blood in the lower body (mainly leg) veins and increases the intrathoracic blood volume.

This raised central blood volume, an increase of about 500 ml in normal individuals, leads to an increased stroke volume and cardiac output. It also contributes to the well-known diuresis of immersion, but the exact reflex mechanism is not entirely clear.

The increased trans-diaphragmatic pressure gradient is enough to overcome gastro-oesophageal sphincter function in susceptible individuals, causing gastric reflux.

Table 3.1 *Comparative depth penetrations*

Diving situation	Depth (in metres)
Human free (breath-hold) diver	
- 'constant ballast'	81
- 'no limits'	150
Sunlight perceptible to human eye	500
Human (compressed gas)	600
Military submarine	900
Sperm whale	1150
Northern elephant seal	1500
Wreck of Titanic	3810
Alvin (manned submersible)	5000
Octopus	5639
Jason (robotic submersible)	6000
Shinkai (three-person submersible)	6527
Deepest known fish	8370
Amphipod crab	9789
Deepest part of ocean	10 912

Depth limits

The ratio of the total lung volume to the residual volume affects the maximum depth that can be reached without lung collapse or blood pooling in the thorax. This situation is reached when the increased pressure has compressed the initial lung volume to the residual volume. Thus, a diver with a total lung capacity of 6 l and a residual volume of 1.5 l should theoretically be able to breath-hold dive to 30 metres (4 ATA), where the total lung volume would be compressed to the residual volume of 1.5 l (Boyle's law). Divers with a larger total lung capacity and/or a smaller residual volume would be capable of greater depths before lung collapse occurs. Significantly greater depths than those suggested by these calculations are regularly achieved.

Compression of the lung during descent allows pooling of blood in the pulmonary vasculature and large intrathoracic veins. Up to 1 l may be shifted into the thorax. This redistribution of blood lessens the reduction in lung volume and significantly increases the maximum depth that can be reached by trained breath-hold divers. Descent to a depth where the lungs are damaged by compression causes a form of pulmonary barotrauma known as 'lung squeeze'.

Duration limits

The total oxygen stores in a 70-kg man at resting lung volume have been calculated to be 1.5 l. If nearly all of this oxygen can be extracted, one might

predict that resting man who has an oxygen consumption of 300 ml/min would completely deplete his oxygen stores in 5 minutes. Most untrained humans can only breath-hold for approximately 1 minutes. The limit or breaking point of breath-hold diving is determined by the interactions of lung volume, carbon dioxide and oxygen blood gas tensions as well as psychological drive. Arterial carbon dioxide is the most important.

The difference in response of trained divers may result from a decreased sensitivity of the medullary centres to carbon dioxide, a conscious disregard for the carbon dioxide stimulus, or both. A depressed ventilatory response to carbon dioxide has been found in submarine escape training personnel and in professional breath-hold divers such as the Ama. Their vital capacity and maximum breathing capacity are also much larger than those of non-divers. The increase in vital capacity is due to an increase in the inspiratory reserve volume.

Respiratory gas changes

Blood gases during breath-hold dives will change with the partial pressure of the gases in the lungs (Dalton's law), as well as with changes caused by oxygen (O_2) consumption and carbon dioxide (CO_2) production. When the breath-hold diver descends, the partial pressures of the gases in the lungs increase and their volumes decrease. The reverse takes place during ascent back to the surface. This leads to concomitant rises and falls in arterial and tissue O_2 and CO_2 pressures.

Alveolar gas pressures during (a) a breath-hold period, (b) a breath-hold dive to 10 metres and (c) a breath-hold dive to 10 metres with prior hyperventilation are illustrated in Figure 3.1. During **breath-holding**, CO_2 pressure rises and O_2 falls as expected.

With **descent**, ambient and thus partial pressures rise according to Boyle's law. The rise in O_2 is somewhat reduced because of continued consumption. Because of the high alveolar P_{O_2} at depth there is a sufficient alveolar–arterial gradient to allow continuing oxygen uptake for a considerable time.

During **ascent** the rapid fall in alveolar O_2 tension is greater than expected from gas laws alone, showing that oxygen was still being extracted from the alveolar air. A very low partial pressure of O_2 developed at the end of the dive preceded by hyperventilation. The dangers of breath-hold diving and hyperventilation are discussed further in Chapter 16.

CO_2 elevation during descent reverses the normal gradient from blood to alveoli. This reversed CO_2 gradient was maintained during the period at 10 metres (2 ATA), except for the last 20 seconds of the dive preceded by hyperventilation. This indicated continued tissue storage of CO_2. The final alveolar CO_2 concentrations were greater than those predicted, indicating that the normal gradient had been re-established and that CO_2 had passed back into the lungs.

There would be some nitrogen absorption from the lungs during descent and the time at depth. This would be followed by its release during ascent and the following period on the surface.

The diving response

Face immersion causes a reflex apnoea if not already initiated voluntarily. This instinct can be very strong, sometimes totally preventing the novice from inspiring from a snorkel or demand valve despite the exhortations of the diving instructor.

The bradycardia in diving mammals (described later in this chapter) has also been observed in humans during actual and simulated diving, and in face-only immersion. In some cases the heart rate may drop to as low as 10–15 beats per minute after 50 seconds face immersion. There may be a slightly increased heart rate during the first few seconds of the dive, but it then slows to about 60 per cent of the pre-dive rate. The tachycardia may be due to sympathetic stimulation, or to a rise in central blood volume (Bainbridge reflex). In some trained young individuals the rate may be even lower in spite of underwater exercise. The degree of induced bradycardia declines with age. Simple face immersion with simultaneous breath-holding is almost as effective in producing diving bradycardia as total body immersion.

The two basic stimuli precipitating the diving response are apnoea (voluntary or involuntary) and water on the face. Colder water produces a greater fall in heart rate. Fear is also thought to heighten the response.

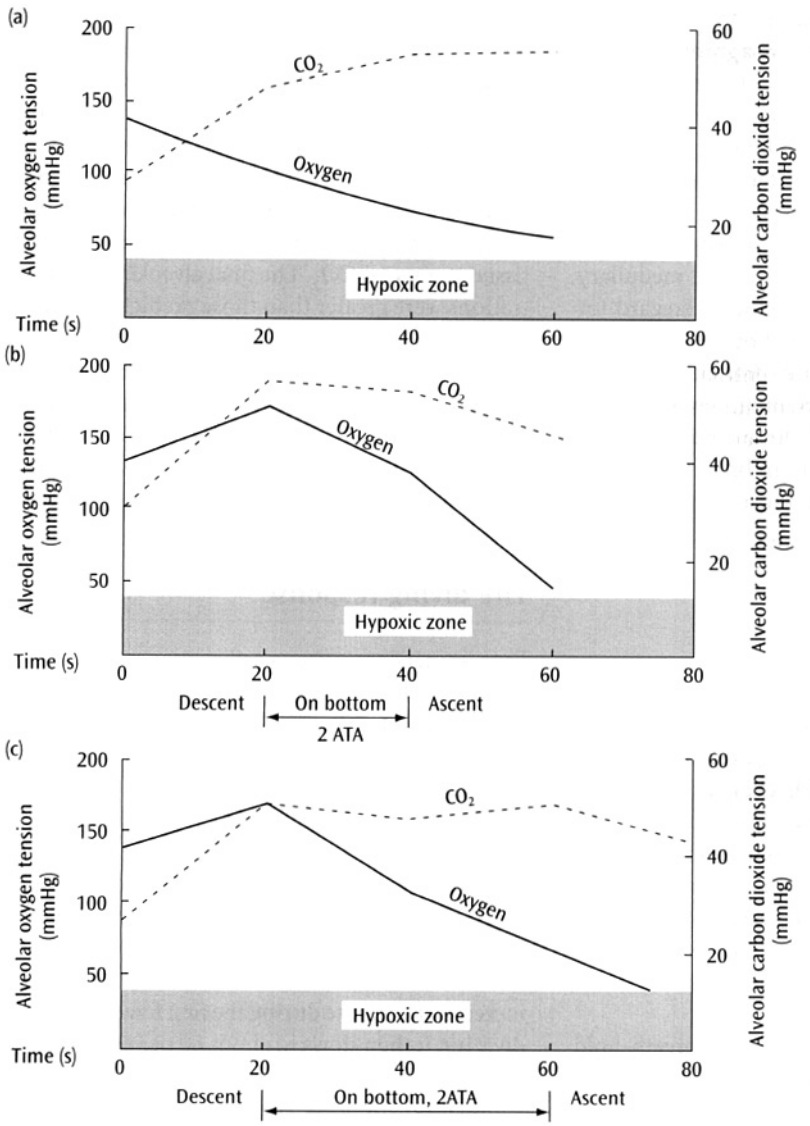

Figure 3.1 *Alveolar gas tensions during breath-holding and breath-hold dives. (a) Breath-hold on surface; (b) breath-hold dive; (c) breath-hold dive preceded by hyperventilation. These graphs are drawn from a series of breath-hold dives in a chamber to 10 metres reported by Lanphier and Rahn (1963). The dives involved a 20-second descent, 20 or 40 seconds at depth, and then a 20-second ascent. These graphs show that the partial pressures of both gases increased during descent. The alveolar gas tensions did not double as might be expected from Dalton's law, indicating that oxygen was being extracted from alveolar air and carbon dioxide had passed back into the tissues from the lung, i.e. the normal carbon dioxide gradient was reversed.*

There are other components of the diving response as well as the more obvious 'diving bradycardia'. Despite the progressive decrease in heart rate there is a coincident increase in the arterial blood pressure. Intense peripheral vasoconstriction is also initiated within the first few seconds of exposure. Forearm blood flow is decreased more during face immersion than during breath-hold alone. The vasoconstriction occurs in peripheral arterioles and has also been demonstrated in arteries greater than arteriolar size during diving.

Certain individuals, usually fit and young, display a marked bradycardia and a progressive reduction in forearm blood flow to almost zero during simulated dives. There is a direct linear relationship between reductions in heart rate and forearm blood flow.

The diving response, elicited by breath-holding and immersion, is characterized by bradycardia, reduction of limb blood flow, and a gradual rise in mean arterial pressure.

Blood lactate increases by a small amount during the latter part of the dive, but levels usually peak during the first minute of the recovery period.

Cardiac dysrhythmias are common during breath-hold diving, and again the incidence is higher in cold water. The commonest abnormalities include abnormal P waves and atrioventricular nodal rhythms. The more severe dysrhythmias generally occurred towards the end of the apnoeic period. Atrial, nodal and ventricular premature contractions have been observed, as has sinus bradycardia, sinus arrest, nodal escape rhythms, atrioventricular (AV) block, and idioventricular rhythm. The T waves often become tall and peaked.

The question remains as to whether the diving response has any protective effect conserving oxygen in humans.

'HUMAN AQUATIC POTENTIAL'
Extracts from 'Pipin Productions' by noted free diver Francisco 'Pipin' Ferreras (http://www.freediving.net/potencia.htm)

. . . My heart, under direct control of the central nervous system begins a rapid slowdown. This diminution of my cardiac output is a result of the body's decreasing needs for oxygen and energy consumption. This efficiency in energy conservation is of vital importance for survival in the undersea environment while in a state of apnea. As an example, when I begin my pre-immersion preparations my resting heart rate is 75 bpm, 10 minutes after entering a state of deep relaxation it drops, to 55 bpm. As I begin my descent, in a matter of seconds it has slowed to 30 bpm. My cardiovascular performance is influenced by other factors, foremost being my physical conditioning, and mental preparation . . . Once I have reached a depth of 110 m., I institute one last command to my heart to slow down. At this point my heart is down to a mere 10 to 14 bpm. On several immersions when all of the above mentioned factors are ideal I have obtained readings of an incredible 7 bpm! Obviously these findings are augmented by the power of mind over body that I have developed over the years, through the study and practice of Yoga . . .

. . . Finally, the subject would be incomplete without mentioning the most intriguing of all these physiologic adaptations, that of the 'Blood Shift Phenomenon'. This theoretical 'shift' has not been proven directly although enough indirect evidence exists to attribute my ability to withstand these pressures that theoretically would cause 'Thoracic Squeeze'. This theory implies that the maximum achievable depth while breath holding is limited by the individuals' maximum lung volume TLC (Total Lung Capacity) and his Residual Volume . . .

. . . The only viable explanation to my markedly surpassing this theoretical limit is that of this 'Blood Shift'. Panic engrossed me the first time that I experienced the sensation of my lungs drowning in liquid. This has now become a welcomed and pleasant sensation that accompanies me every dive. As the human body approaches the depths, the 'weight of the ocean' begins to exert its pressure, peripheral blood begins to fill the lungs, the compressed volume of air merely filling the frontal and paranasal sinuses. Blood is the only liquid that can withstand the column of hydrostatic pressure and thus prevent the collapse of the thorax.

Now that you have read my premise, can you consider that a part of us belongs in the aquatic environment? I truly believe it, and this gives me the moral obligation to discover man's maximum potential . . .

Record diving

Trained breath-hold divers have been able to achieve remarkable underwater feats, and in certain societies these divers are accorded celebrity status.

Records are attempted for various categories of diving involving depth, duration and underwater distance. Because of the risks involved these attempts are not sanctioned by most national and international diving organizations. Nevertheless, physiologists and physicians need to be aware of these remarkable achievements. The purest form of depth record is referred to as **constant ballast** and involves return to the surface with the same weights carried down (if any). The record is currently 81 metres. **Variable ballast** involves using weights and even a sled to reach the depth target, which are then shed for a mechanically unassisted return to the surface. The record is of the order of 115 metres. It should be noted that this depth is well beyond the limits of safe compressed-air scuba divers.

No limits free diving is the most extreme category, and requires no swimming at all. Divers hold on to a weighted, rope-guided sled. On reaching the target depth, they detach themselves from the sled and pull a pin that releases compressed air from a cylinder into a balloon, which they grab to speed themselves back to the surface. Although the greatest depths are achieved this way, many in the sport regard it as somewhat artificial. Depths of 152 metres have been reached.

The absolute limit of these hazardous 'experiments' remains unknown. Death may be precipitated at depth by pulmonary haemorrhage, pulmonary oedema or cardiac dysrhythmias. Cerebral hypoxia is an invariable development during the latter stages of ascent. Quite often these divers require rescue by standby scuba divers because they become unconscious due to rapidly developing hypoxia as they approach the surface.

Records are also held for **static apnoea**, which is a motionless, energy-conserving underwater duration exposure. Periods of over 7 minutes have been reported. Underwater breath-hold horizontal distances (**dynamic apnoea**) of 164 metres have been achieved in 50 metre swimming pools, using fins for propulsion. However, divers attempting these latter two records risk unconsciousness from hypoxia, especially with prior hyperventilation, but this can be obviated by prior inhalation of oxygen.

Exponents of all these types of underwater exposures frequently demonstrate reduced sensitivity to CO_2 when tested in a laboratory.

DIVING MAMMALS

Marine mammals have the same characteristics that separate mammals from all other animals, i.e. they breathe air exchange gas through the lungs, are warm-blooded, have hair or fur, bear live young, and produce milk. Marine mammals are classified as follows:

I. Order Carnivora
   Sub-order pinnipedia – sea lions, fur seals, true seals, walruses, sea otters.
II. Order Cetacea – whales, dolphins, porpoises; this is the most diverse group.
III. Order Sirenia – dugongs, manatees.

The northern elephant seal and the sperm whale can dive to 1500 metres. The Southern elephant seal can stay submerged for 2 hours, although usual dives are of 20–30 minutes duration. The Weddell seal regularly dives for food to greater than 100 metres, and can remain submerged for up to 60 minutes. Humans, with some practice, can breath-hold underwater for about 2 minutes and descend to 10–15 metres.

The majority of dives by animals in the wild are brief and aerobic. Such dives are apparently dependent largely upon oxygen-conserving rather than anaerobic defences. Longer dives invoke anaerobic responses, which are more exhausting and require a longer recovery. Less often utilized, anaerobic mechanisms may nevertheless be essential for survival. It is not known how the animal modifies its physiological response to allow for longer, deeper dives.

How are these animals able to achieve these remarkable underwater depth and/or duration exposures that appear to defy conventional wisdom with respect to limits of hypoxia? How also do they achieve these feats without developing some of the disorders (such as shallow water blackout, barotrauma, decompression sickness, nitrogen narcosis, oxygen toxicity or high-pressure neurological syndrome) that are the subject of subsequent chapters in this book? Mechanisms for avoiding these problems are interlinked and involve anatomical and physiological adaptations. This has been the subject of extensive research over the years, and although much is now known, some mysteries remain.

Obvious anatomical adaptations include: a streamlined shape, low-friction body surface (skin or

fur), and the development of flippers or fins. Dolphins can reach speeds of 20 knots with remarkably low energy consumption. A dorsal blowhole in whales and dolphins also aids energy efficient respiration.

Less evident but more interesting to the diving physician and physiologist are the mechanisms to cope with an interrupted oxygen supply. A marked variation in susceptibility to hypoxic damage exists not only among species but also among different tissues of the body. For example, the tissues in human limbs can be deprived of blood for up to 2 hours or more without damage. On the other hand, it is obvious that the central nervous system – especially those parts of it involved in maintenance of consciousness – cannot sustain activity for more than a few seconds without a continuous supply of blood which is rich in oxygen.

The dramatic physiological changes that take place during mammalian birth provide an illustration of mechanisms designed to protect the central nervous system during asphyxia. These responses include the use of anaerobic reserves and cardiovascular adjustments that favour the central nervous system at the expense of less sensitive tissues. Analogous mechanisms come into play in breath-hold diving.

Adaptations that allow diving animals to achieve long periods underwater are both physiological and biochemical. These adaptations are not new from an evolutionary aspect; rather, they are qualitatively similar to processes that exist even in a rudimentary form in all vertebrates, though species differ in their quantitative development. They can perhaps be best considered as developments of the normal responses that ensure survival.

Oxygen stores

All diving mammals have an increased total body oxygen store. The relative contribution of the lungs, blood and muscles storage areas depends on the diving pattern of the animal.

Deep-diving mammals probably do not dive at full lung capacity for reasons discussed later, so the bulk of oxygen is stored in blood and muscle. Such animals have increased blood volume, red cell volume and haemoglobin concentration. They also have a markedly increased myoglobin concentration, especially in the swim muscles, and this myoglobin increase is proportional to the diving capacity of the animal.

Seals store an abundance of oxygen – twice as much per kilogram of body weight as a human being does. The oxygen is mainly held in the blood and, to a lesser extent, in the muscles. Only 5 per cent is stored in the lungs as opposed to 70 per cent in the blood. The oxygen-carrying molecule myoglobin binds about 25 per cent of the animal's oxygen in the muscles. An increase in blood volume and haemoglobin contributes to the seal's impressive oxygen supply, but oxygen still needs to be conserved.

The shunting of oxygen-rich blood from capacious spleens, hepatic sinuses and abdominal vasculature is not thought to play as large a part in oxygen storage as was previously believed.

Oxygen consumption

The resting metabolic rates of seals are somewhat higher than those of comparably sized terrestrial mammals, while body temperatures are similar. The elevated metabolic rate and thick subcutaneous blubber (representing 25–50 per cent of body weight) provide the heat production and insulation necessary for maintenance of normal internal temperature in the cooling water environment. It can be readily calculated that if the submerged seal continued to metabolize at the same rate as before diving, its oxygen supplies would not be sufficient during long dives.

The duration of the dive is determined by the rate of depletion of oxygen stores and the level of tolerance to low blood oxygen. Oxygen depletion is affected by individual organ metabolic rates. The metabolic rate of the heart, for example, is affected by heart rate and bradycardia is a feature of diving animals (see below). Hepatic and renal oxygen extraction is affected by perfusion. In Weddell seals, renal and splanchnic blood flow is reduced in stressed or forced dives, but not in routine foraging dives.

The degree of muscle perfusion also affects blood and muscle oxygen utilization. Blood may be preferentially perfused to swimming rather than non-swimming muscles. Studies indicate that pinniped

skeletal muscles have an enhanced oxidative capacity to maintain aerobic lipid metabolism under the hypoxic conditions associated with diving, and that these adaptations are more pronounced in swimming than in non-swimming muscles.

Diving response

The term 'diving reflex' usually refers to the bradycardia of immersion. In 1870, the French physiologist Paul Bert observed that ducks experimentally submerged in water exhibited a dramatic slowing of the heart rate – an observation which initiated a long and continuing series of investigations. All vertebrates exhibit this phenomenon, which is part of a complex of cardiovascular and metabolic adaptations to conserve oxygen during diving.

In contrast to land mammals that increase ventilation and cardiac output during exercise, marine mammals breath-hold, reduce cardiac output, and limit peripheral blood flow during diving.

The term 'diving response' refers to a sequence and collection of physiological events, including apnoea, bradycardia and redistribution of cardiac output, which are all under the control of multiple reflexes rather informing a single 'diving reflex'. Oxygen conservation as opposed to storage is accomplished by selective redistribution of circulating blood.

Rapid onset of bradycardia at start of the dive is most profound in diving species. This enables the work of the heart to be reduced and hence require less oxygen. It also slows catabolism. Tissues that are most critical for survival (e.g. retina, brain, spinal cord, adrenal glands and, in pregnant seals, the placenta) are well supplied with oxygen by constriction of the arteries during the maximum supply of blood to these areas. The seal essentially shuts off the flow of blood to certain tissues and organs, such as the kidneys, until it resurfaces.

Some major components of the diving response are determined by the intention, conditioning and psychological perspective of the animal being studied. Thus, the animal produces the most intense diving response when the need for achieving maximum diving duration is anticipated.

Cardiac output is either normal or falls during apnoeic dives. Up to 90 per cent reduction in cardiac output has been shown in experimentally dived Weddell seals, and about 30 per cent reduction in stroke volume. The maintenance of **arterial blood pressure** is essential if adequate perfusion of vital organs is to take place during the profound decrease in heart rate and cardiac output. The mean arterial pressure remains almost constant, despite the prompt heart slowing from 110 to 9 beats per minute. The energy for maintaining arterial pressure is provided by the stretching of the elastic arterial walls during and immediately following systole, and its recoil during diastole.

This function is augmented in many species of marine mammals by a bulbous enlargement of the root of the aorta, the aortic bulb. This approximately doubles the diameter of the ascending aorta in harbour and Weddell seals, providing a compensating reservoir for maintaining pressure and flow into the constricted arterial tree during the long diastolic intervals characteristic of diving. The entire human aorta contains less volume than the aortic bulb alone in seals of a similar bodyweight. The increase in left ventricular afterload that would be expected as a consequence of elevated peripheral resistance and decreased large artery compliance is reduced. The result diminishes peaks systolic pressure development, thus tending to lessen cardiac work and oxygen consumption while at the same time maintaining stroke volume.

Muscular sphincters in the inferior vena cava alter blood flow during dives adjusting cardiac output to the reduced diving requirement. Sometimes in the latter portion of a dive, blood oxygen may be higher in the inferior vena cava than in arterial circulating blood.

The **electrocardiogram** of the diving animal shows some progressive changes during prolonged apnoeic dives. These include, besides the general bradycardia with prolongation of diastole, sometimes the gradual diminution or even abolition of the P wave. Cardiac rhythm is then apparently set independently of the sinoatrial node, and is established by a ventricular pacemaker site. Other cardiac dysrhythmias occasionally appear.

Anaerobic metabolism

With prolonged dives certain tissues will switch to anaerobic metabolism, which produces lactic acid as a by-product. There is an increased tolerance to lactic acid in the muscles through increased buffering capacity. High levels of lactic acid, however, lower the pH of the blood and can lead to acidosis, causing a weakening of the heart's ability to contract. Acidosis is avoided by confining anaerobic metabolism to the skeletal muscles and other tissues isolated from the blood supply. When the animals resurface, these tissues will release the lactic acid into the blood and the liver, lungs and other organs will flush out the by-product. Also, immediately upon resurfacing, heart rate and cardiac output temporarily exceed the resting levels. Recovery periods at the surface allowed Weddell seals to clear lactate from the blood either by oxidation or by a resynthesis by oxygen to glucose or glycogen. Long anaerobic dives are not necessarily always followed by long surface recoveries. On some occasions, long dives by the Weddell seal are followed by sequences of short aerobic dives during which lactate clearance has continued.

Diving technique

Modified diving behaviour to limit muscle activity and thus oxygen consumption has been demonstrated in Weddell seals. Prolonged downward gliding, with minimal muscular effort, due to reducing buoyancy with lung compression at depth can result in up to a 60 per cent reduction in energy costs. Gliding is utilized during dives exceeding 18 metres in depth, and occupies 78 per cent of the descent.

Pressure changes

Structural adaptations to accommodate thoracic compression during deep dives include a flexible rib cage, stiffened alveolar ducts, and attachments of the diaphragm so as to permit some shifting of abdominal contents into the thorax. The resultant quarantining of pulmonary gas from perfusing blood minimizes accumulation of nitrogen (decompression sickness), which might occur in repetitive diving.

Deep-diving mammals do not dive on a full lung volume. As well as limiting nitrogen uptake this means that the animal is not exposed to oxygen toxicity because the partial pressures never reach dangerous levels.

The above structural changes also help the animal avoid pulmonary barotrauma of descent, though how the elephant seal and sperm whale avoid the high-pressure neurological syndrome during their impressive diving feats is not yet understood.

Hypothermia

A thick layer of blubber and a relatively low surface area to reduce heat loss maintains core temperature. A reduction of blood flow to the skin increases insulation of the fat layer and allows surface cooling, which is not transmitted to the internal core. Well-developed counter-current heat exchange systems also aid in conserving heat by cooling arterial blood and heating venous blood as it returns to the core. Examples can be found in the fins and flippers of whales and seals. Working muscles are close to the surface and have little fat insulation. Also many animals, when not diving, have a raised metabolic rate to produce heat.

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Kooyman, G.L. (1989) *Diverse Divers; Physiology and Behavior.* Springer-Verlag.

Kooyman, G.L. and Ponganis, P.J. (1998) The physiological basis of diving to depth: birds and mammals. *Annual Reviews of Physiology.* **60**, 19–32.

Free-diving websites

- http://www.multimania.com/aidafrance/AIDA/Tableau.htm
- http://www.geocities.com/~freediver/freediving-page.htm

4

Diving equipment

JOHN PENNEFATHER

INTRODUCTION

The first part of this chapter deals with the equipment used by amateur divers. The more complex and unusual types of diving equipment that are used by commercial or military organizations are dealt with in the second part of the chapter. Attention is paid to the problems that the equipment can cause, particularly for the learner diver. This is important in understanding the medical problems that are related to the diving equipment, and may also help the reader to understand the stresses experienced by the novice diver.

EQUIPMENT FOR AMATEUR DIVING

Breath-hold diving equipment

The simplest assembly of diving equipment is that used by many children, coral reef tourists and spearfishermen – a mask, snorkel, and a pair of fins or flippers. In colder climates a wet suit may be added for thermal insulation and a weight belt to compensate for the buoyancy of the suit.

Mask

A mask is needed to give the diver adequate vision while underwater. The mask usually covers the eyes and nose, and seals by pressing on the cheeks and forehead with a soft rubber edge to prevent entry of water. Goggles which do not cover the nose are not suitable for diving. The nose must be enclosed in the mask so that the diver can exhale into it to allow equalization of the pressure between the mask and the water environment. It should be possible to block the nostrils without disturbing the mask seal, to allow the wearer to perform a Valsalva manoeuvre. Full facemasks, that cover the mouth as well as the eyes and nose, or helmets that cover the entire head, are more commonly used by professional divers and are considered in the later section on professional diving equipment.

A diver with visual problems may obtain a mask with the **optical corrections** ground in, but others mount ordinary spectacles (with arms removed) into their mask. Ocular damage can occur if hard corneal lenses are used for diving (see Chapter 30). Contact lenses may also be lost if the mask floods. The lens part of the facemask should be hardened glass. People who are susceptible to allergies may

prefer a mask with the rubber portion made from silicone rubber in order to reduce allergy problems.

All masks cause a restriction in vision, but in most cases the diver can see about one-third of his/her normal visual field. The restriction is most marked when the diver tries to look down toward their feet, and this can be a danger if the diver becomes entangled. The more nervous beginner finds the visual restriction worrying, often fearing that there is a lurking predator just outside his vision. The visual field varies with the style of mask. Experimentation is also needed to find which mask provides a good seal to minimize water entry. The diver also needs to master a technique to expel water from the mask; if this is not learned, a leaking mask can become a major problem.

Snorkel

The typical snorkel is a tube, about 40 cm long and 2 cm diameter, with a U-bend near the mouth end. A mouthpiece is fitted to allow the diver to grip the tube with his teeth and lips. The tube is positioned to lead past his ear so that the diver can breathe through the tube while floating on the surface, looking down. Any water in the snorkel should be expelled before inhaling through it. Many attempts have been made to 'improve' the snorkel by lengthening it, adding valves, etc, but so far these appear to have been unsuccessful. All snorkels impose a restriction to breathing, a typical snorkel restricting the maximum breathing capacity to about 70 per cent of normal. The volume of the snorkel also increases the diver's anatomic dead space. Because of this, increasing the diameter to reduce the resistance is not a viable option. These problems add to the difficulties of a diver who may be struggling to cope with waves breaking over him (and into his snorkel) and a current that may force him to swim hard. There have also been anecdotal reports of divers inhaling foreign bodies that have previously lodged in the snorkel. (The senior author's reported incident involving a cockroach has been discredited as a cockroach of the claimed size would not fit in a snorkel.)

Fins

Fins, or flippers, are mechanical extensions of the feet. Fins allow the diver to swim faster and more efficiently, and free his arms for other tasks. The fins are normally secured to the feet by straps or moulded shoes. Various attempts have been made to develop fins that give greater thrust with special shapes, valves, controlled flex and miracle rubber competing for the diver's dollar. Divers often get cramps, either in the foot or calf, with fins that are the wrong size, or if they are out of training. The loss of a fin may also cause problems for a diver if they have to a swim against a current.

Weight belt

Even without the buoyancy of a wet suit, some divers require extra weights to submerge easily. The weights are made from lead and are moulded to thread onto a belt. Usually, the belt should be fitted with a quick-release buckle, to allow a diver to drop the weights quickly and so aid return to the surface. The situations where a quick-release buckle should not be fitted are those where it would be dangerous to ascend, e.g. in caves where there is no air space above the water.

Unfortunately, divers often neglect to release the belt if they are in difficulties, and the reason for this omission is not clear. It is possibly caused by defective training, because they almost never drop it during a drill. There have been cases of people in a panic touching the buckle, as is commonly done in a drill and leaving the belt on. The alternative drill of taking the belt off and holding it in one hand is recommended because the diver will drop it if he loses conciousness. If he needs two hands to correct his problem he can replace the belt and remedy it. Another, less frequent difficulty with weight belts, is that the weights can slip on the belt and jam the release mechanism.

In most fatal diving accidents the diver did not release his weight belt.

This basic free-diving equipment is adequate to dive in shallow, warm water, and experience with this gear is excellent training for a potential scuba diver. The diver can gain the basic skills, without the extra complications caused by scuba gear. It will also allow a more realistic self-assessment of the desire to scuba dive. With the confidence gained in free diving, he is also less likely to become dependent on his breathing apparatus. In cold climates, a free diver needs a suit to keep warm (suits are discussed in the section on thermal protection).

Breathing apparatus

The simplest form of breathing apparatus consists of a gas source and a tap which the diver turns on to obtain each breath of air. This system works, and was in use until the 1930s, but much of the diver's time and concentration may be taken up in operating the tap. In the most common breathing apparatus, the **Aqualung** or **scuba**, the tap is replaced by a one- or two-stage valve system.The flow of gas to the diver is triggered by the diver's inspiratory effort, and closed by expiration or cessation of inspiration.

The operating principles of a simple demand valve system are shown in Figures 4.1 and 4.2 (a,b). The air is stored in a cylinder at a maximum pressure that is determined by the design of the cylinder. For most cylinders this pressure – called the working pressure – is 150–200 ATA (2200–3000 psi).

The first stage of the valve system (Fig. 4.1) reduces the pressure from cylinder pressure to about 10 atmospheres greater than the pressure surrounding the diver, and regulates its outlet pressure at this value. The valve is held open by the force of a spring until the pressure above the first-stage piston builds up and forces the valve seal down on the seat, shutting the gas off. The first-stage valve opens and closes as gas is drawn from the system by the diver. The water can enter the water chamber, and this helps the spring to hold the valve open. This adjustment of the supply pressure with water pressure is termed 'depth compensation'. It is designed to prevent the flow decreasing as the diver descends.

When the diver inhales he reduces the pressure in the mouthpiece, or second-stage valve. As he does so, the diaphragm curves in and depresses the lever (Fig. 4.2(a)). The inlet valve opens and remains open until inhalation ceases. At this stage the diaphragm moves back into the position shown in Figure 4.2(b).The second stage valve is often called the 'demand valve'.

Expired air passes out of the mouthpiece through an expiratory valve. In the demand valve, air flow increases with respiratory effort because the valve opens more, allowing the diver to breathe normally. The purge button allows the diver to open the inlet valve to force any water out of the regulator. He may need to do this if he takes his regulator from his mouth while underwater. An example is when the diver has to share the air supply with another diver, a practice called 'buddy breathing'.

The scuba regulator provides the diver with a gas supply matched to his respiratory needs.

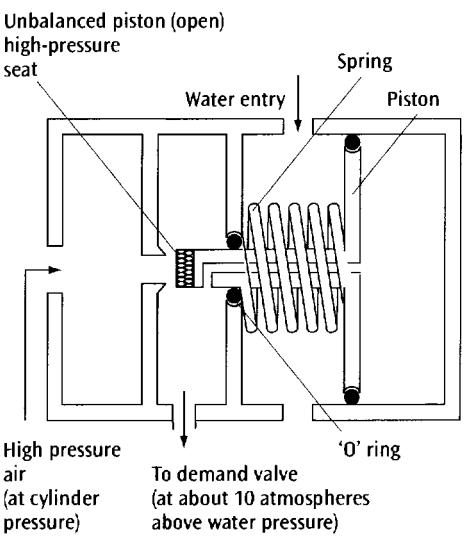

Figure 4.1 *First-stage reducer valve: the gas escapes from the cylinder until the pressure above the piston increases to a level where the force on the pressure can compress the spring, pushing the first-stage valve seat down and shutting the gas flow off. The valve opens again when the pressure above the piston (and in the hose to the second-stage valve) falls. This is normally because the diver has taken another breath.*

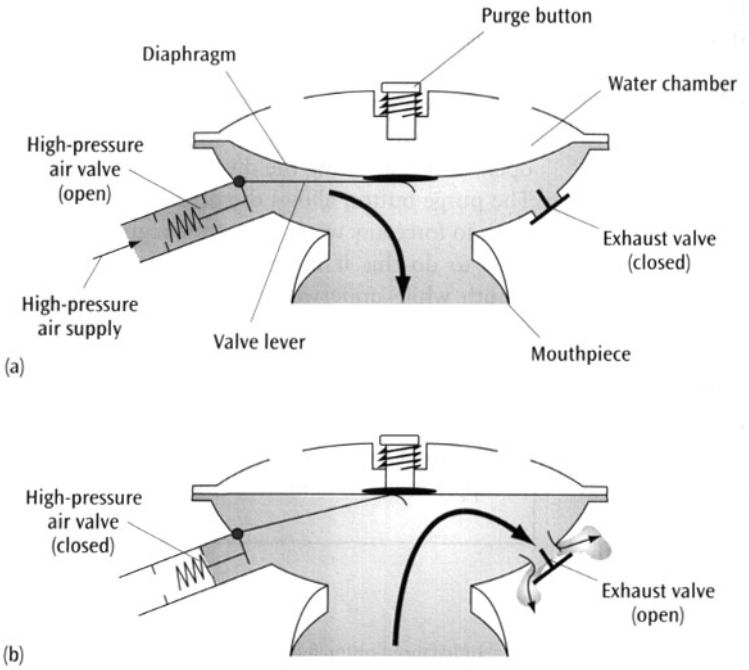

Figure 4.2 *Scuba demand valve during (a) inspiration and (b) exhalation. The arrows indicate air flow. During inspiration the diver decreases the pressure in the mouthpiece. This causes the diaphragm to curve in and tilt the air supply valve open. At the end of inspiration air continues to flow until the pressure in the mouthpiece equals the pressure in the water chamber; at this stage, the diaphragm will return to the position shown in (b), and the air supply valve shuts. During exhalation the pressure in the mouthpiece is greater than that in the surrounding water. This pressure difference forces the exhaust valve open and allows exhaled air to escape. The purge button is used to trigger a flow of gas from the supply without the need to inhale from the regulator.*

Most divers have little difficulty using scuba. When they first don it, the weight and bulk will make them awkward, and may aggravate back problems. However, in the water, the buoyancy of the set balances its weight.

The lips should seal round the mouthpiece and prevent the entry of water. Water can also enter through the diaphragm or the expiratory valve if either are faulty. A leak can generate an aerosol if the water reaches the inlet valve of the second-stage valve, and such an aerosol can cause distress to the diver and may cause 'salt water aspiration syndrome' (see Chapter 24).

Another problem associated with demand valves is that they may cause pain in the temporomandibular joint; this condition is considered in Chapter 30.

In very cold water the first stage of the regulator may freeze up. This occurs because the air cools as it passes through the first stage and can ice up with the piston frozen in the open position. The problem can be reduced by using a first stage that is designed for operation in cold water.

Because the first stage regulates the pressure to the second stage, the inspiratory effort required to cause a flow does not vary until the cylinder is almost empty. Then, the pressure in the hose to the second stage falls and the flow decreases. The diver's first warning of the cylinder being almost empty would be an increased resistance on inhaling.

Most divers have a pressure gauge connected to the cylinder by a hose which gives them a measure of their remaining air supply. The contents of the cylinder is proportional to the pressure, so the gauge is often called the 'contents gauge'. Divers tend to say they have 50 bars left rather than the volume this represents. A major problem is that a diver who is entranced by the scenery or is concentrating on his task may run out of air because he forgets to check the gauge.

Another common system to prevent divers running out of air is a reserve valve. In operation, it resembles a boiler safety valve – the air escapes to the diver until the cylinder pressure falls to the level at which the reserve valve seats. The remainder of the

air can be released by pulling a lever that opens the reserve valve. The problem with this is that the valve lever may be bumped into the 'on' position, so that the diver uses the reserve of gas without being aware of the valve being opened.

A diver can also use a demand valve with air supplied by a hose from the surface. This equipment, **surface supply breathing apparatus** (SSBA), restricts the diver's range and depth to the length of his air supply hose. Its advantages are that the diver is freed from the cumbersome air tanks, and his air supply can be as large as needed – instead of being restricted by his carrying capacity and need for mobility. The air for SSBA may be stored in large tanks or compressed as required. The use of a compressor, often called a '**Hookah**' system, is economically attractive because the air is compressed to a lower pressure than that required for storage tanks. The major disadvantage is that the diver gambles on the reliability of the compressor.

Two modified forms of SSBA have found support in some circles. In one, a small motor and air compressor is supported on a float on the surface. It supplies air to one or two divers. In the other the diver(s) tow a float that supports an air cylinder. An advantage of these systems is that, if the hoses are short, the divers are unable to reach the depth needed to develop decompression sickness. A significant problem is that the user has no indication of when the gas supply will fail. Also, the users may forget that they are still exposed to the other hazards of scuba diving. In some resort areas people are renting these devices to novices who have had no training and who may be medically unfit to dive.

Safety and protective equipment

The best safety measures available to a diver are correct training and common sense. Almost all accidents are preventable, and the authors do not ascribe to the popularly held belief that these accidents are attributable to an 'Act of God' – they involve human, predictable and thus correctable, mistakes. This point is developed in Chapter 46, where deaths and accidents are considered. Several items of equipment which reduce the hazards of diving, or assist with coping with them, are discussed below.

Emergency air supplies

This can take a variety of forms. In the early days it was common to rely on buddy breathing, but an analysis of diving accident statistics showed that this often did not work in an emergency. The use of a second regulator attached to the scuba set, often called an **octopus rig**, has many supporters. Neither buddy breathing nor an octopus rig will be of use if the diver with gas is not available or is unwilling to cooperate. For this reason a second source of air that is available to each diver without assistance is now favoured. For cave divers this may be a second scuba set. For divers who can reach the surface more easily, a small cylinder of air with an attached regulator – such as the device sold under the name **Spare** ™ – is becoming a popular option. Other people rely on the air contained in their buoyancy vest. Indeed, in some vests there is a separate air supply and a valve that allows the diver to inhale air from the vest.

Thermal protection

This is needed in cold water or on prolonged dives to minimize the risk of hypothermia. This protection is normally provided by insulated clothing which reduces heat loss. The most common protection is a **wet suit**, made from air-foamed neoprene rubber. The water that leaks into spaces between the suit and the diver soon warms to skin temperature. Foamed neoprene has similar insulation properties to wool felt, and its effectiveness is reduced by loss of heat with water movement, and increasing depth. Pressure decreases insulation by reducing the size of the air sacs in the foam; at 30 metres the insulation of a wet suit is about one-third of that on the surface. Compression of the gas in the foam also means that the diver's buoyancy decreases as he goes deeper, but this can be compensated for if a buoyancy vest is worn. If this is not the case, the diver needs to consider reducing his weights, but this will mean he is too buoyant when closer to the surface.The buoyancy and insulation of a wet suit decreases with repeated use.

The other common form of thermal protection is the **dry suit**. This is watertight, and has seals round the head and hand openings; these openings have a waterproof seal to allow the diver to get into the suit. The dry suit allows the diver to wear an insulating

layer of warm clothes. A gas supply and exhaust valve is needed to allow the diver to compensate for the effect of pressure changes on the gas in the suit. The gas can come from the scuba cylinder or from a separate, dedicated supply.

The diver needs training in the operation of a dry suit or he may lose control of his buoyancy. This can lead to an uncontrolled ascent, when the excess of gas expands, speeding the ascent. If the diver trys to swim down the excess gas may accumulate round his legs, and it cannot then be vented off through the exhaust valve. The excess gas can also expand the feet of the suit and cause the diver's fins to pop off, whereupon he may find himself floating on the surface with the suit grossly overinflated – a most undignified posture!

Heat can also be supplied to a diver help him keep warm. The commonly used systems include hot water pumped down in hoses, but various chemical and electrical heaters are also available. External heat supplies are more often used by commercial divers.

Buoyancy compensators

This consists of an air-filled bag attached to the scuba, or an inflatable jacket, called an **ABLJ** (adjustable buoyancy life jacket) worn by the diver. It allows the diver to adjust their buoyancy or to bring them to the surface and support them. The ability to change buoyancy allows the diver to hover in the water and adjust for any factor that causes his density to increase (e.g. picking up an object on the bottom). A buoyancy compensator with a reduced maximum lift is needed in cave and saturation diving. In these situations it may be dangerous to ascend.

The best buoyancy compensators can be inflated from the scuba or a small separate air bottle which can also be used as an emergency air supply. A vent valve is fitted so that the diver can reduce buoyancy by venting gas from the compensator. Divers can lose control of their buoyancy while ascending. As the diver starts to ascend, the gas in the vest expands; thus, the lift increases, as does the rate of ascent. This may lead to the diver imitating a leaping whale, and an air embolism may result from the rapid ascent. The second problem is that the diver surfaces when he should have stopped for decompression.

Depth gauge

A watch and waterproof decompression tables are needed if an unsupervised diver is operating in a depth/time zone where decompression stops may be needed. Electronic, mechanical and capillary gauges are all used as depth gauges by divers. (Capillary gauges measure pressure by the reduction in volume of a gas bubble in a graduated capillary tube.) Some gauges record the maximum depth reached by the diver during the dive, a feature which aids the diver in choosing the correct decompression table. The need to check the accuracy of some types of gauges is often overlooked; faulty gauges have lead to divers developing decompression sickness.

Decompression meters

These have been designed to replace tables and to shorten decompression from a dive where time is spent at several depths. In using decompression tables the time limits and decompression stops are generally calculated assuming that the diver spent all the dive at one of a tabulated selection of maximum depths and a selection of times at this depth. The meters can save decompression time as they calculate the decompression needed for the actual depths reached and the times spent at these depths. During the 1990s the choice of decompression meters has rapidly expanded. The meters now available are electronic, and combine data from the depths and durations of the dive (and any recent dives). The meter has a display that advises the diver when they should ascend to the surface and if they need to pause during the ascent to the surface for excess gas to be eliminated from their tissues. The meter generates this information by comparing the dive conducted with a program and decompression data stored in its memory. The design of these meters is considered in greater detail in Chapter 10, when the reader will be given a better understanding of the decompression theory involved.

Contents gauge

The role of this gauge has been discussed earlier. It indicates the pressure and, by extrapolation, the amount of gas remaining in the supply cylinder.

Communication lines

Because of the risks in diving it is generally considered to be foolhardy to dive without some method of summoning assistance. Most commercial divers do this with an underwater telephone or signal line. However, divers who do not want the encumbrance of a link to the surface can dive in pairs – commonly called a 'buddy pair'. Each has the duty to aid the other if one gets into difficulty. The common problem in the use of the buddy system is to attract the attention of the buddy if he/she is looking elsewhere. The partner can be called to assist if the pair is linked with a buddy line – a light, 2–4 metre-long line that is clipped to each diver. A tug, or the drag of a body, soon attracts attention. The line causes little inconvenience to a considerate, capable pair and is less of a distraction than looking to see if the buddy is alright every few seconds. Some divers oppose buddy lines, placing their faith in other methods of calling for assistance. When diving in caves, a continuous line to the surface is needed. This can be followed if the team of divers becomes disorientated, or when visibility is lost due to torch failure or disturbed silt forms an opaque cloud. In such a situation, each diver should be clipped to the main line.

A buddy line is a simple reliable method of summoning assistance.

A **floating line**, dragging downstream or downtide from the boat, will aid recovery of divers when they surface downstream. Some call this the 'Jesus line' as it saves sinners – the divers who have erred and surfaced down current from the dive boat. This is not needed if a lifeline or pick-up boat is being used, or if the current is insignificant.

A **shot line** or **shot rope** is a line that hangs down from the dive boat to the bottom, with a weight at the bottom end. It may be used to guide the diver to his work and back. It can also be the centre for a circular pattern search. It is often marked with depth markers, which can be used to show the decompression stop depths. The diver can hold onto the line at the depth mark. A **lazy shot** line is a weighted line that does not reach the bottom and is used for decompression stops.

A **lead line** is often used to assist the diver on the surface. It leads from the stern of the boat to the anchor chain, and allows a diver who has entered the water at the stern of the boat to reach the anchor when the current is too strong for them to swim to it.

Dive boats

Boats used for diving range from canoes to large specialized vessels that support deep and saturation diving. The facilities required depend on the nature of the diving, but there are minimum requirements. In some conditions a second safety boat may be needed, perhaps when divers may need to be picked up after drifting away from the main vessel.

Propellor guards, or a safe propulsion system such as a water jet, is desirable if there is any chance of the engine being needed during diving operations.

A **diving platform** or ladder is needed on most boats to facilitate the diver's return from the water. Consideration should also be given to the recovery of an unconscious or incapacitated diver. This can be very difficult from a craft with enough freeboard to be used for diving in a seaway. Recovery into an inflatable craft is often a safer alternative as the body can be dragged, rather than lifted, into the boat. The air-filled hull is also less likely to injure a diver than a rigid hull.

Diving flags, lights or other signals as required by the local maritime regulations should be available. These offer legal, if not physical, protection from the antics of other craft. In most areas boat 'attacks' cause more deaths than shark attacks.

The **first aid kit** and **emergency medical equipment** (see Chapter 48) should be chosen depending on local hazards and the distance from assistance.

EQUIPMENT FOR PROFESSIONAL DIVING

This section deals with the more specialized equipment used by professional and military divers, most of whose tasks involve comparatively shallow depths. The tasks could be conducted with scuba gear of the type described above. Equipment fitted with communications allows the diver to confer with the

surface support. Communications operate better in air, and so are commonly fitted into a helmet or full facemask. In these devices the air flow may either be continuous or on demand.

More specialized equipment is used for some military diving where an element of stealth is required. For these tasks an oxygen rebreathing system which can be operated with no telltale bubbles may be used. In dealing with mines, stealth is again required, to avoid activating the noise or magnetically triggered circuits. If the mine may be too deep for an oxygen set, a rebreathing system with an oxygen/nitrogen mixture may be used.

For even deeper tasks, where oxygen/helium mixtures are used, some method of reducing the gas loss provides cost and logistic savings. This can be achieved by the diver using a rebreathing system or returning the exhaled gas to the surface for reprocessing.

Breathing systems

For most tasks the professional diver is working in a small area for long periods, and consequently does not need the mobility of the scuba diver. His air normally comes from the surface in a hose, either supplied from storage cylinders, or compressed as needed by a motor-driven compressor. The cable for the communication system and a hose connected to a depth-measuring system are often bound to the air hose. Another hose with a flow of hot water may also be used to warm the diver. It is normal for the diver to have an alternative supply of air in a cylinder on his back; this supplies him with air if the main supply should fail.

Free flow systems

These were used in the first commercial air diving apparatus. The diver was supplied with a continuous flow of air that was pumped down a hose to him by assistants turning a hand-operated pump. These pumps have been susperseded, but the same principle is still in use. In the most common system, called **standard rig**, the diver's head is in a rigid helmet, joined onto a flexible suit that covers his body. The diver can control his buoyancy by controlling the amount of air in the suit. The main problem with the system is that the flow of fresh air must be sufficient to flush carbon dioxide from the helmet. The flow required to do this is about 50 L/min measured at the operating depth – well in excess of that needed with a demand system.

The other problem associated with free flow systems and the high gas flow is the noise that this generates. In the early days the diver was also exposed to the risk of a particularly unpleasant form of barotrauma. If the pump or air supply hose broke, the pressure of the water tends to squeeze the diver's soft tissues up into his helmet. Fitting a one-way valve, to stop flow back up the hose stops this. For deep dives, where oxygen/helium mixtures are used, the cost of gas becomes excessive, and a method of reducing gas consumption may be fitted. For example, the U.S. Navy Mark 12 rig can be fitted with a rebreathing circuit incorporating a canister of carbon dioxide absorbent to purify the gas. The gas flow round the circuit is generated by a venturi system that does away with the need for valves to control gas flow. The rig is converted into a rebreathing system, which has a separate set of problems that are considered in a later section.

Demand systems

These have been developed to gain the reduction in gas consumption expected from a demand valve system. They also have the facility for the diver to talk underwater. Several types of equipment are in common use. One type uses a full facemask that seals round the forehead, cheeks and under the chin. The back of the diver's head may be exposed to the water (as in the **Aga**™ mask), or covered with a wet suit hood that is joined onto the facemask, as with the **Bandmask**™ and derivatives. Another type is fitted in a full helmet, in which an oronasal mask reduces rebreathing of exhaled air. The helmets are often less comfortable than the facemasks, but provide better thermal and impact protection. In each case a demand valve reduces the gas consumption compared to a free flow system. These helmets may also be used at greater depths, where helium mixtures are used. A return hose may be used to allow collection of the exhaled gas at the surface for reprocessing.

All the systems mentioned above have the major advantage, compared to a demand valve held in the mouth, of reducing the chance of the diver drowning. This is important if he loses consciousness or has a convulsion while breathing oxygen. Some decompression schedules allow the diver to breathe oxygen to reduce decompression time. The increased safety and the advantages of a clear verbal communication system has led to the adoption of helmets by most diving firms.

Sets that use helmets and a full facemask reduce the risk of drowning, and can allow the diver to converse with people on the surface.

Rebreathing systems

The major advantage of rebreathing systems is their economy of gas usage. This is shown by the following example. In a demand system, a diver on the surface may have a respiratory volume of 20 l/min and an oxygen consumption of 1 l/min. He inhales 4 litres of oxygen per minute, and exhales 3 litres. This is lost into the water and therefore wasted. A diver at 40 metres (5 ATA) consuming the same amount of oxygen, would have a minute volume of 20 litres at that depth, which is equal to 100 litres at the surface. This contains 20 litres of oxygen, 19 of which are exhaled into the water and wasted. The oxygen wastage inherent in demand breathing apparatus is considerable even on the surface, and increases with depth. With closed-circuit rebreathing equipment, the diver can extract all the oxygen in the supply. With semi-closed rebreathing systems the wastage can be reduced to a fraction of that with a demand system.

The main disadvantages of rebreathing systems is that they are more complex than free flow or demand systems. In particular, they have a carbon dioxide-absorbing agent which is prone to failure.

Because of the similarity between semi-closed and closed rebreathing sets their common features will be discussed. The points peculiar to each type are then discussed separately.

The usual gas flow pattern found in rebreathing sets are shown in Figure 4.3. The movement of inhaled and exhaled gas is controlled by valves as the gas flows round the circuit. Gas enters from a storage

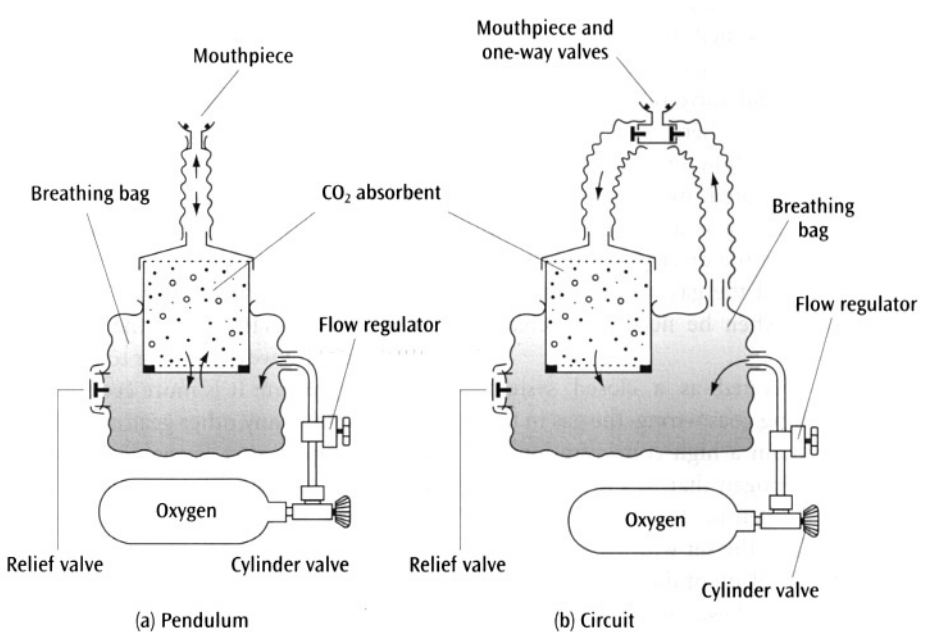

Figure 4.3 *Rebreathing sets.*

cylinder, through a flow-regulating device into the breathing bag or counterlung, and then to the diver. In most sets the flow regulator adds gas automatically. A manually controlled valve allows the diver to add extra gas. In this way the diver can increase his bouyancy by adding the volume of gas in his breathing bag; he can also add fresh gas to dilute any contaminating gas in the set.

The breathing bag or counterlung is a gas storage bag that expands and contracts as the diver breathes. It normally incorporates a relief valve that releases surplus gas into the water and prevents excess pressure building up. Venting of excess gas is needed in closed rebreathing sets when the diver ascends and the gas in the counterlung expands. In semi-closed circuit sets there is a loss of excess gas through the relief valve.

The carbon dioxide absorbent is usually a mixture of calcium and sodium hydroxides, both of which react with carbon dioxide to form carbonates and water.

$$M(OH)_2 + CO_2 \rightarrow MCO_3 + H_2O$$

Closed-circuit oxygen systems

These are the simplest closed rebreathing sets. The breathing bag is filled with oxygen from the cylinder and as oxygen is consumed, the volume of the bag decreases. In some sets a trigger mechanism that operates like a demand valve releases more gas into the bag. In other sets there is a mechanism that releases a continuous flow of oxygen into the circuit. A manually operated method of adding oxygen to the breathing bag is also usually fitted. This will be needed when the diver puts the set on, when he goes deeper and the gas in the breathing bag is compressed, or when he needs to increase his buoyancy.

The set can be operated as a closed system because, unless something goes wrong, the gas in the breathing bag will contain a high concentration of oxygen, diluted with nitrogen that was in the lungs and body of the diver when he put the set on. It is standard practice to flush the set with oxygen at set intervals to prevent a build-up of diluting gases.

Possible problems with these sets include carbon dioxide toxicity if the absorbent fails, dilution hypoxia if the oxygen is impure or the diver neglects to flush nitrogen from his lungs and the counterlung, and oxygen toxicity if he descends too deep. To reduce the risk of oxygen toxicity a depth limit of about 8 metres is often imposed on the use of these sets.

A closed oxygen rebreathing apparatus has the particular advantage that a small set may provide a long endurance. A set weighing less than 15 Kg can allow dives of over 2 hours. The lack of bubbles and quietness of this set is also important in some specialized roles such as clandestine operations.

Rebreathing sets are quieter and have a greater endurance than compressed-air sets. The extra hazards and costs involved restrict their use.

Closed-circuit mixed gas systems

These are comparatively new types of sets in which oxygen and a diluting gas are fed into the breathing loop at rates required to keep the oxygen partial pressure within safe limits, and to provide an adequate volume of the mixture. The fundamental features of this system are shown in Figure 4.4. As with the oxygen set, the diver inhales gas from the storage bag and exhales through the carbon dioxide absorber back into the bag. As he uses oxygen the partial pressure of oxygen in the bag will fall, and this fall is detected by the oxygen sensor. At a certain level a valve lets more oxygen into the circuit. If the volume of gas in the bag falls, a volume sensor triggers a second valve that adds diluting gas. Manual controls, and readouts indicating the oxygen concentration, may be fitted to allow the diver to override the controls if the automatic valves fail. In some sets he can also change from normal gas mixture to high oxygen mixtures to shorten the decompression time.

This system would appear to be the most efficient breathing system. It is more economical in terms of gas usage than any other gear apart from the oxygen breathing apparatus. It enables a diver to go deeper for longer, and with less encumbrances than other equipment. As an example of the efficiency of this type of equipment, it has been stated that in a helium saturation dive program involving a prolonged series of dives to 180 metres, the cost of helium for semi-closed diving apparatus was $US 40 000. With closed rebreathing apparatus, the cost would have been less

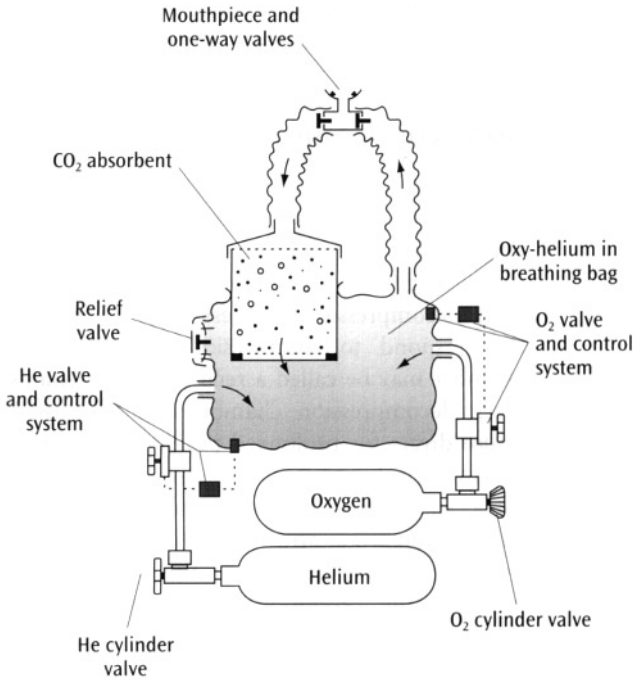

Figure 4.4 *Closed-circuit mixed gas rebreathing set.*

than $US 1000. However, these advantages must be balanced against the greater cost and, more importantly, the complexity of the system, which can lead to fatal malfunctions.

Semi-closed rebreathing systems

These provide some of the savings in gas achieved by the closed systems while avoiding the depth limits of the oxygen sets and the complexity of the closed mixed gas sets. The basic system as shown in Figure 4.3 can also be used for a semi-closed system, the main change being that a gas mixture is used instead of oxygen. The other component of the mixture, generally nitrogen or helium, dilutes the oxygen and increases the depth capability of the equipment.

In a typical semi-closed rebreathing system the gas flow and composition are chosen for maximum efficiency for the proposed dive. First, the composition of the gas is chosen, with as high an oxygen concentration as possible. A partial pressure of oxygen less than or equal to 2 ATA is generally used to prevent oxygen toxicity at the maximum depth. This level may be changed depending on the duration of exposure. The flow is then chosen so that the diver will receive sufficient oxygen while working on the surface.

These steps can be used in reconsidering the dive to 40 metres described above. A 40 per cent oxygen mixture could be chosen (at 5 ATA the partial pressure of oxygen would be 2 ATA). If the maximum expected oxygen consumption on the surface was 3 l/min, then the flow would be set at about 12 l/min.

The oxygen concentration in the diver's inspired gas is an equilibrium between flow into the system, the diver's consumption and loss through the relief valve. It ranges from close to that in the supply bottle when the diver is resting, down to about 20 per cent when the diver is working at the maximum expected rate. This ensures that the diver does not become hypoxic if he needs to surface rapidly.

A variety of methods have been devised to meter the fresh gas into the circuit. The example used above assumed a constant flow; this may be obtained with a jet or valve if the flow through the metering device meets certain design conditions. In other sets the flow is determined by the amount of gas the diver breathes, a small portion of each breath being lost into the water.

The semi-closed system with a flow of 12 l/min gives an eight-fold saving of gas compared with a demand system when the diver is consuming 1 litre of oxygen per minute. This saving would increase if

the scuba diver was working harder and consuming more air. The high oxygen concentrations in these sets means that the diver may not absorb as much nitrogen as he would had he been breathing air. This can lead to a decrease in the decompression needed.

Military divers are the main users of semi-closed sets, as the reduced gas flow with these sets means that they can be designed to make little noise. If they are constructed from non-magnetic materials they can also be used for dives near mines.

The main problems with these sets have much in common with closed-circuit equipment. For example, carbon dioxide accumulation can occur if the absorbent fails, while oxygen toxicity can occur if the diver exceeds his depth limit, or uses a mixture containing too much oxygen. Hypoxia may result if the gas flow decreases, if the diver works harder than expected, or if a mix with too little oxygen is used.

Chambers, habitats and underwater vehicles

Divers may use several special types of vehicles and living facilities, including vehicles which are hoisted and lowered to transport them to and from deep dive sites, propelled vehicles to increase the diver's range and endurance, and machines to carry underwater equipment. The accomodation to be considered include underwater houses and pressurized houses at the surface.

Submersible decompression chambers (SDC)

These are often called personnel transfer capsules, and are used to transport divers and any attendants from the surface to the work site. They may also be used as a relay station and store for gas and equipment. The most complex SDC may carry the diver at constant pressure from a deck decompression chamber to his work site, and back. The simplest SDC consists of a bell chamber that is open at the bottom and allows the diver to decompress in a dry environment, exposed to the same pressure as the surrounding water.

Habitats are underwater houses that accommodate divers in air or gas-filled environments. They are used by divers to rest between excursions. Divers have lived in some of these habitats for weeks at a time.

Deck decompression chambers (DDC)

These can be small and used for surface decompression, a procedure that allows a diver to be decompressed in a dry chamber instead of in the water. Larger chambers can be used to treat divers with decompression sickness and other diseases that respond to compression, in this case the chamber may be called a **recompression chamber**. Deck decompression chambers are also used to house divers for prolonged periods under elevated pressure. In this case they are carried to their work by a submersible decompression chamber or a small submarine which keeps the diver in a pressurized environment. At the end of the job, possibly after several weeks, the pressure in the DDC is lowered slowly to return the diver to atmospheric pressure.

Transport vehicles

These can carry the divers at normal atmospheric pressure, at ambient pressure in a dry environment, or in a wet environment. These include vehicles towed by a boat. A small motor and propellor that pulls the diver along gives increased speed with reduced effort. Some submarines have a lock system to allow divers to leave and enter underwater.

A **1 atmosphere diving equipment** such as the **JIM** suit seals the diver in a pressure-resistant compartment. It has flexible arms with tools on the 'hands' to allow work underwater. The early types of suit had legs that gave the diver the ability to walk on firm surfaces if there was little current. However, the diver had no control in mid water and had to be lowered and hoisted from the surface. In other designs, such as the **WASP** system, the diver controls a set of propellers, making him a cross between a diver and a one-man submarine.

Life support systems

These are required to provide the occupants of all these vehicles, habitats and chambers with a

respirable atmosphere. They work on the same principles as a diver's breathing apparatus, and in some vehicles the diver may even be wearing a breathing apparatus. The system must be self-contained for transport vehicles, but for habitats and submersible decompression chambers the gas is generally supplied from the surface.

Gas from the surface can be supplied in a free flow and escape from the bottom, or be recirculated through a purifying system. Simple gas-purifying systems can involve a hand-powered pump to force gas through a carbon dioxide absorption canister with a manually operated system for adding oxygen. The most complex systems are those found on large submersibles, nuclear submarines and chambers used for deep saturation dives. These have automatic closed systems with provision for removing trace contaminants and odours; they also regulate temperature, pressure and humidity.

Gas reclaimers

These are mainly used to recover helium to be reused again, and in doing so they help to reduce costs. One type cools the gas until the other gases are liquified, leaving pure helium to be stored and used again. Other types use a chromatographic technique to separate the gases.

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5

Undersea environments

CARL EDMONDS

INTRODUCTION

For the diver who is adequately trained and physically fit, who is aware of the limitations of his equipment, and who has given attention to the specific requirements of different environmental diving conditions, the sea is rarely dangerous. Nevertheless, it can be hazardous and unforgiving if attention is not paid to all of these factors.

The induction of fear in the inexperienced diver, and physical stress in the more skilled diver, is only appreciated when one examines each specific environmental situation and is instructed in the specialised techniques recommended to cope with them. These techniques will be referred to in this and other chapters. The reason for including them in a medical text is that, unless the physician comprehends the problems and dangers, the diving medical examinations and assessments of diving accidents will be less than adequate.

Some aspects of the environments have physiological and pathological sequelae, and therefore have specific chapters devoted to them, e.g. the effects of cold (Chapters 27 and 28), altitude and fresh water diving (Chapter 2), explosives (Chapter 34), depth (Chapters 2, 15, 20, 46 and 68), marine animal injuries (Chapters 31 and 32), and these receive little mention here. Others, that are covered more fully in diving texts, are considered in this chapter.

WATER MOVEMENT

Because of the force of water movement, a diver can become a hostage to the sea.

White water

White water – the foaming effect of air bubbles – dramatically interferes with both visibility and buoyancy, as well as implying strong currents or turbulent surface conditions. A diver in white water is a diver in trouble, and under these conditions the recommendation is usually to dive deeper.

Surge

Surge is the to-and-fro movement of water that produces disorientation and panic in inexperienced divers, who often try to swim against it. Others use the surge by swimming strongly with it, then hold on to rocks or corals when the surge moves in the opposite direction.

Occasionally there is a continuous water flow, because of a **pressure gradient** through a restricted opening, which can siphon and hold (or even extrude) the diver. This is encountered in some caves, blue holes or rock areas near surf (an underwater 'blow hole'), in man-made structures such as the water inlets in ships hulls, and outlets in dams and water cocks (taps). The pressure gradient may slowly draw the diver into its source and then seal him, like a bath plug. Protection is by avoiding the area or covering it with a large grating.

Tidal currents

These are very important to the diver and, if used correctly, can take him where he wants to go. Otherwise they are likely to take him where he does not want to go. The latter event can be both embarrassing and terrifying, and it can also be very physically demanding.

Divers are frequently lost at sea because of currents. Sometimes these can be vertical as well as horizontal, and are unable to be combated by swimming or buoyancy. Certain popular diving areas, such as at Palau, Ras Muhammad, the Great Barrier Reef and Cozumel experience these, and multiple fatalities are not uncommon.

Divers sometimes relate their successful swims against 4- to 5-knot currents, but in fact, the average fast swim approximates 1.2 knots. For brief periods, it may be possible to reach up to 1.5 knots, and the average swimmer can make very slow or no progress against a 1-knot current. A half-knot current is tolerable, but most divers experience this as a significant problem, and so it is. They tend to exaggerate its speed as the hours go by, and especially during the après-dive euphoria.

Tidal currents are usually much faster on the surface than they are on the seabed, due to friction effects. A helpful observation is that the boat will usually face the current with its anchor upstream and the stern of the boat downstream. Any diver worth his salt knows that it is safer to swim against the current for the first half of his usable air, and allow the current to bring him back to the boat for the second half of the dive. The **'half tank' rule** is worked out by taking the initial pressure, say 200 ATA, subtract the 'reserve' pressure, say 40 ATA, i.e. 160 ATA and divide this by 2, i.e. 80 ATA. Thus, for this example, 80 ATA is used on the outward trip, and the return is then made with ample air to allow for misadventure (such as navigational error).

Untrained divers tend to make unplanned dives. They submerge and 'just have a look around'. While they are having their look around they are being transported by the current, away from the boat, at a rate of 30 metres (100 ft) every minute in a 1-knot current. When they consider terminating the dive, after they have used most of their air, they have a very hard return swim against the current. They surface because of their diminished air supply, well downstream from the boat, and having to cope with a faster, surface current.

This is a very difficult situation, and far more hazardous than that of the experienced diver who used the 'half tank' rule, who surfaced upstream from the boat and floated back to it – but who also had enough air to descend underwater and return with ease if he wished, or to rescue his companion.

The **lines** attached to the boat are of extreme importance when there are currents. First, there is the **anchor line**, and this is the recommended way to reach the seabed upstream from the boat. The anchor chain should not be followed right down to the anchor because this may occasionally move, and can cause damage to the adjacent divers. More than one diver has lost an eye from this 'freak accident'. How may the diver reach the anchor line? A line may be attached to the top of the anchor line, with the other end to the stern of the boat. It should have enough play in it to allow divers to sit on the side of the boat and to hold it with one hand – the hand nearest the bow of the boat – while the other hand is used to keep the facemask and demand valve intact. On entry, the diver ensures that he does not let go the line; he then pulls himself forward to the anchor line, and descends.

Perhaps the most important line, if there is a current, is a **float** or **'Jesus' line**. This drags 100 metres or more behind the boat, in the direction of the current, and has some floats to ensure that it is always visible to divers on the surface. It is often of value to have one diver on this line while the others are entering the water. He virtually acts as a backstop to catch the odd stray diver who has not followed instructions and is now floating leeward. The Jesus line is also of immense value at the end of the dive when divers have, incorrectly, exhausted their air supply or when they come to the surface for some other reason and find themselves behind the boat. This would not have happened had a dive plan been constructed and followed correctly. Occasionally, however, it does happen to the best divers, and it is of great solace to realize that the Jesus line is there, and ready to save the 'sinner' – irrespective of their religious persuasion!

Even divers who surface only a short way behind the boat in a strong surface current may find that it is impossible to make headway without a Jesus line. Under these conditions they can descend and use their compass to navigate back to the anchor line or inflate the buoyancy compensator, attract the attention of the boat lookout, and hope to be rescued.

Buddy breathing, while swimming against a strong current, is often impossible. Even the octopus (spare) regulator causes problems at depth, or when two people are simultaneously demanding large volumes of air, typical of divers swimming against a current. An alternative air supply (pony bottle) is of value, if it has an adequate capacity.

In dive planning, there should be at least one accessible fixed diving exit, easily identifiable, that serves as a safe haven. This may be an anchored boat, in areas with tidal currents. The safety boat is a second craft – not anchored – and this, like any boat that is used among divers, needs a guard on its propeller. A pressure-tested distress flare (smoke) may be needed to attract the safety boat. Another recently developed rescue device is an inflatable elongated 2 metre (6 ft) bag, called the 'safety sausage'.

There are other problems with currents, and these are especially related to general boat safety and ensuring that there is a stable anchorage. When the current is too strong or the depth too great for an anchored boat, a float or **drift dive** may be planned, but this requires extreme care in boat handling. Divers remain together and carry a float to inform the safety boat of their position. It allows the surface craft to maintain its position behind them as they drift.

The concept of 'hanging' an anchor, with divers drifting in the water near it and the boat being at the mercy of the elements, has little to commend it. The raising of the diver's flag under such conditions, although it may appease some local authorities, is often not recognized by the elements, reefs, or other navigational hazards, including other boats which are moored.

Some currents are continuous, e.g. the standing currents of the Gulf of Mexico, the Gulf Stream off Florida and the Torres Strait, but tidal currents are likely to give an hour or more of slack water. At these times diving is usually safer and also more pleasant as there is less sediment to reduce visibility. To ascertain the correct time for slack water, reference has to be made to the tidal charts for that area. The speed of the current can be predicted by the tidal height.

Surf

Entry of a diver through the surf is loads of fun to an experienced surf diver. Otherwise, it can be a tumultuous, moving experience, and is a salutary reminder of the adage 'he who hesitates is lost'. The major problem is that people tend to delay their entry at about the line of the breaking surf. The diver, with all his equipment, is a far more vulnerable target for the wave's momentum than any swimmer.

The recommendation is that the diver should be fully equipped before entry into the surf, and not readjust facemasks and fins until he is well through the surf line. The fins and facemask must be firmly attached beforehand, as it is very easy to lose equipment in the surf. The diver walks backwards into the surf, looking over his shoulder at the breakers and also towards his buddy. The facemask and snorkel have to be held on during the exposure to breaking waves. The regulator must be attached firmly to the jacket, with a clip, so that it is easily recoverable at all times.

When a wave does break, the diver presents the smallest possible surface area to it, i.e. he braces

against the wave, sideways, with his feet well separated, and crouches and leans, shoulder forward, into the wave. As soon as possible he swims (in preference to walking) through the wave area, going under the waves. If he has a float, then this is towed behind. It should never be pushed between the diver and the wave.

Exit should be based on the same principle as entry. However, the wave may be used to speed the exit by swimming immediately behind it or after it has broken. The float then goes in front of the diver.

ENTRAPMENT ENVIRONS

Being held underwater, with a limited air supply, will result in drowning. A variety of materials can trap the diver, including kelp, lines (even 'safety' lines), fishing nets and fishing lines. If there is not a compromised air supply, then a buddy, a calm state of mind and a diving knife or scissors, will cope with most circumstances. All three may be needed.

Kelp

Giant members of this large brown algae or seaweed may grow in clear water to depths of 30 metres, but growth is less in turbid or unclear water. Kelp usually grows on hard surfaces, e.g. a rocky bottom, a reef or wreck. It is of interest commercially as it is harvested to produce alginates, which are useful as thickening, suspending and emulsifying agents, and also in stabilizing the froth on the diver's glass of beer!

Kelp has caused many diving accidents, often with the diver totally bound up into a 'kelp ball' which becomes his coffin. The danger of entanglement is related to panic actions and/or increased speed and activity of the diver while in the kelp bed.

Divers who are accustomed to kelp diving usually take precautions to ensure that there is no equipment that will snag the strands of kelp, i.e. they tend to wear knives on the inside of the leg, tape the buckles on the fin straps, have snug quick-release buckles, and do not use lines. They descend vertically feet first, to where the stems are thicker and there is less foliage to cause entanglement. The epitome of bad practice in kelp diving is to perform a head-first roll or back roll, as it tends to result in a 'kelp sandwich with a diver filling'.

The kelp is pushed away by divers as they descend and ascend, i.e. they produce a clear area within the kelp, into which they then move. They ensure that they do not run out of air, as this situation will produce more rapid activity. If they do get snagged, they avoid unnecessary hand and fin movements. Kelp can be separated either by the use of a knife or by bending it to 180° when it will often snap (this is difficult to perform while wearing gloves). It is unwise to cut kelp from the regulator with a knife without first clearly differentiating it from the regulator hose. Some divers have suggested biting the strands with one's teeth. This may be excellent as regards dietary supplementation (the kelp being high in both B vitamins and iodine), but it does seem a little over dramatic!

Kelp can be useful in many ways to the diver. It allows a good estimate of clarity of the water by assessing the length of plant seen from the surface. The kelp blades tend to lie in the direction of the prevailing current. In kelp beds there is usually an abundance of marine life, and the kelp provides other benefits such as dampening wave action both in the area and on the adjacent beach. It can also be used as an anchor chain for people to use when they are equalizing their ears, as well as to attach other objects such as floats, diver's flags, surf mats and specimen bags.

Kelp does float, and it can often be traversed on the surface by a very slow form of dog paddling or crawl, i.e. one actually crawls along the surface of the water, over the kelp. This can only be done if the body and legs are kept flat on the surface, thus using the buoyancy of both the body and the kelp, and by using the palms of the hands to push the kelp below and behind as one proceeds forward. Any kicking that is performed must be very shallow and slow.

Cave and wreck diving

These produce dangerous situations for recreational divers. Caves are always more complex than they first appear, and planning involves not only primarily the setting of goal oriented objectives, but also the delineation of maximum limits. Maximum safe depths for cave diving with compressed air is 40 metres.

The diver descends, often through a small hole, passes down a shaft, goes around a few bends and is faced with multiple passages, in total darkness. Under these conditions, and to make this particular type of diving safe, it is necessary to be accompanied by a diver who has considerable cave experience – in that cave – and whose judgment is trustworthy. It is equally important that the equipment is both suited to cave diving and is totally replaceable with spares during the dive. Apart from the obvious environmental and navigational difficulties inherent in diving through a labyrinth of caves, there are added specific problems.

Air pockets found in the top of caves are sometimes non-respirable, due to low oxygen and high carbon dioxide levels. Sometimes the roof is supported by the water, and when this is replaced by air from the divers tanks, it can collapse.

Safety in cave diving is not usually achievable by immediate surfacing; thus, all necessary equipment must be duplicated. The minimum extra safety equipment includes a compass, two lights, a safety reel and line. It is a diving axiom that entry into a cave is based on the presumption that the return will have to be carried out in zero visibility.

For **visibility**, each diver takes two lights; however, other factors can interfere with their value. A great danger in cave diving is the silt which can be stirred up when the diver swims along the lower part of the cave. If there is little water movement, the clay silts can be very fine and easily stirred up. It is for this reason that the fins should be small ones, and the diver tries to stay more than a metre away from the bottom of the cave. Visibility can be totally lost in a few seconds as the silt curtain ascends – and it may remain for weeks. Sometimes this is inevitable as the exhaled bubbles dislodge silt from the ceiling. Mixing of salt and fresh waters also cause visual distortion and blurring.

The usual equipment includes double tanks manifolded together, making a common air supply, but offering two regulator outlets. With the failure of one regulator, the second one may be used for the air supply – or as an octopus rig. The second regulator must have a long hose, as often divers cannot swim next to each other. Because of space limitations, buddy breathing is often impractical under cave conditions. An extra air supply (pony bottle) is a bonus. The ideal equipment for recreational divers to explore caves is a reliable compressed air surface supply, with a complete scuba back-up rig.

All the instruments should be standardized, e.g. the watch goes on the left wrist, the depth gauge above it, the compass on the right wrist, and the contents gauge or console (this can include a contents gauge, decompression meter, dive profile display, compass) attached to the harness under the left arm. The gauges and decompression must be modified for freshwater and altitude, if these are applicable. The knife is strapped to the inside of the left leg, to prevent entanglement on any safety lines.

The buoyancy compensator is often bound at the top, to move the buoyancy centre more towards the centre of gravity (as cave divers do not need to be vertical with the head out of water). There is no requirement for excess buoyancy, as safety in cave diving is not usually equated with a direct ascent. Thus, any carbon dioxide cylinders should be removed and replaced with exhausted ones to prevent accidental inflation of vests. A principle of cave diving is that safety lies in retracing the entry path by the use of lines and not as in the normal open ocean diving, by ascent.

The **techniques** of cave diving are also very stereotyped. Specialized training in cave diving, dive planning, the use of reels and lines, and the lost diver protocols, are essential. Usually, no more than three divers should go on a single dive, and on completion of the dive, each should have a minimum of one-third of his initial air supply. If there is water flow within the cave, and the penetration is with the flow, this air supply is not conservative enough – as the air consumption is greater returning against the current.

Vertical penetrations need a heavy shot line moored or buoyed at the surface and weighted or fixed at the bottom. The reel is used for horizontal penetrations, not vertical. Otherwise entanglement is likely with rapid ascents, especially if divers precede the lead diver. Thin non-floating lines especially cause entanglement if they are allowed to be slack.

Most people who have difficulties with cave diving have not followed the recommended rules, and unfortunately cave diving problems tend to lead

to multiple fatalities. The number one enemy of cave divers is panic.

Wreck diving has potentially similar problems to some cave and ice water diving. In addition, it has the hazards of instability of the structure and the dangers of unexploded ordnance, sharp objects, and toxic cargo and fuel.

Silt in wrecks is usually heavier than that in still water caves. Thus, the sudden loss of visibility that can occur when silt is stirred up may be less persistent. The diver should ascend as far as is safe, and wait until the silt cloud settles down.

Cold water and under-ice diving

In these situations the obvious problem is that of hypothermia. It is so obvious that most people will avoid it by the use of dry suits, or efficient wet suits. (See Chapters 27 and 28 for the effects of a cold environment on physiological performance.)

A major difficulty is the tendency of many single hose regulators to freeze, usually in the free flow position, after about 20–30 minutes of exposure to very cold water (less than 5°C). This is aggravated if there is water vapour (potential ice crystals) in the compressed air and if there is a rapid expansion of air, which produces further cooling in the regulator's first and second stages. The first stage or the second stage may then freeze internally.

Expansion of air as it passes from the high tank pressure to the lower pressure demand valve and then to environmental pressures (adiabatic expansion) results in a drop in temperature. It is therefore not advisable to purge regulators if exposed to very cold temperatures. The freezing from increased air flow is also produced with hyperventilation or panic. Octopus rigs become more dangerous to use under these conditions, or at great depth, because of the increased air flow. An emergency air source (pony bottle) has replaced buddy breathing and octopus rigs.

'External' ice is formed in and around the first (depth-compensated) stage of the regulator, blocking the orifice and interfering with the spring. Moisture from the diver's breath, or water in the exhalation chamber of the second stage, may also freeze the demand mechanism, causing free flow of gas or 'internal' freezing with no flow.

Modifications designed to reduce freezing of the water in the first stage, include the use of very dry air and the replacement of first-stage water with silicone, oils or alcohols, which require lower temperatures to freeze, or with an air flow from the regulator. The newer, non-metallic second stages are less susceptible to freezing. Despite all this, regulator freezing is common in polar diving. Surface supply with an emergency scuba or twin tank/twin regulator diving, as with cave diving, is probably safer. It must be presumed, in under-ice diving, that the regulator will freeze.

Under ice there is little use for snorkels, and so these should be removed to reduce the likelihood of snagging. Rubber suits can become sharp and brittle. Zippers are best avoided because they freeze and may also allow water and heat exchange. Buoyancy compensators should be small and with an independent air supply.

As a general rule, and if well-fitting dry suits are unavailable, the minimum thickness of the neoprene should increase with decreased water temperatures:

- at <5°C, use a 9 mm-thick wet suit;
- at <10°C use 7 mm;
- at <20°C use 5 mm; and
- at <30°C use 3 mm.

Hood, gloves and booties should be of a considerable thickness, or heat pads can be used.

Unheated wet suits do not give sufficient insulation at depth (beyond 18 metres) when the neoprene becomes too compressed and loses much of its insulating ability. Non-compressible wet suits, inflatable dry suits or heated suits are then required. In Antarctic diving, in order to gain greater duration we had to employ a wet suit or other thick clothing under a dry suit.

Ice diving is in many ways similar to cave diving. It is essential that direct contact must always be maintained with the entry and exit area. This should be by a heavy-duty line attached to the diver via a bowline knot. The line must also be securely fastened at the surface, as well as on the diver. The dive should be terminated as soon as there is any suggestion of either shivering, diminished manual dexterity, other effects of cold, or a reduced gas supply.

The entry hole through the ice should be at least two divers wide. Allowing room for only one diver to enter ignores two facts. First, it tends to close over by

freezing. Second, two divers may require to exit simultaneously. There should be a surface tender with at least one standby diver. If the penetration under the ice is in excess of a distance equated with a breath-hold swim, then a back-up scuba system is a requirement. A bright light, hanging below the surface at the entry hole, is also of value.

DEEP DIVING

'Divers do it deeper' represents a problem with ego trippers and a challenge to the adventure seekers. Unfortunately, the competitive element sometimes overrides logic, and divers become enraptured, literally, with the desire to dive deeper. They then move into a dark, eerie world where colours do not penetrate, where small difficulties expand, where safety is further away, and where the leisure of recreational diving is replaced with an intense time urgency.

Beyond the 30-metre limit the effect of narcosis becomes obvious, at least to observers. The gas supply is more rapidly exhausted. Buoyancy, due to wet suit compression, has become negative – with an inevitable reliance on fallible equipment, such as the buoyancy compensator. The reserve air supply lasts less time, and the buoyancy compensator inflation takes much longer and uses more air. Emergency procedures, especially free and buoyant ascents, are more difficult. The decompression tables are less reliable, and ascent rates become more critical.

Many of the older, independent instructors would only qualify recreational divers to 30 metres. Now, with instructor organizations seeking other ways of separating the diver from his dollar, specialty courses may be devised to entice the diver to 'go deep'.

NIGHT DIVING

Because of the impaired visibility, extra care is needed for night diving. Emergency procedures are not as easy to perform as during the day. There is also a greater fear at night. For inexperienced divers it is advisable to remain close to the surface, the bottom or some object (anchor, lines, etc.). Free swimming mid-water, and without objects to focus on, causes many divers to be aprehensive.

Preferably the site should be familiar, at least in daylight, without excessive currents or water movements and with easy beach access, or diving between the boat and the shore. From a boat entry the diver sometimes encounters surface debris that was not obvious.

Any **navigational aid** needs to be independently lighted. This includes the boat, the exit, buoys, buddies, etc. A compass is usually required. A chemical luminous/flourescent light should be attached firmly to the tank valve, and at least two reliable torches should be carried. The snorkel should have a fluorescent tip. A whistle and a day/night distress flare are sometimes very valuable for summoning the boatman, who does not have the same capabilities of detecting divers at night as he/she does during day-time.

Signals include a circular torch motion (I am OK, how about you?) or rapid up-and-down movements (something is wrong). The light should never be shone in a diver's face, as it blinds him momentarily. Traditional signals can be given, by shining the light onto the signalling hand.

Marine creatures are sometimes more difficult to see at night. Accidental contact with a submerged stingray and needle spine sea urchins is more likely.

ALTITUDE DIVING

The conventional idea of diving is that a diver descends from the sea surface (1 ATA) and returns when he has finished his dive. A diver might have to dive in a mountain lake where the pressure on the surface is less than 1 ATA. The problems encountered with this type of diving are related to the physics of altitude.

For simplicity's sake, the following description is based on the useful, but questionable, traditional belief that the ratio between the pressure reached during the dive and the final pressure, determines the decompression required. If this ratio is less than 2:1, then a diver can ascend safely without pausing during ascent. This means that a diver from the sea surface (1 ATA) can dive to 10 metres (2 ATA) and

ascend safely. A diver operating in a high mountain lake, surface pressure 0.5 ATA, could only dive to 5 metres (1 ATA) before he had to worry about decompression. This statement ignores the minor correction required in a freshwater lake (freshwater is less dense than salt water).

Another pressure problem occurs when a diver, who dives from the sea level, travels by air or goes up into the mountains after his dive. For example, a 5-metre dive (1.5 ATA) could be followed by an immediate ascent to a pressure of 0.75 ATA, with little risk. Deeper dives or altitude exposure may require the diver to pause at sea level if he is to avoid decompression sickness. If the diver ascends, in a motor vehicle or airplane, the reduced pressure will expand 'silent' bubbles or increase the gas gradient so as to produce larger bubbles, thus aggravating the diseases of pulmonary barotrauma and decompression sickness.

Another problem of diving in a high-altitude lake is the rate at which a diver may have to exhale during ascent. A diver who ascends from 10 metres (2 ATA) to the surface (1 ATA) would find that the volume of gas in his lungs has doubled. Most divers realize this and exhale at a controlled rate during ascent. They may **not** realize that a similar doubling in gas volume occurs in the last 5 metres of ascent to the surface, where the pressure is 0.5 ATA. The same effects are encountered with buoyancy control, which can more rapidly get out of control at altitude.

The diver's equipment can also be affected or damaged by high altitudes. Some pressure gauges only start to register when the pressure is greater than 1 ATA. These gauges (Bellows and Bourdon tube types) would try to indicate a negative depth, perhaps bending the needle, until the diver exceeded 1 ATA. Thus, the dive depth would have to reach over 5 metres at an altitude of 0.5 ATA, before the gauge even started measuring.

The other common depth gauge, a capillary tube, indicates the depth by an air/water boundary. It automatically adjusts to the extent that it always reads zero depth on the surface. The volume of gas trapped in the capillary decreases with depth. For a diver starting from 0.5 ATA this gauge would start at zero, but show that the diver had reached 10 metres depth when he was at 5 metres. In theory, the diver could plan his dive and decompression according to the 'gauge' depth, but only if he was very courageous.

Divers who fly from sea level to dive at altitude, as in high mountain lakes, may commence the dive with an already existing nitrogen load in excess of that of the local divers, who have equilibrated with the lower pressures. Thus the 'sea level' divers are in effect doing a repetitive dive.

Decompression tables that supply acceptable modifications for altitude exposure, include the Buhlmann tables and the Canadian DCIEM tables. Some decompression meters are damaged by exposure to altitude (e.g. as in aircraft travel), and none is yet applicable to altitude diving or saturation excursions.

FRESHWATER DIVING

The main problem with freshwater is that it is not the medium in which most divers were trained; thus, their buoyancy appreciation is distorted. Acceptable weights in seawater may be excessive in fresh water.

There are also many organisms that are killed by seawater, but thrive in warm freshwater.

RECOMMENDED READING

Australian Antarctic (ANARE) Diving Manual, (1990).

British Sub-Aqua Club Diving Manual. Current edition.

Exley, S. (1981) *Basic Cave Diving.* National Speleological Society, Jacksonville, Florida.

NOAA Diving Manual. 4th edition, 2001.

US Navy Diving Manual. Current edition.

6

Pulmonary barotrauma

ROBYN WALKER

INTRODUCTION

Pulmonary barotrauma (PBT) of ascent is the most serious of the barotraumas, and causes concern in all types of diving operations. It is the clinical manifestation of Boyle's law as it affects the lungs and is the result of overdistension and rupture of the lungs by expanding gases during ascent. It is also called 'burst lung' or pulmonary overinflation. It happens in both compressed-air divers and submariners, especially when exposed to free ascent or buoyant ascent training. Free ascent refers to an ascent without equipment; buoyant ascent refers to an ascent using specialized buoyancy equipment.

PBT occurs more frequently in novice divers than in experienced divers, and much of our knowledge of this condition results from submarine escape training.

A recent review[1] of submarine escape training collected data on injury from 11 nations. Despite careful selection procedures and extremely high standards of training supervision, hooded buoyant ascent (where the trainee's head is enclosed by a hood providing a breathable air space) had an incident rate between 0.1 and 0.6 per 1000 escapes and a fatality rate 10 to 50 times lower than that. For buoyant ascents (the trainee wears a life jacket, his head is in water, and he must breathe out continually during the ascent) the incident range was 1 to 19 per 1000 escapes. Under recreational scuba diving conditions, these figures are likely to be underestimates.

PATHOPHYSIOLOGY

All gas-filled spaces within the body obey Boyle's law. For highly compliant organs such as the gastrointestinal tract the contraction and expansion of gases with descent and ascent is accommodated with ease. However, the lungs are not so compliant, and if gas breathed at depth is not vented from the lungs during ascent differential pressure gradients sufficient to

cause injury to local structures may result. This is known as PBT of ascent. The condition may involve much of the lung, such as when the expanding lung gases are not exhaled during ascent. Alternatively, it may involve only localized areas of pathology following obstructed airflow or altered compliance in associated airways.

A diver whose total lung volume is 6 litres at 10 metres depth (2 ATA) will need to exhale 6 litres of gas surface equivalent in order to maintain his normal 6-litre lung volume at the surface (1 ATA). If he commenced his ascent with the lungs only half-full, at only 3 litres, he could have surfaced in safety. Once the total lung capacity (TLC) is reached, the lung tends to stretch against an increasing resistance until the elastic limit of the pulmonary tissues is exceeded, and tearing results.

There is evidence that the degree of overpressure required to cause lung tissue injury is dependent on the extent to which the lung is splinted by its surrounding structures.[2] Experimentally, cadaver lungs have been shown to burst with a positive inflation pressure of 70 mmHg, but if the thorax is confined (e.g. by thoracic binding or possibly by a wetsuit) pressures up to 110 mmHg are tolerated before rupture occurs. Intensive-care literature also reports the risk of rupture with pulmonary inflation pressures of 70 mmHg or more is substantial. Therefore, overstretching of the lung causes the tissue injury of PBT by a transmural pressure change rather than the change in the volume of gas.[2]

Generalized PBT may therefore result when the ambient water pressure falls by 70 mmHg or more below the intrapulmonary pressure, i.e. with an ascent from a depth of about 1 metre to the surface.

Denison[3] has reported cases of pulmonary rupture occurring with deep inspiration at 1 ATA, and suggests this may be an asymptomatic yet frequent event. He postulates that when the lungs are at close to TLC, sneezing, coughing or taking a deep breath generates enough pressure to exceed the elastic limits of the lung, resulting in a tear. While this may remain asymptomatic and go unreported at 1 ATA, the leakage of gas from the lung into the mediastinum or chest cavity and its subsequent expansion with ascent in a diver is likely to be symptomatic and may be life-threatening. Novice divers, due to inexperience with their equipment and the environment, tend to swim with lung volumes close to TLC. 'Skip breathing', a procedure used by many divers in an attempt to conserve air, is a voluntary reduction in breathing rate, but usually associated with close to maximal lung volumes. Both these situations, where the lungs are held close to TLC, may predispose the individual to PBT.

Scarring within the lung parenchyma has been associated with an increased risk of PBT; however, Calder[4] reported that the site of injury was inconsistently related to the site of the scar. This may be explained by the fibrous tissue within the scar generating abnormal tensions on the adjacent 'normal' lung parenchyma. As the scar cannot stretch, abnormally high pressures are generated at a site distant to the scar, and it is at this distant site that the lung ruptures.

There are two types of alveoli: partitional and marginal. The marginal ones are those that rest on blood vessels and are the source of interstitial gas in PBT. The gas escapes into perivascular sheaths over the pulmonary blood vessels. When the pressure gradient is created, the gas takes the line of least resistance, tracking along the sheaths to the hilum of the lung, giving rise to mediastinum emphysema or pneumomediastinum.

Predisposing factors

Predisposing pathology includes disorders that may result in local compliance changes, gas trapping or airway obstructions. These include previous spontaneous pneumothorax, asthma, sarcoidosis, cysts, tumours, pleural adhesions, intrapulmonary fibrosis, infection and inflammation.

Precipitating factors include inadequate exhalation caused by panic, faulty apparatus or water inhalation.

Although many cases of PBT may be due to voluntary breath-holding during ascent or to the pathological lesions mentioned above, attitudes towards these etiologies are changing. About half the ascent trainees who develop PBT have been observed to carry out correct exhalation techniques. Many of these divers were also passed as medically fit before the dive, and showed none of the causative pathology afterwards. A frequent finding with some of these subjects is a reduction of compliance at maximum

inspiratory pressures, i.e. the lungs are less distensible (more stiff), and are exposed to more stress than normal diver's lungs, when distended.

Brooks *et al.*[5] demonstrated that a lower than predicted forced vital capacity (FVC) was associated with PBT in submarine escape trainees, and this further supports the suggestion that reduced pulmonary compliance is an important factor in the causation of PBT. Interestingly, many medical standards refer to the requirement for the forced expired volume in 1 second (FEV_1)/FVC to be greater than 75–80 per cent of predicted levels, yet, this has not been shown to be causally related to PBT in trainees who have no evidence of lung disease. Spirometry, does however, provide an indication of lung function and the presence or absence of obstructive lung disease.

There are four manifestations of PBT of ascent, which may occur either singly or in combination:

1. Pulmonary tissue damage.
2. Mediastinal emphysema.
3. Pneumothorax.
4. Air embolism.

In a Royal Navy series[6] of 109 non-fatal cases of PBT submarine escape training accidents, the pathology in the majority of the cases was cerebral arterial gas emboli (CAGE); however, 15 cases with arterial gas emboli also had mediastinal emphysema, seven with arterial gas embolism also had pneumothorax (three bilateral, four unilateral), four had only mediastinal and cervical emphysema, and one case had only unilateral pneumothorax.

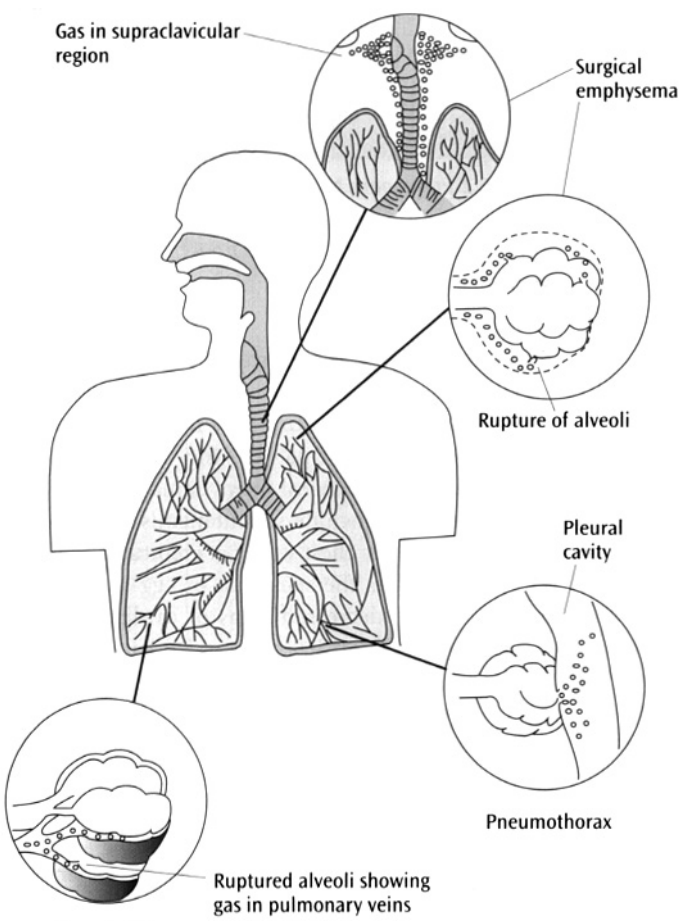

Figure 6.1 *Pulmonary barotrauma of ascent.*

CLINICAL FEATURES

Pulmonary tissue damage

After the diver surfaces, an explosive exhalation of expanded gases may be accompanied by a characteristic sudden high-pitched cry. Dyspnoea, cough and haemoptysis are symptoms of lung damage, and widespread alveolar rupture may cause death from respiratory damage.

Abnormal investigations include arterial gas measurements, a hematological assessment and chest X-ray examinations, etc. Perfusion imaging of the lungs has also been employed. These tests are usually not possible during the all-important few minutes after ascent.

Mediastinal emphysema

Mediastinal emphysema is more likely to present in scuba divers who have a history of breathing against the high regulator resistance from a low-on-air tank, and who are not exposed to the depths and extremely rapid ascent rates of the free-ascent trainees in the submarine escape training tank. It also tends to be delayed in onset, perhaps due to:

- the gradual tracking of the gas;
- increase in pulmonary pressures during coughing or exertion; and/or
- the nitrogen movement from tissue to bubble – due to a nitrogen load from the diving exposure.

In some cases there may be hours between the dive and the patient presenting with symptoms.

After alveolar rupture, gas escapes into the interstitial pulmonary tissues. This gas may track along the loose tissue planes surrounding the airways and blood vessels, into the hilar regions, and thence into the mediastinum and neck (subcutaneous emphysema). It may also extend into the abdomen as a pneumoperitoneum. When the pleura is stripped off the heart and mediastinum, a pneumoprecordium may be misdiagnosed as a pneumopericardium (Fig. 6.2)

Symptoms may appear rapidly in severe cases, or may be delayed several hours in lesser cases. They may include a voice change into hoarseness or a brassy monotone, a feeling of fullness in the throat,

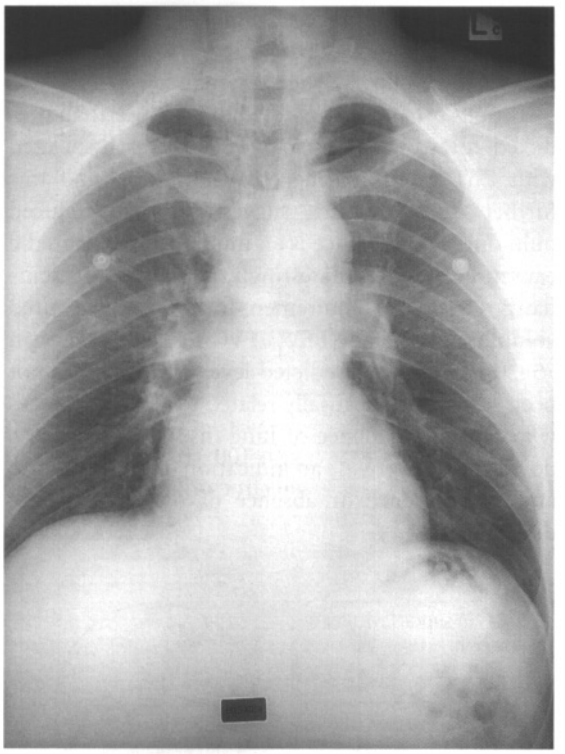

Figure 6.2 *Pulmonary barotrauma of ascent: chest X-ray showing mediastinal emphysema causing the 'tram track' sign, from air stripping the pleura from the edge of the cardiac shadow.*

dyspnoea, dysphagia, retrosternal discomfort, syncope, shock or unconsciousness. The voice changes are described as 'tinny' and have been attributed to 'submucosal emphysema' of the upper airways and/or recurrent laryngeal nerve damage.

Clinical signs include subcutaneous emphysema of neck and upper chest wall, i.e. crepitus under the skin (described as the sensation of egg-shell crackling, by divers), decreased areas of cardiac dullness to percussion, faint heart sounds, left recurrent laryngeal nerve paresis, and cardiovascular effects of cyanosis, tachycardia and hypotension.

Precordial emphysema may be palpable and give the pneumoprecordium or Hamman's sign – crepitus related to heart sounds. Sometimes it can be heard at a distance from the patient; other times it can only be diagnosed on auscultation. An extension of the mediastinal gas into the tissues between the pleura and the pericardium, rather than gas in the

CASE REPORT 6.1

RJN, a 19-year-old, was having his second dive in scuba equipment at a depth of 5 metres when he noted a slight pain in his chest. He then noted a restriction in his air supply, and thought he had exhausted his gas. He opened his reserve valve and ascended to the surface. He was asymptomatic after the dive but later, during physical training, he noted that he was breathing heavily and felt weak. A few minutes later he noted slight retrosternal chest pain. During lunch, he developed a fullness in his neck (a 'tightness') and dysphagia.

An hour-and-a-half after the dive, he decided to see the doctor because he was not feeling well. It was then noted that his voice was altered in quality and that he had subcutaneous emphysema in both supraclavicular fossae, bilateral generalized crepitus over the chest and a positive Hamman's sign. Chest X-ray showed gas in the upper mediastinum and neck. An electrocardiogram showed ischaemic changes in leads II, III and aVF.

He was treated with 100% oxygen and improved rapidly.

Chest X-ray and electrocardiogram were normal six days later. Subsequent lung function studies showed that pulmonary compliance was reduced below predicted values.

Diagnosis: PBT with mediastinal emphysema and coronary artery embolism.

CASE REPORT 6.2

TC, an experienced navy clearance diver developed epigastric discomfort towards the end of a 90-minute, 11-metre scuba work dive. The dive was otherwise unremarkable, although he had at times worked hard and he made four controlled ascents during the dive to change his tools.

Approximately 15 minutes after leaving the water he developed retrosternal chest pain, which increased in intensity over the next few hours. The pain extended from the epigastrium to the base of the throat. The pain was pleuritic in nature and aggravated by inspiration, coughing and movement. He was not dyspnoeic, and there was no associated cough or haemoptysis.

Examination was unremarkable; in particular, there was no palpable subcutaneous emphysema and no positive neurological signs. There was no clinical evidence of pneumothorax.

Chest X-ray revealed the presence of surgical emphysema in the neck and superior mediastinum. No pneumothorax was seen and the lung fields were clear. Computed tomography (CT) of the chest was reported as showing 'air in the mediastinum. Inferiorly, this is seen around the esophagus in the retrocardiac recess. Superiorly, it is seen surrounding the descending aorta at the level of the carina. It also extends along the major branches of the aortic arch adjacent to the trachea and esophagus superiorly into the base of the neck on both sides. The spread of air appears to be mainly along the major vessels of the aortic arch into the base of the neck'.

TC was treated with 100% oxygen and bed rest with complete resolution of his symptoms. He was considered permanently medically unfit to dive.

Diagnosis: PBT with mediastinal emphysema.

pericardial sac, has produced cardiac tamponade with its clinical signs.

There may be radiological evidence of an enlarged mediastinum with air tracking along the cardiac border or in the neck.

Pneumothorax from diving has the same clinical features and management as pneumothorax from other causes.

Pneumothorax

If the visceral pleura ruptures, air enters the pleural cavity and expands during further ascent. It may be accompanied by haemorrhage, forming a haemopneumothorax. The pneumothorax may be unilateral or bilateral, the latter being more common from the dramatic emergency ascents (Fig. 6.3).

Symptoms usually have a rapid onset, and include sudden retrosternal or unilateral (sometimes pleuritic) pain, with dyspnoea and an increased respiratory rate. Clinical signs may be absent, or may include diminished chest wall movements, diminished breath sounds and hyper-resonance on the affected side; movement of trachea and apex beat to the unaffected side with a tension pneumothorax; signs of shock; X-ray evidence of pneumothorax; and arterial gas and lung volume changes.

If necessary, chest X-rays can be performed through the windows of most recompression chambers, giving results satisfactory enough to confirm the diagnosis. It is prudent to conduct a few trials before being confronted with a genuine case, and this will indicate suitable adjustment of exposures, which vary for different distances and penetration materials.

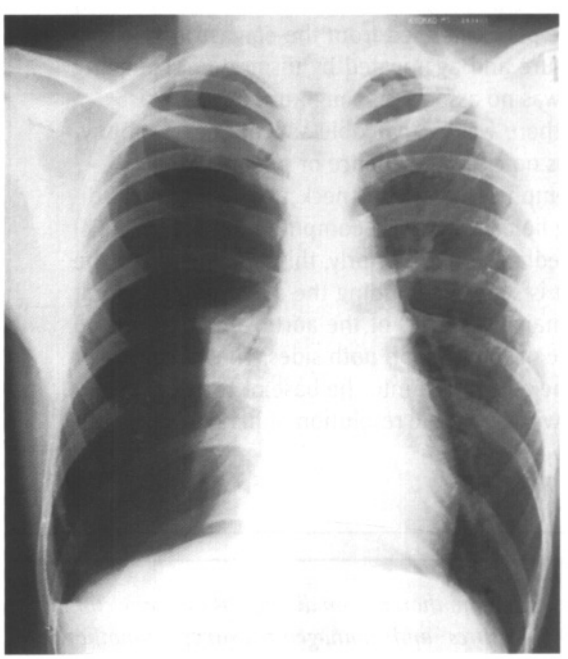

Figure 6.3 *Pulmonary barotrauma of ascent, causing a large, right-sided pneumothorax with a slight hemothorax.*

A pneumothorax under pressure becomes a tension pneumothorax during ascent. The ascent should always be halted and the patient examined critically for a pneumothorax if there is any sign of increasing respiratory distress or deterioration in the patient's condition. Tension pneumothorax may also develop from coughing or exposure to altitude or aviation pressures. Rarely a pneumoperitoneum may accompany the pneumothorax.

Air (gas) embolism

This is a dangerous condition, and is the result of gas passing from the ruptured lung into the pulmonary veins and thence into the systemic circulation. Here, it can cause vascular damage or obstruction, hypoxia, infarction, and activation of an inflammatory cascade. Air embolism is more commonly associated with mediastinal emphysema than with pneumothorax.

In one large series[6] of CAGE, the longest interval to onset of symptoms and signs was 8 minutes in a single case, with all others showing evidence of CAGE within 5 minutes of completing the dive. There were no cases occurring in excess of 10 minutes.

During overdistension of the lung, the capillaries and small vessels are stretched and may tear, along with other lung tissue. Since these vessels are small and often compressed by distended air sacs, air embolism does not usually result until overdistension is relieved by exhalation. Only a small volume in the systemic arterial system is necessary to produce severe disturbances. Serious effects may result from blockage of cerebral or coronary vessels by bubbles in the order of 25 μm to 50 μm in diameter, or by otherwise interrupting blood flow (perfusion injury). Death may follow coronary or cerebrovascular embolism. Other tissues affected may include the spleen, liver, kidneys or limbs.

Most of the clinical series refer to CAGE as the dominant site of pathology. Due to the buoyancy of circulating bubbles, CAGE is more likely to occur with the subject in the vertical position (common, under the diving situation, with ascent). Air emboli are more likely to enter the coronary system when the subject is in a horizontal position. (See Chapter 11 for a full discussion of the pathophysiology of embolic gas injury.)

Serious symptoms, which develop immediately after ascent, must be regarded as air embolism and treated accordingly, until a definitive diagnosis has been made.

Manifestations

The manifestations of CAGE are usually acute and may include:

- Loss of consciousness; other neurological abnormalities such as confusion, aphasia, visual disturbances, paraesthesiae or sensory abnormalities, vertigo, convulsions, varying degrees of paresis; gas bubbles in retinal vessels; abnormal electroencephalograms (generalized slowing or flattening of waves) and brain scans, etc.
- Cardiac-type chest pain and/or abnormal electrocardiograms (ischaemic myocardium, dysrhythmias or cardiac failure).
- Skin marbling; a sharply defined area of pallor on the tongue (Liebermeister's sign).

The cardiovascular symptoms following arterial air embolism could result from five or more mechanisms acting alone or in combination:

1 Air enters the coronary arteries leading to myocardial infarction, with resulting dysrhythmia and ischaemic damage.
2 A bolus of air in the ventricle produces an air lock that interferes with cardiac pumping action.
3 Air enters the cerebral circulation, initiating hyperactivity of the automatic nervous system, and resulting in changes in blood pressure, cardiac dysrhythmias, and even ventricular fibrillation and death.
4 Cerebral air embolism produces apnoea and other respiratory problems that could aggravate the cardiovascular changes.
5 Air bubbles in the vascular system cause ischaemia, haemorrhages, coagulopathies, endothelial damage in vessels, perfusion and reperfusion injury affecting cerebral or cardiac function (see Chapter 11).

Some of these mechanisms could theoretically be overcome by adequate cardiopulmonary resuscitation, which has often been administered to the fatal cases. Recompression is the mainstay of therapy, and could affect all the above mechanisms, except for the last.

In one series of 88 cases of CAGE,[7] mainly from free-ascent practices, 34 per cent of divers had collapsed unconscious within seconds of surfacing, 23 per cent had become confused, disoriented or uncoordinated after emerging from the water, and 17 per cent had presented with a paresis (six cases with an upper monoparesis and six with a hemiparesis).

In another series, presented by Pearson,[6] 15 per cent of divers had complete remission within 4 hours, 53 per cent had some spontaneous improvement pretherapy, and 77 per cent with coma improved to some degree before treatment. These spontaneous improvements were not always sustained, and 15 per cent died. Four per cent had a previous incident of CAGE.

Of divers who experience symptoms of CAGE, many will show a partial, or even a complete, recovery within minutes or a few hours of the incident. This recovery presumably reflects a movement of the embolus through the cerebral vasculature. Even those who become comatose may improve to a variable degree after the initial episode.

Unfortunately, the recovery is unreliable in that it may not occur or it may not be sustained. Recurrence of symptoms has an ominous prognostic significance, and this or deterioration of the clinical state may be due to:

- new embolization (coughing, straining, Valsalva manoeuvre);
- recycling of gas emboli that pass through both arterial and venous systems;
- redistribution of gases trapped elsewhere (heart, pulmonary vessels, etc.);
- positioning effect or buoyancy determining bubble movement;
- local vascular damage, perfusion and reperfusion injury; and/or
- local cerebral damage, blood–brain barrier disruption, oedema.

Differential diagnosis

The differential diagnosis for a patient, who ascends rapidly from 30 metres or more and becomes

unconscious or develops neurological signs within some minutes of ascent, but with no respiratory manifestations, can cause problems. Some would label the case as air embolism, whereas others would consider fast-tissue decompression illness with intravascular bubbles a possibility (see Chapter 12).

It is also possible that gas emboli may trigger off a decompression sickness (DCS) incident if these emboli are seeded into a supersaturated solution, e.g. in venous blood taking nitrogen from the tissues to the heart and lungs. The presence of 'combined CAGE and decompression sickness' – Type 3 DCS (see Chapter 12) – is well recognized, but fortunately not very common.

Electroencephalogram (EEG) changes are frequently detected, even after initial therapy, and neuropsychometric tests may indicate brain damage that is not always obvious clinically. Both are of immense value as baseline investigations for subsequent assessments.

Enzyme levels may indicate specific tissue damage associated with CAGE, such as creatine kinase, lactic acid dehydrogenase, serum glutamic oxaloacetic transaminase and serum glutamic pyruvate transaminase.

Haemoconcentration may also be found.

Previously, it was considered important to differentiate between CAGE and DCS because the initial recompression regimes were different. DCS was treated with a shallow 18 metre oxygen table, e.g. US Navy Table 6, while CAGE was treated on a USN Table 6A (includes an initial deep excursion on air to 50 metres). A number of studies have not been able to show an advantage in the initial deep excursion and most centres now manage CAGE and DCS patients identically (see Chapter 13). Consequently, recompression has become the priority rather than establishing the 'correct' diagnosis. It is still considered important to determine whether or not PBT has occurred as this diagnosis may render the diver permanently medically unfit to dive.

TREATMENT

Aggravation of PBT

Once PBT has resulted in the distribution of gas within body tissues, it may be aggravated by other factors. Further ascent in a chamber or underwater, or ascent to altitude during air transport, will expand the enclosed gas and cause deterioration in the clinical state of the patient.

Physical exertion, increased respiratory activity, breathing against a resistance, coughing, Valsalva manoeuvre, etc. may also result in further pulmonary

Table 6.1 *Presenting signs and symptoms in 114 Royal Navy submarine escape training accidents and 74 diving accidents involving arterial gas embolism*[6]

Signs and symptoms	Percentage incidence	
	Submarine escape training	Scuba diving
Coma with convulsions	7	18
Coma without convulsions	29	22
Stupor and convulsion	14	24
Collapse	8	4
Vertigo	14	8
Visual disturbance	6	9
Headache	2	1
Unilateral motor changes	17	14
Unilateral sensory changes	10	8
Unilateral motor and sensory changes	6	1
Bilateral motor changes	1	8
Bilateral sensory changes	1	1

Data compiled from case histories and records of initial post-accident examinations. In the case of submarine escape training accidents, the examinations were always carried out within 5 minutes of onset of symptoms.

damage, or in more extraneous gas passing through the lung tissues or into the pulmonary vessels.

If the diver has exposed himself to depths and times resulting in tissue loading by inert gas, this gas will have a pressure gradient between the tissue and the bubbles, leading to gas diffusion into the latter. A situation develops which has facets of both PBT and decompression sickness and may require more energetic recompression therapy.

Another way in which the entrapped gas from PBT may be temporarily increased in volume is by breathing a lighter, more rapidly diffusible gas, e.g. helium, in the otherwise correct belief that this may improve ventilation. The anaesthetic, nitrous oxide, rapidly diffuses into tissues, causing expansion of bubbles.

Pulmonary tissue damage

Treatment involves the maintenance of adequate respiration with 100 per cent oxygen to ensure acceptable arterial gas levels. The treatment is similar to that of near-drowning or the acute respiratory distress syndrome. Positive-pressure respiration could increase the extent of lung damage and should be used only if necessary. Support for the cardiovascular system may be required, and attention should be paid to the electrolyte and fluid balance.

Mediastinal emphysema

The need for therapy may not be urgent in mediastinal emphysema. However, exclusion of air embolism or pneumothorax is necessary and, if in doubt, treatment for these should take precedence. Management of mediastinal emphysema varies according to the clinical severity. If the patient is asymptomatic, only observation and rest may be necessary. With mild symptoms, 100 per cent oxygen administered by mask without positive pressure will increase the gradient for removal of nitrogen from the emphysematous areas. This may take 4–6 hours.

If symptoms are severe, therapeutic recompression using oxygen may be useful. A shallow oxygen table, such as Comex 12 m without air breaks (to prevent further nitrogen entry), is appropriate. This shallow table avoids the complications of oxygen toxicity, which can be confused with lung or cerebral damage from PBT.

Pneumothorax

Treatment depends on the clinical severity and the depth at which it is diagnosed. The possibility of associated air embolism must be excluded. The treatment follows the standard principles used in treating a pneumothorax from any other cause.

Mild cases at the surface may resolve with the administration of 100 per cent oxygen, without positive pressure. This will often appreciably reduce the size of the pneumothorax within a few hours. More serious cases, where there is associated dyspnoea will require aspiration and possibly the insertion of an intercostal catheter. The presence of a pneumothorax is not a contraindication to recompression if other sequelae of PBT such as CAGE are present. Therapeutic recompression is likely to provide rapid initial relief from the pneumothorax. However,

CASE REPORT 6.3

Al was a relatively inexperienced diver, aged 19 years and in good health. He was performing a free ascent from 10 metres. On reaching the surface, he gave a gasp, his eyes rolled upward, and then he floated motionless. While being rescued from the water it was noted that blood and mucus were coming from his mouth and that he was unconscious. Resuscitation was commenced immediately, using oxygen. He was noted to be groaning at this time but soon after appeared dead. Resuscitation was continued while he was rushed to the nearest recompression chamber. Thirty minutes after the dive he was compressed to 50 metres but with no response. Autopsy verified the presence of PBT.

Diagnosis: air embolism resulting from PBT of ascent.

Figure 6.4 *Pulmonary barotrauma of ascent: pathological sequence.*

because the pneumothorax is likely to re-expand during ascent, aspiration or thoracocentesis may be indicated. The use of a Heimlich valve is preferable to an underwater seal drain, which requires careful observation and management during ascent or descent, or air and water may flood the thoracic cavity. Pneumothorax may be aggravated or reproduced by coughing, which is sometimes produced in chambers, due to condensation while decompressing.

Pneumothorax must always be considered if a patient's condition deteriorates on ascent. The ascent should be halted and careful clinical examination undertaken – this is often difficult in the noisy confines of a recompression chamber.

Offshore chambers may be heavily contaminated with infective organisms, especially *Pseudomonas* sp. and *Staphylococcus aureus*. Such problems need to be considered in making the decision to aspirate the chest cavity or to insert an intercostal drain.

Air embolism

Treatment of air embolism is urgent, must be instituted immediately, and usually takes precedence over other manifestations of PBT. The effect of delay on treatment outcome is to increase mortality and morbidity. The likelihood of achieving a cure is reduced to 50 per cent when delay of recompression exceeds 4 hours.

Positioning

The 'modified Trendelenburg' position or the head-down left lateral position was recommended in the past. Some authorities even recommended a 45° angle, which is virtually impossible to maintain even in a conscious cooperative patient, let alone a seriously ill victim requiring resuscitation. This position was recommended to prevent the re-embolization of the brain – as bubbles distribute with buoyancy in large vessels and therefore away from dependent tissues. However, this is no longer recommended due to the possibility of compounding the embolic brain injury by cerebral oedema.

To prevent further brain damage, and possibly to reduce the likelihood of a further CAGE, the patient should be nursed horizontally, on his back or lying on his side in the 'coma' position (preventing the tongue from causing airway obstruction, or if there is a possibility of aspiration of stomach contents or seawater). The legs should not be elevated, as this may increase cerebral venous pressures as well as central venous pressure, possibly causing paradoxical embolism.

A similar position should be maintained in transit to the chamber, while the chamber is being compressed, and for an uncertain period of time (usually one to two oxygen periods) while breathing oxygen. The patient is initially allowed to sit or stand while still at the initial treatment depth. A sudden deterioration in the clinical state, with an apparent redevelopment of the embolism, may follow the resumption of an erect (sitting or standing) position. This would suggest the continued existence of gas emboli.

Oxygen

Oxygen (100 per cent), via a close-fitting mask, should be administered in transit to the chamber to:

- improve oxygenation of hypoxic tissues;
- dissolve the mobile emboli;
- ensure that any subsequent ones are composed of oxygen, instead of nitrogen; and
- dissipate the blocked emboli more rapidly.

Recompression

Immediate recompression is necessary, and a recompression chamber should always be available near surfacing positions for all free-ascent or submarine escape training. The choice of therapy is limited by the facilities available.

The patient is kept horizontal for at least the first 30 minutes of 100 per cent oxygen breathing in the recompression chamber before being allowed to move, as this might redistribute emboli.

Many treatment facilities use the conventional 18 metre oxygen tables (e.g. USN Table 6). By reducing bubble size, this may assist the bubbles to pass through the arterial circulation into the capillary and venous systems, where they may become trapped in the lungs or redistribute to other areas. The denitrogenated state of the blood then assists in rapid bubble resolution. Oxygenation of damaged tissues and a reduction of cerebral oedema are bonuses.

A variation in this technique is to expose the patient to an initial 50 metre short exposure on air (e.g. USN Table 6A), to enhance the redistribution of blocked arterial emboli by decreasing their length to less than 20 per cent, and proportionately reducing the resistance to forward flow, prior to the oxygen tables. More traditional clinicians extend the duration at depth, and may even employ long air and saturation exposures, foregoing the value of hyperbaric oxygen.

The 30-metre 50 per cent oxygen/nitrogen Comex tables may be an acceptable compromise between these opposing concepts. Many units now substitute heliox for nitrogen to avoid adding further nitrogen to the bubble load.

Repetitive hyperbaric oxygen treatment may be of value in those neurologically impaired patients who have not recovered fully. These are continued until improvement has stopped.

Symptomatic therapy

A possible cause of death from air embolism is from a cardiac lesion, and so cardiopulmonary resuscitation before and during recompression may be necessary. Despite the common hazards of hyperoxia and highly conductive wet environments, some chambers are equipped for using defibrillation techniques. With appropriate precautions these are claimed to be safe.

Circulatory and respiratory support may be necessary while the recompression facilities are being obtained. Prevention and/or treatment of secondary complications such as myocardial infarction, dysrhythmias, renal failure, cerebral oedema or haemorrhages and respiratory insufficiency should be carried out. The treatment of these disorders is based on conventional medical principles.

Rehydration may be both important and needed. Intravenous fluids (saline, electrolytes) should correct haemoconcentration, and may contain glucose only if long-term infusion is needed. Dextrose or glucose is not usually indicated, because hyperglycemia may decrease neuronal survival by increasing lactic acid levels and provoke glycosuria and more haemoconcentration.

Neuropsychological testing may demonstrate transitory or permanent cerebral damage after CAGE, and many divers have abnormal EEGs during and after treatment. The abnormality is excessive or disorganized slow wave activity – localized or generalized. This tends to improve over some weeks, at least in cases effectively treated.

Brain scans, such as CT, magnetic resonance imaging (MRI) and single photon emission computed tomography (SPECT) may assist in the diagnosis and management of decompression sickness and CAGE. These are most helpful in post-recompression diagnosis and evaluation of treatment. These studies may show areas of infarction and oedema, but would take second place to recompression therapy in the acute phases.

Drugs are not very valuable in most eases of CAGE, despite many attempts to affect the complications of blood–bubble interactions. Heparin, aspirin and steroids are not indicated (see Chapter 13).

Other regimes to reduce cerebral oedema include diuretics such as mannitol and frusemide. However, their value is unproven and they have not received much clinical support.

The use of controlled hyperventilation has not been shown to be of value in CAGE, and the reduction in blood flow in ischaemic areas may be detrimental.

Lignocaine has been shown to improve neuronal recovery in humans following air embolism[8] as a consequence of open-heart surgery, but its use in diving-related cases is yet to be demonstrated.

Relapses

In a Royal Navy series of CAGE patients,[6] some degree of relapse was seen in 32 per cent of cases. The relapse may be profound and delayed by as much as 68 hours. It may be due to:

- regrowth of emboli during decompression;
- re-embolization from the initial causative lung lesion;
- redistribution of existing emboli (possibly when repositioning the patient);
- vasogenic oedema secondary to the blood–brain barrier damage; and/or
- reperfusion abnormalities.

Endothelial damage is an initiating factor in a complex chain of events leading to progressive failure of perfusion, despite an initial restoration of blood

flow after the initial embolic blockage (see Chapter 11).

DIVING AFTER PULMONARY BAROTRAUMA

In general, an incident of PBT is a contraindication for further scuba diving. The reasons are two-fold: first, the diver has demonstrated a pulmonary abnormality or predisposition; and second, pulmonary damage has been sustained and will produce local scarring on healing and predispose to further problems by alterations of compliance.

Recurrences of PBT tend to be worse than the first incident, with an increased risk to life. Being neurologically incapacitated in the water is a serious situation.

PREVENTION

Attempts to prevent PBT, or to reduce its incidence, have centered on increased standards of fitness for divers, modification of training and diving techniques, and the development of safer equipment.

Dive training

Dangerous diving practices to be avoided include; delayed or skip breathing, buddy breathing at depth and during ascent, ditch and recovery training and emergency free-ascent training when there are no experienced medical staff and full recompression facilities on site.

Deep diving increases the danger by exhausting the air supply more rapidly, causing narcosis and less attention to the air contents gauge, and results in a deeper and longer free ascent.

The faster the ascent, the greater the danger of PBT. Certain diving equipment will reduce the likelihood of out-of-air situations and subsequent uncontrolled ascents, e.g. the use of tank pressure (contents) gauges, octopus rigs, and carrying an alternative and independent air supply ('pony bottle').

Medical selection

Predisposing pathology includes previous spontaneous pneumothorax, asthma, sarcoidosis, cysts, tumours, pleural adhesions, intrapulmonary fibrosis, infection and inflammation, etc. These disorders may result in local compliance changes or airway obstructions.

Cases of penetrating chest wounds may increase the risk of CAGE or pneumothorax. Pleurodesis ensures a protection from pneumothorax, at the expense of an increasing risk of CAGE and mediastinal emphysema.

Basal plural thickening may indicate adhesions, which have been shown to be a cause of pulmonary tearing during the overinflation of the lungs of fresh cadavers.

The medical standards are dealt with in Chapters 53 and 54 and involve: the exclusion of candidates with any degree of significant pulmonary pathology as described above; the imposition of respiratory function tests demonstrating efficient capability of exhalation; and often a pre-diving chest X-ray. In most cases, a single full-plate chest X-ray is acceptable, but some groups insist upon both maximum inspiratory and maximum expiratory X-rays, hopefully to demonstrate the air trapping in the latter view. If this is a serious consideration, more sophisticated lung function tests are indicated. High-resolution or spiral CT scans of the lungs, without contrast, are useful in demonstrating emphysematous cysts, pleural thickening, etc.

SYNCOPE OF ASCENT

The so-called syncope of ascent is a cause of a transitory state of confusion, often described as either disorientation or lightheadedness, and associated with a sensation of imminent loss of consciousness. It is caused by inadequate exhalation of the expanding lung gases during ascent, with resultant distension of the lungs and an increase in intrathoracic pressure causing an impairment of venous return. It is analogous to cough syncope.

Syncope of ascent most commonly occurs during rapid ascents, when the pressure gradients are

magnified, and also when the diver attempts to retain the air in his lungs, instead of exhaling it. In the past, free ascent training from 18 to 30 metres was carried out by divers and submariners, and was a typical situation in which this disorder occurred – it caused considerable problems with differential diagnosis.

As there is no actual lung pathology, it is technically incorrect to describe this as pulmonary barotrauma, but it could sometimes be a step in the progression to this disease.

PULMONARY BAROTRAUMA OF DESCENT

This is known by the divers as 'lung squeeze'. **Descent barotrauma** is not common in breath-hold diving, and very rare with open-circuit diving apparatus. The actual depth limit for breath-hold diving is probably determined by two factors:

1 *Residual lung volume:* the total lung volume decreases with increasing depth, in accordance with Boyle's law. Once the actual volume approximates the residual volume, lung compressibility is limited, and subsequent descent results in pressure gradients, which are equalized by pulmonary congestion, oedema and haemorrhage. Further descent may also result in collapse of the chest wall.

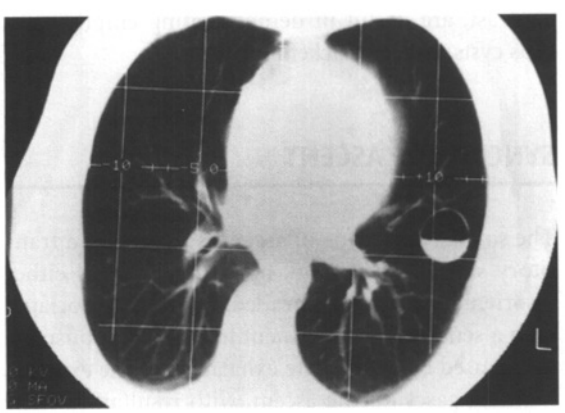

Figure 6.5 *Lung cyst produced by pulmonary barotrauma. This computed tomogram of the chest shows an axial scan through the lower zones. In the left lower zone, there is a 3-cm diameter cyst, which contains a fluid level. It resolved within a month.*

2 Individual variations in the dilatory response of the pulmonary vascular bed to an increased pulmonary vascular-to-alveolar pressure gradient. It has been found that, in deep breath-hold dives, the pulmonary venous bed dilates and blood displaces air in the thorax, decreasing the effective residual volume. This extends the depth, which can be reached in safety.

Pulmonary barotrauma of descent is rare.

An average full lung contains 6 litres of air at the surface, but this is compressed to 1.5 litres at 30 metres. This approximates the normal residual volume, and further descent may be hazardous. The individual pulmonary vascular response determines the final volume limitation. Breath-hold dives to a depth of 133 metres have been achieved because of a combination of increased dilatation of the pulmonary venous bed, a large vital capacity and a small residual volume. The minimal residual volume, which if further reduced will result in pulmonary damage, has not been determined.

In diving with open-circuit apparatus, inhaled gases are at the same pressure as the surrounding environment and the diver, thus preventing PBT of descent.

PBT of descent is possible in the following situations:

- Breath-hold diving.
- Loss of surface pressure supply with failure or absence of a non-return valve. This may occur with surface supply and standard diving.
- Failures of the gas supply to compensate for the rate of descent. This is more common with standard diving in which there is no automatic relationship between the gas supply and the ambient pressures, and in which the diver is overweighted and has negative buoyancy.

Clinical features are poorly documented, but include chest pain, haemoptysis with haemorrhagic pulmonary oedema and death. Treatment is based on general principles. Intermittent positive-pressure respiration may be needed. Initially, 100 per cent oxygen should be used with replacement of fluids, treatment of shock, etc.

CASE REPORT 6.4

(described by a diver/doctor, in his incident report)

On Day 1 the diver using a helium/oxygen system carried out a bounce dive to 492 feet. The dive job was carried out successfully and was completed without incident in 13 minutes. During decompression upon reaching 90 feet, the diver reported tightness in his chest, some shortness of breath and discomfort while breathing.

The diver was recompressed to 100 feet where he had complete relief and felt normal. The chamber atmosphere was at this point changed over to a saturation atmosphere and the diver was decompressed at a saturation decompression rate. The Diving Superintendent at this point informed Mr. A. on shore that a treatment procedure was being carried out.

When the diver reached 85 feet the symptoms redeveloped and other treatment procedures were instituted. The diver was recompressed to 185 feet and brought out on a treatment schedule.

Decompression was uneventful with the diver feeling fine until Day 2 at 02:53 hours where, at 105 feet, the diver had the first recurrence of symptoms. The diver was recompressed according to the treatment schedules and then decompressed. He experienced a second recurrence of the symptoms at 85 feet during decompression and he was once more recompressed to 185 feet for therapeutic decompression at 14:33 hours. At this point a special treatment was instituted at Mr. A's instructions. He had now diagnosed the case as a burst lung problem and discounted any kind of bend.

On Day 3 at 13:00 hours, upon reaching 75 feet during his decompression, the diver complained of restriction to his breathing whereupon he was recompressed to 125 feet where he obtained complete relief. It was decided to attempt decompression once more to see if the diver could be decompressed all the way or if there would be a further recurrence of symptoms. At 23:25 hours while reaching 83 feet in the decompression the diver again complained of breathing difficulties. Recompression to 135 feet relieved all symptoms.

At this point Mr. A. decided that the problem could not be an ordinary decompression problem and was reasonably certain that the symptoms were the result of a pneumothorax. A doctor was called and arrangements were made to go to the rig in the morning of Day 4. The doctor was informed of the treatment to date and of the diagnosis and was asked to bring the necessary needles with him to vent a pneumothorax.

On Day 4 at 10:49 hours Mr. A. and the doctor arrived at the rig. At 13:49 hours while the diver was at 80 feet the doctor made a cursory examination of the diver without taking his temperature and diagnosed the diver's condition as 'full blown pneumonia and pleurisy of the left lung' and ruled out the possibility of a pneumothorax. The doctor was challenged on the fact that the diver obtained relief by recompression; however, he stated that this would be the case with pneumonia and that he had previously treated a very similar case.

At this point the doctor took over the treatment and instructed the diver to be decompressed at the rate of 3 feet per hour and emphasized the fact that the diver would experience severe chest pains during decompression due to the pneumonia. By the afternoon of Day 4 the diver was treated with penicillin injections and, due to severe pain, the rig medic administered an injection of painkiller at 22:45 hours of Day 4.

The doctor left the rig by evening of Day 4 stating that it was a routine case and that he would be available ashore for consultation. By the morning of Day 5, the diver had been decompressed to a depth of 60 feet and his condition had steadily deteriorated. Mr. A. at this point requested the opinion of a second doctor regarding the diver's treatment and condition. Attempts were made by Mr. B. to obtain another doctor to go to the rig but he was unsuccessful.

The attending doctor was notified of these attempts and of the worsening of the diver's condition. During Day 5 the diver received injections of penicillin and painkiller with little apparent effect. During the early hours of Day 6, further drugs were administered and the diver's condition was worsening. The doctor had been summoned and examined the patient at 03:40 hours while the diver was at 39 feet.

The doctor stated that the diver's condition had improved, that the pneumonia was disappearing and that the decompression rate was to be increased so that the diver could be transferred to a hospital as soon as possible.

At 09:00 hours the diver's pulse had stopped and by 09:15 he was pronounced dead by the doctor.

Cause of death:

1. Death resulted from a tension pneumothorax of the left lung (postmortem finding).
2. Cause of pneumothorax unknown; however, it was learned that the diver had a slight chest cough on the day before the incident and complained to the rig medic of some pain on the left side of his chest, and over the central area.

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7

Ear barotrauma

CARL EDMONDS

INTRODUCTION

Barotrauma is defined as the tissue damage caused by expansion or contraction of enclosed gas spaces, due to pressure changes. It is a direct effect of these changes causing tissue distortion.

The volume change in gas spaces with depth is proportionally greatest near the surface, and so it is in this zone that ear barotrauma is more frequently experienced. It is probably the most common occupational disease of divers, experienced to some degree by almost all.

Ear (also called otological or aural) barotrauma may affect the:

- external ear (when a sealed gas space exists);
- middle ear (which encompasses an enclosed gas space); and/or
- inner ear (which adjoins a gas space).

Middle-ear barotrauma is the most common form. Barotrauma problems may contribute to panic and diving deaths in novice divers, or to permanent disability – tinnitus, balance and hearing loss.

In the earlier literature on caisson workers' and divers' disorders, the otological barotrauma symptoms were hopelessly confused with decompression sickness symptoms, and this confusion persists in many clinical reports today.

Barotrauma refers to damage to tissues resulting from changes in volume of gas spaces, which in turn is due to the changes in environmental pressure with descent and ascent (Boyle's law).

Barotrauma of **descent** is a result of a failure or an inability to equalize pressures within the ear cavities as the volume of contained gas decreases. Because enclosed cavities are surrounded by cartilage and bone, tissue distortion is limited, and the space may be taken up by engorgement of the mucous membrane, oedema and haemorrhage. This, together with the enclosed compressed gas, assists in equalizing the pressure imbalance. It is commonly called a 'squeeze'.

Barotrauma of **ascent** is the result of the distention of tissues around the expanding gas within the ear, when environmental pressures are reduced, i.e. on ascent. Divers use the misnomer 'reverse squeeze' to describe it. Similar problems are encountered with aviation and space exposure, in hypo/hyperbaric chambers and by caisson workers.

Middle-ear barotrauma of descent is the most common disorder encountered by divers.

Barotrauma is classified according to the anatomical sites and whether it is due to ascent or descent. It may occur in any combination in the external, the middle, or the inner ear. With helium/oxygen diving, middle ear and sinus autoinflation seems easier than with air breathing, and less barotrauma has been noted. General information on the ear in diving, including many references to barotrauma, is included in Chapters 35–38.

EXTERNAL-EAR BAROTRAUMA OF DESCENT

This ia also referred to as 'external ear squeeze' or 'reversed ear'. Because the external auditory canal is usually open to the environment, water enters and replaces the air in the canal during descent. If the external ear is occluded, then water entry is prevented. Contraction of the contained gas is then compensated by tissue collapse, outward bulging of the tympanic membrane, local congestion and haemorrhage. This is observed when a pressure gradient between the environment to the blocked external auditory canal is +150 mmHg or more,

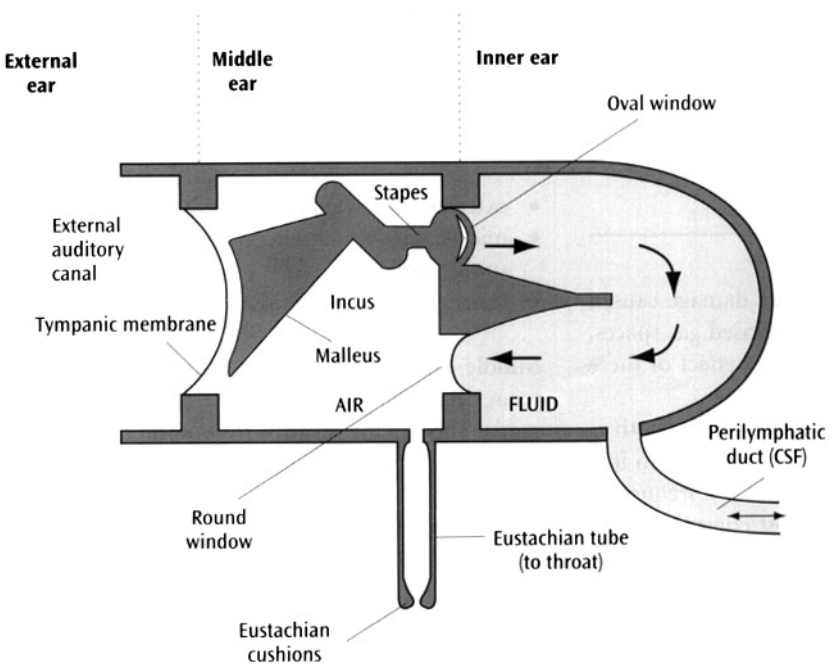


Figure 7.1 *Diagrammatic representation of the anatomy of the ear.*

i.e. 2 metres descent in water. The common causes of blockage of the external auditory canal include cerumen (ear wax), large exostoses, foreign bodies such as mask straps, tight-fitting hoods and mechanical ear plugs.

Clinical symptoms are usually mild, but occasionally a slight difficulty in performing the Valsalva manoeuvre is experienced. Following ascent there may be an ache in the affected ear and/or a bloody discharge.

Examination of the external auditory canal may reveal petechial haemorrhages and blood-filled cutaneous blebs which may extend onto the tympanic membrane. Perforation of this membrane, if it does occur, is rare.

Treatment for this condition includes maintaining a dry canal, removal of any occlusion, possibly cleansing of the canal with 1.5 per cent hydrogen peroxide solution warmed to body temperature, and prohibition of diving until epithelial surfaces appear normal. Secondary infection may result in a recurrence of the pain, and require antibiotics and local treatment (see Chapter 29).

This condition is easily prevented by ensuring patency of external auditory canals and avoiding ear plugs or tight-fitting hoods which do not have apertures over the ear to permit water entry.

External ear barotrauma of ascent, following barotrauma of descent, is theoretically possible.

MIDDLE-EAR BAROTRAUMA OF DESCENT

Known as 'middle ear aqueeze', this is by far the most common medical disorder experienced by divers, and it follows the failure to equilibrate middle ear and environmental pressures via the eustachian tubes, during descent. An abnormal pressure difference (gradient) causes the tissue damage. Diving marine animals avoid this disorder by having an arteriovenous plexus in the middle ear, which responds to the pressure changes. This fills during descent and empties on ascent, accommodating the volume changes.

Any condition which tends to block the eustachian tube, predisposes to middle-ear barotrauma. More commonly it is caused by faulty technique of voluntary middle-ear autoinflation.

Pathophysiology

The eustachian tubes may open when the pressure gradient between the pharynx and middle-ear cavity reaches 10–30 mmHg, which equates to an underwater depth of 13–39 cm. Equalization of pressure occurs when the eustachian tubes open, and this can be achieved normally by yawning, moving the jaw or swallowing, or by voluntarily inflating the middle ear cavity by the Valsalva manoeuvre. It is termed 'equalizing' or 'clearing the ears' by divers, and 'middle-ear autoinflation' by otologists.

If the eustachian tubes are closed during descent, a subjective sensation of pressure will develop when the environmental pressure (external to the tympanic membrane) exceeds that in the middle-ear cavity by 20 mmHg, or after about 25–30 cm descent in water.

Discomfort or pain may be noted with a descent from the surface to 2 metres, a 150 mmHg pressure change and causing a volume reduction of less than 20 per cent in the middle-ear cavity. If the middle-ear pressure is then equalized, for another 20 per cent middle-ear volume reduction (and its associated ear pain to occur) the diver must descend to 4.4 metres, then to 7.3 metres, then to 10.8 metres, etc. Thus, the deeper the diver goes, the fewer autoinflation manoeuvres are required per unit depth, to prevent symptoms.

If equalization is delayed, a eustachian locking effect may develop and prevent successful autoinflation. This is due to the tubal mucosa being drawn into the middle ear, becoming congested and obstructing the tube.

If a diver continues descent without equalizing, mucosal congestion, oedema and haemorrhage within the middle-ear cavity follows inward bulging of the tympanic membrane. This partly compensates for the contraction of air within the otherwise rigid cavity. The tympanic membrane will become

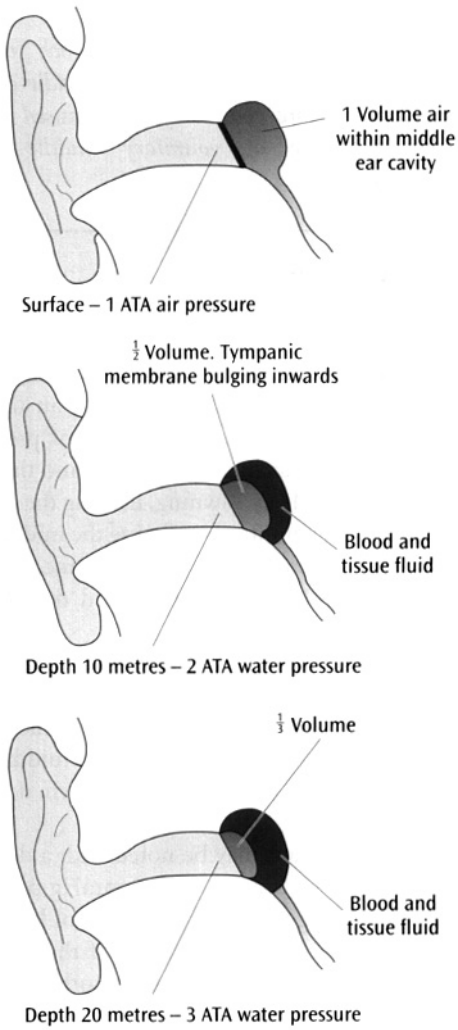


Figure 7.2 *Middle-ear barotrauma of descent.*

haemorrhagic (the 'traumatic tympanum' of older texts), and eventually it may rupture.

It is commonly inferred that perforation is the ultimate damage from not equalizing the pressure in the middle-ear cavity, and follows the extreme degrees of haemorrhage described in gradings of middle-ear barotrauma of descent. Many tympanic membrane perforations due to diving are not associated with gross haemorrhages in the tympanic membrane. It is likely that perforation competes with middle-ear haemorrhage as a pressure-equalizing process – the former demonstrating tympanic membrane fragility, and the latter demonstrating vascular capillary fragility. Perforation is more likely with rapid descents or old perforations and scarring.

There is a time factor in the development of middle-ear congestion and haemorrhage, with greater degrees resulting from longer exposure to unequalized middle-ear pressures.

Middle-ear barotrauma of descent has two major causes:

- pathology of the upper respiratory tract obstructing the eustachian tube; and
- incorrect autoinflation techniques.

Blockage of the eustachian tubes may be due to mucosal congestion as a manifestation of upper respiratory tract infections, allergies, otitis media, some drugs, respiratory irritants, venous congestion, mechanical obstructions such as mucosal polyps, or individual variations in size, shape and patency of the tube.

For some divers, with very patent eustachian tubes, attention to autoinflation is not of much importance. For others – especially novice divers and those with less patent eustachian tubes – early and positive middle-ear autoinflation techniques should be instituted.

Opening of the eustachian tubes is more difficult in the inverted position, when the diver swims downward, this having been attributed to increased venous pressure. It is easier if the diver descends feet first, when air flows more readily upwards in the vertical tubes.

Factors leading to blockage of the eustachian tube include:

- *upper respiratory infections and allergies;*
- *alcohol ingestion;*
- *premenstrual mucosal congestion;*
- *gross nasal pathology, septal deviation, mucosal polyps, etc.;*
- *delay in autoinflation during descent;*
- *descent to the point of 'locking';*
- *horizontal or head-down position;*
- *cigarette or marijuana smoking, respiratory irritants; and*
- *drugs – cocaine, beta-blockers, parasympathomimetics.*

Symptoms

These consist initially of a sensation of pressure or discomfort in the ear followed by increasing pain if the descent continues. This pain may be sufficiently severe to prevent further descent.

Occasionally, a diver may have little or no symptomatology despite significant barotrauma. This occurs in some divers who seem particularly insensitive to the barotrauma effects, and also when a small pressure gradient is allowed to act over a prolonged time, e.g. when using scuba in a swimming pool or when not autoinflating the ears on the bottom, following the final metre or so of descent. Some divers reduce the symptoms (but not the pathology) by slowing the descent or engaging in repeated short ascents after they notice discomfort (the 'yo/yo' descent).

Difficulties are more frequently encountered within the first 10 metres due to the greater volume changes occurring down to this depth.

Eventually, rupture of the drum may occur, usually after a descent of 1.5–10 metres (100–760 mmHg pressure) from the surface. This causes instant equalization of pressures by allowing water entry into the middle-ear cavity. After an initial shock, pain is automatically relieved; however, nausea and vertigo may follow the caloric stimulation by the cold water (depending on the spatial position of the head; see Chapter 38). Unless associated with vomiting or panic, this situation is seldom dangerous as it quickly settles when the water temperature within the middle-ear cavity warms to that of the body.

Occasionally, there is a sensation of vertigo during the descent, but this is not as common as in middle-ear barotrauma of ascent or inner-ear barotrauma (see below), both of which can follow and be due to middle-ear barotrauma of descent. It may also result from the Valsalva manoeuvre.

Blood or blood-stained fluid may be expelled from the middle ear during ascent, and run into the nasopharynx (to be spat out or swallowed) or appear from the nostril on the affected side (epistaxis). Blood is occasionally seen in the external ear, from a haemorrhagic tympanic membrane.

Following a dive which has resulted in descent barotrauma, there may be a mild residual pain in the affected ear. A full or blocked sensation may be felt which is sometimes associated with a mild conductive deafness involving low frequencies, and is due to haemotympanum, fluid in the middle ear or some dampening effect on the ossicles. This condition is usually only temporary (hours or days), but in severe cases fluid may be felt in the middle ear for longer periods, possibly with crackling sounds, before full resolution.

Tympanic membrane perforation, if it occurs, is usually either an oval- or crescent-shaped opening below and behind the handle of the malleus or adjacent to previous scarring.

Middle-ear barotrauma is classified into six grades based on the otoscopic appearance of the tympanic membrane. The grades are:

Middle ear barotrauma of descent: Tympanic membrane grading (see Plate 1)

- *Grade 0 – Symptoms without signs.*
- *Grade I – Injection of the tympanic membrane, especially along the handle of the malleus.*
- *Grade II – Injection plus slight haemorrhage within the substance of the tympanic membrane.*
- *Grade III – Gross haemorrhage within the substance of the tympanic membrane.*
- *Grade IV – Free blood in the middle ear, as evidenced by blueness and bulging.*
- *Grade V – Perforation of the tympanic membrane.*

Remember that damage involves the whole of the middle-ear cleft and not just the tympanic membrane.

Recent overt or subclinical middle-ear barotrauma of descent results in congestion of the middle-ear spaces and subsequent eustachian tube blocking. Autoinflation becomes progressively harder with repeated descents, with delayed or omitted middle-ear autoinflation, until the middle ear is almost totally full of fluid. Then there is little problem with diving, but at the expense of middle- or inner-ear damage.

Sometimes the eustachian tube may be narrowed and produce a 'hissing' sound during autoinflation, as opposed to the normal 'popping' sound of the tube opening, or the tympanic membrane movement. A

patulous eustachian tube (see Chapter 37) can also follow either descent barotrauma or forceful attempts at Valsalva techniques.

Treatment

The clinical management consists of:

- avoiding all pressure changes such as diving and forceful autoinflation techniques, until resolution;
- systemic or local decongestants occasionally (very rarely); and/or
- antibiotics where there is evidence of a pre-existing or developing infection, usually with gross haemorrhage or perforation, and possibly with culture and sensitivity tests.

In treating many thousands of middle-ear barotrauma cases, these authors rarely use decongestants or antibiotics.

Serial audiometric examination should be undertaken to exclude any hearing loss, and to assist in other diagnoses (especially inner-ear barotrauma) and management if such loss is present.

Impedance audiometry (tympanometry) may be used to follow the middle-ear pathological changes, if there is no perforation (see Chapter 36), and occasionally it is needed to verify a tympanic membrane perforation.

> *Serial audiograms should be performed on all but the most minor cases of middle-ear barotrauma.*

Diving can be resumed when resolution is complete, and autoinflation of the middle-ear cleft has been demonstrated during otoscopy. If there is no perforation (Grades 0–4), recovery may take from one day up to two weeks. However with perforation (Grade 5) it may take one to two months, if uncomplicated and managed conservatively. Although the tympanic membrane may appear normal much earlier, recurrent perforation frequently results from premature return to diving. There is rarely an indication for such active procedures as tympanoplasty, unless healing is incomplete, or if the lesion recurs with minimal provocation.

It is important to clearly identify and correct the contributing factors (pathology and technique) in each case, before resuming diving or flying.

Prevention

Prevention of this disorder consists of ensuring patency of the eustachian tubes prior to diving, and appropriate training in autoinflation techniques, to be used while diving. Autoinflation is best confirmed by otoscopic examination of the tympanic membrane during a Valsalva manoeuvre, when the tympanic membrane will be seen to move outwards. The degree of force needed to autoinflate, and the degree of movement of the drum, will provide an estimation of the probable ease of pressure equalization.

If one or other tympanic membrane appears to move sluggishly, or if much force is necessary, then decongestant nasal drops or sprays may improve the patency of the eustachian tubes – as can attention to technique (see below).

The use of **decongestants** to improve eustachian tube patency prior to diving is to be discouraged. They may reduce descent barotrauma problems, but the evidence for this is debatable. In one prospective comparison of topical decongestants, they did not seem to be any more valuable than placebos in preventing middle-ear barotrauma.

The rebound congestion of the mucosa is cited by otologists as a reason for the avoidance of decongestants, but the diving clinician is also concerned with the systemic problems of sympathomimetics and the increased incidence of middle-ear barotrauma of ascent encountered with these medications. The reason for the latter may be that decongestants are more effective in improving nasal airflow and thereby affecting the pharyngeal cushions of the eustachian tube, than influencing the tubal mucosa or middle-ear orifice, which may be affected by the same pathology. Decongestants, both local and general, will only be effective in the marginally obstructed tube, thereby allowing slow descent and permitting some degree of descent barotrauma and resultant congestion of the middle-ear orifices of the tube, which block on ascent and cause middle-ear distention and further barotrauma (see below).

CASE REPORT 7.1

JQ performed three scuba dives, to a depth of 5 metres. He was not able to equalize the pressure in his middle ear during descent, but in the first dive he did manage to achieve this after he had reached 5 metres. Following this first dive his ears felt 'full' or 'blocked'. He then went down to 3 metres, 'to see if I could clear them' for his second dive, with the same result. On the third dive he felt pressure in his ear during descent, and again could only equalize them once he had reached the bottom; considerable pressure was required for autoinflation. After ascent he again noted that his ears felt blocked and he again attempted to equalize them, this time using considerable pressure. Suddenly pain developed in the right ear, and it gave way with a 'hissing out'. On otoscopic examination of the left ear there was a Grade 3 aural barotrauma with a very dark tympanic membrane, haemorrhage over the handle of the malleus and the membrana flaccida, and a small haemorrhage anterior to the handle of the malleus. The right ear had similar features, with a large perforation posterior to the tip of the handle of the malleus. Daily audiograms revealed a 15 dB loss in this ear throughout the 150–4000 Hz range. This hearing loss disappeared after two weeks when the perforation had almost healed over.

Diagnosis: middle-ear barotrauma of descent.

From the safety aspect, it is more prudent to have difficulties with descent than ascent.

We do not recommend the use of decongestants before diving, but they are of value to trainees who can practise middle-ear autoinflation techniques and improve this skill on land, prior to diving.

In most cases – and especially in the novice diver – practise and instruction in middle-ear autoinflation and the use of correct diving techniques, is much more effective than drugs in improving eustachian tube patency.

It is possible to measure the force or pressure necessary to open the eustachian tubes; tube patency and middle-ear pressure changes can be measured if impedance audiometers are employed clinically (see Chapter 36 for more information).

When dealing with divers who have not adequately autoinflated their middle ears during descent – despite the ability to perform this in the clinic – the following errors are commonly encountered:

1 Not autoinflating early enough, i.e. waiting until the sensation of pressure is felt. This results in relatively negative middle-ear pressure referred to above. Commonly, the novice diver, instead of performing a Valsalva manoeuvre before descent, will concentrate on his struggle to descend, and will often be 2–3 metres underwater before he 'remembers his ears'. This is referred to as 'equalizing behind the dive', and is overcome by autoinflating on the surface before descent and with each metre of descent. Alternately, autoinflation can be employed after each breath during descent. Open-water diving, without use of a descent line or anchor line, interferes with control of the descent and thus contributes to barotrauma.
2 Attempting to autoinflate while in the horizontal or head-down position. If only one ear causes difficulty, it is advisable to tilt that ear toward the surface while attempting autoinflation. This stretches the pharyngeal muscles and puts the offending tube in a vertical position, capitalizing on the pressure gradient of the water.
3 Diving with problems which cause eustachian tube obstruction, such as mucosal congestion from such factors as infections, irritants such as cigarette smoke, drugs or allergies. After an upper respiratory tract infection has cleared, another week or two is necessary before diving is resumed safely. Divers who have an allergic diathesis should avoid the allergens (e.g. avoid dairy products for 12–24 hours before diving).

Divers should be advised of the dangers of delaying middle-ear autoinflation, and also of using excessive force in achieving it.

Correct middle-ear autoinflation for divers 'equalizing ahead of the dive'

- *Practise and ensure reliable middle-ear autoinflation, on land. Only then, consider diving.*
- *Autoinflate middle ear on surface before dive.*
- *Autoinflate every 1 metre of descent. Use a descent line.*
- *Autoinflate with head upright.*
- *Do not descend if pressure is felt on ears. Abort dive.*
- *Do not use multiple ascents (Yo/Yo) or waiting, to.*
- *Do not dive with upper respiratory pathology* *(see p. 79).*

Other options

Some physicians have found the use of proteolytic or allegedly mucus-softening enzymes to be of value. Papain tablets can be chewed, and there is also a papain/bromelain mixture which contains calibrated amounts of the enzymes. It is taken three times a day, dissolved through the buccal pouch. These treatments may be commenced the day before the dive and continued during the diving trip. Well-controlled experimental trials are required to demonstrate the efficiency of these preparations.

Hyperbaric ear plugs. Recently, ear plugs have been promoted to reduce the symptoms of middle-ear barotrauma. The principle on which these are based is as follows: a small malleable, plastic, compressible and porous plug is fitted with an airtight seal into the external ear. This allows for air to move more slowly into the external ear space, during pressurisation (descent) in a chamber. If, for some reason, the middle ear is not adequately inflated, then the subject is likely to experience the pain of middle-ear barotrauma of descent, i.e. the pain associated with a tympanic membrane being drawn into the middle-ear space, and pulling on the sensitive tissues around its circumference.

The use of these 'hyperbaric plugs' will prevent the extreme inwards distortion of the tympanic membrane, being pulled into the middle ear – as they tend to move it in the opposite direction, i.e. outwards. Thus, the discomfort and pain of the 'negative' pressure in the middle ear, is less.

Nevertheless, these plugs do not change the pathology of middle-ear barotrauma, other than the effect on the tympanic membrane. Thus, the damage to the middle-ear mucosa, the oval and round windows and the inner ear all remain (being dependent of the pressure gradient between the middle-ear space and its surrounding body tissues). The only thing that has really changed is that the symptom of pain with middle-ear barotrauma has been lessened.

The literature on this product that a pressurization of 1 psi/min to 15 psi will result in a delay in the pressurization on the tympanic membrane from 15 minutes to 31 minutes, i.e. a 16-minute delay in equalization of middle and external ear pressure. This results in the tympanic membrane bulging outwards, if the middle-ear pressure is successfully equalized by autoinflation.

The potential pay-off for reducing the symptomatology of middle-ear barotrauma is as follows:

- The production of a mild external-ear barotrauma of descent.
- The persistence of pathology of ear barotrauma affecting the middle-ear mucosa and inner ear.
- Possible aggravation of ascent barotraumas affecting the ear, because of the pathology induced in the middle ear during descent.
- Vertigo from unequal middle-ear pressures when the plugs are not inserted equally into both sides.

Whether the masking of middle-ear pathology by reducing the symptoms is a wise move is open to question.

An alternative to the hyperbaric ear plugs is to pressurize more slowly (i.e. the same effect on the middle ear, without inducing an external ear barotrauma to achieve it).

'Diving' ear plugs, where a restricted opening replaces the ceramic filter, slow the barotrauma

pathology as described above, and increase the possibility of ascent barotrauma.

Various gadgets that connect the oral cavity with the external ear have been used in the false belief that they overcome the effect of impaired middle-ear autoinflation. This could only happen if the diver has a tympanic membrane perforation – in which case they should not be diving.

In patients who are unconscious, and need hyperbaric treatment (in a recompression chamber) middle-ear barotrauma is particularly frequent and *myringotomy* is often required. Professor Joe Farmer states that myringotomies are required for hyperbaric exposure in patients who are comatose or who have a tracheostomy or orotracheal or nasotracheal tubes. If repeated treatments are considered likely, tympanostomy tubes can be inserted.

In conscious divers who will only need one such hyperbaric treatment session, an alternative to myringotomy is to have repeated pauses or a very slow descent, accepting slower barotrauma or giving more opportunity for autoinflation.

MIDDLE-EAR AUTOINFLATION TECHNIQUES

Passive opening of the eustachian tubes is the ideal and natural way to equalize pressure between the middle ear and the nasopharynx, although it is not always possible. Most amateur divers need to use an active technique which will inflate the middle ears and prevent the pain and discomfort during descent. During ascent, passive equalization of ear pressures is more common, and active techniques are rarely needed.

Sometimes reluctant trainees use the failure to autoinflate ears as an acceptable excuse to avoid diving. Sometimes they are scared to use sufficient nasopharyngeal pressure, for fear of causing damage.

It is part of the routine diving medical examination to ensure that the diving candidate can autoinflate the middle ear actively. This is achieved by using a positive-pressure manoeuvre described to the diver, while the examiner is observing the tympanic membrane and its movement. The latter is seen either by focusing on the otoscopic light reflex or on another part of the tympanic membrane which reflects light (either the membrane flaccida or the circumference). As the candidate autoinflates the middle ear, the tympanic membrane moves outwards.

Sometimes the diver will not adequately pressurize the nasopharynx, and in these cases there will be no flaring of the nostrils as the diver occludes the nares and 'blows'. The following techniques are recommended, though different candidates perform them with varied ease. In each case, practise of the technique is recommended on land, before subjecting the novice to hyperbaric and aquatic conditions which interfere with application of this new skill.

Valsalva manoeuvre

This is probably the most easily understood. It involves occluding the nostrils, closing the mouth and exhaling so that the pressure in the nasopharynx is increased. This separates the cushions of the eustachian tube and forces air up this tube into the middle ear. The pressure required to achieve this is usually 20 to 100 cm H_2O.

The force necessary for the successful autoinflation will vary with the diver's body position. Using the Valsalva technique, novice divers average 40 cm H_2O in the head-up, vertical position, and in the horizontal ear-up position. In the horizontal ear-down position they need 50 cm H_2O. In the vertical, swimming-down position they average about 60 cm H_2O.

Note that the Valsalva used by divers does not include closure of the glottis (larynx) – as employed originally by Antonio Valsalva to increase intrathoracic pressure – and usually only for 1–2 seconds. Trials performed on divers indicate that they do not produce the high thoracic pressures often encountered by cardiothoracic physiologists.

The problems induced by inappropriately high Valsalva pressures, prolonged for many seconds, include cardiac arrhythmias, hyper- and hypotension, arterial and venous haemorrhages, pulmonary and otological barotrauma, gastric reflux, stress incontinence and the possible shunting of blood through right-to-left vascular shunts (atrial septal

defects and patent foramen ovale) increasing the possibility of paradoxical gas embolism.

Frenzel manoeuvre

This involves closing the mouth and nose, both externally and internally (this is achieved by closing of the glottis) and then contracting the muscles of the mouth and pharynx upwards ('lifting the Adam's apple'). Thus, the nose, mouth and glottis are closed and the elevated tongue can be used as a piston to compress the air trapped in the nasopharynx and force it up the eustachian tube. A pressure of less than 10 cm H_2O may accompany this manoeuvre.

Toynbee manoeuvre

This involves swallowing with the mouth and nose closed, and is of value in relieving the over-pressure in the middle ear during ascent. It is also of value during descent when movement of the eustachian cushions produce a nasopharyngeal opening of the eustachian tube, with an equalization of pressures between the nasopharynx and the middle ear. Thus, the final pressure in the middle ear with the Toynbee manoeuvre may be negative (less than environmental).

Lowry technique

A combination of the techniques have also been proposed. A very successful one is the combination of the Toynbee and Valsalva, known as the Lowry technique. This involves occlusion of the nostrils, then a swallowing movement which is made continuous with a Valsalva manoeuvre. The diver is thus advised to 'hold your nose, blow and swallow at the same time'. Despite the rather confusing (and impossible to achieve) instruction, the technique is extremely valuable in resistant cases. It is easily learnt with practice, on land.

Edmonds technique

This is rather similar to the Lowry technique, and involves the opening of the eustachian cushions by rocking the lower jaw forward and downward (similar to the start of a yawn) so that the lower teeth project well in advance of the upper teeth and performing the Valsalva manoeuvre at the same time.

In the Edmonds No. 2 technique, the advice to the diver is to 'Block your nose, close your mouth, then suck your cheeks in, then puff them out – quickly'.

BTV technique

The voluntary opening of the eustachian tubes (beance tubaire voluntaire, BTV) refers to the opening of the eustachian cushions and allowing the pressure difference between the middle ear and nasopharynx to equalize. This tends to be performed by experienced divers who, over the years, have developed this muscular skill.

An interesting variation of the BTV, is the Roydhouse technique. Here, the diver is asked to identify the uvula hanging down from the posterior of the hard palate, then raising it as he moves the back of the tongue downwards. This opens the eustachian tubes, a situation which can be verified by the diver when he hears his own humming sounds reverberate in his ears.

Which technique?

As divers become more experienced, they tend to utilize such techniques as jaw movements, commencing a yawn, swallowing, lifting the soft palate, etc. which allow for equalization of the middle ear without pressurizing the nasopharynx.

When examining potential divers, attempts by them to demonstrate either the Frenzel or the BTV are not usually successful. In our practise, during otoscopy, the Valsalva manoeuvre is tried first, followed by the Toynbee, the Lowry and then the Edmonds techniques. If there is any difficulty remaining with equalization, then the candidate is advised to repeat the most effective procedure a few times a day, and to achieve success – determined by hearing both ears click – before commencing the diving course.

Academic arguments abound as to which is the best technique. In truth, whichever works is the best. The major problem is not the danger of middle-ear autoinflation, but the dangers of **not** autoinflating.

Some techniques (Valsalva, Lowry, Edmonds) have the disadvantage of a transitory pressure that

may extend into the thorax, but this is not usual with divers. They have the advantage of distending the middle ear, thus allowing further descent without the problems of a negative middle ear pressure developing and producing middle-ear congestion and eustachian tube obstruction. These are therefore better for divers who have trouble with middle-ear autoinflation. They also assist in equalizing sinus pressures and avoiding these barotraumas.

Other techniques (Toynbee, BTV) are effective if there is easy and frequent middle-ear autoinflation. They either equalize the pressures passively or produce negative middle-ear pressures. Wave action or descent can also cause a negative middle-ear pressure with congestion and eustachian tube obstruction, and these techniques may aggravate this situation.

Experienced divers, who may have mobile tympanic membranes like small spinnakers, can often descend to great depths before they need to equalize their middle ear pressures. They also autoinflate their ears using less pressure.

Most patients referred to us with inability to autoinflate their middle ears, have suffered more from inadequate diver instruction than eustachian tube obstruction. To ascertain the extent of this problem, 200 consecutive otoscopic examinations were recorded on potential diving candidates. Autoinflation was successful using either the Valsalva, Toynbee, Lowry or Edmonds techniques in 96 per cent of subjects, with 4 per cent being unsuccessful in one or both ears.

Decongestants probably do work on the mucosal membranes and do improve nasal air flow, but their effect on the eustachian tube is greater at the nasopharyngeal orifice and thus may be more effective in reducing descent than ascent middle-ear barotrauma.

Some otologists have recommended the use of the **pneumatic otoscope** to demonstrate passive tympanic membrane movement. This is certainly of value in assessing middle-ear pathology, but it is inadequate for diving assessment, as it fails to demonstrate the procedure that is required, i.e. *voluntary* autoinflation of the middle ear.

Other investigations, of more value in assessing middle-ear autoinflation, include the modified tympanogram (see Chapter 36) and examination of the eustachian cushions with a fibreoptic nasopharyngoscope.

MIDDLE-EAR BAROTRAUMA OF ASCENT

This refers to symptoms caused by distention of enclosed gases within the middle ear, expanding with ascent. Because it may prevent ascent, it is usually more serious than middle-ear barotrauma of descent – which allows an unhindered return to safety.

During ascent, the middle ear opens passively, with a pressure gradient of around 50 cm H_2O (70 cm H_2O in the head-down position). If the eustachian tube restricts its release, symptoms may include sensations of pressure or pain in the affected ear, or vertigo due to increased middle ear pressure difference (alternobaric vertigo). Occasionally these co-exist.

Middle-ear barotrauma of ascent usually follows recent, but sometimes mild, middle-ear barotrauma of descent and/or the use of nasal decongestants. In each case the common factor is probably a congestion and therefore blockage of the eustachian tube.

The **mild vertigo** is often rectified by further ascent, which may open the less patent eustachian tube. When the pressures in both are equalized with

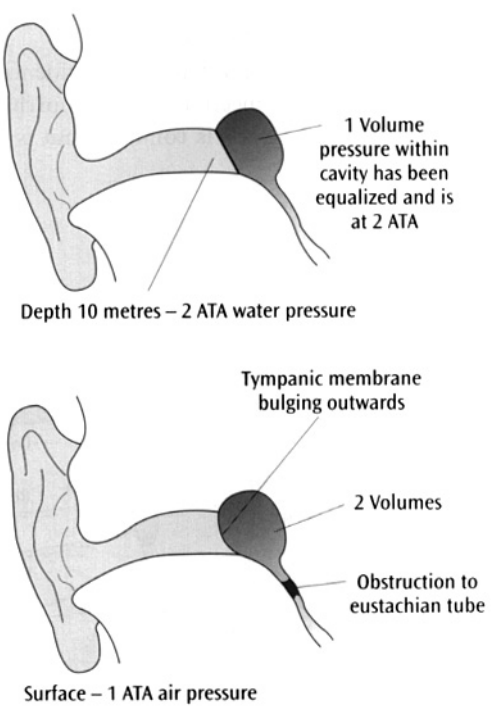


Figure 7.3 *Middle-ear barotrauma of ascent.*

the ambient pressure, the stimulus to vertigo ceases. Subsequent opening of the tube is also easier. Other cases may reach the surface while still having an asymmetry of pressure within the middle-ear cavities, or residual damage from excessive middle-ear pressure, and they may experience vertigo following the ascent.

Most cases of middle-ear barotrauma of ascent are mild, but this is not always so and there have been instances of inner-ear damage, of progressive pain during ascent, or perforation of the tympanic membrane.

The vertigo is most pronounced when the diver assumes the vertical position, and least in the horizontal. The spinning is towards the ear with the higher pressure, and it tends to develop when the middle-ear pressures differ by 60 cm H_2O or more.

The diver may be able to take remedial action. Occasionally, the Valsalva technique, jaw movements or performing a Toynbee manoeuvre (see above) will relieve the discomfort, as may sudden pressure applied to the external ear (by inward pushing of the water column in the external ear, with the tragus or middle lobe). Equalization may be easier if the affected ear is facing the sea bed, thereby utilizing the pressure gradient from the external ear to the throat.

Otoscopic examination often reveals evidence of tympanic membrane injection or haemorrhage. Congestion of blood vessels is common, but is less than with descent barotrauma. It is more pronounced around the circumference of the tympanic membrane than along the handle of the malleus. The tympanic membrane may appear to be bulging.

Hearing loss in the affected ear, if present, may be conductive and follow damage to the tympanic membrane or the middle-ear structures. The tympanic membrane may rupture occasionally. Inner-ear barotrauma with sensorineural hearing loss, is a possible complication (see Chapter 37). Seventh nerve palsy is also a possible complication.

Fortunately, most effects are short lived, and **treatment** should consist of prohibition of diving until clinical resolution has occurred, normal hearing and vestibular function are demonstrated, and prevention of future episodes is addressed.

Rarely, the diver will be seen soon after the event and if the middle ear is still distended, the procedures described above for the diver to perform may be satisfactory. Otherwise, the use of oxygen inhalation or minimal recompression are effective.

Antibiotics are used if there is evidence of infection, and decongestants are sometimes used to improve the eustachian tube patency. Usually neither are indicated. **Decongestants**, especially topical ones, are rarely of use in **preventing** this disorder, unless they prevent a causal middle-ear barotrauma of descent. Usually they have the opposite effect. Systemic decongestants are more effective, but they have other

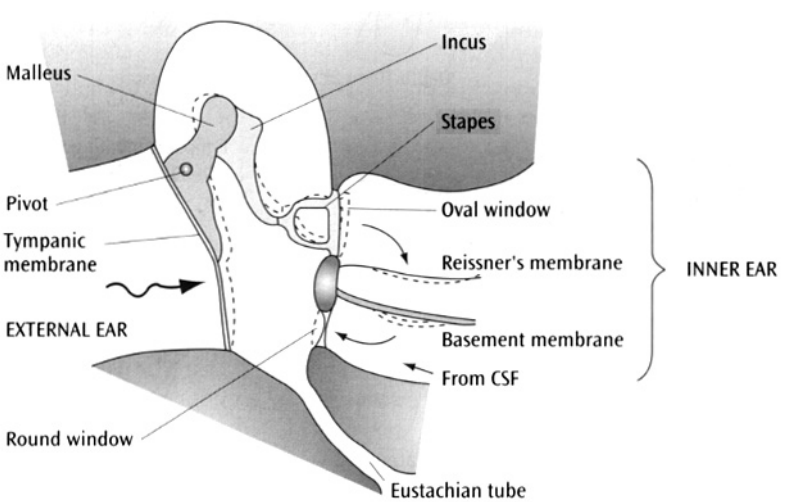
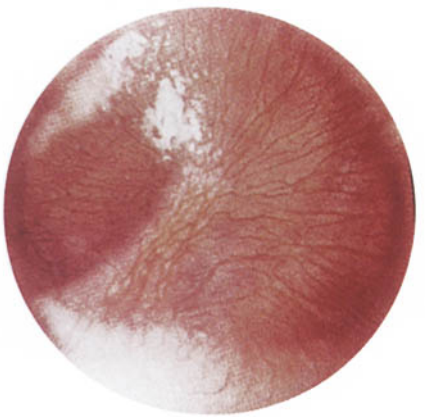
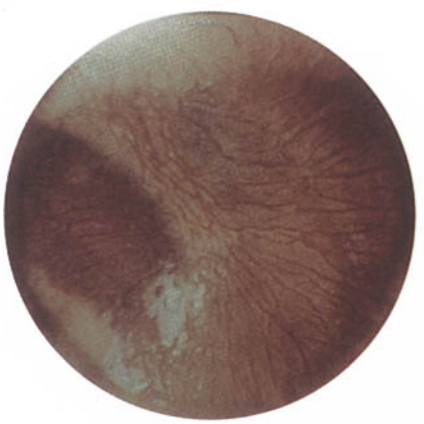


Figure 7.4 *Pressure wave (e.g. sound) passing from external, through middle, to inner ear.*

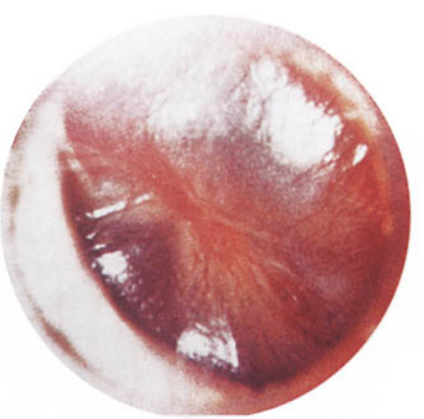
Grade 0



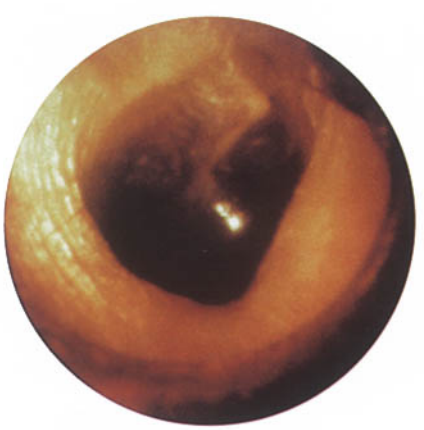
Grade 1



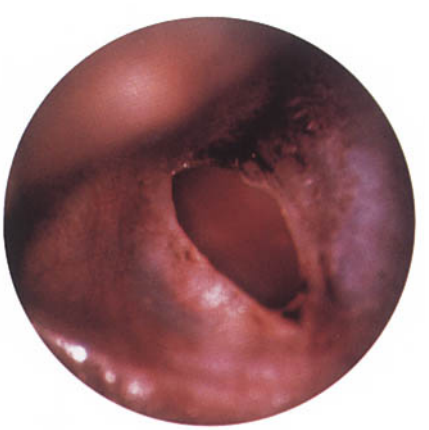
Grade 2



Grade 3



Grade 4



Grade 5

Plate 1 *Middle-ear barotrauma of descent: grades 0 to 5, graded by otoscopy.*

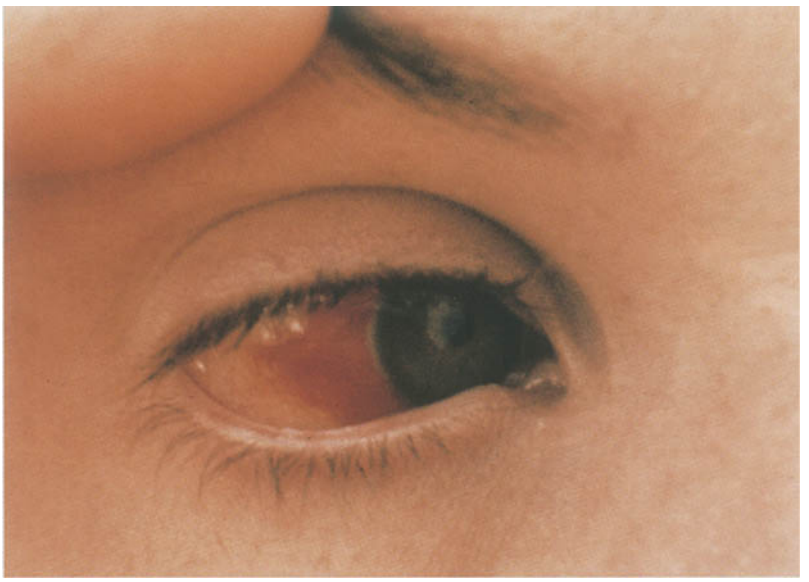


Plate 2 *Facial barotrauma of descent (mask squeeze), causing subconjunctival hemorrhages and facial edema when this diver did not equalize the pressures within the mask during descent to 15 metres.*

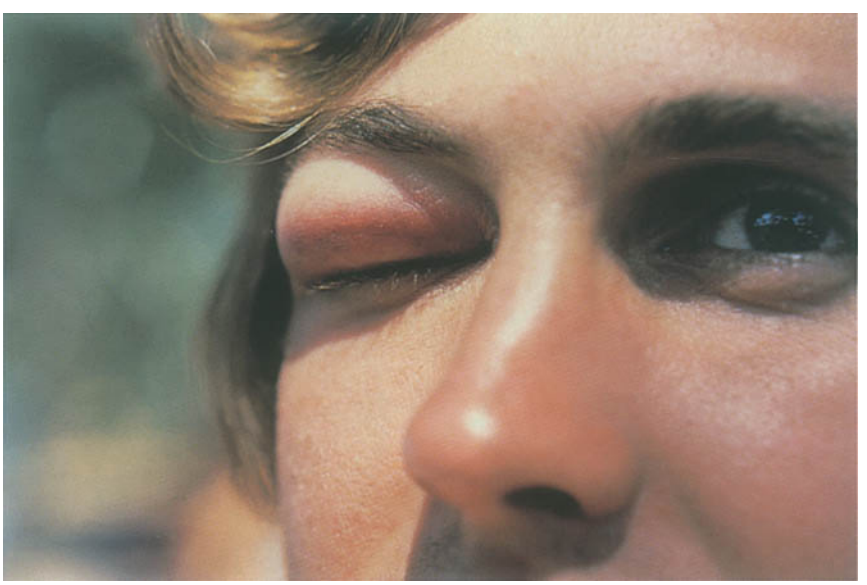


Plate 3 *Orbital surgical emphysema. Following a recent vehicle accident, this diver performed a Valsalva maneuver at a depth of 9 meters. The otherwise asymptomatic fracture to the lamina papyracea allowed gas to pass from the nasal cavity to the orbit, causing surgical emphysema which expanded on ascent, occluded the palpebral fissure, and produced subcutaneous hemorrhage.*

disadvantages, permitting the diver to descend with marginal improvement in eustachian tube patency and inadequate autoinflation of the middle ear.

Prevention is best achieved by avoiding nasal decongestants and by training the diver in correct middle-ear equalization techniques during descent (see above). Unless descent barotrauma is prevented, ascent barotrauma is likely to recur.

Once middle-ear barotrauma of ascent has been experienced, particular care should be taken to ensure that if it does recur, the diver will always have adequate air to descend briefly, use the techniques described above, then gradually ascend. A low-on-air situation could cause extreme discomfort or danger, if the diver's ascent is restricted by symptoms.

Vestibular function has been tested experimentally while undergoing pressure changes in a recompression chamber, to replicate the sequence of events and verify the aetiology and diagnosis (see Chapter 38). It is not generally required.

INNER-EAR BAROTRAUMA

Overview

There is always the possibility of inner-ear damage in divers who have:

- experienced ear barotrauma of any type; or
- who have had difficulty in equalizing middle-ear pressures by autoinflation; or
- who subsequently apply excessive force to achieve this.

In these cases, sensorineural hearing loss may immediately follow the dive, or may develop over the next few hours or days. **Tinnitus** is a common association, while some patients (but certainly not all) may complain of vertigo, nausea and vomiting. There may be no otoscopic signs.

Combined cochlear/vestibular injury is experienced in 50 per cent, only cochlea injury in 40 per cent, and only vestibular in 10 per cent. It has been reported from dives as shallow as 2 metres and has even been observed in a surfer who dived under a wave. Animal experiments reproduce the pathology with equivalent depths of 1–6 metres.

In the event of otological barotrauma, a sensorineural or combined hearing loss, tinnitus or demonstrable vestibular damage, implies inner-ear barotrauma.

Pathophysiology

Middle-ear barotrauma is the commonest cause of inner-ear damage in diving, and a variety of inner-ear pathologies have been demonstrated (for anatomical background, see Chapter 35).

A **perilymph fistula** is a common pathological entity of inner-ear disease. The perilymph leak is variable in volume and may come from the round window (most often), the oval window or a membrane rupture within the labyrinth. Perilymph fistulae from the labyrinthine windows are now well-recognized by otologists, and result in a leakage of perilymph into the mastoid or middle-ear space. In general medicine it may be related to congenital syphilis or other infections, cholesteatoma or any sudden increase in intercranial or labyrinthine pressure. It can develop spontaneously, or be due to trauma, especially with head injury, weight lifting and physical straining. The pressure wave so produced can be transmitted into the inner ear, by the cochlea and possibly the vestibular aqueduct. An increased pressure of 120 mm H_2O in the cerebrospinal fluid (CSF) is sufficient to induce this in some patients.

Any procedure which involves manipulation of the ossicular chain can cause an oval window perilymphatic fistula, and this occurs in 7 per cent of post-stapedectomy patients.

The hearing may fluctuate, depending on the replacement of perilymph, or it may progress slowly or suddenly as the perilymph leaks out. The quicker the ear replenishes its perilymph, the less likely it is to sustain permanent damage. The prognosis is better when only the low or mid frequencies are affected. Both the loss of pressure within the perilymphatic system, and the possible electrolyte imbalances, affect the dynamics of the neural elements of the hearing and vestibular systems, and the damage may become permanent if not corrected.

Exposure to environmental pressure change is possibly one of the commonest causes, and this

includes ear barotrauma of diving and aviation exposure.

There are two postulated mechanisms for this disorder in diving. In one, with descent the tympanic membrane moves inward because of the pressure gradient, resulting in the footplate of the stapes being pushed inwards. This causes a displacement of perilymph through the helicotrema, so that the round window membrane bulges outwards. If at this stage a forceful Valsalva manoeuvre is performed, there is an increase in the pressure within the middle-ear cleft. This leads to the tympanic membrane being very rapidly returned to its normal position, the stapes moving outwards, and the round window being pushed inwards. The reversed flow of perilymph may not be sufficiently rapid to avoid damage to the inner-ear structures, e.g. rupture of the round window membrane.

The other explanation involves a pressure wave transmitted from the CSF through a patent cochlea aqueduct during the Valsalva manoeuvre, and 'blowing out' the round window into the middle ear. This has been demonstrated in animal experiments, with a rise of CSF pressure. The aqueduct constricts with age, and may explain why children are more susceptible.

Inner ear barotrauma has occurred in unconscious patients and guinea pigs, indicating that a forceful Valsalva manoeuvre is not a necessary prerequisite. Animal experiments suggest *multiple pathologies* for the inner-ear damage. End artery spasm, thrombosis, gas or lipid embolism, are aetiological proposals which have little experimental or clinical support.

It is likely that cochlea and vestibular haemorrhages and internal inner-ear membrane ruptures are common – but are not amenable to treatment. A tear of Reissner's membrane results in an isolated loss in one or two frequencies (tested on 100-Hz increments between 400 and 1300 Hz). Stretching of the round window, with the entry of air into the cochlea and resultant sensorineural hearing loss, has been demonstrated. A progressive sensorineural loss or vertigo which develops hours or days after the barotrauma incident is most likely to be due to a **fistula of the round window**, with leakage of the perilymph into the middle ear, or air into the perilymph. This can develop at any stage of the dive, or afterwards. Expansion of gas during ascent or a rise in pressure in the middle ear by forceful middle ear autoinflation, may force air across the round window into the perilymph.

Symptoms

Many of these cases develop the first symptoms after the completion of the dive, while performing energetic tasks, e.g. pulling up the anchor. This may be because the middle ear (including the round window) has been damaged by the earlier barotrauma. The subsequent fistula follows a rise of pressure in the CSF, the cochlea aqueduct and the perilymph. Sudden tinnitus and hearing loss may be more frequent with inner-ear haemorrhages.

Progressive deterioration of sensorineural hearing, over hours or days, fluctuating hearing loss and positionally induced hearing loss indicate a perilymph fistula. Persistence of vestibular symptoms may indicate perilymph fistula. Deafness is of the sensorineural type, either a total (all frequencies) or selectively high-frequency loss (4000–8000 Hz). It also may be variable and altered by changing head positions – possibly because of the buoyancy of air in the perilymph or increased leakage into the middle ear. **Tinnitus**, with a ringing, roaring, popping or running water-type sound is frequent. Aural fullness and hyperacousis are described. If left untreated, the sensorineural hearing loss may become total and permanent.

Impairment of speech discrimination may precede or overshadow the delayed and progressive hearing loss. There is often an associated conductive or lower frequency hearing loss which resolves over the subsequent one to three weeks, and may be mistakenly interpreted as a therapeutic success. Bone conduction audiograms are indicated to identify this.

Oval window fistulae, probably due to damage from the stapes footplate, have been observed, often with a severe vestibular lesion that may persist until surgical repair. It is more likely in divers who have had surgery for otosclerosis.

The symptoms of inner-ear barotrauma may include those of vestibular origins such as vertigo, nausea, vomiting and ataxia. Vestibular symptoms vary from almost unnoticeable to incapacitating. In the cases which initially, predominantly, or solely

involve vestibular function, the symptoms may progressively diminish as adaptation occurs. Even though the symptoms may diminish, the lesion may progress to destruction of the vestibular system. In others, vertigo may persist or recur while the fistula persists or recurs.

Hennebert showed that an increase in pressure in the ear canal could produce **nystagmus**, in patients who were known to have perilymph leakage. Tullio described a similar response with loud sounds. Similar clinical experiences have been observed in patients with perilymph fistula, their vertigo being induced by any activity which increases the pressure in the ear canal (ascent and/or descent, loud sounds of low frequency, Valsalva manoeuvre, tragus pressure, pneumaticotoscopy, or tympanometry).

Symptoms associated with inner-ear barotrauma may include:

- *Sensation of blockage or fluid in the affected ear.*
- *Tinnitus of variable duration.*
- *High-frequency hearing loss.*
- *Vestibular disturbances such as nausea, vomiting, vertigo, disorientation and ataxia.*
- *Clinical features of an associated middle-ear barotrauma (± conductive hearing loss).*

Unfortunately, the clinical differential diagnosis between cochlear/vestibular trauma, haemorrhage and perilymph fistula, based on the above criteria, is by no means certain.

Once inner-ear barotrauma has been experienced, the diver is more predisposed to similar incidents, which further aggravate both the tinnitus and the hearing loss. Cochlea injury is permanent in over half the cases, whereas vestibular symptoms are usually temporary. Meningitis is a possible complication of perilymph fistulae.

Inner-ear barotrauma is suspected in the presence of hearing loss, tinnitus, vertigo or ataxia.

Investigations

In order to demonstrate inner-ear barotrauma, serial investigations may be necessary. Any combination of middle-ear barotrauma symptoms, nausea, vertigo, tinnitus and hearing loss should be immediately and fully investigated by serial measurements of clinical function, daily audiometry up to 8000 Hz (with bone conduction if the loss is in the <4000 Hz range) and positional electronystagmography (ENG). Caloric testing is indicated only if the tympanic membrane is intact, or if the technique guards against pressure or fluid transmission into the middle ear. Other tests are less valuable.

A test proposed to support the diagnosis of perilymphatic fistula, as opposed to other causes of inner ear damage, is that of **positional audiometry**. The patient lies horizontal with the affected ear uppermost, for 30 minutes, and the hearing improves more than 10 dB in at least two frequencies. The theoretical explanation for this improvement is that air is displaced from the perilymph-leaking windows.

Other investigations are sometimes thought to be even more sensitive than the ENG. In diagnosing perilymph fistulas, Kohut suggested that the presence of Hennerbert's sign (or its equivalent Tullio phenomenon) is required before vestibular symptoms are attributed to a perilymph fistula. Other tests, being investigated to verify this pathology include dynamic posturography, vestibulospinal response (body sway) reactions to stress (Hennerbert's or Tullio) and electrocochleography.

Investigations that may be of value include temporal bone polytomography, computed tomography (CT) and other imaging techniques. Until now they have not been particularly helpful in diagnosis or treatment, but their discrimination is improving rapidly.

Treatment

Treatment should be initiated promptly, applying the following principles.

1 Avoid any increase in CSF pressure, from performing Valsalva manoeuvres, sneezing, nose-blowing, straining with defecation, sexual activity,

coughing, lifting weights, fast movement or physical exertion. Divers very commonly perform middle-ear autoinflation, almost as a matter of habit. Advise the patient that under no circumstances should autoinflation be performed. Otherwise, the already damaged round window may not withstand the pressure wave.

2 Almost total bed rest with the head elevated and careful monitoring of otological changes; this is given irrespective of which of the other treatment procedures are followed.

3 Bed rest should continue until all improvement has ceased, and for up to a week thereafter, in order to allow the inner-ear membranes to heal and the haemorrhages to resolve. Loud noises should be avoided.

4 If there is no improvement within 24–48 hours in cases of severe hearing loss, or if there is further deterioration in hearing, operative intervention must then be considered.

5 Reconstructive micro aural surgery is indicated when there is deterioration or no improvement with bed rest, with severe hearing loss or incapacitating vertigo. With developing hearing loss, repair to the round or oval window will prevent the further leakage of perilymph and has proved curative in some cases, sometimes restoring hearing acuity. It cures vertigo and reduces tinnitus, both of which may be disabling. If a fistula is not visualized during middle ear exploration, a graft should still be applied to the window, as sometimes the fistula is intermittent. Some surgeons use fluorosaline to make the leakage more obvious. Others employ techniques to increase the CSF pressure. Middle-ear exploration may cause further hearing loss. Surgery, which was employed in most cases in earlier years, is rarely needed now if conservative treatment is given conscientiously. After two weeks delay surgery will rarely improve hearing. Middle-ear surgical exploration is not indicated in cases of inner-ear haemorrhage, as it is not a harmless procedure, and in rare cases it can induce further or complete hearing loss.

6 Prohibition of diving and flying is essential for the first few weeks following a perilymph fistula. If medical evacuation by air is required, an aircraft with the cabin pressurized to ground level is necessary. For most cases, but especially those precipitated by minimal provocation and who have poor eustachian tube function and/or nasal pathology, it is prudent to advise against any further hyperbaric (diving) exposure. The same applies if permanent hearing loss, tinnitus or vestibular asymmetry persist.

7 Treatment for vertigo is based on routine medical principles. It is usually suppressed by cerebral inhibition within a few weeks – but may be precipitated by sudden movement or other vestibular stimulation (caloric or alternobaric). It may persist if the fistula remains patent.

8 Other regimes. Vasodilators (nicotinic acid, carbogen, etc.) have been recommended by some, but little evidence exists to show any favourable effect. Aspirin is to be avoided, or discontinued, because of its anticoagulant effects. Steroids have no verified place in the treatment of this type of hearing loss.

9 Air entry into the perilymph, as a cause of the pathology, has yet to be quantified. As a relatively harmless procedure, we sometimes add 100 per cent oxygen breathing to the conservative treatment regime for 4–6 hours a day for three days.

10 Hyperbaric oxygen therapy has been used and recommended by some experienced hyperbaric therapists, and warrants further investigation. The present authors have tried it, but had subsequently to proceed to surgery. Hyperbaric oxygen has the potential for aggravating the fistula and increasing the perilymph flow into the middle ear during descent – both from the relatively negative middle-ear pressures and the need for Valsalva manoeuvres. It has been responsible for 'cures' in some cases of middle-ear barotrauma with conductive generalized hearing loss, misdiagnosed and reported as inner-ear barotrauma. In these cases, if the middle-ear is autoinflated with descent, the gas expansion removes middle ear fluid on ascent. It may be of value in other forms of sudden hearing loss, with or without steroids.

Prognosis

After inner-ear barotrauma there may be an apparent complete cure, or persisting residue. The cochlea acuity (especially lower-frequency hearing) may improve for a few weeks, and then the remaining

CASE REPORT 7.2

This diver, who had been exposed to gunfire in the past, experienced considerable pain and difficulty in equalizing both middle ears during a dive to 10 metres. He continued to dive despite the pain, and performed forceful autoinflations. He noted tinnitus, and also experienced ear pain and vertigo during ascent. Otoscopic examination of the tympanic membrane revealed the effects of barotrauma. The diver became progressively more deaf, with a sensorineural pattern in both ears, over the next few days. Transient episodes of vertigo were noted. Exploratory surgery was performed. A fistula of the round window was observed, together with a frequent drip of perilymph fluid into the middle ear. The round window was packed. A similar procedure was performed five days later in the other ear, with the same result. Subsequent audiograms over the following month revealed a considerable improvement in hearing.

Diagnosis: inner-ear barotrauma (with perilymph fistula of the round window) due to middle-ear barotrauma of descent and forceful autoinflation, resulting in sensorineural hearing loss.

high-frequency loss is usually permanent – to be aggravated by the effects of ageing.

Tinnitus often improves over the next 6–12 months, presumably the effect of damaged sensory endings either being repaired or dying.

If the vestibular system is damaged and an asymmetry persists, the patient will never be able to dive or fly safely, because of alternobaric vertigo. He may continue to have occasional vertigo, aggravated by sudden head movement, which is then a hazard in all occupations that involve balance, exposure to heights or driving. Associated cochlea damage is also present in most cases.

The present authors would advise against piloting aircraft because of the danger of alternobaric vertigo, which has followed some cases of unilateral inner-ear damage. Too many cases of inner-ear barotrauma have recurred, for these authors to agree to the resumption of diving – either free or with equipment – once permanent inner-ear damage has been demonstrated.

MIDDLE-EAR DISEASE COMPLICATIONS

Seventh nerve palsy

The seventh or facial cranial nerve may be affected, causing 'facial baroparesis'. Recorded in both aviators and divers, this is more frequent following middle-ear barotrauma, and presents as a unilateral facial weakness similar to Bell's palsy. It tends to recur in the same patient if the cause is not corrected.

The reason for this disorder is found in the anatomy of the facial canal, which opens into the middle ear in some people, and shares its pathology. Paralysis of the facial nerve makes frowning impossible, prevents the eye from closing on that side, and causes drooping of the lower lid (which may result in tears running down the face because they do not drain into the nasolacrimal duct). The cheek is smooth and the mouth pulled to the normal side. Whistling becomes impossible, and food collects between the cheek and gum. A metallic taste may be noticed at the start of the illness, as may impaired taste in the anterior part of the tongue on the same side, from chorda tympani involvement. Hyperacousis may be due to paralysis of the stapedius muscle.

Both physicians and otologists frequently omit to interrogate Bell's palsy patients regarding their swimming, diving and aviation exposure.

Otitis media

Although not frequent, this is an occasional complication of middle-ear barotrauma, with the middle ear collecting fluid which forms a medium for growth of organisms (see Chapter 29). Thus, ear pain develop-

ing hours or days after middle-ear barotrauma, should be considered to be a middle-ear infection. This is not only a serious illness in its own right, but also a possible cause of narrowing of the Eustachian tube and further middle-ear barotraumas.

Mastoiditis

The mastoid, being part of the middle ear cleft, will respond in the same way as the middle ear, to a negative pressure situation. Thus, the production of fluid and blood in the mastoid, especially during descent, can develop and produce the conditions conducive to an infection. Under these circumstances there is usually pain and tenderness over the mastoid, and the pathology can be demonstrated by CT of the temporal bone.

Meningitis

Although rare, this is a possible extension of otitis media, mastoiditis, sinusitis, etc., and a complication of both labyrinthine fistula and pneumocephalus.

Pneumocephalus

Another complication from the mastoid is a pneumocephalus, due to the expansion of gas in the mastoid air cells, into a space which is now also occupied by blood and fluid from the descent barotrauma. A rupture into the cranial cavity, with air and/or fluids, produces an excruciating and sudden headache, with the pathology demonstrated by CT brain scans and magnetic resonance imaging (MRI) (see Fig. 9.4)

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8

Sinus barotrauma

CARL EDMONDS

AVIATION EXPERIENCE

Almost sixty years ago sinus barotrauma from aviation exposure was well described by Campbell.[1,2] The injury was due to the changes in volume of the gas spaces within the paranasal sinuses during ascent or descent – when those changes could not be compensated by the passage of air between the sinus and the nasopharynx. It was the clinical manifestation of Boyle's law, as it affected the sinuses.

The **pathological** changes found within the sinuses included: mucosal detachment; submucosal haematoma; blood clots in membranous sacs; small haemorrhages within the mucosa; and swelling of the mucous membrane.

Weissman *et al.*[3] described a series of 15 cases of **frontal sinus barotrauma** (mostly Grade III) in aviators, using a grading systems as follows:

- Grade I: a transient discomfort which cleared promptly and had only a slight mucosal oedema, but no X-ray changes.
- Grade II: characterized by pain over the affected sinus for up to 24 hours. There was thickening of the mucosa seen on X-ray. If such a sinus was opened, small amounts of blood-tinged fluid were found. Serosanguinous fluid sometimes drained from the sinus, with or without the use of decongestants.
- Grade III: a severe pain or a 'bee-sting' or 'being shot' sensation. If the pain was not quickly relieved by the Valsalva manoeuvre, the pilot had to descend rapidly to relieve symptoms.

Usually aviators with Grades I and II did not seek medical aid, and were usually treated simply. Grade III cases resulted in oedema and congestion of the sinus mucosa with submucosal haemorrhages. As the sinus mucous membrane was pulled away from the periostium by the negative intra-sinus pressure, a haematoma formed. X-ray of the sinuses showed an air fluid level or a polypoidal mass, the incision of which brought forth a spurt of old blood, with clots.

DIVING EXPERIENCE

Reviews of the diving-related sinus barotraumas were not easy to find. Flottes,[4] in 1965 described sinus barotrauma in divers.

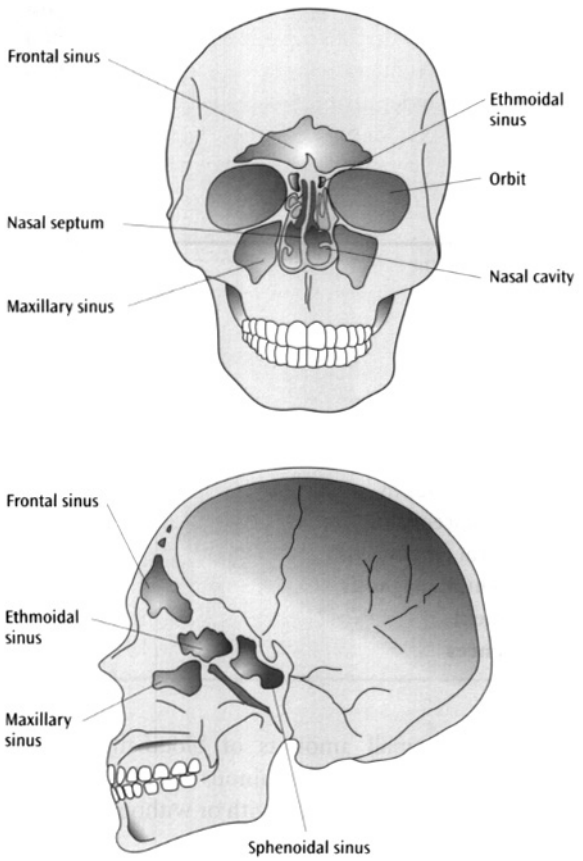

Figure 8.1 *Anatomy of accessory nasal sinuses.*

Sinus barotrauma has been described in various texts on diving medicine,[5] but initially without specific clinical series being documented. A reasonably large clinical series of divers with sinus barotrauma was first described in Australia by Fagan *et al.* in 1976[6] and quoted widely thereafter. This series described minor and acute cases, and was complemented by another series of 50 more serious cases, referred for definitive treatment.[7]

The first Australian series[6]

This series included 50 consecutive cases of sinus barotrauma (equivalent to Campbell Grades 1 and 2 sinus barotrauma) as they were observed in a Navy setting, where all such cases were referred for medical opinion, irrespective of severity. It included many cases that might otherwise have not attended for treatment. Some 68 per cent of the presenting symptoms developed during or on descent, and in 32 per cent during or after ascent.

In the majority of cases, the divers were undergoing their first open-water diver training course. **Pain** was the predominant symptom present, in all the cases on descent, and in 75 per cent of those on ascent. It was referred to the frontal area in 68 per cent, the ethmoid in 16 per cent and the maxillary in 6 per cent. In one case, pain was referred to the upper dental area.

Epistaxis was the second most common symptom, occurring in 58 per cent of cases. It was rarely more than an incidental observation, perhaps of concern to the diver but not usually of great severity. It was the sole symptom in 25 per cent of the cases of ascent barotrauma.

Even though these were inexperienced divers, in 32 per cent there was a history of **previous sinus barotrauma**, produced by scuba diving, aviation exposure or free diving. Half had a history of recent **upper respiratory tract inflammation** and others gave a history of intermittent or long-term symptoms referable to the upper respiratory tract, e.g. nasal and sinus disorders, recurrent infections, hay fever, etc. In 48 per cent of cases, otoscopy showed evidence of middle-ear barotrauma on the tympanic membrane.

Radiologically, the affected sinuses did not replicate the frequency of the clinical manifestations. The maxillary sinus had either mucosal thickening or fluid level in 74 per cent of the cases, the frontal in 24 per cent and the ethmoid in 15 per cent. This contrasts with the clinical manifestations. A fluid level was present in 12 per cent of the maxillary sinuses.

Most of these divers required no treatment, or responded to short-term use of nasal decongestants. Antibiotics were prescribed if there was a pre-existing or subsequent sinusitis. Neither sinus exploration nor surgery was required in any case. This series has been used inappropriately to imply that such intervention is never applicable in the treatment of sinus barotrauma.

This prospective Australian series, by its design, included relatively minor cases of sinus barotrauma. It has, by default, been used as being typical of all sinus barotrauma cases – even those that present

with recurrent or delayed symptoms, or complications, in emergency wards or ear, nose and throat (ENT) consulting rooms. That extrapolation is not necessarily valid. Moreover, it was done over two decades ago, before computer imaging techniques became commonplace.

Sinus barotrauma and its complications remain a common medical problem of diving, the importance having been stressed by many workers including Edmonds *et al.*,[8] Becker and Parell,[9] Neblett[10] and Roydhouse.[11]

The second Australian series[7]

A series of 50 more severe cases i.e. those referred for treatment of sinus barotrauma (equivalent to a Campbell Grade III sinus barotrauma), was presented in 1994. These were seen within one month of the latest incident. The cases were self-selecting because the divers with repetitive or more significant problems were more likely to present for medical treatment. The investigations frequently involved computed tomography (CT) scans of the sinuses, sinus endoscopy and occasionally magnetic resonance imaging (MRI).

These cases were more experienced divers – 88 per cent had in excess of 50 dives. The distribution was skewed strongly to the extremely experienced, with 70 per cent of the divers having over five years' experience, and many being dive masters, dive instructors or professional divers. Because of the extreme amount of diving exposure in this group, it is presumed that the sinus ostia or ducts had become scarred and narrowed from the repeated insults they sustained.

In 12 per cent of the cases, the presenting **headache** developed and progressed whilst at depth. It could usually be made worse with subsequent ascents or descents, but the initial development of the headache during a time in which there was no substantial change in depth, did cause some confusion in the initial physician's assessment.

From the aviation literature, it is believed that a small degree of negative pressure[12] is sustainable within the sinuses, without symptoms. Exceeding this may be sufficient to cause a gradual effusion to develop, and the full or heavy sensation within the sinus may take some time to develop. Extrapolation would suggest that diving-related barotrauma could occur with a reduction in sinus air volume of 5–10 per cent, i.e., at a depth of 0.5–1 metre below the surface.

In 8 per cent of the cases there was a very clear-cut and dramatic sensation of a bursting or popping, during depth changes. Of these, half were on descent and half on ascent. This has been described in aviation medicine as the 'popping of a champagne cork', a 'gun shot', 'like a bee sting over the eye', or 'like being struck on the head with a club or bat'. It is presumed, both from the observations of Campbell[1,2] and Mann and Beck,[12] and from this series, that the sensation is due to a haemorrhage stripping up the mucosa of the sinus, produced by the negative intrasinus pressure with descent.

A similar sudden sensation can also occur from the rupture of an air sac or release of pressure from a distended sinus during ascent. This may be followed by a 'hissing' sensation of air movement, which may then relieve discomfort and pain. One of the cases involved the ethmoidal area, and there was a subsequent small oval-shaped haematoma noted over the ethmoid region, within hours (see Figure 8.2).

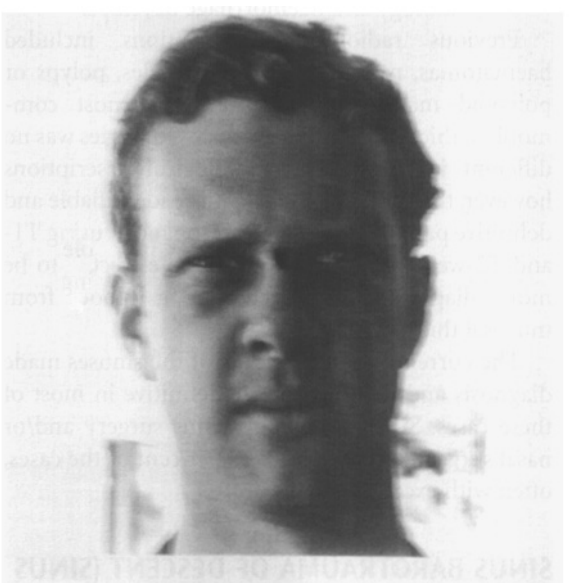

Figure 8.2 *After ethmoidal sinus barotrauma of descent, the sinus burst during ascent with sudden extreme pain, bruising and haemorrhage in adjoining skin.*

In 10 per cent of cases repetitive incidents of sinus barotrauma appeared to be provoked by inappropriate diving and equalization techniques. In these cases there would frequently be a head-first descent, and/or swallowing as a method of middle ear equalization. The substitution of the feet-first descent (preferably down a shot line), together with frequent positive-pressure middle ear equalization manoeuvres, appeared to rectify the situation. These techniques are now described in medical texts[16] used by divers.

A similar problem developed if descents were slow, because of discomfort noted in the sinus. The blood or effusion gradually accumulating in the sinus equalizes the pressure and reduces the degree of pain and discomfort. This might be appropriate for an emergency dive, but is not reasonable if pathology is to be avoided. On the contrary, divers inappropriately used the development of the pathology (blood or effusion, mucosal congestion, etc.) as a 'treatment' to replace a contracting air space in the sinus during descent, and allow the dive to continue.

Divers in these categories were advised of the correct methods of descent and to use positive-pressure middle ear equalization (e.g. a Valsalva manoeuvre). This may have an affect of aerating the sinuses before major pathology and haemorrhage develops.

Previous radiological descriptions included haematomas, mucous cysts, mucocoeles, polyps or polypoid masses, opacification and, most commonly, a thickening of the mucosa. Our series was no different in the various radiological descriptions however, the CT scans showed more identifiable and definitive pathology. Magnetic resonance[19] using T1- and T2-weighted imaging would be expected to be more diagnostic in differentiating blood from mucosal thickening.

The current use of CT scans of the sinuses made diagnosis and treatment more definitive in most of these cases. Sinus endoscopy, sinus surgery and/or nasal surgery was needed in 12 per cent of the cases, often with excellent results.

SINUS BAROTRAUMA OF DESCENT (SINUS SQUEEZE)

If a sinus ostium is blocked during descent, mucosal congestion and haemorrhage compensate for the contraction of the air within the sinus cavity. During ascent, expansion of the enclosed air expels blood and mucus from the sinus ostium. Ostia blockage may be the result of sinusitis with mucosal hypertrophy and congestion, rhinitis, redundant mucosal folds in the nose, nasal polyps, etc.

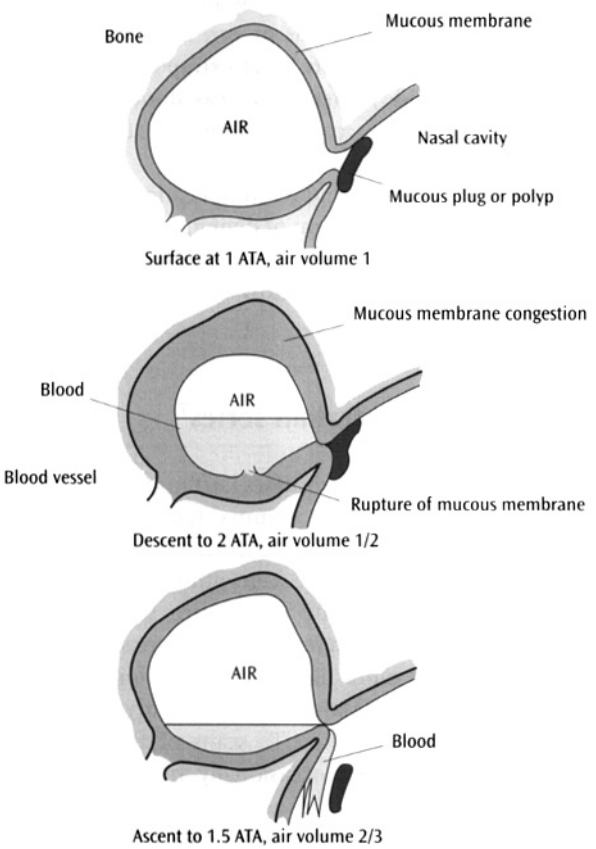

Figure 8.3 *Diagrammatic changes of sinus barotrauma of descent followed by ascent.*

Symptoms include pain over the sinus during descent, and this may be preceded by a sensation of tightness or pressure. The pain usually subsides with ascent, but may continue as a persistent dull ache for several hours. On ascent, blood or mucus may be extruded into the nose or pharynx.

Headache developing during the dive, with the diver neither ascending nor descending, should not exclude the diagnosis of sinus barotrauma. When this develops at considerable depth, the sedative effects of narcosis may distort the clinical features. Also, small changes of depth are not

particularly noticeable, but produce a misleading history.

The pain is usually over the frontal sinus, less frequently it is retro-orbital, and maxillary pain is not common but may be referred to a number of upper teeth on the same side. Although the teeth may feel hypersensitive, abnormal or loose, they are not painful on movement. Coughing, sneezing or holding the head down may aggravate the pain and make it throb. Numbness over the maxillary division of the 5th nerve is possible (see below). The superficial ethmoidal sinuses near the root of the nose occasionally rupture and cause a small haematoma or discolouration of the skin, between the eyes. Discomfort persisting after the dive may be due to fluid within the sinus (remaining from the dive), infection (usually starts a few hours post-dive) or the development of chronic sinusitis or mucocoeles.

Sinus X-ray examination, CT or MRI scanning may disclose thickened mucosa, opacity or fluid levels. The opacities produced by the barotrauma may be serous or mucous cysts, the maxillary and frontal sinuses being commonly involved, though the ethmoid and sphenoidal sinuses may also be affected. The new imaging techniques can clearly demonstrate these, and there is little difference in radiation exposure between the less precise sinus X-rays and the very discriminatory CT sinus scan.

Prevention is achieved by refraining from diving with upper respiratory tract infections, sinusitis or rhinitis. Cessation of smoking will reduce the likelihood of mucosal irritation and sinus barotrauma. Avoidance of allergens might assist in those so predisposed, as may treatment with local steroid nasal preparations. Correction of nasal abnormalities may be needed.

Positive-pressure techniques during descent, such as the Valsalva manoeuvre, assist in aeration of the sinuses as well as the middle ears (as opposed to the passive equalization methods). Feet-first descents are preferable (i.e. head upright); slow descents and ascents will reduce the sinus damage where there is only marginal patency of the sinus ostia.

Some physicians have found the use of proteolytic or allegedly mucus-softening enzymes to be of value (see Chapter 7).

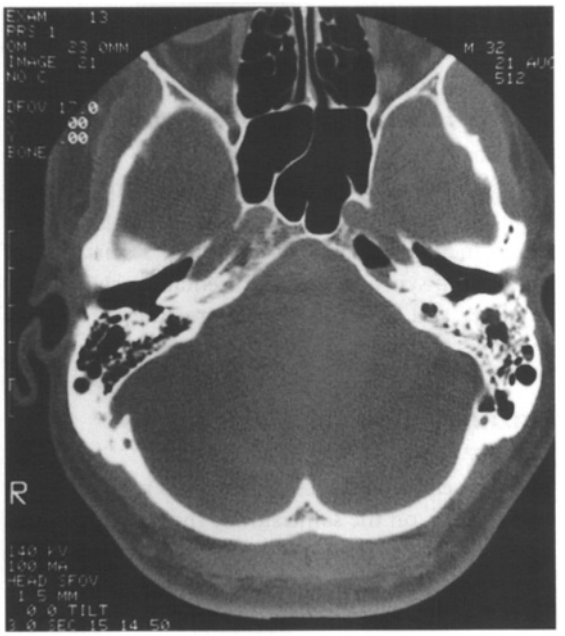

Figure 8.4 *Sinus barotrauma affecting the sphenoid and mastoid regions. Computed tomography (CT) scans have replaced traditional radiology in identification of problematic cases. The CT scan of the brain on bone setting through the skull base shows a fluid level within the antromedial air cell of the left petrous temporal bone line. In addition, there is loss of pneumatization of the left mastoid air cells.*

SINUS BAROTRAUMA OF ASCENT

This may follow the occlusion of sinus openings by mucosal congestion, folds or sinus polyps, preventing escape of expanding gases. The ostium or its mucosa will then blow out into the nasal cavity, with or without pain, and haemorrhage commonly follows. This disease is aggravated by rapid ascent, as in free ascent training, emergency ascents, submarine escape, etc.

If the expanding air cannot escape through the sinuses it may fracture the walls and track along the soft tissues, causing surgical emphysema. Rupture of air cells may cause a sharp and sudden pain of a severe degree, often affecting the ethmoidal or mastoid sinuses, on ascent. Occasionally, the air may rupture into the cranial cavity and cause a pneumocephalus.

OTHER MANIFESTATIONS

General symptoms

In a small number of the cases (8 per cent) some additional symptoms did not appear to be easily explicable on the basis of local sinus pathology. These included nausea or vomiting, a sensation of impending syncope, and disorientation at the time of injury, and occurred in the more dramatic cases of the sinus barotrauma.

Maxillary nerve involvement

In 4 per cent of the cases the pain was referred to the upper teeth, on the same side as the maxillary sinus affected. This is presumably an involvement of the posterior superior alveolar nerve. In another 4 per cent there was involvement of the infraorbital nerve, with numbness over the skin of the cheek on the same side.

Two separate branches of the maxillary division of trigeminal nerve can thus be affected[13,14] as they traverse the maxillary sinus: the infraorbital nerve, as it runs along the wall of the maxillary sinus; and the posterior superior alveolar nerve as it runs along the lateral or inferior wall of the maxillary sinus. The former produces a numbness or paraesthesia over the cheek; the latter a numbness over the upper teeth, gums and mucosa on the same side. In some cases pain and hypersensitivity are noted. Problems with neuropraxes are more common with ascent than descent, suggesting that impaired circulation is more frequent than congestion or haemorrhage of the nerve, as a basis of the pathology.

There is also a possibility of involvement of any division of the trigeminal nerve, including its maxillary division, with involvement of the sphenoidal sinus.[15]

Acute sinusitis

Campbell[1] stated that infection occurs only rarely, and his series may be equitable, in terms of selection, with our initial survey.[6] If, however, one considers our second survey, with its more serious cases, then the infection complications are more frequent.

Acute sinusitis developed some hours after the dive, and extended into subsequent days in 28 per cent of the severe cases – usually with an exacerbation of pain over the affected area. The cases of sinus barotrauma that subsequently developed a sinus infection, possibly did so because of the haemorrhage and effusion in the sinus. This becomes a culture medium for organisms introduced by the flow of air into the sinus during descent. An occasional case of orbital cellulitis may extend from the ethmoidal or maxillary sinusitis, and is a medical emergency.

It is for this reason that we now vigorously treat with antibiotics any persistence of symptoms following sinus barotrauma, i.e. commencing hours after the dive or continuing into the following day.

Chronic sinusitis

The criteria for this diagnosis included a continuation of sinus symptomatology in excess of one month. In 18 per cent of the severe cases there was a continuation of the initial barotrauma episode and acute sinusitis into a syndrome of chronic sinusitis. In another 14 per cent the chronic sinusitis was pre-existent, with recurrent barotraumas developing over it.

Pneumocephalus

The presence of pneumocephalus, in association with sinus injury in general medicine, has been well recorded by Markham,[17] and it is one of the dangers associated with sinus barotrauma.[5] It has been well demonstrated by Goldmann.[18] (see Fig. 9.4).

Surgical emphysema

This has been seen on a number of occasions and was described previously.[5] The tracking of air into the tissues can present as orbital surgical emphysema (usually from the ethmoidal sinus through a fracture of the eggshell-thin lamina papyracea). In other instances the air has passed from other sinuses, and can present as a localized manifestation in the facial tissues.

DIFFERENTIAL DIAGNOSIS

In 6 per cent of the 'serious' cases an initial diagnosis of **decompression sickness** was made, with the case subsequently demonstrated to be sinus barotrauma, often with complicating sinus infection. At the time of presentation – which could be some hours after the dive – the clinical pattern was confused with cerebral decompression sickness, and treated as such. These were understandable mistakes, and there should be no hesitation in administering hyperbaric therapy if there is any doubt regarding the diagnosis.

It would be preferable to miss and mistreat a case of sinus barotrauma than miss and mistreat a case of cerebral decompression sickness.

The only other case of incorrect diagnosis was one subsequently attributed to a **dental** aetiology (barotrauma associated with pneumatization around a carious tooth), and this case was therefore not included in the series.

Lew and his colleagues[15] not only referred to the symptomatology of **sphenoidal sinusitis**, but also referred to its association with 'deep sea diving'. Sphenoidal sinus involvement occurred in 6 per cent of the 'serious' cases. It is important because of the tendency of clinicians to not recognize it and to not appreciate its potentially serious complications. Another case, which is well known to the author but was not part of this series, sustained sphenoidal sinus barotrauma and caused considerable concern because of the proximity of the space-occupying lesion to other important structures in this sinus, and the possibility of the lesion being neoplastic. Although operative intervention was contemplated in that case, the lesion (a mucocele or haematoma) cleared up within a few weeks, following abstinence from diving exposure. Sphenoidal sinusitis is not easy to demonstrate with plain X-rays, but is often obvious with tomography or CT scans.

TREATMENT

Most of the effects of sinus barotrauma rapidly regress if diving is suspended and the underlying or consequential inflammatory pathology of the sinus is treated. Patients with a sinus or upper respiratory tract infection may require antibiotics and decongestants. Surgical drainage is rarely indicated. Even the mucoceles and chronic sinus pathology usually resolve without intervention, if diving is suspended.

In the 'serious' cases (second Australian series) the treatment could be divided into groups:

CASE REPORT 8.1

DN, a 22-year-old sports diver, occasionally noticed a trace of blood from his facemask following ascent. He had often complained of nasal blockage and had various treatments for this, including cautery. His first dive to 12 metres for 10 minutes was uneventful. After a brief surface interval he again descended, but was unable to proceed beyond 6 metres due to a severe tearing headache in the frontal region. He equalized his facemask, and this provided some relief. He then continued the descent feet first but still had some slight pain. On reaching the bottom, the severe sharp pain recurred. During ascent it lessened in severity, but on reaching the surface he noted mucous and blood in his facemask. A dull frontal headache persisted for 3 hours after the dive. Examination revealed a deviated nasal septum to both right and left, with hyperaemic nasal mucosa. X-ray showed gross mucosal thickening in both maxillary sinuses, the right being completely opaque. There was also some slight shadowing on the right frontal sinus. The radiological signs cleared over the next two weeks. As the airways were patent on both sides of the nasal septum, operative intervention was not indicated. The patient's nasal mucosa returned to normal after he abstained from cigarette smoking.

Diagnosis: sinus barotrauma of descent.

1 Those that resolved spontaneously; the patients were advised to not dive until this had happened.
2 Those divers who were using inappropriate diving techniques; these have been described previously and usually responded to appropriate regimes of:
   - feet-first descent;
   - positive-pressure manoeuvres to autoinflate both middle ears and sinuses, on the surface (immediately prior to descent) and then at regular intervals of 0.5–1 metre or so during descent;
   - avoidance of diving exposure with respiratory tract inflammation.
3 Those who responded to medical treatment of the nasal pathologies. This included the topical use of steroid nasal sprays, cromoglycate, topical or generalized decongestants, avoidance of nasal irritants and allergens, and cessation of smoking (tobacco or marijuana).
4 Infective sinusitis requiring treatment of the infections, usually by decongestants and antibiotics.
5 The intractable group required sinus exploration, usually with endoscopy and reconstruction, or nasal surgery. In some cases the surgery was required to produce a patency of the ostia and to remove polyps or redundant mucosa that caused obstruction to the ostia. At other times it was needed to improve nasal air flow. Reference in the literature has been made by Bolger *et al.*[20] in 1990 to the value of surgery in aviators with sinus barotrauma. Their guarded enthusiasm for functional endoscopic sinus surgery is tempered by the possible complications of this procedure. Nevertheless, endoscopic sinus surgery is advancing rapidly, and may offer value to the more serious and chronic cases. With current endoscopic surgical procedures,[21] the maxillary, ethmoid and sphenoid sinuses can be treated so as to widen the sinus ostia, preventing sinus barotrauma. It is considered the treatment of choice in military aviators in the USA. The frontal sinus is less amenable, but may be explored in some cases.
6 The sixth group continued to have difficulties, and usually gave up diving. All were strongly advised to not dive during times of upper respiratory tract inflammation (infections, allergic or vasomotor rhinitis, etc.). As with the original series, over 50 per cent of the divers had a history of diving with such conditions at the time of the barotrauma.

Some clients were moved between treatments, as various measures failed to completely resolve or prevent problems. Our general impression is that approximately equal numbers fell into each 'treatment' group.

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9

Other barotrauma

CARL EDMONDS

DENTAL BAROTRAUMA

This has been called **aerodontalgia** when applied to altitude exposure. Gas spaces may exist in the roots of infected teeth, along dying nerves, in necrotic areas of the pulp, and alongside or associated with fillings which have undergone secondary erosion. The gas may enter around the edge of the filling, adjacent to the tooth or through micro fractures of the enamel and dentine.

Teeth with full cast crowns may be susceptible to air being forced into the cemented material between the crown and the tooth – especially if cemented with zinc phosphate cement or, to a less extent, glass ionomer cement. Micro leakage of gas is less evident with resin cement.

During descent, the contracting gas space is replaced with the soft tissue of the gum or with blood and effusion. Pain may prevent further descent. If, because of slowed descent, symptoms are not noticed, then gas expansion on ascent may be restricted by the blood in these spaces, resulting in distension and severe pain.

Because of the aetiologies described above, the barotrauma is often encountered in older divers, who sometimes experience dental barotrauma reliably at a constant depth – but often without the gas space able to be readily visualized on X-ray. Transillumination with a high-intensity light may reveal the micro fractures.

Another presentation of dental barotrauma occurs in cases involving a carious tooth with a cavity and very thin cementum. As pressure differences across the cementum develops, the tooth may cave in (implode) on descent, or explode on ascent, causing considerable pain. Fast rates of ascent or descent will tend to precipitate this. Pressure applied to individual teeth may cause pain and identify the affected tooth. Sensitivity to cold may also localize the tooth.

A third form of dental barotrauma involves the tracking of gas into tissues, through interruptions of the mucosa, e.g. diving after oral surgery, dental extractions or manipulations. Scuba regulators result in positive oral pressure, forcing gas into tissues.

Preventive measures include: biannual dental checks (including X-ray examinations if indicated),

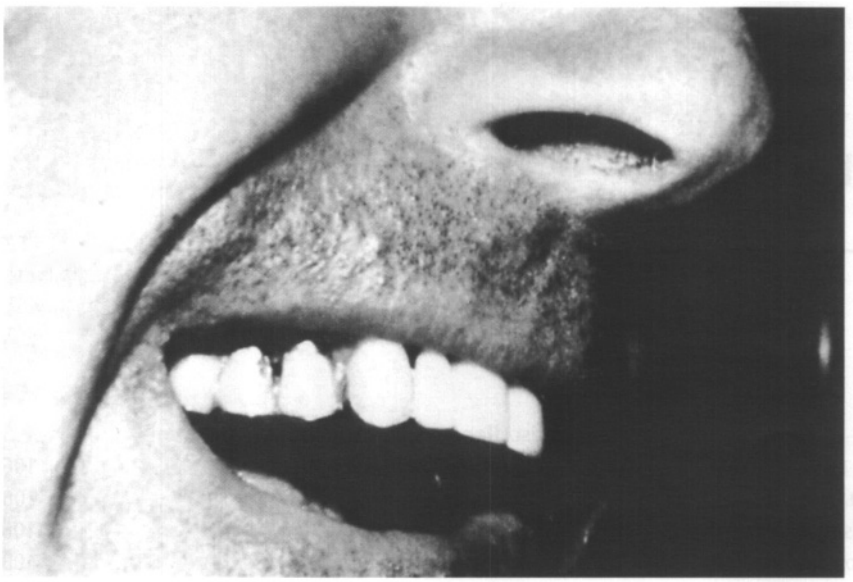


Figure 9.1 *Dental barotrauma showing collapse of the first right bicuspid during a dive to 20 metres. Previous dental treatment converted an open cavity into one covered by a silver amalgam filling.*

avoidance of all diving after dental extractions and surgery until complete tissue resolution has occurred (i.e. intact mucosal surface), slow descent and ascent.

Treatment consists of analgesia and dental repair. The differential diagnosis of sporadic or constant pain in the upper bicuspids or the first and second molars, but not localized in one tooth, must include other dental disorders (see Chapter 42) as well as referred pain from the maxillary sinus or the maxillary nerve (see Chapter 8). This may also present as a burning sensation along the mucobuccal fold.

EQUIPMENT BAROTRAUMA

Facial barotrauma of descent (mask squeeze)

A facemask creates an additional gas space external to, but in contact with, the face. Unless pressure is equalized by exhaling gas into the mask, facial tissues will be forced into this space, during descent.

Clinical features include puffy oedematous facial tissues, especially under the eyelids, purpuric haemorrhages, conjunctival haemorrhages, and later, generalized bruising of the skin underlying the mask (Fig. 9.2 and Plate 2). This condition is rarely serious, and prevention involves exhaling into the face mask during descent. Treatment involves avoidance of diving until all tissue damage is healed.

Skin barotrauma of descent (suit squeeze)

This condition is encountered mainly with dry suits or poorly fitting wet suits. During descent the air spaces are reduced in volume and trapped in folds in the suit. The skin tends to be sucked into these folds, leaving linear weal marks or bruises. The condition is usually painless and clears within a few days.

Figure 9.2 *Facial barotrauma of descent (central figure). This severe 'mask squeeze' developed with failure of the surface supply of compressed air to a full facemask (which did not have a non-return valve). Facial haemorrhage and swelling delineate the mask area.*

Head and body barotrauma of descent (diver's squeeze)

A rigid helmet, as used in standard diving, may permit this trauma. If extra gas is not added during descent to compensate for the effects of Boyle's law, the suit and occupant may be forced into the helmet, causing fractured clavicles, bizarre injuries or even death. The sequence of events may present dramatically if the heavily weighted diver falls off his stage. There is a similar result when the diver loses his compressed air pressure, e.g. due to a compressor or supply line failure. To prevent this, a non-return valve is inserted in the air supply line.

The clinical features include dyspnoea and a heavy sensation in the chest, bulging sensation in the head and eyes, swelling in the areas associated with rigid walls, e.g. the helmet, and then oedema and haemorrhages within the skin of the face, conjunctiva, neck and shoulders, bleeding from the lungs, gastrointestinal tract, nose, ears and sinuses. These pathological changes are due to the effects of barotrauma on the enclosed gas spaces, and to a pressure gradient forcing blood from the abdomen and lower extremities into the thorax, head and neck, because of the negative pressure differential in the helmet. Similarly induced haemorrhages occur in the brain, heart, respiratory mucosa and other soft tissues.

Suit barotrauma of ascent ('blow up')

During ascent in a standard diving ('hard hat') suit, the expanding gas must be able to escape. If it does not, then the whole suit will expand like a balloon and cause increased buoyancy and a rapid and uncontrolled ascent to the surface. This may result in barotrauma of ascent, decompression sickness, imprisonment of the diver and physical trauma.

With the decreasing use of standard diving this emergency is now not encountered very often, but a less impressive manifestation is possible with divers who use an inflatable object, such as a buoyancy vest, dry suit, etc. and inflate this excessively.

A clinically dissimilar and relatively minor symptom is noted by divers in an upright position using equipment which has a counterlung, or breathing bag, positioned below the head and neck. The pressure gradient from the bag to the diver's head results in a sensation of head and neck distension and bulging of the eyes.

GASTROINTESTINAL BAROTRAUMA

Gas expansion occurs within the intestines on ascent, and may result in eructation, vomiting, flatus, abdominal discomfort and colicky pains. It is rarely severe, but has been known to cause syncopal and shock-like states.

Inexperienced divers are more prone to aerophagia, predisposing to this condition. Swallowing to equalize middle-ear pressures is one cause of aerophagia. Performing Valsalva manoeuvres while in the head-down position may also result in air passing into the stomach. Carbonated beverages and heavy meals are best avoided before and during exposure to hyperbaric conditions.

Treatment involves either slowing the rate of ascent, stopping ascent, or even recompression. The simple procedure of releasing tight-fitting restrictions such as belts, girdles, etc. may give considerable symptomatic relief.

Although not common, notable examples of gastrointestinal barotrauma are recorded. Two Norwegian divers were badly affected during 122 metre diving using helium/oxygen, on *H.M.S. Reclaim* in 1961. An Australian lad, responding very well to hyperbaric oxygen therapy for gas gangrene, drank 'flat' lemonade at 2.5 ATA and deteriorated into a shock state with abdominal distension and pains before ascent was terminated. A group of officials celebrating the successful construction of a caisson in the UK experienced a similar embarrassing fate, from imbibing flat champagne.

Gastric rupture. Rarely, with a large and rapid expansion of gas in the stomach, this organ may rupture with ascent. A review of 12 cases were associated with relatively deep dives, over 30 metres, with rapid ascents. The abdominal pain and distension were constant, with various other symptoms including vomiting (25 per cent), belching (16 per cent), haematemesis (33 per cent) and dyspnoea (50 per cent). Guarding of the abdomen, and shock developed occasionally.

On X-ray, pneumoperitoneum was present in all cases, but sometimes this extended to include a

pneumomediastinum and even pneumothorax. These radiological abnormalities can, of course, be also produced by pulmonary barotrauma. Gastroscopy allowed identification and localization of the lesions, and laparoscopy usually showed these to be full thickness, usually on the lesser curvature of the stomach.

Treatment of rupture of the stomach is essentially a surgical procedure; however, breathing 100 per cent oxygen as a first aid measure and even hyperbaric oxygen as an initial treatment, may have some value under some circumstances. As a general rule, decompression of the pneumoperitoneum is best achieved using surgical techniques.

If there has been a full-thickness tear, then gastric contents are likely to be present in the peritoneal cavity, and the treatment must then be on general medical and surgical grounds. Previous work revealed that 4 litres or more of gas were necessary before rupture of the stomach would occur, and usually required a pressure of 96–155 mmHg. The reason given for the localization to the lesser curvature, is that there the gastric wall is composed of only one muscular layer, compared to the three elsewhere. It is postulated that rapid distention of the stomach will increase the angle of His and compress the cardia against the right diaphragmatic pillar, making the oesophageal gastric junction act like a one-way valve, preventing eructation.

MISCELLANEOUS BAROTRAUMA

Localized surgical emphysema

This may result from the entry of gas into any area where the integument, skin or mucosa is broken and in contact with a gas space. Although the classical site involves the supraclavicular areas in association with tracking mediastinal emphysema from pulmonary barotrauma, other sites are possible.

Orbital surgical emphysema, severe enough to completely occlude the palpebral fissure, may result from diving with facial skin, intranasal or sinus injuries. The most common cause is a fracture of the nasoethmoid bones. The lamina papyracea, which separates the nasal cavity and the orbit, is of egg-shell thickness. Thus, when these bones are fractured, any increase in pressure in the nasal cavity or ethmoidal sinus from ascent or Valsalva manoeuvre, may force air into the orbit (Plate 3).

Surgical emphysema over the mandibular area is common with buccal and dental lesions. The surgical emphysema, with its associated physical sign of crepitus can be verified radiologically, as it tracks into loose subcutaneous tissue.

Treatment is by administration of 100 per cent oxygen with a non-pressurized technique, and complete resolution will occur within hours. Otherwise, resolution may take a week or more. Recompression is rarely indicated, but diving should be avoided until this resolution is complete and the damaged integument has completely healed.

Pneumoperitoneum

This has been observed following emergency ascent, with movement of air from a ruptured pulmonary bulla, dissecting along the mediastinum to the retroperitoneal area, and then released into the peritoneum, to track under the diaphragm. It is also possible that previous injury to the lung or diaphragm, producing adhesions, could permit the direct passage of air from the lung to the subdiaphragmatic area. Another possible cause of pneumoperitoneum is, as described above, from a rupture of a gastrointestinal viscus – especially with barotrauma of ascent or underwater explosions.

The condition may be detected by chest X-ray or positional abdominal X-ray (gas under the diaphragm).

Treatment is by administration of 100 per cent oxygen with a non-pressurized technique, whereupon complete resolution will usually occur within hours. Management of the cause (pulmonary or gastrointestinal) is required, and surgical management of a ruptured gastrointestinal viscus may be needed.

Pneumocephalus

Occasionally the cranial gas spaces (mastoid, paranasal sinuses) are affected by an ascent barotrauma, when the expanding gas ruptures into the cranial cavity. This may follow descent barotrauma, when haemorrhage occupies the gas space and its orifice is blocked. The sudden bursting of gas into

the cranial cavity could cause significant brain damage.

The clinical presentation may have all the features of a catastrophic intracerebral event, such as a subarachnoid haemorrhage. Excruciating headache immediately on ascent is probable, although the effects of a space-occupying lesion may supervene. Neurological signs may follow brain injury or cranial nerve lesions.

It is likely that the condition could be aggravated by excessive Valsalva manoeuvres ('equalizing the ears') or ascent to altitude (air travel). Diagnosis can be verified by positional skull X-ray, or computed tomography (CT) scanning (Fig. 9.4).

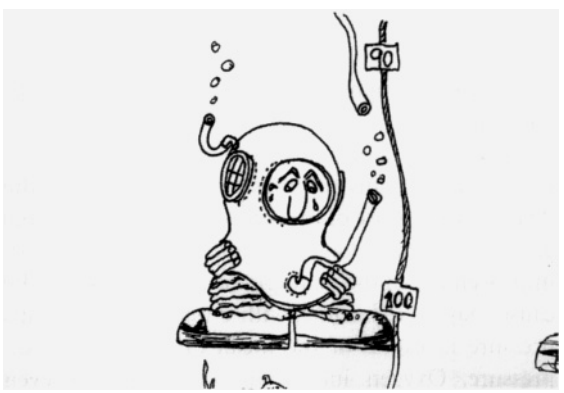
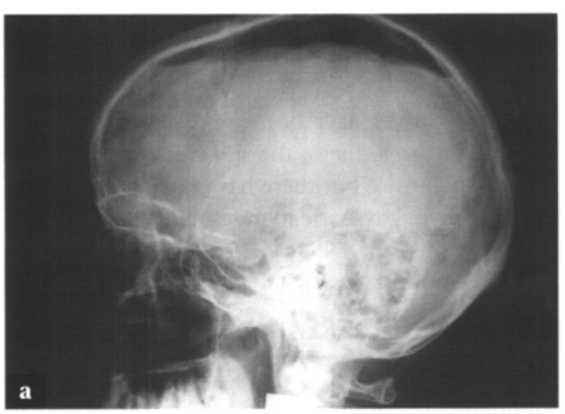
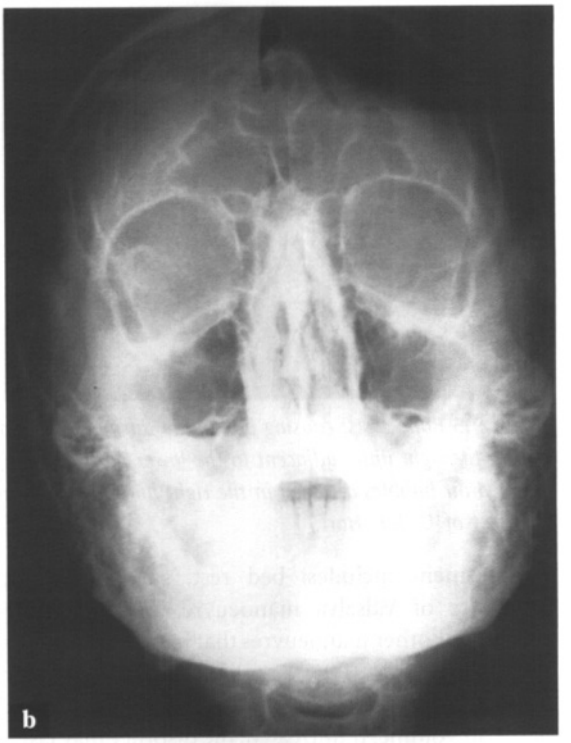


Figure 9.3 *Total body 'squeeze' (barotrauma of descent).*





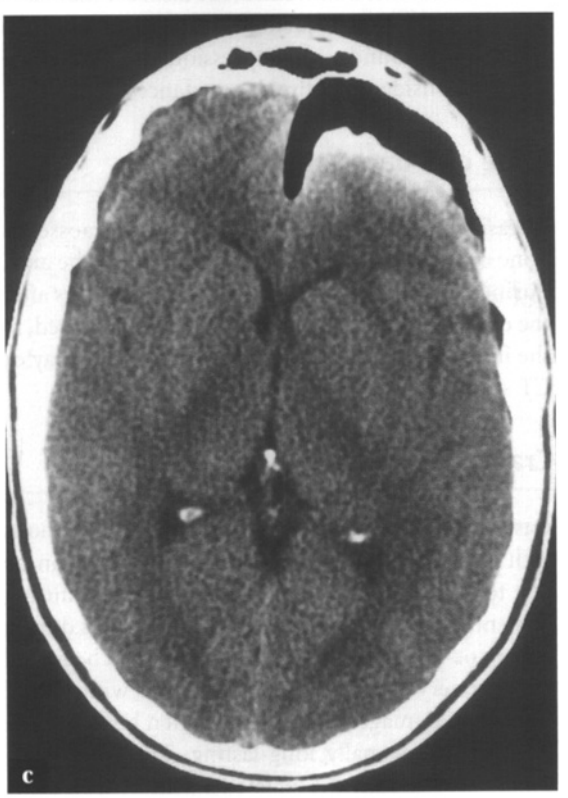


Figure 9.4 *Pneumocephalus from sinus barotrauma: the films include (a) a lateral view, (b) a frontal view and (c) a computed tomography (CT) scan. (c) There is a pneumocephalus which is loculated on the left and has some 'mass effect' causing depression of the underlying brain, on the CT scan. (Courtesy of Dr R.W. Goldman.)*

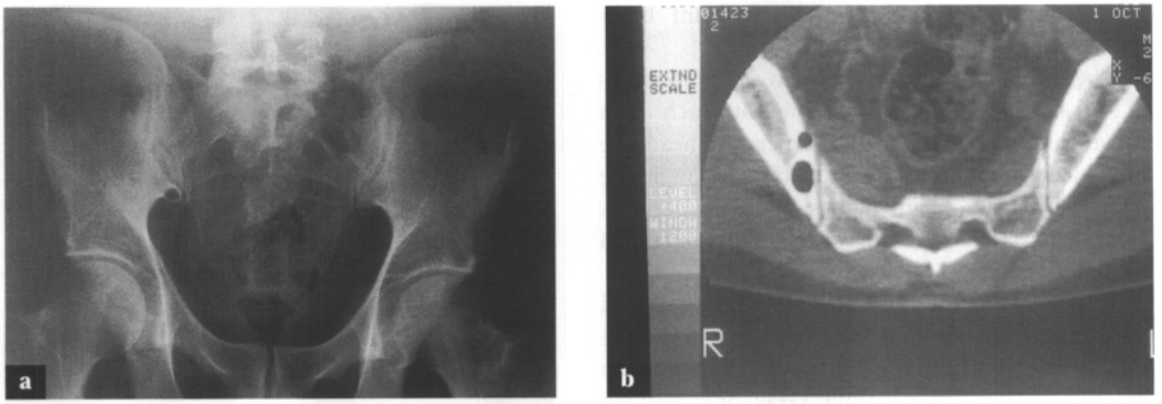


Figure 9.5 *Bone cysts: causing bone pain during descent and/or ascent. (a) X-ray of pelvis: a rounded translucency is seen in the right ilium adjacent to the lower part of the right sacroiliac joint. (b) Computed tomography scan: two rounded air bubbles are seen in the right ilium. One lies anteriorly in the cortex, and the other lies within the medulla. (Courtesy of Dr B.L. Hart.)*

Treatment includes: bed rest, sitting upright; avoidance of Valsalva manoeuvre, sneezing, nose blowing or other manoeuvres that increase nasopharyngeal pressures; 100 per cent oxygen inhalation for many hours and follow up X-rays to show a reduction of the air volume. If untreated, the disorder may last a week or so, and subsequent infection is possible. On theoretical grounds, recompression or craniotomy could be considered in dire circumstances.

Bone cyst barotrauma

Occasionally pain may develop from an intraosseous bone cyst, probably with haemorrhage into the area, during descent or ascent, and may last for hours after the dive. The pelvic bones are most often involved, in the ileum and near the sacroiliac joints. An X-ray or CT scan may demonstrate the lesions.

Cranial nerve palsies

Cases occasionally present with cranial nerve lesions (5th or 7th) attributed to neurapraxis. This can be due to the implosive tissue damaging effects during descent, the distension in enclosed gas spaces during ascent, or both. It is possible that air could be forced into the nerve canals as the gas expands with ascent. The nerve damage varies greatly, often being transitory but occasionally long-lasting. These presentations are usually associated with barotrauma symptoms and signs, as described earlier (see Chapters 7 and 8).

With the cases produced due to ascent, there may be a delay of many minutes after the dive, and the diver may be aware of the feeling of distension of the gas space. The relief as gas escapes may coincide with improvement in the neurapraxis. This infers that the cause may be ischaemic, with a middle ear or sinus pressure in excess of the mean capillary perfusion pressure. Oxygen inhalation may assist, or even recompression may be required.

Other barotrauma

Other gas spaces have been observed in the body, such as in the kidneys, the intervertebral disc and nucleus pulposus, but these have not yet been identified as having dysbaric manifestations.

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10

Decompression sickness: history and physiology

ROBYN WALKER

INTRODUCTION

Bubbles may develop in the body of the diver, aviator or caisson worker, whenever he ascends and is exposed to a reduction in environmental pressure (decompression), causing symptoms of decompression sickness (DCS). These bubbles may affect the neurological system, the musculoskeletal system, the skin, the lymphatics, the inner ear and others. Although DCS was first described in 1670, much argument still exists over why, where and how these bubbles form.

An appreciation of the history and the physiology of decompression is needed to understand this disease.

HISTORICAL ASPECTS OF DECOMPRESSION

Von Guericke developed the first effective air pump in 1650, and this permitted both pressurization and depressurization. Robert Boyle exposed experimental animals to the effects of increased and decreased pressures and, in 1670, reported these experiments. This included the first description of DCS – a bubble moving to and fro in the waterish humour of the eye of a viper. The snake was 'tortured furiously' by the formation of bubbles in the 'blood, juices and soft parts of the body'.

In the 1840s, Colonel **Pasley** noted rheumatism and over-fatigue in divers employed on the wreck of the *Royal George*. These divers were presumably suffering from DCS – a not surprising situation as their bottom times exceeded the accepted limits by a factor of three!

Triger later described cases of DCS in humans in 1841. He designed and constructed a caisson, intending to sink pylons in wet soil for the construction of a bridge across the Loire River near Chalonnes, France. Two labourers experienced severe pains after emerging from a 7-hour exposure to pressure, but relief was obtained by rubbing the affected area with alcohol – a treatment that with some variations in the route of administration has become a tradition among divers. Pol and Watelle, in 1854, published a report indicating the nature of the disease, together with case histories to demonstrate the relationship between pressure, duration of exposure, rapidity of decompression and the development of DCS.

Hoppe-Seyler repeated the Boyle experiments and, in 1857, he described the obstruction of pulmonary vessels by bubbles and the inability of the heart to function adequately under those conditions. He suggested that some of the cases of sudden death in compressed-air workers were due to this intravascular liberation of gas. He also recommended recompression to remedy this.

Le Roy de Mericourt, in 1869, and **Gal**, in 1872, described an occupational illness of sponge divers, attributed to the breathing of compressed air, and equated this with the caisson workers' disease. A host of imaginative theories were proposed during the nineteenth century to explain the aetiology of this disorder.

In 1872, **Freidburg** reviewed the development of compressed-air work and collected descriptions of symptoms of workers given insufficient decompression after exposure to high pressure. He compared the clinical course of severe and fatal cases of DCS to that of the venous air embolism occasionally seen in obstetrics and surgery. He felt that rapid decompression would be responsible for a rapid release of the gas that had been taken up by the tissues under increased pressure. He suggested that the blood was filled with gas bubbles which interfered with circulation in the heart and lungs.

In 1873, **Smith** described 'caisson disease' or 'compressed air illness' as a disease depending upon increased atmospheric pressure, but always developing after reduction of the pressure. It was characterized, he noted, by moderate or severe pain in one or more of the extremities, and sometimes also in the trunk. There may or may not be epigastric pain and vomiting. In some cases, there may be elements of paralysis which, when they appear, are most frequently confined to the lower half of the body. Cerebral symptoms, such as headache, vertigo, convulsions and loss of consciousness may also be present.

Paul Bert, in 1878, demonstrated in a most conclusive manner that DCS is primarily the result of inert gas bubbles (nitrogen in the case of compressed-air divers and caisson workers) which had been dissolved according to Dalton's and Henry's laws, and then released into the gas phase in tissues and blood during or following decompression. He used various oxygen concentrations to hasten decompression, demonstrated the value of oxygen inhalation once the animal developed DCS, and proposed the concept of oxygen recompression therapy.

In 1894, **Andrew Smith**, a surgeon from the Manhattan Eye and Ear Hospital, noted the origin of the term 'bends'. Since pain in the hips and lower extremities was generally aggravated by an erect position, the victims often assumed a stooping posture. Sufferers among the workers on the Brooklyn Bridge caissons in New York were the objects of good-natured ridicule by their comrades, who likened their angular postures to a fashionable stoop in walking, termed the 'Grecian bend', which was practiced by sophisticated metropolitan women at the time. He was aware of the value of recompression, but this was unacceptable to some of his patients. Instead, he used hot poultices, ice packs, hot baths, ergot, atropine, whiskey and ginger – or morphine if the others failed. Moreover, he constructed the first specialized treatment chamber.

Zuntz in 1897 discussed the factors which control bubble size and bubble resorption:

> 'The nitrogen bubbles circulating in a blood grow in size as a result of nitrogen diffusion from tissue fluids. Once formed, a gas bubble will diminish in size only gradually because the tension of nitrogen in the bubble is only slightly higher than alveolar nitrogen tensions. As circulation stops, the resorption of bubbles can only be effected by very slow diffusion through bloodless tissue layers to the nearest free blood vessels. It is no wonder that under such circumstances, Paul Bert as late as the fourth day after decompression still found bubbles in the blood vessels of the spinal cord centres'.

In 1896, **Moir**, while working on the Hudson River caisson tunnel, reduced the DCS death rate from 25 per cent of the work force to under 2 per cent by the use of recompression therapy. In addition, **Snell**, in 1897, was one of the first investigators to observe that the risk of DCS was increased when, due to faulty ventilation in caissons, the concentration of carbon dioxide in the atmosphere was increased. The two-volume monograph published in 1900 by **von Schrotter** and his colleagues is one of the most comprehensive and detailed reports published on the histopathology of DCS.

During the early part of the twentieth century there was considerable controversy regarding the speed and manner in which divers and caisson workers should be decompressed. An English physiologist, **John Scott Haldane**, proposed his critical supersaturation ratio hypothesis, and most of the current decompression tables have their basis in his work. Boycott, Damant and Haldane (1908) submitted a Report to the Admiralty of the Deep-Water Diving Committee and set down the knowledge and hypotheses on which their decompression table calculations were based.

TISSUE GASES AND BUBBLES

Haldane hypotheses

Haldane exposed goats to 2.36 ATA for up to 2 hours before rapidly decompressing them to the surface (1 ATA). With this pressure drop (1.36 ATA), a few of the animals just started to develop bends. However, the same pressure drop from 6 ATA did not produce bends – in fact, the animals did not develop bends despite a drop of 3 ATA! Thus, the bends was not thought to be due to the constant pressure drop.

Haldane's first hypothesis, based on theoretical assumptions, was that the uptake and elimination of nitrogen followed an exponential curve, and he postulated that the body could be represented by the five separate tissues having theoretical half-times of 5, 10, 20, 40 and 75 minutes. Tissue half-times are explained thus: if a subject is exposed to a gas, then half its absorption in any specific tissue will occur in a specific time, e.g. in 5 minutes the T_5 tissue will absorb half the total volume of gas that it is able to absorb in unlimited time. Over the next time interval of 5 minutes, the T_5 tissue will again absorb half of the remaining gas potential that it needs to become saturated. In 10 minutes, the T_{10} will absorb half the total volume of gas that it is able to absorb, etc. Thus, in 20 minutes the T_5 tissue will have received one-half plus one-quarter plus one-eighth plus one-sixteenth, i.e. it will become 15/16 saturated with nitrogen. The T_{20} tissue will only be half-saturated in the same 20-minute period. It was presumed that the elimination of gas during decompression could be expressed by a similar, but opposite, exponential function.

Haldane's second hypothesis followed the observation that divers could tolerate a rapid decompression producing a supersaturation of gas within tissues, yet still not produce DCS (i.e. the tissue has more than enough gas in solution to saturate it). It was believed that the environmental pressure could be halved, with the gas tensions in tissues being as much as twice the environmental pressure, producing a supersaturation and yet not resulting in bubbles or DCS. Haldane believed that decompression could be performed from 2 atmospheres to 1 atmosphere, 4 atmospheres to 2 atmospheres, and 6 atmospheres to 3 atmospheres, i.e. a 2:1 ratio of maximum depth to the first decompression stop, in safety. Haldane then devised methods of ascent whereby the nitrogen pressures in each of the five hypothetical tissues never exceeded the environmental pressure by more than a 2:1 ratio – now known as a 'critical ratio' supersaturation hypothesis.

Haldane devised two commonly used tables. Table 1 involved decompressions of less than 30 minutes, and was later found to be far too conservative, compared to what could be achieved in practice. Table 2 involved longer decompressions, but an unacceptable number of bends. The tables involved a rapid ascent to the first one or two stops, followed by a slower, staged ascent back to the surface.

Neo-haldanian developments

The Haldane concept was carried to other countries, including France, Russia and the United States of America, where variations were made to the tables to make them safer.

The US Navy experience was that the 2:1 critical ratio hypothesis of Haldane was too conservative for dives where the fast tissue limits decompression (short dives), and not conservative enough for dives limited by the slow tissues (long-duration dives). Changes were then made to the allowable critical ratio, such that divers could come up from short dives at considerable depths to a much more shallow first stop, thus increasing the nitrogen gradient which thereby increased elimination of the nitrogen from body tissues.

Yarbrough found that the critical supersaturation ratio seemed to vary not only with each hypothetical tissue, but also with the duration of exposure, and changed the tables used by the US Navy. Des Granges, Dwyer and Workman showed that the ratios also changed with depth – so that a simple 2:1 ratio developed into a series of tables of '**M values**', produced by **Workman**, showing the maximal allowable supersaturation for each hypothetical tissue at each depth. The result was a valiant and valuable attempt by the neo-haldanian workers to fit the physiology of diving to the Haldane model. Incorporating the work of Van Der Aue and including a 120-minute tissue resulted in the US Navy tables of 1956 which, with slight modifications, are used today.

However, a number of observations threw doubt on the adequacy of these revised tables. Slower tissues, such as muscles, tendons or joints, sometimes act paradoxically. Even if bubbles do not develop, uptake of nitrogen may continue in these tissues even after the diver has surfaced; the nitrogen coming from the faster tissues which had taken up more nitrogen (Fig. 10.1). This may happen at any depth after an ascent, with uptake of gas continuing into 'slower' surrounding tissues from the 'faster' ones.

Other observations also did not fit the classic haldanian theories. To overcome some of the problems that arose with the decompression tables, it was found necessary to increase the number of hypothetical tissues from the original five, some workers using three

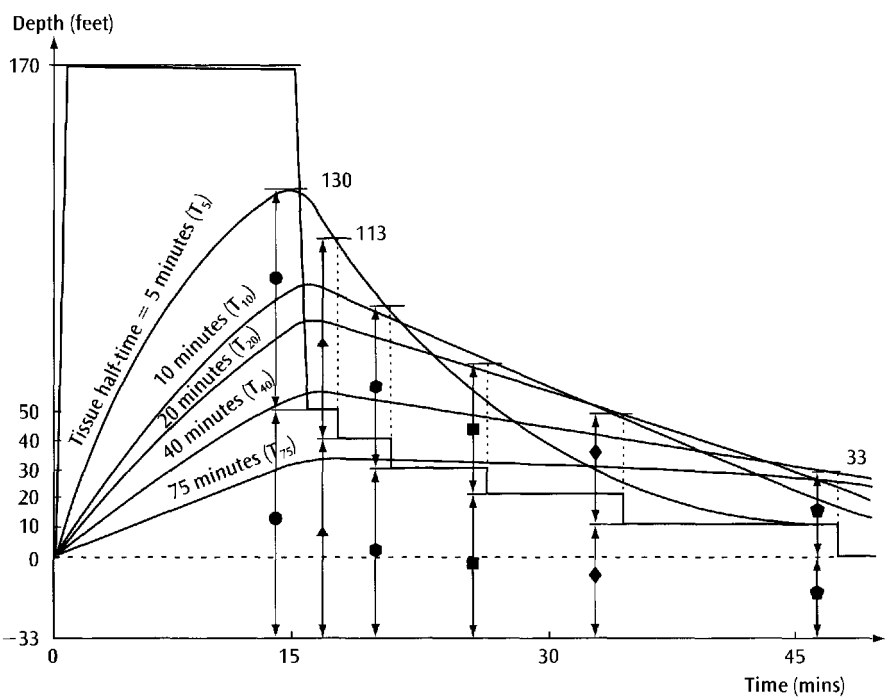

Figure 10.1 *Example of Haldane's decompression procedures for a dive to 168 feet for 15 minutes, based on five tissues: T_5, T_{10}, T_{20}, T_{40} and T_{75}. It presumes a critical supersaturation ratio of 2:1 to determine the duration of each decompression stop in 10-foot increments. The curved lines represent tissue nitrogen levels ('foots-worth').*

times this number. It was also necessary to consider tissues even slower than T_{75}, some having a half-time of up to 1000 minutes for extreme exposures.

Radioactive xenon studies suggest that gas takes much longer to leave tissues than predicted by the perfusion-limited models described above – and on which the conventional decompression tables are based.

Other hypotheses

Leonard **Hill**, in 1912, produced both experimental and theoretical evidence questioning the value of stage decompression over continuous uniform decompression. In the arguments between Haldane and Hill, the practical value of the Haldane tables in reducing decompression time made these more acceptable. The time at depth was less, and Hill could not adequately explain why Haldane's technique avoided DCS. Hill's technique is now applied to decompression for saturation exposures. Sir Leonard Hill's concept was that, to prevent bubble growth, decompression would depend on a maximum safe gradient between the tissue gas tension and the environmental or ambient pressure, ΔP rather than the ratio between these two. This is therefore a **critical pressure hypothesis**:

$$\Delta P = p - P$$

where p equals tissue gas pressure, P equals ambient or environmental pressure and ΔP equals the gradient moving gas from tissues to the environment.

The ΔP hypothesis suggests a linear, and not a staged, decompression. This concept is now used in long-exposure dives, and is also included in other theories of decompression. Albano of Italy suggested a minimum allowable pressure gradient across a bubble surface before it would enlarge, thereby applying this principle to bubbles as well as to gas in solution.

Three related concepts were proposed, which appear to have considerable importance in both decompression theory and DCS therapy. These were the **oxygen window** (Behnke), the **partial pressure vacancy** (Momsen) and the **inherent unsaturation hypothesis** (Le Messurier and Hills). Behnke describes it thus: 'During the course of blood transport through the capillaries, the oxygen is unloaded in different quantities to the various tissues. This results in available space for transfer of inert gas from tissues to lungs'. A similar but even greater oxygen utilization takes place in these tissues. It is this, the difference between the environmental pressure and the tissue gas tension, which allows divers to ascend a safe distance without the tissue gas tension exceeding that of the environment, i.e. without supersaturation and therefore without the likelihood of bubble formation.

During the early 1960s, two Australian workers, **Hugh Le Messurier** and **Brian Hills**, observed the type of diving performed by Okinawans who reached depths of up to 90 metres on air for as long as an hour, twice a day, six days a week. During the peak of the pearl diving industry, there were as many as 900 divers working out of the small coastal township of Broome, in Western Australia. These people had accumulated great experience, without preconceived scientific knowledge, and empirically produced a decompression regime, which cost thousands of lives during its development, but finally resulted in relatively safe decompression tables, which were very economical of time.

These decompression schedules were significant in that they required about two-thirds the time needed by the US Navy decompression tables. Also, when the US Navy tables caused DCS, the remedy used was to extend the shallow decompression. Hills suggested that the problem developed during the initial ascent, and therefore the added stops should be at greater depths. He also used deeper decompression stops than customary, but then surfaced directly from 7.5 to 9 metres. Even the relatively minor variation of transferring the conventional 3 metre stop to 6 metres reduced the DCS incidence by 40 per cent in a UK trial.

Hills, in 1966, developed his 'thermodynamic' model of DCS from these observations and introduced a most important concept of 'unsaturation' in the understanding of bubble prevention and resolution. Hills believed that the gas bubbles developed during decompression with the US Navy tables and that these merely controlled the size of the bubbles, similar to the view expounded by Behnke in 1951.

The concepts of unsaturation can be illustrated by considering representative values for the partial

pressures of respiratory gases for a person on the surface (see Fig. 10.3a and Table 10.1).

It will be noted that the total gas tension in tissues and venous blood is less than the barometric pressure; thus the tissues are unsaturated. Other workers suggest that tissue unsaturation is greater than the 60 mmHg (760 – 700) or 8 kPa indicated here. The importance of this tissue unsaturation in diving is that an instantaneous reduction of the total pressure caused by ascent equivalent to at least 60 mmHg must take place before the tissues become saturated with gas. After this ascent, the alveolar nitrogen partial pressure is less than the tissue nitrogen partial pressure, so a nitrogen pressure gradient is established. Elimination of nitrogen and the re-establishment of tissue unsaturation results. The clinical importance of the different tissue uptake speed is seen in two examples.

In astronaut exposures to 0.2 ATA, DCS can be prevented by denitrogenation of the body through the prior breathing of 100 per cent oxygen for 3 hours. If, however, the astronaut breathes air for 5 minutes after the denitrogenation, DCS will develop at altitude. The fast tissues are the only ones that could be involved in this sequence. Similarly, the occasional DCS from a brief exposure to 5 ATA by divers, with a sudden ascent to the surface, should not cause surprise.

Once the diver has reached the surface from a non-saturation dive, 'slow' tissues such as joint structures can continue to absorb nitrogen from the faster ones, until they equilibrate.

Table 10.1 *Partial pressures of respiratory gases at 1 ATA*

Sample	Gas partial pressure				
	O_2 (mmHg)	CO_2 (mmHg)	N_2 (mmHg)	H_2O (mmHg)	Total (mmHg)
Inspired air	158	0.3	596	5.7	760
Expired air	116	32	565	47	760
Alveolar air	100	40	573	47	760
Arterial blood	100	40	573	47	760
Venous blood	40	46	573	47	706
Tissues	≤30	≥50	573	47	700

In 1951, Harvey postulated the importance of bubble nuclei in the formation and development of bubbles in DCS. Hawaiian workers, including Kunkle and Yount, integrated this with the unsaturation hypothesis and extended the concept of bubble formation and growth. An oversimplified explanation is as follows: the pressure in a bubble (P_b) is due to the gas molecules it contains. If P_b is greater than the surrounding pressures, the bubble will grow. If P_b is less, it will be reduced in volume. The surrounding pressures are the environmental or ambient pressure (P_a), the tensions due to the tissue displacement (P_t) and the surface tension of the bubble (P_y). All these tend to constrict the bubble.

If $P_b > P_a + P_t + P_y$, the bubble will grow.

Bubble growth can follow decompression, when the pressure of the gases (especially nitrogen) in the bubble reflects the pressure at greater depth, and the P_a has been reduced by decompression, decreasing the right side of the equation and producing bubble growth. Localized falls in tissue pressure may be caused in areas of turbulence and during tissue movements.

The effect of surface tension will vary with the size of the bubble:

$$P_y = 2y/r$$

where P_y = pressure due to the surface tension, y = surface tension and r = radius of bubble.

For larger bubbles, P_y is reduced and may become negligible. For smaller bubbles, P_y is increased and may become so great as to force the bubble back into solution. It is difficult to understand how extremely small bubbles can persist, except in protected areas. Thus, tissue bubbles of less than 1 μm will probably disappear, whereas those greater than 1 mm may cause symptoms.

It is believed that for bubbles to form during or after decompression, 'nuclei' of gas need to be present for the other gas molecules to pass into and enlarge. Supersaturation as such will not produce bubbles in pure water (without nuclei) until this supersaturation is about 1000 atmospheres! The 'nuclei' or gas pockets occur in the natural state, and may be illustrated by the bubbles of carbonated beverages developing more frequently on the rough surface of the glass.

In Figure 10.2, the gas is trapped in a small crevice, producing a gas nucleus. The walls of this are hydro-

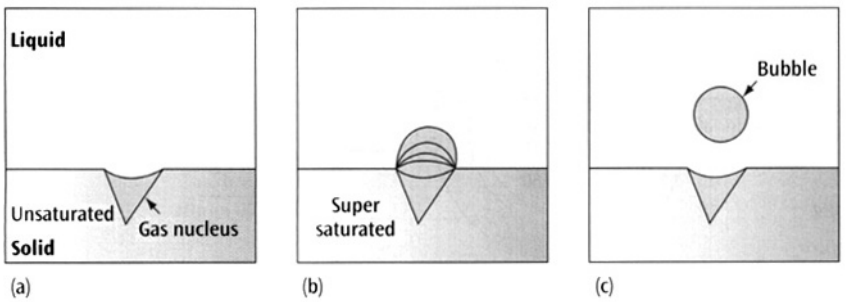

Figure 10.2 *Nucleation of gas emboli with decompression.*

phobic (non-wettable), thereby preventing water from running in and eliminating the gas cavity. The gas–liquid interface tends to flatten due to surface tension (which, in the case of a free bubble, would tend to collapse it). As the gas pressure in the bubble is in equilibrium with tissue gas tensions, it is probably slightly less than the hydrostatic pressure in the liquid, and therefore the gas interface will be concave.

When supersaturation of gas develops and gas molecules diffuse into the gas nucleus, it expands with its interface bulging into the fluid. Surface tension will tend to oppose the growth, but if a critical gas volume is reached a bubble will bud off the nucleus and be carried away in the liquid (e.g. in the blood). In the tissues, the gas bubble may remain stationary, but in either case the nucleus is left to generate more bubbles while the state of supersaturation exists.

Other ways of attracting gas molecules out of solution are by producing local areas of very low pressure. Such may be seen with 'tribonucleation' – the shearing of joint surfaces over each other, in tendons and in the turbulent and vortical motions in the circulation.

High pressures may reduce the size of gas nuclei to such an extent that water enters the crevice and the nucleus disappears. When the walls of the crevice become wet, the gas forms a bubble and the bubble's surface tension is great enough to compress it further and force it back into solution.

Exposure to pressure, for a time insufficient to produce DCS, may be adequate to remove some nuclei. This may account for the reduced incidence of DCS in divers and caisson workers after they have been exposed to repeated and regular diving. This is called 'acclimatization', and may be lost after a week of non-exposure. It may also explain the necessity to gradually 'build up' to deep diving exposures.

Studies with **gelatin**, initiated by Le Messurier in Australia and further developed by Strauss and Yount in Hawaii and Vann at Duke University, have clarified the behaviour of bubble nuclei when exposed to pressure changes. There is some agreement that the differences in pressure between the gas in the tissue and gas in the environment need to be kept small to prevent bubble production. This therefore supports the critical pressure ΔP hypothesis. The bubble-free profiles would then have a small first 'pull', to take up the unsaturation, and then be linear to ensure that the meniscus of the bubble nucleus never bulges sufficiently to cause a separated bubble.

Van Liew and others later demonstrated these concepts by experiments utilizing gas analyses in induced gas pockets. Figure 10.3 illustrates some of these changes, and is the basis of much of our recompression procedures. It demonstrates the gas gradients used to dissolve bubbles.

Nitrogen is lost much faster from solution in tissues, than from bubbles. Thus, if an air-breathing diver has been saturated at 2 ATA and then rapidly ascends to the surface (1 ATA), and if a bubble has not developed in tissue A, but has in tissue B, then the gradient from the tissue to the arterial blood is as shown in the box:

Tissue A:	*N_2 pressure*	*= 1179 mmHg*
Blood:	*N_2 pressure*	*= 573 mmHg*
Gradient:		*= 606 mmHg*
Tissue B:	*Bubble N_2 pressure*	*= 633 mmHg*
Blood:	*N_2 pressure*	*= 573 mmHg*
Gradient:		*= 60 mmHg*

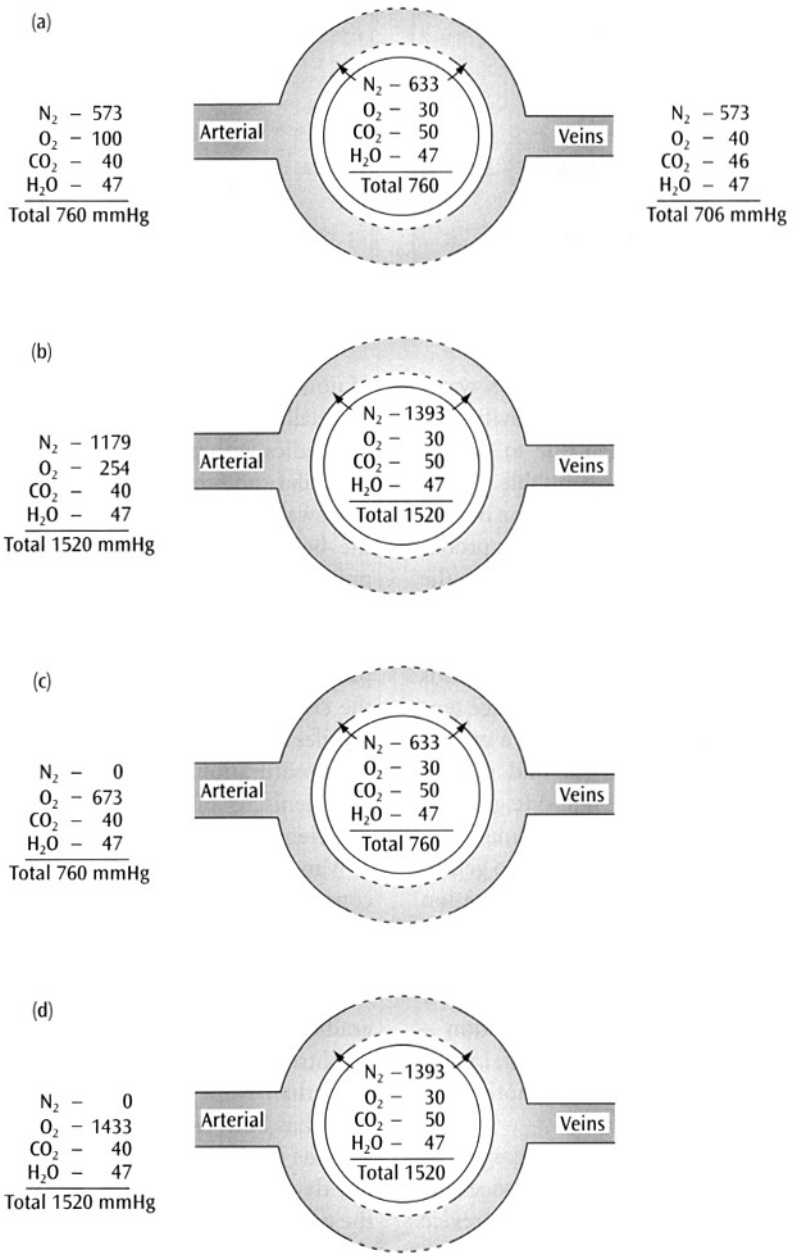

Figure 10.3 *Theoretical gas pressures in bubbles and blood, when the diver breathes air or oxygen at 1 ATA and 2 ATA. (a) N_2 gradient of 60 mmHg, breathing air at 1 ATA; (b) N_2 gradient of 214 mmHg, breathing air at 2 ATA; (c) N_2 gradient of 633 mmHg, breathing O_2 at 1 ATA; (d) N_2 gradient of 1393 mmHg, breathing O_2 at 2 ATA.*

Ignoring the other factors, such as distortion effects from the bubble, it is seen that the gradient to remove nitrogen from solution in the tissues is 10 times that from a tissue bubble, and therefore it will take much longer to get rid of the nitrogen in bubble form.

Even with identical pressure exposures, various tissues will have different factors determining bubble development, including vascularity of the tissue, solubility of the gas and its diffusion coefficient.

Recent work by Danish researchers on bubbles in adipose tissue showed that the breathing of Heliox after air dives resulted in rapid resolution of nitrogen bubbles from some tissues. The inflow of helium, because of its lower (1:4) solubility in adipose tissues, was less than the outflow of nitrogen. The bubbles, which grew while the animals were breathing air, were eliminated with either oxygen or Heliox breathing. They also cast doubt on the concept of gas nuclei persisting after the bubbles had shrunk to submicroscopic levels (10–20 μm), because they were unable to re-expand such bubbles with nitrous oxide breathing. This gas has a solubility of 30 and 45 times that of nitrogen and helium respectively, and should rapidly expand such bubbles.

Behnke had already realized the value of the unsaturation or **oxygen window** concepts by using oxygen inhalation to hasten decompression. He treated DCS with oxygen at 9 metres, and then proposed use of oxygen/nitrogen mixtures at greater depths. Donald brought the value of oxygen into question by a description of 'oxygen bends'. However, Donald was wrong as subsequent analysis by Brian Hills revealed that the animals so afflicted had large quantities of inert gas and that the contribution of oxygen to the disease was minimal. The next important development in comprehending the physiology of decompression was the appreciation of the importance of bubble production during apparently normal uneventful dives. Note was made in Haldane's original thesis that the body could withstand various pressure reductions, which would produce a specific volume of gas. However, his decompression schemes relied on the belief that gas phase separation (bubbles) did not occur with his staging techniques.

One of the main tenets of Haldane's concept was that gas elimination followed the same exponential manner as gas uptake. In 1942, Behnke first proposed the presence of 'silent' bubbles, while discussing DCS due to altitude exposure. Silent bubbles were so-called because they had no clinically obvious effects, and were not causally associated with DCS. If verified, the presence of these bubbles would not be consistent with the exponential gas elimination curve, because bubbles interfere with the movement of gas along pressure gradients in solution.

In 1960, **Hannes Keller** introduced his own approach to decompression theory, which he demonstrated by carrying out a dive to 210 metres in the lake at Zurich. A dive to 300 metres in the open ocean was not as successful, resulting in the death of his companion. Keller's dives were followed by his Swiss colleague, Buhlmann, exploring these new avenues.

In 1961, **Goodman** suggested that future developments in decompression should include the use of different gases during decompression staging, with each one being breathed in turn, until the zero decompression limit is reached for that pressure–time relationship. He also suggested exploration of the use of nitrogen/oxygen mixtures for the treatment of DCS – an idea that was independently exploited by both the French and Australian workers during the following decade.

Buhlmann pioneered the use of mixed-gas diving using various helium, nitrogen and oxygen mixtures in the belief that the gases would be taken up and released independently from solution. He developed curves relating the allowable safe supersaturation ratios for different half-time tissues at depth. The curves were interrelated in accordance with Graham's law (the diffusion coefficient is inversely proportional to the square-root of the molecular weight of the gas). Buhlmann used fast compression rates for his deep dives. He progressively changed from helium to nitrogen as the inert gas during a continuous ascent, but as he approached the surface he would then switch to the maximum tolerable oxygen pressure. He also supported the belief that if work was performed at depth, then decompression times had to be extended.

Idicula and Lambertsen first described **inert gas counterdiffusion** in 1973 in subjects who breathed one inert gas mixture while being surrounded by another. It was also termed 'isobaric counterdiffusion', deriving its name from the Greek prefix 'iso' (meaning the same) and 'baric' (referring to pressure). Isobaric counterdiffusion therefore means the diffusion of gases in different directions, while at a

fixed environmental pressure, i.e. without decompression. The production of bubble formation occurring without decompression introduced a new facet to DCS and its management. There are various forms of isobaric counterdiffusion.

Superficial isobaric counterdiffusion

This develops whenever the inert gas breathed diffuses more slowly than the inert gas surrounding the body. This could occur when the diver breathes air while surrounded by a helium/oxygen mixture. The nitrogen moves from the skin capillary blood through the skin tissue into the environment at a slower rate than the fast diffusing helium passes from the environment into the skin capillary blood. This inequality of diffusion leads to a supersaturation at certain sites within the superficial tissues and presumably also in the capillary blood. It was first noted at a stable depth of 370 metres in a chamber filled with normoxic helium and the diver breathing a neon/oxygen mixture. It has also been observed in a helium environment, when the subjects changed the inert gas being breathed from helium to hydrogen. The lesions produced by the developing bubbles produce an intense itching of the skin (Plate 4).

The vestibular system is prone to decompression disease at great depths. This is thought to be due to counterdiffusion between the middle-ear space and the fluids of the inner ear through the round window, although others have suggested that the continuous gas embolism, which accompanies superficial counterdiffusion, may be the cause of the vestibular disease.

Deep tissue isobaric counterdiffusion

With the entire body exposed to the same gas as that being breathed, superficial counterdiffusion cannot occur. Different rates of tissue uptake and elimination of the various gas mixtures breathed in sequence may possibly lead to tissue supersaturation or subsaturation in the isobaric state. After prolonged exposure to a nitrogen/oxygen mixture, breathing helium/oxygen results in more rapid entry of helium than loss of nitrogen from certain tissue sites. The sum of inert gas partial pressures therefore increases to above ambient, and supersaturation will exist.

Gas switching

DCS may be precipitated or worsened when a helium/oxygen breathing diver converts to air breathing. This is difficult to explain by counterdiffusion principles. It may be that air breathing interferes with pulmonary dynamics, because of the increased density, and that this may potentiate the DCS by reducing helium elimination. Some workers have found that in tissue, and specifically the central nervous system, oxygenation is less with the breathing of nitrogen/oxygen mixtures than with helium/oxygen mixtures at the same oxygen partial pressure.

INTRAVASCULAR BUBBLES

Behnke raised doubts regarding the haldanian hypotheses in 1951, when he conjectured 'it may well be that what appears to be a ratio of saturation tolerance is in reality an index of the degree of embolisation that the body can tolerate'.

Large quantities of air can sometimes be tolerated in the venous system, if infused slowly. Up to 1 litre has been suggested, without causing death. However, less than 1 ml can cause death if it enters certain areas of the arterial system, such as the coronary or cerebral arteries. The difference in volumes is explained by the very capable pulmonary filtration of the bubbles preventing them from entering the arterial circulation, unless right to-left shunts are present.

Because the obstruction of vessels by bubbles is dependent on the relative sizes of the bubbles and the vessels, and also on the blood pressure forcing the bubbles through, obstruction is much more likely in the pulmonary circulation than in the systemic arterial systems.

Venous gas emboli (VGE)

Behnke's 'silent' bubbles became very audible following the development and application of sophisticated ultrasonic techniques. Using these in 1963, Mackay detected bubbles in rats during decompression. Spencer and Campbell, in 1968 demonstrated gas emboli in decompressed sheep by the use of the

Doppler ultrasonic bubble detection system. They also demonstrated bubbles in humans exposed to certain US Navy decompression tables. Evans and Walder, working in the UK, and Powell and Kent Smith working independently in the USA, verified and then extended these concepts. The following is a resume of Doppler developments, and their relationship to DCS.

Bubbles can be detected once they reach a size of 40–50 μm (sometimes as small as 20 μm, especially if there are a lot of them); even single bubbles of 150 μm can be detected. The characteristic sound of a bubble signal is a 'chirp' or 'whistle', which can be confused with other sounds such as blood components, lipid and platelet emboli, superimposed on a background noise. The latter can be particularly troublesome, especially when the diver is active, shivering or cold. To obtain a good recording, an experienced observer is required. It is not a perfect instrument for use in the field and it is important to remember that it demonstrates bubbles, which are not synonymous with DCS.

Nevertheless, the Doppler technique is a valuable research technique and has increased our understanding of decompression and its sequelae. The use of bi-directional ultrasonic localization for the demonstration of extravascular bubbles complements the Doppler technique for the detection of intravascular bubbles, and provides a more complete picture of how the body handles bubble development and movement during and after decompression.

Although bubbles may appear in the arterial or capillary systems, most of the current measurements relate to their movement within the venous system. They are released freely into the veins, either due to their own growth or because of disruption by movement of the tissue or limb. The bubbles then pass through the right side of the heart, and in many cases become lodged in the pulmonary arterioles and capillaries. Up to 6 per cent of the absorbed gas may be eliminated by having the bubbles trapped in this pulmonary filtration system. Most of the eliminated gas still passes in solution from the tissues to the pulmonary blood and then diffuses into the alveolar air.

For short deep air dives of 30–60 metres, VGE can be detected within a couple of minutes of surfacing, or while performing decompression stops. The number of bubbles heard reaches a peak within the first hour and then gradually diminishes over the next few hours. The specific time intervals will vary with different dive profiles, diver activity, etc.

If there is an excess number of bubbles in veins, the pulmonary filtration system may become overloaded. Pulmonary hypertension then develops and, if this exceeds a rise of 120 per cent, then the bubbles will tend to pass through the pulmonary system into the systemic system. Another effect of pulmonary hypertension is to open potential shunts in the atrial or ventricular septa, which may also allow paradoxical emboli into the arterial system. Clinically, when large quantities of gas emboli pass into the pulmonary filtering system and overload it, the syndrome of 'chokes' with right ventricular failure and circulatory collapse may develop. This is more likely when the pulmonary hypertension exceeds a rise of 150 per cent. Spinal cord involvement is also associated with greater pulmonary gas loads. A large volume of gas in the right side of the heart may interfere with cardiac contraction and form an air lock.

Venous bubbles may influence specific organs in different manners (Fig. 10.4). Spinal cord paraplegia may result from venous stasis produced by a gross accumulation of venous bubbles in the spinal venous plexus. Bubbles in the systemic arterial system may produce local brain damage, although there is some degree of tolerance to gas emboli within the cerebral system. Cardiac action may also allow some bubbles to pass through the coronary arteries, without consequence. It may be that the high flow rate and the squeezing action of the heart acting on its own blood vessels may protect the heart from the effects of some of these emboli.

Gas emboli have been demonstrated under many practical diving conditions. They were initially noted following the US Navy Tables of Exceptional Exposure, but have also been observed among sport divers performing no-decompression dives from the US Navy Standard Air Decompression Tables. Bubbles developed in slow decompressions from saturation dives, although they tended to be feeble and fairly constant – unlike those described during the dives that are more likely to produce serious decompression cases. They were also noted in breath-hold dives performed by the Japanese Ama divers after 30 successive open ocean dives to 15 metres, with a total duration of each dive averaging just over 65 seconds.

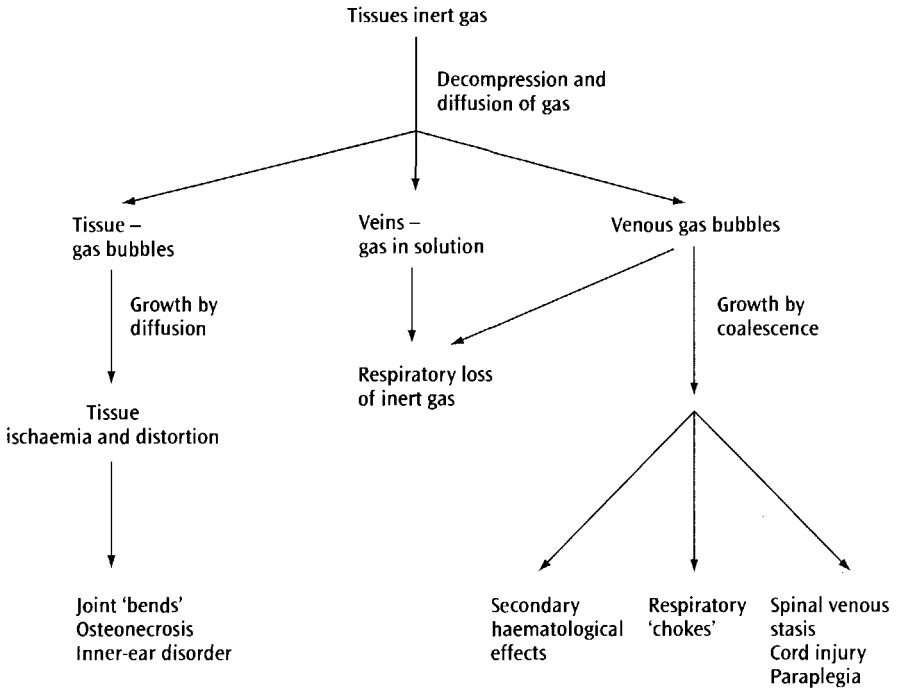

Figure 10.4 *Decompression sickness sequence.*

Bubbles have been observed, as has DCS, from saturation dives to 9 metres and 7.5 metres. The current depth for unlimited exposure is about 7 metres, and most experienced diving physicians are aware of cases of DCS after diving at such depths – usually with aggravating factors such as very long exposure, extreme exertion, etc.

Provocation of bubble formation and movement has been demonstrated by Doppler techniques in certain other environmental situations. This was observed under isobaric conditions (without any change of pressure) when animals were switched from air to helium/oxygen breathing at a depth of 40 metres. Numbers of emboli may be increased by limb manipulation, promoting the dislodging of gas bubbles held in the peripheral veins of that limb.

When a dive pattern is interrupted by a brief ascent to the surface and then a return to the bottom, the overall volume of nitrogen absorbed by the diver should be less than if he had remained at depth throughout. Despite this, there are a greater number of gas emboli with the interrupted dive profile. The second decompression will produce more bubbles than would otherwise have been expected to occur had there been no intervening period at normal pressures!

In this bimodal profile, bubbles may be produced during either or both decompressions, and the bubbles produced by the first ascent, and trapped in the pulmonary circuit, may be compressed and therefore pass through the pulmonary bed into the systemic arterial system during the second compression. They may then expand in the tissue vessels during the second ascent – producing rapidly developing DCS.

There is a considerable individual variation in the likelihood of producing both gas emboli and DCS, but these two results are generally quantitatively related. Occasionally, bends or DCS will develop without VGE being present, especially with saturation exposure or 'slow tissue' DCS.

Under good conditions, it is possible to use the presence of venous emboli as a practical guide to prevent DCS, avoid provocative conditions and indicate the need for specific preventive and therapeutic measures, which include the use of oxygen and

recompression. Gas emboli, without DCS, were removed by breathing 100 per cent oxygen at 1 ATA after some dives (Spencer *et al.*, 1976). Recompression to 9 metres on 100 per cent oxygen cleared both gas emboli and DCS produced from deep exposures. Others showed the value of inwater oxygen decompression to reduce both VGE and DCS.

There is a much higher incidence of VGE than there is of DCS. The statistical relationship between the two is, nevertheless, strong. A dive schedule, which produces bubbles in 20 per cent of divers, may have a 5 per cent incidence of DCS.

There are two bubble classification and scoring systems commonly in use: the methods described by Spencer, and by Kisman–Masurel.

The Spencer grading system

This monitors the precordium for bubbles with the subject sitting quietly:

Grade 0	No bubble signals on Doppler.
Grade I	An occasional bubble, but with the great majority of cardiac periods free.
Grade 2	With many, but less than half, of the cardiac periods containing Doppler signals.
Grade 3	Most of the cardiac periods contain showers or single bubble signals, but not dominating or overriding the cardiac motion signals.
Grade 4	The maximal detectable bubble signals sounding continuously throughout the heart cycle and overriding the amplitude of the normal cardiac signal.

Grade 0 infers that there is little or no chance of DCS. Grades 1 and 2 are infrequently associated with DCS, whereas the higher grades of 3 and 4 are associated with a high incidence of DCS. The syndrome of 'chokes', together with the passage of bubbles into the arterial system through the pulmonary filter, is especially likely with grade 4 bubble detection.

The Kisman–Masurel classification system

This was designed with the aim of the bubble signal being easily incorporated into a computer program. The bubble signal is divided into three separate categories: frequency; percentage of cardiac cycles with bubbles/duration of bubbles; and amplitude. Each component is graded separately and a single-digit bubble grade is then awarded. The primary site for monitoring is the precordium, preferably over the right ventricle. This system has greater flexibility as it also measures during movement. Monitoring is conducted at rest and after a specified movement, typically a deep knee bend (squatting up and down in a continuous fashion). Although this system appears more complicated than the Spencer system it is the preference of most researchers and is easily learnt.

Both systems suffer from inter-observer error, with some difficulties in subjectivity. Although computer-based counting programs are being developed, human observers are still more accurate than the automated models.

Doppler technology is being used increasingly to assess the extent of bubble formation following multiday, multilevel recreational diving, ascent to altitude after diving and in searching for a patent foramen ovale in divers with severe 'unearned' decompression sickness.

Methods of decompression, and even of treating DCS, have been proposed on the basis of the quantification of bubbles by Doppler techniques. Although of value in research applications, clinically a patient is not a bubble. Bubbles and DCS may develop in tissues, and even in the less accessible vascular system, without being detected by Doppler.

Arterial gas emboli (AGE)

Gas emboli may enter the arterial blood from:

1 Decompression of the blood.
2 Pulmonary tissue disruption, such as pulmonary barotrauma.
3 From venous (paradoxical) gas emboli, by:
   (a) patent foramen ovale or other cardiac septal defect;
   (b) failure of the pulmonary filter due to:
      (i) massive embolization;
      (ii) excessive pulmonary artery pressure
      (iii) Valsalva technique, coughing, straining, etc.;
      (iv) arteriovenous anastomoses.

(c) bronchopulmonary anastomoses bypassing the pulmonary filter.
(d) re-embolization after already passing through the arterial system.

Factors reducing the likelihood of in-situ development of AGE include the higher arterial blood pressure and the relatively short circulatory time.

Even if bubbles do enter the arterial system, a calamity is not inevitable. The distribution will depend on the buoyancy of the bubbles, and many sites are either not critical, or may have alternative circulation. Possibly bubbles below 25 μm diameter may pass through cerebral and cardiac circulation, without effect. Many current surgical procedures produce small quantities of arterial bubbles without causing obvious damage.

BUBBLE INTERACTION WITH TISSUES OR BLOOD

Bubbles may impede circulation and cause congestion and oedema from the backpressure (venous emboli) or ischaemia (arterial emboli). Other effects of gas emboli include:

- Damage to epithelium of vessels, with reduction in blood flow.
- An inflammatory reaction (neutrophils, platelets, fibrin) between the bubble and tissue/blood.
- Release of chemicals that have influence on blood flow and tissue viability.
- Destruction of surfactant.
- Coagulation and complement system activation.
- Increased local pressure, when there is a limit to tissue compliance without damage, e.g. in cells, osteoclast spaces, myelin sheaths, inner ear, spinal column, brain.

From these pathological changes, there may be more persistent damage from DCS than that expected of a treatable gas bubble.

DECOMPRESSION TABLES, TECHNIQUES AND COMPUTATION

Every compressed air dive will require decompression – unless the diver remains under water for ever! Nitrogen will be absorbed in excess of the norm, and it must be offloaded. Decompression may occur at either shallower depths (staging) from the maximum, in which case the specific depths and durations are critical, or during ascent, in which case the rate of ascent is critical.

During the past two decades, there has been a plethora of **computer-designed decompression schedules**. Initially, they were based on the haldanian models, with variations in tissue numbers and half-times, in an attempt to explain anomalies of decompression. Subsequently, as with the University of Hawaii decompression tables, allowance has been made to include cavitation concepts in saturated solutions, a degree of inherent unsaturation from oxygen utilization, and the production of bubble-free decompressions using the gelatin models. Variations of breathing gas mixtures can be considered in the program, and attempts are made to produce bubble-free schedules, which avoid both DCS and dysbaric osteonecrosis. As anticipated, the computer calculations propose dives with deeper first stops, but with duration not dissimilar to the conventional tables for deep diving.

Significant changes have also taken place since the Haldane experiments, clarifying the safe rate of ascent for compressed air scuba diving. Haldane used a 9 metres per minute ascent rate, slow enough to avoid bubble development in the fastest tissues and logistically to permit ascent in standard gear (hard hat). With the freedom of ascent with scuba, these rates were increased. With experience, they have now been decreased.

1956 – US Navy	18 metres/min
1957 – Workman	18 metres/min
1968 – RNPL	15 metres/min
(Hempleman)	(3 metres/min between stops)
1966–76 – Hills	12 metres/min
1975 – Buhlmann	10 metres/min

Apart from the greater opportunity to off-load nitrogen during the ascent, other advantages of slow ascents probably include: reduced rate of VGE production; less pulmonary filter obstruction; and less chance of pulmonary damage (barotrauma or seeding of bubbles).

Not only is the total volume of gas very relevant, but so is the time over which it is released. Bolus doses of gas, such as produced during rapid ascents from short deep dives, are more likely to overload the pulmonary filtration system and thus cause early development of cerebral DCS than the same volume of gas from a shallow long dive, which may be in the slower deep tissues and cause a late joint bend.

The shallow safety stop, usually 5–10 minutes between 3 and 5 metres, is of value as it not only increases the total decompression time, but also reduces by up to 50 per cent the bolus volume of gas that the pulmonary filter has to cope with during that period. Moreover, it also reduces the nitrogen gradients between different tissues.

Oxygen can be used, both on the surface and under water, to increase inert gas elimination. Paul Bert, in the 1800s, observed the protective and therapeutic use of oxygen, breathed on the surface after diving. The duration of the oxygen inhalation needs to increase with the increased load in slower tissues, i.e. with longer dives. When this is used as an added safety factor, the duration of decompression is not lessened. At other times, it is used as a means of reducing decompression time, when the advantages and disadvantages need to be more carefully assessed.

There has been a move towards using VGE detection by Doppler as a device to demonstrate safety in diving table development. It is reasonable to denigrate a decompression table on the basis of its production of excessive VGE, because this is roughly correlated with DCS incidence in short-duration dives. The ideal table will produce no bubbling, but this cannot be presumed from the absence of VGE, as these are probably late manifestations of bubble formation in tissues. DCS may occur without VGE being detected, especially in long-duration and repetitive dives.

All currently used tables rely more on experience than knowledge, and are more effective in reducing slow tissue 'joint bends' than fast tissue neurological DCS.

Problems in developing a reliable mathematical model to predict DCS in humans were reiterated by Brian Hills, who was instrumental in proposing many such models during the perfusion versus diffusion arguments of the 1960s. Tissue perfusion observations reveal that arterioles and the associated capillary system will frequently and periodically shut down, whilst adjacent systems may perfuse normally. Thus, in the one tissue there may be both inert gas retention and inert gas washout! As Hill stated, one bubble was enlarging while a similar bubble in the same tissue was contracting. This observation would be difficult to incorporate into relatively rigid mathematical models.

RNPL/BSAC decompression tables

Hempleman, working at the Royal Naval Physiological Laboratory, introduced his single-tissue model of decompression in 1952. There were several assumptions on which these tables were based. They are almost diametrically opposed to Haldane's concepts, the principles on which the US Navy tables are based. They are as follows:

1 Only one tissue type is involved in the production of type 1 (joint) bends.
2 The rate of uptake of gas in that tissue is limited by perfusion (blood supply).
3 The rate of uptake is greater than the rate of elimination because silent bubbles form in that tissue and interfere with maximal gas elimination, even in trouble-free dives.
4 A certain critical volume of gas can be tolerated without symptoms (compare the critical ratio hypothesis of Haldane).
5 Gas diffusion is analogous to the situation where the affected tissue adjoins arterial blood.

A 'limiting line' was proposed to demonstrate the dangers with deeper dives and longer decompressions. Although the allowable descent rate was 30 metres/minute, the ascent rate was reduced to 15 metres/minute.

Hempleman's tables result in longer decompressions and deeper first stops. They compared favourably with the US Navy tables in safety, at least for recreational diving depths (30 metres).

Unfortunately, because there are different mechanisms involved with various categories of DCS, the exclusion of one tissue DCS (limb bends) does not ensure protection of others, such as neurological DCS, and these are more likely if the RNPL/BSAC tables are used at greater depths.

New schedules were devised in 1968 and were known as the '1968 Air Diving Tables', incorporating a variable ratio of tissue nitrogen tension to environmental pressure. However, these tables were considered too conservative, and the Royal Navy modified them, metricated them and presented them as the RNPL 1972 Tables.

For the RNPL/BSAC tables, the initial no-stop limits are sometimes considered too long. This is especially so with the deeper dives. Some surface intervals were also too short.

In 1988, Hennessy modified the BSAC procedure for calculating repetitive dives and deleted the concept of the 'limiting line', by deleting such exposures. **The BSAC-88 tables** are based on more conservative no-stop limits and a safe no-decompression depth of 6 metres (compared to 9), and makes allowance for the ambient pressure effects on bubbles during repetitive dives, as a source of extra inert gas. The rate of 'off-gassing' is presumed to decrease with each repetitive dive; thus each repetitive dive requires a more conservative decompression than the last. They consist of a set of seven tables, with Table A being used for the initial dive and other tables being determined by the presumed nitrogen retention and the surface interval.

These tables are comparable with the current Buhlmann and Defence and Civil Institute of Environmental Medicine (DCIEM) of Canada tables for non-decompression diving. For dives requiring decompression stops they are often, but not always, more conservative than the US Navy tables, but usually less conservative than the Buhlmann or DCIEM tables.

Bassett tables

The US Air Force commissioned Dr Bruce Bassett to validate schedules for flying after diving. This led him into believing that the US Navy tables had too high an incidence of DCS if they were 'pushed to the limit'. The DCS incidence was about 6 per cent and Doppler bubbles were detected in approximately 30 per cent. These findings were similar to those of Dr Merrill Spencer in Seattle.

With this information, Bassett reduced the no-decompression limits from the US Navy tables by reducing the allowable supersaturation in the various half-time tissues. For the shorter no-decompression dives, Bassett recommended an ascent rate of 10 metres/minute, with a 3- to 5-minute safety stop at 3–5 metres for all dives greater than 9 metres, and a total time under water, rather than the bottom time, being used to calculate the repetitive group after a dive.

Knight and Lippmann modified the Bassett tables for repetitive dive use.

DCIEM tables

The DCIEM of Canada uses a different model for decompression, based on tissue compartments arranged in a series, rather than in parallel as with the US Navy (originally Haldane) model.

It is a very well-researched table and, although it is based on the theoretical model, it has been modified greatly by extensive human testing with Doppler monitoring. The testing was performed in both cold water and under hard working conditions. The single no-decompression times, and most repetitive dives, are more conservative than the US Navy tables.

Buhlmann tables

The Swiss decompression expert, Professor Buhlmann, has conducted many theoretical and practical investigations on the Swiss model which includes 16 theoretical tissue compartments with half-times of 4 to 635 minutes. The testing of the tables at altitude has been more careful than for most other tables.

The permitted duration at deep stops often exceeds the US Navy tables, but the ascent rate is reduced to 10 metres. All no-decompression dives require a stop of 1 minute at 3 metres depth.

It is believed that shorter initial dives will minimize bubble formation on the first dive and so improve the nitrogen off-loading during ascent. This allows longer times for the repetitive dives.

PADI tables

The initial PADI tables were, like the Bassett tables, more conservative than the US Navy tables.

The PADI Recreational Dive Planner is different, and embraces a repetitive system based on the T_{40} or T_{60} tissue compartments rather than on the T_{120}, as in the US Navy tables. The result is that it allows longer repetitive dives after shorter surface intervals.

A small amount of somewhat selective and restricted practical testing of the tables was performed, using Doppler monitoring.

The repetitive dive planner comes in two forms: there is a table version, similar in design to the original tables; the other version is the 'wheel', and can be used for multi-level diving.

'New' tables

The development of tables involves such a heavy cost, in terms of time and money, that it is most unlikely that any new tables could be tested comprehensively enough to be acceptable in the diving community, without many years of effort and huge financial backing.

For table development, there not only needs to be a model on which the tables are based (to allow extrapolation to other situations), but also a testing of the tables against both a decompression database (such as is available in the USA and Canada) and also practical testing of the tables under conditions more likely to produce DCS. When such tables are tested, they will need to have a predictable decompression incidence allocated for each dive profile.

Testing tables to determine their DCS safety is very demanding. If one required a 99 per cent certainty that a specific profile has a <1 per cent DCS incidence, then 456 DCS free dives would need to be achieved. For a much more liberal 20 per cent DCS incidence with a 95 per cent confidence, then only 14 DCS dives would be needed.

US Navy EL decompression model

The US Navy developed the EL decompression model during development of decompression tables to support the MK-15 UBA which supplied a fixed 0.7 ATA partial pressure of oxygen in nitrogen. This algorithm was an extension of the typical Haldane approach in which gas uptake and elimination is exponential, but if the supersaturation exceeds a certain amount, gas elimination becomes linear, greatly slowing offgassing. Decompression stops were determined by a table of 'M values', much the same as in the Haldane approach, and for this reason it is called the deterministic EL algorithm. This deterministic EL algorithm was man-tested using a fixed partial pressure of oxygen in nitrogen, fixed partial pressure of oxygen in helium and air. Currently a version of the deterministic EL algorithm for air and fixed partial pressure in nitrogen breathing gases has been programmed into an off-the-shelf decompression computer which is undergoing evaluation in the USN experimental diving unit.

In 1988 the work was extended to recast the EL algorithm as a probabilistic model. A complete set of decompression tables for air and 0.7 ATA fixed partial pressure of oxygen in nitrogen were computed, but these have not been put into use by the Navy. The main reason for this was that the no-decompression limits were seen as too short, and some decompression times as excessively long. Due to its mathematical complexity the probabilistic EL algorithm has not been programmed into a computer.

Multi-level diving

There are a number of different techniques that can be used to allow for diving at multiple levels. In all multi-level diving it is necessary that the deepest part of the dive be carried out first, and that the dive should progressively become shallower. Multi-level diving in the other direction, i.e. going from shallow to deep, is particularly hazardous because any bubble formed will expand rapidly when the diver returns to the surface, as a result of the higher pressure gradient between the tissues and the bubble.

From current statistics it appears that, for a set nitrogen load induced by a single dive, a square-wave pattern is probably more likely to cause DCS than a multi-level dive, if the latter is carried out from deep to shallow.

> *Any decompression table or dive computer, which permits less time for decompression, will cause a greater need for recompression chambers.*

Repetitive diving, when combined with multi-level diving, becomes a mathematical nightmare,

and dive computers only supply answers to theoretical tissue models not divers.

DECOMPRESSION COMPUTERS

These instruments have been in widespread use since the late 1960s, but none has yet received approval by conservative and safety conscious organizations. Indeed, many have been withdrawn despite extensive publicity and acceptance by the general diving population. Three concepts have been used:

1 **Mechanical models** of gas uptake and release, such as the movement of gas through restricted orifices. The SOS and Farallon meters were of this type. These models were gross oversimplifications of the dynamics of decompression and were inappropriate for the possible range of diving exposures – nevertheless, the later SOS meters were moderately safe for single, shallow, no-decompression dives of less than 24 metres.
2 Electronic models of the established **decompression tables**, such as the Suunto USN meter. As these offered no duration advantage over the tables, and no ability to 'cheat' the tables, the diving community did not generally accept them.
3 Electronic models using the **decompression theories** on which different tables were originally based (usually quoting the US Navy or Buhlmann tables), and integrating this with a theory of **multi-level diving**. Because these models excluded the many safety factors of 'rounding up' the depths, durations and surface intervals used in the established tables, they allowed much greater underwater exposure with less decompression. In an attempt to reduce the inevitable increased danger of DCS, shorter no-decompression limits were often included and slower ascent rates were advised. Nevertheless, the dives permitted by these devices were shown to be dangerous, and this was supported by the clinical observations.

Subsequently, diving computer manufacturers have included more and more safety factors in an attempt to counter the many defects inherent in the meters. One of the major manufacturers, in 1989, released a more conservative booklet for their users, stressing the additional need for:

- training in diving computer use;
- acceptance of a DCS incidence;
- not diving in excess of 39 metres, without specialized training and professional supervision;
- no repetitive dives deeper than 24 metres with surface intervals of less than 1 hour;
- in repetitive diving, successive dives should be shallower;
- in multi-level diving, use an ascending profile;
- always add safety margins;
- on all dives, stop at 3 metres for at least 5 minutes;
- do not begin using a computer if diving has been undertaken within the previous 24 hours;
- extra care in altitude diving and flying after diving; and
- back-up equipment and preparation for equipment failure.

The warnings are timely and address some of the problems with the metres. One major problem common to all models, and which has both safety and financial implications, is the high incidence of faults developing during underwater exposure.

In 1994, Acott reported an increased association in the rate of DCS in dive computer users when compared with those divers using a dive table. This was particularly evident in dives to 30 metres or deeper.

In the twenty-first century, dive computers tell divers their dive time and the time left before they exceed decompression limits, record accurately maximum depth and total duration, and include ascent rate monitors and audible warning alarms if this rate is exceeded. Some also display remaining air pressure and remaining time based on the diver's air consumption and depth. However, no computer model is foolproof, and all divers must be aware of their deficiencies and learn to dive with the assistance of a computer and not be governed by them.

RECOMPRESSION THERAPY TABLES

The history of this subject is dealt with elsewhere. Suffice it to say that the reverence with which some

of these tables are held is a triumph of faith over knowledge.

In 1873, A.H. Smith first reported the treatment of DCS by recompression during the construction of the Brooklyn Bridge. Moir, during the construction of the Hudson River tunnel, reduced a 25 per cent death rate in the workforce to less than 2 per cent by recompression therapy.

The 50 per cent recurrence rates observed following treatments in the first half of this century were reduced by the introduction of three principles:

1 Depth of treatment being the depth of relief plus 1 ATA, with a 6 ATA maximum.
2 A 12-hour 'soak' at 9 metres.
3 The later addition of oxygen at shallow depths.

The US Navy air tables 1–4 were promulgated in 1945 – before they were tried on patients. All tests were performed on asymptomatic, recently dived, healthy subjects. The high incidence of DCS production resulted in modifications, with Table 1 becoming 1 A, Table 2 becoming 2A, etc.

As the US Navy became more involved in the treatment of civilian divers, with their less controlled diving and often a delay in recompression, the failure rate moved from 6 per cent to 43 per cent in 1963.

Previously Behnke, in 1939, obtained excellent results by treating DCS with oxygen at 2 ATA. This was not acceptable to the Navy at that time, probably for the same reason that the experience of Edgar End, who treated 250 cases with oxygen at 45 feet, was rejected. The regime was not considered 'sailor proof'. As an alternative to the oxygen treatment, Behnke also used Heliox (30 per cent oxygen, 70 per cent helium) at approximately 30 metres for treatment of caisson workers with air-induced DCS, in 1942. Half a century later, this was still considered very avant garde.

Goodman and Workman reduced the failure rate to 3.6 per cent by using oxygen at 18 metres for 90 minutes – a success rate that many today would envy. Later this was arbitrarily extended to 2 and 4 hours and, later again, air breaks were added in the belief that this would reduce the possibility of oxygen toxicity. A risk–benefit analysis of these modifications has still to be carried out.

Many would question whether the duration of oxygen breathing is not of more importance than the depth.

ACKNOWLEDGMENTS

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11

Decompression sickness: pathophysiology

ROBYN WALKER

INTRODUCTION

There is little disagreement today that the symptoms and signs of decompression sickness (DCS) are a direct consequence of the effects of gas bubbles within the tissues and bloodstream. Robert Boyle reported the first case of DCS in 1670 when he described a bubble moving to and fro in the waterish humour of the eye of a viper after decompression. The snake was 'tortured furiously' by the formation of bubbles in the 'blood, juices and soft parts of the body'.

Yet some 330 years later it is still not known with certainty how and where bubbles form in the body. No one proposed mechanism could account for all types of DCS, and it is possible a number of different mechanisms are involved dependent upon the affected tissue.

Decompression sickness is a multisystem disease, and single organ involvement is uncommon. The definitive pathophysiology of DCS remains to be elucidated but most likely is mutifactorial in origin.

BUBBLE FORMATION

Bubbles may form within the intravascular space or within the tissues.

Arterial bubbles

Whilst it is possible for bubbles to form *de novo* in the arterial system, this is thought to be an unlikely mechanism in all but explosive decompression. The combined effects of arterial blood pressure and the equilibration of arterial blood with alveolar inert gas tensions will reduce the arterial pressures of these, and avoid bubble formation.

With decompression, venous bubbles may become arterialized. The lungs normally act as an efficient bubble filter, the bubbles being trapped and undergoing resolution. This bubble filter may be overwhelmed in the presence of a large number of bubbles, resulting in the passage of venous emboli through into the left heart chambers and consequently into the arterial circulation.

Venous bubbles may also gain access to the arterial circulation through the passage of emboli via a

patent ductus arteriosus (present in 30 per cent of the population), septal defects or patent arteriovenous anastomoses, especially when the pulmonary artery pressures increase with emboli blockage of the pulmonary circuit, producing a right-to-left intracardiac shunt (paradoxical emboli). Similar effects could be induced by straining, sneezing, coughing, Valsalva manoeuvre, etc.

The greater the number of pulmonary emboli, the greater the pulmonary artery pressure and the greater the likelihood of paradoxical gas embolism and arterial gas embolism (AGE).

The importance of a right-to-left shunt in the heart, through a patent foramen ovale, allowing venous gas emboli (VGE) to become AGE, has been previously observed in autopsies. Moon *et al.*[1] elegantly demonstrated a higher than expected incidence of this disorder among severe DCS cases. They examined 30 patients with a history of DCS for the presence of a patent foramen ovale by bubble contrast, two-dimensional echocardiography and colour-flow Doppler imaging, the former technique being found more sensitive. Of the 18 patients with serious signs and symptoms of DCS, 61 per cent had shunting, compared to a 5 per cent prevalence with healthy volunteers as controls (this increased to 8.5 per cent during the Valsalva manoeuvre).

Marabotti *et al.*[2] have demonstrated a post-dive significant right ventricular overload and a possible impairment of ventricular relaxation in recreational divers with so-called asymptomatic silent bubbles. An increase in right atrial pressure may cause cardiac dilatation and open a potentially patent foramen ovale, allowing paradoxical emboli.

Arterial bubbles may also occur as a result of pulmonary barotrauma (see Chapter 6).

Venous bubbles

Venous bubbles may form *de novo* or result from the intravascular release of tissue bubbles. As stated previously (see Chapter 10), bubbles form from supersaturation combined with micronuclei, and as a result of tribonucleation and cavitation.

Venous bubbles are detectable post-diving, by Doppler (see Chapter 10). After typical near-decompression dives they are observed some minutes after ascent and persist for some hours. For short deep dives, they occur early and are not long-lasting, but for longer shallower dives they take longer to develop and may continue to be produced for many hours.

Extravascular bubbles

Autochthonus bubbles are those which form within the tissues. How these bubbles are generated also remains conjecture, and attempts to visualize them histologically have been fraught with difficulties. Autochthonous bubbles are most likely to form in tissues with a high gas content (high solubility of gas in tissue and low diffusivity) and relatively poor perfusion, e.g. spinal cord white matter, adipose tissue and perhaps periarticular tissue.

EFFECTS OF BUBBLES

Cerebral DCS

Arterial bubbles distribute according to buoyancy relative to blood in large vessels, and with blood flow in small vessels.[3] This explains the preponderance of cerebral symptoms in DCS, as in the erect diver the bubbles will distribute preferentially to the cerebral circulation via the cerebral vessels. Thus, small multivessel pathology is more common.

Arterial gas bubbles may do one of three things. They can:

1 Obstruct the vessel permanently.
2 Temporarily obstruct the blood vessel and then eventually redistribute through the venous circulation.
3 Pass through the vessel directly into the venous circulation.

If a vessel becomes permanently blocked by gas emboli this is most likely to occur at the junction of the white and grey cerebral matter at the arteriolar level. An ischaemic infarct will follow, with the clinical signs and symptoms dependent upon the vessel distribution. Histologically, punctate multivessel pathology and haemorrhages are seen at the border between the grey and white matter.

Arterial bubbles may initially trap in a cerebral vessel. Locally, a reflex increase in blood pressure is

observed as successive cardiac contractions attempt to push the bubble through into the venous circulation. The majority of gas emboli are redistributed in this way. Alternatively, the gas bubble may pass directly through the arterial circulation, through the capillary bed and into the venous system.

Experimentally[4] there has been shown to be a great increase in blood–brain permeability with the passage of gas emboli. This disturbance is reversible and may, by inducing cerebral oedema, account for the delayed symptoms of cerebral DCS. Langton[5] reports that DCS involves a bubble-mediated endothelial injury that leads to neutrophil activation and adherence. The neutrophil response occurs secondary to endothelial cell dysfunction with activation of specific cellular adhesion molecules and/or chemotactic cytokine production. Intracerebral vasospasm leads to the clinical syndrome of cerebral DCS. Experimentally, the importance of neutrophils in the pathogenesis of injury has been confirmed. Both leucocyte depletion and pretreatment with doxycycline (which is known to inhibit a range of neutrophil activities) reduces the early electrophysiological and neurological abnormalities of AGE.

Simply put, during their passage the arterial emboli damage the vascular endothelium, setting in place a neutrophil-mediated inflammatory cascade which opens up the blood–brain barrier, resulting in localized decreased cerebral blood flow and haemoconcentration. This inflammatory reaction is thought responsible for the 'constitutional' symptoms of cerebral DCS such as generalized malaise, headache, lethargy, fever and vague aches and pains.

Cerebral DCS is thought largely to occur as a result of this embolic form of bubble injury; however, other pathological possibilities for cerebral DCS include myelin sheath damage and autochthonous bubbles. These are also multifocal and predominantly affect the white matter.

Pathology may also be induced by other emboli (lipid, platelet, etc.), while aggravated damage may result from raised intracranial pressure, coagulopathies, etc.

Spinal cord DCS

Spinal cord DCS is not thought to be principally embolic, and possible mechanisms for this condition include venous infarction of the cord, autochthonous bubble formation and embolic disease. The spinal cord changes occur predominantly in the white matter, and are most often observed in the mid-thoracic, upper lumbar and lower cervical areas, with the lateral, posterior and anterior columns suffering in that order. Often there is sparing of some long sensory tracts.

Venous infarction of the cord

This theory proposes that when VGE block the pulmonary arterioles there is a rise in intrathoracic pressure (pulmonary hypertension) which interferes with the venous drainage from the spinal cord, through back pressure in the anastomoses of the spinovertebral-azygos system. This interference with venous drainage causes engorgement and thrombosis of the spinal cord with infarction in the comparatively poorly vascularized areas.

This theory explains why spinal DCS is more common than cerebral DCS, despite the much greater mass and blood supply (and thus emboli) to the brain.

Autochthonous Bubbles

Autochthonous or tissue bubbles – gas bubbles evolved from the nitrogen dissolved in white matter of the cord – may arise because the white matter has a high lipid content and this absorbs more nitrogen than the grey matter, which is relatively spared.

Francis[6] proposed that autochthonous bubbles may result in spinal cord dysfunction by:

- destruction of the axons around the growing bubble;
- stretching and compression of axons around the growing bubble;
- a biochemical interaction between blood and bubble (i.e. activation of the inflammatory cascade); and/or
- haemorrhage into the tissue secondary to damage to the microvasculature, persisting after resolution of the bubble.

The spinal cord is a 'soft' tissue, but with a limited compliance, because of its confinement by the vertebrae and dura. If the volume of the canal is increased by 10 per cent or more, the slack is taken up and the

pressure within the cord escalates rapidly. This can develop by either the engorgement of blood, production of gas or experimental injection of saline. Once the slack has been taken up, the escalating pressure could compress the venous system. This mechanism explains how spinal DCS cases can respond to recompression (reducing bubble size and cord pressure), but deteriorate with ascent (expanding bubble size and cord pressure).

The autochthonous bubble theory therefore can account for rapid-onset spinal DCS, yet the autochthonous bubble theory for spinal cord disease also has its critics.

Notably, tissue bubbles have been rarely demonstrated pathologically, and this theory does not account for spinal cord disease developing hours after surfacing from the dive. For bubbles to form there must be a degree of supersaturation (see Chapter 10). The time frame for spinal cord bubbles to form will be limited to minutes rather than hours; thus, for these bubbles to remain silent for some hours before symptom onset suggests that another mechanism such as activation of the inflammatory cascade may be involved.

Gas Emboli

Embolic disease is considered to be a less frequent cause of spinal cord DCS, since AGE resulting in ischaemia is more likely to affect the more vascular grey matter of the cord rather than the white. In addition, the spinal cord is relatively poorly perfused when compared to the brain, and redistribution of VGE to the spinal cord will take a finite period of time. Because of this delay critics suggest that VGE cannot account for the rapidly progressive spinal cord disease observed in some divers soon after surfacing from a dive.

No single mechanism of disease completely explains the varying forms of spinal cord DCS. Most likely, a combination of mechanisms coexist to explain the observed clinical condition.

Musculoskeletal DCS

Although the musculoskeletal symptoms are common presentations of DCS in humans, the pathology is not well understood. Radiological evidence of gas in joint spaces, periarticular areas, fascial planes and tendon sheaths is occasionally seen, but this is not necessarily the causative lesion. Gas in a joint space is not usually painful.

Extravascular bubbles in the subperiosteal area, tendons, ligaments, joint capsule, fascia and muscles are thought to cause the pain of 'bends'. These tissues are tight, and the development of a bubble is likely to distort and stretch the tissue and/or its nerve supply. Conventional teaching suggests that application of local pressure may reduce this effect and produce relief in 60 per cent of cases, though this high incidence is not our experience. Crepitus can sometimes be felt and this, with the associated pain, can be massaged away.

Bubbles in the articular vascular supply have been proposed, but are unlikely as recurrences tend to be in the same site. Bubbles in the myelin sheaths of peripheral nerves and referred neurological pain have also been incriminated, and verified in some cases.

Inner-ear DCS

Several non-exclusive explanations have been proposed for inner-ear DCS that is more frequently associated with deep dives using Heliox mixtures:

1 Rupture of bone lining the otic spaces by bubbles developing within the enclosed osteoclasts. This current theory proposes that osteoclast cells lie in bone cavities that do not permit expansion of bubble nucleation and growth during decompression. This causes elevated pressures which rupture the bone lining into the otic fluid spaces. Haemorrhages and blood protein exudates are seen in the vestibular and/or cochlear systems. Petrous bone fractures attest to the considerable local pressures generated. Later there is a growth of ectopic bone and fibrous tissue into the semicircular canals, and this has been demonstrated radiologically and at autopsy in both experimental animals and humans.
2 Supersaturation and helium bubble formation: the inner ear can receive helium from its own blood supply, and from gas which passes across the round and oval windows bordering the middle-ear space. The perilymph can thus become saturated with this gas, and can reach a

steady state of gas content quite rapidly. The bubbling that develops during decompression may then disrupt the delicate inner-ear structures. Pressure fluctuations in the middle ear reflect Eustachian tube functioning as well as ambient pressures, and this may aggravate the release of dissolved helium into gas phase.

3 Supersaturation by counterdiffusion across membranes, such as the round or oval window, results in bubbles at the interface between the middle and inner ear. The inner ear is unique in having the only localized extracellular fluid space that may not be dependent on the circulation for inert gas transport. Helium still occupies the diver's middle-ear space, even after he changes from Heliox to air breathing, and the situation across the otic fluids is not dissimilar to the classic counterdiffusion situation breathing a slow heavy gas which saturates the blood and fast tissues, while a fast gas diffuses inwards from the environment (middle-ear space). This hypothesis explains the additive effects of the inert gas switching and decompression. Although decompression is not required to fulfil these conditions, it would be expected to aggravate the situation by causing the bubbles to expand.
4 Vascular emboli, either gaseous, lipid or thrombotic, in the end-arteries of the inner ear (without the anastomoses of other cerebral arteries) cause ischaemia. The rarity of inner-ear disease with generalized intravascular emboli makes this hypothesis questionable.
5 Impaired perfusion from the severe haematological effects of DCS.
6 Gas-induced osmosis: transfer of fluid between endolymph (perfused with blood) and perilymph (saturated with gas in the middle ear) could result in vestibular membrane damage.
7 Inner-ear membrane ruptures associated with tissue damage or hydrostatic forces consequent on bubble formation in an enclosed space. This would explain the confusion between DCS and inner-ear barotrauma, permitting both to exist and, in the future, the possibility of inner-ear reconstructive surgery must be considered.
8 Inner-ear haemorrhages develop rapidly, within hours of the above pathologies, and are likely to convert a potentially correctable lesion to a permanent one.

Peripheral nerve DCS

Autochthonous bubble formation in the myelin of peripheral nerves will result in a patchy sensory damage or motor impairment, predominantly involving the limbs. In severe cases, there may be a glove-and-stocking distribution, but the usual patchy presentation is with paraesthesiae, numbness and weakness. Pain may be related to a major plexus, and may be long-lasting. The differentiation between peripheral nerve and an incomplete spinal lesion is important, because the prognosis is less worrisome if the clinical symptoms are due only to peripheral nerve involvement.

Cardiopulmonary DCS

A small amount of bubble production may actually enhance the body's ability to expel nitrogen, when they become blocked in the pulmonary vascular bed, and diffuse into the alveolar spaces. As a general rule, however, it is probably safer that gas is transferred to the lungs in its dissolved form, and is then equilibrated with the alveolar gas pressures. After uneventful dives, bubbles that have originated in the tissues and venous system may be entrapped in the lung circulation for an hour or two.

Clinical manifestations are noted when approximately 10 per cent or more of the pulmonary vascular bed is obstructed. The effect of gas in the pulmonary vessels is to displace blood and inflate the lungs intravascularly. This may reflexly produce a shallow rapid breathing, due to the inflation of the vascular tree, and thereby reduce both alveolar ventilation and compliance. Pulmonary oedema develops.

AGE may enter the coronary arteries, especially if the diver is horizontal. These bubbles are thought to produce similar effects as AGE in the brain, namely activation of the inflammatory cascade via endothelial damage or obstruction of the vessel.

Cardiac manifestations may be initiated by cerebral DCS affecting the brainstem.

Cutaneous DCS

Cutaneous DCS takes many forms, and is thought to occur as a result of bubbles within the skin,

cutaneous blood vessels or lymphatics (see Plate 5).

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12

Decompression sickness: clinical

ROBYN WALKER

INTRODUCTION

Decompression sickness (DCS) is a dysbaric disease caused by the liberation of gas bubbles from solution, into tissue or blood. Its sequelae range from death to permanent neurological impairment to full recovery.

Bubbles may cause direct mechanical effects such as tissue distortion or disruption, or ischaemia by blocking blood vessels or increasing tissue pressure sufficiently to impair perfusion. Bubbles also are known to stimulate the inflammatory cascade via neutrophil activation; a consequence of endothelial or cellular damage (see Chapter 11).

Doppler studies tell us that the so-called 'silent' bubbles which do not produce clinical features of DCS are present under many practical diving conditions, including dives conducted well within so-called non-decompression table limits. There is a much higher incidence of venous gas emboli than DCS, although 'silent bubbles' precede the overt manifestations in many cases. They may also be the foundation on which DCS develops during subsequent dives.

Individual susceptibility to DCS varies enormously and varies from day to day, making mathematical modeling of DCS very difficult. Indeed, no computer model has been found to predict a true incidence of DCS, and most have 'fudge' factors added to account for this variability.

PREDISPOSING FACTORS

Some physiological and environmental factors are thought to increase the likelihood or severity of DCS. Most of these influence the blood supply to tissues and therefore the speed of gas uptake or release. These include exercise, environmental temperature, age, obesity, dehydration and carbon dioxide retention.

Exercise

Exercise has various effects, which are sometimes contradictory. Exercise performed while at depth is likely to increase the blood supply to the muscular tissues, and increase the rate of inert gas absorption at that site, which is then the site of the DCS. In fact, exercise can increase decompression requirements by a factor of 3.

During or after decompression, **severe exercise** results in an increase in the speed of bubble development and in the number of bubbles – perhaps due to increased cavitation from tribonucleation of tissues, or to turbulence (similar to shaking a bottle of champagne!).

Mild exercise during recompression is of value in increasing the rate of gas elimination, perhaps by increasing tissue perfusion, if supersaturation and bubble growth have not been incurred.

Resting after decompression may give the body a longer time to liberate the inert gas it has absorbed during the dive, although occasional movements are warranted to ensure that paralysis or incoordination has not developed without the diver being aware of it. Thus, the routine practice of periodically walking the length of the compression chamber during decompression and therapy is to be commended.

Physical fitness

Physical fitness, perhaps due to its relationship to more efficient muscular use and blood flow, seems to be of some protective value to divers.

Environmental temperature

Temperature may influence DCS in a complex manner, by its influence on perfusion (increased temperature producing increased blood flow) and solubility (lowered temperature producing increased gas solubility). Divers who are exposed to cold at maximum depth may have less tissue perfusion and DCS in no-decompression dives than divers in warm water and with hot-water suits.

If the diver becomes mildly hypothermic – which is not uncommon in longer dives – the ability to eliminate the nitrogen is decreased, and DCS is more likely. In some studies the perfusion rate in muscle was shown to be halved, thus doubling the required duration of decompression.

Divers who become cold during decompression have a lowered perfusion of tissues during ascent, less gas uptake from the tissues, and more DCS. The opposite occurs in warmer conditions. During decompression it is better to be warm, because the nitrogen elimination is increased.

After the dive, exposure to sudden and excessive heat (such as with a hot shower) produces increased superficial blood flow and lowered solubility of gas, resulting in a bolus of nitrogen being mobilized, with gas phase separation and delivery to the lungs. Both skin and generalized DCS manifestations could develop.

Age

Increasing age increases DCS incidence, possibly due to impaired perfusion or to already damaged vessels being more susceptible to other flow interferences. Abnormalities and degeneration within joint surfaces also increase the likelihood of tribonucleation in the aged.

Obesity

Obesity increases the tissue mass available to absorb more inert gas, and hence may increase DCS in longer-duration dives. Not all surveys demonstrate an association between fat content and DCS among normals (excluding the obese). Nitrogen is 4.5 times more soluble in fat than in water and non-fatty tissue. In active adult populations, women have 20–30 per cent body fat compared to 10–20 per cent for men.

Dehydration

Dehydration, caused by the environment, exercise, water loss from respiration and immersion, and the impracticability of fluid replacement while diving, will reduce perfusion of tissues and thus the elimination of inert gas.

Carbon dioxide retention

Increased carbon dioxide pressures, from the effects of pressure, exercise or breathing resistances with equipment, may cause increased perfusion during the dive, with increased nitrogen loading. It is also a factor with inadequate ventilation in caissons, chambers and helmets.

Other factors

In altitude exposures, **females** have been shown to have up to a four-fold greater increase in DCS incidence than males, but this can not be readily generalized to diving exposures. There is no support for the hypotheses that the contraceptive pill or menstruation increases the incidence of DCS in females (see Chapter 60).

Alcohol over-indulgence may influence judgement at the time, but more commonly the dehydration, vasodilatation and heat loss which develops in the hours afterwards will aggravate DCS.

Physical injury, such as a sprained joint or a previous episode of DCS, may predispose to DCS due to scarring and alterations in local tissue perfusion. Thus, some clinicians are concerned that spinal operations, such as a laminectomy, may be associated with spinal DCS risk.

Dive profiles may influence the likelihood of DCS: the deeper the dive and the more decompression required, the greater the incidence of decompression sickness. Surface decompression (returning to the surface before being recompressed) is often more dangerous.

For a single-depth/duration exposure, DCS is more likely with a direct ascent to the surface than a gradual ascent interrupted by reasonable and shallower stops, i.e. with a safety stop or multilevel diving. The opposite will occur if there are increased depths at successive levels.

The no-decompression dive durations allowed by the US Navy tables, if followed without any added safety factors, have a 1–5 per cent incidence of DCS. No-decompression dives allowed by the DCIEM air tables have a 2 per cent incidence of DCS.

Multiple ascents during the dive may, by initiating venous gas emboli that are trapped in the pulmonary filter but escape to the arterial system during subsequent compressions, increase the likelihood of DCS.

Repetitive dives: if a diver exposes himself to increased pressures within 24 hours of a previous dive, the residual nitrogen remaining within his tissues will increase the likelihood of DCS. Mild or insignificant cases may be made much worse. Advice is often given to recreational divers to take a day off after every three days of repetitive diving. If diving has produced asymptomatic bubbles, then subsequent diving even longer than 24 hours afterwards will probably increase the likelihood of DCS.

Travelling to altitude to dive, or exposure to altitude after diving, may provoke DCS by producing or expanding existing bubbles. Air travel may also predispose to DCS because of the dehydration effect in the cabin altitude, if not rectified by a compensatory fluid intake. More often, aviation exposure can induce DCS days after a dive, especially from repetitive, long or saturation dives. Flying is not advisable for at least 24 hours after normal, non-decompression, recreational diving.

Breathing different gas mixtures, such as nitrous oxide after air diving, decompressing on air after diving on Heliox, or breathing a slow diffusing gas while in a fast diffusing gas environment, may produce local or general pressure gradients which cause bubbles to develop. Existing bubbles may also expand if faster gases are breathed, e.g. breathing Heliox after producing air bubbles (see Chapter 11).

Decompression staging in a **horizontal position** results in an increased rate of gas elimination, as compared with the vertical or seated positions. In water, decompression has similar advantages over dry decompression, although this is not mirrored in practical situations because of the many adverse factors during diving compared to dry compression.

One factor reducing the likelihood of developing DCS is **adaptation or acclimatization** – the repetitive and recent exposure to increased pressures. DCS is more probable during the first week of diving operations, and following lay-off periods of more than a week. Although the more susceptible divers would be selectively eliminated at an early stage, this is not the whole explanation. It appears as if, with regular diving, a slight degree of resistance to DCS can develop for that diving depth. For example, the

incidence of DCS in caisson workers is halved in the second week, and again in the third week.

Factors influencing DCS

- *Exertion*
- *Physical fitness*
- *Temperature – cold water, hot shower*
- *Female gender*
- *Age*
- *Obesity*
- *Dehydration*
- *Increased carbon dioxide pressures*
- *Alcohol intake*
- *Physical injury*
- *Adaptation*
- *Dive profile*
- *Rapid and multiple ascents*
- *Repetitive and multi-day diving*
- *Altitude exposure*

The currently fashionable explanation for adaptation is the removal of naturally occurring gas nuclei, which are thought to be the nidus on which the bubble develops. This is also the reason why divers and caisson workers are advised to work up to their maximum exposures gradually.

Many other possible aggravating factors have been proposed, both endogenous (serum complement, lipids, smooth muscle activating factors, etc.) and exogenous (smoking, migraine), but these require further investigation before their confirmation.

CLASSIFICATION

The classification into type I (mild) and type II (serious) DCS and cerebral arterial gas embolism (CAGE) was introduced as an attempt to differentiate cases, so that identification, prognosis and therapy could be more standardized. Unfortunately, this clinical classification was not well defined or applied appropriately.

Type I was described as (musculoskeletal) pain-only bends. Skin manifestations were also allocated to this subdivision. Recently, the US Navy Diving Manual pragmatically defined type I DCS as having the following characteristics: (i) extremity pain only, skin rash or lymphatic disease, with (ii) a normal neurological examination before treatment and (iii) symptoms resolved within 10 minutes at 2.8 ATA on oxygen.

Type II includes those presenting with symptoms other than pain or skin involvement, or with abnormal physical signs.

Although the use of type I is reasonably clear when it is applied to acute DCS affecting the musculoskeletal system ('joint bends'), investigations reveal many subclinical neurological manifestations – depending on the sophistication of the investigation. Also, some skin manifestations are of ominous significance.

Potentially serious type II cases involve the central neurological, cardiovascular, respiratory and gastrointestinal systems. Nevertheless, in most DCS series, peripheral nerve symptoms are allocated to the same group as spinal and cerebral manifestations.

An additional classification – type III DCS – was then introduced to describe the clinical picture observed with combined arterial gas embolism (AGE) and DCS. This scenario occurs when intravascular bubbles (AGE) from pulmonary barotrauma seed the circulation and tissues, which are loaded with inert gas from previous dive exposures. The pressure gradient from the supersaturated tissues causes inert gas to diffuse into the bubbles, which are at environmental pressure. The result is that a rapidly developing DCS (often spinal) evolves from an existing or resolving CAGE case.

DCS grading

Type I ('pain only')	*Type II ('serious')*
• *Limb or joint pain – dysfunction*	• *Central nervous system disorder*
• *Itch*	• *Inner ear*
• *Skin rash*	• *Lungs*
• *Localized swelling*	• *Cardiac*
	• *Type I symptoms developing under pressure*
	• *Other manifestations*

Critics of this classification system argue that distinguishing between DCS and CAGE is often very difficult and artificial, as the initial management is

identical. Symptoms of CAGE can occur prior to the diver reaching the surface, and the arterialization of DCS-induced venous gas emboli may result in CAGE. Retrospective studies also revealed a lack of inter-observer consensus in differentiating DCS from CAGE. The lack of consensus between experienced clinicians in reaching a diagnosis also hinders useful interpretation of treatment outcomes, and attempts at multicentre trials have failed due to the lack of appropriate identification of the study group.

As mentioned previously, many of the so-called cases of type I DCS also showed evidence of neurological signs on detailed examination, and follow-up studies revealed a high incidence of psychological or personality abnormalities in individuals treated for type I disease.

This led to the development of a descriptive classification system for dysbaric disease that makes no attempt to distinguish between DCS and CAGE, and is based on the clinical presentation. This classification aims to provide a system by which individual subgroups of decompression illness (DCI) can be identified such that comparisons of treatment outcome in multicentre trials can be achieved.

Symptoms and signs of DCI are described with both an evolutionary and an organ system term, and describe the development of the condition prior to recompression. The presence of barotrauma is also recorded. For example, a diver presents within hours after a deep dive with weakness of his left foot. The weakness rapidly ascends to involve the whole leg, and is associated with urinary retention. This patient is described as having progressive neurological (spinal) DCI. A second diver presents unconscious on the surface after a rapid ascent, and then regains consciousness. This patient is described as having spontaneously resolving neurological DCI with presumption of pulmonary barotrauma and CAGE.

Both classification systems remain in use around the world, although there is a growing tendency among clinicians and researchers to use the DCI system. It is still considered important to decide on whether or not there is evidence of pulmonary barotrauma as this has profound implications on future fitness to dive.

Decompression illness (DCI) classification

1 *Evolution*
- *spontaneous recovery – substantial improvement in the clinical condition.*
- *static – the condition does not change substantially.*
- *relapsing – cases in which spontaneous improvement has occurred, only to relapse.*
- *progressive – if the number or severity of clinical signs is increasing.*

2 *Organ system term*
- *neurological*
- *limb pain*
- *cutaneous*
- *cardiopulmonary*
- *lymphatic*
- *audio-vestibular*
- *constitutional*

3 *Time of onset – the time interval between the end of the dive and symptom onset.*

4 *Gas burden – estimation of the residual gas burden, e.g. repetitive dive group, estimation based on depth, time and surface interval.*

5 *Evidence of barotrauma*
- *pulmonary – requires evidence such as pulmonary tissue damage, pneumothorax, surgical emphysema or X-ray evidence of extrapulmonary gas.*
- *ear*
- *sinus*

PRESENTATION

Onset

DCS develops after the subject has commenced decompression or ascent. Most cases present within 6 hours of the dive.

Over 50 per cent of cases of DCS develop symptoms within 1 hour of the dive, and 90 per cent within 6 hours.

DCS is more likely to develop rapidly under the following conditions:

- Deep dives, in which fast tissues are loaded and release gas rapidly.
- Rapid ascents, increasing the inert gas gradient.
- With pre-existing bubbles from other dives.
- Gross decompression omission.
- With the predisposing factors referred to above (see pp. 137–140).

A slower onset of symptoms is observed with:

- More shallow dives.
- Marginal decompression omission.
- Slower ascents.

A well-regulated diver who develops DCS from a conservative diving profile is more likely to develop a milder DCS well after the dive. A diver who dives repetitively, deeper, with omitted decompression and less controlled ascents, often develops a more serious DCS, more rapidly.

Constitutional

Fatigue, lethargy, malaise, headache, generalized aches and pains are the most commonly reported symptoms of DCS. Patients often report overwhelming tiredness despite many hours sleep, and often describe relief of these symptoms with recompression as 'if the lights were suddenly turned on' or 'like a veil being lifted'. These symptoms are thought to be a consequence of bubble-induced tissue and endothelial inflammatory process activation.

Musculoskeletal

This has been described as 'joint bends', 'type I', 'pain-only bends', decompression arthralgia or limb pain DCI. First, there is an ill-defined discomfort or numbness poorly localized to a joint, periarticular or muscular area. The subject may protect or guard the affected area, although in the early stages he may obtain some relief by moving the limb. Over the next hour or so the discomfort develops into a deep dull ache, then a pain with fluctuations in intensity, sometimes throbbing and occasionally with sharp exacerbations. Limitation of movement is due to pain, and the limb is placed in a position that affords the most relief. The duration of pain is often related to the severity of symptoms. Usually there are no signs of joint inflammation or effusion.

The shoulder is the more common joint affected in recreational divers, in approximately one-third of cases. In caisson workers, aviators and deep saturation divers, the knee 'bend' is more common. Other joints, about equally affected, are the elbows, wrists, hands, hips, knees and ankles. Often, when two joints are involved, they are adjoining ones, and frequently the localization is between joints, over the scapula, on tendon insertions, etc. The involvement is rarely symmetrical.

The application of local pressure, by means of a sphygmomanometer cuff, has been reported to result in considerable relief and thus be of diagnostic value. However, this is not common or universally believed. The site of pain can sometimes be transferred by massage of the area. Occasionally, a recent or old injury might predispose to DCS.

In the mild cases, fleeting symptoms are referred to as 'niggles', and may only last a few hours. The pain of the more severe cases usually increases over 12–24 hours and, if untreated, abates over the next three to seven days to a dull ache. Local skin reactions may occur over the affected joint.

An uncommon presentation of DCS, in which the gas is in the joint capsule, is seen with shallow long-duration air dives, such as those performed by professional shell fishermen (see Chapter 65). These divers dive for many hours on consecutive days to depths of less than 15 metres, usually without decompression. The DCS is painless and develops many hours after the ascent. The only symptom is the loud crunching noise with joint movement. Abalone divers refer to it as the 'squelching shoulder', and the condition usually clears by the next morning. It can be detected by X-ray or computed tomography (CT) scan of the joint. A similar condition has been described in aviators.

Neurological

Civilian divers, who perform deep air dives (especially with fast ascent rates), tend to develop cerebral symptoms that reflect fast tissue DCS. Divers who do

CASE REPORT 12.1

PR, a 37-year-old sports diver, performed dives to 30 metres and 15 metres on Saturday. There were several hours between the dives, but the duration of each was unknown. The same dives were repeated on Sunday. His decompression meter did not prescribe decompression stops, and all dives were incident-free. After the last dive he drove home 200 km, feeling tired and happy. On getting out of bed the next morning, he developed a sudden severe sharp pain in the right ankle. The pain steadily increased in intensity and prevented any weight-bearing. There was no history of injury to the joint, no history of arthritis and no other symptoms or signs. Recompression to 2.8 ATA breathing 100 per cent oxygen provided improvement within 15 minutes. After 30 minutes there was no pain at rest, but it was still present on forced extension. Ascent was commenced at 12 metres/minute and was uneventful. After reaching the surface he was completely asymptomatic, able to walk and bear weight with no pain.

Diagnosis: musculoskeletal DCS (type I)

CASE REPORT 12.2

LS, aged 34 years, dived on compressed air to 50 metres for 10–12 minutes. He surfaced without decompression and, after having lunch, dived again to 50 metres for 6 minutes. The surface interval was approximately 105 minutes. His decompression meter prescribed a 10-minute stop at 3 metres on the second dive. He was well after surfacing, but 3 hours later felt a few twinges in his right elbow and right shoulder, and some tingling in the right hand. Ten hours later he was awakened from sleep with severe pain in the right elbow and shoulder joints. He also noted some loss of power in the right arm. Examination revealed diminished sensation to pin-prick and light touch over the right arm from the deltoid region. There was some loss of motor power in the flexors and extensors of the elbow and wrist. Administration of 100 per cent oxygen resulted in slight diminution of pain in half an hour, but no improvement in power or sensation. Recompression in water to 9 metres breathing 100 per cent oxygen resulted in considerable improvement. After 30 minutes his symptoms were completely alleviated, but there was some slight tenderness around the shoulder joint. He was asymptomatic after decompression at 12 minutes per metre, and the only residual sign following therapy was slight muscle tenderness and stiffness over the posterior deltoid. The next day he was completely symptom-free.

Diagnosis: musculoskeletal DCS (type I bends), with peripheral nerve involvement.

repetitive air dives for moderate durations produce more spinal problems, often with cerebral manifestations. Cerebral DCS often presents earlier than spinal DCS.

The shorter the time between surfacing and developing DCS, the greater the likely severity and the worse the prognosis.

Cerebral DCS is fastest in onset, reflecting fast tissue bubble development and haemodynamic effects. Peripheral nerve tends to be slowest, reflecting the release of inert gas from myelin. Spinal DCS is intermediate, reflecting both pathologies. Myelin sheaths, being lipid structures, are capable of absorbing nitrogen more than helium. Cerebral, spinal and peripheral nerve DCS are more common with air diving.

With the very deep helium diving, and especially with excursions from deep saturation depths or from the initial pull during decompression from great depths, inner-ear disorders are more frequent.

The clinical subdivisions of neurological presentations are cerebral, cerebellar, spinal, inner ear and peripheral nerve.

Cerebral

The clinical manifestations depend on the site of vascular injury and collateral arterial supply, and are largely a matter of chance (see Chapter 11). They tend to affect multiple sites in the frontal and parietal lobes, and represent impaired perfusion in the anterior and middle cerebral artery distributions.

Any cerebral tissue may be damaged by gas bubbles, and this causes a great variety of manifestations, analogous to those of the diffuse cerebrovascular disease of general medicine. Especially noted are the homonymous scotomata, unilateral or bilateral, single or multiple. Others include hemiplegia, monoplegia, focal or generalized convulsions, aphasia, alexia, agnosia, hemisensory or monosensory disturbances, migraine and confusional states.

Retinal lesions with intravascular bubbles and haemorrhages have been described. Vision is more commonly affected by interference with the neural pathways, with appropriate visual field defects, either directly from bubble development or indirectly by the cardiovascular effects of DCS. Long-term retinal lesions, with low retinal capillary density at the fovea, microaneurysms and small areas of capillary non-perfusion, are said to be related to DCS incidence (see Chapter 44).

Raised intracranial pressure has been observed, and may be associated with severe headache. Sludging of blood in the venous sinuses, oedema or other cerebral pathology may also produce this effect.

Serial, non-cultural, psychometric assessments of cognitive function may be of value if given before, during and after treatment. They provide measurements of mental impairment and response to treatment. Permanent mental impairment has been claimed as sequelae of cerebral DCS. Brainstem involvement may also result in cranial nerve and pupillary abnormalities.

Even after treatment of DCS, there is frequently evidence of neuropsychological dysfunction – as well as abnormal psychometric, electroencephalographic and brain imaging studies. This may last for many weeks or months, and is similar to the post-concussion syndrome, with or without a symptomatic depression.

Cerebellar

These lesions produce ataxia, incoordination with typical neurological signs of hypotonia, diminished or pendular deep tendon reflexes, asynergia with dysmetria, tremor, dysdiadochokinesis, rebound phenomenon, scanning speech and nystagmus. The 'staggers', which is variously described as vestibular, posterior column, spinal cord and cerebral DCS, is probably more often due to cerebellar lesions, usually without nystagmus.

CASE REPORT 12.3

SD, a 35-year-old clearance diver, was participating in a series of dives at an altitude of 315 metres above sea level. The dive was to 37 metres (which corrected to 42 metres) and conducted in accordance with the DCIEM tables. SD reported that as he was climbing the ladder to exit the water his left leg felt heavy. He then stated he was unable to lift his left foot as he tried to remove his dry suit. As he walked, his toes dragged along the ground, and he was unable to wriggle his toes and flex his ankle. He then noted the weakness quickly spread proximally and he was no longer able to walk. He was immediately recompressed in the on-site six-man portable recompression chamber to 18 metres. His symptoms resolved within minutes of reaching 18 metres, and he completed a USN Table 6. He made a full recovery and returned to diving after four weeks.

Diagnosis: spinal cord DCS (type II)

Spinal

The spinal cord changes are predominantly in the white matter, and are most often observed in the mid-thoracic, upper lumbar and lower cervical areas, with the lateral, posterior and anterior columns suffering in that order. Often there is sparing of some long sensory tracts (see Chapter 11).

Local spinal or girdle pains may precede other symptoms, developing into serious spinal cord disease. It is more common in patients who also have respiratory symptoms ('chokes'). The symptoms and signs are those of paraplegia or paraparesis, and include urinary retention with overflow incontinence.

The lower abdominal pain due to a distended bladder from spinal DCS is frequently misdiagnosed.

> *Spinal DCS often follows pulmonary involvement.*

Inner Ear

Inner-ear DCS is characterized by cochlear damage (tinnitus, sensorineural hearing loss) and/or vestibular disorder (prostrating vertigo, nausea, vomiting, syncope). It is especially noted with deep helium diving (in excess of 100 metres), excursions from saturation diving or switching inert gas mixtures during a dive. It is sometimes precipitated by a helium-breathing diver changing rapidly to air breathing. In helium diving, inner-ear DCS may occur in isolation whereas it usually occurs in conjunction with other organ or system involvement if diving on air.

In deep helium diving, the diagnosis is relatively easy. It is often an isolated DCS symptom, presenting deeper than most symptoms, but others may supervene at more shallow depths. Ear barotraumas are less common with helium diving, probably due to the rapid diffusibility of the gas.

In shallow scuba air diving, isolated inner-ear DCS is less well documented and probably rare. Isolated inner-ear disease with shallow air diving is much more likely to be due to inner-ear barotrauma (see Chapter 7).

The consequences of vertigo, such as near-drowning, vomiting, dehydration, electrolyte disturbances and distress, are more important in a patient who is already seriously ill with DCS, but are dramatic enough in their own right to endanger a diving operation.

If incorrectly diagnosed during decompression (e.g. if the symptoms are incorrectly attributed to seasickness), then further decompression may result in more damage to the vestibular apparatus.

A delay of onset of symptoms is not uncommon in cases of inner-ear barotrauma and in DCS. The development of symptoms during an action which increases intracranial and otic pressure, such as coughing, lifting anchors, etc. makes inner-ear barotrauma more likely.

In cases of generalized neurological DCS, vestibular symptoms are misdiagnosed and often confused with cerebellar disease. Investigations, including electronystagmography, clarify the peripheral (vestibular) or central (cerebellar) nature of the disease. In these cases, the auditory involvement is only a minor part of the disease, and other manifestations dominate the clinical presentation and treatment.

Inner-ear haemorrhage, as a consequence of inner-ear DCS, may result in a failure to respond to recompression.

Table 12.1 *Differential diagnosis of inner-ear disease due to diving*

	Inner-ear barotrauma	Inner-ear DCS
Dive exposure	Any	Near or exceeding decompression limits
Onset	Descent, ascent or post-dive	At depth, ascent or post-dive
Associations	Ear barotrauma	Other DCS symptoms, deep or saturation dive
Gas breathed	Mainly air	Mainly helium or hydrogen
Treatment	Conservative/surgery	Recompression/oxygen

DCS, decompression sickness

Peripheral Nerve

Bubble formation in the myelin of peripheral nerves will result in a patchy sensory damage or motor impairment, predominantly involving the limbs. In severe cases, there may be a glove-and-stocking distribution, but the usual presentation is with paraesthesiae, numbness and weakness. Pain may be related to a major plexus, and may be long-lasting.

Cutaneous

Skin manifestations range from being local and innocuous, to generalized and ominous, with a complete spectrum in between. If they develop with water exposure, they are more likely to be serious than with chamber exposures, in which the inert gas can be absorbed through the skin. They have been variously described as follows.

Pruritis

This is a common complication of diving in compression chambers and dry suits, more than in water environments and wet suits. It may be partly or wholly due to gas passing into the skin from the hyperbaric gas environment, when the temperature is elevated. The increased temperature produces vasodilatation and accelerates the cutaneous uptake of nitrogen during compression. The elimination of gas is restricted by the vasoconstriction that accompanies the drop in environment temperatures during decompression.

It is often a transient effect, presenting very soon after decompression, and is not considered a systemic or serious manifestation of DCS. It is noticed mostly after short deep exposures. The areas affected are the forearms, wrists and hands, the nose and ears, and the thighs. There is usually no objective sign. In other cases, a slight folliculitis may be observed as red punctate areas, when this presentation merges with the next. The symptoms are attributed to small gas bubbles in the superficial layers of the dermis, and especially near its entry via the epidermis and the sebaceous glands.

Scarlatiniform rash

This is a progression of the above. Piloerector stimulation, and perhaps tissue histamine release, produces a red punctate rash. The distribution is predominantly over the chest, shoulders, back, upper abdomen and thighs, in that order, the rash perhaps lasting for several hours.

Erysipeloid rash

This is a further extension of the above and occurs over the same distribution, but with the involvement of endogenous gas interfering with venous drainage. It is a definite sign of systemic DCS. Some of the skin appearance is thought to be a reflex vascular reaction. The lesions are collections of papules, which may merge to form large plaques with flat and firm borders. Coughing or performing the Valsalva manoeuvre will accentuate the venous markings (Mellinghoff's sign) (see Plate 5).

Cutis marmorata marbling

This commences as a small pale area with cyanotic mottling, but may spread peripherally, becoming erythematous with extension of cyanotic mottling. The area is warmer than the surrounding skin, and the swelling and oedema result in a mottled appearance. Recompression provides dramatic relief. The area may become tender to palpation in a few hours, but the other signs may have diminished or disappeared by then. Marbling of the skin is a cutaneous manifestation of what is occurring elsewhere in the body, and this is a serious sign of DCS. Gas bubbles are present in both tissues and blood vessels.

Subcutaneous emphysema

This has the typical crepitus sensation on palpation, either in localized areas or along the tendon sheaths. It can be verified radiologically and should not be confused with the supraclavicular subcutaneous emphysema extending from the mediastinum, due to pulmonary barotrauma.

Lymphatic obstruction

This presents as a localized swelling which may be associated with an underlying DCS manifestation. If it involves hair follicles, a peau d'orange or pigskin appearance with brawny oedema is characteristic. It is common over the trunk, but it is also seen over the head and neck.

Counterdiffusion of gases

There have been occasional reports of skin and mucosal swellings due to counterdiffusion of gases. This results in bubbles forming in tissues from gases diffusing at different rates, but with the total gas pressure at the interface exceeding the environmental pressure. This is only likely when the subject's body is exposed to a readily diffusible gas, while he breathes a slower diffusing gas (see Chapter 10 and Plate 6).

Other manifestations

Formication may be the presentation in any of the skin manifestations described above, or due to involvement of the peripheral nervous system or the spinal cord. The neural involvement may also result in numbness, hypoaesthesia, paraesthesia or hyperaesthesia of the skin. Signs of inflammation may also occur over affected joints. Bruising is sometimes described over the chest and abdomen in serious cases, but this is not due to genuine tissue haemorrhages, because it blanches on local pressure.

Gastrointestinal

Mildly affected patients may present only with anorexia, nausea, vomiting or retching, abdominal cramps and diarrhoea. When the condition is severe, local ischaemia and infarction of bowel, with secondary haemorrhages, may result. In such cases the use of drugs that encourage haemorrhage, such as aspirin or heparin, could be detrimental. In some of the DCS fatalities, gastrointestinal haemorrhage was the final cause of death.

CASE REPORT 12.4

CT, a 24-year-old recreational diver, dived to 27 metres for 39 minutes. As he surfaced he reported he had blacked out momentarily, but this went unreported at the time as his dive buddy had developed acute spinal and cerebral DCS and was evacuated to the nearest recompression chamber. CT was left to take the dive boat home.

That night he felt lethargic, vomited his dinner, and had difficulty sleeping. He did not feel well enough to go to work and remained at home by himself for three days. On the fourth day he presented to work at which time his colleagues immediately arranged for CT to be transported to the local hospital.

On arrival at the recompression facility, CT was complaining of pain in his knees and left elbow and extreme lethargy. He was found to be mildly confused with slow mentation. He displayed poor short-term memory. His left pupil was dilated and reacted sluggishly. He had reduced power (4/5) in both upper limbs and lower limbs. His gait was unsteady and he was unable to walk unaided. There was decreased sensation to light touch and pin-prick over the left upper arm and ulnar aspect of the left forearm. There was decreased light touch, pin-prick over the left leg. His bladder was grossly distended.

A urinary catheter was inserted (residual urine 1300 mL) and the patient underwent an extended USN Table 6 with minimal improvement. He underwent daily soaks for 12 days with fluctuations in his clinical signs. No further improvement was documented after this, and daily hyperbaric treatments were ceased.

A MRI brain scan was reported as showing no abnormality. CT was reviewed by a neurologist 12 days post dive, who found evidence of residual left hemiparesis, decreased light touch and pin-prick on the left, autonomic changes affecting his left foot and frontal lobe damage resulting in a gait apraxia and a Korsakoff-like syndrome. At discharge he was still unable to walk unaided and discharged to an in-patient rehabilitation facility.

Diagnosis: neurological DCS (type II)

Cardiorespiratory

The presence of gas bubbles in the blood may hamper the microcirculation and produce both local hypoxia and generalized haematological sequelae.

Local ischaemic effect

This may follow cerebral, coronary, gastrointestinal, renal or splenic occlusion, etc. The result of these occlusions may be tissue ischaemia and infarction, and the clinical manifestations will vary according to the organs involved. Specifically, an infrequent but troublesome cardiac manifestation of DCS is the development of a ventricular arrhthymia, which may not respond to recompression therapy. It is not clear whether these all represent coronary emboli, or whether they result from extravascular bubbles interfering with the myoneural conducting mechanism of the heart.

Pulmonary involvement ('chokes')

Clinical manifestations are noted when approximately 10 per cent or more of the pulmonary vascular bed is obstructed. The effect of gas in the pulmonary vessels is to displace blood and inflate the lungs intravascularly. This may reflexly produce a shallow rapid breathing, due to the inflation of the vascular tree, and thereby reduce both alveolar ventilation and compliance. Pulmonary oedema develops.

Tachypnoea is significant. The initial symptom of chest pain is aggravated by inspiration, sometimes with an irritating cough, which may be precipitated by cigarette smoking. Interference with the pulmonary circulation can result in a decrease in pulse rate and a decrease in blood pressure, progressing to circulatory collapse in severe cases.

The pulmonary effects usually appear early, and can be reduced by shallow respiration, oxygen inhalation or recompression. Without therapeutic intervention they are followed by either a rapid resolution (with pulmonary shunting of blood away from the obstruction and a loss of gas from the lung's vasculature by diffusion into the alveoli) or a progression of symptoms due to increased numbers of bubbles, vascular stasis and the blood/bubble interactions mentioned previously (see Chapter 11). These may cause deterioration in the clinical state irrespective of recompression and present as the well-recognized respiratory emergencies ('shock' lung syndrome, adult respiratory distress syndrome or ARDS, disseminated intravascular coagulation). Right heart failure may occur.

Investigations are often not possible, but electrocardiographic evidence of right axis deviation, high-peaked P wave, and right ventricular strain may be found. Radiological evidence of pulmonary oedema may be detected.

Post-decompression shock

In very severe cases, e.g. in explosive (very rapid) decompression or following grossly inadequate decompression, there may be a generalized liberation of gas into all vessels, resulting in rapid death. The presence of gas bubbles in the circulating blood results in a bubble/blood interaction that leads to all grades of vessel wall damage and haematological reactions from haemoconcentration to disseminated intravascular coagulation (DIC).

The effect of hypotension, combined with air, platelet and lipid emboli, causes secondary damage to capillaries, increased capillary permeability and extravasation of fluid into tissues. The signs and symptoms of hypovolaemic shock, such as haemoconcentration, postural hypotension, syncope, low urinary output, etc., are not uncommon. Similar to the pulmonary manifestations, they are either resolved quickly or proceed ominously.

The consequences of both haemoconcentration and DIC are discussed below.

COAGULOPATHIES AND OTHER LABORATORY FINDINGS

Although rare, certain biochemical and haematological abnormalities (the 'coagulation cascade') are reported in association with severe decompression and DCS. A generalized stress response, from physical, environmental and psychological factors, may result in elevations of blood catecholamines, free fatty acids, isoenzymes, leucocytes and cortisol. A reduction in erythrocyte numbers is related to the aquatic environment and weightlessness. The additional

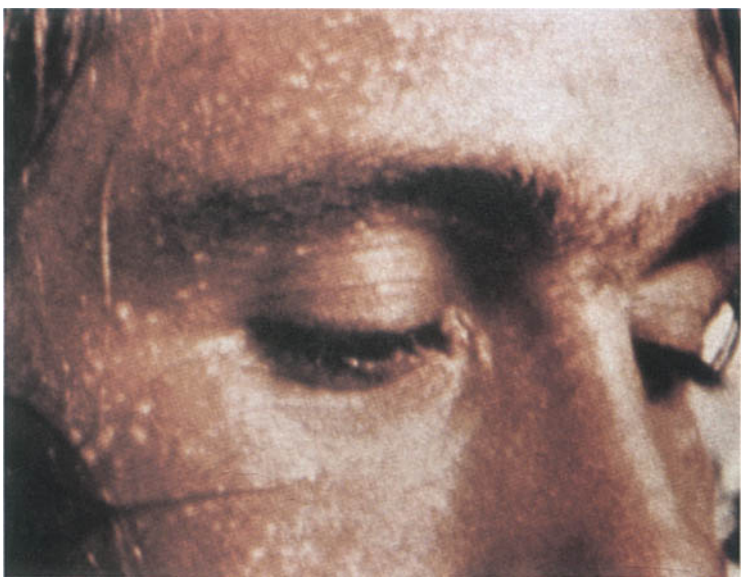

Plate 4 *Decompression sickness: skin lesions of counterdiffusion. The subject breathed a neon/oxygen mixture at 360 meters (1200 feet), while exposed to a chamber of helium/oxygen. Gross itching accompanied the intradermal bubbles. (Photograph by courtesy of Professor C.J. Lambertsen.*

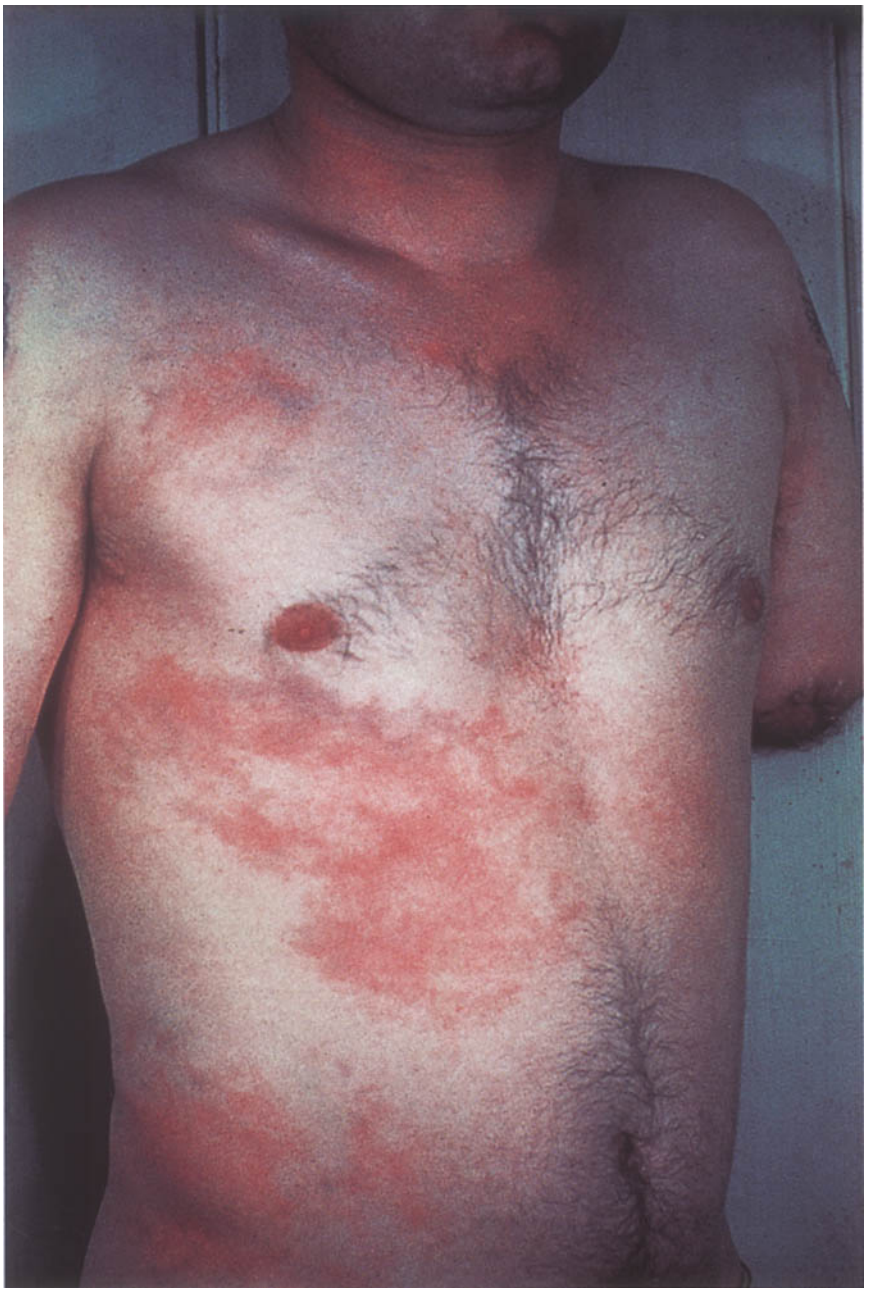

Plate 5 *Skin lesions of decompression sickness. This diver, who had had an upper-limb amputation, developed 'bends' pain in the phantom limb, and skin bends over the body. Both responded rapidly to recompression therapy. (Photograph courtesy of Dr Ramsay Pearson.)*

CASE REPORT 12.5

PB, a 41-year-old technical diver presented after a dive to 78 metres using Heliox as his bottom mix and Nitrox and oxygen for his decompression stops. The oxygen was secured to a staging platform. He experienced difficulty switching to 100 per cent oxygen due to the strong current and so remained on a Nitrox mixture for 20 minutes longer than planned. On surfacing, he experienced pain in his right shoulder, right elbow and left knee. He developed disabling vertigo and nausea on attempting to stand. He was treated on board the boat with 100 per cent oxygen and was transported immediately to the naval recompression chamber.

On examination, there was nystagmus on looking to both the left and right, grade 4/5 power in his right wrist extensors, clonus of the left patella reflex, and he was unable to stand or sit unaided, secondary to his vertigo. There was no associated tinnitus or hearing loss.

He was immediately recompressed to 18 metres with minimal change in his symptoms after three oxygen periods. The chamber was then dived to 30 metres with the patient breathing 50/50 oxygen/helium. A lignocaine infusion was commenced. Post-treatment the patient was much improved and able to sit unaided. His nausea resolved. PB received a further six hyperbaric oxygen soaks.

On review at six weeks post-discharge the patient still experienced some disequilibrium with sudden movements, but felt that these were decreasing. He was very eager to continue his technical diving career despite contrary medical advice.

Diagnosis: cerebellar DCS (type II) with musculoskeletal manifestations.

influences of intravascular bubbles on these changes are not yet clarified. Abnormal red blood cells may be produced, with spiculated echinocytes originating at depth and becoming more marked with the development of DCS, from deep dives.

An increase in the erythrocyte sedimentation rate is noted in the latter stages of decompression from deep Heliox saturation dives, and to a lesser degree from the more shallow air saturation dives. It is not directly related to depth or oxygen partial pressures.

Complement activation has been described with DCS, and it is postulated that DCS produced by unstressful dive profiles may be predicted in some divers on the basis of this mechanism.

Biochemical changes are sometimes noted. There is a decrease in plasma cortisol associated with decompression, and a decrease in serum sodium and blood lactate in DCS. Some of the reports of biochemical changes with DCS are suggestive of tissue damage. Decompression *per se* does not produce these enzymatic changes. With moderately severe DCS, serum enzymes will reflect the organs affected.

Decompression, and especially inadequate decompression, results in thrombocytopenia, increased fibrin formation and haemoconcentration. Many decompressions are associated with overt or 'silent' intravascular bubble formation. Blood reacts with bubbles as it does to other foreign bodies – with a deposition of protein (especially fibrinogen) and coalesced plasma lipids. The protein deposition is thrombogenic and attracts platelets, which aggregate around it and release clotting agents. With these developments, there is a drop in plasma lipids and circulating platelets. Hypovolaemia may result from a reduction in blood flow, or hypoxia, associated with the blood/bubble complex. Expansion of the intravascular volume, inactivation of the complement system and reduction in platelet adhesion and aggregation has been used therapeutically.

If the fibrin-clotting mechanism is activated, then all manifestations of DIC may result. Once this cycle has commenced, the disease does not necessarily respond to recompression therapy. Thus, such cases may deteriorate even while at an initially adequate recompression depth. Attention to intravenous fluid replacement and correction of coagulation defects follows general medical principles, and may improve the recompression results.

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13

Decompression sickness: treatment

ROBYN WALKER

INTRODUCTION

> No-one who has seen the victim of compressed air illness, gravely ill or unconscious, put back into a chamber and brought back to life by the application of air pressure, will forget the extraordinary efficiency of recompression, or will be backward in applying it to a subsequent case of illness.
>
> *Robert Davis (1935)*

This chapter will deal with the definitive management of decompression sickness. (For information on the first aid management of the diving accident victim, see Chapter 48.) The ideal treatment will vary as the disease varies, there often being little similarity between the decompression sickness (DCS) syndromes. Compare the following cases:

- Saturation DCS as the diver very slowly approaches the surface.
- The same diver subjected to an extreme excursion from saturation.
- Inner-ear or helium-based DCS.
- The cerebrovascular incident after a short bounce to 50 metres.
- The joint bend developing hours after a long shallow dive.
- The dramatic crises involving pulmonary, haematological and neurological systems after explosive decompression from saturation or from gross omitted decompression.
- Respiratory symptoms followed by rapid development of spinal paraplegia.
- The mild joint DCS of a shallow diver who has remained well within the established tables.

These cases cannot be optimally managed by a single regime, yet the approach to their management is similar.

The mainstay of treatment for DCS is recompression followed by a slow decompression back to atmospheric pressure, the patient hopefully being

devoid of symptoms and signs. Oxygen is breathed to increase the washout of the inert gas and promote bubble resolution. Fluid replacement is recommended, as divers are often dehydrated as a consequence of cold water diuresis, seasickness and bubble-induced fluid shifts out of the intravascular compartment. Adjuvant therapies such as antiplatelet agents, corticosteroids, heparin and dextrans have been used without success, although the use of lignocaine appears promising.

If left untreated, the pain of joint DCS resolves spontaneously, usually within days or weeks. There have been reports of spontaneous resolution of cases of neurological DCS without recompression therapy; however, the majority require treatment or remain symptomatic. It is not known whether untreated DCS increases the likelihood of dysbaric osteonecrosis or subclinical neurological injury.

Care should be taken to avoid circumstances that will aggravate the 'bubbling' of DCS. These include excessive movement of the patient, exposure to altitude and the breathing of certain gases (e.g. nitrous oxide anesthesia).

RECOMPRESSION

The aim of recompression treatment[1] is to produce:

- an immediate reduction in bubble size, which will
  - cause the surface tension pressure acting on the bubble to increase, and may cause its resolution;
  - increase the surface area of the bubble relative to its volume, enhancing gas diffusion out of the bubble;
  - reduce the length of the gas column that is trapped intravascularly and hence may allow perfusion pressure to push it into the venous circulation;
  - reduce the compression of adjacent tissues; and
  - reduce the bubble–tissue and bubble–blood interface and the secondary inflammatory reactions.
- an increase in the diffusion gradient of gas out of the bubble (see Fig. 10.3);
- relieve ischaemia and hypoxia; and
- restore normal tissue function.

There are three factors to consider after deciding that a diver needs recompression therapy:

1 The depth required for therapy.
2 The gas mixture to be used.
3 The rate of decompression.

Depth of recompression

In deciding the depth of recompression, there are three different approaches that may be made: (i) recompress to a pressure (depth) dependent upon the depth and duration of the original dive; (ii) recompress to a depth that produces clinical relief of symptoms, and then tailor the gas mixtures for decompression from that depth; and (iii) recompress to a predetermined fixed depth, i.e. according to standard tables of recompression therapy. Only the third is in common practice, but each has some logic to it under certain circumstances. These are now elaborated further.

Recompress in relation to dive depth and duration

This technique uses the principle that if a dive to 4 ATA produces DCS, then recompression to 4 ATA should relieve the symptoms. This approach was best typified by the now defunct concept of treating aviator DCS merely by descent to ground level.

This is not a particularly satisfactory technique, because it is designed to cope with the total quantity of gas dissolved in the body during the original dive, irrespective of its distribution. Because DCS is the clinical manifestation of a gas bubble lodged in a vulnerable area, it is necessary to recompress in order to reduce the size of that particular bubble, irrespective of the total quantity of inert gas dissolved in the body. There is also evidence that once a bubble is formed it may not completely disappear, and bubble nuclei remain.

The one advantage of this approach, apart from its simplicity, is seen when a diver develops DCS very soon after surfacing from a deep dive. Under these conditions, a prompt return to the original depth will ensure that there is no tissue-to-bubble pressure

gradient that could cause bubble growth at a lesser depth.

Recompress to a depth of relief

This empirical approach was first used in underwater recompressions, to reduce as far as possible the depth exposure. It is still applicable, both underwater and in chambers, to achieve this end. The freedom to be able to choose any depth, that results in an acceptable clinical result, and then select an appropriate breathing gas, is invaluable in the very serious cases.

In deep and saturation exposures the patient was recompressed to the depth at which all major symptoms disappeared, plus one additional atmosphere. This presupposes that the additional pressure would result in a reduction of the subclinical bubbles size, with the resultant increase in surface tension promoting bubble resolution.

This approach is not without difficulty, as it requires a sound knowledge of saturation decompression plus the ability to mix and administer different gas mixtures. Nitrogen narcosis, central nervous system and pulmonary oxygen toxicity all become important considerations. The recompression chamber (RCC) should have a mixed gas capability to support such operations.

Recompression using standard recompression schedules

This approach uses standard recompression tables that can be roughly divided into the following groups:

- air tables;
- oxygen tables;
- oxygen tables with deep excursions; and
- saturation decompression.

The air tables start at 30 and 50 metres, the oxygen tables at 9–18 metres, the deep excursions are commonly to 30 metres, and the saturation treatments depend on the depth of symptom relief.

The standard tables of recompression clearly state the gas mixture to be used (usually air or oxygen) and the rate of decompression. Standard air tables, oxygen tables and gas mixture tables are presented in the Appendices (pp. 671–698).

Standard recompression tables

A prudent diving physician will advise non-experts to adhere to strict treatment guidelines, as depicted in the manuals, but retain his own flexibility to treat the cases which did not respond, or when the facilities may not be appropriate to apply the guidelines. It is for this reason that more than one set of treatment techniques is covered in this text. The standard tables are as follows.

Air tables (US Navy 1A, 2A, 3 & 4, RN 52, 53, 54, 55, 71, 72, 73)

The USN first published their air recompression tables 1–4 in 1945, and these were the standard treatment tables for over 20 years. What is remarkable about these tables is that they were never tested on subjects with DCS, but the test subjects made normal dives, which required decompression, and then they were subjected to a treatment table. The treatment tables were deemed satisfactory, if the subjects did not surface with DCS after one of the treatment tables!

During the 1960s failure rates of up to 50 per cent on the air tables resulted in the development of the shallow oxygen tables.

Many experienced clinicians are reluctant to use air tables because of the problems they bring and their variable benefits. The difficulties with air tables may include: logistical problems with prolonged decompression; aggravation of symptoms during ascent; nitrogen narcosis and DCS in the attendants; and respiratory distress due to the increased density of air under pressure, especially if pulmonary involvement is already present. The results are often not adequate unless the symptoms are mild, recent and not produced from gross omitted decompression.

However, if oxygen is unavailable and the RCC can logistically support a treatment table lasting up to 40 hours, then air tables may be considered.

Oxygen tables (USN 5 & 6, RN 61 & 62, Comex 12)

The introduction of standard oxygen tables using 100 per cent oxygen interspersed with air breathing gave more flexibility and improved results. They are shorter in terms of use, needing only 2–5 hours. The

physiological advantages are in the speed of bubble resolution and increased oxygenation of tissues, and in countering some of the pulmonary arteriovenous shunting effects.

Disadvantages include the less immediate reduction in bubble size, i.e. to less than half the volume achieved with the 50-metre standard air tables, the fire hazard, oxygen toxicity, and the occasional intolerance of a distressed patient to oxygen or to a mask. Although the pressure gradient of nitrogen in the intravascular bubble-to-blood is increased with the oxygen breathing, if the diver has previously dived in excess of 18 metres, there could well be a gas pressure gradient from tissue-to-extravascular bubble during the early phase of recompression.

With the above qualifications, the use of oxygen tables has received worldwide acceptance as the starting point for all standard recompression therapy. Whilst a number of authors have reported significant failure rates with them, no better performing alternative has been identified.

Whenever oxygen is used at atmospheric or greater pressures, attention must be paid to oxygen toxicity. Unless following established safe protocols, it is usually suggested that the oxygen parameters should not exceed those likely to result in neurological or pulmonary toxicity (see Chapter 17). In cases of potential disability, these risks may be acceptable.

Oxygen tables with deep excursions (USN 6A, Comex 30)

USN Table 6A involves the addition of an initial period of breathing air at 50 metres followed by a standard Table 6. This table was initially introduced as a treatment of cerebral arterial gas embolism (CAGE) in submarine escape trainees (with a low nitrogen load), although later studies have not shown any benefit from this deep excursion over the shallow 18-metre oxygen tables in divers.

Most centres now recommend starting with a standard USN Table 6 at 18 metres; however, in severe cases of DCS or cases not responding the option exists to then go deeper using a mixed gas table such as a Comex 30. Heliox mixtures (50/50) are substituted for oxygen at depths greater than 18 metres. In refractory cases there is then opportunity to go to 50 metres using either a modified USN Table 6A or RN Table 64 breathing Heliox mixtures.

Saturation tables (USN 7)

Saturation treatments are applied when divers have developed DCS during or just after decompression from saturation exposures. The customary treatment tables are inappropriate due to the extreme gas loads in 'slow' tissues, the contributions that this can make to developing bubbles, the presumed slow development of symptoms and the often excessive oxygen exposure. In general, increased pressure is applied and the oxygen percentages are less. They may also be used after recompression to depth of relief in severe cases and when other tables have failed.

Standard oxygen therapeutic tables have the following advantages over the standard air tables:

- *Economy of time*
- *Increased speed of bubble resolution (increased nitrogen gradient)*
- *Increased oxygenation of tissues*
- *Flexibility of combining with mixture and air tables*
- *Better results than air tables*

Disadvantages include:

- *Less immediate reduction in bubble size*
- *Oxygen toxicity*
- *Intolerance of patient to oxygen or mask*
- *Increased risk of fire*

Gas mixture

Recompression results in a reduction of bubble size, but may also be associated with a further uptake of gas into the bubble. Ideally, a breathing mixture would be selected that diffuses into the bubble at a slower rate than the inert gas diffuses out, resulting in bubble shrinkage. This is one reason why the oxygen tables are preferred to the air tables, as no further inert gas can be absorbed.

There is anecdotal evidence that recompression using Heliox has been beneficial in treating DCS after air dives. Hyldegaard *et al.*[2] have demonstrated that, in some tissues, while breathing air there is a steady increase in bubble size. Oxygen breathing resulted in an initial increase and then decrease in bubble size, whilst Heliox breathing resulted in a progressive shrinkage in bubble size.

The initial increase in bubble size with oxygen may be explained by the fact that at equal partial pressure differences the flux of oxygen in fat is twice that of nitrogen and four times that of helium.[2] This may explain the initial worsening of symptoms seen occasionally when patients with DCS are recompressed on oxygen.

There are currently no definitive studies available to suggest that Heliox tables are more effective than the oxygen tables, although many centres will move to a Heliox table for the difficult or non-resolving acute case. It is also easier to breathe at depth than air – this is especially relevant to patients with respiratory 'chokes'.

Replacement of 'air breaks' with '20/80 Heliox' breaks during oxygen recompressions retains the advantage of reducing oxygen toxicity while continuing to reduce bubble size and not adding to the nitrogen load.

In determining which therapeutic table should be selected the following should be remembered:

- *The natural history of the disease*
  - *with increasing surface interval prior to symptoms, the less likely they are to worsen;*
  - *neurological symptoms leave sequelae, many others do not.*
- *The value of pressure*
  - *recompression to depth of dive will prevent bubble growth, and may hasten resolution;*
  - *if air tables are used, decompression is made more difficult as nitrogen load is increased;*
  - *with increasing duration of symptoms, the recompression depth effect is of less value.*

Delay to recompression

Once a manifestation of DCS has developed, the subsequent progress may be more related to the speed of recompression than the specific treatment table selected. With prompt treatment the destructive tissue distension effects of bubbles are lessened, as are the effects of ischaemia and the chemical and cytological reactions to the bubble.

Even using air tables, Rivera[3] demonstrated that if initial treatment was administered under 30 minutes there was a 95 per cent probability of relief. This falls to 77 per cent if the delay exceeds 6 hours. The delay between the dive, the development of symptoms and the presentation for treatment allows the clinician to assess the clinical importance and propose more rational therapy.

The approach to the patient who presents 30 minutes after a dive with an ascending paralysis is different to the patient who presents 48 hours after a dive with shoulder pain. The first patient's neurological injury is likely to progress with time, whilst the shoulder pain will not and may undergo spontaneous resolution. The first patient should be treated aggressively, commencing with an USN Table 6, but consideration given to the options of going deeper and changing gas mixtures if early or full recovery is not evident.

Patients who delay seeking medical assistance may benefit from recompression even up to 14 days post injury; however, most diving physicians would not consider deep or saturation tables for these late presentations. Consider the two illustrative case reports 13.1 and 13.2.

RECURRENCE OF SYMPTOMS

The fact that some authority has promulgated a therapeutic table does not make it effective, and there have been many modifications and deletions made to these tables during the professional lives of the author. As a good general rule, if symptoms recur during treatment, both the recompression schedule and the clinical management should be seriously questioned. Attention should be directed to ensure that there has been adequate recompression and supportive therapy, including correct positioning,

CASE REPORT 13.1

This man dived to 18 metres for 60 minutes on scuba, but had not dived for a month prior to this. The isolated symptom of a left shoulder pain developed 5 hours after the dive and had been present for 24 hours before the diver presented for medical treatment.

Diagnosis and treatment: This is not only a mild case of DCS, it is not going to get significantly worse, if the patient avoids further exposure to hypo- or hyperbaric conditions. By the time the medical assessment was made (29 hours post-dive) the tissues will have equilibrated fully with the atmospheric pressure, and thus there will be no significant pressure gradient pushing nitrogen into the bubble. On the contrary, there will be a mild gradient in the opposite direction. The administration of 100 per cent oxygen will enhance this gradient even more.

The authors' approach to such a mild case would be to attempt to relieve the patient's symptoms and perhaps to reduce the possibility of subsequent bone damage, although there is no definite evidence that this is possible with such delayed treatment. A shallow oxygen table (e.g. Comex 12) would be more than adequate for treatment. Others would be satisfied with surface oxygen if recompression were logistically difficult.

CASE REPORT 13.2

This diver had identical symptoms to case report 13.1, but with left shoulder pain. The main difference in the two presentations is that the left shoulder symptoms in case 13.2 followed 10 minutes after a 30-metre dive for 30 minutes with a rapid ascent and omitted decompression. The diver then presented for assistance immediately after the symptom developed.

Diagnosis and treatment: A very different situation exists from case 13.1. Even assuming that the left shoulder pain is musculoskeletal and not referred neurological or cardiac, the likelihood of progression of this case from the theoretical 'minor symptom' to a major case of DCS, is much higher.

First, the symptom developed soon after the dive, and thus it is likely to become worse. Second, more symptoms are likely to develop (remembering that DCS manifestations continue to arise over the next 24 hours). Third, the tissues surrounding the bubbles might well have nitrogen supersaturation pressures of almost 4 ATA. The bubble, existing on the surface, will have a nitrogen pressure of approximately 1 ATA, as the bubble is at the same ambient pressure as the body. Under these conditions, there will be a gradient between the tissues and the bubbles, increasing the size of the latter until the tissue gas tension becomes equated with the bubble gas tension.

The therapeutic approach to this diver is to recompress him to the maximum depth at which 100 per cent oxygen can be used therapeutically, i.e. USN Table 6 at 18 metres, and if a satisfactory response is obtained and maintained, to decompress him from that depth. Recompression treatment at 18 metres will not necessarily reverse the tissue-to-bubble nitrogen gradient and, if other more serious symptoms develop, it may be necessary to recompress him deeper on an inert gas/oxygen. These authors would select a 30-metre 50 per cent Heliox treatment schedule (e.g. Comex 30). At this depth there would be no tissue-to-bubble nitrogen gradient.

rehydration, etc. Reassessment of diagnoses should be made, considering:

- Pulmonary barotrauma and each of its clinical manifestations (see Chapter 6).
- Complications of DCS, affecting target organs.
- Non-diving general medical diseases.

Nevertheless, DCS patients do sometimes deteriorate during recompression therapy. The composition of the breathing mixture should be confirmed, as should the efficiency of the mask seal.

One table (USN 4, RN 54) frequently caused DCS in attendants, who did not even have a nitrogen load to start with. It is difficult to understand how it could then improve patients, at the shallow stops. Oxygen is now used by both patient and attendant from 18 metres, to prevent this. The short air embolism 'Table 5A', which many of us believed to be a contributor to deaths during treatment, has now been removed from the US Navy Manual.

If similar and significant symptoms recur, it must be presumed to be a re-expansion of a bubble, which was not completely removed, and treated accordingly. However, occasionally there may be other explanations, such as:

- The inflammatory tissue reaction to the bubble produced pathology.
- Lipid/platelet/fibrin deposits or emboli.
- Re-perfusion injury.
- Redistribution of gas emboli.

Only the last of these could be expected to respond to recompression therapy, and it would be a great coincidence if it were to reproduce the same symptoms as the original lesion.

Recurrences of the original symptoms, or the development of other serious symptoms, should be looked upon as due to inadequate treatment or the aggravation of the problem by the re-exposure to nitrogen at depth or on the surface. Recurrence of symptoms requires surface oxygen (if mild), hyperbaric oxygenation or a conventional therapy table.

Paraesthesia and other symptoms developing while undergoing recompression therapy may be due to the development of oxygen toxicity (see Chapter 17), and therefore are not necessarily an indication to extend the therapy.

It is not necessary to recompress repeatedly for minor and fluctuating symptoms, unless these have some ominous clinical significance. Minor residual musculoskeletal or peripheral nerve pathology is very common, and chasing these symptoms to obtain a complete 'cure' becomes demoralizing and exhausting for both patient and attendants.

Both recompression and altitude exposure alter blood gases (O_2, CO_2, pH) and may affect these minor symptoms, presumably by affecting marginal ischaemia or nerve irritability from myelin sheath damage. Patients should be reassured that such minor symptoms – often developing for months after DCS – are not uncommon and do not require intervention.

FOLLOW-UP TREATMENTS

Despite aggressive initial recompression therapy (pushing the limits of pulmonary oxygen toxicity if necessary), there will be cases in which there are residual symptoms and signs that can be attributed to the sequelae of bubble damage to the tissues. Hyperbaric oxygen therapy, repeated daily, may be of value in severe and refractory cases. With spinal cord or cerebral damage, it is mandatory to continue with intermittent hyperbaric oxygen therapy until all subjective and objective improvement has ceased. These authors employ oxygen at 9–12 metres for 1–2 hours, then ascend at 3 minutes/metre, without air breaks, daily. Other regimes may be applied, but the use of repeated diving therapeutic tables, such as extended Table 6 (USN) with its hyperbaric 'air breaks', is illogical and has increased complications to both attendant and patient.

CEREBRAL ARTERIAL GAS EMBOLISM

As stated above, recompression treatment of CAGE involved a deep excursion to 50 metres followed by a standard USN Table 6. Most diving physicians now follow the approach as listed above for cerebral DCS, i.e. USN Table 6. If the CAGE is not due to DCS and is a manifestation of pulmonary barotrauma, then other factors should be considered, *viz*:

- there may be little or no inert gas loading in the tissues (making long saturation-type treatments inappropriate and brief deep excursions more valuable);
- the presence of pulmonary tissue damage;
- the possibility of pneumothorax; and
- fitness for further diving and its investigations.

ADJUNCTIVE THERAPY

General medical treatment is required, and this will vary according to the DCS manifestations.

Position

Previously a head-down or Trendelenburg position was adopted in patients suspected of having CAGE in order to prevent re-embolization. The head-down position was originally used to divert emboli from the brain, by the effect of buoyancy. It is no longer recommended because the increased venous return causes increasing intracranial pressure, decreasing cerebral perfusion, aggravating the neurological pathology – as well as increasing the possibility of paradoxical gas embolism through a potentially patent foramen ovale (see Chapter 11). For similar reasons, the legs should not be raised, the patient being maintained supine or in the coma position and advised not to strain or perform Valsalva manoeuvres.

General care

The haematological effects of DCS may aggravate dehydration from immersion and cold-induced diuresis. This increases blood viscosity and reduces blood flow to the major organs. It may require correction, orally or intravenously. Rehydration, with a urinary output of 1–2 ml/kg per hour, should be achieved, preferably with electrolyte fluids.

Hartman's solution, Ringer's lactate or physiological saline is preferable, until the serum electrolytes and plasma osmolarity can be determined. Intravenous colloids may be of value, and low-molecular weight dextran in saline has been used in the past to prevent rouleaux formation, expand the blood volume rapidly and to reduce the likelihood of intravascular coagulation. There are occasional problems with dextran use, however, including fluid overload, anaphylaxis, renal failure and bleeding, and to date no advantage over crystalloids has been reported.

Glucose and other carbohydrate fluids should be avoided if other fluids are available, as cerebral injury may be exacerbated by hyperglycemia.

Urinary catheterization will be required for most spinal DCS, as will careful skin and body maintenance.

Monitoring of vital signs and electrocardiogram, are sometimes indicated and should not cause difficulties in most chambers. Cardioversion is rarely required, and can be performed in some specialized recompression facilities.

Chest X-rays can be performed through the ports or walls of many chambers, but this usually requires practice and pre-planning. Water aspiration and pulmonary barotrauma can both mimic and complicate DCS.

Arterial gases are invaluable in severe cases, and biochemical and haematological studies can be performed on blood taken under pressure.

Drug therapy

As an adjunct to recompression therapy, drugs and intravenous fluids may be used to correct some of the sequelae of DCS. Over the years there have been many such drug treatments recommended, but few have stood the test of time. They are often based on animal experimentation, usually specifically related to very rapid decompressions and are often of more value if administered before the actual decompression accident, or at least very soon afterwards. There is a considerable degree of logic in the use of pharmacological agents to reduce platelet aggregation, microthrombi, haemoconcentration, neurological oedema, etc. Others have been proposed to increase tissue perfusion and/or expedite inert gas elimination. Although the logic is understandable, the clinical value is less than remarkable.

Anti-platelet drugs

There is evidence that anti-platelet agents such as aspirin or dipyridamole, when given prophylacti-

cally, modify platelet action following decompression, though there are no controlled studies to support its use in the treatment of DCS. There are more arguments against the use of aspirin than for it, with the increased likelihood of aggravating inner ear or spinal cord haemorrhagic pathology. Aspirin has a variety of other negative influences on susceptible individuals (such as bronchospasm), and there are reports in animal studies linking aspirin with an increased risk of dysbaric osteonecrosis.

Aminophylline

Aminophylline, probably with other sympathomimetic drugs, may be contraindicated in dysbaric diving accidents – and was removed from the North Sea medical packs. It results in the dilatation of the pulmonary vasculature and a profuse release of bubbles trapped in the pulmonary circuit, to enter the systemic circulation.

Anticoagulants

Heparin and coumarin derivatives have been advocated because of their effect on the coagulation pathway. They were said to be indicated in cases of disseminated intravascular coagulation (DIC) that had no evidence of systemic infarction and bleeding. Anticoagulation is now rarely, if ever, used. Correction of specific coagulation defects seems a more logical approach to the rare complication of DIC in DCS. Anticoagulation can be harmful following the haemorrhagic pathologies of the spinal cord, inner ear and other DCS manifestations.

Anti-inflammatory Agents

There are no adequate clinical trials to support the use of non-steroidal anti-inflammatory drugs (NSAID) or prostaglandins in the treatment of human DCS. It has been postulated that, because of their effect on platelet activation, NSAID use may modify the subsequent activation of the coagulation pathway by bubbles. There is one double-blind, randomized controlled study under way assessing the value of NSAIDs in DCS.

Corticosteroids

The use of corticosteroids has previously been justified on the belief that this class of drugs may reduce cerebral oedema and modify the inflammatory process. This experience came not from treating DCS but from treating cerebral oedema associated with traumatic and vascular brain injury. Just as the use of corticosteroids has now been discredited in brain injury, there are no definitive studies to support its use in DCS brain injury.

However, high-dose methylprednisolone initiated within 8 hours of traumatic spinal injury has resulted in a significantly greater neurological recovery (NASCIS 2 trial[4]) and has rekindled the interest in steroids of many diving physicians. Although to date there are no published trials supporting its use, high-dose methylprednisolone (30 mg/kg bolus followed by 5.4 mg/kg per hour for 23–48 hours) should be considered in refractory early cases of severe spinal DCS. Some of the disadvantages of using this high-dose regime include severe sepsis, haemorrhage, hyperglycemia, anaphylaxis and an increased susceptibility to oxygen toxicity.

Diazepam

Diazepam (Valium®) has also been recommended for use in DCS. It may be of considerable value in reducing the incidence and degree of oxygen toxicity, especially in the serious cases, which require extensive exposure to oxygen under pressure, during transport and after recompression therapy. It is also of considerable value in the occasional patient with a toxic-confusional state, because of the involvement of the neurological system from either DCS or CAGE. These patients can cause much concern, as well as being very difficult to handle in the recompression chamber, and may not tolerate the oronasal mask without an anxiolytic. In the latter circumstance, the dosage must be regulated according to the clinical state of the patient, but otherwise a 10 mg initial dose may be supplemented by 5 mg every few hours, without causing any significant drowsiness, respiratory depression or interference with the clinical picture.

Vestibular DCS may require suppressants, such as diazepam. Others counsel against its use for fear of masking the clinical signs that are used to guide the response to recompression.

Lignocaine

Lignocaine (lidocaine) has been recommended in the same dosage as used for cardiac dysrhythmias, in both cardiac and cerebral DCS (see pp. 148,142), on experimental grounds. When used therapeutically, a beneficial effect of lignocaine has been demonstrated in animal models of air embolism.[5,6] Lignocaine has also been demonstrated to have a cerebral protective effect when used prophylactically in patients undergoing left heart valve surgery.[7] These patients are at high risk of gas emboli as a consequence of their time spent on cardiac bypass. Possible mechanisms for cerebral protection by lignocaine include:[7]

- Deceleration of ischaemic transmembrane ion shifts.
- Reduction in cerebral metabolic rate.
- Modulation of leucocyte activity.
- Reduction of ischaemic excitotoxin release.

Lignocaine is pro-arrhthymic, and is an epileptogenic drug. There are anecdotal reports of its successful use in cases of neurological DCS, but its value in human DCS requires confirmation in clinical trials.

Surface oxygen administration

> *One area that has been relatively overlooked recently is the administration of oxygen at normobaric pressure.*
>
> *Albert R. Behnke (July, 1990)*

The administration of 100 per cent oxygen, carried out by those trained to do so, will often relieve some of the patient's symptoms and may reduce the likelihood of others developing. It is particularly of value prior to subjecting the patient to altitude. Oxygen has been demonstrated to:

- Enhance the inert gas elimination.
- Prevent venous gas emboli, as detected by Doppler.
- Reduce the size of inert gas bubbles.
- Prevent DCS developing.
- Treat developed DCS.
- Prevent recurrences of DCS.
- Possibly improve oxygenation of damaged tissues.

In one series,[8] surface oxygen was shown to be an effective treatment for DCS. Although this was a highly selected population, it did demonstrate the value of surface oxygen, given early and for some hours, in remote areas where recompression facilities are not readily available.

Used in transit, oxygen has been shown by the Divers Alert Network[9] to result in some DCS cures and a reduction of DCS sequelae incidence following recompression.

Although the value of administering 100 per cent oxygen, with intermittent air breaks, is unquestioned, problems do arise with inexperienced personnel. Commonly, an inadequate mask is used; for example, the Edinburgh or other plastic nosepieces which struggle to produce 40 per cent oxygen in the inspiratory gas. Other risks involve the inflammable nature of oxygen and the contribution to oxygen toxicity.

Some diving physicians prefer to use 100 per cent oxygen post recompression therapy to prevent the recurrence of DCS symptoms and thus avoid repeated treatments. The current practice tends towards repeated hyperbaric oxygen treatments for residual symptoms.

UNDERWATER TREATMENT

In-water air treatments

By far the most traditional of the non-chamber treatments of DCS is underwater recompression therapy. In this situation the water (instead of a recompression chamber) exerts the pressure, while an air supply is usually provided by compressors sited on the diving boat. Although this treatment is frequently disparaged, it has often been the only therapy available to severely injured divers, and has had many successes. This was certainly so in those remote localities such as Northern Australia, in the pearl fishing areas, where long periods were spent under water and only standard diving equipment was used.

The failure of DCS to respond to recompression therapy is often related directly to the delay in treatment. Sometimes chambers are not readily available, and for this reason, underwater air recompression was effectively used in Hawaii, with good results, within minutes of symptoms developing. This was also the experience of professional shell divers of Australia, at least until the underwater oxygen became available.

Despite the value of underwater air recompression therapy, many problems are encountered with it. These are well recognized by both divers and their medical advisers, and include the following:

- Most amateurs or semi-professionals do not carry the compressed air supplies or compressor facilities necessary for the extra decompression. Most have only scuba cylinders, or simple portable compressors that will not reliably supply divers (the patient and his attendant) for the depths and durations required.
- Environmental conditions are not usually conducive to underwater treatment. Often the depths required for these treatments can only be achieved by returning to the open ocean. The advent of night, inclement weather, rising seas, tiredness and exhaustion, and boat safety requirements make the return to the open ocean a very serious decision.
- Because of the considerable depth required, hypothermia from the compression of wet suits becomes likely.
- Seasickness in the injured diver, the diving attendants and the boat tenders is a significant problem.
- Nitrogen narcosis produces added difficulties in the diver and the attendant.

The treatment often has to be aborted because of these difficult circumstances, producing DCS in the attendants, and aggravating it in the diver. Underwater air treatment of DCS is not to be undertaken lightly. In the absence of a recompression chamber, it may be the only treatment available to prevent death or severe disability. Despite considerable criticism from authorities distant from the site, this traditional therapy is recognized by most experienced and practical divers often to be of lifesaving value.

Underwater oxygen therapy

The advantages of oxygen over air tables include: increasing nitrogen elimination gradients; avoiding extra nitrogen loads; increasing oxygenation to tissues; decreasing the depths required for the reduced exposure time; and improving the overall therapeutic efficiency. The same arguments are applicable when comparing underwater air and underwater oxygen treatment.

In 1970, this new option was applied to the underwater treatment of decompression sickness. The method developed in response to an urgent need for management of cases in remote localities – remote in both time and distance from hyperbaric facilities. As a result of the success of this treatment, and its ready availability, it became known and practiced, even when experts were not available for its supervision.

The physiological principles on which this treatment is based are well known and not contentious, although the indications for treatment have caused some confusion. As for conventional oxygen therapy tables, it was first applied mainly for the minor cases of DCS, but was subsequently found to be of considerable value in serious cases.

The techniques and equipment for underwater oxygen therapy were designed to make for safety, ease and ready availability, even in medically unsophisticated countries. It is now in widespread use in the Pacific Islands and the northern parts of Australia, as well as the colder southern waters of Australia, where it is now used by abalone divers who sometimes dive in areas difficult to service by conventional transport.

Hawaiians have included a deep air 'dip' prior to the underwater oxygen treatment, in an attempt to either force bubbles back into solution or to allow bubbles trapped in arteries to transfer to the venous system.

Technique

Oxygen is supplied at maximum depth of 9 metres, from a surface supply. Ascent is commenced after 30 minutes in mild cases, or 60 minutes in severe cases, if significant improvement has occurred. These times may be extended for another 30 minutes, if

there has been no improvement. The ascent is at the rate of 12 minutes/metre. After surfacing, the patient should be given periods of oxygen breathing, interspersed with air breathing, usually on a 1 hour on, 1 hour off basis, with respiratory volume measurements and chest X-ray examination if possible.

Whenever oxygen is given, the cylinder should be turned on and the flow commenced, before it is fitted to patients or divers.

Equipment

No equipment should be used with oxygen if it is contaminated, dirty or lubricated with oil.

The equipment required for this treatment includes the following: a G-size oxygen cylinder (7000 l or 220 ft^3). This is usually available from local hospitals, although in some cases industrial oxygen has been used from engineering workshops. Breathing this volume of oxygen at a depth varying between 9 metres and the surface is usually insufficient to produce either neurological or respiratory oxygen toxicity. A regulator, set at the appropriate pressure for the demand valve connects with 12 metres of supply hose. This allows for 9 metres depth, 2 metres from the surface of the water to the cylinder, and 1 metre around the diver. A non-return valve is attached between the supply line and the full facemask. The latter enables the system to be used with a semi-conscious or unwell patient. It reduces the risk of aspiration of seawater, allows the patient to speak to their attendants, and also permits vomiting to occur without obstructing the respiratory gas supply. The supply line is marked in distances of 1 metre from the surface to the diver, and is tucked under the weight belt, between the diver's legs, or is attached to a harness. The diver must be weighted to prevent drifting upwards in an arc by the current.

An air-breathing diver attendant should always be present, and the ascent controlled by the surface tenders. The duration of the three tables is 2 hours 6 minutes, 2 hours 36 minutes and 3 hours 6 minutes. The treatment can be repeated twice daily, if needed.

Overview

It was originally hoped that the underwater oxygen treatment would be sufficient for the management of minor cases of DCS, and to prevent deterioration of the more severe cases while suitable transport was being arranged. When the regime is applied early, even in the severe cases, the transport is often not required. It is a common observation that improvement continues throughout the ascent, at 12 minutes/metre.

Attendant divers are not subjected to the risk of DCS or nitrogen narcosis at this depth, and the affected diver is not going to be made worse by premature termination of the treatment, if this is required. Hypothermia is much less likely to develop, because of the greater efficiency of the wet suits at these shallow depths.

The site chosen can often be in a shallow protected area, reducing the influence of weather on the patient, the diving attendants and the boat tenders. Communications between the diver and the attendants are not difficult, and the situation is not as stressful as the deeper, longer, underwater air treatments.

The underwater oxygen recompression treatment is not applicable to all cases, especially when the patient is unable or unwilling to return to the underwater environment. It is also of less value in the cases where gross decompression staging has been omitted, and the present author is reluctant to administer this regime when the patient has either epileptic convulsions or clouding of consciousness.

One of the common comments in Australia is that this underwater treatment regime is applicable to the semi-tropical and tropical areas (where it was first used), but not to the southern parts of the continent, where water temperatures may be as low as 5°C. There are certain inconsistencies with this statement. First, if the diver developed DCS while diving in these waters, then he is most likely already to have effective thermal protection suits available to him. Also, the duration under water for the oxygen treatment is not excessive, and it is at a depth at which his wet suit is far more effective than at his maximum diving depth. If he is wearing a dry suit, the argument is even less applicable. The most effective argument is that it is used, and often very successfully, in these very areas.

There has been a concern that, if this technique is available for treatment of DCS, other divers may

misuse it to decompress on oxygen under water, and perhaps run into subsequent problems. However, this is more an argument in favour of educating divers, than depriving them of potentially valuable treatment techniques.

It has been argued that this treatment is unlikely to be of any value for those patients suffering from air embolism and pulmonary barotrauma. Such may be the case, or it may not. The treatment was never designed for this, and nor was it ever suggested that the underwater oxygen treatment be used in preference to recompression facilities where they exist, or where they can be obtained.

The underwater oxygen treatment table is an application, and a modification, of current regimes. It is not meant to replace the formal recompression therapy in chambers. It is an emergency procedure, able to be applied with equipment usually found in remote localities, and is designed to reduce the many hazards associated with the conventional underwater air treatments. The customary supportive and pharmacological adjuncts to the treatment of recompression sickness should still be used, if available, and the superiority of experienced personnel and comprehensive hyperbaric facilities is not being challenged. The underwater oxygen treatment is considered as a first aid regime, not superior to portable recompression chambers, but sometimes surprisingly effective and rarely, if ever, detrimental.

The relative value of proposed first aid regimes (underwater oxygen, underwater Heliox, an additional deep descent and surface oxygen treatment) needs to be clarified.

Use of underwater oxygen treatment

Because of the nature of this treatment being applied in remote localities, many cases are not well documented. Twenty-five cases were well supervised

CASE REPORT 13.3

A 68-year-old male salvage diver made two dives to 30 metres for 20 minutes each, with a surface interval of 1.5 hours, while searching for the wreck of *HMS Pandora* about 100 miles from Thursday Island in the Torres Strait. No decompression staging was possible, allegedly because of the increasing attentions of a tiger shark. A few minutes after surfacing, the diver developed paraesthesiae, back pain, progressively increasing incoordination and paresis of the lower limbs.

Two attempts at underwater air recompression were unsuccessful when the diving boat returned to its base moorings. The National Marine Operations Centre was finally contacted for assistance, but it was about 36 hours post-dive before the patient was flown to the regional hospital on Thursday Island. Both the Air Force and the Navy had been involved in the organization, but because of very hazardous air and sea conditions, and very primitive air-strip facilities, another 12 hours would be required before the patient could have reached an established recompression centre (distance 3000 km, 2000 miles).

On examination at Thursday Island, the patient was unable to walk, having evidence of both cerebral and spinal involvement. He had marked ataxia, slow slurred speech, intention tremor, severe back pain, generalized weakness, difficulty in micturition, severe weakness of lower limbs with impaired sensation, increased tendon reflexes and equivocal plantar responses.

An underwater oxygen unit was available on Thursday Island for use by the pearl divers, and the patient was immersed to 8 metres depth (the maximum depth off the wharf). A 2-hour period was allowed at that lesser depth, after which the patient was then decompressed. There was total remission of all symptoms and signs, except for small areas of hypoaesthesia on both legs.

CASE REPORT 13.4

A 23-year-old female sports diver was diving with a 2000-litre (72 ft^3) scuba cylinder in the Solomon Islands. The nearest recompression chamber was 3500 km away, and prompt air transport was not available. The dive depth was 34 metres, duration was approximately 20 minutes, with 8 minutes decompression. Within 15 minutes of surfacing she developed respiratory distress, then numbness and paraesthesiae, very severe headaches, involuntary extensor spasms, clouding of consciousness, muscular pains and weakness, pains in both knees and abdominal cramps. The involuntary extensor spasms recurred every 10 minutes or so.

The patient was transferred to the hospital, where neurological DCS was diagnosed, and she was given oxygen via a facemask for 3 hours without significant change. During that time an underwater oxygen unit was prepared and the patient was accompanied to a depth of 9 metres (30 feet) off the wharf. Within 15 minutes she was much improved, and after 1 hour was asymptomatic. Decompression at 12 minutes/metre was uneventful, and a commercial aircraft subsequently flew the patient to Australia.

before this technique increased suddenly in popularity, and two such cases are described.

There have now been many hundreds of cases of underwater air and underwater oxygen treatments recorded.[10] Apart from the relative paucity of complications, the major lesson learnt was that prompt re-immersion of the diver allowed shorter duration of treatment and complete resolution of DCS manifestations. Many divers so treated resumed diving within days.

MEDICAL ATTENDANTS

General medical treatment is required during the recompression regimes. Patients should not be left unattended in recompression chambers, and especially while breathing increased oxygen concentrations.

First aid and resuscitation techniques are often required, as are accurate clinical assessments – and for these reasons it is desirable to have a trained medical attendant in the chamber. Most hospital-based hyperbaric facilities require their hyperbaric workers to have undergone a formal training program. It may be necessary to consider the possibility of DCS occurring in the attendants, especially when the patient is subjected to oxygen or oxygen-enriched mixtures. The decompression regimes are based on the gas mixtures being breathed by the patient, and not the air being breathed by the attendants.

It is embarrassing to produce DCS in attendants during recompression therapy.

RETURN TO DIVING AND FLYING

When considering a return to diving after an episode of DCS it is important to consider the following:

- Has there been a good response to treatment?
- Are there any residual symptoms and signs attributable to DCS?
- Was the development of DCS consistent with the diving exposure?
- Does the individual have an increased susceptibility to DCS?
- Was there any evidence of associated pulmonary barotrauma?

Evidence supports the existence of bubbles for some days or weeks post DCS and recompression therapy – a function of the slower rate of gas elimination, especially in the presence of bubbles. For this reason these authors recommend a minimum period of four weeks before a return to diving is permitted.

Bubble micronuclei may exist indefinitely within the tissues, only to re-expand with an exposure to an inert gas load.

Patients with incomplete recovery post recompression should be followed-up clinically with appropriate investigations, e.g. brain imaging techniques, electroencephalogram, bone scans, neuropsychological testing as indicated by the clinical condition.

If there has been a less than complete recovery – particularly with neurological involvement – it is recommended that the individual not dive again. Autopsy evidence suggests that DCS may involve greater areas of the brain and spinal cord than detected clinically – a characteristic due to neurological redundancy. A further insult may result in this subclinical damage extending to become clinically evident. Therefore, the second episode of neurological DCS may result in a significantly worse outcome.

If the episode of DCS occurs after a relatively trivial exposure, a cause for this increased susceptibility (pulmonary barotrauma, patent foramen ovale, etc.) should be actively sought and excluded. Evidence of pulmonary barotrauma (see Chapter 6) renders the individual permanently unfit to dive.

Many episodes of DCS occur due to a complete disregard for decompression schedules; an absolute reliance on computer-based decompression models, rapid and frequent ascents and multi-day repetitive diving. Before a return to diving, the patient with DCS should be counselled on safe diving practices.

Safe diving practices

- *Use a decompression schedule which has been tested and has a known and acceptable risk of DCS, e.g. DCIEM tables (see Appendix A, p. 682).*
- *Addition of a depth/time penalty for future diving, i.e. for a dive to 16 metres for 35 minutes use the decompression limits for a 40-minute, 18-metre dive.*
- *Recognize the limitations of decompression computer algorithms.*
- *Restrict diving to two dives a day, with a long surface interval.*
- *Have a rest day after each three days of diving.*
- *Perform slow ascent rates.*
- *Incorporate a 'safety stop' of 3–5 minutes at 3–5 metres on every dive.*
- *Ensure conservative flying after any diving exposure.*

There is a lack of scientific data as to when it is safe to fly or ascend to altitude following an episode of DCS, with recommendations varying from 24 hours to 28 days. The same bubble micronuclei as discussed above may expand with altitude exposure, with a resultant return of symptoms. As many diving destinations are in remote tropical areas, divers with DCS are usually very reluctant and financially inconvenienced if they cannot return home for four weeks.

It is our policy to recommend to DCS victims as long as possible a delay before flying/ascending to altitude, with a preferred minimum of two weeks.

An alternative technique to remove asymptomatic bubbles and micronuclei is to expose the manifestation-free diver to a few 2-hour sessions of breathing 100 per cent oxygen, prior to aviation exposure. A third possibility is to use aircraft in which the cabin pressures are kept at 1 ATA.

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14

Dysbaric osteonecrosis

CHRIS LOWRY

INTRODUCTION[1–4]

Aseptic necrosis of bone has been described in diving lizards (mosasaurs) of the cretaceous period,[5] although the association with human diving may not be entirely germane. In man, infarction of areas of bone associated with exposure to pressure, be it in air or water, has been recognized since the turn of the century. Twynam[6] first suggested a causal relationship with pressure exposure in 1888 in a case report of a caisson worker constructing the Iron Cove Bridge in Sydney, although in retrospect the man appeared to be suffering from 'septic' necrosis.

In 1912, there were 500 cases of decompression sickness reported among the caisson workers on the Elbe tunnel at Hamburg, and nine had bone changes. Bassoe, in 1913, suggested a relationship between initial joint 'bends' and subsequent X-ray evidence of bone atrophy and sclerosis. Taylor, in 1943, noted that several months elapsed between the hyperbaric exposure and the joint symptoms, and that shaft lesions are usually asymptomatic and may be replaced by new bone. Osteonecrosis has been observed following caisson work at a pressure of 117 kPa (less than 12 metres of seawater equivalent), and also for as short a time as 7 hours, divided into two shifts, at 242 kPa.

Osteonecrosis has been known to develop within three months of the diving exposure, and has occasionally resulted from 'once-only' exposures. Three of five men who escaped from the submarine *Poseidon*, in 1931 in the China Sea after being at a depth of 38 metres for 2–3 hours, subsequently developed osteonecrosis.

The first report of osteonecrosis in a diver appears to have been by Grutzmacher in the German literature in 1941,[7] the disease affecting the shoulder joint. Osteonecrosis has frequently been reported in commercial diving fishermen, affecting the hip and shoulder, but it is rare in recreational sport scuba divers.

Various names (Table 14.1) have been given to this disease, but the term 'dysbaric osteonecrosis' (DON) has gained precedence as it clearly distinguishes the causal relationship with pressure from the other myriad causes of bone necrosis.

Table 14.1 *Some synonyms for dysbaric osteonecrosis (DON)*

- Caisson arthrosis
- Caisson disease of bone
- Hyperbaric osteonecrosis
- Barotraumatic osteoarthropathy
- Avascular necrosis of bone*
- Ischaemic necrosis of bone*
- Aseptic necrosis of bone*
- Diver's bone rot
- Diver's crumbling bone disease

*To be distinguished from other causes of bone necrosis.

INCIDENCE

Detailed studies of the incidence were not undertaken until the 1960s. Figures should be considered cautiously, however, because the radiologists or physicians in each survey may have used different radiological techniques and diagnostic criteria. Other factors influencing the results include the difficulty in obtaining adequate follow-up of workers of divers and the different decompression regimes followed.

For example, at the Clyde tunnel, only 241 compressed-air workers were surveyed of a total of 1362, 19 per cent having lesions, half of which were juxta-articular. By 1972, the UK Medical Research Council Decompression Sickness Council Panel had X-rays of 1674 workers of whom 19.7 per cent had positive lesions. Also, in 1972, a study by Jones and Behnke on the Bay Area Rapid Transit tunnelling project in San Francisco revealed no clinical or X-ray evidence of necrosis. All prospective workers had pre-employment X-rays, and those with lesions were excluded. The pressure ranged from 62–248 kPa (9–36 lb/in^2) with only one decompression per day. However, the follow-up period was relatively short.

The incidence of this disorder reported in divers is exceedingly variable, ranging from 2.5 per cent in the US Navy to a doubtful 80 per cent in Chinese commercial divers. Some representative surveys are listed in Table 14.2. The lower incidences are reported in military series where strict decompression schedules are adhered to, as with many commercial diving operations. However, in the usually self-employed diving fishermen of Japan, Hawaii and Australia, the divers undertake relatively deep dives with long bottom times and, sometimes, with inadequate decompression. These divers appear to have the highest risk of all. There is a higher incidence among divers over the age of 30 years, which may reflect increased exposure rather than age *per se.*

The Medical Research Council Decompression Sickness Central Registry has X-rays for nearly 7000 professional divers and reported that there were only 12 cases of subchondral bone collapse, i.e. about 0.2 per cent. Asymptomatic shaft lesions appeared in about 4 per cent.[8] The prevalence of crippling osteoarthritis leading to total joint replacement has been conservatively estimated at more than 2 per cent in Australian abalone divers.[9] The majority of cases in most series involve shaft lesions, which have no long-term significance to health and well-being, except for the rare possibility of malignant change.

Earlier UK studies on professional divers indicated that lesions occurred significantly more among the older males who had longer diving experience, and also who had exposures to greater depths. Only 0.4 per cent of the compressed-air divers, who had never exceeded 50 metres, had these lesions. The helium-breathing divers who did not exceed 150 metres had an incidence of 2.7 per cent, which rose to 7.6 per cent if they had been deeper. There was a definite increase in incidence among saturation divers and those who had had decompression sickness. Approximately one-quarter of the lesions were of a potentially serious nature, closely associated with joints.

Another UK study of caisson workers, with 2200 subjects, showed an incidence of DON of 17 per cent. The lesions were more often in older men, with more exposure to pressure and also correlated significantly with decompression sickness. The incidence rose to 60 per cent for those who had worked for 15 years in compressed air.

Although rare, several cases have been reported in aviators not exposed to hyperbaric conditions. Whether the incidence of bone lesions is related more to the cumulative effects of hyperbaric exposures than to the statistical chance of a single event increasing with multiple exposures is unknown.

The incidence of avascular necrosis of bone, within the general population not exposed to hyperbaric environments, is also not clearly defined.

Dysbaric osteonecrosis is rare in recreational scuba divers who breathe compressed air at depths of less than 50 metres, and who follow the customary decompression tables.

DON is rare in sport divers; at least two cases (both of shoulder disease) have been reported[11,12] although it is likely that there are many other unreported sufferers.

AETIOLOGY AND PATHOGENESIS

The most common cause of aseptic necrosis of the femoral head is fracture of the neck of the femur.

Table 14.2 *Reported incidence of dysbaric osteonecrosis in divers*

Report	Type of diver	Total no.	Percentage positive
Ohta and Matsunaga (1974)*	Japanese shellfish	301	50.5
Fagan and Beckman (1976)	Gulf coast commercial	330	27
Elliot and Harrison (1976)	Royal Navy	350	4
Harvey and Sphar (1976)	US Navy	611	2.5
Wade *et al.* (1978)	Hawaiian fishermen	20	65
Davidson (1981)	North Sea commercial	4422	4.4
Lowry *et al.* (1986)	Australian abalone	108	25
Kawashima and Tamura (1983)*[10]	Japanese shellfish	747	56.4

*The Kawashima and Tamura survey is an extension of the Ohta and Matsunaga survey and they have divers in common.

The necrotic lesions of high-dose steroid therapy, even though multiple and bilateral, often involve the articular surface of the knee and ankle joints, which is virtually never seen with DON. This variation in distribution suggests that the pathogenesis may be different, even if the pathology is identical. Osteonecrosis is also frequently reported in association with those diseases in which there is some disturbance of fat metabolism, e.g. diabetes mellitus, pancreatitis, alcoholism and cirrhosis, Gaucher's disease and hyperlipidaemia. Trauma and steroid administration are the most common associations. Aseptic osteonecrosis may occur without any known risk factors (idiopathic aseptic necrosis), though certain specific isolated-site bone necrosis disorders, such as Legg–Calve–Perthes disease, may be associated with specific systemic or anatomic abnormalities.

It has been postulated that many of these conditions may lead to fat emboli and obstruct end arteries in rigid haversian canals of bone, leading to osteonecrosis. These fat emboli may arise from a

Table 14.3 *Some causes of aseptic necrosis*

- Decompression sickness or dysbaric exposure
- Trauma (e.g. fractured neck of femur, dislocated hip and unrelated fractures)
- Steroids (Cushing's syndrome and steroid therapy)
- Collagen diseases such as lupus erythematosus, rheumatoid arthritis, polyarteritis nodosa
- Occlusive vascular disease
- Diabetes mellitus
- Hyperlipidaemia
- Liver disease (fatty liver, hepatitis, carbon tetrachloride poisoning)
- Alcoholism
- Pancreatitis
- Gaucher's disease
- Gout
- Haemophilia
- Polycythaemia/marrow hyperplasia
- Haemoglobinopathies (especially sickle cell)
- Sarcoidosis
- Charcot joint
- Specific bone necrosis disorders (Legg–Calve–Perthes, Kienbock's, Freiberg's and Kohler's diseases)
- Radiotherapy

fatty liver, coalescence of plasma lipoproteins, disruption of bone marrow or other fat tissue, or a combination of the above mechanisms. Enhanced coagulability may add to blood vessel obstruction.

The exact mechanism of the production of bone necrosis in association with hyperbaric exposure has not been fully elucidated. The most widely held belief is that it is due to the **decompression phase** and represents a delayed or long-term manifestation of decompression sickness (DCS) (see Chapter 12). There is a definite relationship between DON and exposure to inadequate decompression, experimental diving and clinical DCS.

There are, however, numerous variations on this basic concept. One theory is that the infarction is caused by **arterial gas emboli** produced during decompression. Certainly, 'silent' bubbles can be detected by Doppler techniques during clinically apparently safe decompression schedules. Several series show a relationship to either type 1 DCS or total DCS rather than type 2 (serious) DCS which are, perhaps, more likely to be associated with intra-arterial bubbles. There is also a relationship with 'inadequate' decompression and experimental diving.

Others propose that the fat in bone marrow takes up large amounts of nitrogen during longer pressure exposures. During or after decompression, gas is liberated from the fat and its expansion increases intramedullary pressure, compromising blood flow within non-compliant bone cavities.[13] There is probably a critical period of bone ischaemia after which pathological changes become irreversible – osteocytes are known to die after about 4 hours of anoxia. Some affected areas may recover, while others progress to the typical necrotic lesions.

Bubbles have been found in the large venous sinusoids in animal experiments with DCS at post-mortem examination, and these may well have obstructed venous outflow from marrow, leading to areas of infarction. Bubble formation within bony lacunae is also possible following decompression with destruction of osteocytes.

Changes secondary to intravascular bubbles, be they arterial or venous, may then take place – such as platelet aggregation and intravascular coagulation causing further vascular obstruction. Release of fat, thromboplastin and vasoactive substances could also trigger disseminated intravascular coagulation and exacerbate DON.[14] This is supported by the post-dive observation of increased platelet adhesiveness and decreased platelet count after certain dive profiles.

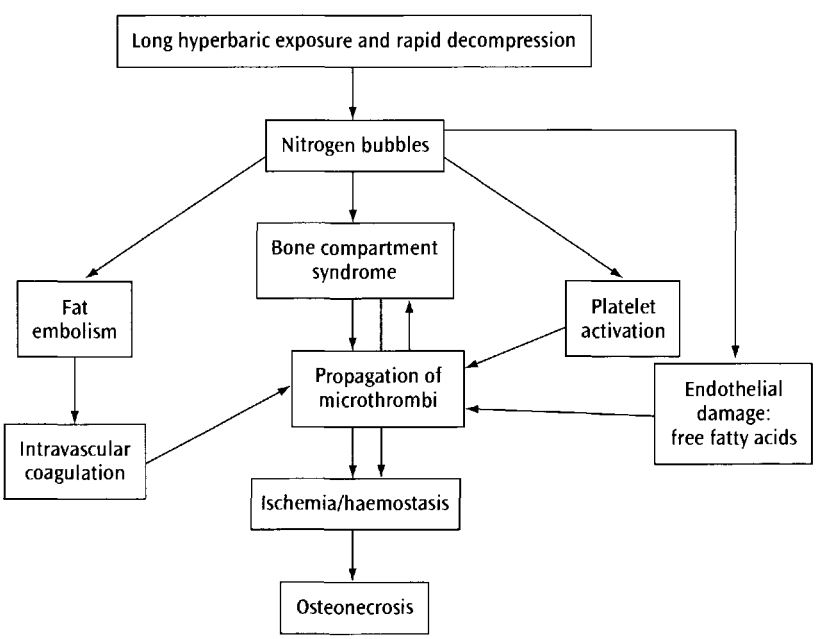


Figure 14.1 *Pathogenesis of dysbaric osteonecrosis. (From Kawashima et al.[15])*

It is possible that a number of factors may combine to produce necrosis in a given situation, and that the aetiology is complex and multifactorial. Experimental evidence is available to suggest that both intravascular and extravascular aetiologies are consistent with the bone pathology, but a direct cause-and-effect relationship has not been proven. The 'silent bubbles', i.e. asymptomatic bubbles during or after decompression, are incriminated in those divers who have had neither DCS nor exposure to hazardous diving practices.

Other theories have been suggested to explain the pathogenesis of bone necrosis. Fat embolism, similar to other causes of aseptic necrosis, has been postulated for dysbaric disease. It is thought that gas (nitrogen) bubbles may disrupt fatty tissue in marrow or elsewhere leading to intraosseous vascular occlusion, either directly or by the initiation of platelet aggregation and intravascular coagulation.

All embolism theories (gas, fat or other) do not adequately explain why other tissues do not appear to be embolized, and why the femur and upper end of the humerus are particularly affected.

Oxygen toxicity is another possible cause for DON, and several mechanisms have been postulated. One suggests that the local vasospastic reaction to high oxygen pressures leads to ischaemia. High oxygen pressures have been shown to cause swelling of fat cells, which may produce increased intramedullary pressure and ischaemia or, if insufficient to obstruct blood flow completely, could inhibit the clearance of gas from the marrow during decompression.

An **osmotic etiology** has also been suggested, incriminating the movement of water into or out of the bone, due to differential gas concentrations causing osmosis. Rapid pressure changes, especially during compression, are thought to be responsible for the large gas gradients. During the compression phase, a gradient exists for all inspired gases across the capillary wall, with the higher partial pressure on the intravascular side. Water would then move into the vascular compartment, and such water movements within the rigid bone structure are thought to lead to local bone ischaemia.

Various animal models have been developed to study the aetiology of DON because of the obvious difficulties in early detection and monitoring of such a capricious and chronic disease. A great deal of research thus involves the experimental induction of bone necrosis in animals such as mini-pigs and mice, but it is difficult to be convinced that these lesions are strictly comparable to those of divers and caisson workers. Studies in larger animals such as sheep who have a large fatty marrow compartment in long bones similar to humans have been more successful. These latter studies in sheep and human post-mortem studies tend to support the raised intramedullary pressure mechanism combined with hypercoagulability.[13,15]

Dysbaric osteonecrosis is thought to be a long-term effect of inadequate decompression.

Any theory must account for the following observations:

- DON may follow a single exposure to pressure.
- Although there appears to be a relationship between decompression sickness and DON, not all divers with DON have a history of decompression sickness.
- Not all divers who suffer decompression sickness develop DON.
- Not all divers at high risk develop DON.

The development of effective strategies for prevention and treatment depends on further research elucidating the precise pathophysiological mechanisms involved.

PATHOLOGY

When examined histologically, the area of necrosis is usually much more widespread than is evident radiologically. Necrosis is first recognized by the absence of osteocytes in the bone lacunae, and this probably starts within a few hours of infarction.

Revascularization then commences from areas of viable bone to form an area of vascular granulation tissue that extends into the infarcted area. Necrotic trabeculae are effectively thickened and strengthened by this new growth, and some lesions even disappear. The revascularization may be arrested before all

areas of necrosis have been invaded. Continuing formation of new bone forms a zone of thickened trabeculae separated from necrotic bone by a line of dead collagen. This area of increased bone bulk is the first sign seen radiologically.

The necrotic trabeculae, not strengthened by the revascularization process, may eventually collapse under a load. It is at this stage that clinical symptoms – that are not necessarily related to recent hyperbaric exposure – may be noted. With lesions near articular cartilage, there is some flattening of the articular surface and, with further load stress, fractures appear in the subchondral bone. The underlying necrosis causes a progressive detachment of the articular surface from its bed. This process resembles that of late segmental collapse, as seen in ischaemic necrosis following fractures of the neck of the femur. Secondary degenerative osteoarthritis often develops in affected joints.

Cases of malignant fibrous histiocytoma, superimposed on DON, may develop in conjunction with the prolonged reparative process set in train by the necrosis.

CLINICAL FEATURES

There may be a history of DCS or repeated inadequate decompression leading to investigation for possible pathology. However, a definite connection between the site of DCS and the site of bone lesions has been notoriously difficult to establish. Early lesions are usually completely asymptomatic, and may currently be detected only by bone scintigraphy (radioactive isotope scan), magnetic resonance imaging (MRI) or radiological examination. However, there are reports of persistent limb pain prior to the development of X-ray changes, in some cases quite severe. Occasional cases seem to have pain in the area of subsequent necrosis dating from the decompression sickness incident. Persistent limb pain may be indicative of a bone compartment syndrome, which may progress to typical DON.[16]

Symptoms of pain and restricted joint movement, usually affecting the hip or shoulder joint, may develop insidiously over months or years, and are due to secondary degenerative osteoarthritic changes.

An increase of 50 per cent in the total mineral content of the bone is necessary before it can be recognized as an area of increased density on X-ray, and these changes may take between three and six months to develop from the time of initial insult. Bone scintigraphy and MRI permit much earlier diagnosis.

There are two major sites for the lesions, which are classified because of their prognostic implications and may be present alone or in combination. Lesions are usually found in the large long bones of the upper and lower limbs, and are classified as juxta-articular lesions and head, neck and shaft lesions.

Juxta-articular lesions

These are also referred to as joint lesions or 'A' lesions, and they are potentially disabling (Figs. 14.2–14.6). They occur near weight-bearing joints where constant pressure is exerted, and may eventually result in the collapse of the articular surface. The most common sites are the hips and shoulders, but rare cases have been reported in other joints, e.g. the ankle. These lesions predominate in caisson workers and divers working in undisciplined or experimental conditions. It is estimated that about one in five articular lesions progress to articular surface collapse, and up to one in five of these proceed to arthroplasty or other surgery.

Head, neck and shaft lesions

Lesions away from the articular surface are also referred to as medullary or 'B' lesions (Figs. 14.7–14.10 and Plate 6). They are usually asymptomatic and are seldom of orthopaedic significance, the most common sites being the shafts of the femur and humerus. They do not extend beyond the metaphysis or involve the cortex of the bone; hence the shaft is not weakened, and pathological fracture is a rare complication. New bone replacement has been observed in these lesions. Their importance is that their presence indicates a greater risk of further DON, though this has not yet been proven statistically.

In assessing the radiological diagnosis of these lesions, it is important to realize that the X-ray will

show only a fraction of the total lesion, and that some bone necrosis areas revealed by scintigraphy never become apparent on the X-rays.

Symptoms

Symptoms referable to juxta-articular lesions depend on the position and severity of the bone damage. Usually there is **pain** over the joint; this may be aggravated by movement and may radiate down the limb. There is often some **restriction of movement**, although a useful range of flexion may remain. In the shoulder, the signs are similar to those of a rotator cuff lesion, i.e. painful arc from 60° to 180° abduction with difficulty in maintaining abduction against resistance. Lifting heavy weights may precipitate the onset of pain. Secondary degenerative osteoarthritis follows collapse of the articular cartilage, further reducing joint movement. The siting of these lesions is approximately in the ratio of femur: humerus, 1:2 to 1:3.

Neoplasia

Malignant tumours of bone (usually fibrous histiocytoma) have been reported in cases of aseptic necrosis, many of which were asymptomatic. The risk appears greatest with large medullary lesions.

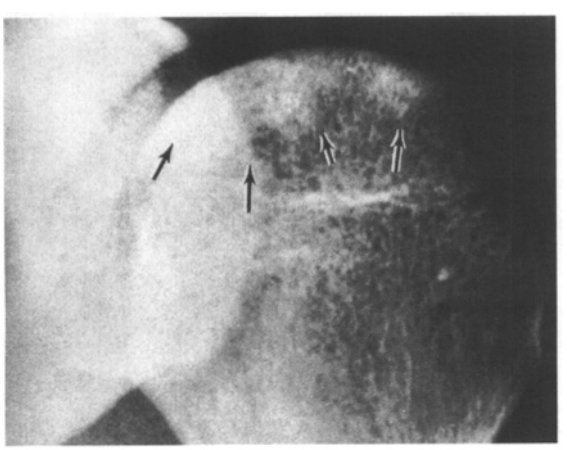


Figure 14.2 *A1 lesions: dense areas, with intact articular cortex. At the top of the humerus are two areas where the trabecular pattern is blurred. The edge of the cortex looks 'woolly'.*

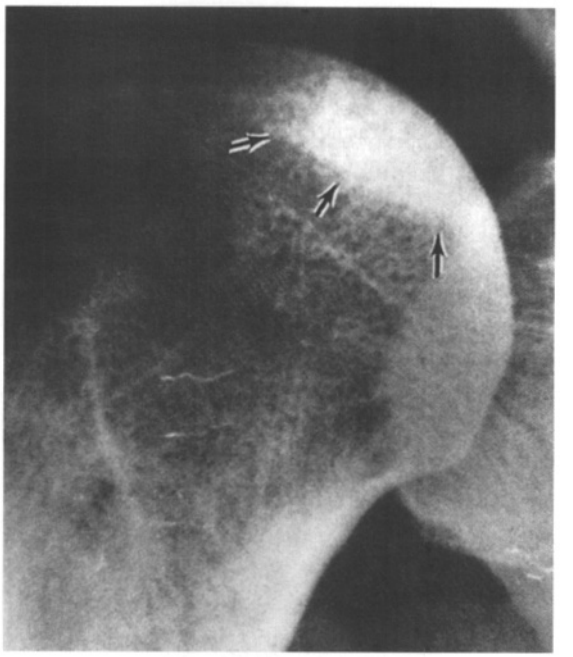


Figure 14.3 *A2 lesion: spherical segmental opacity. Originally called a 'snowcap lesion', this may remain symptomless.*

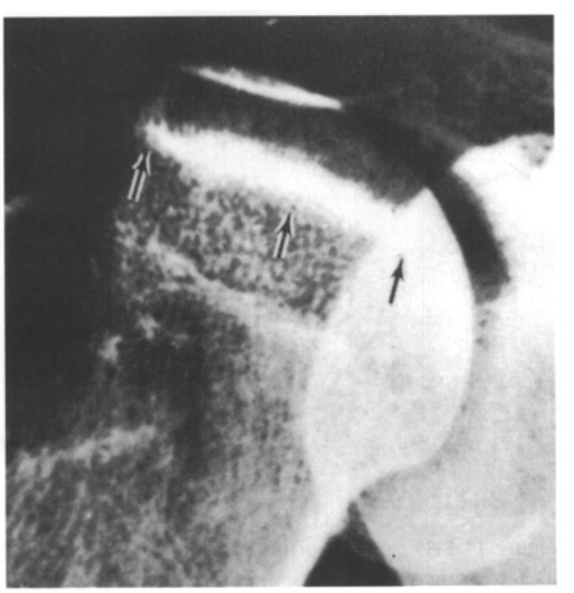


Figure 14.4 *A3 lesion: linear opacity. The dense line marked with arrows represents the lesion. The extremities of such linear opacities characteristically extend to the cortical margin.*

RADIOLOGY AND DIFFERENTIAL DIAGNOSIS[17–19]

The danger of irradiation from X-rays contributing to neoplasia cannot be neglected. With good equipment and technique, the diver has one-third of the annual maximum recommended dose of body irradiation for one long bone series. This amount will increase with poor equipment, extra exposures, slower screens, etc.

Two main questions may arise in early or atypical lesions. First, is the radiological lesion under examination either a variant of normal bone structure, or perhaps a minor dysplasia of bone? Second, does the osteonecrosis have a cause other than the dysbaric environment?

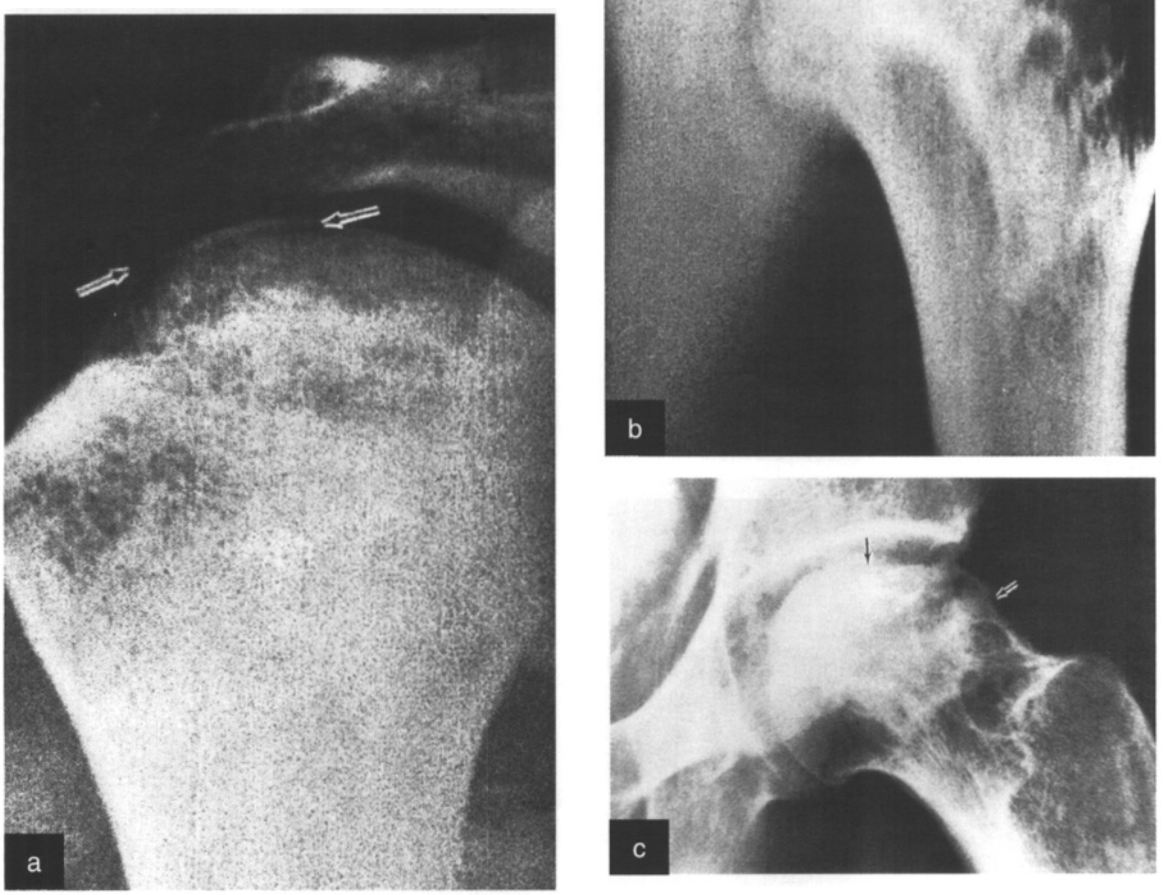


Figure 14.5 *A4 lesion: structural failures. (a) Translucent subcortical band: this lesion (between arrows) is sometimes called a 'crescent sign'. Situated just under the articular cortical surface, the translucent line indicates that a sliver of the cortical surface is about to detach. (b) Collapse of the articular cortex or subchondral depression: the tomogram shows a fracture line (arrows) developing between the sclerotic part of the bone above, which is being depressed into the humeral head, and surrounding bone cortex. (c) Sequestration of the cortex: a loose piece of dead articular cortex has been pushed into the body of the femoral head, causing the latter to appear flattened (arrows).*

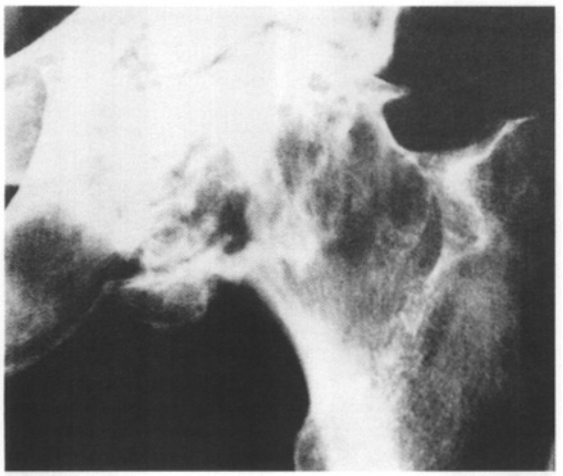


Figure 14.6 *A5 lesion: osteoarthritis. This condition can supervene on any lesion in which disruption of the articular surface has occurred. In osteonecrosis, the cartilage often remains viable so that a joint space of reasonable size often continues to be radiologically visible despite signs of severe osteoarthritis.*

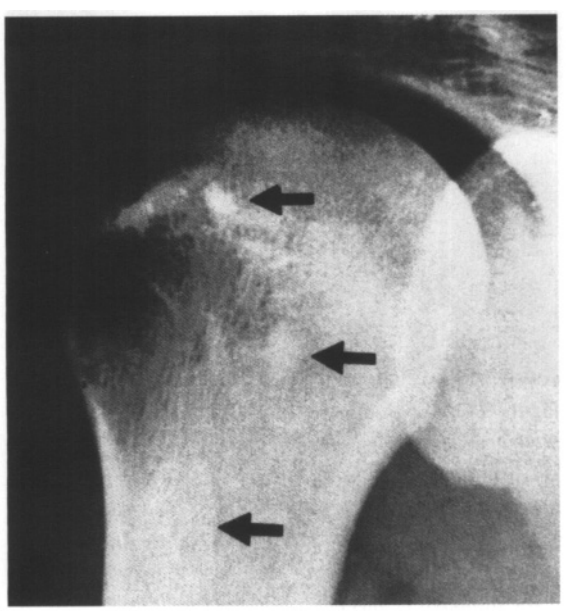


Figure 14.7 *B1 lesion: dense areas. These areas can be seen just at and below the junction of the humeral head and shaft. They are typical of the osteonecrotic lesions seen in such sites, and it is unlikely that they will ever cause disability.*

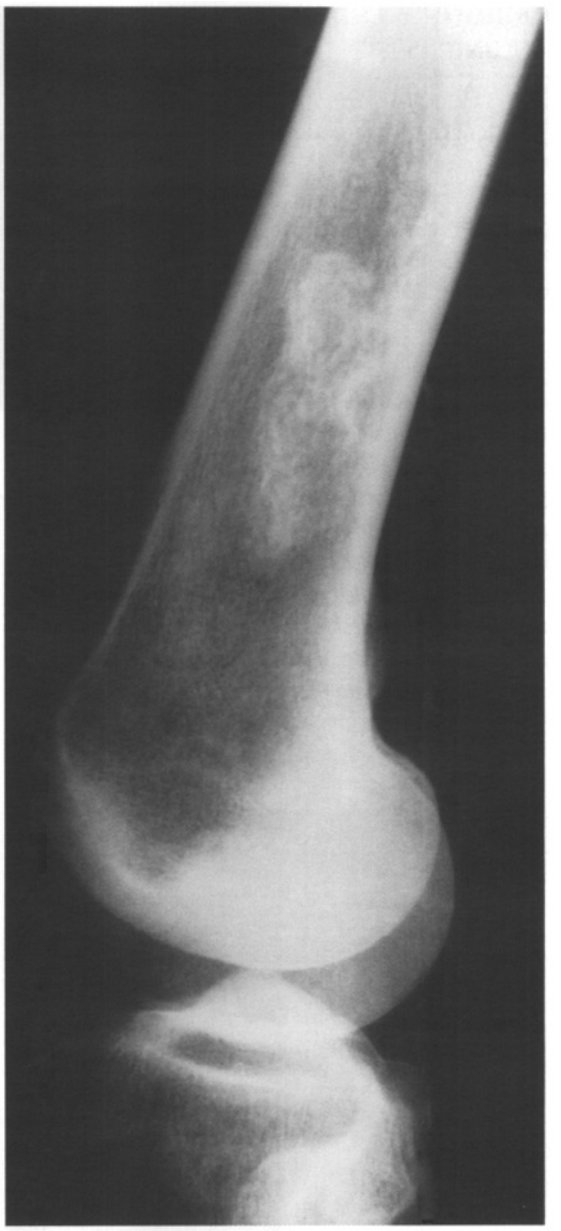


Figure 14.8 *B2 lesion: irregular calcified areas. This condition is commonly seen in divers. Sometimes the appearance is that of rather foamy areas in the medulla at the lower end of the femur, often with a calcified margin. Sometimes femoral lesions have a hardish, scalloped edge around a translucent area. Endosteal thickening frequently accompanies these lesions.*

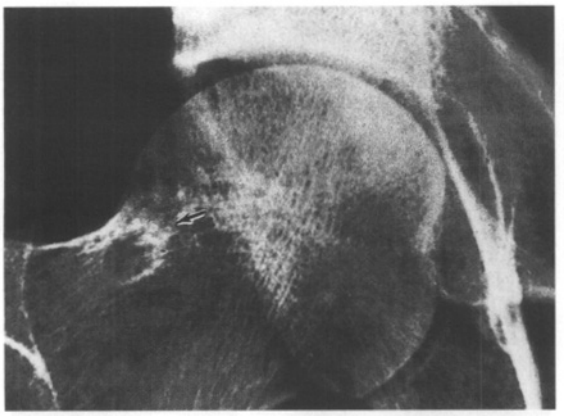
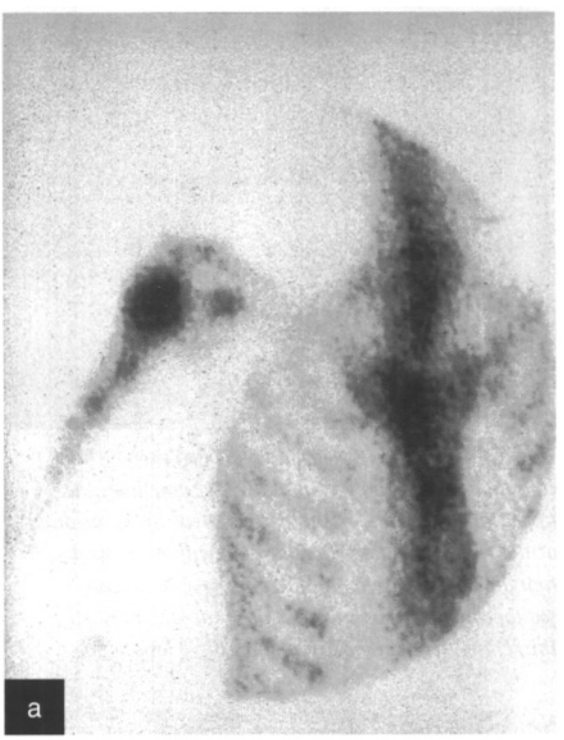


Figure 14.9 *B3 lesions: translucent areas and cysts. A single cyst (arrow) is usually seen in the femoral neck. Sometimes a line of small cysts appears at the point where the hip-joint capsule attaches to the femoral neck. These irregularities may also be found at the junction of the shoulder-joint capsule and humeral neck. Some believe that these multiple lesions are not osteonecrotic, but rather that they relate to past damage at the point of a capsule's insertion into the neck of a bone.*



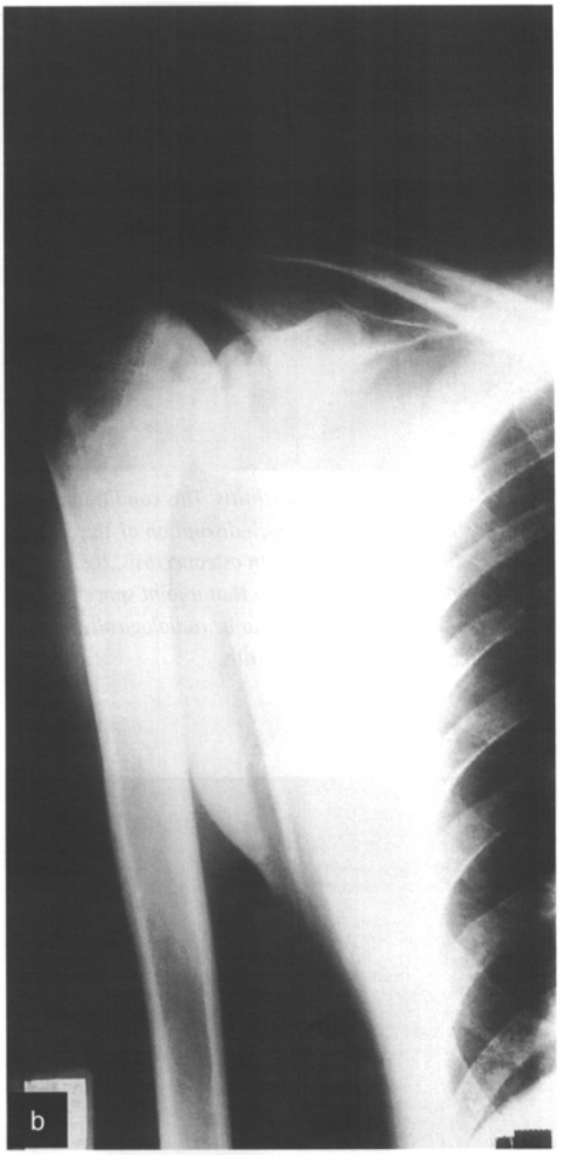


Figure 14.10 *(a) Qualitative scintigram using ^{99m}Tc-labelled methylene diphosphonate (MDP) which shows a 'hot spot', i.e. increased concentration of technetium with increased uptake of MDP, in the right shoulder of a 38-year-old diver who had performed many deep bounce dives on Heliox. The great majority of these were experimental dives, although he has never had treatment for decompression sickness and would only admit to the odd minor discomfort in a variety of joints after dives. Routine screening X-rays at this time showed a normal shoulder. (b) X-ray of the right shoulder 4 months after the first scan. (Photographs courtesy of Dr Ramsay Pearson.)*

Early diagnosis is based on minor alterations in trabecular pattern of bone, resulting in abnormal densities or lucencies. Early detection of asymptomatic lesions may only be verified by serial radiological examinations, showing the progression of the lesion. As considerable skill is required in these assessments, and independent observers from a specialized radiological panel (the members of which then compare their written reports, to exclude subjective errors) perform them. An excellent example was in the UK, where a central registry for cases and X-rays was sponsored as a government body (Medical Research Council)[20]. The classification of lesions is shown in Table 14.4, this being valuable when comparing results between different studies.

Table 14.4 *The UK MRC radiological classification of dysbaric osteonecrosis*

A lesions (juxta-articular)
A1 Dense areas with intact articular cortex
A2 Spherical opacities
A3 Linear opacities
A4 Structural failures
(a) Translucent subcortical band
(b) Collapse of articular cortex
(c) Sequestration of cortex
A5 Secondary degenerative osteoarthritis
B lesions (head, neck and shaft)
B1 Dense areas
B2 Irregular calcified areas
B3 Translucent and cystic areas

The first decision to make is whether the bone is normal.[21] Cysts and areas of sclerosis occur sporadically not only in otherwise normal persons, but also in other diseases. Hence, chance cortical bone defects must be eliminated, and recognition of the normal bone islands is essential. These are dense areas of bone within the cancellous bone structure, but which are sharply defined, round or oval, with the long axis running parallel to the long bone, usually towards the ends of the bone. They have a normal trabecular pattern around them and have no clinical significance. They are thought to develop early in life. It was once believed that bone islands occurred more frequently in subjects exposed to hyperbaric environments, but this is no longer believed to be the case.

Causes of radiological anomaly that may cause confusion in diagnosis include:

- **Bone islands** (see above).
- **Enchondroma** and other innocent tumours: these may calcify, causing an opacity in the shaft of the long bone. Medullary osteochondroma may show foci of calcification, which are more circular, whorled, and in closer apposition than the foci of calcification of DON.
- **Normal variants**: these include sesamoid bones, the shadow of the linea aspera and its endosteal crest.
- **Osteoarthritis**: osteoarthritis, not associated with juxta-articular DON, usually has a reduction of the joint space, with sclerosis of the underlying bone on both sides of the joint. In DON, the cartilage space is not narrowed unless osteoarthritis has supervened.

Other causes of osteonecrosis that are not due to dysbaric exposure must be excluded (see Table 14.3). Both the radiological features and medical history are important in establishing the diagnosis. These causes should be rare in a fit, active diving population who had undergone medical assessment (see Chapters 53 and 54). Among the more important are:

- **Trauma**: a history and its localization to one area may be relevant, but it has also been reported remote from multiple fractures.
- **Alcohol**: a history of heavy consumption or other organ damage may be obtained.
- **Steroid therapy**: the likelihood of this increases with the increased dose of steroid, the minimum being 10 mg prednisone or its equivalent per day for 30 days. Short courses of high-dose 'pulse' therapy have also been incriminated. It is especially noted with rheumatoid arthritis, renal transplantation and asthma.
- **Haemoglobinopathies**: sickle-cell anaemia, thalassaemia and other variants. Diagnosis is made by haematological investigations and by demonstrating lesions in the spine and skull.
- **Specific bone necrosis syndromes**: such as Kienbock's disease (spontaneous avascular necrosis of the carpal lunate), Freiberg's disease (second metatarsal head). The osteochondroses of epiphyseal heads, such as Legg–Calve–Perthe's disease

(hip) and Kohler's disease (tarsal scaphoid), have specific age and clinical parameters.
- **Collagen diseases:** systemic lupus erythematosus and rheumatoid arthritis are associated with a very high incidence of osteonecrosis of the hip, both with and without steroid therapy.

The initial diagnosis of DON must be reasonably certain because it has serious implications for the professional diver. Solitary lesions especially require careful assessment, whereas multiple lesions make the diagnosis easier.

The radiological changes are relatively late manifestations. The first radiological signs may be noted within three to six months, but may take much longer, perhaps even years. The density usually increases, due to an overall increase in the amount of calcification present. Apparently, as a result of the reactive changes to the presence of dead tissue, new bone is laid down on the surface of the dead bone.

The pathological lesion may never produce radiological changes, or may take some time to become apparent. A ten-year radiologic follow-up of 15 caisson workers revealed lesions in previously normal areas and worsening of known lesions despite cessation of further hyperbaric exposure.[22] Autopsy cases reveal that the pathological areas are often far more extensive than the radiological demarcation. Diagnostic radiological parameters include the following:

- Juxta-articular lesions (A lesions)
  - dense areas with intact cortex (usually humeral head).
  - spherical opacities (often segmental in humeral head).
  - linear opacities (usually humeral head).
  - structural failure showing as transradiant or translucent subcortical bands (especially in the heads of the femur and humerus), and often collapse of articular cortex with sequestration.
  - secondary degenerative arthritis with osteophyte formation.

There is usually no narrowing of the joint spaces until later stages. These lesions appear to be quite different from other causes of avascular necrosis.

- Head, neck and shaft lesions (B lesions)
  - dense areas, usually multiple and often bilateral, commonly in the neck and proximal shaft of the femur and humerus; these must be distinguished from normal 'bone islands'.
  - irregular calcified areas in the medulla; these are commonly seen in the distal femur, proximal tibia and the proximal humerus, but they may be bilateral.
  - translucent areas and cysts, best seen in tomograms of the head and neck of the humerus and femur.
  - cortical thickening.

Emphasis is on minor variations of trabecular structure, and special radiographic techniques combined with skilled interpretation are required. Cylinder cone and tomography may be used, while computed tomography or bone scintigraphy may clarify a questionable area.

Dysbaric osteonecrosis and pregnancy have certain features in common:

- *There may be no awareness of the condition until it is too late.*
- *There is sometimes a degree of doubt regarding the aetiology.*
- *It cannot be detected radiographically until three months after the causal incident.*
- *It is feared much more than experienced.*
- *Precautionary techniques usually, but not always, work.*

OTHER INVESTIGATIONS

The value of the plain X-ray in early diagnosis is being questioned, and other imaging techniques are being increasingly used.

Imaging techniques

BONE SCINTIGRAPHY ('BONE SCANS')

This now has an established role in the early detection of bony reaction to osteonecrosis, before they are seen on X-rays. Any lesion, which stimulates bone formation, will be shown up as a 'hot spot' by the radioactive bone-seeking tracer, on the scintigram. ^{99m}Tc-labelled Osteoscan or ^{99m}Tc-labelled

methylene diphosphonate (MDP) may be injected intravenously and serially imaged with a gamma camera. Following up the scintigraphy 'lesions' produced in animals as early as two to three weeks after decompression, with autopsy three months after the event, show that these lesions have a pathological counterpart of necrotic bone with osteogenesis – even though X-ray changes still had not developed in most cases.

Similar findings in humans with biopsy or radiological follow-up indicate that the 'hot spots' occur far earlier and are more numerous than the radiological changes. These 'hot spots' may resolve with no apparent radiological change or may disappear despite classic radiological findings.

Bone scintigraphy in early lesions is thus much more sensitive than radiology but has low specificity, as any bone reparative reaction will be detected, no matter what the cause.

Single position emission computed tomography (SPECT)

This is said to improve specificity, as sections can be obtained in three planes, thus eliminating over- or underlying activity plane of reference. 'Cold' areas occurring immediately after occlusion of blood supply may be detected. SPECT can be performed after the bone scan; it thus does not increase the radiation dose.

Computed tomography (CT)

This provides greater definition revealing both structural collapse and areas of new growth. CT scans may help in the diagnosis of early or doubtful changes on plain X-rays. It is essential if some of the surgical techniques, such as rotational osteotomy, are being contemplated.

Magnetic resonance imaging (MRI)

This can detect necrosis of marrow fat within two to four days of the ischaemic episode, and thus offers the best opportunity for early diagnosis. MRI studies may indicate far greater necrosis than conventional plain radiography. The technique can also reveal bone lesions at other sites when a lesion has been detected on plain X-ray. MRI of the shoulder joint has been suggested as the best surveillance technique for professional divers who are exposed deeper than 15 metres.[23]

Bone-imaging techniques

- *Plain radiography*
- *Scintigraphy*
- *Computed tomography*
- *Magnetic resonance*

Invasive investigations

Invasive investigations have been undertaken to aid in earlier diagnosis, and guide to therapeutic intervention. These techniques include arteriography, intraosseous venography, intramedullary pressure measurement and core biopsy. The latter three are often combined in a technique described by Ficat[24] as 'functional exploration of bone'. Although some claim that intramedullary pressure elevation is an indication of osteonecrosis, others report a wide range of normal values.

PREVENTION

Early recompression of experimental limb bends in sheep prevents progression to DON.[25] This finding

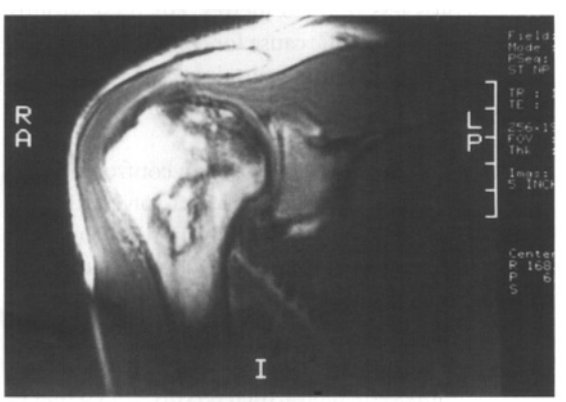


Figure 14.11 *Magnetic resonance imaging of the right shoulder: on these slightly 'TI-weighted' images there is evidence of marked distortion of the marrow signal with areas of necrosis indicated by the dark signal. There is also cortical irregularity. This process involves the articular surface of the glenohumeral joint.*

validates the clinical practice of recompression of all cases of DCS to reduce long-term damage.

Early recognition is imperative, and the following investigations are recommended for all professional divers exposed to frequent hyperbaric conditions at depths greater than 15 metres.

- Baseline long bone X-rays.
- CT or MRI examination in doubtful plain X-ray findings, or to define extent of lesions.
- Bone scintigraphy for any minor arthralgia or bursitis after pressure exposure.
- X-ray examination (baseline) and bone scintigraphy at one to two weeks and six months after decompression sickness.
- MRI, as an alternative to baseline X-ray examination.
- Periodical MRI in high-risk groups.

Follow-up surveillance may be carried out by bone scintigraphy (after initial baseline X-rays). Although scintigraphy has a lower specificity, it detects changes earlier, is more sensitive, and exposes the diver to less radiation. Follow-up long bone X-rays may be required if scintigraphy is not available.

Image-guided biopsy of suspicious lesions (or even surgical biopsy) may be appropriate in special circumstances.

DON is rare in recreational scuba divers who follow decompression tables and use only compressed air to depths less than 50 metres. For these groups, unless there is a specific cause for concern, serial radiological investigation is certainly not warranted, because of the unnecessary irradiation hazards and expense.

The problem of what to do when confronted with an asymptomatic B lesion is not yet solved. If the B lesion is thought to be provoked by non-adherence to established diving tables, these should clearly be followed in the future. Under these conditions, it is assumed that the B lesion is induced because of excessive provocation. If the diver has adhered to normal decompression tables, then it is presumed that he is particularly predisposed to DON, and diving should be restricted to shallow depths. It is generally accepted that the need for decompression should be avoided, as should experimental or helium diving. Doubtful cases should be treated as if positive, until further radiological assessment clarifies the issue.

If a juxta-articular lesion is present, then all exposure to compression should cease. Heavy work and sporting activities that may put unnecessary stress on the joint should be avoided. If the diver can restrict himself to less than 12 metres, and the work is not physically stressful, the medical adviser could be more tolerant.

TREATMENT

Although healing is seen histologically, the possibility of resolution of radiologically positive asymptomatic lesions is controversial. There is at least one case of aseptic necrosis of the hip (which may have been diving and/or steroid induced), which resolved symptomatically and showed MRI improvement with a long course of hyperbaric oxygen therapy.[26] Nevertheless, the treatment of juxta-articular (A) lesions should be based on the fact that DON often progresses through the stages outlined below. The asymptomatic head, neck and shaft lesions require no active therapy.

Surgical treatment

Surgical treatment of disabling aseptic osteonecrosis must be based on the aetiology, which may have a rational basis in DON that is obviously absent in idiopathic disease. Attempts at curative treatment relate to idiopathic osteonecrosis. It is not known if extrapolation of that experience to DON is valid.

Most experience is with idiopathic osteonecrosis of the femoral head – the site at which osteonecrosis produced the most devastating disability – and also the most common site. Some procedures are also applicable to the shoulder joint.

The type of treatment is determined by the staging of the disease process (as described by Ficat[24]), the age of the patient, and the joints involved.

Staging

Staging of the disease process is as follows:

0 Asymptomatic, pre-radiological (i.e. high index of suspicion confirmed by raised intramedullary pressure or positive scan).

1 Symptomatic, pre-radiological.
2 Symptomatic, radiological pre-destruction.
3 Collapse of articular surface.
4 Destruction of joint.

Curative Treatment

Core decompression has its advocates, but the value of the procedure is still questionable. If accepted, it is indicated for stages 0–2. The results are, as expected, better for the earliest stages. A **vascularized fibular graft** procedure has been used for stage 0–2 disease, mainly that affecting the hip, but the results are not known.

Reconstructive treatment

When gross damage to the articular surfaces exists, reconstructive techniques offer the best chance of rehabilitation. **Osteotomy** of the femoral neck, either rotation or wedge, endeavours to move the weight-bearing axis away from a localized necrotic area. **Arthrodesis** is possible for a young patient, with destruction of one hip only. Finally, **arthroplasty** is indicated for 'end-stage' joints, especially if the patient is old or the disease is bilateral. Total joint replacement has proved useful in replacing severely affected hip and shoulder joints, but the concern about this form of surgery is that the life of the prosthesis is unknown as it is employed in a relatively young population with a long life expectancy.

RADIOLOGICAL TECHNIQUE

This must be approached in step-wise fashion:

1 Good definition of the trabecular structure of the bone is important.
2 The gonads must be protected from ionizing radiation in young divers by the use of a lead shield, although this may hinder interpretation.
3 The following projections are required:
   – **Shoulder**: an anteroposterior projection of each shoulder joint; a 30 × 25 cm film is recommended. The patient is placed in a supine position with the trunk rotated at an angle of approximately 45° to bring the shoulder to be radiographed in contact with the table. This arm is partially abducted and the elbow is flexed. Centre 2.5 cm below the coracoid process of the scapula, and cone to show as much humerus as possible, bringing in the lateral diaphragms to show only the head and shaft of the humerus. This view should show a clear joint space, and the acromion should not overlap the head of the humerus.
   – **Hip**: an anteroposterior projection of each hip joint; a 30 × 25 cm film is recommended. The patient is placed in a supine position with the feet at 90° to the tabletop. The edge of the gonad protector should be as near the femoral head as possible, but not in any way obscuring it. Centre the cone over the head of the femur, i.e. 2.5 cm below the midpoint of a line joining the anterior, superior iliac spine and the upper border of the pubic symphysis.
   – **Knee**: an anteroposterior and lateral projection of each knee; an 18 × 43 cm film is recommended. Centre at the level of the upper border of the patella. The field should include the lower third of the femur and the upper third of the tibia and fibula.

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15

Inert gas narcosis

CHRIS LOWRY

INTRODUCTION

Inert gas narcosis (IGN) refers to a clinical syndrome characterized by impairment of intellectual and neuromuscular performance and changes in mood and behaviour. It is produced by an increased partial pressure of inert gas. In compressed-air exposure, these changes, which have been observed for over 100 years, are now known to be largely due to nitrogen. The effects are progressive with increasing depth, but not with increasing time at the same depth. The word 'inert' indicates that these gases exert their effect without undergoing metabolic change in the body, rather than inert gas in the biophysical sense.

Similar effects have been described with other metabolically inactive gases such as the rare gases (neon, argon, krypton, xenon), hydrogen and the anaesthetic gases, although at different partial pressures. Xenon is 'anaesthetic' at sea level, but no narcotic effect due to helium has been directly demonstrated at currently attainable pressures.

The 'inert' gas in compressed air is nitrogen, and its effects are also called 'nitrogen narcosis', depth intoxication, 'narks', rapture of the deep (l'ivresse des grandes profondeurs), the latter term coined by Cousteau. The narcosis, although highly variable, places a depth limit to safe diving with compressed air at approximately 40–50 metres. Effective work at greater depth requires the substitution of a less narcotic respiratory diluent such as helium or hydrogen.

Many 'unexplained' scuba deaths are likely to be induced by nitrogen narcosis.

HISTORY

The first recorded description of symptoms suggestive of air intoxication and related to hyperbaric exposure was by Junod, who, in 1835, reported that 'thoughts have a peculiar charm and in some persons, symptoms, of intoxication are present'. He was conducting research into the physiological effects of compression and rarefaction of air. In 1861, J.B. Green observed sleepiness, impaired judgement and hallucinations in divers breathing compressed air at 5.8 ATA, sufficient to warrant an immediate return to the surface. Paul Bert, in 1878, also noted that divers became intoxicated at great depth. In 1903, Hill and McLeod described impairment of intellectual functioning in caisson workers at 5.5 ATA pressure, while Damant, in 1930, likened the mental abnormalities and memory defects observed in men at 10 ATA to alcoholic intoxication, and postulated that it was caused by the high partial pressure of oxygen.

Hill and Phillips,[1] in 1932, suggested that the effects might be psychological due to claustrophobia, or perhaps due to impurities in the air from the compressors.

The Royal Navy appointed a committee to investigate the problems of deep diving and submarine escape, and their report in 1933 contained a section entitled 'semi-loss of consciousness'. Between 7 and

CASE REPORT 15.1

These are divers' descriptions of their experiences at 75–90 metres (250–300 ft) reported by Hill and Phillips (1932).[1]

'You notice the dark more although it may not be darker; the light is a comfort and company. You notice things more if there is nothing to do; I get comfort from seeing the fish, it takes your mind off everything else.'

When asked for a description, an old hand at diving gave the following account:

'You have to be more careful in deep water; in deep water you know that you are concentrating... You think of each heave as you turn a spanner... If you go down with a set purpose it becomes an obsession; it will become the main thing and you will forget everything else.'

He described how he thinks very deliberately; he says:

'I have finished my job, what shall I do next? – Of course, I have finished and now I must go up.'

He described how he was aware of every action:

'If my hand goes out I think of my hand going out.'

He gave the following as an analogy:

'If I saw a thing of value, say half-a-crown, in the street, I would pick it up. Down below I would look at it and think, "What is that, shall I pick it up? Yes, I will pick it up" and then I feel my hand go out.'

A second diver related that:

'I left the ladder determined to get to the bottom. At 250 feet I got a recurrence of the tingling and a feeling of lying on my back. I decided to rest a couple of minutes and then go on. I slid 10 feet and felt I was going unconscious. I made signals to be pulled up and kept repeating them. I lost the use of my limbs and let go everything. While hanging on to the rope I saw my own face in the front glass; it was outside the glass and looked all greenish; I was dressed in my shore-going suit. I heard the order, "Pull the diver up", again and again, as if someone in the suit was saying it. When I got to the DSDC I did not appreciate the oxygen as usual, I wanted fresh air.'

A third diver described how:

'Suddenly I came over rather "funny"; it was a distinct "different" feeling; I stood up, the tank wire in my right hand, and thinking it was a touch of CO_2, I began to breathe deep and hearty, thinking of course that in a couple of minutes I would be able to resume work. Then I seemed to go quite limp, a feeling of "no life or energy". This was new and strange to me, whether it was a part of CO_2, I didn't know, because I had never experienced a real dose of CO_2; anyhow, after stopping and doing the drill for CO_2, I thought I would be alright, but suddenly something definitely seemed to say "snap" inside my head and I started to, what I thought, go mad at things.'

A description of an interview of the above diver after an aborted deep dive:

Practically no hypnoidal effort was required to produce the horrors of that morning's dive, and the picture of stark, mad terror....left an impression which is very difficult to describe. The impression was of sitting in the stalls and watching the acting of Grand Guignol. To such a pitch did he arouse his emotions that he clawed his face to remove the imaginary face-glass and tore his clothes which he mistook for his diving suit.

11.6 ATA divers answered hand signals, but in many cases failed to obey them. After returning to the surface, they could not remember the events of the dive. It was noted that all divers regained full consciousness during the return to 1 ATA. The report also noted great individual variation in divers' reactions, but was unable to elucidate the problem.

It was not until 1935 that Behnke *et al.*[2] proposed the now generally held theory of the cause of this compressed-air intoxication. They stated that the narcosis was due to the raised partial pressure of the metabolically inactive gas, nitrogen. At a depth of 30 metres (4 ATA), compressed air produced a state of 'euphoria, retardation of the higher mental processes and impaired neuromuscular coordination'. This effect was progressive with increasing pressure, so that at 10 ATA stupefaction resulted, while unconsciousness developed between 10 and 15 ATA. These authors also invoked the Meyer–Overton hypothesis (see Aetiology, p. 190) to relate the narcotic effect to the high ratio of solubility of nitrogen in oil to water.

It was not long after this major breakthrough that Behnke and Yarbrough reported that the substitution of helium for the nitrogen in compressed air eliminated that narcosis.

The **nitrogen partial pressure** theory was not universally accepted, and the 1933 Deep Diving Committee Report raised the possibility of carbon dioxide retention being implicated. Case and Haldane, in 1941, reported that the addition of carbon dioxide to compressed air worsened the mental symptoms, although up to 6 per cent concentrations at 1 ATA had little mental effect. Bean, in 1947, demonstrated a reduction in arterial pH during compression and later also showed increased alveolar carbon dioxide concentrations. He explained these changes as being due to reduced diffusion of carbon dioxide in the increased density of the air. He postulated that carbon dioxide was an alternative cause of the depth narcosis, an idea which was later supported by Seusing and Drube in 1961. Buhlmann (1961) felt that increased airway resistance led to hypoventilation and hypercapnia. (These reports are cited in Bennett, 1993; see Recommended reading.)

Rashbass (1955) and Cabarrou (1959) have refuted the carbon dioxide theory, observing signs of narcosis despite methods to ensure normal alveolar carbon dioxide levels. Later work (Hesser, Adolfson and Fagraeus) showed that the effects of nitrogen and carbon dioxide are additive in impairing performance. Normal arterial carbon dioxide and oxygen levels, while breathing air and helium/oxygen at various depths, demonstrate the key role of nitrogen in the production of this disorder, and the relative insignificance of carbon dioxide

CLINICAL MANIFESTATIONS

'Martini's' law: Each 15 metre (50 feet) depth is equivalent to the intoxication of one Martini.

Although there is marked individual variation in susceptibility to IGN, all divers breathing compressed air are significantly affected at a depth of 60–70 metres. The minimum pressure producing signs is difficult to define, but some divers are affected subjectively at less than 30 metres.

The **higher functions**, such as reasoning, judgement, recent memory, learning, concentration and attention are affected first. The diver may experience a feeling of well-being and stimulation similar to the overconfidence of mild alcoholic intoxication. Occasionally, the opposite reaction – **terror** – develops, though this is more probable in the novice who is apprehensive in his new environment. Further

elevation of the partial pressure of the inert gas results in impairment of manual dexterity and progressive deterioration in mental performance, automatisms, idea fixation, hallucinations and, finally, stupor and coma. Some divers complain of a restriction of peripheral visual field at depth (tunnel vision). They are less aware of potentially significant dangers outside their prescribed tasks (perceptual narrowing).

From a practical point of view, the diver may be able to focus his attention on a particular task, but his memory of what was observed or performed while at depth may be lost when reporting at the surface. Alternatively, he may have to abort his dive because of failure to remember instructions. Repetition and drills can help overcome these problems.

Anxiety, cold, fatigue, sedatives, alcohol and other central nervous system depressant drugs aggravate narcosis.

Nitrogen narcosis has often been likened to alcoholic intoxication, especially the euphoria, lightheadedness and motor incoordination. There is some evidence that correlates subjective feelings of alcohol and inert gas narcosis, especially the variation in intensity experienced between individuals.[3] At much greater depths, the parallel with hallucinatory drugs is probably closer.

Some of the reported observations at various depths when breathing compressed air are shown in Table 15.1.

The narcosis is evident within a few minutes of reaching the given depth (partial pressure), and is not progressive with time. It is said to be more pronounced initially with rapid compression (descent); however, the effect is rapidly reversible upon reduction of the ambient pressure (ascent).

Other factors have been observed to affect the degree of narcosis. Alcohol, fatigue, anxiety, cold, reduced sensory input, and oxygen and carbon dioxide disturbances are interrelated in impairing the diver's underwater ability. In experimental conditions, with an attempt to control variables, alcohol and hard work have been shown to enhance narcosis. Moderate exercise and amphetamines may, in certain situations, reduce narcosis, but some studies have shown unpredictable or increased narcotic effects with amphetamines. Increased carbon dioxide and nitrogen tensions appear to be additive in reducing performance, but task learning and positive motivation can improve performance. Frequent or prolonged exposure produces some acclimatization, but this may, rather than true adaptation, be due to a reduction in psychological stress. A recent study[4] showed no behavioural adaptation after brief, repeated exposures to narcotic levels of hyperbaric air. However, air saturation diving allows for safer excursions to greater depths than is possible from surface-oriented diving.

Direct pathological injury to the diver due to high pressure of inert gas is unlikely at less than 10 ATA.

Table 15.1 *Some observations on the effects of exposure to compressed air at increasing pressure/depth*

Pressure (ATA)	Effects
2–4	Mild impairment of performance on unpractised tasks Mild euphoria
4	Reasoning and immediate memory affected more than motor coordination and choice reactions Delayed response to visual and auditory stimuli
4–6	Laughter and loquacity may be overcome by self-control Idea fixation, perceptual narrowing and overconfidence Calculation errors; memory impairment
6	Sleepiness; illusions; impaired judgement
6–8	Convivial group atmosphere: may be terror reaction in some; talkative; dizziness reported occasionally Uncontrolled laughter approaching hysteria in some
8	Severe impairment of intellectual performance Manual dexterity less affected
8–10	Gross delay in response to stimuli Diminished concentration; mental confusion
10	Stupefaction Severe impairment of practical activity and judgement Mental abnormalities and memory defects Deterioration in handwriting; uncontrollable euphoria, hyperexcitability; almost total loss of intellectual and perceptive faculties
>10	Hallucinogenic experiences Unconsciousness

The danger is rather a result of how the diver may react in the environment, under the narcotic influence of nitrogen. Impaired judgement can lead to an 'out-of-air' drowning sequence, with no other apparent cause of death found. The diver affected by IGN may be at increased risk of insidious hypothermia (see Chapter 28) because of decreased perception of cold[5] and decreased shivering thermogenesis.[6]

MEASUREMENT OF CENTRAL NERVOUS SYSTEM EFFECTS

Although suitable and reliable indices of IGN are not yet available, the search continues. Such tests would be useful in: predicting individual susceptibility (diver selection); comparing the relative narcotic

CASE REPORT 15.2

A personal description of nitrogen narcosis by Jacques Cousteau:[7]

We continue to be puzzled with the rapture of the depths, and felt that we were challenged to go deeper. Didi's deep dive in 1943 of 210 feet had made us aware of the problem, and the Group had assembled detailed reports on its deep dives. But we had only a literary knowledge of the full effects of l'ivresse des grandes profondeurs, as it must strike lower down. In the summer of 1947 we set out to make a series of deeper penetrations.

. . .I was in good physical condition for the trial, trained fine by an active spring in the set, and responsive ears. I entered the water holding the scrap iron in my left hand. I went down with great rapidity, with my right arm crooked around the shotline. I was oppressively conscious of the diesel generator rumble of the idle Elie Monnier as I wedged my head into mounting pressure. It was high noon in July, but the light soon faded. I dropped through the twilight, alone with the white rope, which stretched before me in a monotonous perspective of blank white signposts.

At 200 feet I tasted the metallic flavour of compressed nitrogen, and was instantaneously and severely struck with rapture. I closed my hand on the rope and stopped. My mind was jammed with conceited thoughts and antic joy. I struggled to fix my brain on reality, to attempt to name the colour of the sea around me. A contest took place between navy blue, aquamarine and Prussian blue. The debate would not resolve. The sole fact I could grasp was that there was no roof and no floor in the blue room. The distant purr of the diesel invaded my mind – it swelled to a giant beat, the rhythm of the world's heart.

I took the pencil and wrote on a board, 'Nitrogen has a dirty taste'. I had little impression of holding the pencil, childhood nightmares overruled my mind. I was ill in bed, terrorised with the realisation that everything in the world was thick. My fingers were sausages. My tongue was a tennis ball. My lips swelled grotesquely on the mouth grip. The air was syrup. The water congealed around me as though I were smothered in aspic.

I hung stupidly on the rope. Standing aside was a smiling, jaunty man, my second self, perfectly self-contained, grinning sardonically at the wretched diver. As the seconds passed the jaunty man installed himself in my command and ordered that I unloose the rope and go on down.

I sank slowly through a period of intense visions.

Around the 264 foot board the water was suffused with an unearthly glow. I was passing from night to an imitation of dawn. What I saw as sunrise was light reflected from the floor, which had passed unimpeded through the dark transport strata above. I saw below me the weight at the end of the shotline, hanging twenty feet from the floor. I stopped at the penultimate board and looked down at the last board, five metres away, and marshalled all my resources to evaluate the situation without deluding myself. Then I went to the last board, 297 feet down.

The floor was gloomy and barren, save for morbid shells and sea urchins. I was sufficiently in control to remember that in this pressure, ten times that of the surface, any untoward physical effort

was extremely dangerous. I filled my lungs slowly and signed the board. I could not write what it felt like fifty fathoms down.

I was the deepest independent diver. In my bisected brain the satisfaction was balanced by satirical self-contempt.

I dropped the scrap iron and bounded like a coiled spring, clearing two boards in the first flight. There, at 264 feet, the rapture vanished suddenly, inexplicably and entirely. I was light and sharp, one man again, enjoying the lighter air expanding in my lungs. I rose through the twilight zone at high speed and saw the surface pattern in a blaze of platinum bubbles and dancing prisms. It was impossible not to think of flying to heaven.

potencies of different respiratory diluents for oxygen; delineating the role of factors other than inert gas in producing depth intoxication; and monitoring the degree of impairment during practical tasks.

Attempts to quantify the effects of IGN can be roughly divided into two methods. The first is a psychological behavioural approach measuring performance on tasks such as mental arithmetic, memory, reaction time, and manual dexterity. The second relies on observing a change in some neurophysiological parameter. Some representative studies will be discussed to illustrate points.

Behavioural approach

The aspects of behaviour usually studied may be divided into three categories: cognitive ability, reaction time and dexterity. The cognitive functions are the most affected and dexterity the least. One early study measured the performances of 46 men on simple arithmetic tests; reaction time and letter cancellation were measured at pressures from 3.7 to 10 ATA. This demonstrated quantitatively the previously observed qualitative progressive deterioration with increasing pressure of compressed air. It also showed that individuals of high intelligence were less affected. The impairment noted on arrival at the given pressure was exacerbated by rapid compression.

Another study using simple arithmetic tests of manual skill showed that narcosis was maximal within 2 minutes of reaching depth, and continued exposure did not result in further deterioration, but rather there was a suggestion of acclimatization. Muscular skill was much less affected than intellectual performance.

Other studies, involving reaction time, conceptual reasoning, memory and psychometric tests, showed progressive deterioration with increasing pressure.

Narcosis has been measured by tests of intelligence, practical neuromuscular performance, the electroencephalogram and alpha blocking, flicker fusion frequency, visual and auditory evoked responses.

Some work on open-water divers suggested a greater impairment of performance on manual tasks at depth when anxiety was present. Plasma cortisol and urinary adrenaline:noradrenaline (epinephrine: norepinephrine) excretion ratios were used to confirm the presence of anxiety noted subjectively. Divers were tested at 3 and 30 metres at a shore base and in the open sea. Intellectual functions, as assessed by memory test, sentence comprehension and simple arithmetic, showed evidence of narcosis in both 30-metre dives, but the decrement was greater in the ocean dives. This may be due to the greater psychological stress in the open sea. However, these effects have not been reproduced in the laboratory.

Many of these tests have been criticized because the effects of motivation, experience and learning, etc. are difficult to control. A card-sorting test using caisson workers showed some impairment at 2–3 ATA, especially in those who had relatively little exposure to pressure. However, with repeated testing, i.e. practice, this difference disappeared and no loss of performance was noted even deeper than 3 ATA. These experiments were repeated using 80 naval subjects at 2 ATA and 4 ATA breathing air

and helium/oxygen mixtures. The only significant impairment was found at 4 ATA breathing air.

The effects of IGN on behaviour, as measured by the psychologist have been well reviewed by Fowler *et al.*[8] Human performance under narcosis is explained using the 'slowed processing' model. Slowing is said to be due to decreased activation or arousal in the central nervous system, manifested by an increase in reaction time, perhaps with a fall in accuracy. Increases in arousal, such as by exercise or amphetamines, may explain improved performance. Manual dexterity is less affected than cognitive functioning because dexterity requires fewer mental operations and there is less room for cumulative slowing of mental operations (processes). Although memory loss and impaired hearing are features of narcosis, these effects are more difficult to explain (as yet) using the slowed processing model.

Studies of the subjective symptoms of narcosis have indicated that the diver can identify these symptoms, and that they could relate the effect to the 'dose'. Euphoria, as described by 'carefree, cheerful', etc., is only one of these symptoms and may not always be present. Other descriptive symptoms such as 'fuzzy', 'hazy' (state of consciousness) and 'less efficient' (work capability), 'less cautious or self-controlled' (inhibitory state) may be more reliable indicators of effect on performance.

Behavioural studies have cast doubt on some traditional concepts of narcosis. True adaptation to narcosis has not been found in many performance tests. Where adaptation has been found, it is difficult to distinguish learned responses or an adaptation to the subjective symptoms as opposed to physiological tolerance. Carbon dioxide probably has additive and not synergistic effects in combination with nitrogen, and probably acts by a different mechanism. Behavioural studies have not been able, so far, to confirm potentiating effects on IGN of anxiety, cold, fatigue, anti-motion-sickness drugs and other sedatives (except alcohol).

Neurophysiological changes

Attempts have been made to confirm the subjective experiences and obtain objective evidence of performance decrement, with some neurophysiological parameter. The investigations included electroencephalographic records of subjects exposed to compressed air in chambers. Contrary to the expected findings of depression, features suggesting cortical neuronal hyperexcitability were noted at first. These included an increase in the voltage of the basal rhythm and the frequent appearance of low-voltage 'spikes' elicited by stimuli, which do not have this effect at 1 ATA. Experiments, in which the partial pressures of oxygen and nitrogen were controlled, showed that in compressed air these changes are due to the high oxygen partial pressure. If nitrogen/oxygen mixtures containing 0.2 ATA oxygen are breathed, these changes are absent, and the depressant effects of nitrogen are then revealed. These consist of a decrease in the voltages of the basal rhythm and the appearance of low-voltage theta waves.

Blocking of electroencephalographic alpha rhythm by mental activity can be observed in half of the population. The observation that there is an abolition of this blocking on exposure to pressure introduced the concept of 'nitrogen threshold'. It was found that the time (T) to abolition of blocking was inversely proportional to the square of the absolute pressure (T is proportional to $1/P^2$) for an individual, although there was marked variation between subjects. In some persons abolition of blocking was noted as shallow as 2.5 ATA, where no subjective narcosis was evident.

Flicker fusion frequency was investigated in an attempt to obtain a measurement that could be applied to the whole population. Subjects were asked to indicate when the flickering of a neon light, at a steadily increasing rate, appeared continuous. This is termed a 'critical frequency' of flicker. After a certain time at pressure, the critical frequency dropped. The same relationship, T is proportional to $1/P^2$, resulted.

A more direct measure of central nervous system functioning may be obtained by observing the effect of inert gas exposure on cortical evoked potentials. Evoked potentials are the electroencephalographic response to sensory stimuli, and a depression of auditory evoked responses on exposure to hyperbaric air has been shown to correlate with the decrement in mental arithmetic performance under the same conditions. It was therefore concluded that auditory evoked response depression was an experimental measure of nitrogen narcosis. However, other work

was unable to support this hypothesis and concluded that there is a complex relationship between hyperbaric oxygen, nitrogen narcosis and evoked responses.

Auditory evoked response as a measure of narcosis is criticized because of sound alteration with pressure and the ambient noise during hyperbaric exposure. Therefore, visual evoked responses were utilized in an attempt to produce more reliable information. Visual evoked responses as a measure of nitrogen narcosis were studied in US Navy divers, the investigators concluding that there were reliable and significant differences in visual evoked responses, while breathing compressed air at depths, which were not apparent on breathing helium/oxygen. A further study using visual evoked responses during a shallow two-week saturation exposure with excursion dives suggested that some adaptation to narcosis occurred, but was not complete. Reduction of frequency and amplitude of alpha activity when compared to pre- and post-exposure surface levels were also noted. Nevertheless, the value of current methods of measurement of IGN, by the use of neurophysiological changes, is questionable.

AETIOLOGY

Inert gas narcosis is thought to be produced by the same mechanism as general anaesthesia with gases or volatile liquids. Psychological studies suggest that the behavioural effects of all inert gases which produce narcosis are identical. These agents are simple molecules with no common structural feature, and do not undergo chemical change in the body to exert their effect. This suggests that biophysical methods must be involved and most research is based on the hypothesis that the mechanism is the same for all agents (unitary hypothesis of narcosis). Thus, an explanation of the mechanism of IGN awaits the unravelling of how general anaesthetics work. Research in each area is mutually constructive.

Many workers have tried to identify the physical nature of site of action by relating anaesthetic potency to the physical properties of the agents. The closest correlation is with **lipid solubility**. At the turn of the century, Meyer and Overton noted that there is a parallel between solubility of an anaesthetic in lipid and its narcotic potency, i.e. the Meyer–Overton hypothesis. Later, Meyer and Hopf (1923) stated that 'all gaseous and volatile substances induce narcosis if they penetrate cell lipids in a definite molar concentration which is characteristic for each type of cell and is approximately the same for all narcotics'. This means that the higher the oil–water partition coefficient, the more potent the inert gas. The inert gas molecule is thus thought to be taken up by brain lipid and somehow interfere with cell membrane function. However, there are some discrepancies in this approach (Table 15.2): for example, although both neon and hydrogen have been shown to be narcotic, neon appears to be more so. Also, argon is about twice as narcotic as nitrogen but has a similar oil:water solubility ratio. There are also anomalies among the general anaesthetics but, in general, the relationship is much closer than with other physical properties.

The lipid solubility hypothesis has been extended by the **critical volume concept**[9]. Narcosis is said to occur when the inert gas or anaesthetic agent causes

Table 15.2 *Narcotic potencies and physical properties of simple gases*

Gas	Molecular weight	Volume	Solubility in oil at 37°C	Partition coefficient (oil:water)	Relative narcotic potency
Helium	4	2.370	0.015	1.70	0.23
Neon	20	1.709	0.019	2.07	0.28
Hydrogen	2	2.661	0.040	3.10	0.55
Nitrogen	28	3.913	0.067	5.25	1.00
Argon	40	3.218	0.140	5.32	2.33
Krypton	83.7	3.978	0.430	9.60	7.14
Xenon	131.3	3.105	1.700	20.00	25.64

a lipid portion of the cell to swell to a certain volume. Thus, there is a lipid volume change that differentiates the anaesthetized from the unanaesthetized state. Other factors, in particular pressure compressibility of the lipid, will also affect their volume. That some narcotic effects can be reversed by application of increased hydrostatic pressure lends weight to this hypothesis [see also high-pressure neurological syndrome (HPNS), Chapter 20].

Exceptions in both animal and human studies have led to a further refinement – the **multi-site expansion model.**[10] This model postulates that expansion occurs variably at more than one molecular site, and that pressure does not act equally at the same sites. Thus, hydrostatic pressure effects (see Chapter 20) or narcotic effects may predominate.

Human studies using nitrogen, nitrous oxide and sulphur hexafluoride at varying pressures to produce mild to moderate narcosis confirm the positive relationship to lipid solubility, and refute the alternative aqueous hydrate theory.[11]

The physical theories, in general, support the concept that the site of action is a hydrophobic portion of the cell, the traditional view being that this is the cell membrane. Many studies show that membranes are resistant to the effects of anaesthetics and other sites have been sought, such as the hydrophobic regions of proteins or lipoproteins. Recent studies have suggested that the site of action is a protein rather than a lipid, and that narcotics act by competitive binding to specific receptors, affecting synaptic transmission.

Correlation of lipid solubility, molar concentration, and fractional compression or expansion, with measurements of motor and cognitive function is consistent with the multi-site expansion model. It has further postulated that impairment of cognitive function is a result of the inert gas narcotic effect, but impaired motor ability is a consequence of raised hydrostatic pressure.[12]

The site of action of these biophysical perturbations is most likely at a synaptic level, and many studies have investigated inhibitory and excitatory neurotransmitters and receptors in the central nervous system. Neurotransmitters studied include noradrenaline (norepinephrine), serotonin, dopamine, gamma-amino butyric acid (GABA) and glycine. GABA is the most important inhibitory transmitter in the brain. Potentiation of inhibitory pathway synapse receptors (GABA receptors) is suspected to be a major reaction to IGN/anaesthesia, although action at a wide variety of neuronal sites is likely.[13]

Exposure to narcosis raises extracellular dopamine in the area of the brain controlling the extrapyramidal system, at least in rats. This may account for some of the neuromuscular disturbances of IGN. In contrast, dopamine is increased when HPNS (see Chapter 20) is exhibited.[14]

PREVENTION

In its simplest terms, this means avoidance of exposure to partial pressures of inert gas known to produce intoxication. In practice, safe diving on compressed air requires an awareness of the condition and its effect on performance and judgement at depths greater than 30 metres. The maximum depth limit for an air dive should be between 30 and 50 metres, depending on the diver's experience and the task to be performed. Safe diving at a greater depth requires the substitution of a less narcotic agent to dilute the oxygen, such as helium, neon or hydrogen.

There is a firm belief among divers that adaptation to IGN can develop over repeated daily exposures and that one can therefore 'work up' to deep dives. Several studies have shown that, although subjective adaptation can occur, measurement of standing steadiness[4] or reaction time[15] showed no improvement with repeated exposure. As with alcohol, confidence is not matched by performance, possibly compromising safety.

Saturation at depths between 30 and 40 metres is said to allow the development of adaptation. Excursion dives to greater depths can then be made with more safety and improved work performance. A conventional working dive to 100 metres would be inconceivable using air as the breathing medium. However, operational dives may be performed to 100 metres, if the excursion is from a saturated depth of 40 metres. At that depth, the diver becomes acclimatized to the nitrogen narcosis, with a progressive improvement of his job performance, approaching his 'surface' efficiency.

Currently, in deep diving the effect of IGN is avoided by substituting helium, or helium/nitrogen, as the diluent gases for oxygen. Oxygen cannot, of course, be used alone because of its toxicity at high pressure (see Chapter 17), but it can partially replace nitrogen in various Nitrox mixtures. Hydrogen is also being used as a substitute for nitrogen and would be ideal, except for the formation of an explosive mixture with oxygen.

Evidence that helium also has some narcotic effect arises from the observation that HPNS is not the same under hydrostatic rather than helium pressure. It has been postulated that both helium and oxygen need to be considered when calculating the narcotic effects of respired gases under great pressure.[16]

Factors other than narcosis may limit depth/pressure exposures for these gases, e.g. HPNS (Chapter 20) or gas density.

Although amphetamines ameliorate narcotic slowing of reaction time, the use of drugs to reduce narcosis has, as yet, no place in diving. Conversely, divers should be warned of the risks of taking central nervous system depressant drugs which, in the diver, might include alcohol, antihistamines (in 'cold' and 'sinus' preparations) and anti-motion-sickness drugs. These drugs may act synergistically with nitrogen in impairing performance and judgement, but this has only been clearly shown with alcohol.[17]

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16

Hypoxia

CARL EDMONDS

INTRODUCTION

Hypoxia implies an inadequate oxygen (O_2) availability to bodily tissues. The brain, liver and kidney, which require large amounts of energy, are the first affected. Skin, muscle and bone are less vulnerable because of their lower energy requirements. O_2 is used to liberate energy for cellular metabolism.

Aerobic (with O_2) metabolism is much more efficient in the production of biological energy than anaerobic metabolism (without O_2). For example, one molecule of glucose can produce 38 molecules of the energy storage compound adenosine triphosphate (ATP). In the absence of O_2, the conversion of one molecule of glucose to lactic acid produces only two molecules of ATP. Thus, anaerobic conditions (hypoxia) drastically reduce the available energy.

Dry air, at a barometric pressure of 760 mmHg, has an O_2 partial pressure of 160 mmHg. When inspired, it becomes saturated with water vapour at body temperature, by which dilution the O_2 pressure falls to 150 mmHg. Alveolar gas has a lower partial pressure of O_2 than inspired air, as it diffuses into the pulmonary blood, normal levels being in the region of 105 mmHg.

In the blood, O_2 carriage is mainly by attachment to haemoglobin in the red blood cells. In healthy subjects, the tension in arterial blood is slightly lower (100 mmHg) than in the alveoli due to venous admixture. After passage through the tissues the O_2 is utilized and the tension falls to approximately 40 mmHg in mixed venous blood.

O_2 moves passively down partial pressure gradients from the lungs to the tissue cells, where it is an active component of metabolism (Fig. 16.1 and Table 16.1).

The amount of O_2 stored in the body is limited, as are the high-energy phosphate bonds used to store

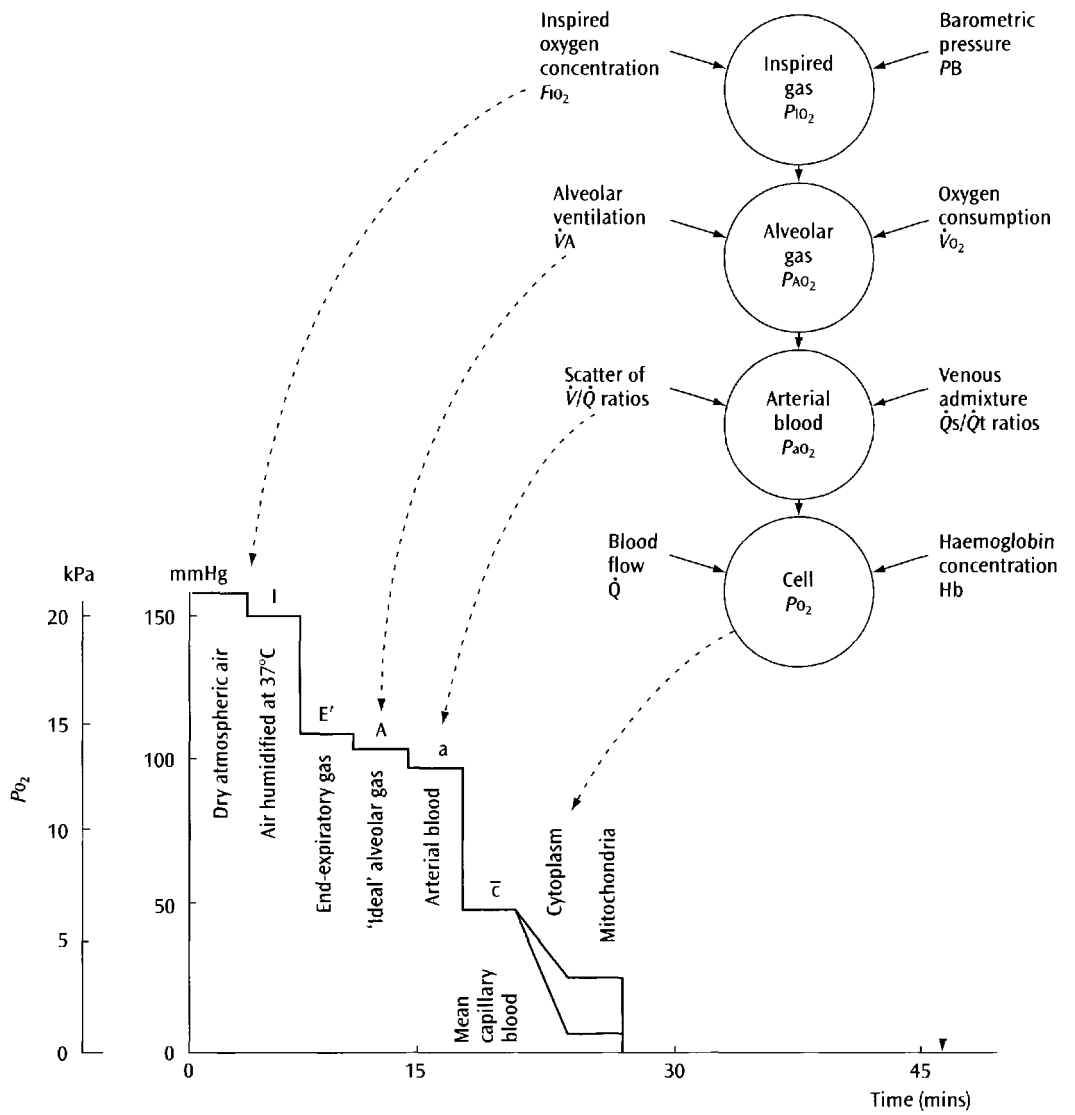

Figure 16.1 *The oxygen cascade: on the left is shown the cascade with Po_2 falling from the level in the ambient air down to the level in mitochondria, which is the site of utilization. On the right is shown a summary of the factors influencing oxygenation at different levels in the cascade. (Redrawn from J.F. Nunn (1987)* Applied Respiratory Physiology, *3rd edition, Butterworths, London.)*

energy. A person breathing air at sea level would hold the following amounts of O_2:

In the lungs	450 ml
In the blood	850 ml
Dissolved in body fluids	50 ml
Bound to myoglobin in muscle	?200 ml
Total =	1550 ml

Not all of this O_2 is readily available for use in vital tissues such as brain and heart.

Basal O_2 consumption is of the order of 200

Table 16.1 *Partial pressures of respiratory gases in normal resting humans at sea level*

Partial pressure	Dry inspired air (mmHg)	Humidified tracheal air (mmHg)	Alveolar gas (mmHg)	Arterial blood (mmHg)	Mixed venous blood (mmHg)
P_{O_2}	159	149	104	100	40
P_{CO_2}	0.3	0.3	40	40	46
P_{H_2O}	0	47	47	47	47
P_{N_2}	601	564	569	573	573

ml/min, but in swimming and diving much higher consumption is possible (up to 3 l/minute). This explains why hypoxia develops so rapidly if respiration has stopped, while exercise continues.

The delivery of O_2 to the cellular level requires an adequate inspired O_2 pressure, adequate lung function, adequate cardiac output and adequate functional haemoglobin for carriage. At arterial O_2 tensions below 60 mmHg, the amount of O_2 given up to the tissue is greatly reduced.

Although impairment of aerobic metabolism of tissues is probably the ultimate mechanism of death in most fatal diving accidents, hypoxia as a primary event is uncommon in conventional scuba diving. It is much more likely with mixed gas and rebreathing equipment.

The diving disorders mentioned here are discussed more fully in their respective chapters.

CLINICAL FEATURES

The physiological associations of hypoxia in general medicine are well known, and will not be discussed here.

Symptoms and signs of hypoxia in diving become more obvious when the arterial O_2 tension drops below 50 mmHg. This corresponds to an inspired concentration at sea level of 8–10 per cent. If the fall in O_2 tension is rapid, then loss of consciousness may be unheralded. With slower falls, an observer may note the diver's in-coordination or poor job performance. Euphoria, overconfidence and apathy have also been reported. Memory is defective and judgement impaired, leading to inappropriate or dangerous reactions to an emergency, and this may also endanger others. The diver may complain of fatigue, headache or blurred vision.

> *There are rarely any symptoms to warn the diver of impending unconsciousness from hypoxia.*

Hyperventilation may develop in some cases, but is usually minimal if the arterial carbon dioxide (CO_2) tension is normal or low.

There are marked individual differences in susceptibility to hypoxia. When combined with hypo- or hypercapnia, a milder hypoxia will impair mental performance. When the CO_2 is controlled, mental performance may not be severely impaired until the alveolar/arterial O_2 tension falls below 40 mmHg. Hypoxia may precipitate or exacerbate other pathological conditions, such as coronary, cerebral or other ischaemia.

Cyanosis of the lips and nail beds may be difficult to determine in the peripheral vasoconstricted 'cold and blue' diver, and generalized convulsions or other neurological manifestations may be the first sign. Masseter spasm is common and may interfere with resuscitation. Eventually, respiratory failure, cardiac arrest and death supervene.

Diagnostic errors may arise because some of the above manifestations are common to nitrogen narcosis, O_2 toxicity and CO_2 retention. The attending physician should also consider cerebral arterial gas embolism and decompression sickness (DCS), should the above features develop during or after ascent by a diver breathing compressed gases.

CLASSIFICATION

Hypoxia ('anoxia') has been classified into four types:

1 Hypoxic.
2 Stagnant.
3 Anaemic.
4 Histotoxic.

Hypoxic hypoxia

This covers all conditions leading to a reduction in arterial O_2 (Fig. 16.2), and a better term might be 'hypoxaemic hypoxia.' This is the common form of hypoxia that is seen in diving, and has a number of causes.

Inadequate O_2 supply

This is due to a decrease in O_2 pressure in the inspired gas, which may result from the use of an incorrect gas mixture or equipment failure. CO_2 retention is not a feature of this hypoxaemia.

Alveolar hypoventilation

This occurs where the amount of gas flowing in and out of functioning alveoli per unit time is reduced. It may be due to increased density of gases with depth, decreased compliance with the drowning syndromes, etc. The extreme example is breath-holding diving. There is associated CO_2 retention.

Ventilation–perfusion inequality and shunt

Perfusion of blood past non-ventilated alveoli results in non-oxygenated blood moving into the systemic circulation (shunt). Lesser degrees of mismatching of perfusion and ventilation also result in arterial desaturation and this may be seen in near-drowning, salt water aspiration syndrome, pulmonary O_2 toxicity and pneumothorax. Inequality of ventilation and perfusion may also occur in pulmonary DCS and pulmonary barotrauma. It is also encountered by deep divers using helium, which promotes lung cooling.

The CO_2 response is variable with ventilation–perfusion disorders, but mild hypocapnia is common if ventilation can be increased by the hypoxic drive.

Diffusion defect

This is due to thickening of the alveolar/capillary barrier, and it may add to the persistent and late hypoxia of near-drowning, and pulmonary O_2 toxicity. CO_2 retention is not characteristic of this hypoxaemia because it diffuses through the barrier much more rapidly than O_2.

There is clearly a great overlap in the different mechanisms by which the various diseases produce hypoxaemia.

Stagnant (ischaemic) hypoxia

This is the term used to describe reduced blood perfusion of tissue, either regional or general, the extreme form being circulatory arrest. Syncope of ascent (see Chapters 6 and 47) is a transitory manifestation due to inadequate cardiac output.

Reduced cardiac output may also be present in DCS. Local ischaemia may result from gas bubbles or gas emboli arising in DCS or pulmonary barotrauma. Many marine venoms induce this effect. Localized ischaemia is the cause of many of the symptoms and signs in those disorders.

Anaemic hypoxia

This refers to any condition in which there is a reduction in haemoglobin, or in its capacity to carry O_2. One cause is traumatic haemorrhage with restoration of blood volume with fluids.

Carbon monoxide poisoning (see Chapter 19), where the formation of carboxyhaemoglobin reduces the O_2 carrying capacity of the blood, is a possible danger with the use of compressed air. The capacity of haemoglobin to carry O_2 is also diminished in the presence of alkalaemia, e.g. low arterial CO_2 tension, and hypothermia.

Histotoxic hypoxia

This refers to the situation where adequate O_2 is delivered to the tissues, but there is a failure of utilization within the cell. Carbon monoxide, as well as the action described above, poisons the cytochrome oxidase enzyme. Histotoxic hypoxia has also been

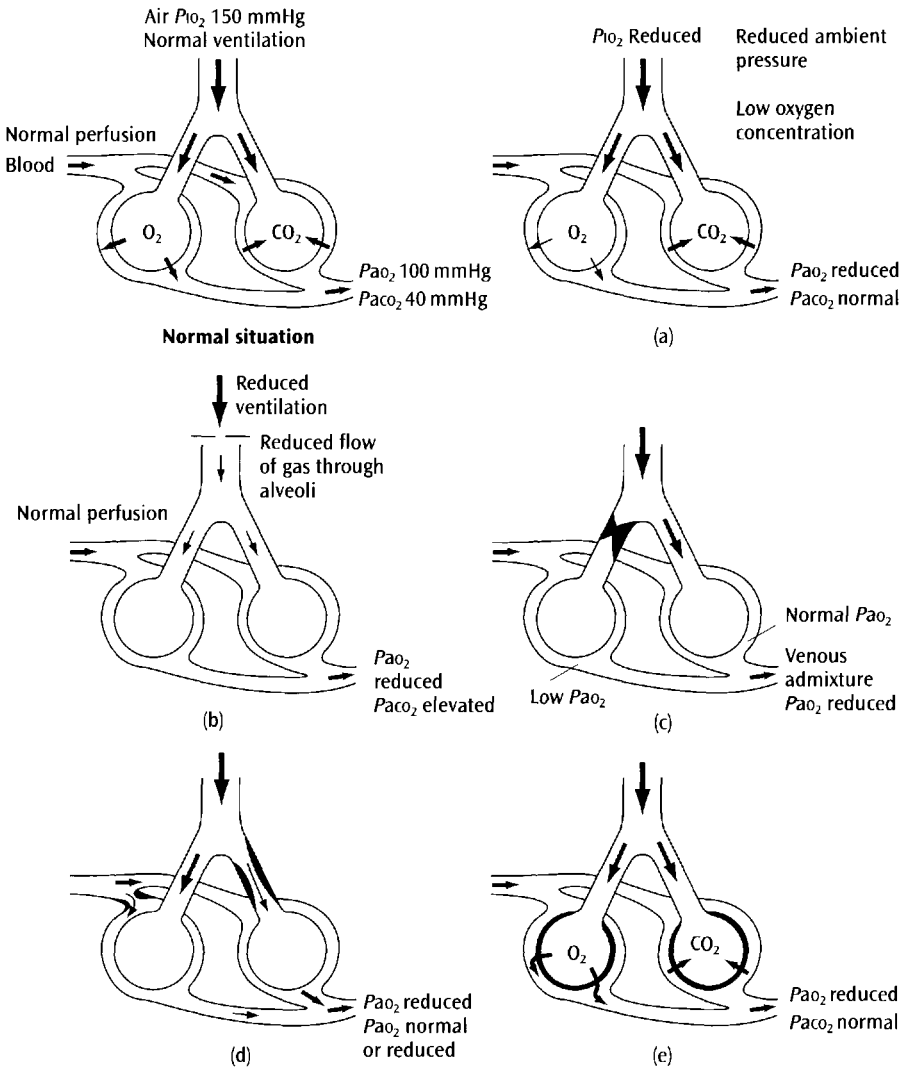

Figure 16.2 *Mechanisms of hypoxaemia (hypoxic hypoxia). (a) Inadequate oxygen supply; (b) alveolar hypoventilation; (c) perfusion of non-ventilated alveoli causing venous admixture; (d) ventilation–perfusion inequality; (e) diffusion defect.* Pa_{O_2} *= arterial oxygen tension;* Pa_{CO_2} *= arterial carbon dioxide tension;* P_{IO_2} *= inspired oxygen tension.*

postulated as a mechanism for inert gas narcosis (see Chapter 15) and O_2 toxicity (see Chapter 17).

HYPOXIA AND DIVING EQUIPMENT

Hypoxia due to inadequate inspired O_2 is due to improper use of (or failure of) the diving equipment, and occurs mainly with the use of closed or semi-closed rebreathing apparatus. Of the following six causative mechanisms, only the first two are possible with open-circuit scuba:

- exhaustion of gas supply;
- low O_2 concentration;
- inadequate flow rates;

CASE REPORT 16.1

Two commercial divers were engaged in making a 110 metre mixed gas dive from a diving bell. The purpose of the dive was to tie in a 6-inch riser. While one diver was in the water at depth working on the riser, the diving bell operator excitedly informed topside that the bell was losing pressure and flooding. The rack operator who was disconcerted by this information opened valves to send gas to the bell. Communication with the bell operator was lost.

The diver who was in the water working on the riser was instructed to return to the bell, which he did. When the diver arrived at the bell, he found the bell operator unconscious and lying on the deck of the bell. The diver climbed out of the water into the bell, took off his Kirby–Morgan mask, and promptly collapsed. When topside personnel realized that they had completely lost communications with the bell, they made ready the stand-by divers. The first stand-by diver was dressed, put on his diving helmet and promptly collapsed unconscious on deck. At this point the bell with the diver and the bell operator was brought to the surface with the hatch open and without any decompression stops. The divers were extricated from the bell and recompressed in a deck decompression chamber. Both the diver and the bell operator died in the deck decompression chamber at 50 metres, of fulminating decompression sickness.

Examination of the rack showed that the rack operator had mistakenly opened a cross-connect valve which should have been 'tagged out' (labelled to indicate that it should not be used). This valve permitted 100 per cent helium to be delivered to the diving bell and the stand-by divers, instead of the appropriate helium/O_2 breathing mixture.

Diagnosis: acute hypoxia and fulminant decompression sickness.

- increased O_2 consumption;
- dilution hypoxia; and/or
- hypoxia of ascent.

Exhaustion of gas supply

The 'out-of-air' situation remains a major cause of diving accidents, despite the existence of contents gauges and reserve supplies, and extensive training.

Low O_2 concentration in the gas supply

Accidental filling of an air cylinder with another gas, such as nitrogen, may result in unconsciousness. Low-percentage O_2 mixtures (10 per cent O_2 or less), designed for use in deep or saturation diving, would lead to hypoxia if breathed near the surface.

Rusting of scuba cylinders has also led to a reduction in O_2 content, causing at least one fatality and several 'near misses'.

CASE REPORT 16.2

MB, a civilian diver, was asked to cut free a rope which was wrapped around the propeller of a diver's charter boat. Because of the very shallow nature of the dive (3 metres maximum), he used a small steel cylinder not often used by divers. After he entered the water, his diving partner noticed that he was acting in a strange manner and swam to him. At this point he was lying on the bottom and was unconscious, but still breathing through his single hose regulator. The diving partner rescued the unconscious diver and got him on deck. His fellow divers prised the

mouthpiece from him and gave him cardiopulmonary resuscitation, and the diver promptly regained consciousness.

On analysis, the gas in the cylinder was found to be 98 per cent nitrogen and 2 per cent O_2. There was seawater present in the interior of the cylinder, together with a considerable amount of rust.

Diagnosis: acute hypoxia due to low inspired O_2 concentration.

Inadequate flow rates

Many rebreathing diving sets have a constant flow of gas into the counter-lung (see Chapters 4 and 62 for an explanation of this equipment). A set designed to use various gas mixtures will have a means of adjusting these flow rates. The flow rate should be set to supply enough O_2 for the diver's maximum requirements, plus that lost through the exhaust valve. The higher the O_2 concentration of the gas, the lower the required flow rate, and vice-versa.

If an inadequate flow rate is set for the O_2 mixture used, then the inert gas, e.g. nitrogen, will accumulate in the counter-lung. Low concentrations of O_2 will then be inspired by the diver.

Other causes include blockage of the reducer by ice, particles, etc.

Increased O_2 consumption

Most rebreathing sets are designed for maximum O_2 consumption between 1 and 2.5 litres/minute depending on the anticipated exertion. Commonly, the maximum O_2 uptake is assumed to be 1.5 litres/minute, but several studies have shown that divers can consume O_2 at higher rates than these. Values of over 2.5 litres/minute for 30 minutes and over 3 litres/minute for 10–15 minutes have been recorded, without excessive fatigue underwater.

This increased exertion may be tolerated due to the cooling effect of the environment and/or greater tissue utilization with increased amount of O_2 physically dissolved in the plasma. The values indicate that it is possible for the diver to consume O_2 at a greater than the often quoted rate under certain conditions. In rebreathing sets, a hypoxic mixture could then develop in the counter-lung due to accumulation of nitrogen (i.e. dilution hypoxia).

Dilution hypoxia

This applies mainly to the O_2 rebreathing sets, and is due to the O_2 in the counter-lung being diluted by inert gas, usually nitrogen. The unwanted nitrogen may enter the system by three methods:

- From the gas supply.
- The diver may not clear the counter-lung of air

CASE REPORT 16.3

AS was diving to 20 metres using a 60/40 O_2/nitrogen mixture in a semi-closed rebreathing system. After 15 minutes he noted difficulty in obtaining enough gas. He stopped to try and adjust his relief valve, and then suddenly lost consciousness. Another diver noticed him lying face-down on the bottom. He flushed the unconscious diver's counter-lung with gas and took him to the surface, after which the set was turned to atmosphere, so that the diver was breathing air. He started to regain consciousness, but was initially still cyanosed. He became aware of his surroundings and did not require further resuscitation. Equipment investigation revealed that CO_2 absorbent activity was normal, but that reducer flow was set at 2 litres/min instead of the required 6 litres/min. This would supply inadequate O_2 for the diver's expected rate of utilization.

before use, thus leaving a litre or more air or nitrogen in it.

- The diver may not clear his lungs prior to using the equipment, e.g. if he breathes into the set after a full inhalation, he may add up to 3 litres of nitrogen to the counter-lung. This may also occur if the diver surfaces and breathes from the atmosphere, in order to report his activities, or for some other reason.

Dilution hypoxia is more likely if O_2 is only supplied 'on demand' (i.e. when the counter-lung is empty), rather than having a constant flow of gas into the bag. As the diver continues to use up the O_2, the nitrogen remains in the counter-lung. CO_2 will continue to be removed by the absorbent, avoiding dyspnoea. Thus, the percentage of O_2 in his inspired gas falls as it is consumed. There is approximately 1 litre of nitrogen dissolved in the body, but the amount that would diffuse out into the counter-lung to cause a dilution hypoxia would be a small contribution.

Hypoxia of ascent

By one of the above mechanisms, the percentage of O_2 being inspired may drop to well below 20 per cent. An inspired O_2 concentration of 10 per cent can be breathed quite safely at 10 metres, as the partial pressure would still be adequate (approximately 140 mmHg).

Hypoxia will develop when the diver ascends sufficiently to reduce this O_2 partial pressure to a critical level. It is therefore most likely to develop at or near the surface.

HYPOXIA AND BREATH-HOLD DIVING

In a simple breath-hold, with no immersion or preceding hyperventilation, the breaking point (the irresistible urge to breathe) is initiated mainly by a rise in CO_2 level, and to a lesser extent by a fall in arterial O_2.

In breath-hold diving, hypoxic blackout is sometimes called breath-hold syncope or shallow water blackout. As the latter term was first used in 1944 to describe loss of consciousness from the use of closed-circuit diving suits, it is best avoided in the breath-holding context. 'Hypoxic blackout' would be a reasonable alternative.

There are two causes for this disorder – hyperventilation and ascent – and as they may occur concurrently, they are often confused. The hyperventilation effect is independent of depth, and may be encountered in 1 metre deep swimming pools, often by children trying to swim greater distances underwater.

Breath-hold divers who train to extend their breath-hold and also dive deep (such as free diving competitors, spear fishing, etc.) risk hypoxia of ascent, with loss of consciousness and subsequent drowning.

With hypoxia there is little or no warning of impending unconsciousness. With increased experience the breath-hold diver can delay the need to inhale by various techniques, without improving the O_2 status. Breath-hold time can be extended (but not with increased safety) by feet-first descent, training (adaptation), swallowing, inhaling against a closed glottis, diaphragmatic contractions, etc.

CASE REPORT 16.4

RAB was diving to 22 metres using a semi-closed rebreathing set with a 40/60 O_2/nitrogen mixture. After 36 minutes he was instructed to ascend slowly. At approximately 3–4 metres he noted some difficulty in breathing, but continued to ascend and then started to climb on board. He appeared to have some difficulty with this, and when asked if he was well he did not answer. He was cyanosed around the lips and his teeth were firmly clenched on the mouthpiece. On removal of his set and administration of O_2 he recovered rapidly but remained totally amnesic for 10 minutes. Examination of his diving equipment revealed that both main cylinders were empty, and the emergency supply had not been used.

Diagnosis: dilution hypoxia and hypoxia of ascent.

One way of avoiding this hypoxia is to inhale 100 per cent O_2 prior to the breath-hold.

Hyperventilation and hypoxia

Craig (1961) observed that swimmers who hyperventilated could stay longer underwater, but then lose consciousness with little or no warning. They were often competing against others or themselves, and often exercising. The hyperventilation extended their breath-holding time, because it washed out a large amount of CO_2 from the lungs, often to half the normal levels.

The build-up of CO_2 is the main stimulus forcing the swimmer to surface and breath. After hyperventilating it takes much longer for this level (the 'breaking point') to be reached. The arterial O_2 pressure drops to a level inadequate to sustain consciousness, if breath-hold time is extended and exercise consumes the available O_2.

It is possible to see the effects of both hyperventilation and exercise in reducing the O_2 level to a dangerous degree, when breath-holding, in the experiment shown in Table 16.2.

Effect	Without hyperventilation	With hyperventilation
Measurements while resting		
BH time (s)	87	146
End-tidal Pco_2 (mmHg)		
Before BH	40	21
Breaking point	51	46
End-tidal Po_2 (mmHg)		
Before BH	103	131
Breaking point	73	58
Measurements while exercising		
BH time (s)	62	85
End-tidal Pco_2 (mmHg)		
Before BH	38	22
Breaking point	54	49
End-tidal Po_2 (mmHg)		
Before BH	102	130
Breaking point	54	43

Source: Craig (1961a).

When the swimmer is concentrating on some purposeful goal, such as trying to spear a fish or retrieve a catch, then he is more likely to ignore the physiological warning symptoms of an urge to breath (due to the rise in CO_2 level in the blood), and delay the breaking point.

The dangers of hyperventilation and breath-hold diving are diagrammatically illustrated in Figure 16.3. This shows that, with prior hyperventilation, the time to reach the 'breaking point' is prolonged. This extra time may allow the blood arterial O_2 to fall to dangerous levels (hypoxic danger zone).

Hypoxia of ascent

The ascent hypoxia was described first in military divers using gas mixtures, and who lost consciousness as they surfaced with low O_2 levels in their rebreathing equipment.

In breath-hold divers, with descent the pressure rises proportionately in the alveoli gases, increasing the available O_2, CO_2 and nitrogen. Some O_2 can be absorbed and utilized, some CO_2 is absorbed and buffered, and some nitrogen is absorbed and deposited in the tissues.

Thus if a diver, having 100 mmHg O_2 and 40 mmHg CO_2 in his alveolar gases, was immediately transported to 2 ATA, the lungs would halve their volume, the O_2 would be 200 mmHg, and the CO_2 80 mmHg. Both would pass into the pulmonary capillary blood – the O_2 to be used and the CO_2 to be buffered. Thus, the O_2 and CO_2 pressures in the alveoli would decrease rapidly. By the time they were both back to 'normal' levels, with O_2 = 100 mmHg and CO_2 = 40 mmHg, then the diver would appear to be in a satisfactory respiratory status – until he ascended. With an expansion of the lungs to twice their size at depth, the pressures in both gases would halve, i.e. the O_2 would drop to 50 mmHg (approaching a potentially dangerous hypoxia level) and the CO_2 to 20 mmHg – if the ascent was immediate.

As ascents do take time, more O_2 will be consumed, extracted from the lungs during the ascent, and the CO_2 would increase towards normal due to the gradient between the pulmonary blood and alveoli.

The drop in O_2 is then able to produce the loss of

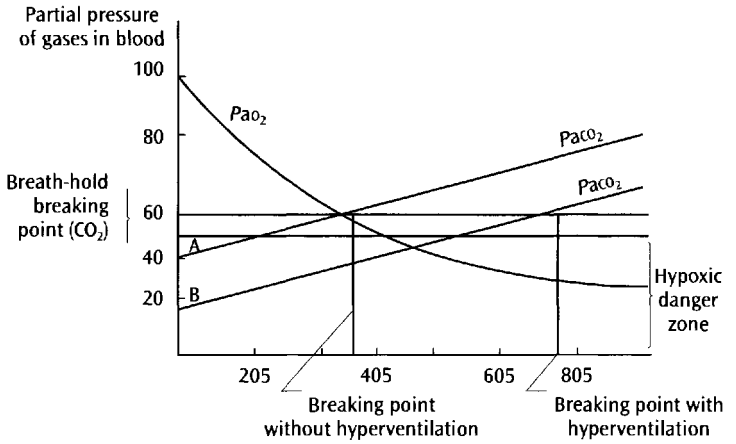

Figure 16.3 *A diagrammatic representation of changes in arterial oxygen and carbon dioxide levels with breath-holding. Point A, without preceding hyperventilation; point B, with preceding hyperventilation. (See Fig. 3.1 for specific data.)*

consciousness, the 'syncope' or 'blackout', commonly noted among spear fishermen. This is referred to as 'hypoxia of ascent'. In deeper dives it becomes more likely, and with some very deep dives, the loss of consciousness may occur on the way to the surface in the top 10 metres (probably an explanation for the 'seven-metre syncope' described by French workers).

Other causes of hypoxia in breath-hold diving include salt water aspiration and the drowning syndromes (see Chapters 21, 22 and 24).

HYPOXIA AND DEEP DIVING

Animal experiments at great pressures and breathing different mixtures are being undertaken regularly to determine the limits of human exposure and thus ocean penetration. Ventilatory capacity is likely to be limited by restricted gas flow or increased work of breathing, both due to the effects of increased gas density, or from pulmonary damage due to the cooling effects on the lungs.

Hypoxia might be expected, due to such factors as an increased 'diffusion dead space', caused by a slowed diffusion of alveolar gases or incomplete mixing of fresh inspired gases and alveolar gases despite adequate inspired O_2 pressure and overall pulmonary ventilation.

The **Choutean effect** (a disputed concept) is an apparent clinical hypoxia despite normal inspired O_2 tension which, at least in goats, is rectified by a slight increase in the inspired O_2 tension (i.e. normoxic hypoxia).

Saltzman has an alternative explanation, suggesting that at greater than 50 ATA there is a decreased O_2 uptake, with decreased pH and increasing acidosis. Thus, there is a block in the utilization or transport of O_2.

MANAGEMENT

First aid management involves the basic principles of resuscitation, establishing an airway, ensuring that there is ventilation of the lungs and that the oxygenated blood is being circulated to the tissues; 100 per cent O_2 should be administered as soon as possible. Further management depends on the aetiology of the hypoxia.

First aid

- *Airway – Head extended, lower neck flexed, jaw forward.*
- *Foreign material – secretions removed.*
- *Breathing*
  - *If breathing, 100 per cent O_2 by mask.*
  - *If not breathing, mouth-to-mouth or mouth-to-nose respiration followed by intermittent positive-pressure resuscitation (IPPR) with 100 per cent O_2 when available.*
- *Circulation – If pulse absent, cardiac massage.*

In many cases there may be an overlap of different causes of tissue hypoxia, and all cases should receive a high inspired O_2 concentration. Recompression or hyperbaric oxygenation may be indicated as a temporary measure to allow the above regimes time to have an effect (see Chapters 6, 13 and 19).

Hypoxic hypoxia

These cases should be given supplemental inspired O_2 or ventilated with 100 per cent O_2 at whatever pressure is needed to ensure adequate arterial O_2 levels. Once these have been achieved, the pressure and percentage of O_2 can be progressively reduced while monitoring arterial gases or tissue O_2 by transcutaneous oximetry.

Stagnant hypoxia

The aim of therapy is to increase perfusion to the affected areas. This may require restoration of total circulatory volume as well as vasodilator drugs, and hyperoxygenation as a temporary measure.

Anaemic hypoxia

Blood loss from trauma may require blood transfusion with packed red cells after crystalloid or colloid resuscitation.

In the case of carbon monoxide poisoning, hyperbaric oxygenation may be life-saving during the early critical period.

Histotoxic hypoxia

This can only be treated by removing the toxic substance and using hyperoxygenation as a temporary measure.

METHODS OF O_2 DELIVERY

There are various devices or apparatus for the therapeutic administration of O_2, and selection of the appropriate mode depends on a number of factors:

- Desired inspired O_2 concentration.
- Need to avoid CO_2 accumulation.
- Available O_2 (i.e. efficiency and economy).
- Need to assist or control ventilation.
- Acceptance of the method by the patient.

Various methods for the administration of O_2 are shown in Table 16.3 (see also Chapter 49). It is important to note that most plastic facemasks deliver less than 60 per cent FiO_2 unless a reservoir bag is incorporated, and this increases the risk of CO_2 retention.

Table 16.3 *Modes of O_2 therapy*

Apparatus	Oxygen flow (l/min)	Concentration (%)
Nasal catheters	2–6	22–50
Semi-rigid mask (e.g. MC, Edinburgh, Hudson, Harris)	4–12	35–65
Venturi-type mask	4–8	24, 28, 35, 40, 50, 60
Soft plastic masks (e.g. Ventimask, Polymask)	4–8	40–80
Ventilators	Varying	21–100
Anaesthetic circuits	Varying	21–100
Demand valves	Varying	21–100
Hyperbaric oxygen	Varying	Varying

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17

Oxygen toxicity

CHRIS LOWRY

Oxygen toxicity is not encountered in routine scuba diving using compressed air. It is a consideration when higher partial pressures of oxygen are used in the inspired gas. Increased oxygen concentration and/or increased ambient pressure lead to higher partial pressures of oxygen. Divers may use high-oxygen gas compositions to reduce inert gas narcosis, reduce decompression obligations or to prolong underwater time.

Central nervous system toxicity, manifested by convulsions, is potentially lethal in the diver.

Pulmonary toxicity is more likely in the longer exposures of saturation chamber diving or hyperbaric oxygen therapy.

Oxygen also has a major role in therapy of many diving disorders.

Toxicity can be avoided by controlling the inspired partial pressure of oxygen and/or the duration of exposure. It can be delayed by intermittent exposure.

INTRODUCTION

The normal partial pressure of oxygen (Po_2) in air is approximately 0.2 ATA. Although essential for survival, oxygen may become toxic at an elevated partial pressure. A rise in the inspired oxygen concentration (Fio_2), an increase in the environmental pressure, or a combination of both will elevate the inspired partial pressure of oxygen (Pio_2).

High Pio_2 has several physiological effects on the body, including an increase in ventilation and a decrease in alveolar and arterial carbon dioxide

buffering tension($Paco_2$). This is due to a rise in central venous Pco_2 due to the reduction in CO_2 carrying capacity of haemoglobin overcoming decreased carotid body excitation resulting from raised Pao_2 itself. Other physiological responses to high oxygen include vagally mediated bradycardia and vasoconstriction of intracranial and peripheral vessels. There is a small rate-dependent fall in cardiac output.

High Pio_2 is known to be associated with retinopathy of prematurity in pre-term infants, and lung damage, convulsions, red cell suppression, visual defects, myopia and cataracts in adults. *In vitro*, toxic effects on cells of many other organs have also been demonstrated.

In diving, toxic effects on cells of the central nervous system (CNS) and lungs are of prime importance, and only these will be discussed in detail. The CNS threshold is above 1.5 ATA and the pulmonary threshold 0.55 ATA. At 1.6 ATA oxygen, pulmonary toxicity is the limiting factor regarding duration of exposure, whereas at higher pressures neurological toxicity is of prime concern.

In both the CNS and lungs there is a latent period prior to the onset of toxicity, and this delay (Fig. 17.1) enables high partial pressures to be used. The length of this latent period depends on the Pio_2.

In diving and diving medicine, oxygen toxicity is possible in the following situations:

- *Closed and semi-closed rebreathing equipment.*
- *Use of high Fio_2 mixtures ('technical diving').*
- *Saturation diving.*
- *Where oxygen is used to shorten decompression times.*
- *Oxygen therapy for diving disorders.*
- *Therapeutic recompression.*
- *Respiratory failure (e.g. near-drowning).*

HISTORY[1,2]

Oxygen was discovered in the latter half of the eighteenth century, and immediately excited interest as to its possible therapeutic effects. Priestley, in 1775, was among the first to suggest that there may be adverse effects of 'dephlogisticated air', subsequently named oxygen by Lavoisier. He observed the rapid burning of a candle and speculated that 'the animal powers be too soon exhausted in this pure kind of air'. Later, in 1789, Lavoisier and Sequin demonstrated that oxygen at 1 ATA does not alter oxidative metabolism, but they did note a damaging effect on the lungs.

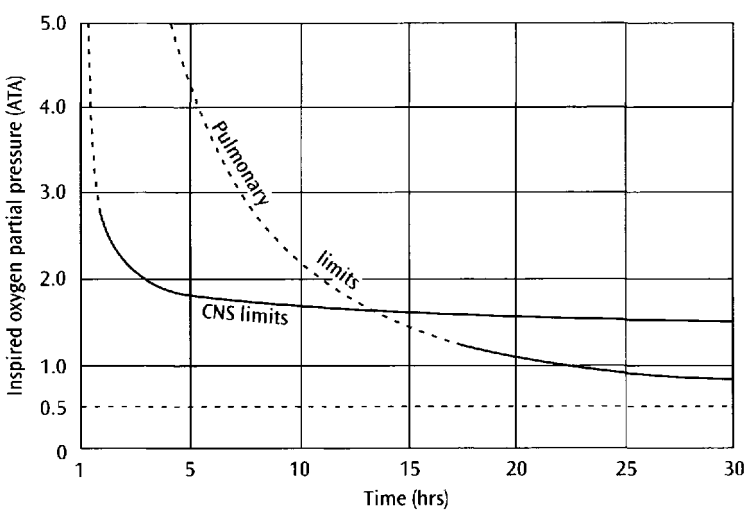

Figure 17.1 *Predicted human pulmonary and central nervous system tolerance to high-pressure oxygen.*

In 1878, Paul Bert published his pioneer work *La Pression Barometrique*[3] in which he presented the results of years of study of the physiological effects of exposure to high and low pressures. He showed that, although oxygen is essential to sustain life, it is lethal at high pressures. Larks exposed to air at 15–20 ATA developed convulsions, and the same effect could be produced by oxygen at 5 ATA. Bert recorded similar convulsions in other species, and clearly established the toxicity of oxygen on the CNS, which is also known as the **Paul Bert effect**. He did not report respiratory damage, however.

In 1899, the pathologist J. Lorrain Smith noted fatal pneumonia in a rat after exposure to 73 per cent oxygen at atmospheric pressure. He conducted further experiments on mice and gave the first detailed description of pulmonary changes resulting from moderately high oxygen tensions (approximately 1.0 ATA) for prolonged periods of time. Smith was aware of the limitations that this toxicity might place on the clinical use of oxygen, and also noted that early changes are reversible and that higher pressures shortened the time of onset. Pulmonary changes are also called the **Lorrain Smith effect**.

Although numerous animal studies were performed, evidence of the effect of high-pressure oxygen on humans was sparse until the 1930s. In 1933, two Royal Naval Officers, Damant and Philips, breathed oxygen at 4 ATA, and reported convulsive symptoms at 16 and 13 minutes. Behnke then reported a series of exposures to hyperbaric oxygen. Exposure at 4 ATA terminated in acute syncope after 43 minutes in one subject, and convulsions at 44 minutes in the other. At 3 ATA no effects were seen after 3 hours, but at 4 hours some subjects noted nausea and a sensation of impending collapse. At that time it was believed that 30 minutes' exposure at 4 ATA and 3 hours' exposure at 3 ATA were safe for men at rest.

Becker-Freyseng and Clamann, in 1939, found that 65 hours' exposure to 730 mmHg oxygen at normal atmospheric pressure produced paraesthesiae, nausea and a decrease in vital capacity. In 1941, Haldane reported a convulsion in less than 5 minutes at 7 ATA oxygen.

At the beginning of World War II, a number of unexplained episodes of unconsciousness were noted in divers using closed-circuit rebreathing oxygen sets at what were considered safe depths. This prompted Donald, in 1942, to commence a series of experiments on oxygen poisoning,[4] and his observations in over 2000 exposures form the basis of current oxygen diving limits. Unfortunately, many of his experiments were performed using rebreathing equipment, without carbon dioxide measurement. Among the more important findings were the marked variation of tolerance and the aggravating effects of exercise and underwater exposure. He suggested a maximum safe depth for oxygen diving of 8 metres.

Research over the past 30 years has been directed primarily at the elucidation of the mechanism of this toxicity. Workers have examined such factors as the role of inert gas and carbon dioxide, blockage of airways and atelectasis, changes in lung surfactant, changes in cellular metabolism, inhibition of enzyme systems and the role of the endocrine system. Further efforts to delineate the pulmonary limits of exposure have also been undertaken. This has become increasingly important with saturation diving involving prolonged stays under increased ambient pressure and the use of oxygen mixtures to shorten decompression time.

AETIOLOGY

The precise mechanism of oxygen toxicity is unknown, but it is clear that oxygen has an effect on the regulation of blood flow, tissue oxygenation and energy metabolism in the brain. These effects are pressure-dependent and are involved in the development of toxicity. There are a great many sites at which oxygen acts on metabolic pathways or on specific cellular functions. These sites may involve cell membranes, 'active transport', synaptic transmission, mitochondria or cell nuclei. Rather than causing an increase in metabolism, as suggested by early workers, hyperoxia has been demonstrated to depress cellular metabolism.

Many enzymes are inactivated by high P_{O_2}, particularly those containing sulphydryl groups (–SH). It is postulated that adjacent –SH groups are oxidized (possibly by oxygen free radicals; see below) to form disulphide bridges (–S–S–), thus

inactivating the enzyme. Enzymes which contain –SH groups, and are known to be susceptible, include glyceraldehyde phosphate dehydrogenase (a key enzyme in glycolysis), the flavoprotein enzymes of the respiratory chain, and the enzymes involved in oxidative phosphorylation.

The **oxygen free radical theory of toxicity** is widely accepted as an explanation at the molecular level. Aerobic organisms have developed antioxidant mechanisms to cope with molecular oxygen exposure, but in the face of hyperoxia these mechanisms may be overwhelmed, leading to the formation of excess reactive oxygen forms. The increased formation of these partially reduced oxygen products or 'free radicals' by hyperbaric oxygenation is thought to be responsible for the cellular toxicity. If produced in excess, they may lead to cellular structural damage or enzyme inactivation. The radicals are intermediates formed in many cellular biochemical enzyme-catalysed reactions. Superoxide anion (O_2^{-}) is formed when oxygen accepts a single electron and hydrogen peroxide (H_2O_2) two electrons. The final reaction is the acceptance by oxygen of four electrons to form water or stable hydroxyl anion. Superoxide and peroxide can react to form the hydroxyl radical (OH^-). All these species of oxygen referred to as oxygen radicals are highly oxidative.

Cells have a system of enzymes to scavenge these radicals known collectively as the **tissue antioxidant system.** Two of these enzymes, superoxide dismutase and catalase, are involved in maintaining adequate supplies of reduced glutathione (which contains –SH groups) to deal with the free radicals. However, hyperoxia may cause this system to be swamped and the excess free radicals may then cause cell damage. Examples of unwanted oxidation reactions are the peroxidation of lipid in cell membranes and protein oxidation in the cell membrane and cytoplasm; both have been demonstrated in rat brain after hyperoxia.[5] Aerolized (recombinant human manganese) superoxide dismutase preserves pulmonary gas exchange during hyperoxic lung injury in baboons.[6] Antioxidants such as glutathione and disulfiram have also been shown to offer some protection.

The characteristic feature of chronic pulmonary oxygen toxicity is pulmonary fibrosis (see later), and in animal studies, paraquat, bleomycin and ozone – all of which are known to produce oxygen free radicals – have all been noted to produce this condition.

Further evidence for the role of the superoxide and hydroxyl free radicals is the demonstration by electron paramagnetic resonance of their increased production in pulmonary endothelial cells.[7]

The molecular disturbance produced by the free radicals is thought to affect cellular metabolic function or, in the CNS, neurotransmitter function. DNA damage has also been postulated to occur, but the exact cellular site of action is still uncertain.

Gamma-aminobutyric acid (GABA) is known to be a transmitter at CNS inhibitory nerve synapses, and one of the demonstrated consequences of enzymatic changes induced by hyperoxia is a reduction in the endogenous output of GABA. This decrease is thought to produce the convulsions by allowing uncontrolled firing of excitatory nerves. Agents, which raise brain levels of GABA, appear to protect against convulsions. Lithium, which has been useful in the treatment of manic-depressive psychosis, has proved to be effective in inhibiting convulsions in rats, as well as preventing the decrease in brain GABA that normally precedes the convulsions. In the rat lung, lithium inhibits the development of oedema.

Exercise, hypoventilation and carbon dioxide inhalation predispose to convulsions, and hyperventilation protects against them. Carbon dioxide may play a role in lowering seizure threshold both at the cellular level, but more likely by influencing cerebral blood flow, and hence the 'dose' of oxygen delivered to the brain.

At greater than 3 ATA Pio_2, oxyhaemoglobin is not reduced on passing through capillaries, and so is not available for the carriage of carbon dioxide as carboxyhaemoglobin. Therefore this route cannot eliminate carbon dioxide. The resultant increase in brain CO_2 tension has proved to be small (2.5–6 mmHg); an equivalent rise is caused by breathing 6 per cent $FiCO_2$and does not cause convulsions in the presence of a normal Pio_2, but the slight rise in PCO_2may reduce the cerebral vasoconstrictive effects of hyperoxia.

CO_2 retention is also unlikely to contribute to pulmonary toxicity, as previously thought, but related changes in acid–base balance may modify the syndrome. These modifying influences may act via

neurogenic and endocrine mechanisms. Very high levels of inspired CO_2 actually protect against pulmonary damage.

Atelectasis is a frequently described change in aviation pathology and is due to absorption collapse of alveoli during 100 per cent oxygen breathing. This has also been suggested as a contributory mechanism to oxygen toxicity in divers. Although absorption collapse has been demonstrated, it is not an initiating factor, as toxicity develops in the presence of inert gas. If the inert gas is at narcotic levels, it actually enhances the onset of toxicity.

Human studies show no difference in the progression of pulmonary oxygen toxicity when comparing pure oxygen or diluted oxygen at the same partial pressure. Rat studies indicate that the risk of CNS toxicity is enhanced by the presence of even small amounts of inert gas in the inspired mixture.

Endocrine studies show that hypophysectomy and adrenalectomy protect against hyperoxia. Adrenocorticotrophic hormone (ACTH) and cortisone reverse this effect and, when given in normal animals, enhance toxicity. Adrenergic blocking drugs, some anaesthetics, GABA, lithium, magnesium and superoxide dismutase each have a protective effect, while adrenaline, atropine, aspirin, amphetamine and pentobarbital are among a host of agents that augment toxicity.

Light, noise and other stressful situations also affect CNS tolerance. Thus, the general stress reaction – and more specifically adrenal hormones – may have a role in enhancing CNS (and pulmonary) toxicity. Several observations suggest a role of the autonomic nervous system in modifying the degree of toxicity. Convulsions have been shown to hasten the onset of pulmonary oxygen toxicity in some animal studies; this may be related to an activation of the sympathoadrenal system during convulsions.

Table 17.1 *Factors increasing oxygen toxicity*

Physiological states
- Physical exercise
- Hyperthermia
- Immersion
- Stress response

Pathological states
- Fever
- Congenital spherocytosis
- Vitamin E deficiency

Drugs
- Amphetamines
- Acetazolamide
- Aspirin
- Atropine
- Disulfiram
- Guanethidine

Gases
- Carbon dioxide
- Nitrous oxide
- ? Inert gases

Hormones and neuro transmitters
- Insulin
- Thyroxin
- Adrenocorticotrophic hormone (ACTH)
- Cortisol
- Epinephrine (adrenaline), norepinephrine (noradrenaline)
- Gamma-amino butyric acid (GABA)

Chemicals
- Paraquat
- NH_4Cl

Trace metals
- Iron
- Copper

CENTRAL NERVOUS SYSTEM TOXICITY (the 'Paul Bert effect')

In diving, CNS oxygen toxicity is more likely when closed or semi-closed circuit rebreathing sets are used, and is the factor limiting depth when oxygen is used. With compressed air, the effect of increased partial pressure of nitrogen (see Chapter 15) usually prevents the diver from reaching a depth and duration at which oxygen will become a problem. 'Technical' diving (see Chapters 62 and 63), in which a higher Fio_2 than air is used, permits CNS toxicity. High oxygen pressures are used in therapeutic recompression for decompression sickness and air embolism (see Chapters 6 and 13). Therefore, the diving physician is more likely to encounter toxicity under these conditions.

Clinical manifestations

A wide range of symptoms and signs has been described, the most dramatic of which is a grand mal-type convulsion. Consciousness is maintained up to the time of convulsion, and there are apparently no changes in the electroencephalogram prior to convulsion.

There is no reliable warning of impending convulsions, but any unusual symptom should be suspect. The following manifestations have been reported, singly and in combination: nausea; vomiting; light-headedness; dizziness; tinnitus; vertigo; incoordination; sensations of impending collapse or uneasiness (dysphoria); facial pallor; sweating; bradycardia; constriction of visual fields; dazzle; lip

CASE REPORT 17.1

A 47-year-old experienced underwater cave diver, with no significant medical history, was diving with two tanks – one containing compressed air, the other a 50 per cent mixture of oxygen and nitrogen (Nitrox). Towards the end of the 47-metre, 19-minute dive, he was seen floating head down, unresponsive, with his mouthpiece out of his mouth and 'his fins (flippers) moving as if he was shivering' (as reported by another diver to the Coroner). The body was carried up to 15 metres depth and then allowed to ascend freely as the other divers decompressed. Cardiopulmonary resuscitation was attempted, but abandoned after 43 minutes, as there was no response.

Examination of the subject's diving equipment revealed that he had been breathing the 50 per cent oxygen/nitrogen mixture for most of the dive. Each tank had a separate first stage connected in an unusual fashion by a two-way switch, which the diver had had made by a local engineering shop. This allowed the diver to switch from one tank to another rapidly. This switch supplied a single second-stage mouthpiece. The two tanks were different colours; the circuit from the black (compressed-air) tank was marked with yellow tape, while the circuit from the yellow (Nitrox) tank was unmarked. The regulator had a small tear and a bite mark in the mouthpiece. The diver wore a facemask and separate mouthpiece rather than a full facemask, which covers eyes, nose and mouth.

The *cause of death*, as determined by the Coroner, was drowning after oxygen toxicity.

twitching; dilatation of pupils; twitching of hand; muscular twitching elsewhere; hiccups; paraesthesiae (especially fingers); dyspnoea; retrograde amnesia; illusions; disturbance of special senses; and hallucinations and confusion.

Facial twitching is a common objective sign in chamber exposures to oxygen greater than 2 ATA, and signifies an imminent convulsion. Lip twitching may be seen if a mouthpiece is being used. Nausea, retching and vomiting are particularly noted after prolonged exposures between 1 and 2 ATA.

The facial pallor is thought to be due to the intense peripheral vasoconstriction of hyperoxia, and not necessarily a sign of cerebral toxicity. Similarly, the paraesthesiae in fingers and toes do not necessarily indicate an impending convulsion; they may persist for hours after exposure and may represent an effect of local vasoconstriction on peripheral nerves.

An important aspect of toxicity is the great variation in susceptibility. As well as the wide range of tolerance between individuals, there is marked variation in one person's tolerance from day to day. Therefore, in any one diver, the time to onset of symptoms cannot be related to a predictable depth or time of exposure. Despite this variation, the greater the partial pressure and the longer the time of exposure, the more likely is the toxicity to develop.

CNS oxygen toxicity

- *Twitching (especially of the lips)*
- *Nausea*
- *Dizziness*
- *Tinnitus*
- *Tunnel vision*
- *Dysphoria*
- *Convulsions*

Exposure in water rather than in dry chambers markedly decreases the tolerance of oxygen. Many of the previously listed clinical features are much less apparent in the water, where convulsions are more often the first manifestation. A convulsion is much more dangerous under water because of the added complications of drowning and pulmonary barotrauma. Therefore, most authorities have set a maximum safe depth for pure oxygen diving at about 10 metres. Short dives may be safe at greater depths and prolonged ones at shallower depths (Table 17.2).

Table 17.2 *US Navy oxygen depth – time limits in water*

Normal operations			Exceptional operations		
Depth in feet	**(m)**	**Time (min)**	**Depth in feet**	**(m)**	**Time (min)**
10	(3)	240	30	(9)	45
15	(4.6)	150	35	(10.7)	25
20	(6)	110	40	(12)	10
25	(7.6)	75			

Factors lowering threshold to CNS toxicity

- *Severe exercise*
- *Immersion in water, rather than chamber*
- *Hypothermia*
- *Increased arterial carbon dioxide from any cause*

Compression chamber exposure is considered to be less hazardous for an equivalent dive profile. Current decompression procedures, if performed in chambers, prescribe oxygen exposures at 18 metres (2.8 ATA).

Exercise has also been shown to hasten the onset of symptoms. Shallower maximum safe depths have been set for 'working' as opposed to 'resting' dives on oxygen. This observation is also of importance when oxygen is used to shorten decompression times in the water. Divers undergoing decompression should be at rest, e.g. supported on a stage – not battling swell, current and buoyancy to maintain constant depth.

Hypothermia is likely to hasten the onset of symptoms.[9]

Carbon dioxide build-up during exercise has been suggested as a potentiating factor in producing convulsions. Increased inspired carbon dioxide tension may develop with inadequate absorbent systems and in poorly ventilated helmets and chambers, thus rendering the diver more susceptible to oxygen convulsions.

The role of inert gas in the exacerbation of oxygen toxicity needs to be more fully elucidated.

CASE REPORT 17.2

BL, a 20-year-old trainee naval diver, was taking part in air diving training to a depth of 21 metres. He was using surface supply breathing apparatus (SSBA), which consists of a demand valve and a hose to the surface connected to a large cylinder via a pressure regulator adjusted according to the depth. After approximately 20 minutes, he was signalled with a tug on the hose to return to the surface because the cylinder was running low. He remained in the water at the surface while his hose was connected to another cylinder, and then recommenced his dive. Some 12 minutes into the second dive BL's surface attendant noted that there were no surface bubbles. The instructor told the attendant to signal BL via the hose tug system, but there were no answering tugs on the line. The stand-by diver was then sent into the water to check BL. He found BL floating a metre off the bottom with his demand valve out of his mouth. He was brought rapidly back to the diving boat and CPR resuscitation was commenced using a portable oxygen resuscitator. After some time, probably about 15 minutes, the small oxygen cylinder of the resuscitator was noted to be low, so one of the group was instructed to connect the resuscitator to the emergency large oxygen cylinder. The oxygen cylinder was then found to have a line already attached to it – BL's SSBA! BL failed to respond to intense resuscitation carried on for more than 2 hours.

Diagnosis: death due to central nervous system oxygen toxicity (presumably convulsions).

CASE REPORT 17.3

AM and his buddy, both military divers, were practising night-time underwater ship attack using closed-circuit 100 per cent oxygen rebreathing sets. While approaching the ship, they exceeded the maximum safe depth to avoid being spotted by lights, and had to ascend to the ship's hull (depth 9 metres). While escaping from the ship, AM had difficulty in freeing his depth gauge and, when he finally did examine it, discovered he was at 19 metres. He started to ascend and remembers 'two or three jerkings' of his body before losing consciousness. The buddy diver noted that AM 'stiffened' as he lost consciousness and then started convulsing, which continued while being brought to the surface. Total time of dive was 28 minutes. At the surface, AM was pale with spasmodic respirations, and the lug on the mouthpiece had been chewed off. Artificial respiration was administered. AM was incoherent for 20 minutes and vomited once. A headache and unsteadiness in walking persisted for several hours after the incident. An electroencephalogram three days later was normal. (The buddy diver was exhausted on surfacing, felt nauseated and was unable to climb into the boat, but recovered quickly.)

Diagnosis: near-drowning due to central nervous system oxygen toxicity.

Table 17.3 *Incidence of symptoms resulting from exposure of divers to 'end-point' in water*

Symptoms	388 resting divers (%)	120 working divers (%)
Convulsions	9.2	6.8
Lip twitching	60.6	50.0
Vertigo	8.8	20.8
Nausea	8.3	17.5
Respiratory disturbances	3.8	5.0
Twitching of parts other than lips	3.2	1.7
Sensation of abnormality (drowsiness, numbness, confusion, etc.)	3.2	—
Visual disturbances	1.0	—
Acoustic hallucinations	0.6	—
Paraesthesiae	0.4	—

The danger of convulsions prevents divers breathing 100 per cent oxygen in safety, deeper than 8–10 metres of seawater.

The frequency of presenting symptoms in 'wet' divers resting and working is shown in Table 17.3, based on the studies of Donald.[2,4] In all cases exposure continued until the first symptoms developed ('end-point').

Convulsions, which may be the first manifestation of toxicity, are indistinguishable clinically from grand mal epilepsy. A review of neurological toxicity in US Navy divers showed that convulsions were more likely to be the presenting feature in inexperienced divers breathing oxygen, compared with trained divers. It is inferred that some of the so-called premonitory symptoms may be due to suggestion rather than oxygen. Of 63 divers, 25 had convulsions as the first clinical manifestation, 10 had focal twitching, and 13 more progressed to convulsions despite immediate reduction of partial pressure. A more recent study revealed nausea as the most common manifestation, followed by muscle twitching and dizziness.

The following description of a typical fit has been given by Lambertsen,[10] who performed much of the original work in the USA on this subject.

Localized muscular twitching, especially about the eyes, mouth and forehead usually but not always precedes the convulsion. Small muscles of the hands may also be involved, and incoordination of diaphragm activity in respiration may occur. After they begin, these phenomena increase in severity over a period, which may vary from a few minutes to nearly an hour, with essentially clear consciousness being retained. Eventually an abrupt spread of excitation occurs and the rigid tonic phase of the convulsion begins. Respiration ceases at this point and does not begin again until the intermittent muscular contractions return. The tonic phase usually lasts for about 30 seconds and is accompanied by an abrupt loss of consciousness. It is followed by vigorous clonic contractions of the muscle groups of head and neck, trunk and limbs, which become progressively less violent over about 1 minute. As the uncoordinated motor activity stops, respiration can proceed normally. Following the convulsion, hypercapnia is marked due to accumulation of carbon dioxide concurrent with breath-holding. Respiration is complicated by obstruction from the tongue and by the extensive secretions, which result from the autonomic component of the central nervous system convulsive activity. Because the diver inspired a high pressure of oxygen prior to the convulsion, a high alveolar oxygen tension persists during the apnoea. The individual remains well oxygenated throughout the convulsion. Due to the increased arterial carbon dioxide tension, brain oxygenation could increase the breath-holding period. This is in contrast to the epileptic patient who convulses while breathing air at sea level.

The latent period before the onset of toxic symptoms is inversely proportional to the inspired oxygen tension. However, it may be prolonged by hyperventilation and interruption of exposure, and shortened by exercise, immersion in water and the presence of carbon dioxide.

The 'oxygen off-effect' refers to the unexpected observation that the first signs of neurological toxicity may appear after a sudden reduction in inspired oxygen tension. Existing symptoms may also be exacerbated. The fall in inspired oxygen pressure is usually the result of removing the mask from a subject breathing 100 per cent oxygen in a chamber, but it may also occur when the chamber pressure is reduced, or when the diver surfaces. It has been postulated that the sudden drop in cerebral arterial oxygen tension in the presence of persisting hyperoxic-induced cerebral vasoconstriction results in cerebral hypoxia in a brain already impaired by oxygen poisoning.

Typical grand mal epilepsy has been observed up to several hours after exposure to high oxygen pressure. It has been suggested that there may be a

CASE REPORT 17.4

TL was using a semi-closed circuit rebreathing apparatus rigged for 60 per cent nitrogen. After 17 minutes at 22 metres, he suddenly noted a ringing noise in his head. He flushed through his set, thinking that his symptoms were due to carbon dioxide toxicity, but then noted that his surroundings were brighter than usual, and decided to surface.

On surfacing he was noted to be conscious, but pale and panting heavily. He moved his mouthpiece and while being brought on board 'went into a convulsion, where his whole face changed shape, his eyes rolled up into his head, his face turned a dark colour and his body began to cramp'. He recovered within 3–4 minutes, and 30 minutes later there was no abnormality on clinical examination.

Equipment examination revealed that the emergency oxygen cylinder was nearly empty, i.e. that he had used 64 litres of 100 per cent oxygen in addition to approximately the same amount of 60 per cent oxygen. The oxygen in his breathing bag would therefore have approximated 80 per cent and the inspired oxygen tension 2.4 ATA. The carbon dioxide absorbent was normal.

Diagnosis: central nervous system oxygen toxicity.

relationship between the manifestations of latent epilepsy and oxygen exposure. In hyperbaric oxygen therapy, known epileptics have convulsed at less than expected pressure exposure.

Some animal experiments have shown that older and male animals are more susceptible to poisoning, but this has not been conclusively demonstrated in humans.

The major differential diagnosis of neurological toxicity is cerebral arterial gas embolism (CAGE) producing neurological features. After reducing the oxygen partial pressure, recovery is to be expected from oxygen toxicity. Repeated daily exposures can produce a syndrome of fatigue, headache, dizziness and paraesthesiae in operational divers.

Pathology

No pathological changes in the CNS that are directly attributable to oxygen toxicity have been observed in humans. Animal experiments with intermittent or continued exposure cause permanent neurological impairment with selective grey matter and neuronal necrosis (the **John Bean effect**).[11] By light and electron microscopy, changes have been reported in studies in rats at 8 ATA oxygen. Lesions, which are characterized by pyknosis, hyperchromatosis with vacuolization and lysis of the cytoplasm, and caryorrhexis, are found in specific areas, such as in the reticular substance of the medulla, the pericentral area of the cervical spinal grey matter, the ventral cochlear nuclei, the maxillary bodies and the inferior colliculi.

Pharmacological control of convulsions and pulmonary oedema does not alter the findings. Severe exposure eventually leads to haemorrhagic necrosis of the brain and spinal cord, but even single exposures (30 minutes at 4 ATA) produce ultrastructural changes in anterior horn grey matter.

Prevention

Predictable prevention of cellular changes by administration of drugs is not yet feasible. In animal experiments, many different pharmacological agents have been shown to have a protective effect against the toxic effects of oxygen, including disulfiram, glutathione, lithium, iso-nicotinic acid, hydrazide, GABA and sympathetic blocking agents. None of these agents is in prophylactic clinical use at present, however. Prevention of convulsions by anaesthetics or anticonvulsant agents removes only this overt expression of toxicity, and damage at the cellular level will continue.

The only current safe approach is to place limits on exposure. These limits depend on the partial pressure of oxygen, the duration and the conditions of exposure (such as 'wet dive' or in a dry chamber, at exercise or rest, intermittent or continuous).

The Royal Navy and Royal Australian Navy place a limit for pure oxygen diving of 9 metres for a resting dive and 7 metres for a working dive.[12] The US Navy relates the maximum duration of exposure to the depth (see Table 17.2).[13]

The US Navy formerly required divers to undergo a test exposure of 30 minutes at 18 metres (60 feet) breathing 100 per cent oxygen to eliminate those who are unusually susceptible.[14] This does not take into account the marked variation in individuals from day to day, or the marked influence of the exposure environment. Some individuals may be excessively oxygen-sensitive, but the variability makes this uncertain and difficult to screen for. The oxygen tolerance test probably has no value in assessing normal dive candidates.

An awareness of levels at which toxicity is likely, and close observation for early signs such as lip twitching, should reduce the incidence of convulsions. If early signs are noted, the subject should signal his companion, stop excessive exertion, and hyperventilate. However, premonitory symptoms or signs are unlikely in the working diver in the water.

In chamber therapy, most therapeutic tables do not prescribe 100 per cent oxygen deeper than 2.8 ATA. Periods of air breathing are used to interrupt the exposure to high levels of oxygen and thus reduce the likelihood of toxicity. Animal studies have demonstrated that interrupted exposure delays the onset of CNS toxicity by up to 100 per cent.

Electroencephalographic monitoring has not proved useful in predicting imminent convulsions.[15]

If the decompression illness is very serious, and the risks of oxygen toxicity are acceptable, drugs such as diazepam may be used to reduce the toxicity effects.

Oxygen-breathing divers and hyperbaric workers are advised to avoid:

- *Exposure while febrile.*
- *Drugs that increase tissue CO_2, e.g. opiates, carbonic anhydrase inhibitors.*
- *Aspirin, steroids, sympathomimetics.*
- *Stimulants such as caffeine (e.g. coffee).*
- *Fluorescent lights.*

Treatment

The initial aim is basically to avoid physical trauma associated with a grand mal convulsion. A padded tongue depressor to prevent tongue biting may be useful in a chamber. In contrast to epilepsy hypoxia is not a concern, at least initially.

In the water, the diver should be brought to the surface only after the tonic phase of the convulsion has ceased. The same action is indicated in compression chambers, but with allowance made for decompression staging. If it is against the interests of the patient to ascend, it is usually a simple matter to reduce the oxygen in his breathing mixture.

Table 17.4 *The use of oxygen in recompression therapy*[16]

IF OXYGEN INTOLERANCE OCCURS OR IS ANTICIPATED:

- Halt ascent, remove mask at once, maintain depth constant.
- Protect a convulsing patient from injury due to violent contact with fixtures, deckplates or hull, but do not forcefully oppose convulsive movements.
- With a padded mouthbit protect the tongue of a convulsing patient.
- For non-convulsive reactions, have patient hyperventilate – with chamber air for several breaths.
- Administer sedative drugs upon direction of a medical officer.
- 15 minutes after the reaction has entirely subsided, resume the schedule at the point of its interruption.
- If the reaction occurred at 18 metres, on the 135-minute schedule, upon arrival at 9 metres switch to 285 minute schedule (15 minutes air–60 minutes oxygen, 15 minutes air–60 minutes oxygen).

Anticonvulsants may be used in exceptional circumstances. For example, phenytoin has been successfully used to stop convulsions in a patient with cerebral air embolism being treated with hyperbaric oxygen; diazepam is also very effective.

PULMONARY TOXICITY (The 'Lorrain Smith effect')

Clinically obvious pulmonary oxygen toxicity does not manifest in short-duration, surface-based oxygen diving. Rather, it assumes greater importance in saturation and long chamber dives, especially where high partial pressures of oxygen are inspired, such as in therapeutic recompression. Prolonged exposures to partial pressures as low as 0.55 ATA (such as in space flight) have been found to produce significant changes. A Pio_2 of 0.75 ATA has produced toxicity in 24 hours.

In animals, pulmonary oxygen poisoning causes progressive respiratory distress, leading to respiratory failure and finally death. The wide variation of tolerance among different species invalidates direct extrapolation of animal studies to humans, but early signs in humans are similar to those in animals. In patients receiving high concentrations of oxygen therapeutically, it is sometimes difficult to distinguish between the conditions for which the oxygen is given and the effects of oxygen itself (e.g. shock lung, respiratory distress syndrome).

Clinical manifestations

As in neurological toxicity, the factors affecting the degree of toxicity are the Pio_2, the duration of exposure, and individual variation in susceptibility. Exposure to 2.0 ATA oxygen produces symptoms in some normal humans at 3 hours, but the odd individual may remain symptom-free for up to 8 hours.

The earliest symptom is usually a mild tracheal irritation similar to the tracheitis of an upper respiratory tract infection. This irritation is aggravated by deep inspiration, which may produce a cough. Smoking has a similar result. Chest tightness is often noted, then a substernal pain develops which is also aggravated by deep breathing and coughing. The

cough gets progressively worse until it is uncontrollable. Dyspnoea at rest develops and, if the exposure is prolonged, is rapidly progressive. The higher the inspired oxygen pressure, the more rapidly do symptoms develop and the greater the intensity.

Physical signs, such as rales, nasal mucous membrane hyperaemia and fever have only been produced after prolonged exposure in normal subjects.

Pulmonary oxygen toxicity

- *Chest tightness or discomfort*
- *Cough*
- *Shortness of breath*
- *Chest pain*

The measurement of forced vital capacity (FVC or VC) is one monitor of the onset and progression of toxicity, although it is less sensitive than the clinical symptoms. Reduction in VC is usually progressive throughout the oxygen exposure. The drop continues for several hours after cessation of exposure, and may occasionally take up to 12 days to return to normal. Because measurement of VC requires the subject's full cooperation, its usefulness may be limited in the therapeutic situation. It has been used to delineate pulmonary oxygen tolerance limits in normal subjects (see Fig. 17.2, which relates partial pressure of oxygen to duration of exposure and indicates the percentage fall in VC). The size of the fall in VC does not always indicate the degree of pulmonary toxicity as measured by other lung function tests, such as other lung volumes, static and dynamic compliance, and diffusing capacity for carbon monoxide. In fact, changes in diffusing capacity may be the most sensitive indicator.

Exposure at 3 ATA for 3.5 hours caused chest discomfort, cough and dyspnoea in most of 13 subjects. There was no significant change in post-exposure FVC, and maximum mid-expiratory flow rates were reduced; however, airway resistance did not change.[17]

Some studies in divers have indicated that reduction in forced mid-expiratory flow rates may persist for at least three years after deep saturation dives, and also after shallow saturation dives with the same hyperoxic exposure profile. It was concluded that the observed pulmonary function changes, which indicate the development of small airways obstruction, are due to oxygen toxicity.[18] Forced expiratory volume in one second (FEV_1) and FVC were not significantly altered.

Certain subjects, especially at higher inspired oxygen pressures (2.5 ATA), demonstrate a rapid fall in VC. The recovery after exposure is also more rapid than that after an equal VC decrement produced at a lower oxygen pressure for a longer time.

Although chest X-ray changes have been reported,

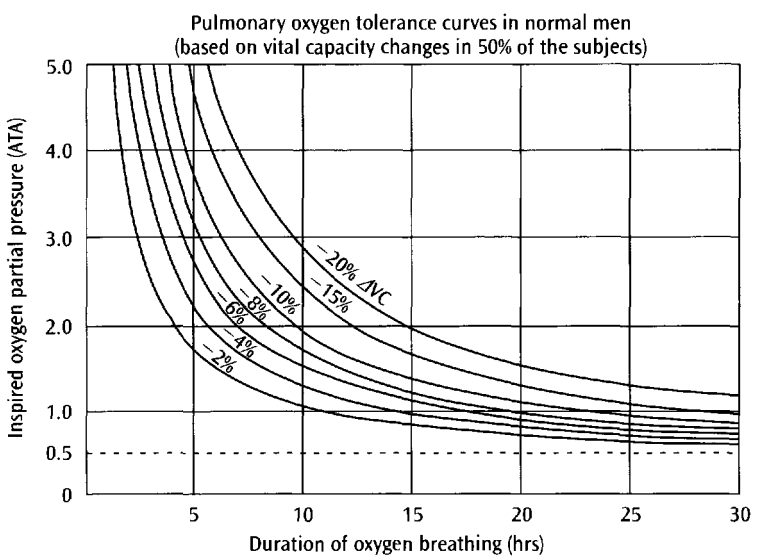

Figure 17.2 *Relationship of partial pressure of oxygen breathed, and duration of exposure, to degree of pulmonary oxygen damage.*

there is no pathognomonic appearance of oxygen toxicity. Diffuse bilateral pulmonary densities have been reported. With continued exposure, irregularly shaped infiltrates extend and coalesce.

Pathology[19,20]

The pathological changes in the lung as a result of oxygen toxicity have been divided into two types; acute and chronic, depending on the inspired pressure of oxygen.

Pressures of oxygen greater than 0.8 ATA cause acute toxicity that has been subdivided into exudative and proliferative phases. The **exudative phase** consists of a perivascular and interstitial inflammatory response and alveolar oedema, haemorrhage, hyaline membranes, swelling and destruction of capillary endothelial cells and destruction of type I alveolar lining cells. (This phase was the type described by Lorrain Smith.) Progression of the disease leads to the **proliferative phase**, which, after resolution of the inflammatory exudate, is characterized by proliferation of fibroblasts, and type II alveolar cells. There is an increase in the alveolar-capillary distance. Pulmonary capillaries are destroyed and some arterioles become obstructed with thrombus.

The chronic response usually follows inspired oxygen tensions between 0.5 and 0.8 ATA for longer times. It is characterized by hyperplasia of type II cells, replacing type I cells and progressive pulmonary fibrosis, especially affecting alveolar ducts rather than alveolar septa. These features are also found in the adult respiratory distress syndrome (shock, drowning, trauma) for which high oxygen tensions are given. Whether oxygen actually causes the damage in these situations or exacerbates the condition by interacting with the initial pulmonary damage is not clear.

A consequence of these effects on pulmonary physiology is to increase ventilation–perfusion inequality. Obstruction of arterioles will result in an increase in dead space.

Prevention

No specific therapy is available which can be used clinically to delay or modify the pulmonary damage caused by hyperoxia. Intermittent exposure may delay the onset of toxicity. Delay of pulmonary toxicity has been demonstrated in humans. It has been suggested that the rate of recovery is greater than the rate of development of cellular changes leading to toxicity.

When toxicity is evident, the oxygen partial pressure should be reduced. Hence, it is important to be aware of the earliest signs of the syndrome.

Traditionally, the monitoring of VC has been employed as an indicator of the progression of toxicity, and the maximum acceptable reduction in VC is dependent upon the reasons for the exposure. Although a 20 per cent reduction may be acceptable in the treatment of severe decompression sickness, a 10 per cent reduction would cause concern under operational diving conditions.

The degree of oxygen toxicity equivalent to a 2 per cent decrease in VC is completely reversible, asymptomatic, and very difficult to measure under ordinary circumstances. With the elevated pressures of oxygen used in the treatment of serious diseases, such as severe decompression sickness or gas gangrene, it may be reasonable to accept a greater degree of pulmonary toxicity in order to treat the patient. The primary requirement of any therapy is that the treatment should not be worse than the disease.

The degree of pulmonary toxicity, which produces a 10 per cent decrease in VC, is associated with moderate symptoms of coughing and pain in the chest on deep inspiration. This degree of impairment of lung function has been shown experimentally to be reversible within a few days. It is suggested that a 10 per cent decrement in VC be chosen as the limit for most hyperbaric oxygen therapy procedures.

VC is a relatively crude measure of toxicity, and forced mid expiratory flow measurements or the less practical diffusing capacity for carbon monoxide may prove to be more sensitive indicators for repeated or long-term exposures.

Intermittent rather than continuous exposure to high oxygen pressure delays the onset of both neurological acid pulmonary oxygen toxicity.

Adherence to proposed pressure-duration limits for pulmonary oxygen toxicity is difficult where extended durations and changing partial pressures of oxygen are involved. Methods for calculating cumulative pulmonary toxicity have therefore been devised, and probably have a role in prolonged decompression and hyperbaric oxygen therapy.[21]

As discussed in the prevention of neurological toxicity, many drugs have been shown to be effective in animal experiments, and these may have a future role in the prevention of pulmonary and other oxygen toxicity, at least for hyperbaric therapy exposure.

OTHER MANIFESTATIONS OF TOXICITY

It has been suggested that oxygen, although essential for survival of aerobic cells, should be regarded as a universal cellular poison. All organs and tissues of the body are susceptible to damage from oxygen free radical production. Nevertheless, in other organs receiving a high blood flow such as heart, kidney and liver, no toxicity has yet been detected in humans. It is possible that CNS and pulmonary toxicity pre-empt its development in other organs.

Haemopoietic system

In space flight exposures, oxygen has been shown to have a deleterious effect on red blood cells manifested by abnormal cell morphology and/or a decrease in circulating red blood cell mass. This may be due to depression of erythropoiesis, inactivation of essential glycolytic enzymes or damage to red cell membranes resulting from peroxidation of membrane lipid. Mice studies show irreversible damage to haemopoietic stem cells after 24 hours' exposure to 100 per cent O_2.

There have also been occasional reports of haemolytic episodes following hyperbaric oxygen exposure, but these seem to be related to individual idiosyncrasies such as specific enzyme defects.

Eye

This has been reviewed by Butler and colleagues.[22] In 1935, Behnke reported a reversible decrease in peripheral vision after oxygen breathing at 3.0 ATA, while Lambertsen and Clarke later demonstrated a progressive in peripheral vision after 2.5 hours' breathing oxygen at 3.0 ATA, reaching about 50 per cent after 3.5 hours. Recovery was complete after 45 minutes of air breathing.

Progressive myopic changes have resulted from hyperbaric therapy, and have also been noted in divers. Reversal of this myopic shift usually occurs within a few weeks, but could take as long as a year. Butler *et al.* have demonstrated a myopic shift after 15 days and approximately 45 hours' of diving exposure to 1.3 ATA oxygen.

Cataract formation has also been reported after hyperbaric exposure,[23] with lens opacities not completely reversible. The myopia and cataract formation may represent lenticular oxygen toxicity.

A reduction of the intraocular pressure may represent a toxic effect on the ciliary process. Retinal detachments, retinal micro-infarcts, changes in dark adaptation, photoreceptor damage and a decrease in the amplitude of the electroretinogram have all been recorded.

Other ocular effects of oxygen toxicity include retinopathy of prematurity in infants breathing supplemental oxygen. Initial retinal blanching due to vasoconstriction is followed by vessel obliteration, fibrovascular proliferation, which may lead to retinal fibrosis and traction retinal detachments. Animal studies have demonstrated the death of visual cells and retinal detachments on exposure to 0.9 to 3 ATA oxygen. Irreversible changes in the cornea and lens of guinea pigs develop after exposure to 3 ATA oxygen for between 4 and 16 hours.

Vasoconstriction

Other disorders that may be affected by the hyperoxic-induced vasoconstriction include Raynaud's phenomenon, Buerger's disease and migraine. Risk of closure of the ductus arteriosus has also been proposed in the foetus exposed to increased oxygen.

Ear

Serous otitis media has been noted in aviators exposed to high concentrations of oxygen, this

being caused by absorption of oxygen from the middle ear. A syndrome related to the middle ear has been described in US Navy divers breathing 100 per cent oxygen from semi-closed and closed-circuit diving equipment.[24] The symptoms were fullness, popping or crackling sensation in the ear, and a mild conductive hearing loss. On examination the most common finding was fluid in the middle ear. The syndrome was first noted after rising from a night's sleep rather than immediately after the dive itself, and disappeared rapidly. There was no suggestion of barotrauma.

Cancer

Repeated or chronic exposure to high levels of oxygen free radicals might be expected to enhance tumour development. However, a literature review including human and animal studies failed to support a possible cancer-causing effect of hyperbaric oxygen.[25]

It is likely that, as more sensitive methods of detection are used, evidence of oxygen toxicity in many other cells and organs will be observed.

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18

Carbon dioxide toxicity

CARL EDMONDS

RESPIRATORY PHYSIOLOGY

Carbon dioxide (CO_2) is normally present in the atmosphere in a concentration of 0.03–0.04 per cent volume of dry air. This represents a partial pressure of 0.23–0.30 mmHg. CO_2 is one of the products of metabolism of protein, carbohydrates and fats produced in the mitochondria of cells in approximately the same volume as oxygen is consumed:

$$C_6H_{12}O_6 + 6O_2 = 6CO_2 + 6H_2O + \text{energy}$$

The resultant CO_2 has to be transported from the tissues by the circulation and eliminated by exhalation from the lungs.

The normal CO_2 tension in arterial blood ($PaCO_2$) is 40 mmHg and for mixed venous blood is 46 mmHg, these values being a balance between production and excretion. CO_2 in the alveolar gas is in equilibrium with that of the pulmonary veins; the alveolar partial pressure ($PACO_2$) is therefore also 40 mmHg. Being a product of metabolism, this is constant irrespective of depth – unlike oxygen and nitrogen, which reflect the pressures in the lung.

Carbon dioxide is the most potent stimulus to respiration, the central medullary chemoreceptors in the brain being stimulated by increases in arterial CO_2 and acidosis. In normal conditions, adjustments in ventilation keep the arterial and alveolar CO_2 partial pressures remarkably constant. The peripheral chemoreceptors (carotid and aortic bodies) are primarily responsive to hypoxaemia (increasing respiration), but also respond to increases in acidosis and CO_2 concentration.

The solubility of CO_2 is about 20 times that of oxygen, so there is considerably more CO_2 than oxygen in simple solution. CO_2 is transported in the blood in both plasma and red cells. In each 100 ml of arterial blood, 3 ml are dissolved, 3 ml are in carbamino compounds (with haemoglobin and plasma proteins), and 44 ml are carried as bicarbonate (HCO_3^-).

At rest, approximately 5 ml CO_2 per 100 ml blood are given up from the tissues and liberated in the lungs. About 200 ml CO_2 are produced and excreted per minute. If this CO_2 is retained in the body (e.g. due to rebreathing) the $PaCO_2$ will climb at the rate of 3–6 mmHg/minute.

With exercise, much larger amounts of CO_2 are produced. For example, the working diver can produce over 3 litres of CO_2 per minute for short periods, and 2 litres per minute for over half an hour, usually without serious alteration in $PaCO_2$, this being due to a concomitant increase in respiration.

In diving, although the environmental pressure is increased, the arterial and hence alveolar CO_2 tensions should be maintained at approximately 40 mmHg. This is because the number of molecules of

CO_2 produced, and therefore the alveolar P_{CO_2} is independent of the depth; hence, the alveolar CO_2 percentage decreases with increased depths. The alveolar P_{O_2} and P_{N_2} increase with depth, but the percentages show little change.

With a constant rate of CO_2 production, the alveolar (and hence the arterial) CO_2 tension is inversely proportional to the alveolar ventilation, to which must be added the tension of CO_2 in the inspired gases. This is shown in the following equation:

$$P_ACO_2 \rightarrow \frac{kVCO_2}{Va} + PiCO_2$$

where $PACO_2$ is alveolar partial pressure of carbon dioxide; k is a conversion factor to convert conditions at STPD (standard temperature and pressure, dry) to BTPS (body temperature and pressure, saturated); VCO_2 is the carbon dioxide production (in litres/min STPD); Va is the alveolar ventilation (in litres/min BTPS); and $PiCO_2$ is the inspired carbon dioxide partial pressure.

Alterations in $PaCO_2$ have widespread effects on the body, especially on the respiratory, circulatory and nervous systems. Apart from the hypocapnia produced by hyperventilation prior to breath-hold diving (see Chapters 16 and 61), the more frequent derangement in diving is hypercapnia, an elevation of CO_2 in blood and tissues. This may be an acute effect, or chronic. Where hypercapnia produces pathophysiological changes that are dangerous to the diver, the term 'CO_2 toxicity' (or CO_2 poisoning) is used.

ACUTE HYPERCAPNIA

Causes

Excluding asphyxia and drowning, there are five main mechanisms of CO_2 toxicity in diving:

1 Failure of the absorbent system, e.g. in closed or semi-closed rebreathing apparatus, submarines, saturation complexes, etc.
2 Inadequate ventilation of an enclosed environment, e.g. in standard dress or other helmet diving and compression chamber diving where flushing is required to remove CO_2.
3 Inadequate pulmonary ventilation, e.g. in deep diving where the work of breathing dense gases is greater, or with increased resistance from the equipment.
4 Physiological adaptations to diving ('CO_2 retainers')
5 Contamination of breathing gases by CO_2.

With all these, CO_2 toxicity is much more rapid when the diver is exercising and producing CO_2 in large amounts.

Carbon dioxide toxicity is most commonly encountered in divers using closed or semi-closed rebreathing equipment. It is also seen where there is inadequate ventilation of an enclosed space such as a helmet, recompression chamber or submarine.

In diving operations that rely on recycling of respiratory gases, the most common method of CO_2 removal uses the reaction between alkali metal (sodium, lithium) hydroxide reagents (Protosorb™, Sodasorb™, Baralyme™, Dragersorb™, etc.) and carbonic acid:

$$H_2O + CO_2 \rightarrow H_2CO_3$$

$$H_2CO_3 + 2NaOH \rightarrow Na_2CO_3 + 2H_2O$$

$$Na_2CO_3 + Ca(OH)_2 \rightarrow 2NaOH + CaCO_3$$

Other techniques, some of which are still being developed, include cryogenic freeze-out of CO_2 with liquid air or oxygen, molecular sieves, electrolytic decomposition into carbon and water, and the use of peroxides and superoxides which generate oxygen while removing CO_2.

Failure of the absorbent system

This failure in rebreathing sets may be due to the following causes:

- **Inefficiency of absorbent material**: this may be due to large granule size, low environmental temperature, low alkali content, low water content or seawater contamination.

- Equipment design faults: the canister should be of adequate size and adequate length compared with the cross-sectional area, and should be insulated against extreme temperature changes. The design should prevent 'channeling' of gases through the absorbent. In circuit rebreathing equipment, the gas space between the absorbent granules ideally should exceed the maximum tidal volume, so that there is time for absorption during the next part of the respiratory cycle. In pendulum rebreathing equipment, excessive functional dead space between the diver's mouth and the canister causes inhalation of expired CO_2.
- Operator error: CO_2 build-up may result if the diver fails to pack his canister properly with active absorbent, undertakes excessive exertion, or exceeds the safe working life of the set.

Inadequate ventilation of the environment

In helmet and recompression chamber dives, there must be a sufficient volume of gas supplied to flush the enclosed system of CO_2. In the same way that alveolar P_{CO_2} is dependent on alveolar ventilation, the level of CO_2 in the enclosed space is inversely proportional to the ventilation of that space. When corrected to surface volumes, this means progressively greater amounts of gas must be supplied as the diver or chamber goes deeper.

Inadequate pulmonary ventilation

At depth this is primarily due to the increased density of the respired gases. This causes an increased resistance to gas flow, both in the breathing apparatus and in the diver's own airways. Using the less dense helium as the diluent gas instead of nitrogen reduces

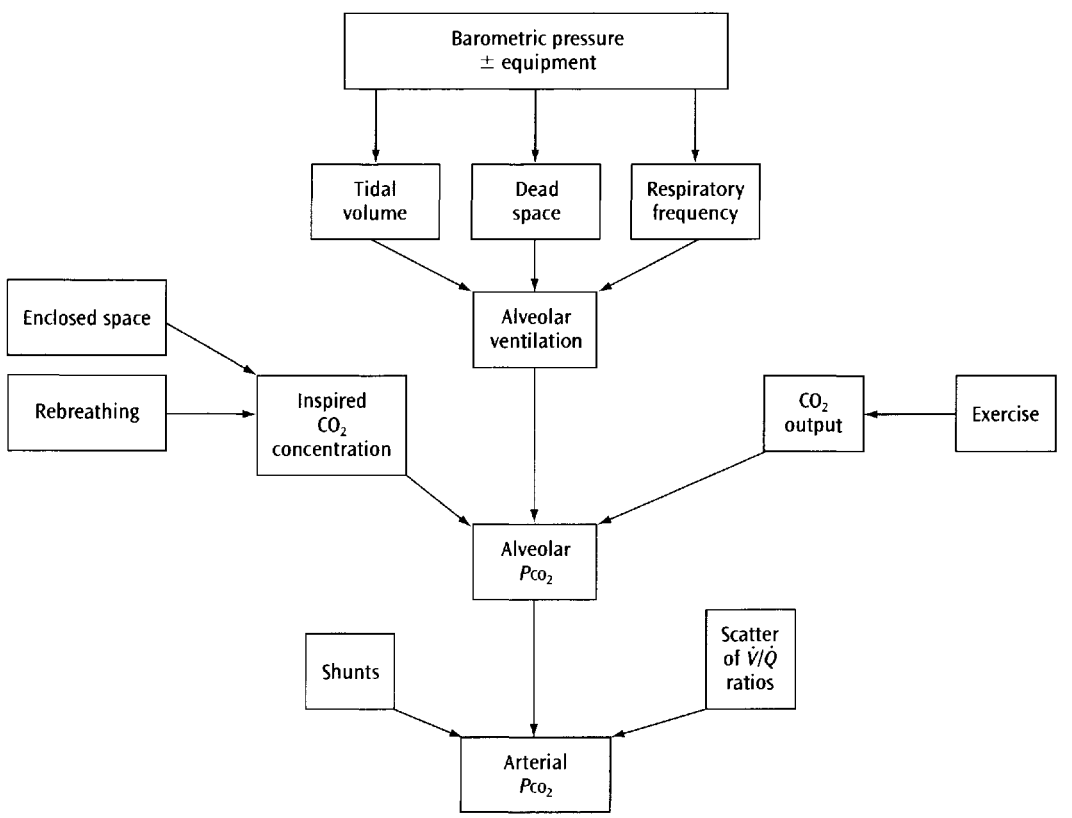

Figure 18.1 *Some factors which influence P_{CO_2}. (Adapted from J.F. Nunn,* Applied Respiratory Physiology, *3rd edition, 1987, Butterworths, London).*

this problem. Tight wet suits, harnesses and buoyancy compensators further restrict thoracic movement, and an increased workload is thus placed on the diver's respiratory muscles. The extent to which this load is overcome varies greatly among divers, but there is often some elevation of the alveolar P_{CO_2}.

Physiological adaptations to diving ('CO_2 retainers')

In the diving literature CO_2 is often incriminated as a major factor producing loss of consciousness, with rebreathing equipment and while employing high oxygen pressures in gas mixtures. There have been ample cases, and adequate investigation of these cases, to verify this.

High oxygen levels in the inspiratory gas have been thought by some (Lanphier) to reduce the respiratory response to CO_2. However, most others show that at an aerobic work rate, ventilation is not altered whether the subject breathes 100 per cent oxygen or air.

Kerem and his co-workers demonstrated that while breathing high oxygen pressures, established divers had significantly lower sensitivity to high CO_2 levels. They concluded that the divers had an inherent or acquired relatively impaired CO_2 response. Kerem also demonstrated that the mean end-tidal CO_2 tension was significantly higher than that in non-divers and diving trainees, when breathing nitrox with 40 per cent O_2 both at 1 ATA and 4 ATA.

In relationship to open-circuit equipment using air (scuba), the relevance of CO_2 as a contributor to diving problems is more dubious.

In the general physiology literature it is believed that the P_{aCO_2} is held remarkably constant during both rest and exercise, within a very narrow range of a few mmHg. Dempsey and Pack, in a recent text on the regulation of breathing, stated that, "significant, sustained CO_2 retention is extremely rare in health, even under the most extreme conditions of exercise intensity and flow limitation".

Yet, significant exercise (O_2 uptake >60 per cent of maximal) under water and using scuba, produces an elevation of P_{ACO_2} and is sometimes marked (P_{aCO_2} >60 mmHg). With increasing depth, a progressively greater amount of energy is required to breathe and rid the body of CO_2.

Different regulators produce varying degrees of resistance to ventilation, with consequent CO_2 retention. This will also rise with the increased breathing resistance due to inadequate maintenance of the regulator, or the deposition of foreign bodies and salt particles. Maintaining regulator performance to an acceptable level can be difficult. The CO_2 retention is usually minor, and does not lead to CO_2 toxicity. It does, however, increase with exposure to depth, and with low scuba cylinder pressure driving the gas through the regulator.

Perhaps more important than the consistent but mild CO_2 build-up with normal scuba diving, is the occasional atypical subject who responds inadequately, either at depth or on the surface, to raised CO_2 levels. It may be that these divers progressively elevate their CO_2, as an alternative to increasing their ventilation, when the resistance to breathing increases. Under these conditions, it is theoretically possible that toxic levels may eventuate.

In diving parlance these divers are referred to as 'CO_2 retainers', a concept based heavily on the work and promotion from Lanphier. This concept has received widespread acceptance in the diving medical fraternity, but is less accepted among more conventional physiologists.

If, indeed, there are such divers who are 'CO_2 retainers', then the explanation for this is conjectual. Some have suggested that ability to tolerate larger than normal CO_2 levels will permit the subject to achieve greater diving successes, e.g. succeed with deep breath-hold diving, or tolerate the high CO_2 levels in helmet diving, breathing resistance from equipment or increased gas density with depth. In this scenario, the 'CO_2 retainer' would be self-selecting – being more successful during the diver training years, and therefore be over-represented in the established diver population.

A more plausible explanation for high P_{ACO_2} levels in divers, for the various reasons referred to previously, is that divers are exposed to higher levels of CO_2 and develop an adaptation towards them.

One diving technique, that of '**skip-breathing**', may be particularly relevant, as it was very commonly employed by divers to reduce their air consumption and therefore prolong the dive duration.

In this, every second breath is voluntarily suspended, so that one breathes much less frequently (or alternatively, with much slower air flows).

One result of this breathing restriction could be an increase in P_{ACO_2} for the same degree of exercise. As the breathing pattern becomes habitual, an increase in tolerance to CO_2 would develop. Although this technique is no longer endorsed by diving authorities, it is still in frequent use as a way of implying diving superiority by prolonging underwater endurance. Also, regulators have less resistance with slow breathing patterns.

'Skip' breathing or 'controlled' breathing not only may produce problems from CO_2 toxicity, oxygen toxicity, nitrogen narcosis and pulmonary barotrauma – it also induces bewilderment among diving respiratory investigators. Nevertheless, it may explain some alleged CO_2 problems encountered by compressed-air scuba divers. The CO_2 levels could be increased because of the other physiological consequences of diving, described above.

Certain divers who have had otherwise unexplained episodes of unconsciousness, have been shown to have a markedly reduced ventilatory response to elevated P_{ACO_2}. In some, the CO_2 toxicity is conjectured to combine with nitrogen narcosis to induce unconsciousness, but in others the depth is too shallow to incriminate significant nitrogen narcosis.

Some of the cases that have lost consciousness with scuba at depths around 30 metres have not only been very experienced divers, but also asthmatics. The traditional CO_2 retention in these subjects may be a contributing factor. Equating the respiratory response to CO_2 among divers, with that of the chronic obstructive pulmonary disease patients or those with sleep apnoea, has little clinical or research merit.

Donald, in 1992, reviewed many of the studies frequently referenced both here and by others, and not only cast considerable doubt on their methodology and conclusions, but also argued against any continuing support for the concept of 'CO_2 retainers' as a separate group of divers.

The relevance of CO_2 retention in the conventional open-circuit scuba diving is still not clear.

Contamination

Some buoyancy vests are fitted with a CO_2 cartridge to inflate the vest in an emergency. A diver who, in a panic situation, inflated and then breathed this gas would rapidly develop CO_2 toxicity.

In limestone cave diving, gas pockets may form under the roof. A diver may be tempted to remove his regulator and breathe in this gas which, not being replenished, will gradually accumulate CO_2. Such a diver may then develop CO_2 toxicity.

Clinical features

These depend on the rate of development and degree of CO_2 retention. They vary from mild compensated respiratory acidosis, detected only by blood gas and electrolyte estimations, to rapid unconsciousness with exposure to high inspired P_{CO_2}. Although CO_2 is a respiratory stimulant, most of its effects are related to the acidosis it produces and are neurologically depressant.

At 1 ATA, a typical subject breathing air to which 3 per cent CO_2 has been added doubles his respiratory minute volume. There is no disturbance of central nervous system function. A 5–6 per cent CO_2 supplement may cause distress and dyspnoea accompanied by an increase, mainly in tidal volume but also in respiratory rate. There is a concomitant rise in blood pressure and pulse rate, and mental confusion and lack of coordination may become apparent. A 10 per cent inspired CO_2 eventually causes a drop in pulse rate and blood pressure and severe mental impairment. A 12–14 per cent level will cause loss of consciousness and eventually death by central respiratory and cardiac depression if continued for a sufficient time ($P_{ACO_2} > 150$ mmHg). A 20–40 per cent inspired CO_2 level rapidly causes midbrain convulsions – extensor spasms – and death.

These effects will occur at progressively lower inspired concentrations with increasing depth, because toxicity depends on partial pressure, and not inspired concentration.

If the inspired CO_2 is allowed to increase gradually (as might occur with a rebreathing set with failing absorbent), the following sequence is observed on land. The subject notices dizziness, unsteadiness, disorientation and restlessness. There is sweating of the forehead and hands, and his face feels flushed, bloated and warm. Respiration is increased in both

CASE REPORT 18.1

WS, a very fit dive instructor experienced two episodes of unconsciousness under similar conditions, about one year apart. They both were associated with diving between 30–50 metres depth, non-stressful and requiring little exertion. They both occurred 10 or more minutes after reaching the sea bed, and there were no problems during descent, and specifically no difficulty with middle-ear autoinflation.

Other divers on the same dives used similar scuba equipment and gases from the same compressor (WS's own dive shop) and experienced no difficulty.

The first episode resulted in a sensation of imminent loss of consciousness, to a severe degree and resulted in him ditching his weight belt and ascending, with help. With the ascent he regained his normal state of awareness. On his second episode he totally lost consciousness and was brought to the surface by one of the companion divers. He was fully conscious and alert within a few minutes of surfacing. Following this dive he was aware of a dull headache.

The only contributory factors that could be ascertained were as follows: He was renowned for consuming extremely small quantities of air and he did admit to employing 'skip breathing' in the earlier part of his diving career – although such a voluntary decision was not made over the recent years. He was also an asthmatic of moderate degree. He had not taken any anti-asthma medication prior to the dives. He then sold his diving practice, and refrained from diving activities.

Provisional diagnosis: a combined carbon dioxide/nitrogen narcosis effect. ?asthma contribution.

Whatever the explanation, remember the maxim that any diving accident not explained and not prevented, will recur under similar conditions.

depth and rate. Muscular fasciculation, incoordination and ataxia are demonstrable, and jerking movements may occur in the limbs. The subject becomes confused, ignores instructions and pursues his task doggedly. Gross tremor and clonic convulsions may appear. Depression of the central nervous system may lead to respiratory paralysis and eventually death if not discontinued.

Under water, the diver may not notice sweating and hot feelings, due to the cool environment. Incoordination and ataxia are much less obvious because movements are slowed through the dense medium, and the elimination of the effect of gravity. Hyperpnoea may not be noted by the diver who is performing hard work or is engrossed in a task. With the rapid development of hypercapnia, there may be no warning symptoms preceding unconsciousness. During the recovery period, the diver may remember an episode of lightheadedness or transitory amblyopia, but these occupy only a few seconds and there is therefore insufficient time to take appropriate action.

A throbbing frontal or bitemporal headache may develop during a slow CO_2 build-up (while still conscious) or after a rapid one (during recuperation).

An exercising diver in the water may have little warning of carbon dioxide toxicity before becoming unconscious.

If the diver is removed from the toxic environment before the onset of apnoea, recovery from an episode of acute CO_2 toxicity is rapid and he appears normal within a few minutes. However, he may complain of nausea, malaise or severe headache for several hours; the headache does not respond to the usual analgesics or ergotamine preparations.

The earlier workers in this field observed the 'CO_2 off-effect', i.e. a brief deterioration in the clinical state when a significant CO_2 exposure is abruptly suspended and the diver breathes normal air.

Carbon dioxide retention enhances nitrogen narcosis (see Chapter 15) and renders the diver more susceptible to oxygen toxicity (see Chapter 17). Conversely, there is evidence that nitrogen narcosis does not exacerbate CO_2 retention by depressing ventilatory response. The hyperoxia of depth may slightly reduce ventilatory drive. It is also believed that CO_2 increases the possibility of decompression sickness by increasing tissue perfusion and by increasing red blood cell agglutination (see Chapter 11).

Prevention and treatment

Familiarizing divers who use rebreathing sets with the syndrome under safe control conditions has been suggested so that appropriate action can be taken at the first indication. In the water there may be few warning symptoms, so this method cannot be relied upon, though it may be of some value in alerting the divers to the problem. Divers should be encouraged to report any unusual symptoms to their companion.

The ideal prevention is by CO_2 monitoring and an alarm system to warn of rising levels. At present, this is practical for recompression chambers, habitats, submarines, etc., but not for self-contained rebreathing apparatus.

If CO_2 levels are not monitored, then attention must be paid to such factors as adequate ventilation of chambers, avoidance of hard physical work, keeping within the safe limits of the CO_2 absorbent system, etc. Even with these precautions, accidents will still happen.

It must be remembered that the percentage of CO_2 in the inspired gas becomes increasingly important as the pressure increases. Although 3 per cent CO_2 in the inspired air at the surface produces little effect, at 30 metres (4 ATA) it is equivalent to breathing 12 per cent on the surface, and would be incapacitating. At very great depth, minimal percentages of CO_2 could be dangerous.

The diver using underwater rebreathing apparatus should be well trained in the immediate action to be taken when CO_2 toxicity is suspected. He should stop and rest, thus reducing muscular activity and CO_2 production. At the same time he should signal his diving partner, as assistance may be required and unconsciousness may be imminent. Either the diver or his companion should flush the counterlung with fresh gas, ditch his weights, and surface by using positive buoyancy. In deep diving, it may be necessary to return slowly to a submersible chamber. On arrival at the surface or submersible chamber, the diver should immediately breathe from the atmosphere.

CASE REPORT 18.2

JF was doing a compass swim using a closed-circuit 100 per cent oxygen rebreathing set at a maximum depth of 5 metres. He had difficulty keeping up with his companion, noticing that his breathing was deep and the gas seemed hot. He ventilated his counterlung with fresh oxygen, but still had breathing difficulty. Just before being called up after 33 minutes in the water, his companion noted that he 'got a new burst of speed, but kept adding more gas to his counterlung'.

On reaching the tailboard of the boat, JF complained that he was nearly out of gas. His eyes were wide open, his face flushed, his respirations panting and spasmodic. He then collapsed and stopped breathing. His facemask was removed and he was given mouth-to-mouth respiration, then 100 per cent oxygen as breathing returned. He was unconscious for 5 minutes and headache and amnesia extended for several hours after the dive.

Oxygen percentage in the counterlung was 80 per cent, and the activity time of the unused absorbent was reduced to 32 minutes (specification 61 minutes). The canister (plus absorbent) from JF's set was placed in another set and a fresh diver exercised in a swimming pool using this. He was unable to continue for more than 5 minutes, due to a classic CO_2 build-up.

Diagnosis: CO_2 toxicity.

Attempts to identify and therefore isolate the 'CO_2 retainers' causing difficulties with open-circuit (scuba and mixed gas divers) have been unsuccessful. Personnel selection is therefore not feasible at this stage. Prevention includes advising against any 'skip breathing' or other gas-conservation techniques, but it is difficult to enforce. Equipment should not impose a significant breathing resistance, even at maximal work loads. The replacement of helium for nitrogen in mixed-gas diving is effective.

First aid treatment simply requires removal from the toxic environment. Maintenance of respiration and circulation may be necessary for a short period. PCO_2 and pH return to normal when adequate alveolar ventilation and circulation are established.

CHRONIC HYPERCAPNIA

The need for defining tolerance limits to CO_2 for long exposures is becoming increasingly important with the development of saturation diving, the use of submersibles and extended submarine patrols (see Chapter 70).

Marked adaptation to exposure to inspired CO_2 levels between 0.5 per cent and 4 per cent has been demonstrated, this adaptation being characterized by an increased tidal volume and a lower respiratory rate. There is a reduction in the ventilatory response to the hypercapnia produced by exercise.

Biochemically, there is a reversal of the initial increase in hydrogen ion concentration, a rise in the plasma bicarbonate, and a fall in the plasma chloride, i.e. a mild compensated respiratory acidosis. There is a slight rise in $PaCO_2$. These latter changes are almost complete in three to five days' exposure, although there is a significant reduction in the ventilatory response in the first 24 hours. There is also a rise in serum calcium and other mineral changes.

While at rest, the average diver can tolerate a surface equivalent of up to 4 per cent inspired CO_2 ($PiCO_2$ of 30 mmHg), without incapacitating physiological changes. During exercise, alveolar ventilation does not increase sufficiently to prevent a significant degree of CO_2 retention, as shown by an elevation of $PaCO_2$. This loss of the ventilatory response to CO_2 (of the order of 20 per cent in submariners) may also be of great significance in the saturation diver, particularly during exercise.

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19

Breathing gas contamination

JOHN PENNEFATHER

INTRODUCTION

In earlier editions of this book, this chapter started by saying that the death of a diver due to breathing a contaminated gas mixture is an uncommon event. Unfortunately, this statement has become less true with the increased use of various forms of mixture diving. Readers need to be aware of the causes of contamination, and also of methods for prevention and treatment of this situation.

AIR COMPRESSORS

The source of compressed air for scuba and some mixed gas systems is a high-pressure compressor system. These take in air at atmospheric pressure and compress it to 200–300 ATA. The air is then filtered, dried and stored in storage cylinders and decanted into scuba cylinders. With small compressor units it may be purified and then discharged into the diver's cylinder. Some commercial divers use a low-pressure compressor which is connected to a large-volume storage vessel and to a hose that supplies the diver; this is a surface-supplied breathing apparatus (SSBA) and is sometimes colloquially called a Hookah.

If the air were to be compressed to high pressure in one step there would be several problems. First, the gas would become very hot, and large amounts of condensation would then form when the gas cooled. It would also be difficult to design seals to prevent air leakage round the large pistons required. Because of these problems most compressors compress the air in three or four stages.

The basic design of a three-stage system is shown in Figure 19.1. In each stage the gas is compressed by a piston that moves up and down in a cylinder with inlet and outlet valves. In the first stage, the air is compressed to about 8 ATA, after which it is cooled and the pressure falls to about 6 ATA. When the gas cools, water vapour will tend to condense, and this is drained off along with any oil that has mixed with the air during the first stage of compression. The second stage of compression raises the pressure to about 40 ATA. Again, the air is cooled and more water is condensed out. The final stage compresses

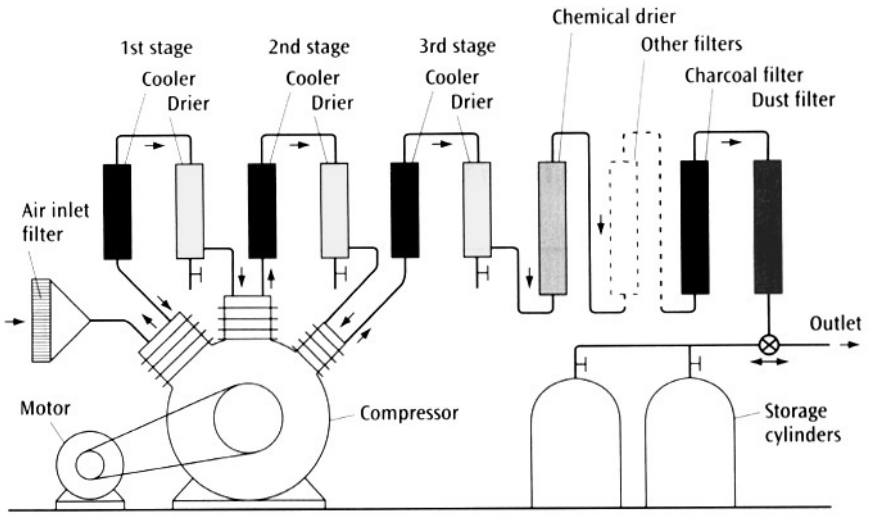

Figure 19.1 *Diagram of three-stage compressor.*

the air to the pressure required for the storage tanks or scuba cylinders. A four-stage compressor differs from the three-stage shown only in that there are four stages, each of which covers a smaller pressure step.

The cylinder, piston and valves in each stage of the compressor is smaller than those of the preceding stage because the volume of gas decreases in accordance with Boyle's law. In the example above, if the volume of the first stage cylinder was 1000 ml, to match the pressures given the second stage needs to be less than 200 ml, and the third stage about 25 ml.

The moisture that condenses is the water vapour in the air that was compressed. If the relative humidity of the air entering the compressor was 50 per cent, condensation would occur when the air pressure is increased to more than 2 ATA (if the temperature remains constant). It is difficult and expensive to remove water from compressed air by chemical means, and because of this it is common practice to have a spring-loaded valve after the final stage. This value only opens to discharge air when the compressor outlet pressure has built up to value more than the working pressure of the storage cylinders. This increases the amount of water that will be removed by condensation. As an extreme example, consider if the discharge valve is set at 400 ATA and the cylinders are filled to 200 ATA. Without any other drier the air going to the cylinders will have a relative humidity of 50 per cent.

A refrigeration system is sometimes used to increase condensation from the air. Both a high outlet valve pressure and refrigeration will also increase the removal of any contaminants that condense with, or in, the water.

There is a divergence of opinion on how to lubricate compressors. Some advocate synthetic oils, while others advocate natural oils. The manufacturer's instructions are the best guide to selecting the lubricant. Some attempts have been made to design compressors that do not need lubrication, while another option is to use water as coolant and lubricant. Neither approach has won general acceptance.

Compressors range in size from small units that can be carried by one person, to large units that weigh several tons.

GASES FOR MIXED GAS AND TECHNICAL DIVING

The gas for military mixture diving is generally prepared in large cylinders and then pumped into the smaller cylinders used by the diver. The large cylinders are generally prepared by mixing gases from a high-purity supply, and the final mix is analysed to check that the composition is correct. This process is costly in time and equipment.

For amateur nitrox (enriched air) diving, the mix is often produced by a modified version of the

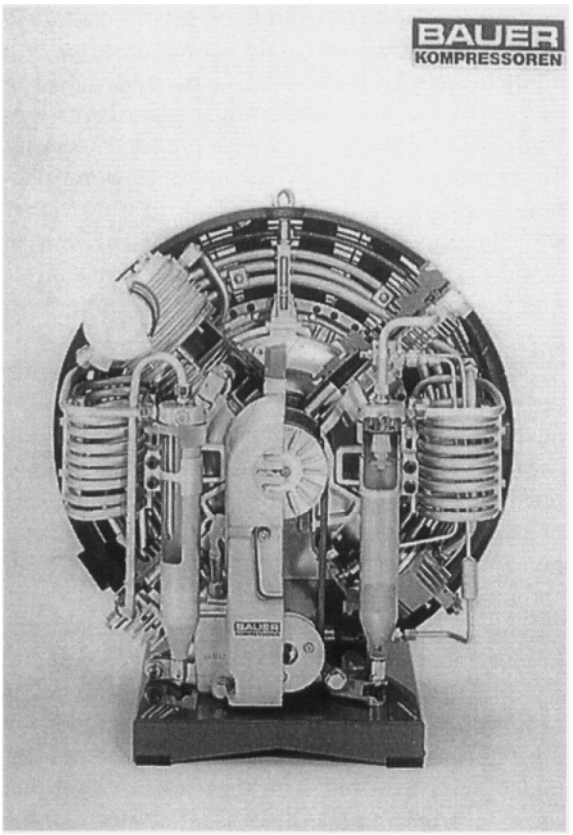

Figure 19.2 *A portable four-cylinder Bauer air compressor in which some components have been cut away to show the internal design. To reduce the size of the compressor, the cylinders are arranged in a radial pattern. The spiral coils are to cool the air between stages of compression; cooling is aided by a fan attached to the far side of the unit. Contaminants are removed from the compressed air by chemicals in prepared packs which fit into the two cut-away vertical cylinders in front of the compressor.*

oxygen generator used for the long-term domestic supply of oxygen. For trimix diving, a mixing process where air, oxygen:helium mixtures are added to cylinders by a formula using pressure readings and Boyle's law calculations. Both processes have resulted in the supply of incorrect mixtures. Divers have failed to verify that correct amount of oxygen in the mixture as they rarely have the equipment to test to confirm that the mixing technique has produced the expected concentrations of the diluting gases.

SOURCES OF CONTAMINANTS

A contaminant in compressed air may have been present in the air before compression, added during compression because of some fault in the compressor system, or have been present or generated within the storage system.

There are many potential contaminants in air, particularly if it is compressed in an industrial area. Carbon monoxide and nitrogen oxides are components of polluted city air, in levels that may be toxic. They may also enter the compressed air if the compressor is driven by, or operated near, an internal combustion engine which produces these compounds.

High temperatures ('hot spots') and high pressures within the compressor produce an ideal environment for contaminants to form. Both these factors promote chemical reactions and hence the production of contaminants. If an unsuitable lubricating oil is used in the compressor it may produce oil vapour, which may contaminate the air as oil, or break down to produce volatile hydrocarbons, or burn and form carbon monoxide. The same trouble can also result if the compressor overheats, causing 'cracking' (oil breakdown) or 'flashing' (oil combustion). The air becomes contaminated with volatile hydrocarbons or combustion products such as carbon monoxide and nitrogen oxides.

> *Contaminants in compressed gas may have been present in the gas before compression, added during compression, or resident in the storage system.*

Some divers believe that using an electric compressor prevents carbon monoxide contamination. Certainly it removes one common cause of carbon monoxide – that of the driving motor exhaust – but it does not reduce the other external and internal sources of carbon monoxide which can develop suddenly, or intermittently. Carbon monoxide can be in the air being compressed, i.e. outside the compressor, such as the exhaust of a nearby engine. It can also be produced when oil in the cylinder of the

compressor overheats due to increased friction between the piston and cylinder. Other hydrocarbons may also be formed in this situation.

Overheating may also be caused by poor design or maintenance of the compressor. For example, a restriction in the compressor intake, a dirty filter, excessive length of intake, or a kinked intake hose can cause problems such as overheating and reduced output. Other problems include leaks between the compressor stages, via piping, loose fittings or head gaskets, or around the pistons. General or local overheating beyond the compressors design limits can cause contamination by causing the lubricant to overheat and burn or decompose.

'I have an electric compressor, so I can't get carbon monoxide poisoning' – Nonsense.

Oil may also contaminate the air as oil vapour by passing around the piston rings to enter the supply. This is most likely when the rings are damaged, or if the air intake is restricted.

Air contamination from residues within the storage cylinder is not common. Residues of cleaning and scouring materials and scale formed by rusting can contribute vapours or dust if the cleaning operation is not conducted properly, or if the cylinder or storage vessel is allowed to deteriorate. Water may be introduced into storage cylinders if there has been a failure of the drying system. There have been problems with paint systems that have been used to protect the interior of steel storage cylinders. The pressure cycling can cause the release of solvent vapour.

The lubricants used on regulators and reducers cause fears of contamination. For example, a diver may taste the lubricant in his regulator and think his air is contaminated. If the wrong lubricants have been used these fears may be justified. With the spread in nitrox diving only lubricants approved for use with high-pressure oxygen should be used because of the risk of fire with other lubricants. Because of the company size, Krytox, a grease produced by E.I. Du Pont, is often more readily available than other products.

The most difficult source of contamination to isolate is intermittent inlet contamination. An example was seen at an Australian firm which had an air compressor inlet on the roof. On rare occasions their air was contaminated with organic chemicals, but only after much work and customer dissatisfaction was the source identified. A nearby factory sometimes used spray painting equipment with an exhaust fan; when the wind was in the wrong direction, these fumes would blow across to the compressor inlet.

There is a low risk of contamination in mixture diving when the source gases are gases purified by liquefaction. This reduces the risk of contamination because most of the potential impurities are separated by their higher boiling points. For mixtures prepared by mixing compressed air with other components there are potential problems with the air and in the mixing process.

There is a possibility of increasing the concentration of trace contaminants when using recycled and reclaimed gases for deep diving operations, and gases compressed in submarines. The compounds that are not removed in any purification process can accumulate with recycling until they reach a level that causes a problem. For this reason, a screening gas analysis program is highly desirable.

With an increase in depth, and hence partial pressure of contaminants, toxicity will increase.

GAS PURITY STANDARDS

Standards have been prepared which specify the composition and the maximum concentration of contaminants in breathing air and for gases used in deep diving. Greater purity is demanded for gases used in deep diving because of the effect of greater pressures.

The levels used in the discussion below are based on the standards used by US Navy and the British Sub Aqua Club. Table 19.1 also shows the Australian and New Zealand Standard. In most countries, the operators will be required to follow a local specification for

Table 19.1 *Air purity standards*

	British Sub-Aqua Club	A/NZ Standard (CZ 2299–1979)	US Navy Diving Manual (1996)
Oxygen (%)	21 + 0.5	20–22 by volume	20–22 by volume
Carbon dioxide (%)	0.03 max. (300 p.p.m.)	900 mg/m^3 max.	(1000 p.p.m.)
Carbon monoxide	10 p.p.m. max	11 mg/m^3 max.	(20 p.p.m.)
Oil (mg/m^3)	1 max.	1 max. (cylinder 12 MPa)	5 max. Oil mist, particulates, 5 mg/m^3
Nitrogen oxides	<1 p.p.m.	—	—
Water	No condensation above 40°F, 5°C	130 max.	—
Odour and taste	Nil	Not objectionable	Not objectionable
Total hydrocarbons as methane	—	—	25 p.p.m.

dry breathing air which is similar to these standards for breathing air. The discussion could lead to the opinion that most limits are rather conservative. Safety margins are incorporated for two reasons. First, the standards are generally based on extrapolation of the effects of the contaminants in isolation at 1 ATA. This might not be entirely valid for contaminants in combination at high pressures. Second, a safety margin will help to allow for any deterioration in the air quality between tests.

It is important to note that standards are only a list of the maximum allowed concentration of some common impurities. Air might meet these specifications and still contain toxic substances. A greater variety of contamination problems occur in caisson work where industrial equipment is being operated, with mineral dust from excavation and blasting the most common problems. Unusual contamination has also occurred in recompression chamber operations, especially if therapeutic or research equipment is used in the chamber. Because exposure times are generally greater in chambers, the toxic contamination has more time to exert its effect, particularly in saturation dives. Toxic substances that may be present include: mercury from manometers; ammonia or freons from leaking air-conditioning plants; and anaesthetic residues and other vapours from pharmaceutical preparations used during hyperbaric treatments.

In using gas mixtures prepared for deep diving, problems can result because of the great pressure at which the mixtures are used. This will increase the risk of toxicity because of the higher partial pressures of contaminants. For example, at 1 ATA a carbon dioxide concentration of 2 per cent in inspired air has little effect. However, at 5 ATA a carbon dioxide concentration of 2 per cent may cause unconsciousness, as would a 0.2 per cent concentration at 50 ATA. Therefore, standards may need adjustment if used for depths and times greater than assumed by their designers.

In avoiding toxicological problems in chambers the basic rule must be, 'if in doubt, leave it out'. Useful guidelines are available from reference to experience with chronic exposures in spacecraft and nuclear submarines.

The reasons for listing the components and the concentrations commonly specified are outlined below.

Nitrogen and oxygen

The concentration of oxygen in compressed air standards is close to the level in clean dry air. Any significant deviation is most unusual. If the nitrogen concentration was elevated, it could increase the risk of decompression sickness, narcosis or hypoxia. If

the oxygen concentration was increased, the risk of oxygen toxicity – and fire hazards in hyperbaric chambers – would increase. The oxygen level may be elevated by connecting to a bulk oxygen supply; this may be accidental, but it has been deliberate in the misguided belief that increasing oxygen concentration will increase the endurance available from a cylinder.

Carbon dioxide

A typical specified carbon dioxide level of 0.03 per cent means that at 10 ATA the partial pressure of carbon dioxide would still be well below that required to cause any physiological effect. The British and Australian standards are probably too strict, because other standards set a maximum limit of 0.1 per cent. This would not be toxic to the depth limits of compressed air diving and would be easier for compressor operators to meet. The carbon dioxide level, even if it is within the specification used, should be considered in relation to the level in the ambient air. An excess may indicate lubricant breakdown or local contamination from an external source.

Carbon monoxide

This toxic gas reacts with haemoglobin to form carboxyhaemoglobin, which cannot carry oxygen. If sufficient haemoglobin reacts with carbon monoxide the diver will become hypoxic. The formation of carboxyhaemoglobin will also interfere with the transport of carbon dioxide by preventing its combination with haemoglobin. Carbon monoxide also has a toxic effect at the cellular level, and this may be more significant clinically.

The frequently described 'cherry red' colour of these victims is an unreliable clinical sign, especially in those with cardiorespiratory impairment. Exertion and increased ventilation will hasten the development of symptoms. Subjects with a low haemoglobin level are more susceptible to carbon monoxide poisoning.

The concentrations of carbon monoxide required for poisoning are considerably greater than the maximum level (10 or 20 parts per million; ppm) normally specified. A limit of 100 ppm is a suggested maximum level for industrial workers exposed for up to 8 hours a day. For divers breathing air, the higher partial pressure of oxygen tends to protect against the effects of increased carbon monoxide partial pressure while at depth. The toxic limits of carbon monoxide at depth, and how they are modified by varying ambient and oxygen partial pressures, have not been established.

It would be expected that divers are at greatest risk as they surface and lose the protection offered by increased transport of oxygen in plasma. This protection occurs at depth when the partial pressure of oxygen in inspired air is elevated. For excursion air diving the standards offers a safety margin in that 20 ppm of carbon monoxide would not cause a serious danger at 10 ATA, even if there was no protection from the increased partial pressure of oxygen.

A lower maximum carbon monoxide concentration is needed for deep and saturation divers; this is

CASE REPORT 19.1

An experienced diver dived in an area subject to tidal currents. He planned to dive at slack water, and anchored his boat a short time before the low tide. The hookah compressor was correctly arranged with the inlet upwind of the exhaust and the dive commenced. After an hour at 10 metres the diver felt dizzy and lost consciousness but was fortunately pulled aboard by his attendant and revived.

Diagnosis: carbon monoxide poisoning, confirmed by blood analysis.

Explanation: as the tide turned, so did the boat. This put the compressor inlet downwind of the motor exhaust. The carbon monoxide from the exhaust was drawn into the compressor inlet and breathed under pressure by the diver.

because the exposure times are longer. Also, the oxygen partial pressure is usually limited to about 0.4 ATA, so the protection from an elevated oxygen pressure is reduced.

Oil*

Oil, occurring as a mist or vapour, can cause compressed air to have an unpleasant odour and taste. Its direct, toxic effects in normal people are not known except that in high concentrations it can cause lipoid pneumonia. In some people low concentrations of oil vapour can trigger asthma. Condensed oil, especially if combined with solid residues, can cause malfunctions of equipment. The other problem with oil is that it can decompose if overheating occurs, generating hydrocarbons and toxic compounds of carbon, nitrogen and sulphur depending on the oil composition.

Some compressed air standards distinguish oil from other hydrocarbons, and specify maximum limits for each. Most hydrocarbons in high-pressure areas can be serious fire hazards, while some have other undesirable effects, such as being carcinogenic.

Water

The control of water vapour is often needed to protect the equipment rather than the diver. Many people find compressed air unpleasantly dry; indeed, the dryness and cooling from the expansion can trigger asthma in people with hyperreactive airways.

The standards are needed to reduce corrosion damage to equipment. A low water concentration is also sometimes claimed to be needed to prevent the formation of ice that might block the supply when diving in cold water. Others working in this field consider that this problem has been overestimated because the areas susceptible to blockage are at lower pressure than the cylinder. The air will not be saturated in such areas because the gas has expanded. Water condensation can also impair the efficiency of the filters used to remove other contaminants; this is more common with certain molecular sieve filter systems.

Deaths have been reported from diving with steel cylinders containing water. If these cylinders are left unused for long periods, rusting occurs which consumes oxygen and leaves a mixture that may cause death from hypoxia. The other problem is that the rusting process weakens the cylinder, perhaps causing it to become an 'unguided missile' if the gas rapidly discharges. Indeed, deaths have been caused by exploding cylinders.

Solid particles*

The presence of these has to be controlled by filters to protect both the diver and his equipment. The effect of the particles depends on their size and composition. For example, particles such as pollens can cause hay fever and asthma in susceptible divers, while other particles have various undesirable physiological effects depending on their size and composition. Any dust which causes coughing could be particularly hazardous, especially for a novice diver.

In diving equipment, abrasive particles such as mineral dust would accelerate wear on the equipment by abrasive erosion. Soluble particles such as salt crystals can accelerate corrosion by promoting electrolysis. Organic dust can also contribute to a fire hazard. There have also been reports of filters breaking down and letting material through so that particles of filter material are contributed to the air supply.

Nitrogen dioxide and nitrous oxide

Some of the oxides of nitrogen – and nitrogen dioxide in particular – are intensely irritating, especially to the lungs, eyes and throat. These symptoms can occur when the subject is exposed to gas with a concentration of nitrogen dioxide greater than 10 ppm. At lower concentrations the initial symptoms are slight, and may not be noticed, or may even disappear. After a latent period of 2 to 20 hours further signs, which may be precipitated by exertion, appear. Coughing, difficulty in breathing, cyanosis and haemoptysis accompany the development of pulmonary oedema; unconsciousness usually follows.

* See note at the end of the chapter

The typical maximum level, 1 ppm, is also the maximum allowed level for 24 hour exposure in other standards. If the effect is increased with pressure then 0.1 ppm may be a more appropriate limit – a level which is often exceeded in industrial cities.

Nitrous oxide is an anaesthetic agent, but only at high concentrations. A low concentration of the gas is specified because if it is generated within the compressor, a precursor – nitric oxide – must have been formed. Nitric oxide can also be converted to nitrogen dioxide at higher pressures and temperatures. Hence, a compressor which adds nitrous oxide to the air being compressed can also form nitrogen dioxide.

Odour and taste

These are controlled to avoid the use of air that is unpleasant to breathe. It is also a back-up for the other standards because if the air has an odour, it contains an impurity.

PREVENTION

Contamination should not occur if clean dry air is pumped by a suitable, well-maintained compressor, into clean tanks. Any deviation from this will lead to the risk of contamination.

Prevention of contamination involves the use of suitable well-maintained compressors, adequate filters, clean tanks and regular analysis of the gas.

Filtering will be necessary to remove any contaminants introduced by compression, but will also be needed if the air that is being compressed comes from a polluted area. Water removal will be needed in most situations. The choice of filtering agents and the frequency of replacement is a specialized field of engineering and should be considered with experts in the field. The following methods and agents are commonly used:

- Silica gel, to remove water vapour.
- Activated alumina, to remove water vapour.
- Activated charcoal, to remove oil mist and volatile hydrocarbons.
- Activated zeolites, and molecular sieves, to remove oil and water.
- Reverse-flow or centrifugal filters, to remove solids and large liquid drops.
- Hopcalite, a mixture of chemicals that oxidizes carbon monoxide.
- Soda lime, to remove carbon dioxide.
- Cryogenic cooling, to remove impurities with a higher boiling point, normally water and carbon dioxide.

A sanitary pad, used by some abalone divers in the air supply line to filter excess water, oil and solid particles, is not suitable for this use.

Some companies incorporate several filtering agents into a cartridge, which in turn simplifies the servicing of the compressor.

TREATMENT

For most of the conditions caused by contaminated air the first step is to replace the contaminated air supply. Rest, breathing 100 per cent oxygen and general first aid measures may be required, but in more severe cases resuscitation may be needed.

It is generally believed that serious cases of carbon monoxide toxicity benefit from hyperbaric oxygen therapy.

Treatment of nitrogen dioxide poisoning requires rest in all cases to help prevent the condition progressing. If the exposure was thought to be to a toxic concentration, or if the patient develops further symptoms, then 100 per cent oxygen is indicated. If pulmonary oedema develops, then this should be appropriately managed.

DETECTION OF CONTAMINATION

The accurate assessment of the concentration of contaminants is best left to specialists, such as air pollution analysts. The tests outlined will give the user a reasonable assessment of air quality. However, some of these tests should not be used for samples where a death or legal action may be involved, because they require large amounts of air for an imprecise answer.

For most compressor operators the purchase of an indicating tube gas analyser system is a sound investment. These are made by Mine Safety Apparatus, Auer, Drager, and other firms. The devices operate by passing a metered volume of air through a glass tube filled with chemicals. These chemicals react with the contaminant to cause a colour change, and a scale on the tube indicates the amount of contaminant present in the sample. Tubes from different makers cannot be mixed because the tube systems use different flows and volumes of gas.

The **oxygen concentration** may be checked using an oxygen electrode, analyser or indicator tube. This is particularly important for diving with gases other than air.

Carbon dioxide can be measured using an indicating tube or a variety of chemical and physical techniques. Infra-red absorption is commonly used.

Oil and dust can be determined by filtering and then weighing the filter, the increase in the dry weight indicating the weight of oil and dust. A solvent such as hexane may be used to dissolve the oil, the remaining weight being particulate matter. This procedure requires an accurate balance as a full cylinder (2000 litres; 72 ft^3) should contain less than 2 mg of oil. An indication of the presence of oil can be obtained by directing a jet of air on to a clean sheet of white paper and then examining the paper under ultra-violet light. Some, but not all, oils fluoresce.

Nitrogen oxides may be detected using indicating tubes. These may also be used for detection of water vapour but a method involving a measurement of the dew point is more suitable.

Combinations of gas chromatography and mass spectrometer systems are often needed to obtain an accurate identification of trace contaminants. However, these instruments need a competent operator and a large stock of reference samples to provide a satisfactory service. Laboratories involved with air pollution may be able to provide these facilities.

RECOMMENDED READING

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20

High-pressure neurological syndrome

CHRIS LOWRY

HISTORY

Alternative respiratory gases to compressed air have been sought because of the restriction to effective diving at depths greater than 40–60 metres of seawater due to nitrogen narcosis (see Chapter 15). Projection of knowledge concerning inert gas narcosis, especially lipid solubilities, suggested that substituting helium for nitrogen would prevent severe narcosis until over 40 ATA. However, in the 1960s difficulties were encountered beyond a depth of 200 metres (pressure of 20 ATA), using a helium/oxygen breathing mixture. This syndrome, which is also characterized by a disturbance to the nervous system, is quite different to the effects of nitrogen (i.e. inert gas narcosis). As the most prominent feature noted was tremors the condition was initially referred to as helium tremors,[1] although it is now realized that helium itself is not the cause.[2]

In retrospect, a series of experiments in the 1880s recorded abnormal excitement, disturbed locomotion and paralysis in marine animals exposed to a high-pressure environment. During the 1920s, a series of publications dealt with the effects of high hydrostatic pressures. Halsey[3] cites a paper published in 1936 where manifestations, which may have been caused by the effects of high pressure on the nervous system, were reported in vertebrate animals. Most of these effects developed at pressures greater than 100 ATA.

In the 1960s British, American, Russian and French investigators noted tremors and performance impairment in divers compressed to depths of 200 to 400 metres of seawater (20–40 ATA).[4] Coarse tremors were often associated with other symptoms such as nausea, vomiting, dizziness and vertigo. Decreased ability to carry out fine movements was also observed. Similar changes were noted in animals which, under further pressure, progressed to generalized convulsions.

This complex of features, which has become known as the high-pressure neurological syndrome (HPNS) or the high-pressure nervous syndrome, is described in terms of neurological, psychological and EEG changes. HPNS should not be confused with inert gas narcosis (see Chapter 15), neurological decompression sickness (see Chapter 12), or central nervous system oxygen toxicity (see Chapter 17) or other gas toxicities.

AETIOLOGY AND LABORATORY STUDIES

HPNS has been observed in all animals studied that have a central nervous system at least as complex as that of a flat worm.[5] Studies in non-air-breathing aquatic animals have demonstrated that the effect is not dependent on elevated gas pressure, and is at least partly due to increased hydrostatic pressure.[3] In air-breathing animals, including humans, HPNS develops while breathing helium and oxygen under high ambient pressure. These gases may modify the manifestations of HPNS,[6] complicating comparative studies.

Animals breathing helium/oxygen under increasing pressure develop fine, and then coarse tremors. These proceed to localized myoclonic episodes and then to generalized clonic seizures. If compression is stopped, the animal will continue to show this seizure activity for up to 12 hours. A reduction in pressure will relieve the symptoms, but if compression is continued tonic–clonic seizures may develop and proceed to death. HPNS is reversible up to a certain stage. Using a slower rate of compression can increase the depths at which convulsions occur.

The addition of nitrogen, hydrogen or nitrous oxide to helium/oxygen mixtures significantly delays the onset of both convulsions and tremor. The antitremor effect is only about one-half that of the anticonvulsant effect. The potency of these gases in alleviating some features of HPNS is proportional to their narcotic potency.

Increased hydrostatic pressure increases excitability of the central nervous system,[7] and this may also be counteracted to some degree by the use of narcotic drugs.[8] These agents appear to act at different locations, and therefore a different clinical pattern develops if HPNS is modified by nitrogen, barbiturates or ketamine. Barbiturates and anticonvulsants also significantly elevate the tremor and convulsion threshold pressures, and may be synergistic with narcotic and anaesthetic gases. Studies in rats indicate that some of the adverse symptoms of HPNS can be reduced by intravenous alcohol; however, at higher doses a characteristic pattern of unsteady locomotion was observed.[9]

The exact mechanism of production of HPNS is not understood. In the past, some aetiological factors that have been proposed include a temperature effect, gas-induced osmosis, a modified form of inert gas narcosis, and neurological hypoxia or hypercapnia caused by the respiratory limitations imposed by increased gas density. However, Halsey presented evidence that tends to discount these theories.[3]

There is considerable evidence for the role of neurotransmitters in the pathogenesis of HPNS. These include gamma-amino butyric acid (GABA), dopamine, serotonin, acetylcholine, and *N*-methyl-D-aspartate (NMDA). The mono-amine-depleting drug reserpine lowers the pressure required to produce convulsions. Drugs which enhance the activity of GABA (e.g. sodium valproate) prevent or reduce some of the changes associated with the syndrome.

Focal injection of NMDA antagonists in rats has been shown to be protective against convulsions. At 81 ATA, primates pre-treated with an NMDA receptor antagonist showed a delayed onset of face tremor and myoclonus with abolished severe whole body tremor and seizure activity. The EEG increase in alpha activity was also abolished, indicating that NMDA transmission plays a significant part in the manifestations of HPNS.[10]

The serotonin syndrome has features similar to HPNS. In rats, a modified form of the syndrome appears at increased pressure – consistent with the hypothesis that elevation of 5-hydroxytryptamine (5-HT) or activation of 5-HT receptors has occurred. Elevation of striatal dopamine in rats exposed to pressure can be blocked by 5-HT receptor antagonists and, concurrently, observable motor features of HPNS reduced.[11]

Changes in neuronal calcium ions induced by high-pressure helium have been postulated as a mechanism for the excitatory phenomena of HPNS.[12]

CLINICAL FEATURES[4,8,13]

The components of the breathing gas mixture, the rate of compression and the time for adaptation all influence the presentation of HPNS. When breathing helium/oxygen, mild effects are seen at pressures of 100 metres, become marked at 185 metres and debilitating at over 300 metres. Adding 5–10 per cent

nitrogen (or hydrogen) to the helium/oxygen respiratory gas mixture permits useful work to be performed after more rapid compression and/or deeper exposure. There is a marked individual variation in susceptibility, however.

Symptoms

A wide variety of symptoms have been reported from various studies, but most seem to involve a disturbance of central nervous system function. Effects reported include: tremor of the hands and arms even extending to the whole body; occasional muscle jerks; light-headedness or dizziness; headache; euphoria; drowsiness; and impending loss of consciousness. There is a tendency to fall asleep if not stimulated. Incoordination, feelings of 'tenseness' or 'nervousness', fatigue, visual disturbances, and dyspnoea are also common. Gastrointestinal symptoms such as nausea (sometimes progressing to vomiting), epigastric sensations, diarrhoea, loss of appetite and aversion to food (leading to weight loss in prolonged exposures) and abdominal cramps may be due to a disturbance of the vestibuloocular reflex.

Dyspnoea at depths in excess of 300 metres may be a manifestation of HPNS, the syndrome often developing or intensifying suddenly and perhaps being precipitated by exercise. The distress is greater during inspiration, but surprisingly it is ameliorated by using nitrogen in the breathing mixture, increasing gas density and thus the work of breathing. Dyspnoea might be related to incoordination of neuromuscular control of breathing, or involvement of the reflex control and feedback systems with a mismatch of afferent and efferent impulses. It can develop or intensify suddenly, and it may be precipitated by exercise and/or increasing the pulmonary ventilation to close to the maximum voluntary ventilation. The distress is greater during inspiration, even though the increased gas density objectively restricts expiratory flow. Higher oxygen concentration does not affect this dyspnoea. Arterial blood gases and pH are usually normal.

Dyspnoea due to HPNS should be differentiated from the diving-associated dyspnoea, which has other causes (see Chapter 42).

Signs

Tremor may appear as shallow as 150 metres (16 ATA), and intensifies progressively with increasing depth/pressure. The tremor is seen both at rest and on movement. The tremor frequency is 8–12 Hz, which differs from that due to Parkinson's and cerebellar disease, which have a frequency of 3 to 8 Hz. It may be thought of as an extension or exaggeration of the normal physiological resting tremor. The amplitude, but not the frequency, of the tremor increases with faster rates of compression or increasing absolute pressure. There is a gradual return towards normal following cessation of compression, but this may not be complete until the diver is decompressed. Divers learn to adapt to the tremor, which leads to an apparent improvement after a day or two.

Opsoclonus is an involuntary, constant, random jittering of the eyes, and is said to be one of the earliest signs of HPNS. It develops at a depth of 160 metres (17 ATA).[8]

Hyper-reflexia and dysmetria are described at 25 ATA, and myoclonus at 50–60 ATA. Other neurological disturbances include fasciculations and myoclonic jerks. Convulsions, described in animals, have not been seen in humans.

Disturbances of long-term memory and a decrease in psychomotor performance have been reported following exposures that produced HPNS. The performance impairment abates somewhat during a stay at constant pressure, but at depths greater than 300 metres full recovery has not been recorded. Other neuropsychological changes have been reported in some divers. The question remains as to what degree of cognitive performance decrement is acceptable from an occupational and safety standpoint.

Psychomotor tests involving manual dexterity reveal a considerable performance decrement, correlated with the tremor, and averaging 1 per cent, for each 20 metres of depth. Performance gradually starts to return towards normal levels after 90 minutes at a constant pressure.

Electrophysiological changes

The electroencephalographic (EEG) records during exposure of divers reveals an increase in theta activity

and a decrease in alpha waves. Increased theta activity may be seen from depths of 60 metres while breathing air, or 150 metres while breathing helium/oxygen.

Sleep disruptions such as an increase in awake periods, a decrease in sleep stages iii and iv, and rapid eye movement (REM) sleep have been reported at depths of 450 metres.

Somatosensory evoked potentials increase in amplitude, but are accompanied by an increase in threshold for sensory stimulation. Shortened latency of peaks following the initial cortical spike is consistent with a state of hyperexcitability in the brain.

The evoked cortical responses may also be altered during deep dives. A progressive decline in the auditory evoked response, by as much as 50 per cent at 457 metres, has been observed. This may be due to increased sound conduction in high-density gas. Somatic evoked responses increase in amplitude, but are accompanied by an increase in threshold for sensory stimulation.

Visual evoked responses have not shown any consistent changes.

PREVENTION AND CONTROL

It is unlikely that HPNS can be prevented entirely, but several approaches are possible either to delay its development or to modify its clinical presentation.

Reduction in the rate of compression

This reduces the incidence and severity of HPNS, and can be achieved either by a slower overall rate, or by slowing exponentially (punctuated by stoppages to allow for acclimatization as greater depths are reached). Very slow rates of compression improve or even prevent symptoms of HPNS in some subjects. Indeed, nausea can be virtually eliminated. Nevertheless, at depths exceeding 300 metres, even with six days of compression, some signs of HPNS are still present. With increasing depth, symptoms become more severe and greatly limit the ability to perform useful work.[14] Performance decrements induced by compression improve during a stop at constant pressure, but total recovery has not been recorded at pressures greater than 300 metres of seawater (msw).[15]

Modification of the breathing gas mixture

This has been used to postpone or modify both nitrogen narcosis and HPNS. Interestingly, the narcotic effect of nitrogen has been used to counter some of the symptoms of HPNS. Small amounts of nitrogen (5–10 per cent) introduced into the helium/oxygen mixture have been shown markedly to reduce tremor, but not EEG abnormalities.

Hydrogen has also been studied in the breathing gas mixtures.[13,15] Hydrogen is less dense than helium and thus would be better for respiratory mechanics. Being more lipid-soluble than helium, hydrogen has a greater narcotic potency that can be used to reduce some of the symptoms of HPNS. It appears that hydrogen's narcotic potency is too great for it to be used alone with oxygen, but hydrogen/helium/oxygen mixtures with about 50 per cent hydrogen has allowed working dives to 500 metres to be achieved without symptoms of HPNS, and with minimal performance decrement.[13] Electrophysiological changes and sleep disruptions were still present, however.

The advantages of added nitrogen include decreased cost, increased thermal comfort, a reduced distortion of speech, and a reduction in HPNS. The advantages of helium and hydrogen include a reduction in the narcotic effect, and a reduction in the work of breathing.

Drugs

Drugs such as alcohol, anaesthetics and anticonvulsants have been suggested to control HPNS. Ketamine is effective in preventing HPNS in rats, while barbiturates have an anti-convulsive effect over a wide range of pressures. Valproate is effective in baboon experiments in reducing HPNS at pressures above 40 ATA.[16] Other anticonvulsants have only a limited effect. Common anticonvulsant drugs such as phenytoin and carbamazepine had no effect on the prevention of tremor, myoclonus and seizures in rats. This suggests that HPNS seizures are of an unusual type,

and that conventional anticonvulsant treatment would be of limited value for HPNS in man.[17]

Currently, the use of drugs to modify HPNS has no place in human exposure.

Brauer has raised two possible problems in trying to control HPNS. First, the efforts to counter HPNS may only be countering early or type 1 HPNS. By avoiding type 1 HPNS, a situation may be created where the first sign may be so-called type 2 HPNS which, in animals, has been fatal. The second problem is that, in baboons, delaying the development of HPNS can induce a new set of symptoms that may involve brain damage.[5] Also, agents that delay the onset of HPNS may also reduce the acclimatization to the stimulus that is causing the condition.

HPNS is a major limiting factor in deep diving.[15] The extremely long duration of deep dives which involve very slow rates of compression to mitigate HPNS followed by slow decompression to avoid decompression sickness have curbed commercial interest in such diving. Decompression from a 600-metre dive may take up to four weeks. Thus, research into HPNS has not progressed with the same momentum over the past decade as in the previous two decades. Extrapolation from lower primates to humans suggests that human divers are approaching depths at which seizures may be anticipated.

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21

Drowning syndromes: drowning

CARL EDMONDS

TERMINOLOGY

Drowning is defined as the death of an air-breathing animal due to immersion in fluid. When patients lose consciousness due to immersion and aspiration, but subsequently recover, the term 'near drowning' is used. When symptoms are not severe enough to classify as near drowned, another term the 'aspiration syndrome', is employed.

The uncritical acceptance of some animal research, including electrolyte, haematological and pathological observations, have misled many clinicians into management and medicolegal errors. 'Dry drowning' is a post-mortem paradox, especially observed after freshwater drownings.

In divers, and others who submerge after losing consciousness, the pathology of drowning is complicated by the effects of barotrauma in air spaces (middle ear, sinus, facemask, etc.) and decompression artefact.

The terminology of drowning may be applied as follows:

- **Drowning** refers to the death of an air-breathing animal due to immersion in fluid.
- **Delayed drowning** or **secondary drowning** occurs when the victim appears to recover from the incident, but then proceeds to die.
- **Near-drowning** refers to the loss of consciousness from the incident, but not leading to death.
- The **aspiration syndrome** refers to the effects of aspiration of fluid into the lungs, but without loss of consciousness.

There is a continuum in the severity of symptoms and signs between aspiration, near-drowning and drowning. They can be incorporated together as the **drowning syndromes**, for a greater understanding of each. The continuum needs to be appreciated if a rational approach to the management of near-drowning is to be made.

- **Post-immersion syndromes** refer to the disorders that develop after immersion and subsequent rescue; these include pulmonary (infections and inflammations), brain, renal and multisystem complications.

Other classifications have been proposed, based on the type and amount of fluid inhaled, and these will be referred to later.

DEMOGRAPHY

Excellent general reviews on this subject have been presented by Donald,[1] Modell,[2,3] Edmonds,[4] Tabeling,[5] Neuman[6] and others.

The incidence of death by drowning appears to be diminishing in the more civilized countries, from seven per 100 000 in 1978 to around two per 100 000 in the late 1990s. This may not be reflected in the less advanced countries, where statistics are not kept. In the USA, there are about 7000 deaths per year, with about 90 000 cases of near-drowning. Island nations with dense populations, such as Japan and Indonesia, are more vulnerable than large continental nations.

Drowning is second only to motor vehicle trauma as a cause of accidental death in Australia and the USA, and is the major cause in some age groups (since the introduction of seat restraints in motor vehicles). There is an over-representation of young males in most drowning series, both accidental and homicidal, and there is a predictable age distribution for specific types of drowning.

Most swimming pool deaths occur in the very young, surf deaths mostly in teenagers and young adults, ocean deaths of sailors and fishermen throughout the whole adult range, and bathtub drownings are in either babies, the infirm or homicides.

Alcohol consumption is involved in over half of the adult male drownings. This may be due to:

- injudicious risk taking;
- reduced capacity to respond to a threatening situation;
- loss of heat due to peripheral vasodilatation;
- interference with the laryngeal reflex;
- increased tendency to vomit; and/or
- suicidal intentions.

> *Bacchus hath drowned more men than Neptune. (Old English proverb).*

BEHAVIOUR DURING DROWNING

Over the range of animals tested and observed, consciousness is usually lost within 3 minutes of submersion. Observation on experimental drownings[7] showed the typical behaviour of animals to be an immediate struggle for freedom, sometimes with an inhalation. There was then suspension of movement, exhalation of a little air, and frequent swallowing. Later, there was a violent struggle for survival, probable loss of consciousness followed by convulsive movements and the exhalation of air with spasmodic inspiratory efforts, prior to death.

Observations of human drownings[8,9] parallel the animal experiments, involving a panic reaction with violent struggling followed by automatic swimming movements. There may be a period of breath-holding and swallowing of large amounts of water. Vomiting may occur, followed by gasping and aspiration of water. Blood-stained froth develops in the airways and the patient convulses, and then dies.

More recently,[10] in observations on children exposed to drown-proofing – as it is euphemistically called – there is usually a failure of the infant to struggle. Breath-holding and automatic but ineffectual paddling-type movements are evident as the infant sinks to the bottom.

For many years, drowning was associated with a 'fight for survival',[2] but this is now changing.[3,4] When a fully conscious human accidentally falls in the water, he usually fights to survive. In other circumstances, drowning may proceed in a quiet and apparently unemotional manner. Examples of quiet drownings include:

- Hyperventilation prior to breath-hold diving (see Chapters 16 and 61) is a common cause of drowning in otherwise fit individuals who are good swimmers. Craig[11] reported eight cases of hyperventilation before breath-hold diving, which resulted in loss of consciousness due to the development of hypoxia. This occurred before the blood carbon dioxide (CO_2) levels rose sufficiently to force the diver to surface and breathe. In these cases loss of consciousness occurred without any obvious warning, and the underwater swimmer then aspirated and drowned quietly.

- Hypothermia and/or cardiac arrhythmias, leading to loss of function and drowning have been well described by Keatinge[12,13] and others.[14]
- Drugs and alcohol increase the incidence of drowning[3,15,16] by impairing judgement and reducing the struggle to survive. It is likely that nitrogen narcosis may have a similar effect in divers.
- Diving problems may produce hypoxia. These include the dilution hypoxic effects with mixed gas breathing and ascent hypoxia (see Chapter 16) and carbon monoxide toxicity due to the interference with oxygen metabolism. They are all likely to cause loss of consciousness without excess CO_2 accumulation, dyspnoea or distress.[17]
- Water aspiration (see Chapter 24). In animals, 2.2 ml of freshwater inhaled per kg body weight drops the PaO_2 to approximately 60 mmHg within 3 minutes, or to 40 mmHg with seawater.[18,19] A similar situation was observed clinically in the salt water aspiration syndrome of divers.[20]
- Other causes of unconsciousness leading to drowning have been described, e.g. cerebral arterial gas embolism, some marine animal envenomations, coincidental medical illnesses such as epilepsy, cerebral haemorrhage, etc.

ANIMAL EXPERIMENTS

In the 1930s many animal experiments were conducted both in Europe and North America which demonstrated that if an animal was immersed and drowned in water containing chemical traces or dyes, these would spread through the tracheobronchial tree to the alveoli surfaces.[7,21,22] In the case of freshwater, this was also absorbed into the bloodstream.[21]

A consistent fall in arterial oxygen content was observed, followed by a rise in arterial CO_2 and sometimes ventricular fibrillation.[23,24]

Swann and his colleagues from Texas, in a series of accurate but misleading experiments,[25,26] flooded animals' lungs with fresh or salt water, and demonstrated the significant differences between the two, attributable to osmotic pressures. In both cases, flooding of the lungs produced a reduction in PaO_2 and pH, with a rise in the $PaCO_2$; this was attributable to airway obstruction.

Because **freshwater** was osmotically much weaker than blood, it moved into the bloodstream and produced haemodilution – reducing most of the blood concentrations of proteins, sodium, chloride, etc. The subsequent reduction in the

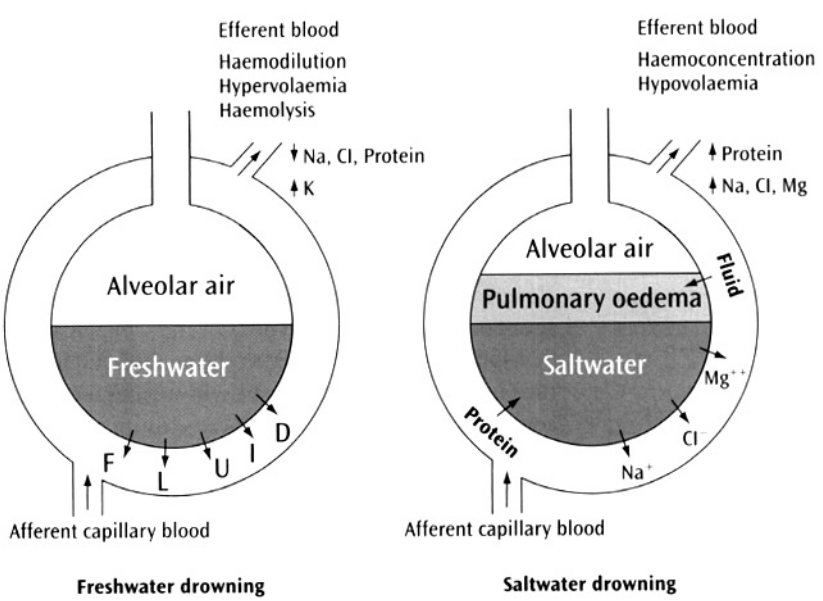

Figure 21.1 *Biochemical and circulatory changes after flooding of animal lungs with freshwater and saltwater.*

osmotic pressure of the blood caused haemolysis and a liberation of both haemoglobin and potassium, with resultant metabolic and renal complications. Deaths were often cardiac in nature and due to ventricular fibrillation.

When, however, the animals' lungs were flooded with seawater – which has a higher osmotic concentration than blood – water was drawn from the bloodstream into the lungs, producing pulmonary oedema and haemoconcentration, and this in turn causing an increase in the haematocrit, blood proteins and electrolytes.

For many years physicians attempted to correct these presumed electrolyte, metabolic and cardiac abnormalities in human drownings, but their cases did not conform to the animal model. Earlier workers had shown that in drowned dogs there were still large volumes of air in the lungs,[7] as occurs in humans – unlike the Texan model.

Colebatch and Halmagyi, working in Australia in 1961, produced an animal model that was more relevant to the clinical management of patients, by aspiration of only 1–3 ml/kg body weight.[27–32] Using these smaller volumes, these workers demonstrated the dominance of arterial hypoxia, not directly proportional to the amount of fluid inhaled. Pulmonary hypertension, vagal inhibition and reduced compliance were also observed. Seawater aspiration usually caused significant pulmonary oedema, but the freshwater was often absorbed from the lungs within 2–3 minutes. Subsequent animal experiments in which intermediate volumes of aspirant were used[18,19,33] showed that shunting of blood was the predominant cause of persistent arterial hypoxaemia, due to perfusion of blood through non-ventilated areas of lung.

Others have described the effects of different types of aspiration on lung surfactant,[33] and shown that freshwater destroys the surfactant, whereas saltwater dilutes and washes it out.

The clinical findings of human drowning do not reflect the experimental observations in many animal experiments.

'DRY' DROWNING

Many references have been made to the paradox of drowning without any aspiration of fluid. It was stated by Cot in 1931[34] that 10 per cent of victims of drowning do not aspirate water. Rather they died from acute asphyxia, whilst submerged. This is now attributed to reflex laryngospasm.

Virtually every review of drowning during the late twentieth century referred to this belief, without question, although the incidence is often increased to 20 per cent. This conflicts with the other animal data obtained in the 1930s, but was given support in 1985 by Pearn[10] in a fascinating philosophical review of pathophysiology.

In a recent review by Modell in 1993,[3] the concept is reiterated. Three relevant references are given, two of which[16,35] refer to autopsy findings; the third reference relates to one of Modell's own reports,[36] but examination of the data reveals little supporting evidence. Modell states that by the time near-drowning/aspiration victims had reached hospital, 10 of the 81 had a Pa_{O_2} of 80 mmHg or greater. No information was available as to whether those cases were fresh or saltwater victims, though the majority of the total number were in freshwater.

As stated earlier, freshwater is absorbed very rapidly from the lungs, and therefore autopsy findings, and indeed the Pa_{O_2}, cannot really be used to imply (let alone prove) the absence of an aspirant. This is especially so when these investigations are performed some time after the event. Thus, there is good reason to question the literature on 'dry' drowning.

The anaesthetic literature is informative,[37] as it defines and describes laryngospasm as the exaggerated and prolonged response of a protective glottic closure reflex. There is no airflow, until it ceases spontaneously when hypoxia and hypercapnia develop. The indications from the anaesthetic literature imply that laryngospasm will not, by itself, be continued until death. The glottic closure will relax prior to death, allowing the passage of gases and fluids into the lungs – a point which has vital implications in resuscitation.

In the absence of more definite information, it would be prudent to presume that all near-drowning

or drowning victims have aspirated, and base one's first aid and management on this presumption. The effects of the aspiration can proceed and develop further, despite the rescue of the victim from the water.[6,10,35,36,39]

'Dry drowning' could be an artefact of fluid absorption from the lungs, or indicate death from other causes. The present author has never witnessed such a case in saltwater divers.

PATHOLOGY

Death may occur during immersion, soon after immersion, or with delayed complications.

> *In recreational scuba deaths drowning is the commonest cause, but it is usually a secondary effect, with the primary cause leading to loss of consciousness. Drowning reflects the fact that unconsciousness occurred in a watery environment.*

Accidents (dysbarism, medical illnesses, trauma, etc.), while immersed or submerged are likely to result in the secondary complication of drowning, with all its pathological sequelae. Drowning then complicates the interpretation of the diving accident, and contributes to a combined pathology.

Certain external characteristics of drowned victims are common, although these are more specific for immersion than for drowning (see Chapter 51). These include evidence of pale, wrinkled, 'washerwoman's' skin; post-mortem decomposition; lacerations and abrasions from impact with rocks, coral, shells, motor boats and their propellers; and post-mortem injuries from aquatic animals, varying from the nibbling of protuberances (fingers, ears, nose and lips) by crustaceans and fish, to the large tearing wounds of sharks and barracudas.

The presence of large quantities of white, pink or overtly bloody froth in the mouth, nose and airways is more specific for drowning, as opposed to immersion. Sea or freshwater may be detected in the sinuses and perforated middle ears. Blood in these cavities is more indicative of **barotrauma** (implying descent while still alive). If the victim was unconscious during his descent to the sea bed, it is likely that all entrapped gas spaces will show evidence of barotrauma of descent. Thus, if a facemask was worn, then the conjunctiva may be haemorrhagic. If a dry suit was worn, then linear skin bruising ('suit squeeze') is possible.

Haemorrhages may be observed in the victim's sinuses, mastoids and middle ears. The latter may be diagnosed either by otoscopy or examination of the temporal bones, and has been mistakenly interpreted as a specific sign of drowning, instead of barotrauma, by pathologists not versed in the effects of hyperbaria. Conventional pathology teaching claimed that mastoid and middle-ear haemorrhages were indicative of drowning. However, it was explained as early as 1969,[39,40] that these haemorrhages are the sequel of barotrauma, not drowning.

Usually the death is due to hypoxia or to a progressive or irreversible **pulmonary damage** associated with the drowning *per se*, and there are various reasons for this. They include progressive surfactant damage despite rescue, pneumonitis from the aspirant or vomitus, and infections. Even victims who appear normal on arrival at hospital, can deteriorate over the next 6–12 hours. Pulmonary oxygen toxicity, associated with prolonged resuscitation attempts, may also be present.

The lungs are heavy, oedematous and may be haemorrhagic, with pink or white froth occupying the airways (and perhaps even the stomach). Other cases have vomitus aspirated into the airways. In freshwater drowning, fluid in the lungs may be less obvious, and if time has elapsed between aspiration of fluid and death, or during resuscitation, the lungs may be dry. Interstitial emphysema may be noted both on the pleural surface and in the mediastinum. Petechial haemorrhages may be seen under the pleura, probably due to overdistension of the lungs.

Respiratory infections, abscesses, etc. are not infrequent, if death is delayed. Otherwise there is little typical to describe macroscopically.

Histological changes may demonstrate toxic effects both of chemicals and the specific aspirant. The surfactant changes, including denaturation, can progress even after apparent clinical improvement. Usually, the epithelial and endothelial changes, with detachment of the basilar membrane and cellular

disruption have been described.[10] Sand, marine organisms, algae and diatoms may be detected in the lungs.

Histological changes in the lungs are said to be helpful in differentiating freshwater from saltwater drowning. In the freshwater drowning, overdistension of the lungs produces an 'aqueous emphysema' and there are endothelial changes, mitochondrial swelling and alveolar disruption. In the saltwater drownings, there is swelling and vacuolation of cells, a perivascular neutrophilic infiltration and increased numbers of peroxidase-positive granules. The picture represents a chemical pneumonitis, varying with the salinity of the water, the presence of other irritants, and the duration between exposure and death.

The major effects on the **neurological** system are those of hypoxic brain damage with petechial haemorrhages and subsequent cerebral oedema and raised intracranial pressure. Autopsies on drowning cases who have submerged while still alive, although unconscious, may also develop other cranial haemorrhages which are sometimes interpreted as a cause of the accident. Meningeal haemorrhages, both dural and arachnoid, are frequently observed; these are usually not extensive and are quite different to the brain haemorrhages of arterial gas embolism or decompression sickness, or the petechial haemorrhages of asphyxia. They are probably derived from the haemorrhages of descent sinus barotrauma, which ruptures into the cranial cavity as the enclosed gas expands when the body is surfaced. If cardiopulmonary resuscitation (CPR) is continued for more than 6 hours, neuronal changes may be seen in the hippocampus, induced by hypoxia.

The stomach often contains free fluid, water inhaled during the incident, together with debris and organisms.

It was stated[38] that autopsy examination of 118 consecutive drowning cases suggested that 85 per cent aspirate 22 ml of fluid or less per kilogram body weight. It was therefore considered unlikely that drowning victims die acutely of electrolyte imbalance and/or ventricular fibrillation. Death is more likely to be secondary to asphyxia.

In the event of delayed drowning deaths, the lungs, brain or kidneys may all be involved. There is often considerable venous congestion of the viscera, especially the brain, kidneys and other abdominal organs. Hypoxic cerebral necrosis and acute renal tubular necrosis, with blood pigment casts, are both described. The cerebral oedema and other changes are thought to be secondary to the low arterial oxygen levels, i.e. secondary to the pulmonary manifestations.

Biochemical tests designed to verify drowning as the cause of death have been employed. The Gettler test uses a difference of 25 mg NaCl per 100 ml in plasma or serum between the right and left sides of the heart during an autopsy performed within 12 hours of death. Comparison of the plasma total solids and/or specific gravities from each side of the heart may be more reliable. However, neither the Gettler chloride tests nor the specific gravity of serum can be relied upon to definitely establish a diagnosis of death by drowning. Magnesium concentration in the vitreous humor is said to provide some indication of the duration of immersion in seawater.

Identification and comparison of environmental and systemic diatoms in the lungs, blood, kidneys and vertebrae, has been recommended, but this is a complex procedure and not frequently performed. It does not prove drowning – merely the aspiration of water while the circulation was still functional.

The theory and the practice of drowning autopsies are surprisingly contentious for such a common disorder. An autopsy can imply, but not reliably prove, that death is due to drowning. More sophisticated variants of the original animal experiments, showing how fluids and particles are transferred from the air spaces to the body are now available,[41] but the basic principles described above have not been materially altered.

OVERVIEW

There is no distinct division in the initial presentation between aspiration, near-drowning and drowning cases. Aspiration syndromes merge with near-drownings – the intensity of the symptoms and the degree of consciousness often depending on various circumstances, the activity of the victim and the administration of oxygen.

'Near-drowning' cases sometimes die hours or days later, and are thereby re-classified as secondary or delayed drowning.

Some of the apparently 'drowned' victims, because of enthusiastic CPR and capable intensive care, surprisingly recover without serious sequelae.

In general, apart from the treatment of the near-drowned, which is frequently and expertly reviewed,[42,43] very little critical analysis has been directed towards the available literature, and most reviews merely replicate previous presentations. Even less attention has been focused on the drowning problem with scuba deaths and accidents,[4,17] but these will be detailed further in subsequent chapters.

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22

Drowning syndromes: near-drowning

CARL EDMONDS

Human series of near-drowning cases do not show the electrolyte, haematological and cardiac changes seen in animals whose lungs are flooded.

Aspiration causes lung changes and hypoxaemia, which in turn may result in acute respiratory distress syndrome (ARDS), hypoxic brain damage, cardiac, multisystem or renal disease.

Many cases have survived without brain damage despite total immersion for durations of 15–45 minutes. Thus, resuscitation must be implemented energetically.

Many patients deteriorate or die hours or days after rescue and resuscitation, and therefore observation in hospital is required over this time.

Most will recover without sequelae, but those with hypoxic encephalopathy may have neurological residual and neuropsychiatric problems.

OVERVIEW

Clinical series described by Fuller[1,2] in 1963, Griffin[3] in 1966, and by Modell *et al.* in 1976[4] have illustrated the considerable differences between near-drowning humans and those studies of animals with flooded lungs.[5,6] Human cases did, however, reflect the animal experiments in which smaller amounts of aspirant were administered.[7–9]

Considerable consensus has been reached on the manifestations of near-drowning. As the major and initial organ involved is the lung, the physiological consequence is hypoxaemia. The major damaging effects are on the lung and brain, but significant electrolyte disturbances and cardiac arrhythmias are infrequent.

The **initial symptoms** are respiratory, followed by the effects of hypoxia that cause subsequent pulmonary and cerebral damage. Aspiration of vomitus, following the ingestion of seawater, is frequent during the early and the resuscitation phases.

Investigations reveal the presence of: hypoxaemia; a variable arterial carbon dioxide (CO_2); acidosis; the effects of reduced pulmonary compliance; patchy and variable consolidation in the lung X-rays; and a polymorphonuclear leucocytosis. Other abnormalities depend on other organ involvement (brain, heart and kidney).

Excellent series of paediatric drownings have been described,[10] but these are not relevant to this text. In addition, many studies on the relevance of hypothermia to drowning will not be dealt with in this chapter.

SURVIVAL FROM NEAR-DROWNING

Treatment at the scene of an accident is often of little ultimate consequence with many disorders, but in drowning it will often determine whether the victim lives or dies. The standard of first aid and resuscitation training of the rescuers therefore influences outcome.

In human drownings, deterioration after initial resuscitation is frequently recorded, and this influences correct management. The temperature of the water and thus the degree of hypothermia may also be a factor. Poorer results are achieved in warm bath water drownings. In addition, whether the drowning occurs in seawater or freshwater may have some influence on the prospects of recovery, and also on the biochemical and pathological abnormalities of drowning. The chances of recovery from immersion in saltwater appear to be better than in freshwater.

In so-called 'dry' drowning (laryngospasm) the patient is hypoxic and, if rescued in time, makes a rapid recovery. When laryngospasm relaxes and fluid aspiration occurs, as it eventually does, the result is either drowning or near-drowning. The differences relate to the degree of aspiration and the body's response to it.

Other factors which influence outcome include: the presence of chlorine and other chemicals and foreign bodies; the aspiration of stomach contents; the subsequent development of pneumonitis; respiratory infection; haemolysis; renal failure; and coagulopathy.

One likely cause for delayed death is the extensive and progressive lung damage of victims.[11] The acute respiratory distress syndrome (ARDS) develops in 40 per cent of near-drowning cases, hours or days after the aspiration. Other causes include cerebral hypoxia, secondary infections (usually of the lungs), renal failure and iatrogenic effects.

Factors which negatively influence survival have been well documented by Modell[12] and include:

- prolonged immersion;
- delay in effective cardiopulmonary resuscitation;
- severe metabolic acidosis (pH <7.1);
- asystole on admission to hospital;
- fixed dilated pupils; and/or
- a low Glascow coma score (<5).

Nevertheless, none of these predictors is infallible, and survival with normal cerebral function has been reported with all of the above factors. A flat EEG may be reversible.

Claims of extended duration underwater without an air supply have been used to encourage rescuers to persevere with resuscitation efforts. Cases have been reported in which people have been submerged for between 15 and 45 minutes,[13–19] and have survived without neurological sequelae, as well as many other similar cases that have not been reported.

The explanations given for such prolonged durations of survival are as follows:

- **Hypothermia**,[10,17–20] which is protective and develops very rapidly with aspiration of water. In swimmers and divers, hypothermia may well commence even before the incident.
- The '**diving reflex**',[10,19] but this explanation remains contentious. Within seconds of submersion the diving reflex may be triggered by sensory stimulation of the trigeminal nerve, and reflex or voluntary inhibition of the respiratory centre in the medulla. This produces a bradycardia and shunting of the blood to the areas more sensitive to hypoxia, the brain and coronary circulations. The diving reflex is independent of baroreceptor or chemoreceptor inputs, but is more intense in the frightened or startled animal than in those that dive or submerge voluntarily. However, it is not known if this is equally applicable to humans. Typically, water temperatures above 20°C do not inhibit the diving reflex, but it is progressively augmented by lower temperatures.
- **Respiratory gas exchange** in the lungs can continue after submersion. With or without the effects of laryngospasm, there are often several litres of air remaining within the lungs, allowing for exchange of respiratory gases. Whether fluid enters the lungs in an unconscious victim will depend on many factors, including the spatial orientation of the body. A dependent position of the nose and mouth, facing downwards, will not be conducive to fluid replacement of the air in the lungs. Increased pressure (depth) might increase the availability of oxygen uptake through Henry's law. In a comatose state, with low oxygen utiliza-

tion and the effects of hypothermia and the diving reflex, a retained respiratory gas volume might add considerably to the survival time – although it is not often considered in the drowning literature.

- **Gas exchange between the pulmonary blood and the aspirated fluid** might have a marginal effect on prolonging life.

Despite the fact that spectacular and successful rescue can be achieved after prolonged submersion, it is more frequent that this is not so. Many victims lose consciousness and die after only a few minutes of submersion.

PATHOPHYSIOLOGY

The effects of near-drowning[21] are multiple, but the initial and primary insult is to the respiratory system, with hypoxaemia being the inevitable result. The sequence of events which occur with near drowning includes:

1 **Initial submersion.** This may be followed by immediate aspiration or by voluntary breath-holding. Duration of the breath-holding depends on several factors – general physical condition, exercise, prior hyperventilation, psychological factors, etc. (see Chapter 61).
2 **Fluid aspiration.** Laryngeal spasm may follow. While laryngospasm is maintained, the lungs may remain dry. Water is often swallowed and subsequent aspiration of fluid or vomitus may complicate the situation. Eventually, the rising arterial carbon dioxide tension ($PaCO_2$) compels inspiration.
3 **Progressive hypoxaemia.** This may be due to oxygen utilization during voluntary breath-holding, laryngospasm, filling of airways with fluid (water, stomach contents, oedema), pulmonary tissue damage and perfusion of blood through non ventilated areas of lungs. It leads to loss of consciousness and eventually cardiac arrhythmias and circulatory arrest. In most cases there is involuntary aspiration of fluid due to diaphragmatic contraction. The cardiac disorders are usually due to the hypoxaemia, at which time hypoxic brain damage is also likely.

The pathophysiology depends on the time before rescue and the effects of resuscitation.

CLINICAL FEATURES

There is no clear-cut distinction between the near-drowning and the saltwater aspiration syndromes as there is a spectrum of severity between the comatose near-drowning patient and the diver who appears to have a mild, transitory, post-dive, inflammatory/respiratory disorder (see Chapter 24). The **respiratory** manifestations include;

- dyspnoea;
- retrosternal chest pain, increased with inspiration;
- blood-stained, frothy sputum;
- tachypnoea and cyanosis;
- pulmonary crepitations and, occasionally, rhonchi; and/or
- ARDS in 40 per cent of the cases hours or days after the aspiration.

Respiratory function abnormalities include decreases in peak expiratory flow, vital capacity, compliance and ventilation–perfusion ratio, causing persisting hypoxaemia despite resuscitation, for hours or days.

Initial chest X-rays may be normal, or show patchy and changing opacities or non-cardiogenic pulmonary oedema. Significant hypoxia may, nevertheless, be present with a normal chest X-ray.

Complications may include pneumonitis, pulmonary oedema, bronchopneumonia, pulmonary abscess and empyema. Severe pulmonary infections with unusual organisms leading to long-term morbidity have been reported.

Central nervous system effects of hypoxia include impairment of consciousness, convulsions, and focal cerebral damage consistent with hypoxia and cerebral oedema. The level of consciousness ranges from awake or blunted to comatose with decorticate or decerebrate responses. Masseter spasm is common.

Cardiovascular manifestations are also largely the result of hypoxaemia on the heart, with cardiac arrest (which may or may not respond to initial resuscitation) not uncommon. After rescue and

CASE REPORT 22.1

Ernie Hazard, age 35. I was thinking, "This is it. Just take a mouthful of water and it's over". It was very matter of fact. I was at a fork in the road and there was work to do – swim or die. It didn't scare me. I didn't think about my family or anything. It was more businesslike. People think you always have to go for life, but you don't. You can quit. . .

The instinct to breathe underwater is so strong that it overcomes the agony of running out of air. No matter how desperate the drowning person is, he doesn't inhale until he is on the verge of losing consciousness. That is called the 'break point'. . .

The process is filled with desperation and awkwardness. "So this is drowning . . . so this is how my life finally ends. . . I can't die, I have tickets for next week's game". . . The drowning person may even feel embarrassed, as if he has squandered a great fortune. He has an image of people shaking their heads over his dying so senselessly. The drowning may feel as if it's the last, greatest act of stupidity in his life. . . The thought shrieks through the mind during a minute or so that it takes the panicky person to run out of air.

****

Occasionally someone makes it back from this dark world. In 1892, a Scottish doctor, James Lowson, was on a steamship bound for Colombo. Most of the 180 people on board sank with the ship, but Lowson managed to fight his way out of the hold and over the side. . .

"I struck out to reach the surface, only to go further down. Exertion was a serious waste of breath and after 10 or 15 seconds the effort of inspiration could no longer be restrained. It seemed as if I was in a vice which was gradually being screwed up tight until it felt as if the sternum of the spinal column must break. Many years ago my old teacher used to describe how painless and easy death by drowning was – 'like falling about a green field in early summer' – and this flashed across my brain at the time. The 'gulping' efforts became less frequent and the pressure seemed unbearable, but gradually the pain seemed to ease up. I appeared to be in a pleasant dream, although I had enough willpower to think of friends at home and the site of the Grampions, familiar to me as a boy, that was brought into my view. Before losing consciousness the chest pain had completely disappeared and the sensation was actually pleasant".

"When consciousness returned I found myself on the surface. I managed to get a dozen good inspirations. Land was 400 yards distant and I used a veil of silk and then a long wooden plank to assist me to shore. On landing and getting on a sheltered rock, no effort was required to produce copious emesis. After the excitement, sound sleep set in and this lasted three hours, when a profuse diarrhoea came on, evidently brought on by the sea water ingested. Until morning break, all my muscles were in a constant tremor which could not be controlled".

Courtesy of Junger, S. *The Perfect Storm.* Pbl. Fourth Estate, London (1997), with quotes from James Lowson, *The Edinburgh Medical Journal.*

resuscitation, supraventricular tachycardias are frequent, but various other dysrhythmias may occur. When the hypoxic acidotic insult has been severe, hypotension and shock may persist despite re-establishment of cardiac rhythm. The central venous pressure may be elevated due to right-sided heart failure or elevated pulmonary vascular resistance, rather than volume overload. Left ventricular filling pressures (estimated by pulmonary artery catheter wedge pressure) may indicate low intravascular volume. Cardiac output and mixed venous oxygen tension may also be low, indicating hypoperfusion.

Gastrointestinal manifestations may include retching, vomiting and diarrhoea – sometimes of severe degree – and complicate both resuscitation and fluid and electrolyte replacement.

Multisystem failure may develop secondary to the hypoxaemia, acidosis and resultant hypoperfusion. Decreased urinary output occurs initially, but occasionally renal failure develops, due to acute tubular necrosis. Haemoglobinaemia, coagulation disorders and even disseminated intravascular coagulation may complicate the clinical features.

Laboratory findings include decreased arterial oxygen with variable arterial carbon dioxide tensions, metabolic and respiratory acidosis, haemoconcentration, leukocytosis, increased lactic dehydrogenase activity; occasional elevated serum creatinine, and haemolysis, indicated by elevated free haemoglobin levels. Serum electrolytes are usually within the normal range.

The arterial oxygen tension is always low, but the carbon dioxide tension may be either low, normal or elevated.

Although recovery from near-drowning is usually complete, residual neurological deficiencies may persist – in the form of either mental impairment or extrapyramidal disorders. They seem more common in warm water drownings of children, as exemplified by the near-drownings of infants in bath tubs.

The long-term neurological and neuropsychological damage may be present in up to 10 per cent of cases. The organic brain damage may be due to the effects of hypoxaemia and/or the cerebral oedema. Frontal lobe ('dysexecutive') disorder, dysmnesic syndromes, extrapyramidal and spastic syndromes are all recorded.

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23

Drowning syndromes: the management of near-drowning

CHRIS LOWRY

Initial resuscitation attempts may be vital in determining the outcome. All divers should be adept at basic resuscitation. Dive leaders and boat operators should have a more advanced training, including the use of oxygen.

Ventilatory support is the mainstay of treatment of hypoxaemia, but to be effective, endotracheal intubation is usually required.

Causes of near-drowning, which require special management, should be considered. Rescuers should be aware of the possibility of cranial or cervical spine injury.

Resuscitation should continue until the return of acceptable physiological parameters, especially core temperature.

Continuing intensive care management for respiratory failure may be necessary.

Central nervous system preservation remains the main therapeutic challenge. Aggressive treatment regimes have not improved cerebral salvage rates.

AT THE SITE

Rescue and initial resuscitation

The efficacy of prompt initial measures in support of the respiratory and circulatory systems greatly influences the eventual outcome of near-drowning. The rescuer should ensure his or her own physical safety in removing the victim from the water. Expired air resuscitation should be started as soon as possible – even in the water if the rescuer has had training.[1,2] A buoyancy vest or other form of flotation is essential to have any chance of success. Aquatic cardiac massage has been described for scuba divers,[3,4] but is not widely accepted and may cause further delay before effective cardiopulmonary resuscitation (CPR) is instituted. The victim should be kept horizontal as much as possible during and after removal from the water.

Consciousness, respiration, circulation and injury should be rapidly assessed (Fig. 23.1). The patient should be moved with the head in the neutral position if cervical spine injury is suspected. Scuba divers

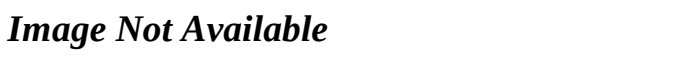

Figure 23.1 *Basic life support. (Reproduced with permission from the Australian Resuscitation Council.)*

are most unlikely to have suffered cervical spine trauma. Vomiting and regurgitation frequently follow submersion, so the patient should be turned on the side for initial assessment and airway clearance. If breathing is detected, the victim is maintained in the 'recovery' position to avoid aspiration of fluid or vomitus. A horizontal position is appropriate for scuba divers who may have suffered gas embolism (see Chapter 6). All patients must be given oxygen if it is available.

Airway

Vomiting and regurgitation frequently follow a submersion incident. Foreign particulate matter causing upper airway obstruction should be immediately removed manually, or later by suction. The patient may now need to be turned supine to continue resuscitation. Obstruction of the upper airway by the tongue is common in the unconscious patient, and either of two methods may be used to do this.

- **Head-tilt/chin-lift** is accomplished by pushing firmly back on the patient's forehead and lifting the chin forward using two fingers under the jaw at the chin. The soft tissues under the chin should not be compressed and, unless mouth-to-nose breathing is to be employed, the mouth should not be completely closed.
- **Jaw-thrust** describes the technique of forward displacement of the lower jaw by lifting it with one hand on either side of the angle of the jaw. Unless cervical spine injury is suspected, this technique is best combined with head-tilt.

Time should not be wasted in clearing water from the lower airways, as only a small amount of water is usually aspirated. Freshwater is rapidly absorbed into the circulation, while saltwater leads to pulmonary oedema where the fluid is at the alveolar level and will continue to form despite attempts at removal.

If airway obstruction is encountered, and has not responded to normal airway management, the **Heimlich manoeuvre** (subdiaphragmatic thrust) has been suggested.[5] This manoeuvre, which was proposed as a routine step to clear water from the airway, has not received the widespread endorsement of resuscitation councils around the world.[1] It should be used with caution and only as a last resort,

because of the risks of regurgitation of gastric contents, rupture of the stomach and causing delay in initiating effective ventilation. Persistent airway obstruction may be due to a foreign body, but other causes include laryngeal oedema or trauma, bronchospasm and pulmonary oedema.

Breathing

Respiration can be assessed by placing one's ear over the victim's mouth while looking for chest movement, listening for air sounds and feeling for the flow of expired air. If breathing is absent, mouth-to-mouth or mouth-to-nose breathing is instituted. Initially, two full breaths of air, with an inspiratory time (for the victim) or 1–1.5 seconds are recommended. For adults, an adequate volume to observe chest movement is about 800 ml. If no chest movement is seen and no air detected in the exhalation phase, then head-tilt or jaw-thrust manoeuvres should be revised. Failing that, further attempts at clearing the airway with the fingers (only if the victim is unconscious!) should be undertaken. With mouth-to-mouth respiration, the rescuer pinches the nose, closing it gently between finger and thumb. Mouth-to-nose rescue breathing may be more suitable in certain situations, such as when marked trismus is present, or when it is difficult to get an effective seal (injury to mouth, dentures, etc).

The rate of chest inflation should be about 12 per minute (one every 5 seconds), with increased rate and decreased volume in young children.

Circulation

The presence of a carotid or femoral pulse should be sought in the unconscious non-breathing victim. This is often difficult because the patient is usually cold and peripherally vasoconstricted. Up to a minute may be required in the hypothermic individual to determine the lack of a pulse. Although it is possible that external cardiac compression (ECC) could precipitate ventricular fibrillation in a hypothermic patient, if in doubt it is safer to commence ECC than not.

If no carotid pulse is detected, ECC should be commenced after two initial breaths. Higher rates of compression are now recommended, with higher outputs achieved at 100 per minute compared with the traditional 60 per minute standard. Controversy still exists over the mechanism of flow in external compression with the evidence for the older 'direct compression' model being challenged by the 'thoracic pump' theory. Abdominal binding, interposed abdominal compression, simultaneous compression–ventilation and active compression–decompression CPR have all been proposed because there is evidence in animal studies of improved cerebral and coronary blood flow. However, these have not yet been evaluated in clinical trials.

Cardiac compression should be performed with the patient supine on a firm surface, and with the legs elevated if possible to improve venous return. The rescuer kneels to the side of the patient, with the heel of their hand placed in line with the sternum. The lower edge of the hand should be about two fingers above the xiphisternum (i.e. compression is of the lower half of the sternum). The second hand should be placed over the first and the compression of the sternum should be about 4–5 cm in adults in the vertical plane. To achieve this the rescuer's elbow should be straight, with the shoulders directly over the sternum. A single rescuer may only be able to achieve rates of 80 per minute because of fatigue, but if several rescuers are present, it may be possible to maintain higher rates.

Further help should be sought immediately, by a third person, if possible, without compromising resuscitation efforts.

Advanced life support and transport

A regional organized emergency medical service (e.g. paramedics) that carries specialized apparatus such as oxygen, endotracheal tubes, suction and intravenous equipment should be activated, if available. In any case, the patient should be transferred to hospital as soon as possible. The early administration of oxygen by suitable positive-pressure apparatus, by people trained in its use, may be the critical factor in saving lives. For this reason such equipment should be carried on all dive boats. Even conscious patients after submersion may have significant pulmonary venous admixture with resultant hypoxaemia. All such patients should receive supplementary oxygen and be further assessed in hospital, as

respiratory and cardiac arrests have occurred after apparently successful rescue.[6]

Although endotracheal intubation remains the best method for securing an airway and achieving adequate ventilation, the necessary expertise may not be available until the victim is transferred to hospital. In such cases, the use of airways such as the laryngeal mask airway may improve ventilation while the patient is being transported to hospital. Other airways such as the pharyngotracheal lumen airway and the Combitube® tube are alternatives; however, these require more training before use, and each has its own problems.

Properly trained and equipped personnel may well undertake some of the procedures described under emergency room management in the field (Fig. 23.2).

IN HOSPITAL

Hospital management is subdivided into initial emergency room management and continuing therapy in the intensive care unit. All patients should receive oxygen (see Chapter 49) while undergoing evaluation.

Emergency room

On arrival, the emphasis is on evaluation and resuscitation of respiratory failure. Preliminary assessment includes airway, circulation and level of consciousness re-evaluation. Continuous monitoring of pulse, blood pressure, pulse oximetry and electrocardiography are commenced.

The severity of the case determines the appropriate care. If submersion victims show no signs of aspiration on arrival in the emergency room, there may be no need for hospital admission. Patients who are asymptomatic and have normal chest auscultation, chest X-ray and arterial blood gases will not subsequently deteriorate,[7–9] and may safely be discharged after 6 hours.

Patients with mild hypoxaemia, auscultatory rales or rhonchi, or chest X-ray changes should be admitted for observation, as they may subsequently deteriorate.

In moderately hypoxaemic patients who have not lost consciousness and who are breathing spontaneously, the use of non-invasive ventilatory support with facemask or nasal continuous positive airway pressure (CPAP) may be an alternative to endotracheal intubation, provided that adequate gas exchange is achieved.[10]

The remaining group should be admitted to an intensive care unit. Patients who are clearly hypoxaemic or who have respiratory symptoms or disturbed consciousness may rapidly deteriorate further due to progressive hypoxaemia.

Patients who are unconscious and/or hypoxaemic will require ventilatory support, and should be intubated and ventilated despite the presence of spontaneous ventilation. Muscle relaxants may need to be administered, and steps should be taken to avoid gastric regurgitation and further pulmonary soiling. This involves the use of cricoid pressure. A nasogastric tube should also be inserted and the stomach emptied.

Some possible causes of drowning in divers

- *Panic, entrapment, equipment failure, etc.*
- *Trauma*
- *Drugs (especially alcohol)*
- *Hyperventilation*
- *Cardiac arrhythmia*
- *Myocardial infarction*
- *Seizure*
- *Subarachnoid haemorrhage*
- *Pulmonary barotrauma, decompression illness*
- *Nitrogen narcosis*
- *Alternobaric vertigo*
- *Syncope of ascent*

Concurrent with resuscitation measures, attempts to establish the cause of drowning should be made (i.e. whether it is a primary or secondary event). A careful search for any other injuries, such as cranial or spinal trauma, internal injuries and long bone fractures should be made. Initial X-ray investigations should include the chest and cervical spine. Cerebrovascular accident, myocardial infarction,

seizure or drug abuse should be suspected if the cause of the incident is not readily apparent. In the scuba diver, other disorders such as pulmonary barotrauma and cerebral arterial gas embolism, may have initiated or complicated the drowning. These conditions may require specific treatment such as recompression, hyperbaric oxygen or drainage of a pneumothorax.

The prime goal of therapy is to overcome major derangement of hypoxaemia, with its subsequent acidosis. The benchmark should be an arterial oxygen tension (Pa_{O_2}) of at least 60 mmHg, and this may be achieved by administration of oxygen by mask in milder cases, possibly with CPAP. However, most will require more aggressive therapy, employing intermittent positive-pressure ventilation (IPPV) with a high fractional inspired oxygen concentration.

High ventilatory pressures may be required to obtain adequate tidal volume. Progress should be monitored by serial arterial blood gas determinations and continuous pulse oximetry.

Hypoxaemia persists in the near-drowning victim due to the pathophysiological changes described and its resultant gross ventilation–perfusion mismatch. These changes do not return to normal for several days in most cases, and much longer in the severely affected.

The institution of continuous positive end-expiratory pressure (PEEP) with either IPPV or spontaneous ventilation (i.e. CPAP), will decrease the pulmonary shunting and ventilation–perfusion inequality and increase the functional residual capacity, thus resulting in a higher Pa_{O_2}. Nebulized bronchodilator aerosols may be used to control bronchospasm. Fiberoptic bronchoscopy can be used to remove suspected particulate matter. Endotracheal suction should not be used frequently to aspirate fluid because it may exacerbate pulmonary oedema and hypoxaemia.

Early on in the resuscitation sequence large-bore **intravenous access** should be established, and warmed crystalloid fluids commenced. Large volumes may be required because of tissue losses, immersion diuresis and dehydration. Simultaneously, haematology and biochemistry laboratory investigations can be performed for baseline assessment, including cardiac enzymes. Intraarterial pressure monitoring is useful, allowing frequent arterial blood gases to guide ventilation and acid–base management.

If **cardiac arrest** is diagnosed, the rhythm should be rapidly determined and defibrillation and/or intravenous adrenaline (epinephrine) administered according to Advanced Life Support (ALS) protocols (Fig. 23.2). Other arrhythmias should also be appropriately treated if they have not responded to correction of hypoxaemia and restoration of adequate tissue perfusion.

Because, in this situation, acidosis has usually developed before cardiac arrest, large doses of bicarbonate (1–2 mmol/kg) may be required. The use of bicarbonate remains controversial,[5] and some clinicians may prefer to hyperventilate a patient in order to create a respiratory compensation for the metabolic acidosis. Although acidosis depresses myocardial function, the administration of buffers may also decrease myocardial contractility.

Some victims have been noted to be markedly hypoglycaemic, and an association with alcohol intoxication, physical exhaustion and hypothermia has been noted. Blood glucose should be rapidly determined along with blood gases on arrival at hospital, and intravenous glucose therapy instituted if appropriate. Untreated hypoglycaemia may aggravate hypoxic brain lesion, but intravenous glucose is otherwise contraindicated as it may exacerbate cerebral oedema. Hyperglycaemia resulting from massive catecholamine release may require insulin infusion.

Hypothermia (see Chapter 28) may complicate the drowning and lead to difficulties in diagnosing cardiac arrest. When in doubt, CPR should be continued at least until the patient is warm. The emergency room management of hypothermia depends on the severity, and low-reading thermometers are required as severe hypothermia may otherwise be overlooked. Warmed intravenous fluids and inspired gases, insulation, heating blankets and radiant heat may be sufficient but, in severe cases, gastric lavage, peritoneal lavage or even cardiopulmonary bypass have been employed. Resuscitation should continue at least until core temperature is normal. Due care should be taken to avoid hyperthermia.

Hypovolaemia may be due to the combined effects of immersion diuresis, and pulmonary and tissue oedema. Circulatory support may be required

Figure 23.2 *Advanced life support. (Reproduced with permission from the Australian Resuscitation Council.)*

in order to provide adequate perfusion of vital tissues. The maintenance of effective cardiac output may require the correction of this hypovolaemia, which may be unmasked by the instigation of body rewarming. Positive-pressure ventilation reduces venous return to the heart, thus lowering cardiac output, but this can usually be overcome by volume restoration or even augmentation. If the patient has not rapidly assumed an adequate cardiac output and tissue perfusion as indicated by blood pressure, pulse oximetry, blood gases and urine output, then careful fluid administration and/or inotropic support will be required.

Although **central venous pressure** monitoring is a useful adjunct, it may not provide a true indication of hypovolaemia or of failure in what may be primary non-cardiogenic pulmonary oedema. Therefore, many clinicians advocate the use of pulmonary artery flotation (Swan–Ganz) catheters to provide information on the left heart filling pressure (pulmonary artery wedge pressure; PAWP), cardiac output and mixed venous oxygen content. Transoesophageal echocardiography may also give useful information about left ventricular function, and this may be essential to guide fluid administration and inotropic support. Colloids may be more useful than crystalloids in the secondary phase of fluid replacement, but this remains controversial.

Once adequate **cardiac output** is established, fluid restriction should be initiated and blood volume

maintained with colloids. Concentrated albumin has been advocated. Diuretics (e.g. frusemide, 1 mg/kg) have also been employed where overhydration is suspected. A urinary catheter with hourly output measurements is essential for indication of renal perfusion and function, and is also a good indication of adequate volume.

Electrolyte disturbances are usually not a significant problem in the initial phases, but any abnormality should be corrected.

Antibiotics, given prophylactically, are of no benefit and should only be employed where clearly clinically indicated, guided by sputum and blood cultures. Routine use may encourage colonization by resistant organisms.

Intensive care

The general principles of intensive therapy are followed, but with special emphasis on respiratory function, as near-drowning is a common cause of the adult respiratory distress syndrome (ARDS), and is managed in the same way. The following clinical baseline measurements, established on arrival in the emergency room, should be regularly monitored:

- Routine clinical observations such as pulse, blood pressure, temperature, respiratory rate, minute volume, inspiratory and PEEP pressures, electrocardiograph, pulse oximetry, urine output.
- Where indicated, invasive monitoring such as central venous pressure, PAWP and cardiac output by a pulmonary artery catheter.
- Blood tests such as estimation of arterial blood gas and acid–base status, haemoglobin, packed cell volume, white cell count, serum and urinary electrolytes, serum and urinary haemoglobin and myoglobin levels, serum creatinine, urea, glucose, protein and coagulation status.
- Regular chest X-ray examination to detect atelectasis, infection, pneumothorax, pulmonary oedema, pleural effusions, etc.
- Serial estimation of pulmonary mechanics, by measurement of airway pressures and compliance are also useful in monitoring progress. In the less severely ill patient, simple spirometry is a useful guide to recovery.

The optimal method of ventilation is that which produces an adequate oxygen tension at the lowest fractional inspired oxygen concentration (preferably Fio_2 of 0.6 or less to avoid pulmonary oxygen toxicity; see Chapter 17), and with the least haemodynamic disturbance. CPAP can be dramatic in improving oxygenation by reducing intrapulmonary shunting. Pressures of 5–10 cm H_2O are usual, but much greater pressures may be required. Patients receiving positive-pressure ventilation tend to retain salt and water, so fluid intake should be reduced to about 1500 ml per day, with low sodium content. Fluid overload may have a disastrous effect on pulmonary function. PAWPs are kept as low as possible.

Various modes of ventilation have been employed, including spontaneous respiration with CPAP, IPPV with and without PEEP, synchronous intermittent mandatory ventilation, pressure-limited cycling, pressure support and high-frequency ventilation. These modes are employed to enable the patient to tolerate the ventilator better, to allow less use of sedatives and relaxants, and to allow weaning from ventilatory support.

Femorofemoral or full cardiopulmonary bypass has been employed both for rewarming hypothermic patients and for establishing adequate oxygenation in severe cases.[11] Severe ARDS has been successfully managed with continuous membrane oxygenation.

The use of exogenous surfactant administered to patients with unresponsive respiratory failure has also been described.

Because of improvements in cardiorespiratory support, preservation of the central nervous system is now the major therapeutic challenge.[12] The application of various brain preservation techniques has not altered cerebral salvage rates.[13] These regimes included deliberate hypothermia, extreme fluid restriction, hyperventilation, barbiturate-induced coma and corticosteroids.[14] In particular, corticosteroids have not been shown to reduce either cerebral oedema or intracranial pressure (ICP).

Central nervous system function is assessed clinically and by electroencephalography. Although its value is debatable, ICP monitoring has been advocated in severe cases, but with prompt therapy for any sudden elevation that most likely indicates irreversible brain damage.

Serial creatinine estimations often reveal mild **renal impairment** in patients requiring intensive care. Severe acute renal failure[15] requiring dialysis is less common, but may develop in those presenting with severe metabolic acidosis and elevated initial serum creatinine. Occasional cases of rhabdomyolysis have also been reported.[16]

Hyperpyrexia commonly follows near-drowning, and its effects on oxygen consumption may be deleterious – especially to the brain. External cooling and

Figure 23.3 *Near-drowning algorithm. Reproduced with permission from* Harrison's Principles of Internal Medicine. *14th edition, 1998. McGraw-Hill.)*

antipyretic drugs to prevent shivering may be indicated.

Continuing hyperexcitability and rigidity may require the use of sedative and relaxant drugs.

PROGNOSIS

It is difficult to suggest the prognosis of individual cases because reported data, much of it from paediatric cases, arises from widely different situations. These range from childhood bath and pool incidents in freshwater to boating, swimming and diving activities in the open sea. Nevertheless, a number of reasonably consistent observations emerge.

Factors that negatively affect outcome include submersion time, time to initiation of effective CPR, severe metabolic acidosis, a Glasgow Coma Scale (GCS) score of < 5, cardiac arrest, and the presence of fixed, dilated pupils. Complete recoveries have been reported despite the presence of one or more of these adverse predictors.

Poor prognostic factors

- *Prolonged submersion*
- *Prolonged time to effective CPR*
- *Cardiac arrest*
- *Absence of spontaneous respiration*
- *Prolonged coma*

Several pooled series indicate that 90 per cent of patients who arrive in the hospital at least arousable, with spontaneous respiration and purposeful response to pain, will survive neurologically intact. In contrast, of those who arrive comatose, 34 per cent died and 20 per cent of the survivors had neurological damage.[17]

Unresponsive coma, decorticate and decerebrate rigidity, areflexia and fixed dilated pupils are not in themselves diagnostic signs of death, although they are, of course, signs of a poor prognosis. Patients who arrive in hospital in asystole usually have a poor prognosis, one series showing 93 per cent mortality after cardiac arrest. Current treatment regimes do not alter the outcome.

The rapid development of severe hypothermia, either before or during the final submersion is probably protective and helps to explain some spectacular recoveries after prolonged periods of submersion.[18,19] The role of the diving response (see Chapter 3) remains controversial.[20]

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24

Drowning syndromes: saltwater aspiration syndrome

CARL EDMONDS

Divers who aspirate small quantities of seawater may develop a respiratory disorder with generalized symptoms mimicking those of an acute viral infection. Severe cases merge into near-drowning.

The symptoms develop soon after the dive, usually persist for some hours, and are aggravated by activity and cold exposure.

Superficially there are similarities between the saltwater aspiration syndrome and other diving disorders, but the characteristics and natural history differentiates pulmonary barotrauma, decompression sickness, Key West scuba divers' disease, hypothermia, infections and asthma.

Treatment includes rest, warmth and oxygen inhalation.

HISTORY

During the 1960s, a common diving illness in the Royal Australian Navy was the saltwater aspiration syndrome (SWAS).[1,2] Its importance lay in the understanding of near-drowning cases, and because of its confusion with other diving or infectious diseases.

This condition, which is due to the aspiration of small amounts of saltwater, may occur because of inexperience, during buddy breathing, or from a faulty regulator. In the 1960s, navy regulators did not have purge valves, and novices were trained in buddy breathing during their first dive, in the open ocean. This led to a frequent aspiration of seawater. In other cases, the aspiration occurred on the surface, after the diver had removed his regulator.

Experienced navy divers often developed SWAS following a fast towed search, while abalone divers did so as a result of using low-pressure surface supply equipment with 'leaky regulators'. Their description 'saltwater fever' indicated that they were well aware of the cause, before SWAS was described by the

clinicians. Other seafarers who present with a similar disorder, though possibly less frequently, are snorkellers, surfers and helicopter rescuees.

A prospective survey[1] was carried out on 30 consecutive cases who presented for treatment. In none of these dives was the depth/duration exposure sufficient to implicate decompression sickness. Nonetheless, the symptomatology was documented and investigations performed. Subsequently, 'volunteers' were encouraged to aspirate seawater through doctored demand valves. The clinical and laboratory manifestations that they developed were consistent with those in the clinical series of SWAS, and thus the aetiology was verified.

CLINICAL FEATURES

The following observations were made on clinical cases.

Immediate symptoms

On specific interrogation, a history of aspiration was given in 90 per cent of cases. Often, the novice diver did not realize the significance of aspiration as being the causal event of the syndrome. Most noted an immediate post-dive cough, with or without sputum, but this was usually suppressed during the dive. Only in the more serious cases was the sputum blood-stained, frothy and copious (as seen routinely in near-drowning cases).

Subsequent symptoms

Subsequent symptoms identified among clinical cases may be classified as follows:

Symptom	Per cent of cases
Rigors, tremors or shivering	87
Anorexia, nausea or vomiting	80
Hot or cold sensations	77
Dyspnoea	73
Cough	67
Sputum	67
Headaches	67
Malaise	53
Generalized aches	33

The signs and symptoms often revert to normal within 6 hours, and rarely persist beyond 24 hours, unless the case is of considerable severity.

Respiratory symptoms

There was often a delay of 1–2 hours before dyspnoea, cough, sputum and retrosternal discomfort on inspiration were noted. In the mild cases, respiratory symptoms persisted for only an hour or so, while in the more severe cases they commenced immediately following aspiration and continued for days. The respiratory rate roughly paralleled the degree of dyspnoea. Physical activity and respiratory stimulants appeared to aggravate the dyspnoea and tachypnoea, as did movement.

Examination of the chest revealed crepitations or occasional rhonchi, either generalized or local, in about half the cases. Rarely, they were high-pitched and similar to that of obstructive airways disease.

Administration of 100 per cent oxygen was effective in relieving respiratory symptoms and removing any cyanosis.

X-rays of the chest revealed areas of patchy consolidation, or a definite increase in respiratory markings, in about half the cases. These usually cleared within 24 hours, but remained longer in severely affected cases. X-rays taken after the incident and repeated within a few hours sometimes showed a variation of the site of the radiological abnormality.

Expiratory spirometry performed repeatedly over the first 6 hours, showed an average drop of 0.7 litres from the baseline in both fixed expiratory volume (1 second) ($FEV_{1.0}$) and vital capacity (VC) measurements. Even those patients who had few or no respiratory symptoms had a reduction in lung volume. Arterial blood gases, when performed, revealed oxygen tensions of 40–75 mmHg with low or normal carbon dioxide tensions, indicative of shunting (perfusion) defects.

Generalized symptoms

The patient usually complained of being feverish, and malaise was the next most prominent feature. Headaches and generalized aches through the limbs, abdomen, back and chest were important in some

cases, but usually not dominant. Anorexia was transitory.

The feverish symptoms were interesting – and are also seen in near-drowning cases. Shivering, similar in some cases to a rigor, and in some cases to generalized fasciculations, was more common during the colder months. It was precipitated or aggravated by exposure to cold, exercise or breathing 10 per cent oxygen (a research procedure, not recommended clinically), and relieved by administration of 100 per cent oxygen. Shivering occurred especially in those exposed to cold because of duration and depth of dive, inadequate thermal clothing, and environmental conditions both during and after the dive. The association of shivering with hypoxia and cold had been described previously.[3] The shivering occurs concurrently with the pyrexia, which also takes an hour or two to develop. Pyrexia was verified in half the cases, up to 40°C [mean ±SD of 38.1 ± 0.6°C], and the pulse rate was elevated [mean ±SD of 102 ± 21 per minute], over the first 6 hours. Some patients obtained relief from these symptoms by either hot water baths or showers, or lying still in a warm bed.

In some patients there was an impairment of consciousness, including a transitory mild confusion or syncope with loss of consciousness on standing. Clinically, these are approaching the near-drowning cases described (see Chapter 22) and were treated accordingly.

Investigations

Haemoglobin, haematocrit, erythrocyte sedimentation rate (ESR) and electrolytes remained normal. The white cell count was usually normal, although a mild leucocytosis (not in excess of 20 000 per mm^3) was observed in a few cases, with a moderate polymorphonuclear leucocytosis and a shift to the left. Lactic dehydrogenase estimations revealed a mild rise in some cases. X-ray and lung volume changes are described above.

Examination of the diving equipment may reveal the cause of the aspiration. Inspection of the second-stage regulator, breathing against the regulator with the air supply restricted, and having another diver use the equipment under similar conditions, may each identify the problem.

DISCUSSION

A detailed investigation into the causes of recreational scuba diving deaths[4,5] revealed that saltwater aspiration was part of the sequence leading to death in 37 per cent of the cases – often a consequence of equipment problems or diving technique. In these cases, 'leaking regulators' were often observed and commented on by the victim beforehand, or demonstrated during the subsequent diving investigation.

The degree of aspiration increases with the volume of air being required (e.g. with exertion, swimming against currents, panic, etc.) and/or with a diminished line pressure to the second stage.

Saltwater aspiration often formed a vicious circle with panic and exhaustion. Hypoxia from saltwater aspiration aggravated the problems of fatigue and exhaustion, and was a precursor to loss of consciousness (with or without dyspnoea) in both near-drowning and drowning cases.

There is no distinct division in the initial presentations between SWAS, near-drowning and drowning cases. Aspiration syndromes merge with near-drownings – often the intensity of the symptoms and the degree of consciousness depending on environmental circumstances, the activity of the victim and the administration of oxygen.

DIFFERENTIAL DIAGNOSIS

In the differential diagnosis of SWAS, the possibility of other occupational diseases of divers must be considered:

- Acute infection: the aspiration syndrome mimics an acute respiratory infection and develops soon after a dive. It is often claimed that a mild upper respiratory infection is likely to be aggravated by diving, but this is countered by the number of divers who continue to dive despite such infections. Differentiation between SWAS and an acute infection can be made from the history of aspiration, serial chest X-rays, spirometry, and a knowledge of the natural history of the diseases. In the first few hours of this syndrome, the possibility of both influenza and early pneumonia are often

considered, but these may be dismissed as SWAS clears within hours.

- **Decompression sickness** (DCS) with cardiorespiratory or musculoskeletal manifestations: if there is any question regarding the possibility of cardiorespiratory symptoms of DCS ('chokes'), then recompression therapy is mandatory. DCS should be considered with subjects who conduct deep or repetitive diving. The specific joint pains and abnormal posturing characteristic of 'bends' is quite unlike the vague generalized muscular aches, involving the limbs and lumbar region bilaterally, seen with SWAS. The immediate beneficial response to the inhalation of 100 per cent oxygen in the SWAS is of diagnostic value. Chest X-ray, lung function and blood gas analyses may be used to confirm the diagnosis. DCS responds rapidly to recompression therapy (as does SWAS to hyperbaric oxygenation). Otherwise, except for the occurrence of a latent period, the clinical history of the two disorders is quite dissimilar.
- **Pulmonary barotrauma**: serious cases of pulmonary barotrauma result in pneumothorax, air emboli and surgical emphysema occurring suddenly after a dive. In minor cases of pulmonary barotrauma, confusion with the SWAS may arise. In these, the diagnosis and treatment of the former must take precedence, until such time as the natural history, chest X-ray, spirometry and blood gas analysis demonstrates otherwise. Hyperbaric oxygen is also effective (but unnecessary) treatment for SWAS.
- **Hypothermia**: the effects of cold and immersion are usually maximal at, or very soon after, the time of rescue. The clinical features are only likely to be confused with SWAS when both conditions exist. The body temperature is higher than normal with SWAS, lower with hypothermia.
- **Key West scuba divers' disease**[6]: this and other infective disorders due to contaminated equipment, may cause some confusion. Fortunately, these illnesses usually take longer to develop (24–48 hours), and to respond to therapy. There is thus little clinical similarity in the sequence and duration of the clinical manifestations.
- **Asthma**: some subjects have hyperreactive airways to hypertonic saline (seawater), analogous to an asthma provocation test. Such patients have the clinical signs of asthma (expiratory rhonchi, especially with hyperventilation, typical expiratory spirometry findings and positive asthma provocation tests). They respond to salbutamol or other beta-receptor agonists.

TREATMENT

Most of the clinical manifestations of the SWAS respond rapidly to rest and the administration of oxygen. Warming the patient is of symptomatic benefit. In general, no other treatment is required.

There is a possibility that some of the clinical manifestations may not be due entirely to the aspiration of water, but to the body's (and specifically the respiratory tract's) response to aspirated organisms, foreign bodies or irritants carried to the lungs with the seawater aspiration.

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25

Drowning syndromes: why divers drown

CARL EDMONDS

Drowning is the cause of death in 74–82 per cent of recreational diving deaths, compared to the more high profile diseases of decompression sickness (<1 per cent) and contaminated air supply (<1 per cent).

Comparisons of divers who drown with those who survive from near-drowning reveal the importance of:

- *personal factors including both medical and physical fitness;*
- *diving experience;*
- *faulty equipment and misuse of equipment;*
- *hazardous environments; and*
- *neutral buoyancy being maintained during the dive, and not being dependent upon the buoyancy compensator.*

Other factors which increased the likelihood of diving problems having an unsuccessful outcome included:

- *an inadequate air supply;*
- *the failure to employ correct buddy diving practices;*
- *inadequate buddy communication;*
- *failure of positive buoyancy after a diving incident; and*
- *inappropriate or delayed rescue and resuscitation.*

BACKGROUND

In a prelude to the 1997 UHMS Workshop on Near Drowning, the Chairman made the following statement[1] in the pre-workshop correspondence: "As you know, the drowning literature ignores diving, whilst the diving literature ignores drowning". It is paradoxical that drowning, which causes more than eighty times the number of deaths in recreational divers than either decompression sickness (DCS) or contaminated air, does not rate more than a paragraph or two in some diving medical texts.

In reviewing the literature on drowning, before the 1997 Workshop, the only papers that could be found which specifically related any of the drowning syndromes to scuba diving, was one on the saltwater aspiration syndrome,[2] and one with an anecdotal review followed by a case report.[3] Nevertheless, of the major seminal reviews presented on this subject, many[4–7] have been by diving physicians.

A normally functioning human, with adequate equipment in a congenial ocean environment, is protected from drowning as he carries his own personal life support with him – his scuba system. Drowning would only occur when there is:

- diver fault (pathology, psychology or technique);
- failure of the equipment to supply air; or
- hazardous environmental influences.

Nevertheless, the commonest ultimate cause of death in recreational scuba divers is drowning. Factual information that clarifies the causes and management, is of value in preventing further fatal outcomes.

Previous surveys[8–12] illustrated the importance of drowning as the ultimate cause in 74–82 per cent of recreational scuba diving fatalities. Of note, in the more detailed surveys,[10–12] was the high frequency of multiple contributing factors to each death. Drowning tended to obscure those preceding factors. The drowning sequelae and drowning pathology were due to the environment in which the accident occurred, and not the initiating or primary cause of the accident. For example, any loss of consciousness or capability when engaging in terrestrial activities is unlikely to cause death, but it would do so more frequently if the victims were diving under water.

SURVEY RESULTS

The aspiration of seawater causing clinical features in scuba divers, while retaining consciousness, has been dealt with previously (see Chapter 24). Sometimes this progresses into the other manifestations of near-drowning and drowning, and these were compared in one survey of fatalities (drownings) and survivors (near-drownings)[13] in recreational diving. The observations were as follows.

Personal contributions

Population

Of the 100 fatalities, 89 per cent were male and 11 per cent were female. Of the 48 survivors, 52 per cent were male, 48 per cent female. Compared to the diving population at the time (30 per cent females, 70 per cent males), males were over-represented in the scuba drowning cases, as they are in almost all other forms of drowning.[6] The surprise was that females appeared to be over represented in the 'survivor' series.

Whether females had more accidents, or whether they only reported them more frequently, could not be deduced. However, it does appear as if the female accidents result in fewer deaths.

Training

Among the fatalities, 38 per cent had no known formal qualification. This group was approximately equally divided between:

- those in whom the documentation was inadequate;
- those without training, but who were experimenting with scuba under their own or their friends' cognisance; and
- those who were engaged in introductory dives, brief resort courses or 'dive experiences' with a recognized commercial organisation.

Of the survivors, 81 per cent had completed basic training and only 4 per cent had no training. A surprising number in both groups were under formal training at the time – 8 per cent of the fatalities and 15 per cent of the survivors.

Experience

This did not entirely correlate with training. In both the fatality and survivor series, the divers were equally represented among inexperienced (less than 5 dives), novice divers (usually 5–20 dives) and experienced divers. Of the fatalities, over one-half were experiencing diving situations to which they had not been previously exposed, whereas one-third had previous experience of the conditions in which they died. The others were unable to be assessed.

The buddy or dive leader appeared to be considerably more experienced than the diver in most of

these cases, possibly explaining why the diver died and the buddy lived.

Victim's behaviour

Analysis of these data revealed the following:

Victim's behaviour	Fatalities (observed) (%)	Survivors (reported) (%)
Panic	21	27
Rapid/abnormal movements	16	31
Nothing unusual	63	42
Loss of consciousness	33	25
Air requested	21	10

In 100 diving fatalities that were observed during the incident, over one-third had either a panic response or rapid/abnormal movements. Over one-half of the survivors reported these sensations. The increased incidence in the surviving group could be attributed to this being a reported sensation, whereas the fatality figure represented the observed behaviour. Over half of the fatalities showed no change in their behaviour, with loss of consciousness being the first objective warning in one-third. It was the first manifestation noted in one-fourth of the survivors.

Of interest was the absence of panic in many of the cases, even though it is a frequent cause of other diving deaths.[8–14] Drowning scuba divers frequently drown quietly – possibly because of the effects of previous aspiration (hypoxia), depth (narcosis) or training ('don't panic').

A request for a supplementary air supply was made by twice as many fatalities (21 per cent) as survivors. This may bring into question the value of relying on a buddy responding to such a request. Alternatively, with the survivors, more frequently buddies offered the emergency air supply – a preferred sequence. Occasionally, there was the apocryphal underwater tussle for a single regulator. When the low-on-air diver went for an air supply, he more frequently sought the companion's primary regulator, than the octopus.

Medical conditions

Analysis of these data revealed the following:

Medical disorder	Fatalities (%)*	Survivors (%)
Asthma	10	19
Cardiovascular	6	2
Drug intake	10	8
Very unfit	5	4
Panic	7	8

* History often not sought.

This is a contentious area, not only regarding the incidence of medical disorders but also their significance. Authors differ in their assessments of this, and none is free of prejudice.

Medical history data from fatality records is inevitably an underestimate. In one analysis,[10] when an attempt was made to acquire the medical history, in less than half of the cases could this be obtained.

In this survey no attempt was made to draw statistical conclusions regarding the correlation between past illnesses and drowning; however, there was no doubt as to the contribution in the survivor group. Some of both groups should not have been classed as medically fit for diving (see Chapters 53–59).

Environmental factors

Water conditions

The adverse influences of water conditions were expected. Probably the only surprise was the frequency with which drowning occurs in calm waters – in over half the cases. Strong tidal currents were slightly more frequent among the fatality group.

Fresh/seawater

Most of the accidents occurred in the ocean, without obvious differences between the fatality (93 per cent) and survivor groups (98 per cent). The extra difficulty of performing rescues in cave diving (2 per cent) was expected.

Depth of incident

The depth of the aspiration/drowning incident was not necessarily the depth of the original problem. Thus, a diver who used most of the air supply and

then panicked and ascended, might not show any evidence of aspiration until he reached the surface.

As in previous surveys,[8,10] many problems developed on the surface. Approximately half the fatalities occurred while on the surface or on the way to the surface. Frequently, the diver no longer had adequate air to remain under water. Another 20 per cent occurred in the top 9 metres, and the rest were distributed over the remaining depths. This implies that just reaching the surface is not enough – successful rescue then requires the victim to remain there.

The survivors more accurately reported the depth at which the incident developed, as opposed to the depth at which the incident was noted by others. Nevertheless, almost two-thirds of the incidents occurred in the top 10 metres.

Among the fatality and the survivor groups, the dive was the deepest of their diving career in 26 per cent and 33 per cent, respectively. In almost half of the 'inexperienced' and 'novice' divers the depth was beyond that which had previously been undertaken, suggesting that these groups are especially susceptible to the various problems associated with depth (panic, air consumption, visibility, narcosis and logistic difficulty with rescue). This demonstrates that it is not so much the environment that is the problem, but the diver's experience of that environment. The danger of 'diving deeper' without extra prudence and supervision is apparent. Any dive deeper than that previously experienced should be classified and treated as a 'deep dive', irrespective of the actual depth.

Visibility

Visibility was usually acceptable, but seemed to be more frequently adverse in the fatalities (38 per cent), compared to the survivors (18 per cent).

Conclusions

The cases, in general, demonstrated the adverse effects of various environments, especially with tidal currents, white (rough) water, poor visibility and deeper diving than previously experienced. There was not a great deal of difference between the two groups, except in the higher incidence of strong tidal currents, night diving and cave diving in the fatalities. The figures, however, were few in number. Such adverse environments may affect the victim directly, or may negatively influence rescue and resuscitation.

Equipment

In most fatalities the equipment showed no structural abnormality, and only in 20 per cent were there significant or serious **faults** contributing to the fatality. This corresponded with the reported incidence by the survivors (18 per cent). Equipment faults were most frequently found with buoyancy compensators and regulators (both first and second stages).

The incidence of **equipment misuse** was more frequent but more difficult to ascertain in the fatality series – and is dependent on one's definition (fatalities 43 per cent, survivors 38 per cent). Misuse of equipment included the use of excessive weights (fatalities 25 per cent, survivors 27 per cent), or the failure to carry equipment that could have been instrumental in survival (e.g. buoyancy compensator, contents gauge, snorkel, etc.) (fatalities 12 per cent, survivors 8 per cent). Difficulties in using buoyancy compensators were also frequent.

Diving technique

Various diving techniques either contributed to the drowning incidents, or influenced rescue and survival. They include a compromised air supply, buoyancy factors, buddy rescue and resuscitation attempts.

Air supply

In 60 per cent of the fatalities either an out-of-air (OOA) or low-on-air (LOA) situation had developed. There was insufficient air in the tank for either continuing the planned dive or returning to safety, underwater. In the survivors there was a lower incidence (35 per cent) of compromised air supply, but it was still very high. The survivors were more likely to have air in their tanks to cope with the emergency.

The failure to use the available contents gauge, in both groups, was a source of concern, though this could sometimes be attributed to the conditions placing other stress on the diver (depth, anxiety, tidal

current, deepest dive ever, etc.) In many more cases there was a voluntary decision to dive until the tank was near reserve or 'ran out'.

One surprising feature was the failure in both groups (fatalities 8 per cent, survivors 13 per cent) to reopen the valve of the scuba tank, after initially testing the tank pressure, before diving. Thus even though there was plenty of air in the tank, it was unavailable other than sometimes to allow a rapid descent to a few metres. Only then was the diver aware that further air was not available. In none of these cases was there a buddy check of equipment – breathing near the water surface and checking the equipment before descent.

In a smaller number of cases there was a failure to ensure the cylinder tap was adequately turned on. With reduction of tank pressure there was a restriction of air supply – sometimes obvious only at depth.

Buoyancy factors

Buoyancy was frequently a vital factor in reaching the surface, and in remaining there as an unconscious diver, and being found, rescued and resuscitated in time. The three major influences on this are the buoyancy compensators (BCs), weights and the companion (buddy) diver practice.

- Buoyancy compensators: in the survivor group the BC was inflated by the victim or rescuer (35 per cent and 25 per cent respectively) in twice as many cases as in the fatality group (15 per cent and 16 per cent). This figure is even more relevant when the delay in producing buoyancy in the fatality group is considered (see below).
- Weights: these were as follows:

Weights	Fatalities (Observed) (%)	Survivors (Reported) (%)
Not worn	1	6
Not ditched	66	48
Entangled	3	2
Victim ditched	10	19
Rescuer ditched	20	25

Although in 30 per cent of the fatality cases the weights were ditched, in practice this was not as valuable as it sounds. In most of the instances in which the rescuer ditched the weights, the victim was probably no longer salvageable, because of the delay (see below).

The survivor group not only ditched the weights more frequently, but often this was done by the victim. When it was done by the rescuer, this usually occurred early in the incident.

- Buoyancy action by survivors: the fatality and survivor groups differed in that the latter more often performed an action (ditching weights, inflating buoyancy compensator) which resulted in them achieving positive buoyancy during and following the incident.

An interesting observation was made that when the victim and buddy were both in difficulty, usually based on a LOA/OOA situation, in the ensuing scenario (irrespective of whose problem developed first) the overweight diver tended to be the one that died, and the buoyant diver the one that survived. Among 14 instances of this situation, the ratio was 6:1 in favour of the overweighted.

These data all provide support to the current Instructor Agencies emphasis on buoyancy training, although one could argue for its inclusion in introductory courses more than an advanced courses.

Companion diver practice, rescue and resuscitation

In most cases of significant aspiration of water, rescue depends on rapid action being undertaken by either the victim or the companion (buddy) diver. Once a diver gets into difficulty, and is unable to carry out safety actions by himself, he is heavily reliant upon his buddy or dive leader. The fatality and survivor populations were very different in this respect.

Fatalities

In the fatality group, less than half of the victims had an experienced buddy available to assist them. In 21 per cent of the fatalities, the dive was a solo one.

In 38 per cent of fatalities the diver had separated from his buddy, and in 12 per cent from the group, before the serious incident. Thus, a voluntary separation happened in 50 per cent of the cases before

the fatality; this was initiated in most because the victim could not continue (usually due to a LOA situation). The victim then attempted to return alone, essentially making it a solo dive.

The diver was separated from his buddy or the group during the actual incident, and often by the incident, in 21 per cent of cases. However, in almost half of these cases the separation was produced because the diver was following his buddy or the group. The others occurred during the 'rescue'.

Thus, separation made early rescue and resuscitation improbable. In 9 per cent of fatalities the victim was swimming behind their companion/s, and thus the victim was not visible to the 'buddy' at the time of the incident.

In summary, 80 per cent of the victims did not have a genuine buddy, by virtue of their elected diving practice. In fewer than one in ten deaths was there a continued contact with the buddy or group during and following the incident.

The victims seemed flagrantly to disregard the 'buddy' system – as did their companions, the organization which conducted the dive, or the 'dive leader'. Group diving conferred little value because the 'leader' often had insufficient contact with individual divers to be classified as a buddy, and the responsibility of others was not clear – especially towards the last of the 'followers'.

In only 20 per cent was the diver reached within 5 minutes of the probable incident time, and thereby had a real chance of successful resuscitation. In another 12 per cent the diver was recovered within 6–15 minutes, when, theoretically, there was a slight chance of recovery – had the rescue facilities been ideal and had fortune smiled brightly.

Resuscitation was not a feasible option for most of the eventual fatalities, who were obviously dead or showed no response to the rescuers attempts, in nine out of ten cases. This is explained by the excessive delay in the rescue in most cases.

Survivors

In the surviving group most were rescued by their companion, and some form of artificial respiration or CPR was required in 29 per cent of the cases. Oxygen was available and used, usually in a free-flow system, in 52 per cent of cases.

No specific data were available on the buddy divers assisting the survivors, other than the subjective assessment as to whether the survivor believed the buddy to be of much value. The buddy:

- was immediately available to the survivor in 71 per cent;
- was considered to be of assistance in 58 per cent;
- supplied an independent air source in 15 per cent;
- inflated the BC in 25 per cent;
- ditched the weight belt in 25 per cent; and
- attempted buddy breathing in 4 per cent.

In 52 per cent of cases the diver surfaced under control of the buddy.

The attitude toward buddy diving practice with the survival group appeared to be very different to those in the fatality group. The frequency of oxygen use probably represented a more sophisticated and organized diving activity, which may also be related to more conscientious buddy behaviour. The axiom is that to successfully rescue an incapacitated diver one must know where he is, and reach him quickly. This implies some form of buddy responsibility. Once reached, the buddy divers seemed to be of considerable value – implying good training or initiative in this aspect of diver safety.

In recent years there has been a promotion of solo diving and reliance upon oneself, as compared to buddy diving practices. The above data indicates that the traditional buddy concept, correctly practiced, is of more value.

CONCLUSIONS

There are many lessons for recreational divers to learn from the data now available, as well as from the diving medical experience and the regulatory requirements of commercial diving, to reduce the incidence of drowning with scuba. They can be summarized as follows.

- **Diver fitness:** ensure both medical and physical fitness, so that there is no increased likelihood of physical impairment or loss of consciousness, or difficulty in handling unexpected environmental stresses.

- **Experience**: ensure adequate experience of the likely dive conditions (dive under the supervision of a more experienced diver, when extending your dive profile).
- **Equipment**: failure to possess appropriate equipment is a danger, but not as much as equipment failure and misuse. The latter includes the practice of overweighting the diver, and his over-reliance upon the buoyancy compensator.
- **Environment**: hazardous diving conditions should be avoided, using extreme caution with tidal currents, rough water, poor visibility, enclosed areas and excessive depths.
- **Neutral buoyancy (dive)**: ensure neutral buoyancy while diving; this implies not being overweighted and not being dependent on the buoyancy compensator.
- **Air supply**: an inadequate supply of air for unexpected demands and emergencies may convert a problematical situation into a dangerous one. It also forces the diver to experience surface situations that are worrying and conducive to anxiety, fatigue, unpleasant decision making and saltwater aspiration. Equipment failure is not as common a cause of LOA/OOA as failure to use the contents gauge and/or a decision to breathe the tank down to near-reserve pressure.
- **Buddy diving**: use traditional buddy diving practice – two divers swimming together. Solo diving, for the whole or part of the dive, is much more likely to result in an unsatisfactory outcome in the event of diving problems. It is the divers who are committed to the traditional buddy diving practices who are likely to survive the more serious of the drowning syndromes.
- **Positive buoyancy (post incident)**: positive buoyancy is frequently required if problems develop. Failure to remove the weight belt during a diving incident continues to be the major omission, and must reflect on training standards. In most situations, unbuckling and then ditching (if necessary) the weight belt is the most reliable course of action once a problem becomes evident. Buoyancy compensators cause problems in some emergency situations, and not infrequently will fail to provide the buoyancy required. They are of great value in many cases – but are not to be relied on.
- **Buddy communication**: if feasible, inform the buddy prior to ascent. If correct buddy diving practice is being carried out, the buddy will automatically accompany the injured or vulnerable diver back to shore.
- **Rescue**: employ the rescue, water retrievals, first aid facilities (including oxygen) and medevac systems which were planned before the dive.

Taken together, these factors differentiate a drowning fatality from a successful rescue.

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26

Seasickness (motion sickness)

JOHN PENNEFATHER

INTRODUCTION

Almost everybody is susceptible to motion sickness.[1] In general, the population can be divided roughly into one-third who are highly susceptible, one-third who react only under rough conditions, and one-third who become sick only under extreme conditions. Although anyone with a normally functioning vestibular system is susceptible, people who are totally deaf and have unresponsive vestibular systems are immune.

In diving there are two major situations that predispose to seasickness. The first is on the boat going to the dive site, and the second is while the diver is in the water – particularly if he is attached to the boat, for example, on a shot line during decompression. Most divers are less susceptible to seasickness swimming under water than when they are on the boat. For this reason many divers hurry to enter the water, after exposure to adverse sea conditions en route to the dive site. Problems develop because they are inadequately prepared and equipped due to haste or the debilitating and demoralizing effects of seasickness.

SYMPTOMS[1]

Usually, the first sign of seasickness is pallor, although this occasionally may be preceded by a flushed appearance. It may be followed by yawning, restlessness and a cold sweat, often noticeable on the forehead and upper lip. Malaise, nausea and vomiting may progress to prostration, dehydration, electrolyte and acid–base imbalance. During this progression, there is often a waxing and waning of symptoms, especially prior to the actual development of vomiting.

Tolerance develops to a particular motion, and a subject may become immune under specific conditions. If there is a change in the intensity or nature of the motion the individual is again susceptible. Continuous exposure to constant conditions will usually produce tolerance within two to three days. This can also develop to repeated shorter exposures. There is a central nervous system habituation, to such a degree that after the subjects disembark and the motion is stopped, the subject feels they are rocking at the frequency of the original ship exposure.

There is considerable variation in susceptibility to seasickness. With increasing age, individuals tend to become more resistant, and at least one study suggests that females are more susceptible.[2] This is said to be due to a lack of experience with the situations which produce seasickness. Overindulgence in food and alcohol prior to exposure, and especially the night before, predisposes to motion sickness. Both the number of meals and their energy content correlate with susceptibility to airsickness.[2] The position on board the vessel can also be important, with least stimuli if the victim is amidships, using the horizon as a visual reference. Any attempt to read will aggravate the motion sickness. Psychological factors play a part, especially with the seasickness that develops prior to boarding the vessel. Once one person becomes seasick, there is often a rapid spreading among the others.

AETIOLOGY

Motion sickness is due to a mismatch or conflict of sensory neural information.[3] Normally, the vestibular stimuli are consistent with the visual and proprioceptive stimuli, all informing the brain of the position of the body – even when it is in motion. When the environment starts moving as well, the information becomes conflicting. The motion sickness occurs at the onset and cessation of sensory rearrangements, when input of vision, vestibular and proprioception is at variance with the stored patterns of recent stimuli information.

Seasickness is mainly a central nervous system reaction to vestibular impulses. It has been claimed that some of the drugs work by reducing the vestibular response to stimuli, and that this variation in sensitivity also explains variation in susceptibility. However, the results[3] show considerable overlap between the groups selected for high and low susceptibility.

PREVENTION

Drugs for general use

There is a wide variation in the suggested drugs for preventing seasickness. Goodman and Gillman[4] state that hyoscine (scopolamine) has been found to be the drug of choice, and cite Graybiel *et al.*[5] as their reference, though these authors compared drugs alone and in combinations. The best combination was found to be promethazine hydrochloride 25 mg + 25 mg ephedrine sulphate. Scopolamine was the most effective single drug, but was more effective when combined with ephedrine sulphate or d-amphetamine sulphate than as a single drug. It is probably the most effective drug available over the counter for short exposures (under 4 hours) and severe motion. Because of the frequency of complications drowsiness (80 per cent), blurred vision and 'vertigo', according to Money,[1] scopolamine is not suitable for divers without trial usage. The transdermal form of scopolamine (Transderm Scop™) has supporters, but has been withdrawn from the Australian market. In a trial reported by Pyykko *et al.*,[6] dimenhydrinate was more effective than one Transderm Scop patch and about equal to two, and had the advantage of needing a shorter period to become effective. According to Graybeil *et al.*, dimenhydrinate was more effective when combined with ephedrine sulphate than when used alone. The antihistamines are effective for longer exposures in moderate conditions. Other studies place cyclizine and dimenhydrinate as equal in performance, but suggest that cyclizine reduced gastric symptoms and drowsiness.[7] Repeated doses during long exposures may be necessary. The use of combined preparations, e.g. hyoscine+ dimenhydrinate[8] may be more effective than a single compound. Others have produced results that support a range of products ranging from drugs such as cinnarizine and phenytoin, through ginger pills, to various forms of acupuncture

Drugs for divers

For susceptible divers, both the drugs chosen and the diving depth limits may need modification. For early-morning dives, promethazine 25 mg taken the night before will still have anti-seasickness effects after the sedation has worn off. Cyclizine 25 mg may be used, if necessary, 1 hour before departure. For later dives, meclozine 25 mg is taken 8 hours beforehand. Cyclizine 25 mg may be used, if necessary, 1 hour before departure.

Table 26.1 *Anti-seasickness drugs*

Drugs	Dose (mg)	Duration (hours)	Condition
Hyoscine + dexamphetamine	0.3–0.6 + 5–10	6	Severe
Promethazine + ephedrine	25 + 25	12	Severe
Hyoscine	0.3–0.6	4	Severe
Promethazine	25	12	Severe
Dimenhydrinate	50	6	Moderate
Cyclizine	50	4	Mild
Meclozine	50	6	Mild

In each of these cases the drugs must be tried previously to ensure there are no untoward reactions. There should be no undue sedation immediately before the dive, and the dive should be shallower than 18 metres (60 feet) to avoid significant narcosis dangers.

In diving, the numerous side effects of Transderm™, including reduced respiratory secretions, dry mouth, blurred vision and drowsiness, make it unacceptable.

TREATMENT

For boat passengers and sailors, acclimatization will develop if progressively increasing periods are spent at sea; otherwise it usually takes two to three days to adapt to new conditions. The sources of vestibular and proprioceptive stimulation should be reduced to a minimum, and this usually means either laying down or being as still as possible. Unnecessary head movements should be avoided. In small craft, staying along the centre line of the craft, towards the stern, incurs the least complex motion.

Conflicting visual stimulation is reduced by keeping the eyes closed, or focusing on the horizon. Both mariners and astronauts can also suffer when they have returned to land.[8]

Inhibition of the conflicting stimuli can be achieved by mental activity (e.g. steering, if allowed, or watching distant videos). Reading requires visual convergence, which will increase conflicting visual stimuli and aggravate seasickness.

If a sufferer is seriously ill or if vomiting has commenced, the pylorus will be constricted, and oral drugs may not reach their site of absorption. Consequently, the drugs must be administered parenterally. Promethazine, 12.5–50 mg is more effective than intramuscular hyoscine.[8] If treatment is delayed, then intravenous fluid and electrolyte replacement may be required. Indeed, sea-going medical officers have observed that the fluid may be more important than the drug.

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27

Thermal problems and solutions

JOHN PENNEFATHER

BASIC TEMPERATURE PHYSIOLOGY

For a diver in the water or a pressure chamber the heat loads are often greater than are normally experienced on land. Most readers of this book will not need a more detailed discussion of basic temperature physiology, but any that do are advised to consult one of the basic texts that deal with the subject.[1]

With the exception of those animals that hibernate, mammals require a relatively stable body temperature to function. For a person to be comfortable, their deep body temperature must remain at about $37 \pm 1°C$. Temperatures above this cause sweating, and higher temperatures may lead to heat stroke and death from hyperthermia. Below the comfort range, shivering can proceed to hypothermia and death (hypothermia is discussed in Chapter 28).

People maintain their body temperature by balancing production and loss of heat. Heat is produced by the biochemical processes that convert food to energy and waste products. The other part of the positive side of heat balance is heat gain from the exterior. This occurs in a hot climate or by touching or being exposed to something that is warmer than the person, and this includes food and drink.

Most heat is lost from a warm body by transfer to a cooler environment. The other avenue of losing heat is in warming material that enters the body. Food and cold drinks form a small part of this, but warming and humidifying air before it reaches the lungs acts as a continuous heat drain.

A constant temperature is maintained if the production and loss of heat remain equal, and the body can do this in a variety of ways. In a warm environment the amount of heat produced is set by the activity of the metabolic processes and the exercise undertaken, so the production side of the equation is fixed. In contrast, heat loss to the environment can be adjusted. It is influenced by skin temperature, which is to some extent under the control of the nervous system. If more heat loss is needed, nervous stimuli cause increased blood flow to the skin and this leads to more heat being transferred. If still greater heat loss is needed, the body can sweat; subsequent evaporation of the sweat causes heat to be lost and this results in cooling of the skin.

In a cold environment, if reducing the blood flow to the skin is not enough to conserve heat, then more heat can be produced by muscular activity such as shivering. The other methods of maintaining body temperature are behavioural; putting on or taking off clothes and moving to a warmer or cooler place are examples of this.

DIVER IN SHALLOW WATER

The temperature of the oceans range from −2° to about 30°C, but in most regions the annual range of temperature in open ocean is less than 10°C. This narrow range is caused by the high heat capacity of water, which damps down the seasonal change in temperature. The thermal environment is predictable enough to allow precautions to be taken when diving.

In most circumstances the diver's problem is to maintain body heat. Cooling occurs because water is a good conductor, and has a high specific heat.[2]

For most dives in shallow water, an increase in insulation will reduce heat loss to an acceptable level. Insulation will also be required for survival during prolonged exposure to cold water. The temperature at which extra insulation is required depends on the duration of the dive, the heat production the diver can maintain, and his internal insulation. This is related to the amount of body fat carried and to the previous exposure to cold, which generates a degree of tolerance.

Wet suits are the most common protective clothing in temperate water. They are made from sheets of rubber that has gas bubbles injected into it as it solidifies, while a backing layer of fabric gives the rubber strength. The fabric adds little to the insulation, which mainly comes from the trapped gas bubbles. Some heat is also conserved because the layer of water between the wet suit and skin is trapped there and warms up, so less heat is lost. Because the bubbles in the rubber obey Boyle's law their volume decreases with depth, so the insulation diminishes as depth increases.

Dry suits are made from a fabric/rubber composite sheet, and are generally preferred to wet suits for colder, deeper or longer dives. They derive their name from the intention that the occupant of the suit should be dry under the water-tight barrier of the fabric. Because of this, a warm layer of clothing can be worn and a layer of air is trapped in the suit. As a gas space, the undersuit layer follows Boyle's law, so gas must be added during descent and vented off during ascent to preserve the insulating layer and control buoyancy.

A wet or dry suit will provide adaquate thermal comfort in shallow water, but a dry suit is the preferred option for colder, deeper and longer dives. A common problem with both suits is the loss of dexterity caused by cooling of the hands in cold water.

The insulating efficiency of these suits is demonstrated by a comparison of the effects on subjects immersed in water at 5°C. Without protection, about most would die within a few hours, but in the same water temperature the core temperature of a diver wearing a neoprene foam wet suit would probably fall 1°C in 6 hours.

Heat loss during immersion is much greater from some body surfaces than others. Areas of high heat loss include the skin over active muscles, and also areas of the body with little subcutaneous fat. In cold water much of the heat loss from a diver in a wet suit takes place through the head, so this should be insulated. It is also difficult to keep the hands warm, as the extra insulation increases the surface area from which heat is lost. This is a difficult problem for long dives in cold water, as it leads to a loss of dexterity. This can be a performance-limiting factor unless active warming is provided.

DEEP DIVER

A diver at depth will generally be breathing an oxygen/helium mixture from a supply of dry gas. Because of the higher specific heat of the helium, extra heat is used to warm the gas. This can cool the diver to such an extent that the use of external heat is required to warm the gas. By Norwegian guidelines, the breathing gas for a diver at 400 metres should be warmed to 30 ± 2 °C, and failure to do this can cause dyspnoea (see below).

Various techniques of diver warming have been utilized, including the use of electrical and chemical energy, and even nuclear energy! Hot water supplied from a boiler on the surface is the most common commercial diving system. This is pumped down to the diver and circulates through the space between the diver and his wet suit. A Swedish military system uses the release of heat from the decomposition of concentrated hydrogen peroxide;[3] this is used as a torpedo propellant and so is readily available.

Because of the high energy density of peroxide it is reasonably portable, and this allows the diver to be free of any connections to the surface. The heat generated by the decomposition reaction in the back-mounted reaction vessel is transferred to the diver through a network of tubes through which warmed water is circulated.

Another novel answer to the problem is the use of suits filled with a foam that is loaded with a chemical that changes phase and in doing so gives off heat to the diver. The latent heat of the solidification process acts as a heat source. However, this research has not yet advanced to the point where its final role in keeping divers warm can be accurately predicted.[4]

If the breathing gas is not heated, a deep diver can suffer from **dyspnoea** induced by the cold gas. Flynn[5] describes this as substernal discomfort and chest tightness that may spread to cover the whole substernal area. The more important response for its effect on safety is the production of large amounts of thick mucus that can plug the airways and equipment. With high heat loss, shivering may be uncontrollable and the diver unable to hold their mouthpiece. Rest and breathing warm gas will cure the dyspnoeic condition; warming the inhaled gas will also prevent it.

Dehydration is a hazard of diving with a heated suit. One study showed that the dehydration could be as high as 4–5 per cent of the body weight of the diver – a level that can cause reduced mental and physical performance.[6]

DIVING IN HOT ENVIRONMENTS

Hot water is a less common problem than cold, but cases of divers overheating have occurred. This may happen when diving in water that is artificially heated, as in a power station. It can also occur if a diver needs the protection of a dry suit when diving in warm water because of a risk of disease. For example, police divers occasionally have to dive in sewerage processing plants. To reduce the high risk of infection they often dive in a dry suit and helmet. However, because of the thermal protection of his dry suit, the diver may overheat.

In some circumstances a wet suit to which cold-water cooling can be pumped down is a satisfactory method of keeping cool. If a dry suit is needed for protection, a cooling vest from which heat is removed to thaw ice pouches is an option. It may also be feasable to circulate cold water through the vest to provide a longer period of tolerance.

There is also a risk of **hyperthermia** on the surface while waiting to dive, particularly if the diver is in the sun or he exercises. In this situation a bucket of cold water tipped over the diver, or a quick immersion to cool off, is generally an adequate method of cooling them. The situation where a diver has exercised in an insulating suit has led to deaths from heat stroke in military diving training, but this is an entirely preventable condition if the instructors are aware of the problem.

DIVER IN A RECOMPRESSION CHAMBER (RCC)

Problems with the heat balance of divers in RCCs has caused deaths from hyperthermia and also difficulties from hypothermia.

In a warm climate, overheating may be a problem in any RCC with a carbon dioxide-absorbing system, and a method of cooling and dehumidification is needed. If the carbon dioxide is removed by reaction with soda lime, water is evolved and this is added to the water produced by the diver as sweat and humidifying the inhaled air. The atmosphere becomes saturated with water vapour and the diver cannot use sweating as a method of cooling his body. As a result, the body temperature increases; this exacerbates the problem because as temperature rises above normal, the body produces more heat as its metabolic reactions accelerate.

Because helium is a good conductor of heat, a diver in a helium atmosphere heats or cools more rapidly than in an air-filled space. In an air-filled RCC a person in light clothes is comfortable in a temperature range of about 20°C to 30°C. For a diver in an oxygen/helium atmosphere the thermal comfort range is narrower and warmer than for an air diver. The acceptable temperature increases with depth, because there is more helium in the

atmosphere as the pressure is increased. A diver will be comfortable at about 29°C at shallow depths, and this increases to about 34°C as the pressure is increased. There is an associated narrowing of the temperature range where a diver is comfortable to less than 1°C. With this narrowing of the comfort range there is also an increase in the rate at which cooling and overheating occurs if the temperature goes outside the comfort zone. Hence, a close control of temperature is needed with a helium-rich atmosphere in the RCC. In any RCC, dehumidification is needed if the RCC is warm.

The problems of humidity and overheating occur least with an RCC in which carbon dioxide is removed by flushing the chamber with compressed air. This also removes water vapour, and the diver can keep cool by sweating.

ACKNOWLEDGEMENT

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28

Cold and hypothermia

JOHN PENNEFATHER

INTRODUCTION

Immersion in cold water causes a complex response. In some cases the exposure may be rapidly fatal due to cold shock. A victim who survives this period may not be able to rescue themself because of a loss of power. Divers need to be aware of hypothermia as a diving problem, but it is also a problem if they become hypothermic during the remainder of their time on the water, when they are likely to be less well protected.

If exposure to conditions in which the heat loss from the body is greater than the heat production continues, then body temperature falls and the casualty becomes hypothermic. In all except the warmest seas, divers need assistance to maintain the balance between heat production and heat loss to the water. As discussed in Chapter 27, a wet suit generally provides adequate insulation, but in colder climates a dry suit – which provides more insulation – may be required. In extreme conditions a diver's body and breathing gas will also need to be heated.

Failure to maintain heat balance results in a fall in deep body temperature. If this is mild (1–2°C), the patient feels cold and may shiver. This, and a loss of dexterity, may affect delicate manual tasks. A continued loss of heat results in the body temperature falling to a level where the diver is incapable of looking after himself and is liable to drown. At still lower body temperatures, death occurs even if drowning is prevented.

Hypothermia is a common cause of death in people who are immersed as a result of a marine accident. None of the 1498 passengers who entered the water after the sinking of the *Titanic* survived. Despite the fact that many could swim and had lifejackets, few lived longer than 40 minutes. Almost all passengers in the life-boats were saved. Figure 28.1 shows the relationship of expected survival time to

water temperatures. Curves of this type should be used with caution; for example, most curves predict that people could not swim the English Channel, but the swimmers don't take much notice!

The realization that hypothermia was one of the most common causes of death of sailors during the Second World War led to the development of covered inflatable life rafts and exposure suits that give better protection in cold water. Information on all aspects of hypothermia, including some not considered in this discussion is available in several reviews. Keatinge's text is famous.[1] A recent treatment from the perspective of cruise medicine will be of use to practitioners in the field.[2] It also treats aspects that are not considered here, such as the role of alcohol (none), the options for temperature measurement and the problem of local cold injury. Two other recent reviews should be consulted by people likely to deal with hypothermia in a clinical setting.[3–5]

Hypothermia is one of the major limitations to deep diving in cold seas, such as the North Sea and off-shore Alaskan oil fields. Similar temperatures are experienced by amateur divers in the waters off northern Europe and North America.

Information on treatment comes from the management of clinical cases of hypothermia and observations made during induced hypothermia in surgical procedures on neurological and cardiac patients.

Other workers are interested in the physiological response to immersion and the development of better protection for divers and for people who may suddenly be dropped in the sea. This group includes sailors, fishermen and occupants of aircraft flying over water. For ethical reasons, the subjects in these experiments can only be chilled to a modest extent, so there is doubt as to the relevance of these data in comparison with a severely chilled subject.

In this chapter the accent is on the clinical features, prevention and treatment of hypothermia as encountered by the diving physician. Previously, little consideration would have been given to the chronic hypothermia that develops in the elderly and malnourished in cold climates because this causes different responses and may require different management. A well-insulated diver should not suffer acute hypothermia. But there will be exposures to the transition between rapid-onset and chronic hypothermia. There is a water temperature range where the body can initially maintain core temperature. Hypothermia develops when the energy sources have been depleted. Sound information on the appropriate management of such cases is not available.

As an example of this transition, Beckman and Reeves[6] planned to immerse 24 subjects up to the neck in a 24°C bath for 12 hours. This should not have induced severe hypothermia, but only six

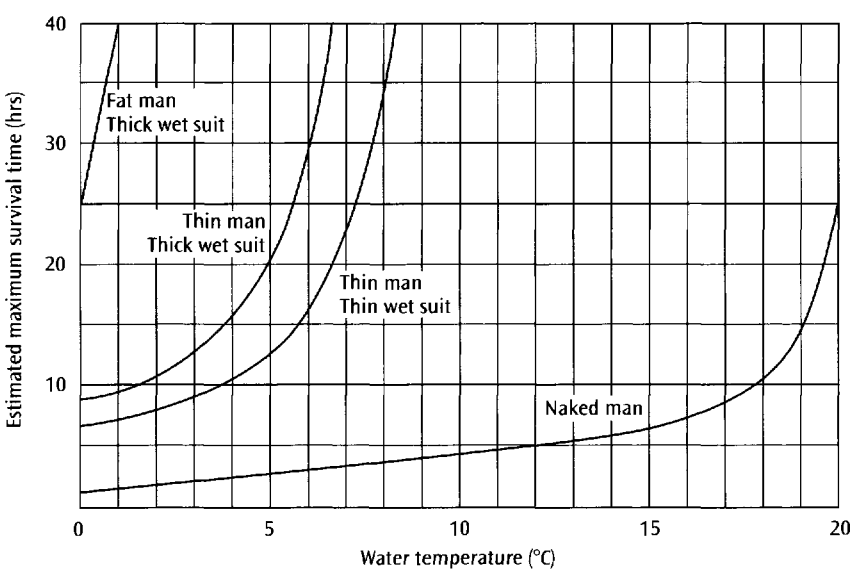

Figure 28.1 *Survival expectancy related to water temperature.*

subjects completed the immersion. Two were withdrawn with mild hypothermia, two suffered nausea associated with hypoglycaemia, and 13 had severe cramps.

The correct management of cases with mild hypothermia and associated symptoms caused by prolonged exposure to cool water is unclear.

INITIAL REACTIONS TO IMMERSION

Until recently the initial responses to cold water immersion have not been well explained. In many cases, good swimmers have died within a few metres of safety after short periods of immersion. Some workers postulated that these deaths were caused by inhaling water, while others suggested a cardiac aetiology. The Royal Navy studies in this area was reviewed by Tipton,[7] who provided an explanation of these fatalities. Within the first 3 minutes a complex series of changes are initiated that may be divided into several groups as summarized below.

Cardiovascular responses

There is an immediate increase in heart rate of about 20 beats/minute and an increase in cardiac output. This is accompanied by an increase in blood pressure and there is a rapid fall in peripheral blood flow as a consequence of vasoconstriction. These responses probably explain some of the sudden deaths.

Subjects with coronary heart disease are at risk because of the increased cardiac workload. The tachycardia will also impair coronary blood flow. The second group at risk are those with arterial disease as the hypertension may trigger vessel rupture and death from a cerebrovascular incident.

Respiratory responses

The initial 'gasp' on entering cold water may be followed by uncontrollable hyperventilation. There may be a ten-fold increase in ventilation, but three- to four-fold increases are common. This response can lead to water inhalation and drowning. This is more likely to occur in rough water, or where there is a period when the head is immersed. The victim cannot hold his breath, so even a good swimmer may aspirate water.

A less obvious problem is that the hyperventilation causes hypocapnia. Tipton cites a study where the arterial carbon dioxide tension fell by 12 mmHg after an iced-water shower for 1 minute. He suggests that this fall could cause enough reduction in cerebral blood flow to explain the disorientation and clouding of consciousness that has been noted.

Increased ventilation can trigger bronchoconstriction in asthmatics. In normal subjects there is an inspiratory shift in end-expiratory volume so that the subject is breathing close to their total lung capacity. This is an inefficient form of respiration, and will rapidly induce fatigue

A more complete version of possible cold shock responses is shown in Figure 28.2

Another response to immersion in cold water is a decrease in swimming performance. Tipton *et al.*[8] had subjects swim at a range of temperatures, only half of them could complete a 90-minute swim in 10°C water. A decrease in stroke length and reduction in distance travelled for a given energy expenditure was observed in the subjects who did not complete the cold-water swim. People with more fat over the arms fared better, suggesting that part of the decrease in performance may have been due to local cooling rather than to generalized hypothermia.

If the subject survives the initial immersion the responses tend to return to normal. Hypothermia will start to develop, but may also develop in subjects who have had a less dramatic immersion.

Immersion in cold water usually causes:

- *Tachycardia*
- *Hypertension*
- *Hyperventilation*

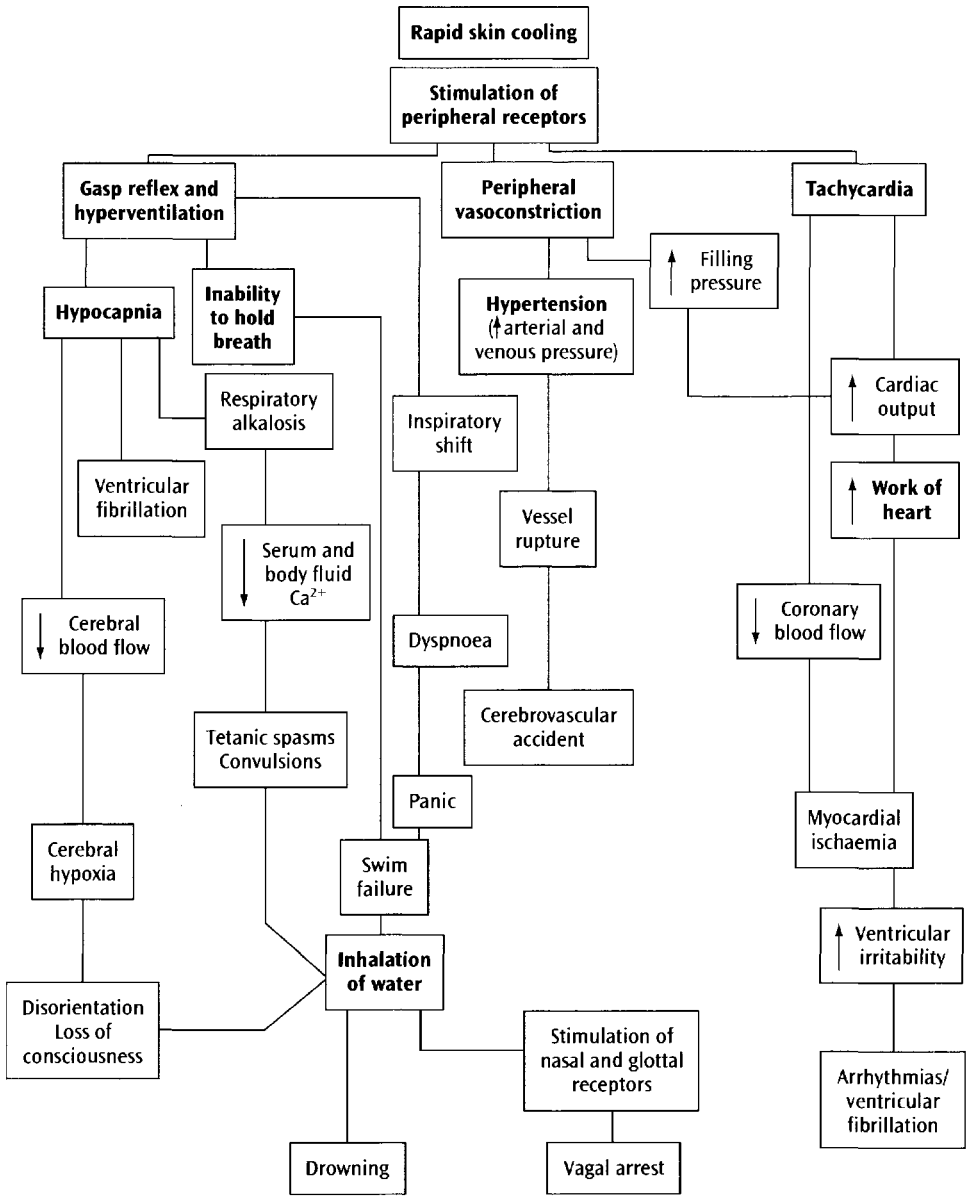

Figure 28.2 *A more complete version of possible cold shock responses. ↑, increase; ↓, decrease. (Adapted from Tipton, 1989.)*

SIGNS AND SYMPTOMS OF HYPOTHERMIA[3,4,9]

The effects of hypothermia in humans are variable, and may be complicated by those of immersion. The degree of hypothermia depends on environmental and physiological factors. Environmental factors include the water temperature and flow, the duration of exposure, the insulating materials (fabrics, fat, grease, etc.) and the gas mixture employed. Physiological factors include somatotype, activity during exposure, the degree of cold adaptation, and the use

of drugs which induce vasodilation or prevent heat-saving vasoconstriction.

With rare exceptions, the lethal lower limit for humans has been 23° to 25°C (rectal). The effects of hypothermia are set out below.

Mild hypothermia

The core temperature is in the range 33–35°C. The victim will be handicapped by the cold but should be breathing and fully conscious. They will probably be shivering and experience local reactions, including the sensation of coldness in the extremities. Numbness occurs as the peripheral sensory nerves are affected. Vasoconstriction, particularly in combination with immersion, leads to a diuresis which can cause dehydration.

Difficulty in performing coordinated fine movements, due to motor nerve involvement, may result in a dangerous situation in which a diver cannot effectively manage a task or the equipment. This loss of control because of cold hands may also be a problem in divers with normal body temperature.

The other major danger is that lethargy and sluggish reactions may lead to an accident or drowning. Other local reactions such as immersion foot (trench foot) and frostbite are more applicable to general and military medicine than to diving medicine.

Moderate hypothermia

With a core temperature between 30°C and 33°C a diver will be slow to respond or unconscious. Shivering is a variable response and, if present, it often ceases in this temperature range and is replaced by muscle rigidity. Heart rate and cardiac output falls. The electrocardiogram (ECG) may show prolonged Q-T intervals, and the QRS complex may develop a terminal complication known as a J wave. Nodal rhythm, atrioventricular (A-V) block or atrial fibrillation can develop. Respiratory frequency falls with the reduction in tissue oxygen needs. Many of the maritime cases of hypothermia succumb at this stage as they are no longer able to contribute to their rescue, to keep swimming, or even keep their head above water.

Severe hypothermia

The victim will have a core temperature below 30°C. They will be unconscious or semi-conscious, and shivering may be replaced by muscle rigidity that can be confused with rigor mortis. Respiration and pulse may be depressed or not detectable, and respiration may be reduced to one or two gasps per minute. There is a high risk of ventricular fibrillation. Any electrical activity on the ECG or electroencephalogram (EEG) is evidence of continued life. Bizarre ECGs should not be considered artefacts. In a field situation the pupillary light reflexes may be helpful, but their absence is not evidence of death. The chilled brain has a greatly increased tolerance to circulatory arrest, 25 minutes at 25°C. The Alaskan dictum, 'do not assume a patient is dead until he is warm and dead' may lead to unsuccessful attempts at revival, but this is preferable to any unnecessary deaths.

The report by Gilbert *et al.*[10] provides dramatic support for the Alaskan dictum, as these authors reported the resuscitation of a victim with a core temperature of 13.7°C. A skier became trapped in an ice gully and was continuously flooded by freezing water. She struggled for about 40 minutess and was trapped for a further 40 minutes before she was extracted and basic first aid started. This was replaced by cardiopulmonary resuscitation (CPR) and positive-pressure ventilation with oxygen during a 1-hour flight to hospital. Given advanced hospital care she has made a good recovery.

In her review, Larach[11] mentions cases where survivors were neurologically intact after over 4 hours of cardiac arrest associated with hypothermia.

Clinical phases of progressive hypothermia:

1 Mild	*35°C to 33°C*
2 Moderate	*33°C to 30°C*

Classification by duration

Lloyd[12] has suggested an alternative classification based on the manner in which the hypothermia is developed. These are:

1 Very rapid onset, of which accidental immersion in cold water is an example.
2 Subacute, or exhaustion hypothermia, in which there is moderate cold stress and exhaustion leads to hypothermia.
3 Subclinical chronic in which mild cold strains a reduced energy supply system and causes hypothermia.

It is Lloyd's opinion that this separation is necessary because it dictates how the patient should be rewarmed. Diving medicine has tended to concentrate on the rapidly cooled patient, but Lloyd's ideas deserve consideration because it is likely that any victim in a protective suit will fall into the second category. In some tropical waters any victim is unlikely to suffer severe hypothermia as the water is too warm, other recent reviews do not adopt this approach.[3–5]

SYSTEM REVIEW[4,9]

Cardiovascular system

The initial response to immersion in cold water has been discussed earlier. Later, as temperature falls the force of ventricular contraction is reduced, and this leads to a reduction in blood pressure, heart rate and cardiac output. Ultimately, cardiac arrest may occur at about 20°C (rectal). Various arrhythmias are common – atrial fibrillation at about 30°C, and ventricular fibrillation usually below 25°C. The blood becomes more viscous, and because hypothermia reduces the effective release of oxygen from haemoglobin, tissue hypoxia may develop.

In the past it has been emphasized that movement of the throat, limbs or chest during rescue or resuscitation may trigger ventricular fibrillation in a hypothermic heart. However, Larach[11] presents data that show this to be a rare complication.

If skin temperature falls below about 10°C the function of the smooth muscles controlling vasoconstriction may be impaired and vasodilatation occurs. This leads to a local increase in temperature and restoration of vasoconstriction. This cold-induced intermittent vasodilation accelerates heat loss and the rate of temperature fall.

For a diver, the effects of hypothermia on gas transport are complex. Cold decreases the blood flow to most areas, and this could be expected to decrease gas uptake. However, cooling increases the solubility of gases and the amount of gas transported to a tissue at elevated partial pressures. A further complication is the effect of rewarming after a dive where an increase in temperature decreases the solubility of inert gases. This can trigger gas release and precipitate decompression sickness.

Central nervous system

With a core temperature below 35°C , there is an impairment of speech, fixation of ideas, sluggish reactions and mental impairment. Depersonalization, amnesia, confusion and delirium are also possible. Unconsciousness may develop at about 30°C, and by 27°C most reflexes are lost. Exposure to cold initially causes reflex hyperventilation, with hypothermia, the respiratory centre is depressed, and this contributes to hypoxia and acidosis. There is also a rare form of sudden apnoea following cold-water stimulation of cutaneous receptors.

Hypothermia may serve a valuable protective function because the harmful effects of reduction of cerebral blood flow are mitigated by a reduction in metabolic rate. At 30°C the blood supply to the brain can be stopped for 10 minutes without neurological damage. Indeed, successful resuscitations may be explained by hypothermic protection.

Gastrointestinal system

There is some slowing of intestinal activity, and retardation of the rate of destruction of bacteria. Paralytic ileus may develop in cases of severe hypothermia.

Renal system

Cold and immersion cause an increase in central blood volume and diuresis; hyponatraemia and hypovolaemia may follow. As the temperature falls further, cardiac output and hence glomerular filtration are reduced, resulting in a decreased urinary

output, while acidosis results from both lactic acid accumulation and respiratory depression. Lysis of red blood cells may result in cold haemoglobinuria.

Liver

There is a decrease in liver function, probably caused by a direct temperature effect on enzyme kinetics. As a consequence, metabolites such as lactate may accumulate. Drugs accumulate because their clearance is slowed or stopped.

> *A chilled liver may not metabolize drugs.*

Locomotor

Shivering is a heat-producing response to cold, and mainly affects the large proximal muscles. However, it also causes a loss of coordination and difficulty in the performance of fine tasks as well as a loss of muscle power. Swimming ability is decreased, with increasing discomfort and fatigue. Apathy and euphoria may combine with fatigue to stop a diver taking appropriate action for their rescue.

Cutaneous reactions

Any prolonged immersion results in softening and swelling of the skin, rendering it susceptible to injury and infection. This 'washerwoman's skin' is characterized by soft ridges, especially over the tips of fingers and toes, where there is a large surface area to mass ratio.

In cold water there may be a sudden release of histamine in susceptible persons, causing cold or allergic urticaria. In some cases the skin rapidly becomes hot, red and oedematous. Symptoms may occur during or after exposure. Occasional deaths have been reported (See Chapter 42).

Cold-induced vasodilation of the peripheral vessels has been discussed earlier. In very cold water this may not stop the skin freezing. There is a variety of values of temperature at which skin freezes, and it is possible for skin to freeze when immersed in seawater near this temperature. Sullivan discusses frostbite and related conditions.[2]

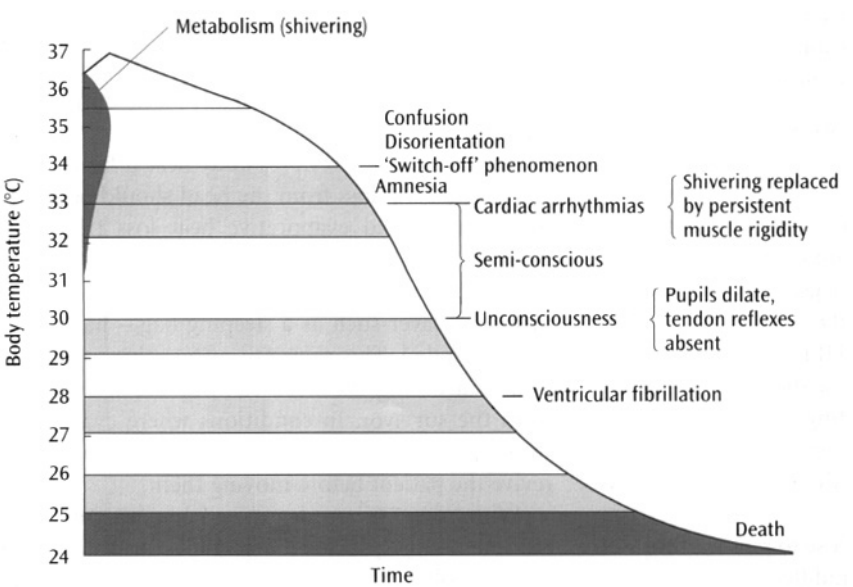

Figure 28.3 *Clinical features of acute hypothermia: the curve represents the behaviour of body temperature during cold-water immersion, with associated clinical features at various body temperatures.*

PREVENTION OF IMMERSION SHOCK AND HYPOTHERMIA

Immersion shock can be reduced by wearing protective clothing which attenuates the stimulus. Cold habituation will also condition the subject. These factors can also be used in the prevention of hypothermia. Another way to help prevent immersion shock is to enter the water slowly. If possible, it is also important to avoid letting the face go under water, so that any involuntary gasp is of air, not water.

The following preventive measures have been used or proposed:

- *Wearing insulating fabrics or coatings.*
- *Increasing the body deposits of subcutaneous fat.*
- *Reducing exercise in the water and assuming a position to minimize the surface contact area.*
- *Acclimatization to cold, over long periods.*
- *Careful desensitization, which may reduce cold urticaria.*

Hypothermia can be delayed or prevented after maritime accidents, and the following advice may assist people in these circumstances:

- Wear a wet suit or a survival suit to reduce heat loss.
- If wearing a life jacket, try to adopt a spheroidal position (foetal position) with the head out of the water, and the legs pulled up to the chest and the arms wrapped round the legs. Hayward and co-workers (see Sullivan, 1999[2]) call this the Heat Escape Lessing Posture (HELP), and they have shown that this may increase the survival time by 50 per cent. 'Drownproofing', where the victim rests with head under water between breaths is not advocated because of the increased heat loss from the head.
- Do not swim unless very close to safety; groups of survivors should remain huddled tightly together to conserve heat (and to give the rescuers a larger target to find).
- Wear clothing to reduce heat loss, in particular a hood or some other head protection is important in conserving heat.
- Avoid or delay immersion if any other options are feasible.

A diver is in a better situation than others as he knows he will be entering the water, and can plan for it.

TREATMENT OF IMMERSION SHOCK AND HYPOTHERMIA

It is desirable to obtain a measurement of the victim's temperature if this is possible. In most facilities a rectal temperature can be obtained; this is the best measurement of those commonly used.[2] When a rectal temperature cannot be obtained, the tympanic membrane temperature offers a useful alternative, with minimal disturbance to either the patient or their protective insulation. Others choose oesophageal temperature as their preferred measure of deep body temperature.[3]

A victim of immersion shock may respond to normal resuscitation techniques. A casualty may suffer cardiac failure, a cerebrovascular accident or drowning. They may also have hypothermia if rescue has been delayed. In mild cases of hypothermia, removal from the cold environment, protection from wind, the use of blankets, and hot-water bottles in a sleeping bag are all remedies that have worked.

Shivering slowly restores body heat if heat loss is prevented. Heat loss from the head should be minimized, as should evaporative heat loss from wet clothing. In an exposed situation, two large plastic bags – one over the victim's body and one over an insulating layer such as a sleeping bag – have been recommended. This prevents evaporative cooling as well as avoiding the need to strip the wet clothes from the survivor. In conditions where evacuation would involve further cooling it is often better to revive the patient before moving them.

With more severe degrees of hypothermia, the treatment depends on the facilities available. The victim probably will not be shivering, but has little heat production. Movement may sometimes trigger ventricular fibrillation, and active treatment can

result in aggravation of core hypothermia and/or hypotension (see below).

Specific treatments of serious hypothermia should include the following approaches.

First aid

It is important to keep the patient horizontal during, and following, removal from the water. A rescue basket, a stretcher or a double strop system, with one loop lifting the patient under the arms and another under the knees, can be used. This prevents a sudden fall in blood pressure that can occur with the loss of hydrostatic pressure on the legs.[13] There have been cases where the patient was alive and responsive in the water, but apparently dead by the time he reached a rescue helicopter after being lifted in a vertical position. This shock reaction is thought to be caused by blood pooling in the legs.

Care should be taken to avoid any unnecessary manipulation of the throat. For example, the use of a sucker can cause reflex vagal slowing of the heart and may precipitate ventricular fibrillation. The airway must be clear, but if suction is necessary, it should be performed with care.

It is important not to give cardiac massage or artificial ventilation just because the spontaneous rates are slow. Sudden over-ventilation and/or trauma to the heart can cause ventricular fibrillation.

> *The pulse may be difficult to detect in a hypothermia casualty.*

The only circumstance in which cardiac massage and artificial ventilation are likely to be beneficial in hypothermia is when the heart has stopped or is in ventricular fibrillation. If drowning has occurred, then normal CPR takes precedence over the management of hypothermia. Cases with hypothermia, or drowning and hypothermia, will normally need rewarming before normal cardiac function can be restored and rewarming can correct the abnormality. Therefore it is probably unwise to treat any electrocardiographic abnormalities except asystole until temperature has returned to normal.

Correction of fibrillation by drugs or defibrillation is not generally effective when the core temperature is below 30°C. The use of drugs is also contraindicated because they will not be metabolized until the victim has been warmed, by which time they may not be needed. A ventilator with a heater and humidifier is needed to prevent respiratory heat loss.

HOSPITAL CARE

The victim with severe hypothermia will need hospital care during and after rewarming. The choice of rewarming method will depend on the skills and equipment available, and some of the options are outlined below. Intravenous fluids should be warmed to at least body temperature, but how hot is a subject for debate. One animal study suggests that centrally administered intravenous fluid can be as hot as 65°C.[14] The use of solutions containing lactate should be avoided, as this will not be metabolized by a cold liver.

Normal saline, containing glucose if the patient is hypoglycaemic, is suggested. Immediate bolus administration of 500 ml, then 100 ml/hour, is a simple guide to overcome haemoconcentration and possible shock as the peripheral vessels expand.

An alternative argument suggests that there may be complications in the use of fluids producing an overload. If hypovolaemia is caused by hypothermia, then it is suggested that the fluid may return to the vascular system during rewarming.[3]

It is important not to treat any abnormalities of blood chemistry unless the blood sample is a reliable one, preferably taken from a central vein, and with all temperature corrections made.

Thick bronchial secretions or aspirated gastric contents may require suction; this requires care to prevent developing arrhythmias. After rewarming, antibiotics may be needed for respiratory infections.

METHODS OF REWARMING

A recent review of therapy has stressed the shortage of trials comparing methods of rewarming patients

with moderate or severe hypothermia.[15] Most victims of acute, mild hypothermia will recover if allowed to rewarm passively. However, to speed the process they can be put into a hot bath and disturbed as little as possible; this seems to be the best rewarming treatment in a small hospital. An initial water temperature of 36°C, to reduce the pain response and risk of atrial fibrillation, and then an increase over 5–10 minutes to 40° to 42°C, until the rectal temperature is above 33°C, has been recommended. Others advocate a 42°C bath on the assumption that a cold body will lower the temperature and more heat will be needed. There is no evidence of any difference in outcome from the two approaches.

It has been suggested that limbs should be placed in an elevated position, outside the bath. Often, a temporary degree of hypotension accompanies vasodilation, but the elevation of the limbs is usually sufficient to counteract this. This position was said to reduce core cooling by cold blood from the extremities during the initial stages of rewarming. Hoskin *et al.*[16] suggest that trunk-only immersion is not superior to whole-body immersion in a hot bath.

In a small hospital which is not prepared for cases of hypothermia, as well as on a boat, hypothermia may have to be treated without even the assistance of a low-reading clinical thermometer. The diver can be left in his wet suit and immersed in warm water until he is asymptomatic and has started to sweat. Removal of the wet suit is unnecessary and involves possibly dangerous manipulation. On the other hand, its presence helps to protect the patient against pain from the hot water and hypotension from peripheral pooling of blood.

In these conditions the use of active treatment must be weighed against the risk of possible complications from the rapid rewarming. Some workers advocate less rapid rewarming. This allows the body more time to initiate its normal corrective responses to any disturbed biochemical parameters. This may be the better approach for a victim who has had a long period of immersion. A glucose infusion may be indicated. In a review, Lloyd[12] suggests that the person who has cooled slowly should not warm faster than 0.5°C/ hour, but others ignore this aspect of the problem and make no reference to the duration of immersion as being a factor for consideration.[5,11]

Rewarming under a cover with forced hot air has become an accepted method of rewarming patients with acute hypothermia.[3,17] It has the advantage of being easy to administer if there is access to mains electricity, and also provides a satisfactory rate of rewarming with comparatively little interference with the subject.

The common response – to have a hot shower – is far less effective. It may also be dangerous as the diver feels he has recovered before he has been adequately rewarmed. Also, injuries have been reported when a diver has fallen in a shower as a result of hypotension and loss of consciousness.

Active core rewarming

This has been achieved by haemodialysis, intragastric irrigation, peritoneal dialysis and extracorporeal circulation, all utilizing a system of warming the perfused fluid. Of these methods, extracorporeal circulation from the femoral artery to vein is the most common, and has given good results. Some consider venoarterial warming with complete cardiopulmonary bypass to be the method of choice as this restores perfusion and oxygenation as well as providing a warming effect.

Central venous infusion of warmed fluids may increase the temperature of the thoracic viscera, though care must be taken not to induce hyperpyrexia with active core rewarming. Warming of the humidified inspiratory gases to 38–42°C is recommended but has not proved to be a successful method of rewarming as originally hoped.

The use of low-power microwave radiation, based on the same principle as the microwave oven, has been used experimentally. This technique offers potential benefits as it could allow the heating of selected areas or organs from the exterior. One could possibly warm the liver, then the aortic blood, and so rewarm the body with less fear of complications than with current techniques.

With this range of options there is a shortage of randomized trials of the therapies. A review of methods has been attempted,[15] but in many of the trials the subjects would have improved with any therapy, and the main index of the performance of the methods was the rate of rewarming produced.

CASE REPORT 28.1

IQ was undergoing a diving course in winter. Following an uneventful dive to 10 metres for 20 minutes he joined the rest of the course in a surface swim. After 15 to 20 minutes he was noted by his instructor to have stopped using his hands. He was told to return to the boat, but showed no response. Soon after this, he submerged twice and required assistance. Back on board he looked pale and exhausted. He was confused, shivering, incoherent in speech and uncoordinated in movement. When the boat was returning to shore, IQ was placed near the engine and massaged, but did not improve. He was then given a hot drink and hot shower. When seen by a doctor 30 minutes later, at which time his temperature was 34.8°C orally and 35.0°C per rectum, he appeared a dull grey ashen colour, was only able to walk with assistance, and was still confused and amnesic. He was placed in a hot bath with arms and legs out and gradually improved. Over the next hour his body temperature returned to normal. Chest X-ray and FEV_1/VC measurements had not changed. Several hours later he was asymptomatic.

One medical humorist reported a comment made by an Alaskan researcher to the effect that people use what they know best. The proctologist will use warm enemas, while the urologist will favour bladder lavage. The major hospital will choose cardiopulmonary bypass, and all this is based on experience and skills rather than sound evidence.

A basic problem in assessing methods of rescuscitation is that we lack answers on the questions of how hot the gas or fluid can be. Most of the people using warmed humidified gas have suggested temperatures of 40–42°C, but in a sauna people are exposed to humid air at over 50°C without injury. Similarly, dogs have been transfused with intravenous fluids at 65°C in an experimental treatment of hypothermia.[14]

PROLONGED IMMERSION

The problems of a diver who has been immersed for a long period have not been investigated in a systematic manner. It is known from rescues of divers with adequate insulation that survival for at least 24 hours can be expected. About one-third of the crew of the *USS Indianapolis* survived for about four days in life jackets after being sunk near the Philippines. The main problems for them was dehydration and mental problems. It is not clear if these stemmed from lack of sleep, anxiety related to the delayed rescue, or were a consequence of severe dehydration.[18]

Beckman and Reeves[6] considered that hypoglycemia, dehydration, muscle cramps, haemoconcentration and adrenocortical stress response were factors to be considered as well as hypothermia in this group. This would suggest that fluids with glucose are needed as well as heating, though how the warming should be administered in these cases is open to question. Probably the best advice is to 'be prepared for complications'.

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29

Local infections

CHRIS LOWRY

INTRODUCTION

A variety of pathogenic organisms may be encountered through contact with water. These include bacteria, viruses, protozoa and parasites. Infection associated with swimming or diving may be acquired in a number of ways. Water-borne pathogens may enter the body through intact or damaged skin, or mucous membrane. Portals of entry include eyes, ears, nose, throat, lungs, and gastrointestinal or genitourinary tracts. The infection may remain localized to the site of entry or progress to severe, systemic disease (see Chapter 30).

The microscopic flora of water is determined by such factors as its proximity to human habitation, the content of organic matter, pH, temperature, light, salinity, oxygenation and rainfall. Some microorganisms are found naturally in water, others are periodically washed into water from soil, and others favour the artificially created environments of swimming spas, baths and aquaria.

The exact mode of penetration of the organism into a body site depends on the interplay of a number of factors involving water, pathogen and host. For example, divers may lose the bacteriostatic benefits of cerumen, normal flora and acidic pH in the external ear with repeated immersion, thus predisposing to external-ear infections. Factors affecting host defenses to permit infection by aquatic microorganisms include trauma, aspiration and immunosuppression.

Marine bacteria were, until recently, thought not to cause infections in humans. In the past 30 years, these organisms have been isolated from a variety of human infections. Such pathogens may obtain entry by trauma from coral, rock or marine animal spines, teeth or shell, or from the ingestion or aspiration of water.

The morbidity resulting from diving with certain pre-existing infections is perhaps more important and certainly more common than infections directly due to swimming or diving. These infections are mainly those affecting the respiratory system and both cause and complicate barotrauma.

Upper respiratory tract infections render the sufferer liable to barotrauma of sinuses and/or middle ear, due to blockage of equalizing air passages (see Chapters 7 and 8). Lower respiratory tract infections, where there is likely to be bronchospasm, mucus plugs and obstruction of smaller airways, are dangerous particularly if a flap valve arrangement exists. Gas can enter the alveoli distal to the 'valve' during descent, but is trapped as it expands on ascent, causing pulmonary barotrauma (see Chapter 6).

It has been suggested that spread of respiratory infections is enhanced by diving, and this does appear to be clinically so in some cases. Sharing of regulators may be a cause of cross-infection. Alternatively, many divers claim that a dive is a good way of 'cleaning the sinuses' of mucus resulting from a mild inflammation. Saline irrigations have a good reputation among sinusitis sufferers.

Special problems of infection control arise in the closed environments of saturation chambers, undersea habitats, submarines and hyperbaric facilities.

AETIOLOGY AND PATHOGENESIS

Infections associated with the marine environment and aquatic activities are common. This is due to increasing use of water for recreational activities and also, unfortunately, to increased sewage pollution of oceans and inland waterways.

Infecting organisms are usually bacteria, but protozoa, viruses, fungi and helminths may also be involved. Seawater contains many bacteria known to be potentially pathogenic for humans (Table 29.1).

The outcome of exposure to water-borne organisms depends on a number of factors. Factors relating to the organisms themselves include the causal organism, inoculum size and virulence. Host factors include site of inoculation, gastric acidity (for gastrointestinal infection) and host immunity. Interaction between the organism and host factors determines the outcome of exposure, i.e. whether infection occurs and, if it does, whether mild or severe disease results.

Many water-borne organisms, which were previously considered non-pathogenic or of doubtful pathogenicity, have been implicated as definite pathogens – some causing severe, even fatal, disease. Certain species may be associated with disease more often. For example, *Aeromonas sobria* and *Aeromonas hydrophilia* cause most clinically significant aeromonas infections. Severe invasive disease due to non-cholera vibrios is most commonly identified with *Vibrio vulnificus*; this can cause overwhelming septicaemia, with rapid progression to death within 48 hours. The production of virulence factors by bacteria (e.g. cytolysins, proteolytic enzymes) may be seen with some organisms and correlate with invasiveness and pathogenicity.

Host factors may potentiate the development of infection. The skin is a most effective protection, and inoculation of organisms below the skin surface by penetrating injuries, or coincident with an apparently trivial abrasion or laceration, commonly enables environmental organisms to establish clinical infection, which would otherwise not occur. Severe or even fatal infections due to *V. vulnificus* predominantly occur in immunosuppressed persons, e.g. chronic renal disease, liver disease or those on immunosuppressive therapy. HIV-infected persons might be at greater risk. A particularly strong relationship is seen with haemochromatosis. Severe infections in normal individuals are not common, but do occur frequently enough to cause concern.

Table 29.1 *Some potentially pathogenic bacteria found in seawater*

Acinetobacter lwoffi	*Klebsiella pneumoniae*
Actinomyces species	*Mycobacterium marinum*
Aeromonas hydrophilia	*Plesiomonas shigelloides*
Aeromonas sobria	*Proteus mirabilis*
Alcaligenes faecalis	*Pseudomonas aeruginosa*
Bacillus subtilis	*Pseudomonas* species
Clostridium botulinum	*Salmonella enteriditis*
Edwardsiella tarda	*Serratia* species
Enterobacter species	*Staphylococcus aureus*
Enterococcus species	*Staphylococcus epidermidis*
Enterococcus faecalis	*Vibrio alginolyticus**
Erysipelothrix species*	*Vibrio cholera*
Escherichia coli	*Vibrio parahaemolyticus**
Flavobacterium species	*Vibrio vulnificus**

*More likely to be involved in marine-acquired infection.

In the external ear and elsewhere in the body, environmental factors such as prolonged immersion resulting in skin maceration or high humidity aid bacterial penetration, even through intact skin. Bacteria have been shown to survive longer in moist skin, and may then gain entry following relatively minimal integumentary damage. The softened macerated skin of immersion is, of course, more prone to damage from minor trauma. These organisms may be either water-borne or a part of the diver's normal flora.

SITES OF INFECTION

Aquatic-derived infections are categorized according to the apparent site of entry of the organism. Some specific organisms are also discussed; note that the order in which organisms are referred to is not meant to imply incidence or importance, nor is the list to be regarded as complete. The diving physician must think laterally when facing an unusual presentation. For example, cutaneous larva migrans (due to a hookworm) has been described on the feet of a scuba diver![1]

Wound infections

Integumentary trauma is a common association of aquatic activities, be they recreational or occupational. Organisms isolated may be found in seawater or are normal human skin flora. They include *Staphylococcus aureus,* pyogenic *streptococci, vibrios, Enterobacter, Pseudomonas* and *Bacillus.* Although potential pathogens may be present either in the water or the skin, many marine infections arise because the pathogen is part of the normal flora of the agent producing the trauma, such as coral (see below), fish spines or seashells. In this context, various species of halophilic or marine *vibrios* are frequently involved.

A vivid illustration of the potential for introduction of infection is a study of the teeth of a great white shark, which were found to harbour potentially pathogenic species of *Vibrio, Staphylococcus* and *Citrobacter.*[2] Swabs of shark bite injuries have grown *Vibrio parahaemolyticus, Aeromonas caviae, Vibrio alginolyticus, Vibrio carcharia, and Aeromonas hydrophila* among others.[3]

Aeromonas hydrophila and *Aeromonas sobria* wound infections have followed diving in polluted waters.[4,5] *Aeromonas* species can cause severe progressive wound infections with cellulitis leading to osteomyelitis.

Marine-acquired wound infections range from mild local inflammation, cellulitis, lymphadenitis and abscess formation to severe spreading infection with systemic effects and even septicaemia. The more severe infections are often associated with *V. vulnificus* (see Chapter 30) or *Aeromonas.* Gas gangrene has been reported following the contamination of wounds received in an aeroplane crash in the Everglades, and also following shark attack, although the organism may have been introduced from terrestrial origins.

Treatment[6]

Wounds should be thoroughly cleaned and debrided of devitalized tissue and foreign material. This may require local or general anaesthesia.

Cellulitis, lymphangitis, etc. indicate the need for a broad-spectrum systemic antibiotic (e.g. doxycline, co-trimoxazole, cephalosporins), after a swab is taken for culture and sensitivity. In such cases, bed rest, elevation of the affected limb and other general supportive measures will also be required.

Wounds suffered in or out of the water, including ulcerations due to poorly fitting fins, are notoriously slow to heal in divers who spend considerable time in the water. Secondary infection is common and is aided by softening and maceration of the skin due to immersion, and also by swimming in contaminated water. A prolonged period of time out of the water with frequent dressings with antiseptic powder or ointments may be required to achieve healing. Prompt drying after immersion may be of prophylactic value.

Coral cuts[5,7]

This is a specific type of wound infection, as corals frequently cause lacerations and abrasions to inexperienced divers. These injuries may initially appear minor in nature, but because of foreign material

such as pieces of coral, nematocysts, infected slime, etc., they often become inflamed and infected.

Clinical features

The laceration, usually on the hand or foot, causes little trouble at the time of the injury, but some hours later there may be a 'smarting' sensation and a mild inflammatory reaction around the cut. This may be due to the presence of discharging nematocysts. In the ensuing one to two days, local swelling, erythema and tenderness develop around the site, but usually this abates in three to seven days.

Occasionally, an abscess or ulcer will form and discharge pus. This can become chronic, and osteomyelitis of the underlying bone has been reported. Cellulitis and/or lymphadenitis often accompany the acute stage. Fever, chills, arthralgia, malaise and prostration occur in some cases, probably reflecting the systemic effects of a severe bacterial infection. *Erysipelothrix rhusiopathiae* may be involved in the infection. Healing may take months to years if complications ensue.

> *Secondary infections of cuts and abrasions, fin ulcers, otitis externa and minor upper respiratory tract infections (such as sinusitis and otitis media) are the most common infections encountered in divers.*

First Aid

The wound should be thoroughly cleaned using a soft brush and a mild antiseptic solution. Bleach is often used in this respect. All foreign material should be removed – measures which incur some discomfort to the patient!

Treatment

When severe wounds have been cleaned and debrided, they should be dressed with a soft absorbent dressing. Antibiotic ointment applied several times daily (e.g. Bactroban) is effective if used early enough, but systemic antibiotics may be indicated if the infection has spread. Tetanus prophylaxis is advisable.

Prevention

Coral cuts can be avoided by the use of protective clothing, gloves and swim fins with heel covers, and prompt treatment of minor abrasions. Good training in buoyancy control for divers will also prevent damage to the coral.

Otitis externa[8,9]

Although otitis externa occurs without indulging in aquatic activities, swimming increases the risk about three- to five-fold. In divers, external-ear infection is one of the most common and troublesome disorders encountered.

Aetiology

Hot, humid conditions such as in tropical climates, standard dress diving or recompression chambers provide ideal conditions for this organism. Retention of fluid within the external auditory canal following immersion, particularly in contaminated water, adds to the risk. Exostoses, which are common in swimmers and divers, predispose to retention of cerumen, epithelial debris and water. Local trauma, such as attempts to remove cerumen by cotton swabs or syringing, are often associated with subsequent infection. Swimmers with dermatological conditions, such as seborrhoea, neurodermatitis and eczema, have an increased incidence.

The bacterial flora is usually mixed, but with *Pseudomonas aeruginosa, Staphylococcus aureus* and *Proteus* species predominating. Less commonly, *diphtheroids, Escherichia coli, Streptococcus faecalis, Aspergillus niger* and *Candida albicans* are involved.

> Pseudomonas aeruginosa *is frequently found in otitis externa in divers.*

Prolonged exposure to water changes the healthy ear flora from Gram-positive cocci and diphtheroids to Gram-negative bacilli, and this change often precedes the acute symptoms of otitis externa. In divers, *P. aeruginosa* is probably the most frequently associated organism in otitis externa, as the

external canal provides an environment that is particularly conducive to the growth of this organism. The organism can also be found in freshwater lakes, some of which are associated with a high risk of infection to swimmers.[10]

Pseudomonas and, *Escherichia* species and fungi are the most common pathogenic organisms in saturation diving environments, where there is commonly a high ambient temperature and relative humidity. Other marine bacteria causing otitis externa include *Achromobacter xylosoxidans, E. coli, Enterobacter* and *Klebsiella* species, and *Proteus* and *Vibrio* species.

Clinical features

The infection may be either circumscribed (furuncle) or diffuse. Symptoms include itching or pain in the ear made worse by jaw movements and traction on the tragus. Examination reveals localized tenderness, moist debris in an oedematous external canal, and a possible conductive hearing loss. The eardrum may not be visualized. Regional lymphadenopathy and, rarely, a purulent discharge may be present. Divers may complain of vertigo due to the obstruction of one canal.

Treatment

Management consists of analgesia with topical and/or oral analgesics, and gentle cleaning of the canal. This should be followed by antibiotic-steroid ointments, which should completely fill the canal or be impregnated in a wick, or regularly applied antibiotic drops. Ciprofloxin-hydrocortisone ear drops may be useful for *Pseudomonas*-mediated otitis externa. Antifungal agents are often included in these preparations. Systemic antibiotics may be required. Severe or unresponsive cases should have culture and sensitivity tests to aid antibiotic therapy. Diving should cease until the condition has cleared.

Prevention

The ears should be rinsed with freshwater to remove saltwater or contaminants. Salt crystallizes on drying in the canal and, being hygroscopic, retains moisture. Recurrence may be prevented by the use of paraffin or olive oil drops prior to diving. Spirit drops following the dive to ensure adequate drying may also be helpful. The prophylactic use of 2 per cent acetic acid in aluminium acetate twice daily and after immersion has been shown to reduce the incidence of otitis externa during saturation dives, which otherwise approaches 95 per cent. Glacial acetic acid 5 per cent in propylene glycol is very effective, as is 5 per cent acetic acid in 85 per cent isopropyl alcohol ('Aqua Ear'). The use of oil-based antibiotic-steroid ear drops might be needed in refractory cases where diving must continue.

Otitis externa can be a major problem in saturation diving, but active prophylaxis can significantly reduce the incidence.

Otitis media

This infection is much less common than otitis externa, but may occasionally complicate middle-ear barotrauma (see Chapter 7), especially when the latter follows an acute upper respiratory tract infection. The most commonly involved organisms are haemolytic streptococci, pneumococci or staphylococci. A severe case of suppurative otitis media in a diver caused by *Vibrio alginolyticus* has been reported. This patient had a long history of upper respiratory tract infections and otitis media since childhood, but continued to dive in warm seawater.[11] These or other mixed flora gain entry to the middle ear via the eustachian tube, or less commonly through a perforation of the tympanic membranes due to the middle-ear barotrauma or blast injury. The presence of water, fluid or blood provides a culture medium. Clinical features and management are similar to those of other causes of otitis media except that it is noted between 4 and 24 hours after diving. Otitis media following severe middle-ear barotrauma can usually be treated by systemic antibiotics.

Sinusitis

Acute sinusitis is also a recognized infection in divers. The aetiology, clinical course and bacteriological findings are similar to those of otitis media. Sinusitis sometimes follows sinus barotrauma (see Chapter 8).

Chronic sinusitis is a possible long-term occupational disease of divers. Orbital cellulitis, with the

CASE REPORT 29.1

AM had been diving intermittently for some years. He had mild symptoms of a 'cold' but was able to equalize his ears satisfactorily and so went ahead with the planned dive. He was unable to descend beyond 5 metres because of pain in both ears and inability to auto-inflate. After surfacing, the pain subsided, but otoscopic examination revealed grade 3 aural barotrauma in the right ear and grade 2 in the left. A few hours after the dive, he developed increasingly severe pain in the right ear, which was accompanied by tinnitus and later pyrexia. On examination, his temperature was 38.2°C and the tympanic membrane appeared lustreless and erythematous with an outward bulge. Audiometry revealed a mild conductive hearing loss in the right ear. The administration of antibiotics and decongestants resulted in symptomatic improvement in 24 hours. Seven days later the appearance of both tympanic membranes was normal, as was the audiogram.

Diagnosis: otitis media complicating middle-ear barotrauma.

infection extending from the sinus has also been observed; this is a medical emergency requiring intensive antibiotic therapy.

Eye infections

Keratitis due to *P. aeruginosa* has been reported following similar exposures to those resulting in pseudomonas folliculitis (see below). *P. aeruginosa* has also been associated with corneal ulcer and endophthalmitis.

Vibrio alginolyticus has also been reported to cause conjunctivitis,[12] as has *Chlamydia* following swimming in certain lakes.

Keratitis due to *Acanthamoeba* has been associated with exposure of the eye to contaminated water (e.g. hot tubs), especially in wearers of contact lens. This organism is ubiquitous in aquatic environments and more cases may be expected. Treatment is with oral ketoconazole and topical miconazole or neomycin or propamidine isethionate. Surgery such as keratoplasty or even enucleation may be required.

Gastroenteritis

Gastrointestinal infection may result from ingestion of water containing pathogenic organisms while swimming.

For such infections, the size of inoculum is important. For example, an inoculum of less than 100 bacteria is unlikely to cause significant infection or disease in normal hosts. An exception to this is infections due to *Shigella* species that may occur with ingestion of as few as 10–100 organisms. Gastric acidity is an important host defense. In people taking antacids or who have achlorhydria, the infective dose of organisms is considerably lower.

The most severe form with severe, profuse watery diarrhoea and dehydration and prostration is caused by *Vibrio cholerae*. Non-cholera *Vibrio* species, found in seawater, can cause gastroenteritis with nausea, vomiting, diarrhoea, fever and abdominal pain.

Other bacteria also found in water, and associated with gastroenteritis, include *Bacillus cereus, E. coli, Salmonella* species and *Campylobacter jejuni*. Severe infections have occurred following the consumption of shellfish, oysters and fish contaminated with marine organisms, especially *Vibrio vulnificus* (see below).

Swimmers in freshwater are also at risk of protozoan parasites such as *Giardia lamblia, Naegleria fowleri* and *Cryptosporidium parvum*.

SPECIFIC PATHOGENS

Vibrio[13]

The genus *Vibrio* (Gram-negative bacillus) tends to live in marine or brackish water, but has also been reported from inland waterways.[14] As well as cholera,

Table 29.2 *Pathogenic marine vibrios causing wound and other infections*

Organism	Infection/condition caused
V. vulnificus	Wound infection, cellulitis, septicaemia, pneumonia, meningitis, endometritis, peritonitis, gastroenteritis
V. parahaemolyticus	Otitis, conjunctivitis, osteomyelitis, gastroenteritis
V. alginolyticus	Cellulitis, epidural abscess, otitis, pneumonia
V. damsela	Septicaemia, otitis
(Non-01) *V. cholerae*	Gastroenteritis, wound infection

several species of *Vibrio* cause infection in humans. All are indigenous to marine environments and are natural flora of shellfish. They thrive in warmer temperatures. *Vibrio vulnificus* is the most virulent species, and is capable of causing severe wound infections, gastroenteritis and severe systemic illness in swimmers and divers (see Chapter 30).

Other *vibrios*, such as *V. alginolyticus*, have also been seen in marine wound infections, but seem less invasive than *V. vulnificus*. Nevertheless, a case of epidural abscess due to this organism has been reported, presenting three months after an open head injury incurred diving in seawater. A *V. damsela* wound infection has followed injury from a stingray barb.

Clinical presentations

These also include gastroenteritis, cellulitis, fasciitis, septic thrombophlebitis, vasculitis, conjunctivitis, and otitis externa and media.

A case of endometritis following sexual intercourse while swimming in seawater known to harbour *V. vulnificus* has been reported, again indicating the virulent nature of this organism.

Diagnosis

There must be a high index of suspicion. Microscopy and culture of wounds and blood should be undertaken, using appropriate media for marine organisms.

Treatment

Extensive surgical debridement and broad-spectrum antibiotic administration prior to confirmation of the organism is usually necessary. Tetracyclines in combination with gentamicin or one of the third-generation cephalosporins, such as ceftriaxone, or a fluoroquinalone, such as ciprofloxacin, may be appropriate, and commenced in severe infections while awaiting results of antibiotic sensitivy investigations.

Early and aggressive treatment reduces the risk of progression to serious infection.

CASE REPORT 29.2

A 20-year-old male struck his forehead on a submerged object while diving off a platform along the coast of Guam. He sustained an 8-cm laceration and lost consciousness for 2 minutes after the incident. The laceration was repaired, and X-rays were taken revealing a comminuted fracture of the frontal bone extending through the frontal sinus, with minimal depression of the fragments. A computed tomography (CT) scan showed the fracture site, but no other evidence of intracranial pathology.

The patient underwent frontal craniotomy with exenteration of the frontal sinus and realignment of the frontal bone fragments three days after the accident. Bacterial cultures of the subdural and epidural spaces were negative. The patient did well postoperatively, but did experience cerebrospinal fluid rhinorrhea. The patient remained asymptomatic for three months until intermittent fever and headache developed. A repeat CT scan revealed a displaced frontal fracture site as well as a large epidural fluid collection. Physical examination was completely normal other than the bony defect. Laboratory studies showed only that spinal fluid cultures were negative.

In the operating room, osteomyelitis of the frontal bone was noted. The entire frontal plate of involved bone was excised. A 25-ml collection of purulent material was recovered from the epidural space. Gram staining of this material revealed pleomorphic, curved, Gram-negative rods.

The aerobic culture of the epidural space and frontal bone tissue revealed heavy growth of *V. alginolyticus* in a pure culture.

The patient was treated with a four-week course of intravenous chloramphenicol at a dosage of 50 mg/kg per day without complication. The patient recovered without neurological sequelae, and was discharged without medication other than phenytoin for seizure prophylaxis. The patient was seen at follow-up six months later and was completely asymptomatic.

(From Opal and Saxon, 1986[15])

CASE REPORT 29.3

A 32-year-old woman presented with a 24-hour history of severe pelvic pain described by the patient as 'worse than having a baby'. She also complained of right lower quadrant pain, low back pain, and frequent urination with burning and constant cramping. Upon physical examination the patient appeared to be 'toxic' with a temperature of 38.4°C. Her lungs were clear and her abdomen was non-tender. Upon pelvic examination, there were no external lesions, but a non-bloody, purulent vaginal discharge was noted. The uterus was also very tender. An intrauterine device, which had been in place for one year was removed through the cervix and sent to the microbiology laboratory for aerobic culture. The patient initially received 4.8×10^6 units of benzyl penicillin (penicillin G) in divided doses intramuscularly. She also received oral doxycycline, 100 mg per day for 14 days. Two days later she was much improved with little discomfort.

After isolation of *V. vulnificus* from this unusual site (endocervix), the patient was interviewed as to possible sources of exposure to this marine bacterium. The patient had not eaten any seafood in the two weeks before the onset of symptoms. However, about 18 hours before the onset of pelvic pain she had been swimming in Galveston Bay and had engaged in sexual intercourse while in the water.

V. vulnificus has repeatedly been isolated from Galveston Bay, with a peak incidence during periods of warm temperatures and moderate salinities.

(From Tison and Kelly, 1984[16])

Erysipelothrix

Infections (erysipeloid) due to the Gram-positive bacillus *Erysipelothrix rhusiopathiae* or *insidiosa* are found worldwide. Abrasions due to contact with fish, shellfish, meat or poultry may lead to the infection which is usually limited to the skin. In the marine context, this organism is involved in crayfish poisoning, coral poisoning, seal finger, whale finger and fish handler's disease.

Clinical features

There is a history of skin injury, which may appear to heal during a one- to seven-day latent period. Then, a sharply defined purplish-red area spreads outward from the injury site, which becomes indurated. There is associated itch, pain or burning sensation. Oedema develops and adjacent joints become stiff and painful. Regional lymphadenitis or systemic manifestations such as endocarditis have been reported but are rare. Secondary infection may result in abscess formation.

Prevention and treatment

All small marine cuts and injuries should be treated actively with antiseptic solutions. Definite lesions are best treated with local antibiotic powder or ointment and systemic penicillin or tetracycline.

Pseudomonas

Pseudomonas aeruginosa has been previously mentioned as a common cause of otitis media. Although this organism is not known to survive in saltwater, it may persist for a long time in freshwater. It thrives in a warm, moist environment.

Pseudomonas folliculitis

This condition, also known as 'splash rash',[17] is usually seen after exposure to whirlpools, spas and hot tubs,[18] but has also been described after swimming pool exposure.[19,20]

A papulopustular eruption develops some 8–48 hours after exposure to a recirculating water environment. The lesions may be pruritic or even tender and usually occur on axillae, groin, trunk and buttocks. Fever, malaise, dizziness, headache, sore eyes and throat and regional lymphadenopathy may develop.

Pseudomonas urethritis and keratoconjunctivitis[21] has also been reported following immersion in whirlpools or spas.

Diving-suit dermatitis

Several similar cases have been reported in divers,[22,23] the distribution of the rash coinciding with the area covered by the suit. The wetsuit, which provides optimal conditions for *P. aeruginosa*, was shown to be the source of the organism in at least one diver.

These conditions are usually self-limiting in one to two weeks, but pustules or abscesses may recur for several months. Cultures of skin lesions and environmental source reveal the same serotype of *P. aeruginosa.*

Prevention and treatment

Prevention requires improved disinfectant and filtering systems and regular water testing. Treatment is usually symptomatic, but, in severe cases, systemic antibiotics may be required.

Tinea

Tinea pedis dermatophytosis, caused by a *Trichophyton* species or *Epidermophyton floccosum,* is common in swimmers and divers due to such factors as moist environment, bare feet on wet decks and floors of communal showers, etc. It is usually of nuisance value only, but secondary infection may lead to lymphangitis and lymphadenitis.

Treatment with topical imidazole or terbinafine is effective. Occasionally, systemic griseofulvin or terbinafine is required.

Pityriasis versicolor

This skin condition due to *Malassezia furfur* will become obvious in swimmers and divers because of their exposure to sunlight when the small patchy areas fail to tan evenly.

Treatment is usually sought for cosmetic reasons only, and include topical fungicides, imidizoles creams and lotions. Rarely systemic terbinafine may be required. Although this eradicates the infection, the areas of discoloration may persist for many months.

Mycobacterium marinum

This acid-fast atypical tuberculous bacillus *Mycobacterium marinum* (previously also called *M. balnei)* is the cause of cutaneous granulomata that have been called 'swimming pool granuloma'[24,25] or 'swimmer's elbow'.[26] *M. marinum* is also known to occur in seawater. This organism may gain entry to the skin via an abrasion from a swimming pool wall or ship's hull. Granulomata usually develop on fingers, hands, elbows or knees. There is one report of the infection following the bite of a dolphin,[27]and it has also been noted in tropical fish tank enthusiasts[28] (fish-fancier's finger). Immunosuppressed individuals appear to be particularly susceptible.

Clinical features

The granulomata usually develop over bony prominences, i.e. sites of abrasion. The onset is noted three to four weeks after the predisposing injury (eight

weeks in the case of the dolphin bite). They may develop as discrete red papules covered with fine scales, and may be large enough to be fluctuant; aspiration may then reveal thick pus. The papules or cysts may become indurated or even ulcerate. Spontaneous resolution can occur in one to two years, but cases have persisted for 45 years. There is no evidence of systemic involvement. Synovitis was a common presenting feature in one series.[29]

Diagnosis

This is by punch skin biopsy of the ulcer and demonstration of the organism either by direct staining (acid-fast) or culture on Lowenstein–Jensen media at 30–33°C. Growth takes up to three weeks. Skin testing with tuberculin is positive in 85 per cent of cases.

Treatment

Drug therapy should be guided by response to in-vitro sensitivity tests. Infections have responded to treatment with trimethoprim-sulphamethoxazole, tetracyclines (especially minocycline) and the tuberculosis drugs, rifampicin and ethambutol. The drugs have to be continued for at least four weeks, with some authorities suggesting 18 months. The use of local warmth may be beneficial, and it has been suggested that the infection is confined to the skin because of the inability to grow at body core temperature. Surgery, either alone or in combination with antibiotics has also been reported to be useful. Antibiotics are essential if the infection has extended to deep tissues such as tendon sheaths, joints or muscle.

Prevention

These measures include adequate chlorination of swimming pools, smooth tiles and, in the case of divers, protective gloves and clothing.

Schistosome dermatitis

This condition, also known as 'swimmer's itch', bather's itch and marine dermatitis, is likely to be contracted near the surface of the water. It is due to penetration of the skin by cercaria of non-human schistosomes. The cercaria is the larval developmental stage in the life cycle of the fluke. The organism is found in certain fresh or brackish lakes and swamps where a suitable ecological niche exists.

The life cycle of the organism involves shore-loving birds and various gastropod molluscs such as snails of the seashore. The adult fluke is a parasite and lives in the mesenteric vessels of vertebrates, including water birds. The fluke lays its eggs, which pass into the bird's gut, and faeces, and are deposited in the lakes that the birds inhabit. They hatch in the water, becoming young miracidiae, which spend most of their life in the body of water snails. In the water snails, the miracidiae develop into free-swimming larvae, termed cercariae, which are capable of penetrating the skin of wading birds – or humans. If the cercariae enter the birds, then the life cycle starts anew. If it penetrates the skin of humans, it dies, due to an active foreign body reaction that it induces in human tissue. The condition may then become manifest.

Clinical features

Humans become involved accidentally in this cycle. The cercariae are able to penetrate human skin, but not blood vessels. This penetration causes a prickling sensation while in the water or soon after leaving it, and is thought to be a mechanical irritation.

The pruritus subsides to return a day later with increasing intensity in association with an erythematous papular eruption. There may be some associated inflammatory swelling. The rash is present for about one week and then fades, leaving a brown pigmentation that persists for some time. The degree of reaction varies greatly, previous exposure causing hypersensitivity to develop in many subjects. The foreign protein of the dead cercariae causes antibody production. This antigen–antibody reaction occurs at the site of each dead larva and is responsible for the itchy papules. It is important to note that lesions will only occur on parts of the body that have been exposed to water.

Prevention and treatment

Persons at risk should wear protective clothing and vigorously rub any exposed areas immediately after leaving the water. Dimethylphthalate is of value as a repellent. Treatment is symptomatic (e.g. calamine lotion).

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30

General infections

CHRIS LOWRY

SWIMMING AND DIVING IN POLLUTED WATERS[1–3]

Ingestion of polluted drinking water has long been known to pose the risk of hepatitis, typhoid, cholera, dysentery and other gastrointestinal diseases.[4] With the greater worldwide awareness of increasing environmental pollution has come awareness that the swimmer or diver may also be exposed to the risk of these and other infections. Although where monitoring is in place, beaches worldwide are periodically closed due to sewage pollution, the safety of many unmonitored locations is uncertain.

In coastal waterways close to large population centres, the water may be heavily contaminated with a wide range of organisms, but especially faecal coliforms and streptococci, *Salmonella* spp., and entero- and rotaviruses.[5,6] Bodies of water that do not 'flush' well, such as enclosed bays and harbours, are more likely to contain significant numbers of pathogens.

'Coliform counts' are used as indication of water quality, and the US Environmental Protection Agency (EPA) recommends that safe recreational water should contain not more than 200 faecal coliforms per 100 ml. High coliform counts correlate with the presence of pathogens such as *Salmonella*, *Shigella* and *Aeromonas*, but not so well with pathogenic viruses.

High faecal streptococcal counts in marine recreational water have been associated with various illnesses, and the US EPA sets a limit of 35 colony-forming units/100 ml for safe exposure.

Viruses (especially enteric) can enter the marine environment in massive quantities in urban sewage disposal, and are not all destroyed by normal sewage treatment processes. Some can survive for long periods of time in seawater and may be associated with enteric disease among swimmers (especially children). Enteric and respiratory viruses shed directly into water from bathers may be a source of infection. One report suggested an association between water quality and gastroenteritis in swimmers at several US beaches. Polio vaccine viruses, adenoviruses and Coxsackie virus have been recovered from sewage-affected coastal waters.

Professional divers may have to work in severely polluted water, and wet suits and normal masks provide little protection to the skin or gastrointestinal tract. Vaccination against hepatitis A should be considered. Special procedures and equipment such as full hoods and occlusive dry suits may be required in such situations (see Chapter 66).

The pathogenic organisms isolated from polluted waters are listed in Table 30.1. The role of these organisms in the production of disease in swimmers and divers requires further epidemiological studies.

Table 30.1 *Potentially pathogenic microorganisms isolated from polluted waters*

Gram-negative bacteria	**Gram-positive bacteria**
Coliforms	*Staphylococcus*
Escherichia coli	*Streptococcus*
Klebsiella	*Bacillus*
Enterobacter	
Citrobacter	**Viruses**
Edwardsiella	Enteroviruses
Legionella pneumophilia	Reovirus
Campylobacter	Adenovirus
Serratia	Hepatitis A virus
Proteus	
	Protozoa
Oxidase-positive group	*Entamoeba*
Aeromonas	*Giardia*
Plesiomonas	*Acanthamoeba*
Pseudomonas	*Naegleria*
Chromobacter	*Hartmanella*
Yersinia	
Vibrio cholerae	
V. parahaemolyticus	
V. alginolyticus	
Group F 'vibrio-like' organisms	
Lactose (+) 'vibrio-like' organisms	
Anaerobes	
Bacteroides	
Clostridium	
Fusobacterium	
Eubacterium	

This list does not imply that all of these microorganisms are present in any given body of water.

VIBRIO VULNIFICUS

Vibrio vulnificus (see Chapter 29) thrives in warm coastal seawater. It is a particularly virulent organism, and severe infections that may proceed to septicaemia have resulted from marine trauma,[7] (especially from shellfish or crabs), as well as from aspiration of seawater, gastroenteritis and aquatic sexual intercourse.

Clinical Manifestations

The patient may present with fever, chills, headache and myalgia. Bullous skin lesions may develop, as may necrotizing fasciitis and myositis. Septicaemia with hypotension and shock has a high mortality. Disseminated intravascular coagulation and respiratory distress syndrome have also been described with *V. vulnificus* infection. Patients succumbing to this infection may have underlying disease, such as diabetes, liver disease (especially haemochromatosis), renal failure, or may be immunosuppressed.

Treatment[8]

Wounds should be thoroughly cleaned as soon as possible, and the penetrating or more serious injuries surgically explored and debrided under antibiotic cover. Appropriate antibiotics to commence while awaiting sensitivities include tetracycline and gentamicin, or an extended spectrum cephalosporin such as cefotaxime. Intensive care support is often required for multisystem failure. The infection is not contagious.

> Vibrio *infections should be suspected in any patient presenting with fever and shock, in association with wound infections, pneumonia or gastroenteritis when there is a recent history of immersion in salt water.*

SCHISTOSOMIASIS

Schistosomiasis (bilharziasis), a disease caused by trematodes (flukes), is one of the most important causes of morbidity in the tropics. Humans are the definitive host to pathogenic schistosomes. The life cycle is similar to that described under schistosome dermatitis in Chapter 29, but with humans replacing birds. *Schistosoma japonica* was described in the Philippines in the Second World War in US servicemen, and it is also endemic in the Yangtse river area of China. *Schistosoma haematobium* and *Schistosoma mansoni* occur in Africa, the Middle East and South America. The infection is derived from contact (bathing or swimming) with infected water. Several

weeks following skin penetration an allergic reaction develops (Katayama syndrome), which may be severe with fever, cough, headache, abdominal pain, splenomegaly and patchy pneumonia. These symptoms then subside for several months. The parasite can damage the liver gut, lung (multiple small abscesses) and bladder. Severe central nervous system involvement may also develop with *S. japonicum.*

If immersion occurs in potentially infected water, then showering and vigorous towelling might prevent penetration of the skin by the cercariae.

Schistosomiasis has been reported in three scuba divers from a dam in Transvaal;[9] however, schistosome infestations are not contracted from saltwater or correctly chlorinated swimming pools.

LEPTOSPIROSIS

Human infection with *leptospirae* usually result from ingestion of water or food contaminated with these spirochaetes. Less often, the organism gains entry via a break in the skin or mucous membrane, and so fishery workers and recreational water users are most at risk. A number of epidemics from swimming in fresh water have been reported, with rats, swine, dogs and cattle being the principal sources of infection. Certain species, e.g. *Leptospira icterohaemorrhagiae, L. canicola, L. pomona* or *L. australis,* may predominate in a given geographical area.

Clinical features

The incubation period is usually one to two weeks, and is followed by a sudden onset of fever, malaise, nausea, myalgia, conjunctival injection and headache. This period, during which the spirochaetes are present in the blood, may be followed by organ involvement, particularly of the liver (jaundice), kidney (renal failure) or lungs. There may also be meningism (benign aseptic meningitis), nausea and vomiting. The disease may persist for up to three weeks. Severe leptospirosis with profound jaundice (Weil's disease) develops in up to 10 per cent of cases.

Treatment

The role of antibiotics in mild febrile forms of the disease is controversial, but doxycline and ampicillin have been advocated. More severe cases respond to penicillin G, ampicillin and amoxicillin intravenously, and erythromycin. Complete resolution of the disease process is usual.

Prevention

This is by avoidance of exposure to potentially contaminated water, rodent and other host control, and possible vaccination of dogs to reduce contamination. Doxycline (200 mg weekly) may be indicated for short-term protection where risk of exposure is high.

PHARYNGOCONJUNCTIVAL FEVER[10]

This is an acute illness caused by several types of **adenovirus.** It has an incubation period of five to nine days and is characterized by fever, malaise, pharyngitis, cervical lymphadenopathy, cough, conjunctivitis and sometimes diarrhoea. Outbreaks of pharyngoconjunctival fever have been reported in swimmers. Similar viruses are probably often involved in swimming pool conjunctivitis epidemics. No serious morbidity and no deaths have been reported.

PRIMARY AMOEBIC MENINGOENCEPHALITIS

The free-living amoeba of the genus *Naegleria fowleri* causes this severe, often fatal, illness. This protozoan organism is found in freshwater and prefers warmer temperatures, being found more frequently in lakes, hot springs, swimming pools or industrial thermal waters. The organism cannot survive long in a marine environment.

Naegleria species gain entry to the central nervous system via the mucosa of the nasopharynx and the cribriform plate. The amoeba then multiplies in the meninges and olfactory bulbs and eventually elsewhere in the brain. Cases have been reported from Australia, Belgium, Czechoslovakia, the UK, New Zealand and the USA. It is likely that many others have been diagnosed as acute pyogenic meningitis with failure to demonstrate the infecting organism.

Clinical features

The incubation period ranges from about three days to two weeks. The presentation is similar to that of acute pyogenic meningitis, the patient being in good health before the sudden onset of frontal headache, mild fever and lethargy, sometimes associated with sore throat and rhinitis. The headache and pyrexia progress over three days with vomiting, neck rigidity, disorientation and coma. The cerebrospinal fluid (CSF) changes are those of bacterial meningitis, usually under increased pressure. The coma deepens, and death in cardiorespiratory failure supervenes on the fifth or sixth day of the illness. A high index of suspicion and absence of the expected pathogenic bacteria in the purulent CSF raises the diagnosis.[11] This is confirmed by observing the motile amoebae in a plain wet mount of fresh CSF.

Pathological findings at post-mortem examination reveal a slightly softened, moderately swollen brain, covered by hyperaemic meninges. There is a purulent exudate over the sulci and in the basal subarachnoid cisterns. Small, local haemorrhages are seen in the superficial cortex, but the olfactory bulbs are markedly reddened and in some cases haemorrhagic and necrotic. On microscopic examination, there is a mild fibrinopurulent meningeal reaction and amoebae may be seen in the exudate. The degree of encephalitis varies from slight amoebic invasion and inflammation to complete purulent, haemorrhagic destruction. The nasal mucosa is severely ulcerated and the olfactory nerves are inflamed and necrotic. There is no evidence of amoebic invasion elsewhere in the body.

Treatment

There is a very high mortality in this condition. Amphotericin B is the drug of choice, in high dosage intravenously and small doses intraventricularly, and has resulted in some cures. Concurrent miconazole and rifampicin have also been used.

Prevention

Pollution of waterways by sewage and domestic wastewater must be controlled if this disease is to be prevented. Swimming and diving should be avoided in potentially contaminated water, especially if the water or environmental temperature is high.

KEY WEST SCUBA DIVERS' DISEASE[12]

This syndrome was described in classes at the US Navy's scuba training establishment at Key West, Florida. It was reported to occur 36 hours after first use of one particular type of scuba regulator, and it was noted in several students at each new course.

Clinical features

The disease is characterized by the onset of malaise, anorexia, myalgia, fever, often greater than 38°C, headache and substernal tightness. One death has been attributed to this condition. Apart from these features, physical examination, chest X-ray, urine examination, throat and blood cultures for bacteria are negative. Viral studies are also non-contributory. The illness subsides spontaneously in 72 hours. Moreover, continued use of the same regulator does not result in recurrence of the illness unless there is an intervening period without diving.

A multitude of organisms, including mainly *Pseudomonas* and *Fusarium,* has been found on the low-pressure diaphragm and interior of the corrugated air hoses of the twin-hose regulators. Decontamination of these parts appears to prevent the illness.

Salt water aspiration syndrome is due to aspiration of small amounts of saltwater during diving, mimics an acute respiratory infection (see Chapter 24), and bears some resemblance to Key West scuba divers' disease. It is not thought to be a contagious disease, but is included here because of its importance in the differential diagnosis of infectious illness. It usually resolves without antibiotic therapy.

NEAR-DROWNING AND PULMONARY INFECTION

During recovery from aspiration of both fresh and saltwater, some patients develop severe infections of

the lungs. This is one of the many possible reasons for the delayed hypoxia seen in such patients. Post-mortem examinations performed on those who die more than 12 hours after near-drowning often show evidence of bronchopneumonia or multiple abscesses. However, the changes are often those of an irritant pneumonitis rather than infection (see also Chapters 21 and 22).

Although in many cases it may be difficult to determine whether the infection was acquired in hospital, there have been numerous reports where organisms causing pneumonia have also been isolated from the drowning site. Organisms isolated in near-drowning victims include: *Aeromonas, Pseudomonas aeruginosa, Pseudomonas putrefaciens, Klebsiella pneumoniae, Burkholderia pseudomallei* (melioidosis), *Chromobacterium violaceum, Vibrio vulnificus, Streptococcus pneumoniae, Legionella* and the funguses *Aspergillus* and *Pseudallescheria boydii*.[13,14] Some of these primarily pulmonary infections have also developed septicaemia and metastatic abscesses.

Rapid development of *Aeromonas* pneumonia and sepsis with high positive blood cultures has been reported after immersion in healthy young men.[15]

Prophylactic antibiotics have not shown to be useful, and may, in fact, encourage infection with resistant organisms. Microscopy and culture of sputum or tracheal aspirate should be performed regularly and appropriate antibiotic therapy instituted.

SPECIAL ENVIRONMENTS

Enclosed environments

The human–microbe–environment relationship is both subtle and complex, in that a change in any one of the elements may have substantial effects on the others.

An increasing problem in the closed environments in undersea habitats, submarines and hyperbaric facilities is contamination by microorganisms and the flora in such situations can be very rich (see also Chapters 67, 68, 69 and 70). Cross-infection of divers through the use of common equipment, diving practices (such as buddy breathing) and habitation in small enclosures aggravate these problems. The concentration of pathogenic organisms may lead to an increased rate of skin, respiratory and systemic infections.

The commonest organism isolated from skin infection in saturation divers is *P. aeruginosa*, but this is seldom found in routine skin infections. The factors encouraging infection are not entirely clear, but the hyperbaric atmosphere may play a role apart from the humidity and temperature. Evidence has been presented to suggest that the organism is not necessarily introduced into the system by the infected diver, and may persist in the freshwater system for several months.[16]

Whenever a group of people live together in close proximity for days or weeks, they undergo an initial period of illnesses. After recovering from these infections, they are then immune to subsequent infections, as long as they live in isolation with their antigenic peers. They are, however, extremely susceptible to infections from exogenous sources, or when the period of isolation ends and they re-establish contact with outside personnel. Such examples are seen with the Polaris submarine crews, people living in Antarctica, etc. This is readily explained by the limited sources of infection.

Other interesting changes in saturation complexes may be found because of the effects of pressure, temperature, gas changes and relative humidity on the survival, selectivity and transport mechanisms of microorganisms. It was found that humidities in the region of 50 per cent were the most detrimental to air-borne bacteria; however, this may not be applicable to marine organisms transported mechanically from the marine environment, which may assume a predominant role in the air flora of submersible habitats. In less-controlled saturation systems, with high humidity, there may be a greatly increased propensity to infection.

The specific changes from Gram-positive organisms to the Gram-negative *Pseudomonas* and *Proteus* spp. was referred to previously.

Hyperbaric effects

Oxygen under high pressure is of value in treating certain infections, and may be life- or limb-saving in cases of clostridial gas gangrene. It has also been of

value in the treatment of chronic osteomyelitis and other infections.

Oxygen under pressure may have a multitude of effects on the human–microbe–environment interaction. Pulmonary oxygen toxicity is thought to impair bacterial defence mechanisms and thus cause increased susceptibility to infections, particularly of the respiratory tract. Oxygen and *Pseudomonas* infection appear to be additive in damaging the lung to produce the adult respiratory distress syndrome.

Enhancement of viral infection by hyperbaric oxygen has been demonstrated in cell cultures, by an acceleration of virus maturation and production of abnormally high yields or faster host cell destruction. These effects do not depend on continual exposure during the infectious cycle, and therefore may be applicable to all types of hyperbaric exposures. The change appears to be produced by changes in the membrane of the cell and lysosomes.

Experimental studies on rats exposed to 100 per cent oxygen at 3 ATA for 15 minutes before being infected with Coxsackie virus, demonstrate an inhibition of the interferon activity, a greater virus proliferation and less leakage of lysosome enzymes, together with increased host mortality.

Hyperbaric changes in the physiology of the host have been inferred from tissue cultures. There appear to be alterations in cell permeability, and in the metabolism of amino acids and ribonucleic acid precursors. The divers' steroid levels are increased, both in saturation and brief diving excursions, increasing susceptibility both to bacterial and viral infections.

There does seem (in some cases) to be a tendency to impede the host's reaction, together with increased susceptibility to infection. Organisms may change, both in incidence and activity, when associated with a hyperbaric environment. Deep diving or a hyperbaric helium environment can increase the resistance to penicillin of *Staphylococcus aureus* and to gentamicin and rifampicin by *Escherichia coli* and *Salmonella typhimurium*.[17] Hyperbaria also seems to increase the effects of some antibiotics, e.g. in increasing permeability of tetracyclines into CSF.

The above information remains patchy, selective and incomplete. The area is a productive field for future developments – especially if the research encompasses in-vivo experiments and does not reflect merely the microbe–environment duet.

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31

Trauma from marine creatures

CARL EDMONDS

INTRODUCTION

For animals not equipped with venoms and poison, defence against predators relies on either camouflage or aggression. Traumatic damage to the predator may be achieved by biting, bumping, spearing, electrical charges or corrosive materials.

Many marine animals avoid humans, but this attitude can be altered by familiarity with humans, usually divers, who do not confront the animals aggressively. Increased proximity promotes the possibility of injury to both species.

Feeding activity is also related to aggression. Many divers make the error of feeding these animals, and this results in them associating humans with food. Often the animal will attack divers to acquire this food. Divers have evoked feeding attacks from animals that would normally not have behaved in this manner. Examples include some small reef sharks, groupers, dolphins and many fish.

SHARKS

General

The majority of the 250 species of sharks are marine inhabitants, but many enter estuaries, and some travel far up rivers, while a few are freshwater species. Most live in the relatively shallow waters off the continents or around islands and inhabit the temperate or tropical zones.

Some, such as the great white shark, are pelagic species, and although poikilothermic, have adapted to colder ocean temperatures. In other species, the activity of the shark may be more related to the environmental temperature. Shark attacks tend to be more frequent when the water temperature reaches 20°C or more, because of these reasons and because of the increased frequency of humans bathing in warm water.

Even though Australia is renowned as one of the most dangerous areas in the world for shark attack,

Figure 31.1 *Shark: this photograph was taken seconds before leaving the water. (Courtesy P. Lane.)*

there is an average of only one fatality per year, from millions of bathers at risk. Rescue and first-aid groups sometimes exaggerate the risk of shark attack.

Shark attack remains a genuine, but unlikely danger to seafaring people. Although rare, the attack is often terrifying in intensity, and the degree of mutilation produced has a strong emotive effect.

Data on shark attacks

There has been little factual research on shark attacks, perhaps due to the understandable difficulty of experimenting with these animals. Basically, the information is obtained from two sources: statistical analysis of shark attack files; and detailed data collection from specific case histories. Neither source is comprehensive, and neither is adequate by itself. The detailed case histories demonstrate the range of possibilities, whereas the statistical information indicates probable behaviour. The difficulty in obtaining accurate details of any specific shark attack is understandable when one considers the suddenness of the accident and the emotional involvement of the participants.

The problem in assessing the statistical information is that much of the data was insufficient and unreliable. Application of the statistics to an open-water situation is not warranted. Nevertheless, a great deal of interesting information is available from the statistics.

Shark attacks are more frequent when there are more people at risk (i.e. during warm weather, on weekends and holidays). Attacks are more likely at the sharks' natural feeding times, at dusk, near deep channels, in turbid waters in estuaries, and where animal products are dumped.

Anatomy and physiology

Of the 350 species of sharks, about 30 have been implicated in attacks on man. Sharks allegedly have a low intelligence, but this has not interfered with their ability to survive far longer than man in the evolutionary time scale. They are well equipped to locate prey and others of their own species, conduct seasonal migrations, and identify specific localities. They react to multiple stimuli, with the sense of smell being a principal means of locating prey. They can detect some substances in minute quantities, e.g. blood in less than one part per million. Although their visual acuity for differentiating form or colour may not be selective, their ability to discriminate movements and minor contrast variations in low light conditions is extremely efficient.

They have an ability to detect low-frequency vibrations (e.g. the flapping of an injured fish). Their hearing is especially sensitive to low-frequency sounds and they have an extraordinary faculty for directional localization of this sound. Their taste is not well developed, but preferences for some foods have been suggested. The lateral line is a multi-sensory system commencing at the head and passing along the shark's body. This system receives a variety of information, including vibrations of low frequency, temperature, salinity, pressure and minute electrical fields such as those produced by other fish or humans in the vicinity.

The feeding response is related more to the presence of specific stimuli than to the nutritional requirements of the animal. The presence of chemical stimuli, such as that released from freshly killed animals, can cause considerable attraction to sharks and may result in the so-called 'feeding frenzy'. Sharks may swim together in an orderly and smooth manner, but when abnormal vibrations are set up (e.g. by one of the animals being shot or hooked), then the abnormal activity of that animal may trigger feeding responses in the others, and this may intensify into a feeding frenzy.

Attack Patterns

There are several different types of attack, and these may be identified by the behaviour of the animals and the subsequent nature of the injury. Four types represent different degrees of a feeding attack, and the others represent a territorial protection.

1 Sharks in a feeding pattern tend to circle the victim, gradually increasing their swimming speed. As the circles begin to tighten, the sharks may commence a criss-cross pattern (i.e. going across the circle). At this stage, they may produce injury by **contact**, when they bump or brush the prey. The shark's abrasive skin can cause extensive injuries, and it is thought that the information obtained by the animal at this time may influence the progression of the feeding pattern.
2 The **shark bite** is usually performed with the animal in a horizontal or slightly upward direction, with the head swung backward and the upper teeth projecting forward. This results in a great increase in the mouth size and a display of the razor sharp teeth. The attacking force involved is considerable and is often enough to eject the victim well clear of the water. The bite force may be up to seven tons per square inch. Once the animal has a grip on the prey, if the feeding pattern continues, the mouthful will usually be torn out sideways or the area totally severed.
3 If other sharks are in the vicinity, they may reflexly respond to the stimuli created by the attack and commence a feeding frenzy. In this instance the sharks are likely to attack both the original prey and the predator or any other moving object. During the feeding frenzy, cannibalism has been observed, and the subsequent carnage can be extensive.
4 A variant, often used by the Great White when attacking a larger animal which could possibly inflict damage on the shark, is the 'bite and spit' behaviour. This behaviour is seen not only against seals and sea lions, but also against other prey which may have a similar silhouette on the surface – such as surfboard riders, surface swimmers, etc. The shark may make one sudden dash, take one bite and then release the prey, which then bleeds to death. Once the prey has stopped moving, the shark can then continue the feeding pattern in relative safety.
5 The fifth type of attack is termed **agonistic**, and is that of an animal having its territorial rights infringed on by an intruder – either a swimmer or a diver. This is quite unlike the feeding pattern. The shark tends to swim in a far more awkward manner, exaggerating a lateral motion with his head, arching the spine, and angling its pectoral fins downward. In this position, it appears to be more rigid and awkward in its movements than the feeding animal. It has been compared, both in appearance and motivation, to a cornered animal, adopting a defensive and snapping position. If the intruder diver vacates the area, confrontation will be avoided and an attack prevented.
6 When a diver jumps from a boat onto a shark (who may be following and scavenging refuse from the craft), the animal may reflexly snap at the intruder, in defence.

Clinical Features

The lesions produced by shark bite are readily identifiable (see Fig. 31.2 and Plates 7 and 8). The rim of the bite will have a crescent shape, delineating the animal's jaw line. There will be separate incisions from each tooth along the line, with occasional fragments of the teeth in the wound. Identification of the shark species is possible from these teeth. There may be crushing injuries to the tissues, and variable amounts of the victim may be torn away. Haemorrhage is usually severe, in excess of that noted in motor vehicle accidents – probably due to ragged laceration of vessels preventing control by vasoconstriction.

A great variety of damage is noted in different attacks. In some cases merely the brushing and abrasive lesions of the skin may be present. In others, the teeth marks may be evident, encircling either the victim's body or even his neck – when the shark has had an appreciable amount of the victim within his mouth, but has still not proceeded with the bite. In most cases, there is a single bite, but occasionally several attacks and bites are made on the one victim. When the latter occurs, adjacent people are rarely attacked. Amputations and extensive body wounds are common. In victims not killed immediately, the major problem is massive haemorrhage and shock.

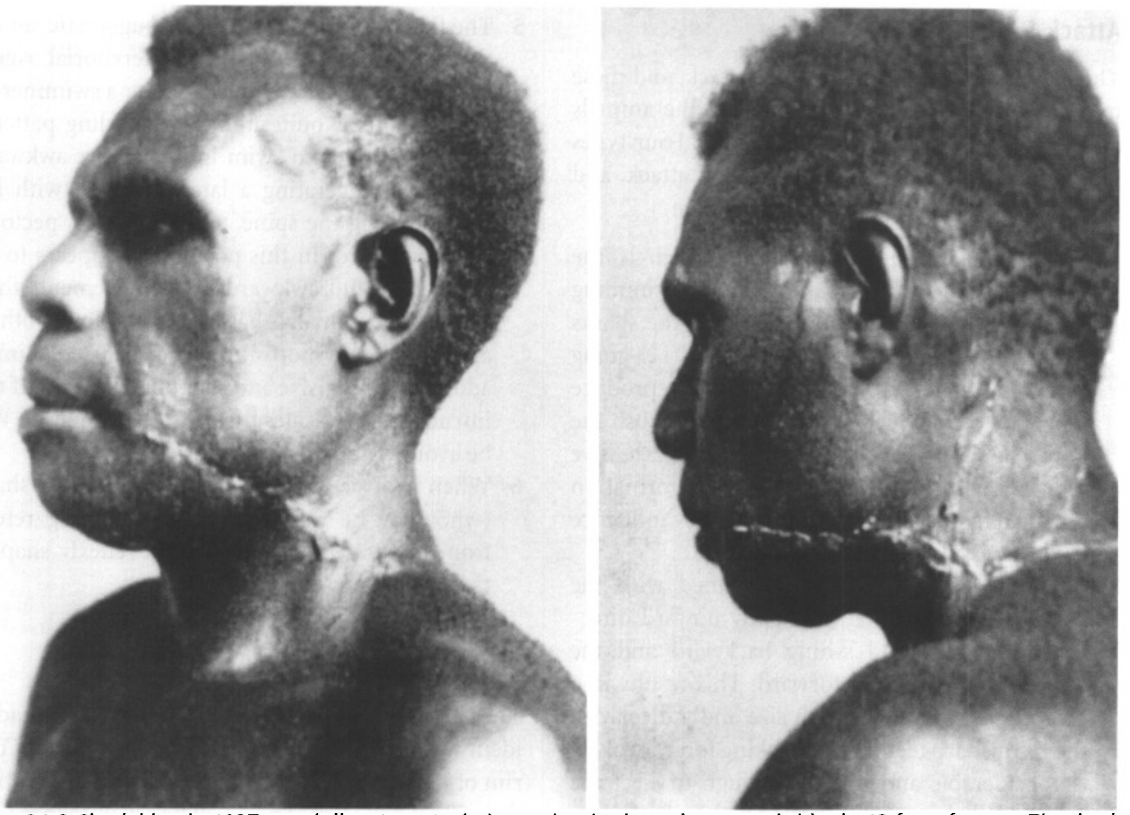

Figure 31.2 *Shark bite: in 1937, pearl diver Iona Asai observed a shark coming towards him in 12 feet of water. The shark enclosed Asai's head within its jaws, and he claims to have felt for and squeezed the shark's eyes – causing it to release him. Asai was pulled on board the boat, bleeding profusely, before he lost consciousness. Two rows of teeth cuts were sutured in hospital (requiring about 200 stitches) and 3 weeks later the tooth of a tiger shark was removed from a neck abscess. Iona had been injured in a shark attack off Cairns 19 years earlier, and is therefore one of the few men to have survived two separate shark attacks. (Courtesy of V.M. Coppleson)*

Treatment

The most valuable first aid is to protect the patient from further attack and reduce or stop the haemorrhage. The rescuer is rarely injured, because the shark tends to concentrate on the original victim. Once the patient is removed from the shark and prevented from drowning, attention should be paid to the prevention of further blood loss. This should be achieved by any means available – pressure on the site of bleeding or proximal to this site, tourniquets or pressure bandages, tying of blood vessels, etc. Any material available should be used. The mortality rate is such that there need be no apprehension regarding either the use of tourniquets or contamination of wounds.

The patient should be lying down with the legs elevated, and covered only lightly with clothing or a towel. They should be reassured as much as possible. Medical treatment is best commenced before transfer of the patient to the hospital, and infusion of blood, plasma or other intravenous replacement fluid should be given top priority until the shock has been adequately controlled. This can be ascertained by the clinical state of the patient, their pulse rate, blood pressure, central venous pressure, etc. The use of morphine intravenously is likely to give considerable benefit, despite its mild respiratory depressant effect.

Recording and assessing vital signs becomes an integral part of the management; they should be

monitored throughout the transfer of the patient to the hospital.

At all stages, first-aid resuscitation takes priority over the need for hospitalization. Transport to hospital should be performed in a gentle and orderly manner as excess activity aggravates the shock state in these patients. Case reports abound with statements that the victims died in transit; they could more accurately state that the victims died because they were transported.

After stabilizing the clinical state, the patient is transferred by the least traumatic means available. The surgical procedures are not significantly different from those used for a motor vehicle accident case. Under anaesthesia, the areas are swabbed, and bacteriological culture and sensitivities obtained. X-rays should be performed, both to show bone damage and also to detect foreign bodies. Surgical excision of the obviously necrotic material is required. The surgical techniques should otherwise be of a conservative type, especially if the blood supply is intact. Tendon suture should not be attempted, unless the wound is very clean. Skin grafting is performed early, whenever possible, to preserve nerves, tendons, vessels, joints and muscles.

Broad-spectrum antibiotics are required, and it should be remembered that the bacteriological contamination is sometimes extensive, both with marine and terrestrial organisms. *Clostridium tetanus* and *Clostridium welchii* have both been isolated from shark wounds, although the contamination almost certainly occurred after the injury.

Prevention

Prevention of shark attack depends on the marine locality. The following procedures will be relevant in different situations.

Heavily populated beaches

The most effective method of reducing the incidence of shark attack is by enclosures or meshing. Total bay enclosures are effective in sheltered areas, if consistent surveillance is carried out to ensure the integrity of the net. Areas exposed to adverse weather or surf are best protected by meshing.

Meshing involves the occasional use of a heavy-gauge net, which is submerged from buoys to the seaward side of the breaking waves for 24 hours and then retrieved. The shark tends to swim into the net, which wraps around it and interferes with its gill function. As the shark is unable to retreat, it will struggle and attempt to push itself forward through the mesh. This results in the shark being further immobilized and leads to death by suffocation. Most of the sharks are dead by the time the mesh is retrieved, and the others are killed at the time of the retrieval.

The shark population becomes decimated when this technique is used. The experience is similar on the relatively heavily shark-populated beaches of both Australia and South Africa. Shark attacks could still occur despite meshing, but, the results are nevertheless dramatic. Not only does the shark population decrease but shark sightings also decrease, and the shark attacks are virtually eliminated. The population develops more confidence in the safety of their surfing area, and increased tourism will often compensate for the cost of the shark meshing.

Alternative techniques include bubble curtains, sound and ultrasonic waves, and electric repellents, but these do not have the same record as enclosure or meshing techniques. Many methods of repelling sharks will, given different conditions and different-sized animals, result in an alerting or an attraction response in the very animals they are meant to deter. This is certainly the case with some electrical and explosive devices.

Survival situation

The crashing of an aircraft or the noises associated with a ship sinking may attract sharks. Thus, the survivors of such accidents may become the victim of shark attack. The most effective way to prevent this is to use life rafts and have the survivors move into them as quickly as possible. As an alternative, the Johnson Shark Screen is very effective. This is a bag of thin, tough plastic with a collar consisting of three inflatable rings. The survivor partially inflates one of the rings, by mouth, and then climbes into the bag. He fills the bag with water by dipping the edge below the water so that as it fills it appears to any shark as a large, solid-looking black object. The other rings can be inflated at leisure. The bag retains fluids and excreta which may otherwise stimulate shark attack, and also attenuates the bioelectric and galvanic fields produced by the survivors.

'Shark Chaser' consists of a black dye and copper acetate; the dye was meant to confuse the shark's visual localization, while the copper acetate was thought to resemble a deterrent chemical that is produced in decaying shark tissues. Unfortunately, this system does not work, but another chemical, produced by the Peacock or Moses sole, is currently under investigation.

Swimmers

Swimmers are advised not to urinate in the water or to swim with abrasions or bleeding wounds. They are also advised to move gently and not thrash around on the surface. They should stay with a group, or at least with a buddy. This is cynically claimed to reduce the chance of shark attack by 50 per cent, but, in fact, it probably reduces it far more. Swimmers are also advised not to swim in water with low visibility, near drop-offs or deep channels, or during late afternoon or night, when sharks tend to be involved in feeding.

Divers

The incidence of shark attacks on scuba divers appears to be increasing, and now comprises one-third of all such attacks. Wet suits offer no protection and may even increase the likelihood, despite popular hopes to the contrary. Divers are advised in the same way as swimmers, but with added precautions. Underwater explosives tend to attract sharks. Shark attacks are more likely with increased depth and can be provoked by feeding, playing with or killing sharks. If one is diving in shark-infested waters, the use of a shark billy (a stout rod with a metal spike) can be effective in pushing the animals away. Powerheads, carbon dioxide darts and the drogue dart (this has a small parachute attached which disrupts the shark's orientation and swimming efficiency) may be appropriate under certain situations. Electric prods and intermittent currents are effective

Divers are also advised not to catch fish or abalone or tether them near their body, as they may attract sharks. If sharks are encountered, it is best to descend to the sea bed or to the protection of rocks, a cliff face, or some other obstacle to interfere with the normal feeding attack pattern described above. If the diver recognizes an agonistic attack pattern from the shark, he should vacate the area, swimming backwards.

Chain mail (stainless steel) suits discourage sharks from continuing an attack, but incur buoyancy problems for divers and swimmers that outweigh the risks of shark attack.

Experiments are being conducted on the use of Kevlar incorporated into wet suits, as a shark-bite-resistant material.

It is sometimes claimed that women should not dive or swim while menstruating, but there is no evidence to support the belief that decomposing blood will attract sharks. In fact, the experimental and statistical evidence indicates the opposite effect.

CROCODILES, ALLIGATORS AND CAIMAN

Crocodiles cause as many human fatalities as sharks, in the areas where both are found. This was not always so, but while there is a diminishing number of shark attacks due to meshing, there is an increased crocodile attack frequency because they have been protected and grow larger. There is also an increase of tourism into remote crocodile territories.

Crocodiles become more aggressive during the breeding times, mainly as the young are hatched from eggs and protected by both parents.

The species considered as man-eaters are the Australian saltwater crocodile and the Nile crocodile which grow to 8 metres; and the American crocodile and alligator, which grow to 3.5 metres. South American caimans are of the same family as alligators and grow up to 5.5 metres, but usually much less. Even the Indian mugger crocodile may attack

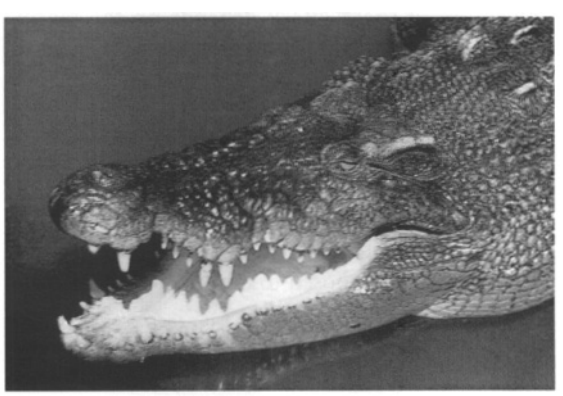

Figure 31.3 *Crocodile.*

humans if provoked while nesting. All crocodilians are carnivorous, and range in size from 1 to 10 metres long, though only the larger specimens are potentially dangerous to humans. The largest crocodiles grow up to a tonne (1000 kg) in weight, and are often believed to compete with fishermen, damaging nets, and may prey on both domestic animals and humans.

Alligators are slower moving than crocodiles, and generally less dangerous to man. Crocodiles have narrower snouts than alligators, and the fourth tooth in each side of the lower jaw is usually visible when the mouth is closed.

Saltwater crocodiles may also be found in freshwater, having swum inland from an estuary or travelled many kilometres overland. Freshwater crocodiles are also found in lakes and rivers that have no connection with the sea, and in some countries they may be both large and dangerous. If humans or other animals intrude into the territory the crocodile sometimes give a warning by exhaling loudly or even growling at the intruder.

As reptiles, they have very complex brains and are intelligent enough to stalk a human, strong enough to destroy a water buffalo, and gentle enough to release its own young from the eggs – with its teeth. It even carries the newly hatched babies in its massive jaws.

Crocodiles tear their food from the carcass, twisting and turning in the water to achieve this. They then swallow it whole. Once an attack pattern has begun, the crocodile will attack repeatedly until the prey is captured, and it may follow the victim from the water if necessary. If the animal captures large prey, it may hide the carcass underwater, entangled in submerged trees or under ledges, until it is ready to resume feeding.

The animal often lies along the banks of rivers, with only the nostrils protruding above water to breathe. The prey, especially land animals such as horse, cattle, giraffe, rhinoceros, kangaroo and wallaby, come to the river bank to drink and may be grabbed in the jaws of the crocodile, and twisted off its feet. This movement will sometimes break the neck of the victim. Once the prey is in the water, it is more vulnerable to panic and drowning. Although this is the classical attack pattern, crocodiles can move quickly on land and in water, and recent attacks in Australia have included attacks with the victim free-swimming in deep water, on dry land and in a canoe.

On land, the attacks are more common at night when the animal stalks for food. They can move surprisingly fast – faster than most humans – issue a hissing sound, and sometimes attack by sweeping the victim with their powerful tail.

The first aid, medical treatment and investigations are the same as for shark attack. Occasionally, a tooth fragment will be found by X-ray of the wound.

BITING FISH

Little space has been allocated to the other marine animals that are said to bite because it is difficult to find more than a few cases of a verified fatal bite on a human.

Barracuda

Barracuda have occasionally be known to attack. They are sometimes attracted by bright-coloured objects and lights, if diving at night.

Grouper

There have been reported cases of a grouper attacking a human, and occasioned deaths. As a general rule, these heavyweight bulldogs of the sea have built up a reputation for friendliness more than

Figure 31.4 *Barracuda. (Courtesy of G. Lewbell).*

forcefulness. They are, however, feared in some areas, e.g. the pearl-diving beds between New Guinea and Australia. They can act aggressively if speared.

Eel

Many eel attacks have been reported. **Moray** eels can grow up to 3 metres in length and up to 30 cm in diameter. They rarely attack without provocation, and the attack is usually precipitated by an intrusion into their domain, or after they have been injured or caught on lines or spear guns. Divers who feed the eels inadvertently encourage them to be more adventurous and less fearful, thus increasing the attack potential – for food. Certainly, once they do attack, they are likely to be difficult to dislodge and may even resume the attack after being dislodged. The wound is likely to be badly lacerated and heavily infected. The medical and surgical treatments conform to those normally used with other damaged and infected tissues.

Piranha

The piranha (*Serrasalmus* spp.), have probably the worst reputation of all small fish, and although they are carnivorous, and can be very ferocious and vicious, they certainly do not deserve their very bad press. They are abundant in the rivers of South America and most of the 20 or more species are harmless. They may grow to 45 cm, but some are only a couple of centimetres long. The black piranha and its relatives are a cause for concern as in sufficient numbers they are believed to be able to remove the flesh from large-sized animals within a few minutes. Although attacks on humans are rare, there are some well-documented cases from the Amazon.

Figure 31.5 *Moray eel. (Photo by P. Lane).*

Miscellaneous biting fish

Taylor or **Blue Fish** (*Pomatomous saltatrix*) also work in large schools and occasionally have caused injury to bathers. They commonly travel in large numbers along the east coast of the United States and have been known to drive swimmers from the water, in Miami, Florida. Fingers and toes can be badly injured, and occasionally even amputated.

Spanish mackerel (*Scomberomorus maculatus*) in shoals have also occasionally attacked and injured swimmers. Other fish which are not commonly known to bite humans, may do under certain circumstances. The very beautiful and famous **bat fish** (*Platax* spp.) around Heron Island in the Great Barrier Reef are unfortunately fed by divers, and because of this and their large numbers, unpleasant nips may be inflicted on divers' exposed skin.

Most **puffer fish** (tetrodotoxic fish) are also able to inflict injury, as their jaws are designed to crunch crustaceans. They are slow-swimming fish and therefore can be approached by divers, with occasional unfortunate results. One such fish, called Thomas the Terrible Toadfish, inflicted multiple injuries to waders, at Shute Harbour, on the Great Barrier Reef. He attacked so many bather's feet that he also became known as Thomas the Terrible Toe Fish.

BITING MAMMALS

Killer whale

This is the largest of the dolphin family, and it acquired its name from its tendency to travel in packs, feeding on other marine creatures such as

seals, larger whales, etc. Killer whales shepherd prey into deep water and cause death by battering and drowning. At least one human fatality is recorded.

Pinnipeds

Even the usually placid **walrus**, if sufficiently provoked by hunters, can retaliate with ferocity. The **Californian sea lion** and its Australian cousins have inflicted minor attacks on humans, usually after intrusion by the latter in the breeding harems. The more infamous **leopard seal** is a solitary animal in the Antarctic and cooler southern waters. It will stalk penguins, seals and other warm-blooded prey, even humans. It swims above them until finally the prey has to surface to breath, and then strikes. If a leopard seal is sighted, diving should be suspended. If underwater at the time of sighting, divers should not surface in midwater, but follow the sea bed to the shore, before departing.

Figure 31.6 *Leopard seal.*

ELECTRIC RAYS

These are slow and very ineffective swimmers, usually lying submerged in the mud or sand at shallow depths, in temperate climates. They can produce an electric discharge between 8 and 220 volts, and this is passed between the electrically negative underside of the ray and the positive topside. They are easily identified by thick electric organs on each side of the spine. The discharge is automatic when the fish is touched, or even during an approach. There is then a latent period before the fish regains its full electrical potential. The electric discharge may well cause much amusement to other divers, but can sometimes be disabling.

OCTOPUS AND SQUID

Octopoda, of the class cephalophoda, have been the source of much folklore. Although the vast majority of these animals will avoid human contact as much as possible, many of them are able to inflict significant bites, puncture wounds from the beak (mouth) or the modified claws on the tips of the tentacles of the giant squid. Other injuries have been produced by the sepia or ink, from the venom associated with salivary glands of the octopus and the animal's ability to adhere to and hold a swimmer or diver underwater, with the suction pads from the tentacles.

Although there has been a great deal of fancy in some of the descriptions of octopus attacks, there have nevertheless been a dozen or so well-recorded episodes in which injury has occurred to humans. Attacks known to this author, including one which resulted in the death of a diver, were consequential to a purposeful intrusion into the animal's territory, usually with a spear gun.

SWORDFISH AND SAWFISH

This group include both the swordfish, sailfish, marlin, garfish and sawfish. There are documented cases of both death and injury from these fish. The trauma is caused by the saw or sword, which is an

extension of the jaw, and even the smaller species – which can skim above the surface of the water – have caused many injuries to fishermen. The garfish can sometimes reach 2 metres above sea level, and are possibly attracted by lights used by fishermen at night. The penetrating injuries from the relatively small spear-shaped jaws, have resulted in both penetration of body cavities and injuries to the face and head.

Both sawfish and the swordfish achieve their damage by a use of the large appendage, either in an attacking or a defensive role. Occasional attacks have occurred under water, in which case death is usually due to blood loss. Others have occurred after the fish has been caught and brought on board. This is especially recorded in fishermen trawling for marlin and swordfish.

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32

Venomous marine animals

CARL EDMONDS

SEA SNAKE

The sea snake is an efficient swimmer and is equipped with a paddle-shaped tail for this reason. It can submerge for 2 hours. They are inquisitive, and sometimes aggressive, especially if handled or trodden on. They are attracted by fast moving objects, e.g. divers being towed by a boat, and under these circumstances they can congregate and become troublesome. They are also caught in trawling nets, especially in the tropics. Land snakes may also take to the water, sometimes causing difficulty with identification. No land snake has the flattened tail.

Sea snake venom is two- to ten-fold more toxic than that of the cobra, but they tend to deliver less of it, and only about one-fourth of those bitten by sea snakes ever show signs of envenomation. It appears that there is some reluctance to inject venom even when they do bite. Nevertheless, the venom able to be injected by one fresh adult sea snake of certain species is enough to kill three men. In most species the apparatus for delivering the venom is poorly developed even though the mouth can open widely, whereas in a few others the mouth is small and the snake has difficulty in obtaining a wide enough bite to pierce the clothing or any other protective layer that the diver may wear.

Sea snake venom appears to block neuromuscular transmission by acting on the post-synaptic membrane, and may affect the motor nerve terminals. It blocks the effects of acetylcholine. Autopsy findings include patchy and selective necrosis of skeletal muscles, and tubular damage in the kidneys if the illness lasts longer than 48 hours.

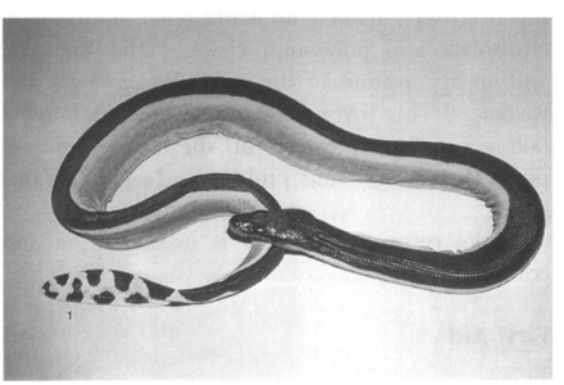

Figure 32.1 *Sea snake: note the flattened, paddle-shaped tail.*

Clinical Features

An initial puncture at the time of biting is usually noted. Fang and teeth marks may vary from one to 20, but usually there are four and teeth may remain in the wound. After a latent period without symptoms, from 10 minutes to several hours, generalized features will develop in approximately one-fourth of the cases.

Mild symptoms include a psychological reaction such as euphoria, anxiety, restlessness, etc. The tongue may feel thick, and thirst, dry throat, nausea and vomiting occasionally develop. Generalized stiffness and aching may then supervene. If weakness does progress into paralysis, then it is usually either of the ascending Guillain–Barré type, with the legs being involved an hour or so before the trunk, then the arms and neck. The other manifestation of paralysis is one which extends centrally from the area of the bite, e.g. from the bite on the hand to the forearm, arm, other arm, body and legs. Usually the proximal muscle groups are the most affected and trismus and ptosis are characteristic. Muscular twitching, writhing and spasms may be seen, and the patient may develop difficulty with speech and swallowing as the paralysis extends to the bulbar areas. Facial and ocular palsies then develop. Respiratory distress, due to involvement of the diaphragm, may result in dyspnoea, cyanosis and finally death in a small number of the cases affected. Cardiac failure, convulsions and coma may be seen terminally.

Myoglobinuria may develop, but when this is seen, one must consider the other possible effects of myonecrosis – namely an acute renal failure, with electrolyte and potassium changes and uraemia, and an aggravation of the muscular paralysis and weakness. This myonecrotic syndrome with renal failure usually supervenes on the other muscular paralysis and may thus prolong and aggravate this state.

When recovery occurs it is usually rapid and complete.

First Aid

The current treatment is the use of pressure bandage (applying a wide strap using about the same tension that one would for a sprained ankle, first wrapped around the area of the bite, and then proximal and distal to it) together with immobilization. This is thought to reduce both venous and lymphatic drainage from the area. Under these conditions it is possible to transport the patient slowly to medical facilities.

Reassurance is needed and exertion is to be avoided. The limb is immobilized, and so should the patient. If possible, the snake (dead, to avoid further problems) should be retained for identification because although it may be harmless, the treatment certainly is not. In the event of respiratory paralysis, artificial or mouth-to-mouth respiration may be required.

Medical Treatment

Once the patient is transported to adequate medical facilities, and the clinicians have reviewed the therapy indicated, the pressure bandage may be removed. Once this happens the envenomation will have its effect on the patient, and preferably the treatment, including the antivenom regime, must be instituted.

Apart from the above first-aid procedures, full cardiopulmonary resuscitation may be required. Fluid and electrolyte balance must be corrected, and acute renal failure is usually obvious from the oliguria, raised serum creatinine and electrolyte changes. A high serum potassium is particularly dangerous, and treatment by haemodialysis is then required. This may result in improvement of the muscular paralysis and the general clinical condition. The acute renal tubular necrosis and the myonecrosis are considered temporary, if life can be maintained.

Treatment may be necessary for the cardiovascular shock and convulsions, and often assisted ventilation is required.

Sea snake antivenom from CSL (Australia) can be used cautiously in serious cases. It contains 1000 units per ampoule, and care must be taken to administer it strictly in accordance with the directions in the brochure. The antivenom can be dangerous to patients who are allergic, and emergency preparations for anaphylactic shock are required, with most authorities advising anti-allergy pre-treatment. The sea snake antivenom is composed of two

antivenoms, each of which has a specific action. Unfortunately, although it does counter the most common sea snake venoms, there are others that are not covered. If it is necessary to use land snake antivenom, then probably the Tiger snake type is to be preferred. Polyvalent land snake antivenom can also be used, although the value has yet to be determined.

Patients with sea snake bite should be hospitalized for 24 hours, because of the delay in symptoms developing. Sedatives may be required, and it is reasonable to administer diazepam as required. This will assist in sedating the patient, without interfering significantly with respiration. Preparation for treatment of anaphylactic shock should be available.

Prevention

This is usually achieved by not handling sea snakes. It is suggested that the feet be shuffled when walking along a muddy sea bed, and that protective clothing is worn when underwater. The wet suit is usually sufficient, and if collecting sea snakes it is wise to use a special sea snake tong.

FISH STINGS

Many fish have spines and a venom apparatus, usually for protection and occasionally for incapacitating their prey. Spines may be concealed, only becoming obvious when in use (e.g. stonefish), or highlighted as an apparent warning to predators (e.g. butterfly cod or firefish; see Plate 9).

Some fish envenomations have resulted in death, especially the stonefish and stingray, and these will be described separately. Others, such as the infamous scorpion and firefish (family Scorpaenidea), catfish (family Plotsidae and Ariidae), stargazers (family Uranoscopidae,) have also been responsible for occasional deaths in humans. As a general rule, fish that have been damaged – such as those in fishing nets – cause less problems clinically, probably because some of the envenomation system may have been previously used. Those wounds that bleed profusely are also less likely to have intense symptoms. Some spines are inexplicably not associated with venom sacs.

Other fish may produce injury by the knife-like spines which may or may not be related to envenomation, e.g. Old Wife (family Enoplosidae), Surgeon and Unicorn fish (family Acanthuridae), Ratfish (family Chimaeridae).

General

Identification of the species of fish responsible is not always possible, but fortunately there is not a great variety in the symptomatology.

Clinical Features

If venom is injected, the first symptom is usually local pain which increases in intensity over the next few minutes. It may become excruciating, but usually lessens after a few hours ('with the change of the tide' – an old mariner's attempt at reassurance). The puncture wound is anaesthetized, and the surrounding area hypersensitive. Pain and tenderness in the regional lymph glands may extend even more centrally.

Locally, the appearance is that of one or more puncture wounds, with an inflamed and sometimes cyanotic zone around this. The surrounding area becomes pale and swollen, with pitting oedema.

Generalized symptoms are sometimes severe, and the patient is often very distressed by the degree of pain, which is disproportionate to the clinical signs. This distress can merge into a delirious state. Malaise, nausea, vomiting and sweating may be associated with mild temperature elevation and leucocytosis. Respiratory distress may develop in severe cases. Occasionally, a cardiovascular shock state may supervene and cause death.

First Aid

The patient should be laid down and reassured, and the affected area should be rested in an elevated position. Arrangements can then be made to immerse the wound in hot (up to 45°C) water for 30–90 minutes – or until the pain no longer recurs. The wound should be washed and cleaned. As well as the wound, some normal skin must also be immersed in the hot water to ensure that scalding is not induced. The injured skin may well be hypoaesthetic, and not give adequate warning of this danger.

Fishermen often make a small incision across the wound and parallel to the long axis of the limb, to encourage mild bleeding and relieve pain if other methods are not available.

FIRST AID TREATMENT OF FISH STINGS (venom injected by spine):

- *Lay patient down with affected limb elevated.*
- *Wash surface venom away and gently remove spine or integument if present.*
- *Immerse in hot water (up to 45°C) to reduce severity of pain, but include normal skin to avoid scalding.*
- *Injection of local anaesthetic without adrenaline into and around the wound.*
- *Clean the wound. Apply local antiseptic or antibiotic.*

Medical Treatment

This includes first aid, as outlined above. Local anaesthetic, e.g. 5–10 mg lignocaine 2 per cent, without adrenaline (epinephrine), if injected through the puncture wound, will provide considerable relief. This may need to be repeated every 30–60 minutes. Local or regional anaesthetic blocks may also be of value.

Symptomatic treatment may be needed for generalized symptoms of cardiogenic shock or respiratory depression. Systemic analgesics or narcotics are rarely needed, but they may be of value in severe cases.

Exploration, debridement and cleansing of the wound, with removal of any broken spines or their integument, is best followed by the application of local antibiotic such as neomycin or bacitracin. Tetanus prophylaxis may be indicated if there is necrotic tissue, or if the wound has been contaminated.

The basic physiological signs (temperature, pulse and respiration, blood pressure, central venous pressure, urine output, etc.) serum electrolytes, electroencephalogram, electrocardiogram are monitored if indicated. For serious or extensive lesions, a soft tissue X-ray or ultrasound may be needed to demonstrate foreign body or bone injury. Broad-spectrum antibiotics (e.g. doxycycline) may be needed. Symptomatic treatment is given for the clinical features present.

Stingrays

General

This vertebrate lies in the sand, and the unwary victim may tread on its dorsal surface or dive over it (see Plate 10). The stingray swings its tail upward and forward, driving the spine into the limb or body of the victim. An integument over the serrated spine is ruptured, and venom escapes and passes along grooves into the perforated wound. Extraction of the spine results in a laceration due to the serrations and retropointed barbs, and may leave spine or sheath within the wound.

The venom is a protein (molecular weight >100 000) that is heat-labile, water-soluble and has an intravenous LD_{50} of 28.0 mg/kg body weight. Low concentrations cause electrocardiographic effects of increased PR intervals associated with bradycardia. A first-degree atrioventricular (AV) block may occur with mild hypotension. Larger doses produce vasoconstriction, second- and third-degree AV block and signs of cardiac ischaemia. Most cardiac changes are reversible within 24 hours. Some degree of respiratory depression is noted with greater amounts of venom; this is probably secondary to the neurotoxic effect of the venom on the medullary centres. Convulsions may also occur.

Fishermen who handle these fish from nets are less seriously affected as the integumentary sheath is probably already damaged.

Clinical Features

Local

Pain is usually immediate and is the predominant symptom, increasing over 1–2 hours and easing after 6–10 hours, but it may persist for some days. Aggravation of pain within days may be due to secondary infection. The pain may be constant, pulsating or stabbing. Bleeding may be profuse, and may relieve the pain. A mucoid secretion may follow.

Integument from the spine may be visible in the wound, which may gape and extend for a few centimeters in length. The area is swollen and pale, with a bluish rim, centimetres in width, spreading around the wound after an hour or two. Local necrosis, ulceration and/or secondary infection are common, and if unchecked may cause incapacity for many months. Osteomyelitis in the underlying bone has been reported.

General

The following manifestations have been noted: anorexia, nausea, vomiting, diarrhoea, frequent micturition and salivation. There is extension of pain to the area of lymphatic drainage. Muscular cramp, tremor and tonic paralysis may occur in the affected limb, or be more generalized. Syncope, palpitations, hypotension, cardiac irregularities (conduction abnormalities, blocks), and ischaemia are possible. Respiratory depression may occur, with difficulty in breathing, cough and pain on inspiration. Other features include nocturnal pyrexia with copious sweating, nervousness, confusion or delirium.

The symptoms may last from hours (the venom effect) to many months (a foreign body and/or infection).

Fatalities are possible, especially if the spine perforates the pericardial, peritoneal or pleural cavities. Death may be due to the envenomation (cardiac arrhythmias), trauma, haemorrhage, or delayed tissue necrosis and infection.

Treatment

See under *First aid* and *Medical treatment* for fish stings (p. 338).

The special problems of penetration of body cavities, immediate and delayed haemorrhage must be monitored with stingray injuries as delayed problems frequently occur.

Prevention

Divers are advised to shuffle their feet when walking in the water, which gives the ray time to remove itself – which it cannot do with a foot on the dorsum! Although wearing rubber boots decreases the severity of the sting, the spine will penetrate most protective material. Care is needed when handling fishing nets.

Stonefish

General

This fish grows to about 30 cm in length and lies dormant in shallow waters, buried in sand, mud, coral or rocks, and is practically indistinguishable from the surroundings (see Plate 11). The 13 dorsal spines, each capable of piercing a sandshoe, are covered by loose skin or integument. When pressure is applied over them, two venom glands discharge along ducts on each spine, into the penetrating wound. The fish may live for many hours out of the water.

The venom is an unstable protein, with a pH of 6.0 and a molecular weight of 150 000 Da. It produces an intense vasoconstriction, and therefore tends to localize itself. It is destroyed by heat, alkalis and acids. The toxin is a myotoxin which acts on skeletal, involuntary and cardiac muscles, blocking conduction in these tissues. This results in a muscular paralysis, respiratory depression, peripheral vasodilation, shock and cardiac arrest. It is also capable of producing cardiac dysrhythmias.

Each spine has 5–10 mg of venom associated with it, and is said to be neutralized by 1 ml of antivenom from the Australian Commonwealth Serum Laboratories. Occasionally, a stonefish spine may have no venom associated with it. It is thought that the venom is regenerated very slowly, if at all.

Clinical features

Whether the local or generalized symptoms predominate seems to depend on many factors, such as the geographical locality, number of spines involved, protective covering, previous sting, first aid treatment, etc.

Local

Immediate pain is noted, and this will increase in severity over the ensuing 10 minutes or more. The pain, which is excruciating in severity, may be sufficient in some to cause unconsciousness, and thus drowning. Ischaemia of the area is followed by cyanosis which is probably due to local circulatory stasis. The area becomes swollen and oedematous, often hot, with numbness in the centre and extreme tenderness around the periphery. The oedema and

swelling may become quite gross, extending up the limb. Paralysis of the adjacent muscles is said to immobilize the limb, as may pain.

The pain is likely to spread proximally to the regional lymph glands, e.g. the axilla or groin, and both the pain and the other signs of inflammation may last for many days. Necrosis and ulceration can persist for many months.

General

Signs of mild cardiovascular collapse are not uncommon. Pallor, sweating, hypotension and syncope on standing may be present. Respiratory failure may be due to haemorrhagic pulmonary oedema, depression of the respiratory centre, cardiac failure and/or paralysis of the respiratory musculature. Bradycardia, cardiac dysrhythmias and arrest are also possible.

Malaise, exhaustion, fever and shivering may progress to delirium, incoordination, generalized paralysis, convulsions and death. Convalescence may take many months, and may be characterized by periods of malaise and nausea.

Treatment

See under *First aid* and *Medical Treatment* for fish stings (p. 338).

Stonefish antivenom may be administered, with 1 ml neutralizing 10 mg of venom (i.e. the venom from one spine). Initially, 2 ml of antivenom is given intramuscularly, although in severe cases the intravenous route can be used. Further doses can be given if required, but it should never be given to people with horse serum allergy. It should be stored between 0-5°C but not frozen, and protected from light. It should be used immediately on opening.

Systemic analgesics and narcotics are seldom indicated or useful, although intravenous narcotics are sometimes used. Tetanus prophylaxis is sometimes recommended. Systemic antibiotics may be used as secondary infection is likely. Debridement should be considered if there is significant tissue damage and necrosis, or if foreign material might be left in the wound.

Appropriate resuscitation techniques may have to be applied. These include external cardiac massage and defibrillation and endotracheal intubation with controlled respiration. Monitoring procedures may need to include records of clinical state (pulse, respiration), blood pressure, central venous pressure, pulse oximetry, electrocardiogram, lung function tests, arterial gases and pH. Clinical complications of bulbar paralysis should be treated as they arise.

Prevention

Thick-soled shoes should be worn when in danger areas, and particular care must be taken when on coral reefs and while entering or leaving boats. A stonefish sting is said to confer some degree of immunity for future episodes.

COELENTERATES

General, including Portuguese man-o'-war

This phylum of 9000 species contains jellyfish, sea anemones, fire coral and stinging hydroids. It constitutes one of the lowest orders of the animal kingdom, and has members which are grossly dissimilar in both general appearance and mobility.

The common factor among the coelenterates is the development of nematocysts or stinging capsules. These capsules are of two types, one which adheres to the animal's prey, either by a sticky mucus or by a coiled spring (see Fig. 32.2), and the other which acts as a needle, penetrating the prey and discharging venom into it. This needle may be as long as 0.5 mm. The triggering mechanism which is responsible for the discharge of the nematocyst is

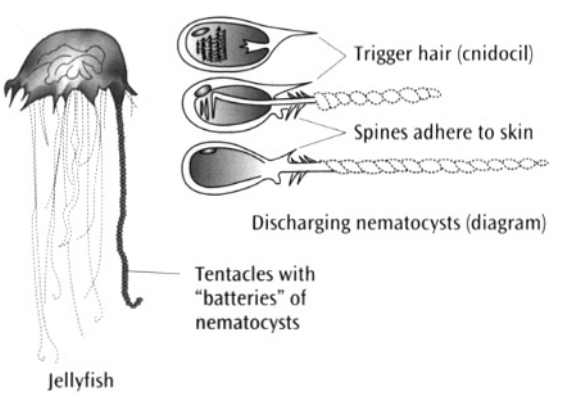

Figure 32.2 *Diagrammatic representation of discharging nematocyst.*

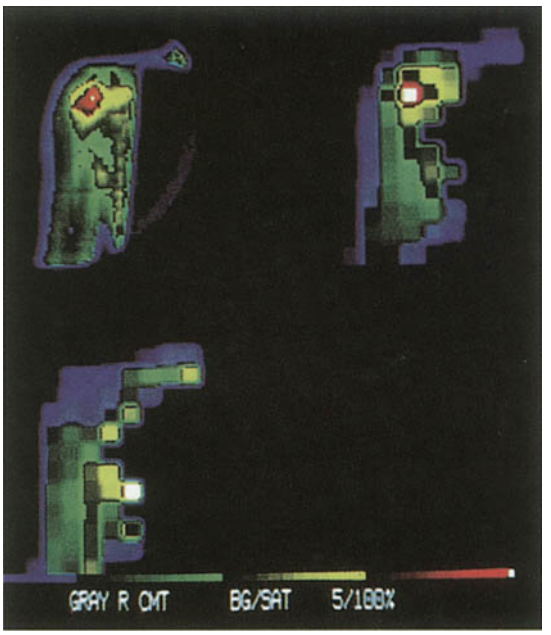

Plate 6 *A further MDP scan taken after Figure 14.10(a), using a technique called 'parametric scanning' or 'functional imaging'. The images are colour-coded on a scale which uses blue for 'cold' and white for 'hot'. The image at the top left is merely a way of quantifying the sort of qualitative image shown in Figure 14.10(a) and is the appearance 2–3 hours after the injection of MDP, when there is less background 'glare' from MDP and has not been taken up by bone, and which has been washed out of other tissues and excreted. The picture at the top right is a scan taken 30 minutes after giving the MDP, and is not a measure of osteoblastic activity. In this case, there is a larger 'hot' area compared to the first image. The third image in the bottom left corner is an expression of the accretion rate constant for technetium measured on a minute-by-minute basis over the first 30 minutes. The accretion rate constant is directly proportional to perfusion. and it is clear that an area in the shoulder joint in this image is 'cold' and therefore not well perfused. This in turn means an area of infarction which, in time, can be expected to become X-ray-positive. This particular lesion was X-rayed again 3 weeks after the scan, and the tomogram in particular showed an affected joint. Pain and restriction of movement were noted at this stage. (Courtesy of Dr Ramsay Pearson.)*

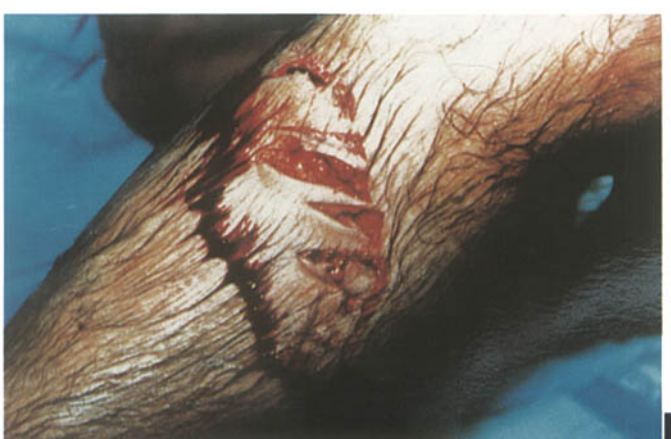

Plate 7 *Triangular teeth marks of shark bite. (Courtesy Dr. C. Barnes.)*

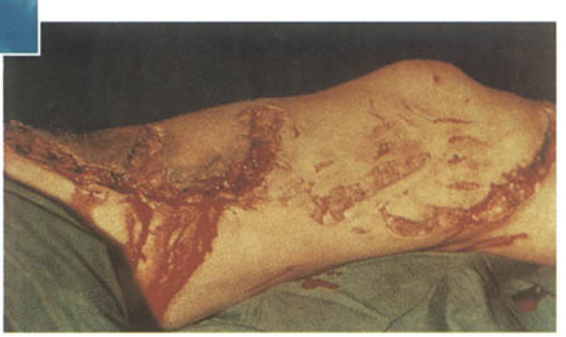

Plate 8 *Curved and concentric lacerations of shark bite – often with teeth left in the wound (see Chapter 31). (Photograph courtesy Dr G.D. Campbell.)*

Plate 9 *Firefish (Courtesy R. Chesher)*

Plate 10 *Stingray (Courtesy R. Chesher)*

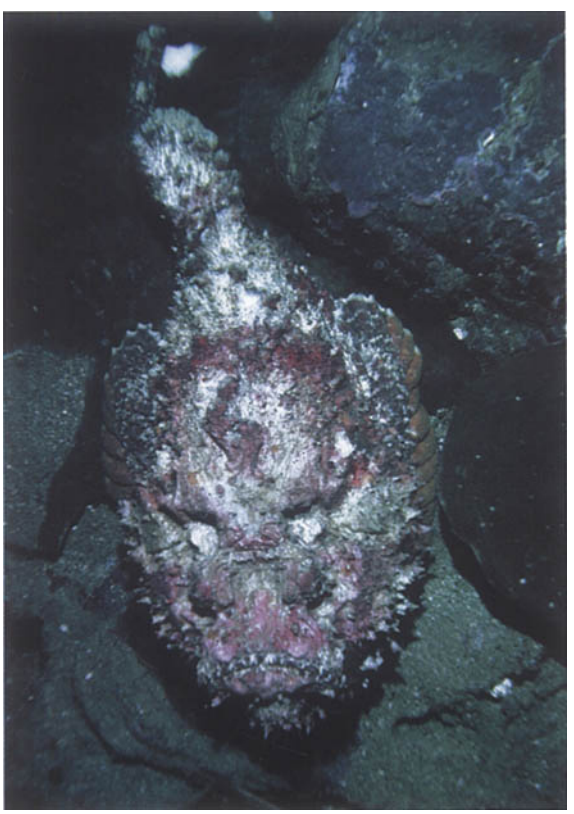

Plate 11 *Stonefish (Courtesy R. Chesher)*

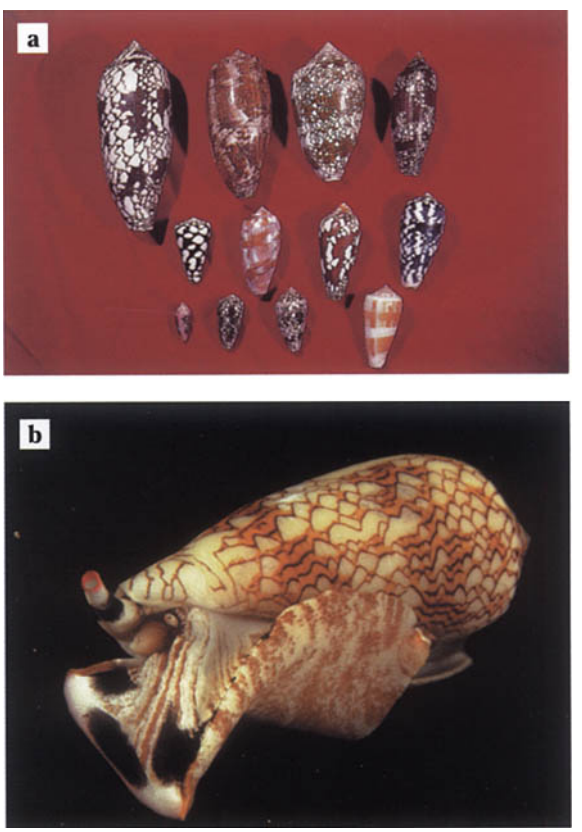

Plate 12 *Poisonous cone shells. (a) Displayed shells; (b) the shells* in situ *(Courtesy R. Chesher)*

thought to be initiated by many factors, e.g. trauma or absorption of water into the nematocyst capsule, causing it to swell.

The function of the nematocysts is to incapacitate and retain prey, which is then used as food by the coelenterate. The nematocysts of different types of coelenterate may be identifiable, and therefore of value in the differential diagnosis of marine stings. In some centres, serological and immunological tests may also be available to identify different species.

There may be a characteristic pattern of nematocyst stings, depending on their aggregation on the tentacle of the coelenterate, and on the morphology of the tentacles. Thus the **Portuguese man-o'war** (see Fig. 32.3) usually produces a single long strap with small blisters along it, whereas the **Jimble** has two to four short red lines, with the **chironex** having multiple long red lines, often with the tentacle adherent due to a thick sticky substance, when the patient is first seen. **Stinging hydroids** and **fire coral**, being non-mobile, sting only when touched by the diver.

Clinical factors may vary from a mild itch locally to severe systemic reactions. The local symptoms vary from a prickly or stinging sensation developing immediately on contact, to a burning or throbbing pain. The intensity increases over 10 minutes or so, and the erythema may develop papules, vesicles or even pustules and necrotic ulcers in severe cases. The pain may spread centrally, with lymphadenopathy, and may be associated with abdominal pain and chest pain.

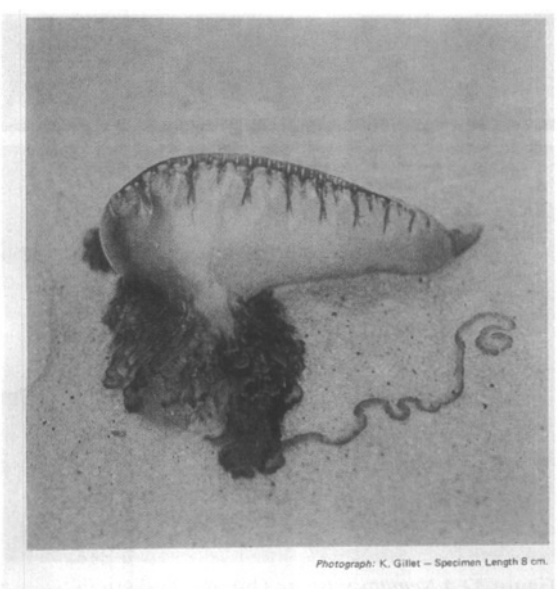

Figure 32.3 *Portuguese man-o'-war (*Physalia physalis*). (Photo by K. Gillett.)*

Generalized symptoms include fever, increased secretions, gastrointestinal disorders, cardiovascular failure, respiratory distress and signs of a toxic-confusional state.

The intensity of both local and generalized manifestations of coelenterate stinging may vary according to: the species involved (chironex is often lethal, whereas the blubber jellyfish can often be handled with impunity); the extent of the area involved; the body weight of the subject, being more severe in children than adults; the thickness of the skin in contact; and individual idiosyncrasies such as allergic reactions, pre-existing cardiorespiratory disease, etc. As the most dangerous coelenterate is the chironex, this is dealt with in detail. The Portuguese man-o'-war or bluebottle (physalia) sting is one of the most common problems encountered by bathers.

The first-aid treatments have been based on the use of empirical and fashionable treatments, such as vinegar, dehydrating agents (alcohols) or denaturing agents (acetic acid, ammonia, papaine in meat tenderizers) to reduce the discharge of further nematocysts. Some of these have aggravated the symptoms. Current treatments include the use of anti-burn preparations, cold packs, and the application of local anaesthetic ointments and steroids to reduce symptoms of pain or itch, respectively. Recently, a 50 per cent water: 50 per cent baking soda mixture has been proposed (see Burnett, 2000), as has the heat treatment applied to fish stings (see Taylor, 2000).

The reason that most coelenterates do not injure humans is that the nematocyst is incapable of penetrating the depth of skin necessary to cause symptoms. Variations of this mode of injury occur in four instances.

1 **Direct entry.** Coral cuts are often experienced in the tropics, and in these cases there is a laceration of the skin, which allows nematocysts to discharge directly into the wound tissues. This is

supplemented by a foreign body reaction to the nematocysts, coral pieces and organisms. Pacific islanders spread coelenterates over spears.

2 **Nudibranchs**, especially the **Glaucus**, ingest certain coelenterates and utilize these nematocysts for their own Machiavellian purposes. This means that humans who come in contact with these nudibranchs may then sustain an injury having a distribution which corresponds to the area of contact with the nudibranch.

3 **Ingestion** and **inhalation.** Allergy and anaphylaxis may develop from contact, inhalation or ingestion. Some are poisonous to eat.

4 **Irukandji.** Some jellyfish produce a minimal sting, but inject a toxin that causes severe generalized muscular spasms, especially affecting the large muscle masses of the spine and abdomen, up to 2 hours later. Because of the latent period the relationship may not be realized and diagnostic problems arise. Although the Irukandji have only been described in the tropical and subtropical parts of the Southern Indo-Pacific, many jellyfish stings may have a muscular spasm/pain component to their injury – as may many other marine animals.

Chironex (box jellyfish, sea wasp)

General

These cubomedusae are restricted to the warm waters of the Indo-Pacific region, with fatalities being more numerous in the waters off Northern Australia. Related species may be found in other tropical areas, such as the equatorial waters and the Middle East.

Chironex is said to be the most venomous marine animal known. It is especially dangerous to children and patients with cardiorespiratory disorders (asthmatics and coronary artery disease). Its box-shaped body can measure 20 cm along each side, and has up to 15 tentacles each measuring up to 3 metres in length on each of its four pedalia. The animal is usually small at the beginning of the 'hot' season, and increases in size and toxicity as it matures during the season. It is especially common after bad weather and on cloudy days, when it moves into more shallow water. It is almost invisible in its natural habitat, being pale blue and transparent. It tends to avoid noise, e.g. speed boats, and turbulence, e.g. the surf – but this should not be relied upon. It is actively mobile, but often drifts with the wind and tide when near the surface.

The severity of the sting increases with the size of the animal, the extent of contact with the victim, and the delicacy of the victim's skin. Deaths have occurred with as little contact as 6–7 metres of tentacle. Adjacent swimmers may also be affected to a variable degree. The tentacles tend to adhere with a sticky, jelly-like substance. They can usually be removed by bystanders, due to the protection afforded by the thick skin on the palmar aspect of the hands. However, this protection is not always

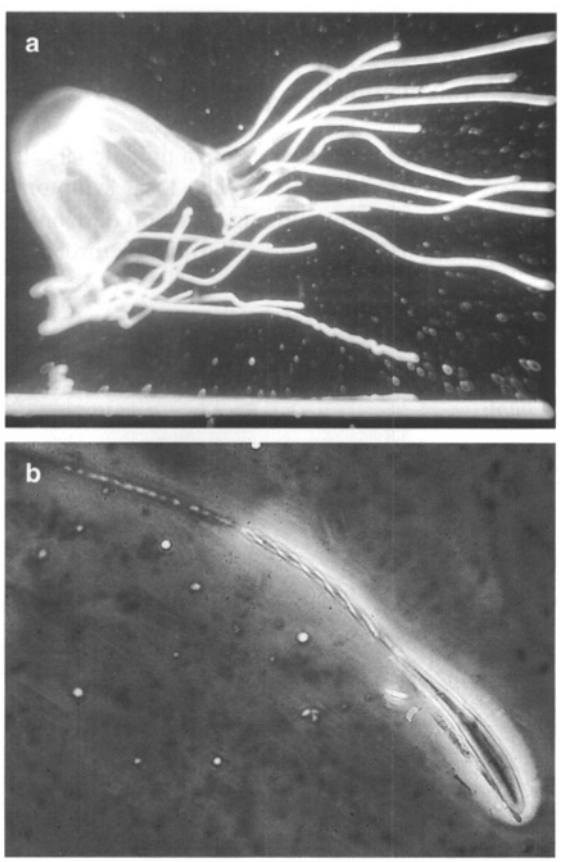

Figure 32.4 *Nematocysts: (a):* Chironex fleckeri. *(b): discharging nematocyst of* Chironex fleckeri *(box jellyfish). (Photo by K. Gillett.)*

complete, and stinging can occur even through surgical gloves.

Toxin

The venom is made up of lethal, dermatonecrotic and haemolytic fractions with specific antigens, and cross-immunity probably does not develop to other species. The effects on the cardiovascular system are an initial rise in arterial pressure which is followed by hypotensive/hypertensive oscillations. It is probably due to interference with vasomotor reflex feedback systems. The hypotensive states are related to bradycardia, cardiac irregularities (especially delay in AV conduction) and apnoea, and these oscillate with hypertensive states. The cardiovascular effects are due to cardiotoxicity, baroreceptor stimulation and/or brain/stem depression. Ventricular fibrillation or asystole will precede cerebral death.

Clinical features

The patient usually screams as a result of the excruciating pain, occurring immediately on contact, and increasing in intensity, often coming in waves. He/she then claws at the adherent tentacles (whitish strings surrounded by a transparent jelly) and may become confused, act irrationally or lose consciousness, and may drown because of this.

> *Chironex stings are excruciatingly painful and possibly fatal.*

Local

Multiple interlacing whiplash lines – red, purple or brown – 0.5 cm wide, develop within seconds. The markings are in a 'beaded' or 'ladder' pattern, and are quite characteristic. These acute changes will last for some hours. They are also described as transverse weals. If death occurs, the skin markings fade, but if the patient survives, the red, swollen skin may develop large weals and, after 7–10 days, necrosis and ulceration develop over the area of contact. The skin lesions may take many months to heal if deep ulceration occurs. Itching may also be troublesome and recurrent. Pigmentation and scarring at the site of these lesions may be permanent.

General

Excruciating pain dominates the clinical picture, while impairment of consciousness may lead to coma and death. The pain diminishes in 4–12 hours. Amnesia occurs for most of the incident following the sting. If death occurs, it usually does so within the first 10 minutes, and survival is likely after the first hour.

Cardiovascular effects dominate the generalized manifestations. The patient may develop cardiac shock, appearing cold and clammy with a rapid pulse, disturbance of consciousness, hypotension, tachycardia and a raised venous pressure. The cardiac state may oscillate within minutes from episodes of hypertension, tachycardia, rapid respirations and normal venous pressure to hypotension, bradycardia, apnoea and elevated venous pressure. The oscillation may give a false impression of improvement just before the patient's deterioration.

Respiratory distress, pulmonary congestion and oedema, and cyanosis may be due to the cardiac effects or to a direct mid-brain depression. Paralysis and abdominal pains may also occur. Malaise and restlessness may persist, with physical convalescence requiring up to a week. Irritability and difficulty with psychological adjustment may take weeks or months to subside. Immunity to the sting is said to occur following repeated and recent contacts, although it is likely that the cross-immunity between the species is incomplete or absent.

Treatment

First aid

The first step is to prevent drowning. Copious quantities of vinegar should be applied to reduce the likelihood of discharge of more nematocysts; this may be repeated if necessary. The tentacles and undischarged nematocysts should be removed gently but quickly, pulling in one direction only. Rough handling and rubbing will cause further nematocysts to discharge.

If vinegar is not available, other materials may be of value, but there is much conflict over which substances may aggravate the condition. Local remedies, such as lemon or lime juice, have yet to be evaluated, but it is possible that stale wine or even cola-type drinks may reduce the nematocyst discharge.

Pressure bandage/immobilization has been proposed by some authorities as a way of reducing venom absorption, but others have been more circumspect – observing that this might result in trauma and induce extra nematocyst discharge and increased concentration of the venom. Currently, there is inadequate experience to recommend this approach, and some indications that it may be hazardous.

Cardiopulmonary resuscitation may be required if the patient has stopped breathing. This should be continued and reapplied whenever there is any deterioration in the patient's condition. Do not assume because there is initial improvement that the patient will not relapse.

Treatment of chironex sting

- *Rescue patient from water.*
- *Apply vinegar in copious quantities.*
- *Mouth-to-mouth respiration and external cardiac massage as required.*
- *Box jellyfish antivenom, if available.*
- *Application of local anaesthetic ointment.*
- *Cardiopulmonary resuscitation techniques.*
- *Intravenous narcotics or general anaesthesia.*
- *Steroids ?*

Medical

Local applications include lignocaine or other local anaesthetic ointment. This may assist even after the first few minutes, during which time the traditional vinegar is believed to be of prophylactic value. Analgesics include morphine 15 mg or pethidine (demerol) 100 mg, intravenously in divided doses. This may also protect against shock.

Hydrocortisone 100 mg is administered intravenously every 2 hours if needed. Local steroid preparations are valuable for treating local manifestations such as swelling, pain and itching. Intermittent positive-pressure respiration, possibly with oxygen, replaces mouth-to-mouth artificial respiration, if needed. This will require constant attention because of the varying degree of respiratory depression. General anaesthesia with endotracheal intubation and controlled respiration is needed if analgesia cannot otherwise be obtained.

Chlorpromazine 100 mg intramuscularly, or diazepam 10 mg intravenously, etc. may be of value after the immediate resuscitation, as they will assist in sedating and tranquilizing the patient without causing significant respiratory depression. Other drugs may be used but are unproven in this clinical disorder. These include noradrenaline (Levophed) or isoprenaline (Isuprel) drips for hypotension, respiratory or cardiac stimulants, verapamil, etc. Continuous electrocardiogram monitoring is indicated, as are pulse rate, blood pressure, central venous pressure, respiratory rate, arterial gases, pulse oximetry and pH levels. External cardiac massage and defibrillation is given if required.

Chironex (box jellyfish) antitoxin has been developed by the Australian Commonwealth Serum Laboratories, and is derived from the serum of hyper-immunized sheep. It is of value against both the local and general manifestations. Local steroid ointment may relieve the severe itching which may follow the acute skin lesion.

Prevention

This includes the wearing of adequate protective clothing (overalls, wet suits, lycra or body stockings, etc.). Swimming or wading should be restricted to the safe months of the year, with care being especially needed on cloudy days toward the end of the hot season. Dragging a section of a beach with a 2.5 cm mesh has been used, though not very successfully, to clear an area for bathing.

Irukandji syndrome

An array of systemic symptoms, known as the irukandji syndrome, may follow an almost negligible sting from a variety of jellyfish.

The name irukandji was given by Dr. Flecker, after a local aboriginal tribe living near Cairns, Australia, where the injury was first described. Similar clinical syndromes have now been reported from many other warm water areas throughout the world. In 1964, another Cairns physician identified the actual cause of the syndrome in that area to be a small box jellyfish, now known as *Carukia barnesi*. Many other

similar disorders were noted along the north Australian and the north-west Australian coastlines, also in tropical waters, producing illness which could be, and has been, confused with decompression sickness.

Similar clinical symptoms may accompany stings from other coelenterates.

This animal is rarely observed by the victim, though the stinging may occur near the surface and in shallow water. It is classically a small box jellyfish with a transparent body about 1–2 cm long and with four tentacles varying from 5 cm to 1 metre in length, depending on the degree of contraction. Nematocysts, appearing as clumps of minute red dots, are distributed over the body and tentacles. The delayed injury is proportional to the duration, extent and location of the sting.

Stingings occur in clusters in the same locality, often in late summer, where clear warm ocean waters approach the land. Others occur well out to sea, in depths of 10–20 metres.

Clinical features

Local

A few seconds after contact, a stinging sensation is felt and this increases in intensity for a few minutes and diminishes during the next half hour. It is usually sufficient to cause children to cry, and adults to leave the water. It may recur at the commencement of the generalized symptoms, but is overshadowed by them.

A red-coloured reaction of 5–7 cm surrounds the area of contact within 5 minutes. Small papules (pimples) appear and reach their maximum in 20 minutes, before subsiding. 'Kissing' lesions occur, where the original skin lesion comes into contact with other skin, for example, near joints. The red colouration can occasionally last up to 3 hours and there is a dyshydrotic reaction (skin dry at first, with excessive sweating later) over the area. Occasionally, in severe cases the area may remain swollen for many hours.

There is usually a latent period of 5 to 120 minutes between contact and the development of generalized symptoms. The patient may not relate these symptoms to the local reaction, unless specifically questioned about this.

Generalized

Pain usually dominates the clinical presentation. Abdominal pains, often severe and associated with spasm and board-like rigidity of the abdominal wall, come in waves. Muscular aches such as cramps and dull boring pains occur, with increased tone and muscle tenderness on examination. This especially involves the spine, but also includes the hips, shoulders, limbs and chest. Headache may also be severe. Profuse sweating, anxiety and restlessness may develop, as may nausea and vomiting.

Respiratory distress with coughing, and grunts preceding exhalations may occur. Pulmonary oedema has been described, usually many hours after the stinging. There may be increased blood pressure and pulse rate, with possible arrhythmias.

Later symptoms include numbness and tingling, itching, smarting eyes, sneezing, joint and nerve pains, weakness, rigors, dry mouth and headache. The temperature usually remains normal, although there may be an increased pulse rate.

Symptoms diminish or cease within 4–12 hours, but occasionally malaise and distress may persist and convalescence may take up to a week.

Prevention

Protective clothing (wet suits, lycra, etc.) should be worn. Once stingings have been reported, the water should be avoided.

First aid

It is currently believed that the copious use of locally applied vinegar for at least a minute may reduce subsequent discharge of nematocysts. The use of compression bandages has also been recommended by some people, but this is contentious. The worry is that these bandages may themselves traumatize the nematocysts and increase their discharge rate.

Medical treatment

This is carried out in step-wise fashion:

1 During the severe phase with abdominal pains, spasms and coughing, administer pethidine intravenous e.g. 0.25–0.5 mg/kg over 5 minutes, to be repeated as necessary (often after 30 minutes).

The response is impressive and occurs within minutes. Sometimes large doses of pethidine, or the use of morphine, may be necessary.

2 Promethazine, with an intravenous dose of 0.25 mg/kg, to a maximum of 25 mg, will not only reduce the symptomatology of nausea and vomiting, but also reduce the subsequent amount of pethidine required. Any patient who has required more than 2 mg/kg of pethidine, implying significant envenomation, should be investigated with a chest X-ray, ECG and echocardiography, because of this risk.
3 Alpha-receptor blockers have been recommended for the control of hypertension, due to catecholamine release. Phentolamine may be given as a bolus dose and subsequent infusion (1–5 mg initially and 5–10 mg/hour). Hydralazine has also been used.
4 Other medications which have been used include diazepam, antihistamines and anaesthesia. General anaesthesia with assisted respiration could be used if the conventional techniques prove insufficient.
5 Monitoring of fluid and electrolyte state, together with cardiorespiratory parameters, would seem indicated, especially if there are any respiratory symptoms. Pulmonary oedema has been treated with intubation and controlled ventilation, high inspiratory oxygen and positive end-expiratory pressure.
6 During the latter part of the illness, when only fleeting neuralgic and arthralgic symptoms predominate, simple analgesics may be effective.

The following recommendations (devised by Little and Mulcahy) appear reasonable.

- If there are only **local symptoms**, treat symptomatically and observe for 2 hours. Observe until the victim is asymptomatic and if no medication is needed for 6 hours, then discharge him with the advice to return to the Emergency Department if symptoms recur.
- If **systemic symptoms** develop at any time, then admit the victim to hospital, give intravenous promethazine 0.25 mg/kg (maximum 25 mg) AND intravenous pethidine (0.25–0.5 mg/kg) every 5 minutes if required. If more than 2 mg/kg pethidine is required, then check the creatine kinase activity, urea and electrolyte levels, and do a full blood count.
- If **further analgesia** is required give fentanyl 0.5 µg/kg and observe closely. This may be repeated.
- A chest X-ray and electrocardiogram may be performed, and echocardiography should be considered. If signs or symptoms of pulmonary oedema develop (which may occur after 10–12 hours), then repeat the creatine kinase measurement and admit to a coronary care/intensive care unit.

CONE SHELLS

These attractive univalve molluscs are highly favoured by shell collectors of the tropics and warm temperate regions (see Plate 12a). They have a proboscis which is extendible from the narrow end, but are able to reach most of the shell. Holding the shell even by the 'big end' may not be safe, and may court a sting with a resultant 25 per cent mortality. The cone shell inhabits shallow waters, reefs, ponds and rubble, and is usually up to 10 cm in size (see Plate 12b). It has a siphon, sometimes ringed with orange, which detects its prey, and this may be the only part visible if the cone burrows beneath the sand. The proboscis, which delivers the coup-de-grace, carries one to ten radular teeth which penetrate and inject venom into its prey, thus immobilizing the victim.

Probably only the fish-eating cones are dangerous to man, and as these are difficult to distinguish at first sight, discretion is recommended. The venom is composed of two or more substances. One interferes with the neuromuscular activity and elicits a sustained muscular contracture; the other abolishes the excitability of muscle fibres and summates with tubocurare, but is uninfluenced by eserine. The major effect appears to be directly on skeletal muscular activity. Children are particularly vulnerable.

Cone shell venom causes skeletal muscle paresis or paralysis, with or without myalgia.

Clinical features

Local

The initial puncture effects may vary from painless to excruciating agony, and may be aggravated by salt water. The puncture region may become inflamed and swollen, sometimes white and ischaemic, with a cyanotic area surrounding it, and it may be numb to the touch.

General

Numbness and tingling may ascend from the bite to involve the whole body, and especially the mouth and lips; this may take about 10 minutes to develop. Skeletal muscular paralysis may spread from the site of injury, and result in anything from mild weariness to complete flaccid paralysis. Difficulty with swallowing and speech may occur before total paralysis. Visual disturbances may include double and blurred vision (paralysis of voluntary muscles and pupillary reactions). These changes may take place within 10–30 minutes of the 'bite'. Respiratory paralysis may dominate the clinical picture, resulting in shallow rapid breathing and a cyanotic appearance, proceeding to apnoea, unconsciousness and death. Other cases are said to result in cardiac failure, although this is probably secondary to the respiratory paralysis. The extent of neurotoxic damage is variable; if the patient survives, he/she is active and mobile within 24 hours. However, neurological sequelae and the local reaction may last many weeks.

Treatment

First Aid

The following recommendations are made:

- **Without** paralysis. The limb must be immobilized and a pressure bandage applied to reduce the speed of venom absorption. The patient should be rested and reassured.
- **With** paralysis. Mouth-to-mouth respiration may be needed, and this may have to be continued for hours, or until medical facilities are reached. This artificial respiration is the major contribution to saving the patient's life. External cardiac massage, as well as mouth-to-mouth respiration is needed if the patient has neither pulse nor respiration. The patient may be able to hear but not communicate and thus requires reassurance. If he is shocked, ensure that he is lying down with his feet elevated.

Medical treatment

With respiratory paralysis, artificial respiration should be administered with intermittent positive pressure adequate to maintain normal arterial gases and pH. Endotracheal intubation prevents aspiration of vomitus and facilitates tracheobronchial toilet, when indicated. Routine care and management of the unconscious patient is required. External cardiac massage, defibrillation, vasopressors, etc., may be indicated by the clinical state and electrocardiogram. Local anaesthetic can be injected into the wound. Respiratory depressants, respiratory stimulants and drugs used against neuromuscular blockade are not indicated.

Prevention

The people at risk, e.g. shell collectors, visitors to the reefs, and school children, etc., need to be educated about this danger. They should avoid contact with the cone shell. Probably no part of it can be touched with impunity, unless the animal is dead. Despite advice to the contrary, touching the 'big end' is not always safe. If these shells must be collected, it is advisable to use forceps and a tough receptacle.

BLUE-RINGED OCTOPUS

This animal usually weighs from 10 to 100 g, and is currently identified only in the Australasian and Central Indo-Pacific region (see Plates 13(a) and (b)). Its span, with tentacles extended, is from 2 to 20 cm, but usually less than 10 cm. It is found in rock pools, clumps of cunjevoi and shells, from the tidal zone to a depth of 10 metres. The colour is yellowish brown, with ringed marking on the tentacles and striations on the body, but these markings change to a vivid iridescent blue when the animal is feeding, becomes angry, excited, disturbed or hypoxic.

The heavier specimens are more dangerous, and handling these attractive creatures has resulted in death within a few minutes. Many such incidents have probably escaped detection by the coroner.

Autopsy features are non-specific and the bite fades after death.

The toxin ('maculotoxin') is more potent than that of any land animal. Analysis of posterior-salivary extracts demonstrates a hyaluronidase and cephalotoxins of low molecular weight, identical to tetrodotoxin. The effects are that of a neurotoxin and a neuromuscular blocking agent. It is not curare-like, and is not influenced by neostigmine and atropine, at least during the acute phase. Hypotension may develop.

The maculotoxin of the blue-ringed octopus is identical to tetrodotoxin from the puffer fish. It is a neurotoxin and a neuromuscular blocker, resulting in painless skeletal muscle paralysis.

Clinical features

Local

Initially the bite is usually painless, and may thus go unnoticed. The 1 cm circle of blanching becomes oedematous and swollen in 15 minutes, but then becomes haemorrhagic and resembles a blood blister. If the patient survives the next hour, he notices a local stinging sensation for around 6 hours. A serous or bloody discharge may occur, and local muscular twitching may persist for some weeks.

General

A few minutes after the bite, a rapid, painless paralysis dominates the clinical picture, which progresses in this order: abnormal sensations around mouth, neck and head; nausea and/or vomiting may occur; dyspnoea with rapid, shallow and stertorous respirations leading to apnoea, asphyxia and cyanosis; visual disturbances, involvement of the extraocular eye muscles results in double vision, blurred vision and ptosis whereas intraocular paralysis results in a fixed dilated pupil; difficulty in speech and swallowing, general weakness and incoordination progresses to complete paralysis. The duration of paralysis is between 4 and 12 hours, but the weakness and incoordination may persist for another day. The patient's conscious state is initially normal, even though he may not be able to open his eyes or respond to his environment. The respiratory paralysis (causing hypoxia and hypercapnia) finally results in unconsciousness and then death, often within minutes of the commencement of symptoms, unless resuscitation is continued. Cardiovascular effects of hypotension and bradycardia are noted in severe cases.

There may be a cessation at any stage of the above clinical sequence, i.e. the effects may cease with the local reaction, a partial paralysis, or proceed to a complete paralysis and death. Less severe bites may result in generalized and local muscular contractions, which may continue intermittently for 6 hours or more. This occurs with a subparalytic dose. Other symptoms noted in mild cases include a light-headed feeling, depersonalization, paraesthesia, weakness and exhaustion.

Treatment

First aid

The following recommendations are made:

- **Before paralysis:** immobilization of the limb and application of a pressure bandage will reduce the absorption of venom. The patient should be rested, preferably lying on his side in case of vomiting, and not left unattended. Only after hospitalization, where preparations have been made for respiratory support, should the pressure bandage be removed.
- **With paralysis:** mouth-to-mouth respiration should be applied to ensure that the patient does not become cyanotic. Attention must be paid to the clearing his airway of vomitus, tongue obstruction, dentures, etc. If an airway is available, this should be inserted – but it is not essential. Artificial respiration may have to be continued for hours, until the patient reaches hospital. If delay has occurred, then external cardiac massage may also be required. The patient, who can hear but not communicate, must be reassured that you understand his condition. Enlist medical aid, but never leave him unattended to obtain this.

Medical

For respiratory paralysis, artificial respiration with intermittent positive-pressure respiration is necessary to maintain normal arterial blood gases.

Endotracheal intubation also prevents aspiration of vomitus and facilitates tracheobronchial toilet, when indicated. Usual management of the unconscious patient is required.

Edrophonium (Tensilon) and neostigmine is of no value during the deeply paralysed state. Other central respiratory stimulants may be of use in borderline cases or during the recovery period. Local anaesthesia infiltration to the painful area will provide local relief of delayed pain. For delayed allergic reactions, intravenous hydrocortisone for systemic effects, subcutaneous adrenaline for bronchospasm, or oral antihistamines for skin lesions are indicated.

Prevention

Contact with the octopus should be avoided, and empty shells should be treated with suspicion. Requests by scientific groups for collection of these specimens should be tempered with caution. A public program on the dangers of this animal should be directed especially to children, who are attracted by the bright colouration.

OTHER MARINE ANIMAL INJURIES

Only a few of these are mentioned in this text. The sea urchin, electric rays and coral cuts are selected for inclusion because of their interest and frequency in tropical and temperate regions.

Sea urchin

Of the 6000 species of sea urchins, approximately 80 are thought to be venomous or poisonous to man. They belong to the phylum Echinodermata, named after the hedgehog (Echinos) because of the many-spined appearance. In some, such as *Diadema setosum*, the **long spined** or **black sea urchin** (see Fig. 32.5), the damage is mainly done by the breaking off of the sharp brittle spines after they have penetrated the diver's skin. Sometimes the spines have disappeared within a few days, but in other cases they become encrusted and may remain for many months, to emerge at sites distant from the original wound. They are commonly covered by a black pigment, which can be mistaken for the actual spine during its removal.

Figure 32.5 *Diadema setosum. (Photo courtesy of R. Chesher.)*

Other sea urchins, such as the **Crown of Thorns** (*Acanthaster planci)* (see Plate 14) can also cause damage by the spines piercing the skin, but seem to have a far more inflammatory action, suggestive of a venom. Vomiting is a frequent accompaniment. Injuries from the Crown of Thorns have been more commonly reported since divers attempted to eradicate them from reefs.

The most potent sea urchins are the **Toxopneustidae**, which have short thick spines poking through an array of flower-like pedicellariae. Deaths have been reported from this urchin. The venom is thought to be a dialyzable acetylcholine-like substance.

Treatment

The long spines tend to break easily, and therefore need to be pulled out, without any bending. A local anaesthetic may be required if surgical extraction is contemplated. Drawing pastes such as magnesium sulphate have been used. Some find relief with the use of heat, and others have removed the spines by the use of a snake-bite suction cap.

A variety of interesting treatments have developed. In Nauru it is claimed that urinating on the wound immediately after the injury produces excellent results; this presumably relieves the bladder, if not the pain! The use of meat tenderizer owes more to good advertizing than to therapeutic efficiency.

One technique which would be described as barbaric, had it not been for the fact that it seems to work, is to apply extra trauma and movement to the area – to break up the spines within the tissue. It does seem as if, in this case, activity is more beneficial than rest and immobilization. With the latter, the limb tends to swell and become more painful. Spines may remain in tissues for several months, sometimes causing little disability before emerging through the skin.

Occasionally patients will present having eaten sea urchins. In Tonga they are used as an aphrodisiac; however, the ovaries may be poisonous and produce both gastrointestinal and migraine-like symptoms.

Sponges

These sedentary animals require some defense from mobile predators, and they have developed a skeleton of calcareous and silicaceous spicules. They also have a form of toxin which is not well understood. About a dozen sponges are toxic, from the 5000 or so species, and they are mainly in the temperate or tropic zones. Skin lesions have developed from sponges which have been deep-frozen or dried for many years.

Clinical features

Local symptoms relate to the contact dermatitis associated with the areas of sponge contact. After a variable time, between 5 minutes and 2 hours, dermal irritation is felt. It may be precipitated by wetting or rubbing the area, and may progress over the next day or so and feel as if ground glass has been abraded into the skin. Hyperaesthesia and paraesthesia may be noted. The symptoms can persist for a week or more with inflammatory and painful reactions around the area. The degree of severity is not related to the clinical signs and some patients may be incapacitated by the symptoms without any objective manifestations.

The dermal reaction may appear as an erythema, with or without papule and vesicle development. There is sometimes a desquamation of the skin in the second or third week, but in other cases the skin lesions have recurred over many months.

Treatment

The only adequate treatment is prevention, using gloves when handling sponges, and not touching anything that has been in contact with them.

The use of alcohol, lotions or hot water will usually aggravate the condition. Local application of cooling lotion such as calamine may be of some value, but the treatment with conventional dermatological preparations has limited success.

Coral cuts

General

Corals, because of their sharp edges combined with man's awkwardness, often cause lacerations. The sequelae of this may well equal the intensity of the more impressive marine animal injuries. Not only is the coral covered by infected slime, but also pieces of coral or other foreign bodies will often remain in the laceration. It is possible that some of the manifestations, especially initially, are due to the presence of discharging nematocysts. There have also been occasional patients who have been infected by the marine organism *Erysipelothrix*.

Certain vibrios may be present in the marine environment and can cause serious infection. These may be cultured in a saline media if identification is to be made.

Clinical features

A small, often clean-looking laceration is usually on the hand or foot. This causes little inconvenience at the time of injury and may go unnoticed, but a few hours later there may be a 'smarting' sensation, especially during washing. At that stage, there is a mild inflammatory reaction around the cut. Within the next day or two the inflammation becomes more widespread with local swelling, discolouration and tenderness. In severe cases there may be abscess formation with chronic ulceration and even osteomyelitis.

After healing there may be a small numb area of skin with a fibrous nodule beneath it; this is a keloid reaction to the foreign body (coral).

Treatment

This involves thorough cleansing of the area, removal of the foreign material and the application of an antibody powder or ointment, e.g. neomycin. One sequel of coral cuts is sometimes a very unpleasant pruritus that can be troublesome for many weeks but which responds to the use of a local steroid ointment.

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Fish poisoning

CARL EDMONDS

INTRODUCTION

Food poisoning from ingested marine animals ('seafood') is a serious hazard to many populations, especially as three-fourths of the world's population live within 15 km of a coastline. Such poisoning is particularly important in tropical or temperate climates, where the outbreaks tend to be sporadic and unpredictable. Commercially valuable industries have been curtailed or prohibited because of the serious threat from this high-protein, readily available food. In cold climates, poisoning from marine and polar animals is also of major importance, but it is more predictable and can be avoided.

Diseases which can destroy whole communities, change the fate of military operations, devastate fishing industries – yet still arise sporadically in a previously safe marine environment – are worthy of considerable investigation and research. Such has not been the case, this subject having been sadly neglected, both in medical research and in medical training.

For those physicians associated with marine medicine, yachting, diving or travel, and those who practise near coastlines, a knowledge of seafood poisoning is essential. It is also important to those involved in public health, industrial medicine, and to the general health of island communities.

In this book, limits of space remit the description of only a few of the more significant fish poisonings, and ciguatera, tetrodotoxin and scombroid poisoning will be dealt with because of their commercial implications. Shellfish and crustacean poisoning will be summarized. No reference is made to barracouta poisoning, hallucinatory fish poisoning, mercury poisoning, other pollutions, seal liver poisoning, shark and ray poisoning, turtle poisoning, and many others. One complicating factor is that there may be more than one type of marine poison responsible for the clinical manifestations in the patient.

Viral and bacterial contamination of fish and shellfish is also common, but is dealt with in other texts.

CIGUATERA POISONING

On a worldwide basis, ciguatera poisoning is the most serious of the marine toxins. It is mainly a disorder of the tropics and, to a lesser degree, the semi-tropical and temperate zones, being mostly found between the 35° latitudes north and south. The fish – which cannot be identified as poisonous by their external appearance – tend to be reef fish, and may either ingest the *Gambierdiscus toxicus* (which is thought to be the origin of the toxin), or they may acquire it by eating other fish. Ciguatoxin is harmless

to the fish , but it tends to concentrate as it travels up the food chain to the more active carnivorous predators. It is for this reason that the larger predator fish tend to be more toxic.

Local knowledge indicates areas in which the fish are poisonous, though this is not entirely reliable as the areas may change. Poisoning is more likely when reefs have been disturbed by natural damage such as hurricanes, or by man-made damage such as constructions, atomic explosions, etc. which disrupt the ecology and permit proliferation of *Gambierdiscus toxicus*.

A variety of techniques have been promulgated by folklore to predict which fish will be safe to eat. Observations which are totally irrelevant, despite parochial beliefs, include

- the presence of worms in the fish;
- whether ants or flies refuse it;
- whether a silver coin will turn black; or
- whether grated coconut will turn green if cooked with the fish.

Perhaps the safest test is to feed a small amount of the fish to a kitten, which is highly sensitive to most fish poisons. If the animal is still alive and unaffected a few hours later, then the fish is probably safe to eat. A traditional variant to this, is the rather pragmatic system of feeding the older members of the family first; if they are unaffected, the remainder of the fish is used to feed the children and the more productive members of the society. (*Note*: this ageing author takes exception to this deplorable practice.)

In a survival situation, the advice is as follows: do not eat the viscera of the fish (e.g. liver, gonads, intestines, etc.). Avoid the exceptionally large reef predators and those species often implicated in ciguatera poisoning. These include barracuda, grouper, snapper, sea bass, mahi-mahi, surgeon fish, parrot fish, wrasses, jacks and many others. Moray eels are particularly virulent. Boiling the fish many times and discarding the water after each boiling may be helpful. Alternatively, the fish may be sliced and continually soaked in water which should be changed every 30 minutes or so. Nonetheless, only small quantities should be eaten.

Symptoms may vary, and be modified by the presence of other marine poisons such as maitotoxin, scaritoxin, okadiac acid, etc. Ciguatoxins increase sodium channel permeability, release norepinephrine (noradrenaline)and may also increase calcium uptake in cells.

Clinical features

Symptoms usually develop 2 to 12 hours after ingestion of the food, although more severe cases tend to occur earlier.

Generalized non-specific symptoms may develop, including weakness and dull aches in the limbs and head. These muscle pains may progress to a more severe weakness, with or without cramps. The pains differentiate this disorder from tetrodotoxin poisoning. Paraesthesiae and numbness are noted around the mouth, and sometimes also in the peripheries. Gastrointestinal problems, including anorexia, nausea, vomiting and diarrhoea, may last for a few days. Severe neurological disturbances may develop in 12–36 hours and include delirium, cranial nerve involvement, incoordination and ataxia, with occasional extrapyramidal disorders, convulsions, coma and even death. Death is likely to be due to respiratory failure, although in severe cases there is evidence of hypotension, cardiac dysrhythmias, and other cardiovascular problems.

Skin lesions are characteristic, and include erythema, pruritus or a burning sensation – sometimes with vesicular formation. These may be very severe for a few days, but then usually subside. Hair and nail loss may supervene. In the severely affected cases, these skin lesions may be troublesome for many weeks. In females, the vagina may be affected, sometimes severely, causing symptoms of cystitis or dyspareunia. Less commonly, males may notice pain during ejaculation, and the toxin may be transmitted in semen, in turn causing local symptoms in the female.

The death rate in different series varies from 0.1 to 10 per cent. In severe outbreaks, the presentation of the disease can be acute and widespread. In most Indo-Pacific regions the disease tends to be sporadic and mild. In these cases the main symptoms can clear within one or two days, although residual weakness and paraesthesiae, together with a reversal of temperature perception, may persist for long periods.

Severe cases may take many months or up to a year for full recovery. Exacerbations can be precipitated by

alcohol, nicotinic acid and other vasoactive drugs. The production of an erythematous area associated with a burning sensation following intake of alcohol is pathognomonic of this disorder. The illness can also recur following stress or the ingestion of certain fish, or animals fed on fish meal. Immunity does not develop, and subsequent poisonings may be even more severe.

Treatment

Treatment includes the removal of unabsorbed material by induction of vomiting, or by gastric lavage in patients who do not have respiratory depression. Activated charcoal may be taken orally. Rest and observation in a hospital are required until the patient has recovered. Respiratory support may be required.

The medical treatment is basically symptomatic. Many different pharmacological remedies have been proposed, but none is consistently effective. A recent treatment is the use of intravenous mannitol, especially if given early. A 250 ml volume of 20 per cent intravenous mannitol given slowly (to a maximum of 1 g/kg), piggy backed to a 5 per cent dextrose infusion, has produced some good results. It is best given as soon as possible after the poisoning, but may still be of value for up to two weeks afterwards.

Drugs which have been suggested and which seem to do no harm include lignocaine, steroids and calcium gluconate 10 per cent intravenous to relieve the neuromuscular or neurological features and perhaps increase muscle tone. As a general rule, pharmacological treatment does not have nearly the effectiveness of general medical care. Symptomatic treatment, while not using vasoactive drugs, seems most valuable. Diazepam can be given safely, and severe cases may require the assistance of a neurologist or an organically oriented psychiatrist for pharmacological advice. A tricyclic antidepressant may be of benefit if given in small dosages, e.g. amitryptiline 25–50 mg nocte.

TETRODOTOXIN POISONING

Of all that are in the waters you may eat these: whatever has fins and scales you may eat. And whatever does not have fins and scales you shall not eat; it is unclean for you.

Deuteronomy 14:9–10.

Tetrodotoxin poisoning follows the ingestion of puffer fish, ocean sunfish or porcupine fish. The name puffer comes from the ability of the fish to inflate itself by taking in large quantities of air or water. The scales have also been modified to form protective plates or spikes. These fish are recognized as poisonous throughout the world, although they are more common in tropical and temperate regions. The toxin is concentrated mainly in the ovaries, liver and intestines. Lesser amounts occur in the skin, but the body musculature is usually free of poison. The toxicity is related to the reproductive cycle.

With two exceptions, these fish are usually considered inedible. The first exception is the uninformed consumer. Captain James Cook on September 7, 1774, sampled this fish in New Caledonia with near-fatal results. The other exception is the Asiatic gourmet consuming 'Fugu'. After a prolonged apprenticeship, specially licensed chefs in Japan are allowed to prepare this fish, receiving considerable kudos by retaining enough of the toxin to produce a numbing effect in the mouth – but not enough to cause death. Nevertheless, accidents do happen, and the death rate from Fugu poisoning is about 50 cases per year.

The toxin interferes with neuromuscular transmission in motor and sensory nerves and in the sympathetic nervous system, by interfering with sodium transfer. It also has a direct depressant effect on medullary centres, skeletal muscles (reducing excitability), intracardiac conduction and myocardial contractility. Hypotension may be due to either the effects on the preganglionic cholinergic fibres or the direct effect on the heart. Respiratory depression precedes cardiovascular depression.

Clinical features

The onset and severity of symptoms vary greatly according to the amount of toxin ingested. Usually within the first hour, the patient will notice muscular weakness and other effects of blockage of the motor and sensory systems. This may progress to total skeletal paralysis, including respiratory paralysis.

Paraesthesia around the mouth may also extend to the extremities, or it may become generalized. Autonomic effects include salivation, sweating, chest pain and headache. Gastrointestinal symptoms of nausea, vomiting, diarrhoea, etc., may develop, and there is sometimes a decrease in temperature, pulse rate and blood pressure.

A coagulation disturbance, which is an occasional complication, may lead to systemic bleeding or desquamation from haemorrhagic bullae.

The **neurological involvement** may commence as muscular twitching and incoordination and may proceed to a complete skeletal muscular paralysis. Bulbar paralysis may produce interference with speech and swallowing. The pupils, after initially being constricted, may become fixed and dilated. The clinical picture is, therefore, one of a generalized paralysis with the patient maintaining a fully conscious state while oxygenation is maintained. This is important in the treatment, because the patient is able to hear and appreciate the statements made by people around him.

The high death rate from this disorder is due to respiratory paralysis and occurs within 24 hours of ingestion. It is a reflection of incorrect diagnosis or inadequate resuscitation techniques, in most cases.

Treatment

Before the patient shows signs of paralysis or weakness, the use of an emetic or gastric lavage may be of value in removing poisonous material. Activated charcoal may be taken orally. Lavage may also be employed if controlled respiration has required the insertion of an endotracheal tube, which will prevent aspiration of stomach contents.

After weakness has become apparent, the treatment is entirely symptomatic, i.e. maintenance of an adequate respiratory state, monitoring of vital signs, measurement of arterial blood gas and biochemical profile. The patient may require a mechanical ventilator for up to 24 hours, before regaining muscular control. Because consciousness is retained in the absence of skeletal or respiratory movement, the periodic administration of a minor tranquilizer such as diazepam seems prudent. Continuous explanation and reassurance should be given, even though the patient cannot respond physically to these.

Various pharmacological treatments have been proposed, including intravenous calcium gluconate 10 per cent, anticholinesterases, respiratory stimulants, steroids, etc., though there is no firm evidence that these are of value.

General nursing care, with special attention to pressure areas and eye and mouth toilets is axiomatic in these paralyzed and debilitated patients.

Prevention

'Scaleless' fish should not be eaten unless they are known to be harmless. If one is forced to eat Fugu in Japan, it should be purchased from a first-class restaurant with a licensed cook. All the viscera and skin must be removed.

In a survival situation, these fish should be eviscerated and only the musculature should be consumed. The meat should be cut or torn into small bits and soaked in water for at least 4 hours. The fish should be kneaded during this time and the water changed at frequent intervals. The toxin is partly water-soluble, and therefore this soaking may help to remove it. Do not eat more than is required to maintain life. Feeding of the fish to test animals has been suggested.

SCOMBROID POISONING

General

This disorder is possible wherever mackerel-like fishes, tuna, bonito or albacore are caught and eaten without adequate care or preparation. It has also occurred in epidemics due to contaminated canned tuna and other large fish.

The fish, which are normally safe to eat, become poisonous when handled incorrectly. If left for several hours at room temperature or in the sun, the histidine in their muscular tissues is changed by bacterial action into saurine, a histamine-like substance. The bacteria implicated include *Proteus morganii*, *Clostridium*, *Salmonella* and *Escherichia*. Laboratory verification of contaminated fish is obtained by demonstrating a histamine content in excess of 100 mg per 100 g of fish muscle.

Clinical features

Initially the fish may have a characteristic 'peppery' taste, but after 30–60 minutes other symptoms characteristic of histamine toxicity develop. Gastrointestinal symptoms associated with headache, palpitations and tachycardia with hypotension, are followed by a typical 'allergic-type' syndrome. The latter may involve the skin with a urticarial reaction, the respiratory system with bronchospasm, or the cardiovascular system in the form of anaphylactic shock.

Treatment

The first-aid treatment includes the removal of unabsorbed material by vomiting or gastric lavage if the patient is not too severely distressed. Treatment involves the customary techniques for handling dermatological, respiratory or cardiovascular manifestations of allergy and anaphyllaxis. These include antihistamines, adrenaline (or other sympathomimatic drugs) and steroids.

Prevention

Prevention is possible by correct care, storage and preparation of the fish. Prompt refrigeration and not leaving the fish exposed to the sun or room temperature has reduced the incidence of this disease. It is believed that pallor of the gills, or an odour or staleness, may indicate that saurine may be present in the fish.

SHELLFISH AND CRUSTACEAN POISONING

Shellfish include oysters, clams, mussels, cockles, etc.; crustaceans include lobsters, crayfish, prawns, crabs, yabbies, etc. Five different types of poisoning may develop from ingestion of these animals.

Gastrointestinal-type

This is the most common, and it develops many hours after ingestion of the contaminated shellfish. Viruses, marine vibrios, *Escherichia coli* and other bacteria and organisms have been implicated. Usually, manifestations last about 36 hours, but vary according to the organism, and are treated along general medical guidelines.

Allergic-type

The allergic reaction appears to be a typical hypersensitivity reaction to a protein in the shellfish. It is likely that the victim has previously been exposed to the same or similar protein to which he has developed an antibody reaction. Symptoms develop after the second and subsequent exposures, and are aggravated by exercise, heat and emotion. There may be a history of allergy to other foreign proteins, e.g. hay fever, antitoxins, horse serum, etc. The clinical features are dermatological, respiratory and/or cardiovascular in type and may, therefore, mimic scombroid poisoning. Antihistamines, adrenaline (or sympathomimetic drugs) and steroids tend to be used in the three manifestations, respectively.

Hepatic disease

There appears to be a hepatotoxin especially concentrated in molluscs, and perhaps related to the presence of a toxic dinoflagellate. This may result in severe hepatocellular disease with the clinical picture of acute yellow atrophy. Viral infection, causing infectious hepatitis, has also been reported.

Paralytic shellfish poisoning (psp)

> *The waters that were in the rivers were turned into blood, and the fish that were in the rivers died; and the river stank.*
>
> *Exodus 7:20–21.*

This disorder is due to the ingestion of a neurotoxin – saxitoxin – which is concentrated in shellfish, and produced by a marine protozoa, a dinoflagellate. Many species of these may be involved, but the commonest is *Alexandrium catanella.* Other species have also been incriminated, as have other toxins, which explains the variability of clinical manifestations seen with this disease. Saxitoxin acts by blocking sodium channels in nerve and muscle cells. It is derived originally from the dinoflagellates, which are filtered by mussels, clams, oysters and scallops, making consumption of these seafoods hazardous when the seawater dinoflagellates count is only 200 per ml. Neither steaming nor cooking affects the potency of the toxin; nor is it eliminated by commercial processing.

PSP is associated with the 'red tide' or other 'water bloom' – a discolouration of the sea caused by masses of dinoflagellates. The Red Sea was so named because of its occasional appearance during these red tides, and the North American Indians avoided shellfish for this same reason. There have been many outbreaks, the first clinical description dating back to 1689. However, there have since been many others, with 54 occurring in Alaska between 1973 and 1992 and affecting 117 individuals.

The symptoms usually come on within 0.5–3 hours (never more than 12 hours), and the prognosis is good for people surviving an additional 12 hours, although weakness and disability can occur for weeks afterwards.

The clinical symptoms are mainly those related to a developing neuropathy, affecting peripheral nerves, the central nervous system the autonomic nervous system and skeletal muscle.

The clinical effects, and treatment, are similar in many ways to tetrodotoxin poisoning.

Amnesic shellfish poisoning

This is, more correctly, domoic acid poisoning. First described in 1958, there have been a couple of severe outbreaks from ingestion of shellfish that have been contaminated with various marine organisms including a pennate diatome, *Nitzschia*. Some phytoplankton outbreaks have also occurred in Japan, Prince Edward Island in Canada, and the west coast of USA.

Domoic acid is an excitatory neurotransmitter which specially affects the hippocampus, thalamus and frontal lobes of the brain, leading to temporary or permanent damage.

Initially there are gastrointestinal symptoms (1–24 hours after ingestion), with neurological manifestations and memory loss developing within 48 hours, possibly progressing to seizures, multiple central nervous system effects and perhaps cardiac disorders. Bronchial secretions may be profuse, and require individuals to be treated with endotracheal intubation and resuscitation.

For those who survive there was often some neurological improvement for up to 12 weeks after ingestion.

The treatment is symptomatic as no antidote exists.

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34

Underwater explosions

JOHN PENNEFATHER

INTRODUCTION

Although most of our information on this topic has been acquired from naval research work, it is also relevant to some civilian divers. Explosives are used in salvage, mining and dredging operations, as well as in war-like activities which involve divers and shipwreck survivors. During the Second World War, there were many deaths of divers and swimmers following air, surface ship or submarine attack. One report claimed that mortality from the underwater blast injuries could approach 80 per cent.

For an explosion with the same energy and at the same distance, an underwater blast is more dangerous than an air blast. This is because in air the blast dissipates more rapidly and tends to be reflected at the body surface; in water the blast wave travels through the body and causes internal injuries.

PHYSICS OF BLAST WAVES

Some knowledge of underwater explosions is of assistance in understanding its clinical manifestations.

An explosion is a very fast chemical reaction, the process propagating through the explosive at 2–9 km/second. The products of the reaction are heat and combustion products such as carbon dioxide. Within the water, a bubble containing gas at a pressure of up to 50 000 atmospheres and a temperature of 30 000°C is produced. The bubble rapidly expands in a spherical form, displacing water. This rapid expansion generates the first pressure wave as the pressure in the gas bubble is transferred into the water, causing a pressure pulse that is transmitted through the water. This is sometimes called the 'short pulse' or 'primary pulse'. The initial pressure change of the wave is steep, with the increase to a peak pressure occurring within a few microseconds. The pressure in the bubble falls as it expands and the gas cools. The fall of pressure at the end of the explosion reflects the end of the expansion of the gases and occurs in milliseconds.

The momentum of the water which has been displaced by the bubble enlarges the bubble past its equilibrium volume, and a series of volume swings can be initiated. These volume oscillations of the bubble cause a series of pressure waves.

Near the point of detonation, the velocity of the first pressure wave is great, and is related to the speed at which the explosive detonates. Some explosives produce high pressures for a short period, while other types, with a slower reaction rate, produce less intense pressure waves that have a longer duration.

At a point some distance from the detonation, the velocity of the pressure waves slows to that of sound in water, about 1.5 km/seconds. From then on, the pressure waves follow the laws of sound in water. The energy of the waves decreases with distance, and they are reflected and absorbed in a similar fashion to sound waves.

In water the pressure pulse is not absorbed as rapidly as in air. In air, the gas surrounding the explosion is compressed and so absorbs energy from the explosion. In water, which is far less compressible, there is little absorption, so the pressure pulse is transmitted with greater intensity over a longer range. Thus, the lethal range of an explosion is normally far greater than the same mass of explosive in air.

A pressure wave is transmitted over a greater range in water than in air.

The energy in a typical blast is distributed in the following proportions: the initial pressure wave has approximately one-fourth of the energy, subsequent waves total one-fourth, and the other manifestations – such as heat and turbulence – comprise the remaining half. In an explosion most of the damage is caused by the initial pressure wave.

Other waves may result from an explosion. If the initial waves reach the sea bottom they may be reflected or absorbed, the proportions depending on the nature of the sea bed. If it is hard and smooth, there is little absorption and much reflection; conversely, a soft bottom absorbs more. The angle of incidence, i.e. the angle at which the waves strike the sea bed, will be equal to the angle of reflection. The reflected wave coming from the sea bed may combine with the other waves, causing increased damage. If the sea bed is distant from the point of detonation, this effect is negligible.

At the water surface the reflected wave is a negative pressure pulse rather than a positive pulse. As a result of this, a diver may experience a less intense pressure wave if he is close to the surface. The negative reflected pulse tends to cancel the positive direct path pulse.

At the surface a series of events may modify the pressure waves. Above a certain intensity the surface of the water will be broken or shredded, and thrown up into a dome, termed the 'dome effect'. This dissipates a small part of the pressure wave, and the remainder is reflected back into the water. Other disturbances which may be observed on the surface following the dome phenomenon include the 'slick' and 'plume'. The slick is a rapidly expanding ring of darkened water due to the advancing of the pressure waves. The plume is the last manifestation of the explosion, and is the result of gas reaching and breaking the surface of the water. Although the plume may be spectacular, it does little damage.

The surface phenomenon varies with the size and depth of the explosion. With a deep or small explosion, the dome may not form, as there may be insufficient energy to shred the water. The slick tends to be retained to a greater depth, being dependent only on the presence of the pulse wave.

Thermal layers may also reflect the pressure waves from the explosion, as may other objects such as large ships. The size of the charge, depth of detonation and distance from target will have an influence on the potential damage by the initial and subsequent pressure waves.

Charge size, distance and risk of injury

The risk of injury or death will be dependent on several variables: the size of the charge, the victim's distance from it, and the nature of the bottom. American sources[1] give the relationship below for estimating the effect of a TNT charge. Other explosives behave differently; the longer pulse from a slower detonating explosion can cause more damage than the same pressure from a more rapidly detonating explosive.

$$P\ (\text{lb/in}^2) \times \frac{\sqrt[3]{13\,000\ 3\ \text{charge size (lb)}}}{\text{Distance from charge (feet)}}$$

where P = pressure. Pressures over 2000 lb/in^2 will cause death, while pressures of over 500 lb/in^2 are likely to cause death or serious injury. Pressures in the range 50 to 500 lb/in^2 are likely to cause injury, but those less than 50 lb/in^2 are unlikely to cause any harm.

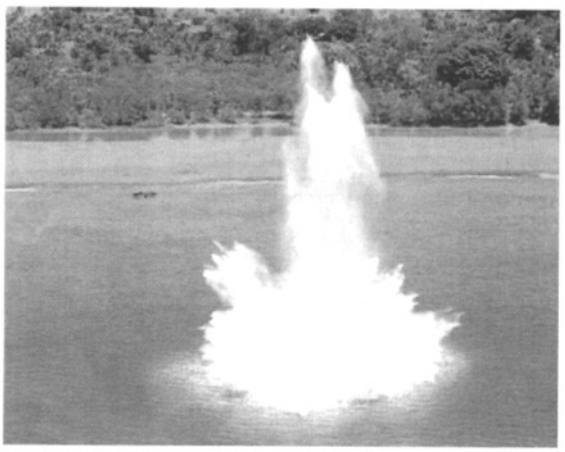

Figure 34.1 *This explosion was a deliberate test of the ship's ability to survive an underwater blast. Any diver in the water near it would have died from injuries caused by the pressure pulses it generated (source: RAN).*

MECHANISM OF INJURY[2–4]

In air explosions, the main cause of damage is from the fragments of the charge container, shrapnel and foreign bodies drawn into the explosive wave, as well as the pressure wave. In the water, particles are retarded by the medium. Also, with air explosions much of the pressure wave is reflected at the body surface, because this is an interface between media of different densities. Much of the blast effect probably acts through the mouth, nose and ears. Intestinal injury as a result of blast damage rarely occurs in explosions in air.

In water, the blast wave passes through the body as it is of a similar consistency to water. The individual molecules are displaced very little except in areas capable of compression, that is, gas spaces. Damage occurs mainly at the air–water interfaces within the body as the gas in the gas-filled cavities is almost instantaneously compressed as the pressure wave passes. The walls of these spaces are torn as the wave passes.

Damage can be expected in the lung, air-filled viscera in the abdomen, sinus cavities and the ear. In the lungs, the damage is not due to pressure transmitted via the upper airways, but is the result of transmission of the wave directly through the thoracic wall. The pressure wave reaches the gas–tissue interface at the respiratory mucosa, and it is there that the tissues are 'shredded' or torn apart.

> *Injury from underwater blast occurs mainly at gas–tissue interfaces, as in barotrauma.*

Animal experiments

Underwater blasts have been studied extensively in animals, and the respiratory and gastrointestinal tracts are seen to be the most significantly affected. Pathological examinations revealed injury to the lungs and gas-filled abdominal viscera. Central nervous system lesions have also been observed.

The respiratory damage included pulmonary haemorrhage, usually at the bases, bronchi and trachea, acute vesicular and interstitial emphysema, pneumothorax and haemothorax. Intestinal injury consisted mainly of subserous and submucosal haemorrhage and perforation of the gas-filled viscera. Renal and hepatic systems were not affected, with no evidence of either gallbladder or urinary bladder damage. If both the thorax and abdomen were immersed, the lungs would be consistently more affected than the intestines. If only the abdomen was immersed, then this would be most affected, with bleeding via the rectum.

The above results show the importance of the air–water interface in damage from an underwater blast. Wolf[5] cites experiments to confirm this phenomenon; if three loops of bowel are prepared and occluded, with one collapsed, another filled with saline and a third filled with air, only the air-containing loop sustained injury.

There is some contention regarding the cause of death in animals exposed to underwater explosions. In most cases early deaths are probably due to pulmonary lesions. These animals usually had a low arterial oxygen saturation, correctable by 100 per cent oxygen inhalation, with a respiratory acidosis and carbon dioxide retention. Some early deaths may be due to central nervous system involvement. Petechial haemorrhage and oedema have been noted in the brain; these might be caused by a rapid increase in the venous pressure, following compression of the chest

and abdominal venous reservoirs by the pressure wave. With this transmission of pressure into the cerebral venous system, small blood vessels may rupture. Another postulate is that pressurizing the air in the alveoli, or rupture of alveoli, may result in the production of air emboli. Air has been demonstrated in the cerebrovascular system of animals who were in the upright position during the blast.

> *Brain damage is thought to be caused by a rapid rise in venous pressure following compression of the chest and abdomen by the pressure wave.*

Late deaths result from the complications of respiratory, abdominal and neurological injuries. These include bronchopneumonia, peritonitis and coma and its sequelae.

CLINICAL FEATURES[2–4]

Information on the clinical features of underwater explosions in humans is mainly derived from case reports. Most of the physical parameters such as distance from the blast, intensity, etc. are not known, and thus clinical correlation with the physical parameters is not possible. Autopsies are usually complicated by the effects of drowning and immersion.

Huller and Bazini[6] reported on 32 casualties from an Israeli destroyer who, while they were in the water, were exposed to blast from an exploding missile. Nineteen had both pulmonary and abdominal injuries, while five had only abdominal injuries and eight had only pulmonary injuries. It is not possible to determine if there was any physical reason for this distribution of symptoms. Among 24 laparotomies performed, 22 showed tears of the bowel and 11 showed subserosal bleeding in other parts of the bowel. In one case the perforations were only found at a second operation, and in one case there was also a lacerated liver. One had an isolated rupture of the spleen, but this may have been due to an incident before the patient entered the water. An unexpected finding was that four cases had ECG changes consistent with myocardial injury. Three cases died from cardiorespiratory failure within 8 hours of surgery, and one case lasted for 48 hours before dying from peritonitis and sepsis.

Table 34.1 *Pulmonary symptoms and radiological findings*

Pulmonary symptoms (27 patients)	Percentage
Haemoptysis	56
Dyspnoea	41
Abnormal auscultatory findings	41
Chest pain	22
Cyanosis	19
Radiological findings	**Percentage**
Mild to severe infiltrates	100
Pneumomediastinum	22
Haemothorax	19
Interstitial emphysema	11
Pneumatocele	11
Pneumothorax	4

From Huller and Bazini (1970).

In other reports neurological involvement has been described. Middle-ear damage with hearing loss is common, and abnormalities of consciousness – varying from a mild delirium to coma – have also been reported. The patients may suffer severe headaches and there may be evidence of an upper motor neuron lesion. There may also be interference with the spinal cord and the autonomic nervous system. It is possible that the paralytic ileus, which is quite common in this disorder, is at least partly due to a neuronal reflex phenomenon. Subdural haematomas have also been reported. Pain has been reported in the testes and legs, as well as the abdomen and chest.

A recent fatality occurred in South Africa during a record-breaking attempt for the number of divers underwater at the one time. The 200 divers were reduced to 199 when a thunderflash (a small explosive charge) was used to recall the divers to the surface. This demonstrated the reduced attenuation of blast when propagated underwater.

MANAGEMENT

Blast injuries cause severe body trauma. Hence, the patient must be admitted to hospital for observation,

even though he may not appear seriously affected in the early stages. There are often no external signs of injury, such as bruising or lacerations, despite the internal damage. Exposure to altitude may aggravate or precipitate respiratory difficulties. Gastrointestinal perforations should also be considered before evacuating by air.

In blast injury the patient may not appear to be seriously injured in the early stages, and there may be no external signs of injury.

The patient should have no oral intake and be maintained on intravenous fluids with gastric suction until the full extent of the damage has been assessed. It is easy to suggest that the appropriate studies to ascertain the degree of lung or abdominal injury should be performed, but for abdominal injury there is no clear recent opinion on what these should be. A recent review[3] suggests that computed tomography (CT), if available, may provide evidence of large collections of fluids and haematomas that require operation. Smaller lesions that may require operation can be missed with CT. Intraperitoneal air suggests injury, but this is a non-specific sign as it may not indicate perforation. The review also considers other options. Peritoneal lavage will provide evidence of perforation and bleeding, but will not identify haematomas. Laparoscopic and endoscopic assessment were not to be considered reliable diagnostic tools.

When there are signs of peritonitis, such as rebound tenderness, rigidity or decreased bowel sounds, a decision must be made on surgical intervention and repair. These signs may be due to the haemorrhagic lesions throughout the bowel, affecting the peritoneal cavity, and do not necessarily indicate a perforation. Before surgical exploration, there should be a reasonable presumption of gastrointestinal perforation. Bleeding from the rectum is common, and is not itself an indication for surgery.

Bowel perforations presenting within the first two days are usually ragged, and require resection and anastomosis. Later perforations normally develop from haematomas and are generally clean-cut and may be oversewn.

Management of blast injury is similar to that of severe body trauma from other causes.

Treatment of the respiratory damage is based on general medical principles. Care must be taken in the use of positive-pressure ventilation, although the administration of 100 per cent oxygen may be needed if there is significant hypoxaemia. It may also be of benefit in cases with signs of cerebral or cardiac air embolism.

Antibiotics and tranquillizers may be indicated, but care must be taken to avoid respiratory depression and the masking of symptoms. Intravenous fluids and transfusions should be decided on the clinical state of the patient, and serial haematological and biochemical studies.

If the signs of peritonitis continue despite the above regime, then surgery is probably needed. A perforated viscus may be present in the absence of radiological signs.

Hyperbaric oxygen therapy has been proposed to reduce cerebral oedema, eliminate cerebral bubbles and improve tissue oxygenation. However, except in sophisticated hyperbaric units, it is likely to delay and complicate the cardiopulmonary support and gastrointestinal surgery. If cerebral arterial gas embolism is the dominant feature, it may be essential.

PREVENTION

The obvious measure is to avoid diving in areas where explosions are possible. If this is unavoidable, the diver should wear protective clothing. A dry suit provides the most protection, but an air-containing vest also gives some protection as it reflects a great deal of the pressure pulse at the first water–air interface and absorbs some of the remainder. If it is possible for the diver to reach the surface and float face-up, the effect of the pressure wave will be decreased. If he is near the surface, the pressure wave may be attenuated by reflected waves.

Elevation of the chest and abdomen out of the water reduces the severity of blast injury.

Lifting the chest and abdomen out of the water will lessen the effect of the blast. This is especially so if the swimmer can lie on some piece of debris or solid support. Facing away from the explosion is also said to provide some protection.

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35

The ear and diving: anatomy and physiology

CARL EDMONDS

INTRODUCTION

This section is included because ear problems are the most common occupational diseases of diving, and the diving physician should have a working knowledge of otology. It is not always possible to obtain specialist assistance at an early stage in the assessment of a diving accident, i.e. when effective therapeutic decisions are made.

The ideal combination is a diving physician and an otologist, both of whom have an appreciation of the other's specialty. It is for the diving physician that this section is included.

EXTERNAL EAR

Anatomy

The external ear comprises the pinna and the external ear canal, which captures sound waves and directs them to the middle ear, which is separated from the external ear by the ear drum or tympanic membrane (See Fig. 35.1).

The external ear canal is approximately 3 cm long; the outer one-third is surrounded by cartilage and the inner two-thirds by bone. It is lined by stratified squamous epithelium which tends to migrate outwards, carrying casts of dead epithelial cells, foreign bodies such as dust, and cerumen.

Cerumen or ear-wax forms in the outer one-third of the canal. At body temperature the wax contains fatty acids which become bacteriostatic. It produces a hydrophobic lining which prevents the epithelium from being wetted, becoming soggy and creating a culture medium for infections of the external canal – referred to as **otitis externa** (see Chapter 29). The pH of the external canal lining is usually slightly acidic, which also serves as a bacteriostatic factor.

Interference with the function of the external canal can be produced by removal of the cerumen, either by **syringing of the ear**, long periods of **immersion**, or **traumatic gouging** of the canal with cotton buds, finger nails, hair pins, etc. The mere presence of cerumen or wax should not be an indication for external ear toilet, or syringing. On the contrary, it is likely to increase the degree of subsequent otitis externa if performed before diving.

By immersion, divers can lose most of the cerumen from their ears, and it is rare to see a subject who is in frequent diving practice who has any significant amount of cerumen blockage, unless he wears a hood that prevents the entry of water.

Patients with seborrhoeic dermatitis (often presenting as dandruff) may have episodes of

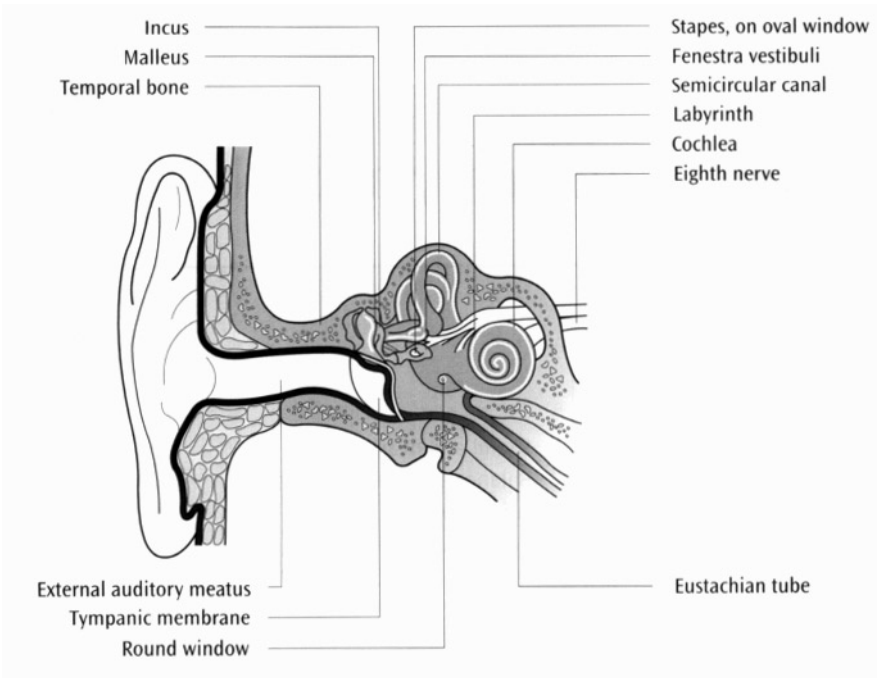

Figure 35.1 *The anatomy of the human ear.*

external-ear itchiness. If they respond to this by scratching the ear, they gouge out furrows of wax and excoriate the skin – breaking the two protective linings. Otitis externa may develop within hours. If the diver refrains from inflicting this trauma, and especially if they treat the inflammation and itch with the use of a non-water based anti-inflammatory steroid ointment (e.g. Ultralan, Kenacomb), then this unpleasant sequence of events does not eventuate.

Patients with an occluding cerumen plug or otitis externa should not dive, but they sometimes do. The obstruction of one external auditory canal may greatly restrict water entry, resulting in asymmetrical caloric stimuli and vertigo while diving, and/or conductive hearing loss on the surface.

A long-term reaction to cold water in the external-ear canal is the development of **exostoses** – usually from three sites around the canal. Being osteomata, they are very hard white masses, and are tightly covered by epithelium. Sometimes they grow to such a size that they occlude the external canal and cause a conductive deafness and may need to be removed by an otological surgeon. Less extensive lesions can still produce problems by interfering with the drainage of cerumen, debris and water – predisposing to otitis externa. Otitis externa may complete the partial occlusion of the canal, initiated by the exostoses.

CASE REPORT 35.1

During a diving medical seminar at Tahiti, a heated discussion ensued on the value of ear syringing to remove cerumen. Of the 44 divers present, only three were required by a Club Mediteranee physician to have cerumen removed from their ears, even though in none of these cases was the external ear obstructed. Otitis externa developed in four ears during the subsequent week, one bilateral and two unilateral – in the three subjects who had their ears syringed. It demonstrated statistically what most of us knew clinically.

MIDDLE EAR

The **tympanic membrane**, which separates the external and middle ears, is a thin but tough and flexible conical membrane. It mirrors pathology of the external and the middle ear.

The **middle ear** is a gas-containing cavity separated from the external ear by the tympanic membrane, and from the inner ear by the round and oval windows. Between the tympanic membrane and the oval window, three small bones are linked together – the ossicular chain, comprising the malleus, incus and stapes. These bones transmit the sound wave pressures on the ear drum across the middle ear and into the fluid-filled inner ear.

The promontory and handle of the malleus can be seen during otoscopy, impinging on the tympanic membrane. The stapes is attached by a strong fibrous band to the oval window. An inward movement of the oval window is transmitted through the inner ear hydraulic system (perilymph) and reflected by a similar but opposite movement of the round window, outwards into the middle ear.

The **middle-ear cleft** extends into the mastoid air cells and the eustachian tube, and includes the middle-ear cavity. It is lined with mucosa which constantly absorbs oxygen from the enclosed space, thereby giving the middle ear a negative pressure (0–20 mm H_2O) relative to the environment. This is intermittently equalized by environmental pressure by the opening of the eustachian tube. This tube is 3.5–4.0 cm long and is directed downward, forward and medially from the middle ear to the nasopharynx. It is lined by respiratory epithelium and is subject to all the allergies, irritants and infections of this system.

The purpose of the eustachian tube is to aerate the middle ear and maintain equal pressures across the tympanic membrane – between the middle ear and the environment. At the potential nasopharyngeal opening the pressure is at environmental or ambient levels, whereas the middle ear opening of the eustachian tube is at middle-ear pressure. If there is a blockage of the eustachian tube, the middle ear cannot be aerated regularly, and therefore a significant negative pressure develops at a rate of 50 mm H_2O per hour, as oxygen is absorbed from this cavity by the mucous membrane. This results in a retracted tympanic membrane and/or an effusion, both of which will partly reduce the negative pressure.

The eustachian tube usually opens once a minute while awake, and every 5 minutes when asleep. Swallowing and yawning will commonly open this tube and allow replenishment of the gas to the middle ear from the nasopharynx, without any active or artificial over-pressurization.

In divers, the eustachian tube may be opened actively by increasing the pressure in the nasopharynx by 50–250 mm H_2O in excess of the pressure in the middle ear (see Chapter 7). The gas then passes from the nasopharynx into the middle ear, alleviating the middle-ear negative pressure produced when the diver descends. If equalization of the pressures is not achieved during descent, by the time a gradient of 400–1200 mm H_2O is reached, this pressure may produce a locking effect on the valve-like cushions of the eustachian tube, preventing further active or passive opening.

If the pressure in the middle ear exceeds that in the nasopharynx by 50–200 mm H_2O, then the eustachian tube usually opens passively and gas passes from the middle ear into the nasopharynx. This occurs when the diver ascends towards the surface.

The pressures above are pressure differences between the spaces mentioned. These should not be directly extrapolated to water depths, as there is considerable tissue movement and distortion which reduces some of the pressure gradients of ascent and descent. Thus, a diver with a very mobile tympanic membrane and a small volume middle-ear cleft, would be able to tolerate a much greater descent without experiencing any change in middle-ear pressures. In practise it is found that if the eustachian tubes are not open, the diver may experience some subjective sensation at a depth of 0.3 metres H_2O. Discomfort or pain may be felt as shallow as 2 metres, and this is also the depth at which the 'locking effect' can develop and the tympanic membrane may rupture from a dive to 2–10 metres.

Abnormalities of eustachian tube function in divers or other groups exposed to variations in pressure result in middle-ear barotrauma and indirectly in inner-ear barotrauma, with mucosal and membrane haemorrhage and ruptures. These clinical

entities are dealt with under barotrauma (see Chapter 7), and in the differential diagnosis of deafness and disorientation, (see Chapters 37 and 38).

INNER EAR

The inner ear is composed of the cochlea, a snail-like structure responsible for hearing, and the vestibular system responsible for the orientation of the animal in space, i.e. appreciation of acceleration, equilibrium, balance and positioning. (Fig. 35.2).

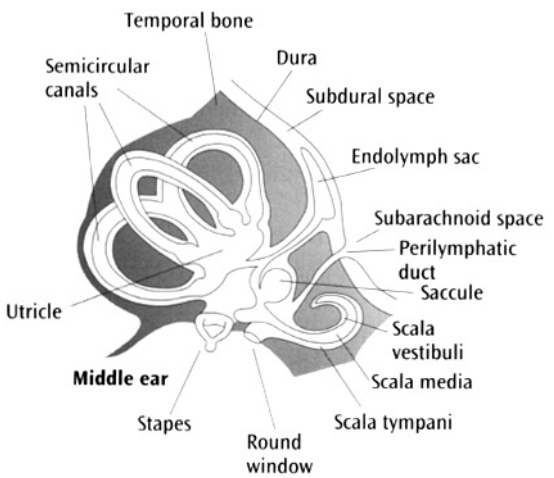

Figure 35.2 *Anatomy of the inner ear.*

Sound and hearing

The hearing part of the inner ear, the cochlea, is composed of three adjoining tubes or scala:

- The scala vestibuli (or inner tube), connected to the middle ear by the oval window membrane and the foot plate of the stapes. This contains perilymph, which is similar in composition to extracellular fluid.
- The scala tympani (or outer) tube, connected to the middle ear via the thin and fragile but flexible, round window membrane. This also contains perilymph, which is continuous with that of the vestibuli. The vestibuli and the tympani communicate at the apex, the helicotrema.
- The media (or middle) tube, lies between the other two channels, demarcated from the vestibuli by a fragile Reissner's or vestibular membrane. This membrane separates the fluid of the two tubes, but is so thin that it does not impede the pressure waves (sound) passing between them. Between the media and the tympani, there is the much thicker basilar membrane, which does impede sound waves and which supports the 25 000 reed-like basilar fibres, which are connected to the sound receptors called hair cells. Each sound frequency causes a vibration at a different sector of the basilar membrane. Near the oval and round windows the basilar fibres are short and rigid, and are sensitive to high-frequency sounds, whereas further up the cochlea they become longer and more flexible and respond to low-frequency sounds. Damage to specific areas produces a specific hearing loss. The fluid within the media – endolymph – is similar in composition to intracellular fluid.

The integrity of the above membranes, together with the different composition and electrical potential of the inner ear fluids, are required for normal hearing.

Sound is caused by the vibrations or pressure waves at frequencies between 20 and 20 000 cycles per second (20 to 20 000 Hertz), transmitted to the ear via air or water.

The **external ear** directs the sound to the tympanic membrane, which vibrates and transmits these vibrations through the ossicular chain of the middle ear to the oval window and thence to the cochlea fluid of the inner ear, distorting the basilar membrane and triggering off the nerve impulses from the hair cells (transducers). Thus, pressure waves are converted into electrical impulses which are transmitted to the brain via the eighth (auditory) nerve, where it is identified and interpreted as sound. The pressure wave disperses by distending the round window into the middle ear. Once the sound stops, these structures resume their normal position.

The hair cells of the cochlea may also be stimulated by vibration of the bones of the skull. Sounds that cause this vibration and are heard in this manner are transmitted by bone conduction. This method of hearing is less sensitive than the air conduction pathway, but it is of importance for hearing

underwater. It is the pathway by which people with external and middle-ear damage can still hear.

A perilymphatic or cochlear duct of variable size may connect the perilymph fluid to the cerebrospinal fluid (CSF) in the brain's subarachnoid space. This also allows transmission of pressure between the two hydraulic systems, and explains why Valsalva manoeuvres, coughing, etc. can cause a pressure wave in the perilymph, transmitted from intrathoracic pressure to the venous system and the CSF.

Symptoms of cochlea impairment include tinnitus, dysacusis (Chapter 37) and hearing loss.

Balance and spatial orientation

The balance organ of the inner ear is called the vestibular apparatus, and includes three semicircular canals, being positioned at right-angles to each other. They measure movement of the enclosed fluid in any plane, by end organs (transducers). The semicircular canals detect change of movement of the head, and supply this information by nerve impulses to the central nervous system.

These canals connect with the utricle and the saccule, which are ballooned fluid-filled cavities, also at right-angles to each other. They contain otoliths (ear stones) – sensory end organs (also transducers) that are able to detect movement of fluid and are also influenced by gravity. They supply gravitational information of the position of the head in space.

The vestibular system is very dynamic, with constant input from all areas at all times. The end organs fire electrical discharges even in the resting state. If there is a stimulus in one area, then there should be a corresponding stimulus in a complementary area on the other side, otherwise conflicting messages are received by the brain. These impulses, giving information regarding position and movement in space, are transmitted to the brain by the vestibular part of the eighth cranial nerve, to the brainstem and finally to the cerebellum, where balance is monitored, coordinated and interpreted.

The membranous labyrinth is somewhat like an inflated inner tube of an automobile tyre, and contains endolymph which also fills the scala media. Around the membranous labyrinth there is perilymph, which also fills the scala vestibuli and tympani, and is connected to the CSF by the perilymphatic duct.

Vestibular damage may lead to symptoms of disorientation and imbalance, vertigo and associated vagal complaints such as nausea, vomiting, syncope, etc. Nystagmus, the clinical sign that corresponds to symptomatic vertigo, is a typical flickering eye movement with a slow component moving the eyes to one side, and a fast or 'correcting' component bringing the eyes back to their original position. With an irritative lesion in one vestibular system, the nystagmus (fast component) is to that side. A destructive lesion causes it to go to the opposite side. Nystagmus, like vertigo, can be suppressed by other sensory input, by cortical inhibition, and also by some drugs.

If the vestibular system is damaged irreparably, the clinical features may diminish over the subsequent weeks or months, as the brain accommodates to the inequality by suppressing responses from the undamaged side (cerebral inhibition). Nevertheless, the damage can still be demonstrated by provocative tests (e.g. caloric electronystagmograms) or movements (sudden head turning). If the damage is more central, such as in the cerebellum, then nystagmus may persist even though the vertigo can be partly inhibited.

Under normal circumstances, balance control is characterized by much redundancy, and so one area of the visual/proprioception/vestibular triad may be damaged without inevitable imbalance or ataxia.

CLINICAL OTOSCOPY

Visual inspection of the external ear canal and tympanic membrane is one of the more valuable clinical examinations to be made on diving candidates. Otological disorders such as cholesteatoma may be diagnosed, or there may be a condition such as otitis externa which is aggravated by diving, or a perforated tympanic membrane which makes diving unsafe. An external ear blockage predisposes to otological diving problems, and external and middle-ear barotraumas are identifiable diving diseases. As well as identifying these clinical disorders, a major value of otoscopy is the verification of a successful middle-ear autoinflation under voluntary control (see page 81).

The tympanic membrane is viewed while the candidate attempts to inflate the middle ear by active manoeuvres (commonly one ear is easier to autoinflate than the other). Physicians experienced in diving medicine will spend considerable time ensuring that diving candidates understand the techniques and sensations experienced during middle-ear autoinflation. Inadequate middle-ear autoinflation is responsible for the most common and preventable of diving accidents – middle-ear barotrauma of descent.

In one Australian series the tympanic membrane was viewed initially in 87 per cent of diving candidates, with 84 per cent being mobile during the autoinflation. Of the remaining 13 per cent, 12 per cent claimed subjective autoinflation. The otoscopic view was obstructed by cerumen in 9 per cent, an unusual canal alignment or structure in 3 per cent, and exostoses in 1 per cent.

Pneumatic otoscopy does demonstrate the mobility of the ear drum, but is of no value in verifying the voluntary control of autoinflation that is required by divers.

RECOMMENDED READING

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Farmer, J.C. (1993) Otological and paranasal problems in diving. In: Bennett, P. and Elliott, D. (eds). *The Physiology and Medicine of Diving*. 4th edition. London: W.B. Saunders.

Freeman, P. and Edmonds, C. (1972) Inner ear barotrauma. *Archives of Otolaryngology* **95**, 556–563.

36

The ear and diving: investigations

CARL EDMONDS

INTRODUCTION

Otological investigations will vary between different clinics depending on their sophistication, skills and clinical orientation. Here, we have tried to confine ourselves to those that have been found to be of value in clinical diving medicine

Simple tests of hearing are outlined in Chapter 37, and of balance in Chapter 38, and are applicable in remote areas. A specialized diving medical unit will employ pure tone audiometry, impedence audiometry and electronystagmograms (ENG). Otological clinics will utilize far more specialized procedures.

PURE TONE AUDIOMETRY

This measures the ability to hear pure tones in octaves between 125 and 8000 Hz (cycles per second). The hearing loss in each frequency is measured in decibels (dB) – a logarithmic scale which means that a loss of 10 dB represents a ten-fold change in intensity of the noise, 20 dB a 100-fold, 30 dB a 1000-fold, etc. Testing is done initially for air conduction, but if this is significantly impaired, then bone conduction should be measured. With sensorineural deafness, the loss is the same for both air and bone conduction.

If bone conduction is adequate and normal, then it infers that the cochlea is picking up sound waves transmitted via the bone, and therefore the sensorineural component of the hearing is normal. If there is a considerable discrepancy, with the air conduction being poor but the bone conduction normal, then this suggests that there is a 'conductive' deafness, i.e. involving the external or middle ear (Fig. 36.1).

A very quiet area, usually a soundproof audiogram booth, is necessary for reliable testing. With a severe unilateral deafness, sound levels of 40–60 dB can be heard by cross-hearing, and therefore masking is needed in the good ear while the damaged one is being tested above this level. As a general rule conductive deafness tends to be first noted in the lower frequencies, (250–3000 Hz), and the sensorineural damage is first noted somewhere in the 4000–8000 Hz range. If there is serious and significant nerve damage then all frequencies may be involved.

More elaborate audiology, utilizing such devices and techniques as recruitment, brainstem and Bekesy audiometry, SISI and pure tone decay are used by otologists – but these are of little interest to the diving physician. Speech discrimination testing is of value if there is distortion of hearing.

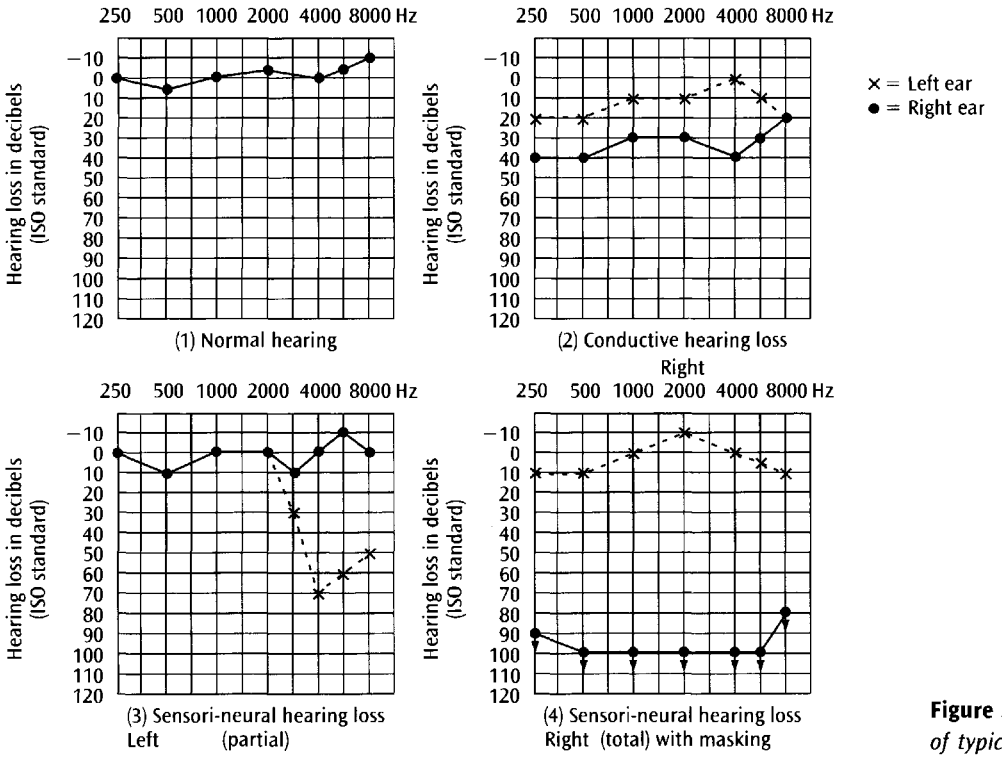

Figure 36.1 *Examples of typical audiograms.*

The most valuable of all ear function tests is the comparison of repeated pure tone audiograms. If these measurements are made before the diver commences his hyperbaric exposures, and routinely during his diving career, then cumulative damage can be readily identified. This comparison should also be performed immediately if there is any subjective complaint of hearing loss, or evidence of inner-ear disorder such as tinnitus or vestibular symptoms. Once evidence of sensorineural impairment is found, then serial audiometry should be performed to ensure that therapeutic measures are having their desired effect.

TYMPANOMETRY

Indirect assessments of the function of the middle-ear cavity and the eustachian tube are obtained from many different otological investigations. Techniques to measure directly the middle-ear pressures and movements are now available to clinicians.

The tympanometer (impedance audiometer) is designed to facilitate the quick, objective testing of middle-ear function, measuring the mobility of the middle-ear system (usually called compliance), the pressure within the middle-ear cavity and the variation in middle-ear mobility reacting to variations of pressure from −300 to +300 mm H_2O applied in the external ear. It can also react to a voluntary autoinflation of the middle ear through the eustachian tube – and herein lies its specific value to the diving clinician.

A graph is produced on an X-Y plotter and is termed a tympanogram, with the peak height on the Y-axis showing the compliance, and its position on the X-axis showing the middle-ear pressure. (Fig. 36.2). The middle-ear pressure can be changed with successful middle-ear autoinflation manoeuvres.

The middle-ear pressures, recorded as the position of the tympanogram peak on the X-axis, changes with different diving environments and accidents. There may be a decrease of middle-ear pressure when 100 per cent oxygen is breathed, or an

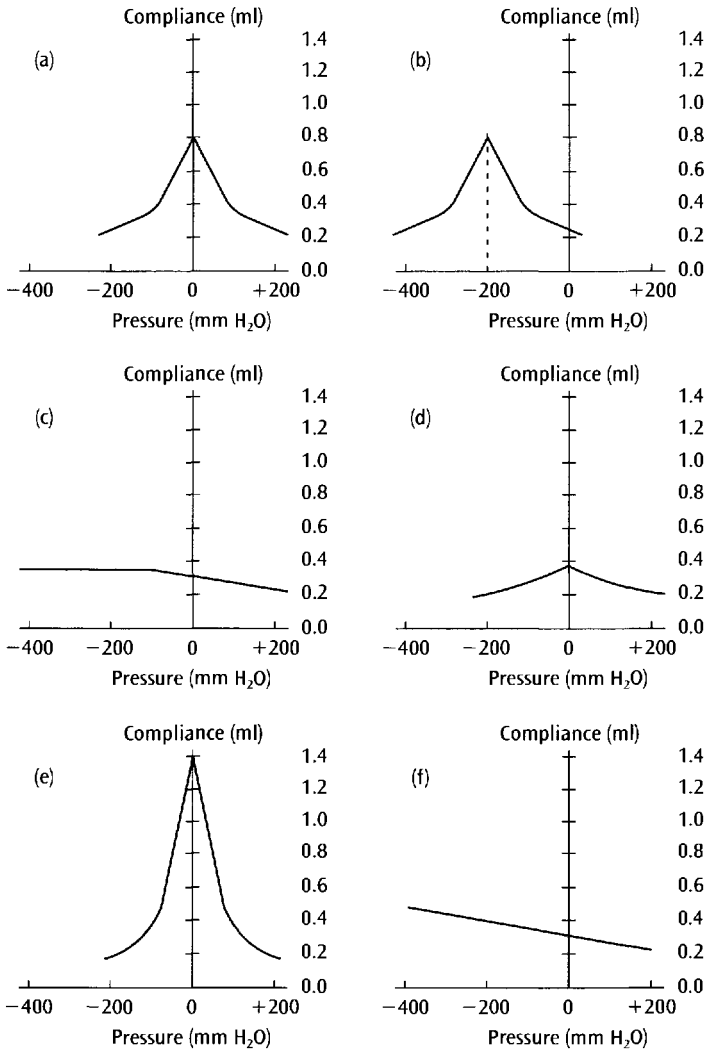

Figure 36.2 *Diagrammatic tympanogram responses: (a) normal; (b) negative middle-ear pressure, e.g. eustachian tube obstruction; (c) fluid-filled, middle-ear cavity, e.g. haemorrhage from severe middle-ear barotrauma; (d) reduced compliance, e.g. otosclerosis or mucosal congestion in the middle ear from barotrauma; (e) increased compliance seen with disarticulation of the ossicles, scarred or very mobile tympanic membranes;(f) perforated tympanic membrane.*

increase in middle-ear pressure when helium/oxygen (heliox) is breathed. In the first case nitrogen is lost and oxygen is absorbed from the middle ear, and this is one of the causes of serous otitis media with oxygen-breathing divers. The increase in middle-ear pressure while breathing heliox is due to the helium diffusing into the middle ear faster than the nitrogen moves out.

Pathological conditions that may cause a **positive pressure peak** are: acute otitis media in the very early stages; middle-ear barotrauma of descent (after subsequent ascent); after haemorrhage and exudation in the middle ear; and during over-pressure from middle-ear barotrauma of ascent (Fig. 36.2).

A **negative pressure peak** may develop with middle-ear barotrauma of descent, while still at depth. Negative pressure peaks are related to poor eustachian tube function and associated absorption of gas from the middle-ear cavity. This may result in effusion, and if the middle ear contains a significant amount of fluid, the pressure peak may disappear and the tympanogram becomes flat.

Middle-ear barotrauma of descent, producing congestion and swelling within the middle ear will

produce a decreased amplitude of the peak of the tympanogram, demonstrating a reduced compliance. This rises as the structures and lining of the middle ear return to normal.

With perforation of the tympanic membrane, no pressure gradient can exist across it, and therefore there is no peak. This information is used clinically to show the patency of grommets inserted into the tympanic membrane, but it is also of value in demonstrating perforated tympanic membranes which cannot be seen otoscopically.

Tympanograms with increased amplitude or notches, may be due to an ear drum abnormality or ossicular discontinuity. An increased amplitude or notching occurs with healed perforations, and a M-shaped pattern can be produced. Ossicular chain discontinuity may cause a high peak, deep or multiple notches.

The presence of a respiratory wave in the tympanogram implies a continuously open (patent) eustachian tube, not uncommon in experienced divers, and an occasional sequelae of middle-ear barotrauma, especially with over-forceful Valsalva manoeuvres.

A way of verifying inner-ear fistula which is currently under investigation integrates an impedance audiometer and an ENG, with both horizontal and vertical leads. The induction of nystagmus during the external-ear pressure change may give some support to the diagnosis, but should only be performed if facilities exist for immediate inner-ear surgery – if the procedure enlarges the fistula. This application remains only a research tool at this stage and is not recommended for general usage.

Diving Tympanogram *(Edmond's technique)*

For voluntary autoinflation assessments on divers, a tympanogram is performed, with the pressure in the external ear passing from −300 to +300 mm H_2O, and this is used as a control. The pressure in the external ear is then moved to zero, and a Valsalva manoeuvre is performed by the subject. A tympanogram is then repeated, moving from −100 to +300 mm H_2O. If the Valsalva has been successful this will demonstrate a significant movement of the peak, illustrating the increase in the middle-ear pressure because of the Valsalva forcing more air into this cavity. On completion of this second tympanogram, at +300 mm H_2O the subject is then asked to perform the Toynbee manoeuvre and the third tympanogram is recorded, passing from +300 mm H_2O to −300 mm H_2O. If the Toynbee manoeuvre is successful, then the peak of the tympanogram will have returned towards the control or even beyond it to indicate a reduction of middle ear pressure (Fig. 36.3).

This allows an objective way of measuring the effects of autoinflation and deflation on the middle ear during simulated conditions of descent and ascent. This technique is somewhat different to the conventional one. The modification ensures that inadvertent opening of the eustachian tube, and consequent release of post-Valsalva pressure, is not caused by the excess external-ear pressure induced during the traditional procedure.

The impedance audiometer is a valuable instrument for use in both the clinical and research aspects of diving medicine.

VESTIBULAR FUNCTION TESTING

When there is an imbalance in the neural activity from the vestibular systems, involuntary eye movements develop, and these are termed 'nystagmus'. This is the physical sign, which corresponds to the sensation of vertigo.

Spontaneous nystagmus is an important sign of vestibular disorder. Positional vestibular testing refers to the effects of certain head, neck and eye positions in producing abnormal vestibular responses in some patients.

The Hallpike caloric tests were the traditional tests used to demonstrate the activity of the vestibular system stimulated by water at temperature of 30° and 44°C, i.e. 7°C above and below body temperature, with the subject lying supine and head elevated to 30 degrees. The temperature change causes convection currents in the horizontal or lateral semicircular canal, and this stimulates the vestibular system. The response to hot and cold water on both sides is compared, and conclusions are drawn as to whether there is unequal function of the two vestibular systems to identical stimuli and whether there is preponderance

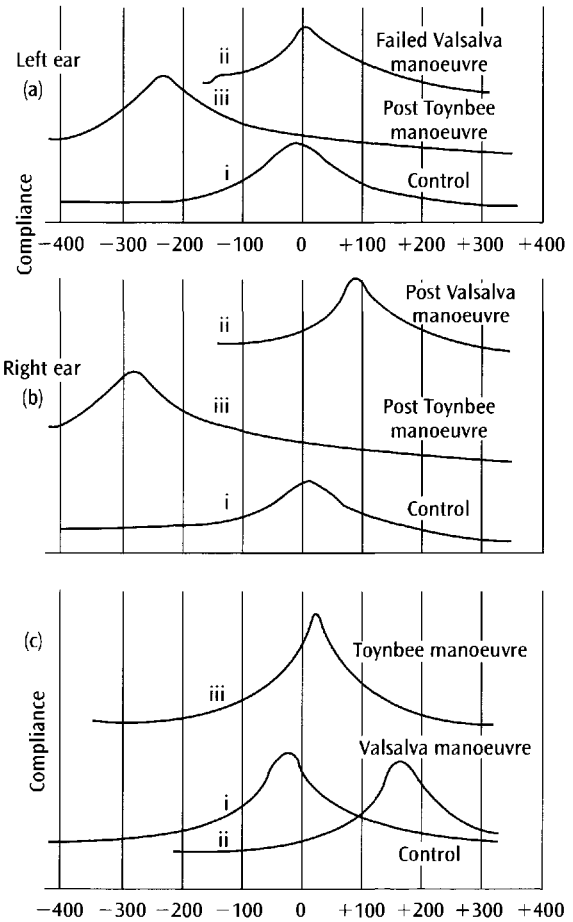

Figure 36.3 *'Diving' tympanogram: the tympanometry demonstration of the effect of Valsalva and Toynbee manoeuvres on middle-ear pressures. (a) Left ear: (i) normal control tympanogram, passing from –400 mm H_2O on the left to +300 mm H_2O on the right (note: opposite to the conventional technique). (ii) Valsalva manoeuvre attempted at the neutral position (peak of control tympanogram) and a repeat tympanogram from –100 mm H_2O to +300 mm H_2O shows no substantial movement of the peak, i.e. middle-ear pressures were not influenced by the procedure. The Valsalva manoeuvre failed! (iii) Toynbee manoeuvre performed at +300 mm H_2O with the subsequent tympanogram showing a 225 mm H_2O negative pressure being produced in the middle ear. The Toynbee manoeuvre was successful!*
(b) Right ear: (i) normal control tympanogram; (ii) Valsalva manoeuvre produced a 80 mm H_2O positive pressure in the middle ear; (iii) Toynbee manoeuvre at +300 mm H_2O produced a 280 mm H_2O negative pressure to the middle ear. The Toynbee manoeuvre was successful. Note: for patient tolerance, the technique has been modified to extend between –300 and +300 mm H_2O.
(c) Diving tympanogram: performed as above. This tympanogram demonstrates an increase in middle-ear pressure from –25 mm H_2O (normal) to +175 mm H_2O with Valsalva manoeuvre, and then a partially successful Toynbee manoeuvre – reducing the middle-ear pressure to +25 mm H_2O).

of responses to one side. The first suggests a peripheral lesion, and the second a more central one.

In the event of no response from one side, greater stimuli may be given, e.g. iced water calorics (more practical than the standard calorics under most operational hyperbaric conditions) or even drug provocation. With peripheral vestibular lesions, the eye movements are sometimes best observed by having the subject close his eyes and perform distracting exercises such as mental arithmetic. The eyes can then be seen rolling with fast and slow components, beneath the closed lids. These primitive techniques of assessing vestibular function in the field are rarely needed nowadays. Vestibular function assessment has been revolutionized by the addition of the ENG to detect spontaneous nystagmus and the effects of both positional and caloric provocation.

Electronystagmogram (ENG)

If it is measured clinically, i.e. by observation or by the use of Frenzel's glasses, nystagmus is a crude sign and is associated with many false-positive and false-negative results. If it is measured by ENG, then it is not only a quantitative measure of the ocular movement, but it is also a sensitive measurement of vestibular dysfunction. The ENG is far more sensitive than the clinical sensation of vertigo, and therefore depicts subclinical levels of vestibular disease. It may demonstrate abnormality long after the symptoms have disappeared.

The recordings are measurements of movement of the eye causing a deflection on their electrical field. This is possible because the retina has a negative charge compared to the cornea, and as the eyes

move so does the electrical field, detected by electrodes adjacent to the eyes. In some units, infra-red tracking devices in a dark room have now replaced electrical leads to measure eye movement.

In clinical practise the conventional ENG is a graphic record of vestibular function made during positional tests and also during caloric stimulation – the Hallpike tests referred to above. Thus, the horizontal electrodes are commonly used. In diving research it is recommended that the tracings include both horizontal movement and vertical.

By the use of this very valuable and objective test, vestibular dysfunction can be demonstrated. The duration of the nystagmus can be observed, reviewed and measured, and the degree of nystagmus can be quantified by different parameters (rate, height and deflection of the slow and fast components). The vestibular responses can be stimulated in a provocative but selective manner by the use of the Hallpike caloric test, positional tests, and even changing pressures – by altering the subject's ambient pressure in recompression and decompression chambers.

Vertigo and nystagmus can be induced in some cases of vestibular pathology (especially perilymph fistulas) by a sonic stimulus to the affected ear (equivalent to the Tullio phenomenon) or by a subsonic stimulus, e.g. pressure from an impedance audiometer to the inner ear (similar to the basis of Hennebert's sign).

Stress factors may be used to increase the sensitivity of the ENG to demonstrate perilymph fistulae. In these cases either pressure (tympanometry) may be applied to the external canal (Hennebert's sign), or a loud noise of 95 dB at 500 Hz (Tullio phenomenon). Alternately, body sway-type tests can be used. The frequencies of the stimuli can be reduced to those more likely to have vestibular effect, such as 50 or 25 Hz, and the intensity can be increased to 130 dB, with the patient standing on a platform to measure postural stability during low-frequency stimulation.

The ENG has revolutionized our approach to vertigo and its relationship to diving, and has allowed us to clarify and verify clinical impressions, often demonstrating significant pathophysiology in cases which may otherwise have been classified as functional. It has an occasional application as an electrodiagnostic procedure in recompression chambers, with the electrode leads under pressure and the equipment outside the chamber, to assist in diagnosis and to monitor the effects of therapeutic intervention such as recompression and gas mixture alterations. Infra-red tracking may be preferred in the future.

ENGs may be used to differentiate end organ (vestibular) disease from central (brain) causes of vertigo and dizziness. The ENG of peripheral vestibular end organ disease produces a spontaneous nystagmus greater than 7 degrees per second, due to a reduction in the tonic discharge from the affected vestibule. The nystagmus (fast phase) is thus toward the side of increased vestibular activity. It is strongest when the subject looks to the same side as the fast phase. It is suppressed by visual fixation (more than 40 per cent) and augmented by eye closing and mental concentration (e.g. mathematical calculations).

Sophisticated computerized balance and movement tests are used in many balance-disorder clinics.

RADIOLOGY AND SCREENING TECHNIQUES

The value of plain X-rays of the otological area is limited to demonstration of middle-ear and mastoid cavities, especially in assessing the safety of deaf divers to undergo training.

More recently the use of computed tomography, magnetic resonance imaging and other scanning techniques have allowed identification of more anatomical and pathological structures. These have been used to demonstrate the degree of barotraumas, the integrity of the round window membrane, and gas and haemorrhagic lesions in the inner ear.

Brain scans and arterial Doppler measurements are applicable in some differential diagnoses.

Future developments in this field are expected, but in practise the value of the sophisticated scanning techniques is limited at this stage.

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37

The ear and diving: hearing loss

CARL EDMONDS

CLINICAL FINDINGS

The symptom of deafness is one of the most common encountered in diving medicine. In many cases, the complaint is not supported by conventional audiometric testing. The diver may be referring to a feeling of 'fullness' or 'blockage' within the middle ear, these sensations being associated with congestion and swelling of the tympanic membrane and mucosal lining of the middle-ear cavity. It is commonly due to middle-ear barotrauma of descent, and frequently resolves within a week or so.

Objective hearing loss may be of four types (Fig. 36.1):

- Conductive due to problems of the conductive apparatus (external ear, tympanic membrane, ossicles, middle ear) (this is common).
- Sensorineural – when the problem lies in the cochlea or the auditory nerve (this is less common, but very important).
- Central – due to lesions of the auditory pathways in the brain, central to the cochlear nuclei (this is rare, as the hearing centres are bilaterally represented).
- Mixed – various combinations of the above.

The type of hearing loss can be inferred from the **patient's speech**. Soft but articulate speech associated with a moderately severe hearing loss is suggestive of conductive deafness, whereas loud clear speech suggests an acquired bilateral sensorineural hearing loss. Slurred, hesitant, inarticulate and inaccurate speech is more compatible with central lesions. Impaired speech discrimination may be obvious during the interview.

The otological examination may include the **tuning fork tests.** The Rinne test becomes abnormal when the fork is heard louder by bone conduction (fork held on the mastoid bone) than air conduction (tines of fork placed over the auditory meatus). This indicates a 'conductive' deafness. A Barany noise box may 'mask' the other ear during the tests, which may extend between 64 and 4096 Hz forks.

The Weber test is performed with a 256- or 512-Hz fork, which is placed on the vertex of the skull; the subject then indicates in which ear he hears the fork.

He will hear it in the affected ear with conductive deafness, the unaffected ear with sensorineural deafness, or both ears in mixed or bilateral deafness.

The Schwarbach test is performed by comparing the patient's and the examiner's ability to hear the tuning fork tests by bone conduction.

Operational hearing tests

In remote environments, use can be made of even more primitive techniques. The ticking or alarms from a wrist watch has a reliable sound intensity and frequency, the latter commonly being about 4000–6000 Hz – this is the region that is less noticeable to the diver than the usual voice frequencies of 250–2000 Hz. Comparison is made between the diver's hearing ability in both ears and the examiner's hearing. The finding that the watch can be heard only in one ear, or that the hearing acuity for the watch ticking is below that of the examiner's, suggests that pure tone audiometry is required. For divers who have subjective sensations of 'dullness', the hearing of the watch gives some reassurance to the examiner that a gross sensorineural loss is less likely.

As a rough general indicator, for use in remote or isolated areas, an approximation of the diving accident victim's hearing can be made by:

- *speech;*
- *associated symptoms (tinnitus, vertigo, etc.);*
- *hearing tests with watch and whispered speech; and/or*
- *'tuning fork' tests – Rinne, Weber and Schwarbach.*

The type and existence of hearing loss is confirmed by audiometric testing (see Chapter 36). Most value is obtained by comparison of the diver's post-incident audiograms with those performed prior to diving training, or previous annual audiograms if he is a frequent or professional diver. If hearing loss is demonstrated, bone conduction should be performed. A classification of the types of hearing loss can be made, based on whether it is predominantly conductive or sensorineural (see Chapter 36).

CLASSIFICATION OF HEARING LOSS IN DIVING (Edmonds *et al*, 1973)

Conductive

Conductive hearing loss may be due to:

- External ear obstruction; this may be due to
  - cerumen;
  - otitis externa; and/or
  - exostoses.
- Tympanic membrane perforation; this may be caused by
  - middle-ear barotrauma of descent;
  - forceful autoinflation; and/or
  - shock wave.
- Middle-ear cleft disorder; this may be caused by
  - middle-ear barotrauma of descent;
  - otitis media;
  - forceful autoinflation;
  - increased gas density in the middle ear; and/or
  - ossicular disruptions.

Sensorineural

Sensorineural hearing loss may be due to:

- Noise damage;
- Decompression sickness; and/or
- Inner-ear barotrauma.

Other causes

Dysacusis (painful hearing) has a variety of causes (see below).

CONDUCTIVE DEAFNESS

External ear obstruction

This is not uncommon in divers, and is easily diagnosed both on history and on otoscopic examination. It may also induce vertigo under certain circumstances (see Chapter 38). The causes are as follows:

- **Cerumen plug:** conductive deafness may result from the movement of cerumen in the external ear, due to gas space alterations with diving. Water may cause swelling of the desquamated tissue and cerumen, possibly obstructing an external canal which was already partly blocked.
- **Otitis externa:** Infection (see Chapter 29) is aggravated by immersion of the ear in water.
- **Exostoses:** These (see page 366) may be single or multiple. Although rarely causing complete obstruction of the external canal by themselves, this may develop in the presence of cerumen or otitis externa.

Tympanic membrane perforation

This is a not uncommon complication of diving, but there is usually a well-defined reason. The patient may complain of a feeling of air hissing through the tympanic membrane, especially when he autoinflates the middle ear.

Caloric stimulation may produce transient vertigo underwater (see page 388). The hearing loss is conductive, but usually only 5–15 dB, with the lower frequencies being most affected. Hearing loss is rectified when the tympanic membrane is almost closed. This may take hours to a few days with small perforations, or weeks with larger ones.

Clinical management includes keeping the ear dry. Divers are advised not to aggravate the problem by further pressure changes, e.g. flying, diving, nose-blowing, or middle-ear autoinflation (which divers with ear symptoms tend to do). Prophylactic oral antibiotics may be indicated. In some cases the diver may become aware of the perforation only at a later date, e.g. when returning from a diving expedition or after performing a Valsalva manoeuvre.

The causes of perforation of the tympanic membrane during diving include:

- Middle-ear barotrauma of descent (see Chapter 7).
- Forceful autoinflation. The Valsalva manoeuvre may cause perforation of the tympanic membrane, especially if it has been damaged previously, e.g. by middle-ear barotrauma, otitis externa, old perforations, etc.
- Shock wave. This may be due to an underwater explosion, or the pressure wave from another diver's fin. These cases often develop vertigo (see page 389), and there are often no signs of middle-ear barotrauma of descent on otoscopy.

Middle-ear disease

Dullness of hearing, dampening of sound, deafness and 'fullness' or 'blockage' within the ear, are common symptoms reported by divers. The most common cause is middle-ear barotrauma of descent.

Middle-ear diseases associated with diving are as follows:

- **Middle-ear barotrauma of descent** ('ear squeeze') (see page 75).
- **Otitis media:** the hearing loss, in cases in which the infection does not extend into the inner ear, is of the conductive type. Infection may follow contamination of the middle ear with nasopharyngeal organisms, introduced with autoinflation. It may progress because of the culture media of blood and effusion, caused by middle-ear barotrauma of descent. The patient usually presents with pain which has developed within 6–24 hours of a dive. Prognosis and treatment are based on general medical principles (see Chapter 29).
- **Forceful autoinflation of the middle ear:** this is associated with middle-ear barotrauma of descent, and may be an indirect cause of some of the damage noted with this disorder. Damage may be to the tympanic membrane, ossicles, oval window, cochlea or round window. The last three may result in a sensorineural deafness. Dysacusis may also develop (see below).
- **Increased gas density:** with exposure to increased pressure and increased depth, more gas molecules pass into the middle ear to maintain its volume. The gas is more dense – causing a depth-related, reversible hearing loss, due to increased impedance of the middle-ear transformer. This explains some of the hearing loss noted in recompression chamber experiments and helmet diving. The use of helium, which will reverse this increased gas density, unfortunately changes the middle-ear

resonant frequency, thereby also interfering with normal hearing. Underwater, bone conduction is the major hearing mechanism.

- **Ossicular disruption:** this may follow middle-ear barotrauma and may also cause a mixed hearing loss. It is identified on the tympanogram (see Chapter 36).

Hearing loss in diving may be either conductive of sensorineural, the latter being more serious, the former more common.

SENSORINEURAL DEAFNESS

Noise-induced deafness

Extreme noise exposure may result in temporary or permanent threshold shifts that present as a sensorineural deafness and which is usually bilateral and partial. This may be experienced in compression chambers, with diving helmets, compressors, etc.

The hearing loss is confined almost entirely to frequencies higher than that of the offending noise, the greatest shift being for tones about half an octave above the exposure tone, but all of the higher frequencies may be more or less affected. Hearing for lower tones remains almost unaffected.

Exposure to a 1000-Hz signal at 120 dB (the threshold of discomfort) for 30 minutes usually causes a temporary threshold shift of about 35 dB over the upper half of the speech range. These changes are normally reversible, but the frequencies which are most vulnerable and least likely to recover are those between 3000 and 6000 Hz.

Frequencies between 3000 and 6000 Hz are particularly sensitive to noise-induced deafness.

Repeated exposure to loud noise may cause a permanent threshold shift in which the multiple threshold shifts summate, especially if there is little time between the exposures. Prolonged and repeated exposure to noise is also likely to occur in attendants who work with compressors and compression chambers. Under these conditions it would be expected that the noise-induced deafness would be bilateral.

Divers in the armed services, are often trained in gunnery and explosives – both are related to conditions that might predispose to sensorineural deafness, which may be unilateral.

Pre-existing sensorineural deafness seems to sensitize the ears to further damage from other causes.

Decompression sickness

Sensorineural hearing loss in relation to decompression sickness is outlined in Chapter 12.

Inner-ear barotrauma

Sensorineural hearing loss in relation to inner-ear barotrauma is outlined in Chapter 7.

Pure tone audiograms should be performed on all divers before exposing them to hyperbaric conditions. Without these pre-incident audiograms an assessment of hearing damage would be most difficult, especially as it predominantly involves the asymptomatic high frequencies in mild or early cases. In mild sensorineural deafness, unlike the conductive deafness that interferes with speech frequencies, the diver may not be aware of the deafness.

DYSACUSIS

This word, which has been used to denote 'faulty' hearing, does not include hearing loss, but the terms are not mutually exclusive – both may exist from the one cause. In diving medicine the most common types of dysacusis are painful hearing and echo hearing.

Dysacusis, presenting as **painful hearing**, may follow injury to the inner ear from noise damage,

secondary to middle-ear barotrauma, or due to successful but excessive Valsalva manoeuvres. There may be an associated hearing loss, either temporary or permanent. The pain, which is usually associated with loud noises, may persist for a variable time.

Any excessive noise may produce pain, especially the sound levels of 120 dB or greater. This is possible in either helmet diving or in compression chambers. It has been known to damage the vestibular part of the inner ear and produce the **Tullio phenomenon** (vertigo and nystagmus associated with excessive noise stimulation).

A dysacusis effect from a **patulous eustachian** tube may present as an echo, excessive awareness of the patients respiration or his speech (causing him to speak softly), or the reverberation of sounds such as his footsteps. The eustachian tube may be overstretched and made 'patulous' by the excessive pressure employed with the Valsalva manoeuvre. Other medical causes should be excluded.

The clinical otoscopic sign of tympanic membrane movement during respiration is considered pathognomonic of a patulous eustachian tube in the general population. This is not necessarily so among divers, many of whom are quite capable of opening the eustachian tube at will and during normal breathing.

The symptoms may be relieved or abolished by reclining or lowering the head between the legs – thus increasing venous and lymphatic congestion in the soft tissue of the eustachian tube. Symptoms are also temporarily relieved by sniffing, or by the nasal congestion associated with upper respiratory tract infections.

The disorder may be transitory, or last for many months. Treatment is not usually indicated and those procedures which have been employed in otological practise – such as paraffin or Teflon paste injections around the eustachian cushions – may not be appreciated by divers who need patent eustachian tubes for their diving activities.

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38

The ear and diving: vertigo and disorientation

CARL EDMONDS

DISORIENTATION

The diver needs an accurate appreciation of his orientation, depth, and distance from the boat or shore, to enable him to return safely. With the advent of free and scuba diving – without an attachment to the surface – the importance of **spatial orientation** underwater has increased.

Under terrestrial conditions, spatial orientation depends mainly on information from three inputs: vision; proprioception; and the vestibular system. Vision usually dominates the other two, unless there is abnormal sensory input, damage or stimulation of the other systems.

> *Awareness of body position in space involves integration of visual, proprioceptive and vestibular input information.*

The most likely cause of disorientation under water is the **reduction of sensory input.** With greatly impaired vision and reduced proprioception in an unfamiliar environment, disorientation is more likely. Also, in contrast to the situation on land, there is an added dimension of vertical movement. This becomes important when the diver has lost sight of the sea bed, and is distant from the surface. Other sensory inputs are also modified, e.g. the speed of sound is increased, making localization and discrimination more difficult.

This lack of orientation may be independent of the vestibular system, and occurs in predictable environment conditions, e.g. at night, diving in murky water, diving without a companion, buddy line, surface line, float or boat contact, or diving far from the surface, sea bed or other marine objects. Under these conditions the novice diver especially may become disorientated and also develop symptoms of an agoraphobic reaction (see Chapter 41) – a sensation of depersonalization, derealization, or isolation, associated with anxiety which may extend to panic.

With experience, clues to spatial orientation are available to the diver underwater. Exhaust bubbles can be felt on the skin, moving towards the surface. In certain positions inhalation or exhalation may be expedited, especially with the use of twin hose regulators and rebreathing equipment. Inhalation is

easier if the breathing bag is below the diver in the water. If it or the relief valve are above the chest, then it is easier to exhale.

Heavy objects with negative buoyancy, for example weight belts, pressure gauge or knife, still obey gravity and press or fall downwards. Buoyancy compensators pull upwards. Gas spaces within the body can be felt to expand during ascent and contract during descent, and this may be noted in the diver's lungs, middle ear or facemask. When the diver is immobile his legs will tend to sink and his chest to rise – fins which are negatively buoyant increase this tendency. These clues are of value to the alert, experienced and composed diver, but are ignored by the trainee or during a panic situation.

Diving reduces visual and proprioceptive input, thus placing greater reliance on the vestibular system.

Psychological factors are believed to influence disorientation. Neuroticism, and its associated anxiety state, may result in neglect of many of the orientation clues that have been learnt, and may interfere with an appreciation of the significance of them.

Disorientation may be an early manifestation of a toxic-confusional state, due to **abnormal gas pressures**, e.g. carbon dioxide toxicity, hypocapnia, oxygen toxicity, hypoxia, nitrogen narcosis, high-pressure neurological syndrome, carbon monoxide toxicity, etc. These toxicities are mentioned elsewhere, and are evident by appraising the diving profile and analysis of the breathing gases. Disorientation in these states, although of extreme importance as an early symptom, is usually overshadowed by the subsequent events – unconsciousness or drowning.

Despite the multitude of causes of disorientation under water, the most common is inadequate sensory input experienced by the novice diver.

In other diving accidents, there may also be a prodromal sensation of disorientation, but this becomes obliterated with the impairment of cerebral function. Such instances are seen with cerebral arterial gas embolism, decompression sickness, and syncope of ascent.

There are many other coincidental disorders that may contribute to disorientation, such as cardiovascular disease, cerebrovascular disease, drugs (sometimes related to the diving, such as anti-seasickness medications, decongestants).

CLINICAL VESTIBULAR DISORDER

The anatomy and physiology of the bilateral and integrated vestibular system is described in Chapters 35 and 36.

The most dramatic and demonstrable cause of disorientation underwater is that due to **vertigo**. The diving induced causes are detailed below. The perennial problem of differentiating vertigo from other disturbances of equilibrium such as dizziness, giddiness, unsteadiness, faintness, lightheadedness, swaying, etc., is nowhere more prominent than in the early diving literature. In this chapter, the term 'vertigo' is reserved for conditions in which there is an hallucination of movement, resulting in the impression that objects are moving in a certain direction (objective vertigo), or the patient is moving in a certain direction (subjective vertigo).

Because vertigo is associated with nystagmus and this can be demonstrated objectively, differentiation is made between the specific causes of vertigo from those of disorientation in general. Even under good diving conditions there is interference of visual cues, and weightlessness causing loss of proprioceptive input. Extraordinary significance is then placed on vestibular responses – greatly in excess of that customary on land. This is aggravated if the vestibular stimulation is increased, as is likely in the aquatic dysbaric environment. If vertigo occurs to such a degree that the diver cannot compensate, or if it is associated with vomiting, visual disturbances or unconsciousness, then safety will be seriously impaired.

When the disorientation of the diver is due to a disorder of the vestibular system, it is commonly a transient and mild effect due to the unequal stimulation of the two labyrinths (due to pressure changes, caloric stimuli, etc.). The disorder may persist for as

long as the unequal stimuli remain, and perhaps for a few minutes longer. During this period however, accidents may occur either directly from the effects of vertigo and disorientation, or by interfering with the dive profile.

Sometimes **peripheral vestibular disorders** may be more severe and longer-lasting, e.g. for weeks until central compensatory mechanisms inhibit the unequal vestibular responses. This may be seen in decompression sickness with deep and helium diving, or the more frequent inner-ear barotrauma. The clinical effects may then be catastrophic and endanger even the habitat or saturation diver – let alone the free swimming recreational scuba diver.

Central vestibular disease interfering with vestibular and cerebellar relationships, may produce very long-lasting signs, but are less common than the peripheral manifestation of vestibular disease, in divers.

Once vestibular disease has developed, a full otological investigation is indicated of middle-ear and inner-ear function, both auditory and vestibular. Investigations of cases of vertigo in diving should include:

- Physical examination – otological and neurological.
- Pure tone audiometry to 8000 Hz.
- Electronystagmography – with positional, sometimes caloric or even dysbaric provocation.

In true vestibular disease there is a nystagmus demonstrable at the time of the abnormal sensation, and for a variable time thereafter.

Vertigo is likely to be even more serious in breath-hold diving, when aspiration and/or vomiting is more significant. With scuba diving there is an adequate air supply to permit the diver to wait and settle – to see if the vertigo is transitory. Orientation cues, such as the direction of bubbles, are less appreciated by free divers.

Apart from the diving-induced causes of vertigo, there are many other causes that may have to be considered, and these may be either unrelated or only peripherally related to the diving. These include:

- ear infections;
- benign paroxysmal positional vertigo (cupulolithiasis);
- vestibular neuronitis;
- Meniere's disease;
- coincidental neurological disease, such as multiple sclerosis;
- cerebrovascular disease;
- acoustic neuroma; and/or
- drugs, such as some anti-malarials.

CLASSIFICATION OF VERTIGO IN DIVING (Edmonds *et al.*, 1973)

Vertigo due to unequal vestibular stimulation

1 Caloric
  (a) Unilateral external auditory canal obstruction
    (i) Cerumen
    (ii) Otitis externa
    (iii) Miscellaneous
  (b) Tympanic membrane perforation
    (i) Shock wave
    (ii) Middle-ear barotrauma of descent
    (iii) Forceful autoinflation
2 Barotrauma
  (a) External-ear barotrauma
  (b) Middle-ear barotrauma of descent
  (c) Middle-ear barotrauma of ascent
  (d) Forceful autoinflation
  (e) Inner-ear barotrauma
    Perilymph fistulae
    Other causes
3 Decompression sickness
4 Miscellaneous
    Tullio phenomenon

Vertigo due to unequal vestibular responses

1 Caloric
2 Barotrauma
3 Gas toxicity
4 Motion sickness
5 Sensory deprivation

Vertigo due to non-diving aetiologies

UNEQUAL VESTIBULAR STIMULATION

Exposure to hyperbaric and diving environments results in stimuli to both vestibular apparatuses.

Under most diving conditions the stimuli on each side will be equal, but in certain situations this is not so, especially when there is a pathological process involving one external or middle ear. Under these conditions a dominant stimulus effect on one side may produce vertigo.

Caloric

When the diver immerses himself there is normally an equal flow of cold water into both external auditory canals. This stimulation is symmetrical, and no vertigo is expected – nor does it occur in the vast majority of dives. If the stimulus is greater on one side, vertigo would be expected – with an intensity and duration related to this inequality.

In the production of **caloric vertigo** the spatial position of the diver is pertinent. In an experimental situation, caloric stimulus will produce the most intense vertigo and nystagmus when the subject is either lying supine with his head elevated at 30 degrees, or lying prone with his head depressed at an angle of 30 degrees. In both these positions the horizontal semicircular canal becomes vertical. This has been demonstrated in the supine position during the conventional 'Hallpike' caloric tests (see page 374).

This 'Hallpike' head position is adopted by divers occasionally, for example when cleaning the hulls of ships, but a more usual position for divers is the prone position. The diver assumes the most vestibularly sensitive 'prone-with-head-down' position when descending obliquely, while attempting to swim horizontally when underweighted or with positively buoyant fins. Resumption of an upright posture terminates the vertigo (Fig. 38.1).

In these positions, vertigo may result when there is unilateral obstruction to water flow into the external canal, when the tympanic membrane perforates, or where there is bilateral and equal caloric stimulus but with unequal vestibular responses (see below).

Unilateral external auditory canal obstruction

This does not cause permanent vestibular damage, and the only effect on audiometry is that due to the canal obstruction, i.e. a remedial conduction deafness. Two common causes observed are **cerumen** and **otitis externa**, although others such as **exostosis**, **foreign body**, **ear plug**, **asymmetric hood** and **air bubble** may allow water to flow into one ear only, and produce this effect.

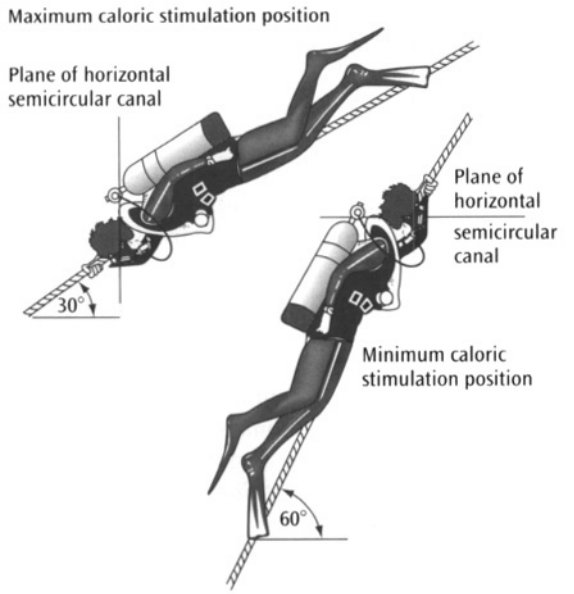

Figure 38.1 *Caloric stimulation related to spatial orientation.*

Tympanic membrane perforation

This is a dramatic cause of transient, but often disabling vertigo. Firstly, there may be a 'pop' or loud noise or pain associated with a sensation of cold water rushing into the middle ear. Vertigo follows almost immediately and usually lasts for less than a minute. This small amount of cold water rapidly warms to body temperature, removing the caloric stimulus. Vertigo is especially likely if the diver continues his descent, encouraging more water flow into the middle ear.

On surfacing, the diver may have bloodstained fluid running from the external ear; this is expelled when the gases which have remained in the middle ear expand during ascent and force blood out through the perforation.

There may be no vertiginous symptoms in those cases in which there is no entry of water into the middle-ear cavity, because this space is occupied by blood, or if the perforation occurs during ascent.

CASE REPORT 38.1

This diver had no difficulty in autoinflating his ears during a descent to 10 metres. While swimming along a horizontal underwater line he felt as if he were rotating to one side around the line. As the line was on the sea bed he knew that his sensation was incorrect, and he decided to surface. The vertigo, which lasted for some 10 to 20 seconds, did not trouble him during the ascent and he had no further difficulty.

On clinical examination there was no abnormality other than the presence of a large plug of hard cerumen in the left ear. Before removal of the plug it was decided to carry out ENG with caloric testing, to ascertain whether sufficient water passed this obstruction. The positional ENG was normal, but the caloric demonstrated a false picture of a total left canal paresis. The cerumen was removed, and no evidence of external- or middle-ear barotrauma was noted. The cerumen plug was large enough to obstruct the free flow of water, but not enough to prevent some water from equalizing the changing pressures within the external ear. The caloric stimulus was therefore much greater in the unobstructed ear, producing transient vertigo. No further incidents occurred after the plug was removed.

Diagnosis: vertigo from a unilateral caloric stimulus, due to unilateral auditory canal obstruction by a cerumen plug.

These patients usually notice the hissing of gas through the perforation with ascent or autoinflation. Occasionally, they first develop their vertigo while driving after the dive. Whether this is due to head movements aggravating positional vertigo, air currents replacing water in producing caloric stimulation, or eddy current movements of the tympanic membrane and ossicles, is not known.

There are three major predisposing causes of tympanic membrane perforation leading to vertigo while diving: a shock wave; middle-ear barotrauma of descent; and forceful autoinflation of the middle ear.

- *Shock wave*: This disorder is easily diagnosed on history, and was common when Navy divers were subjected to underwater explosions, especially when they were faced at right-angles to the source. (see Chapter 34). In recreational diving the most common underwater shock wave causing perforation of the tympanic membrane and vertigo is caused by being 'finned'. When a diver swims past another diver, considerable pressure waves are felt from the fin (or flipper) movements. Perforation of the tympanic membrane is possible, especially if the ear is not adequately autoinflated. The shock wave, which is a water pressure wave, is probably also responsible for the entry of water into the middle ear following the perforation. Most cases have no permanent vestibular or hearing sequelae, although these could occur in more severe cases with inner-ear barotrauma.
- *Middle-ear barotrauma of descent* (see page 75).
- *Forceful autoinflation of the middle ear.*

Barotrauma

As a cause of vertigo there are five clinically important types of barotrauma (see Chapter 7), comprising:

1 External-ear barotrauma of descent.
2 Middle-ear barotrauma of descent.
3 Middle-ear barotrauma of ascent.
4 Forceful autoinflation of middle ear.
5 Inner-ear barotrauma.

Vertigo due to barotrauma is more likely when the diver is upright (vertical) than when he is horizontal or in the 'caloric' position. To verify this clinical observation it is possible, but ethically questionable, to reproduce vertigo due to barotrauma in a compression chamber with ENG monitoring.

This spatial orientation may infer involvement of the utricular/saccular divisions. The unequal pressure gradients may themselves produce vestibular

disorder, or they may – especially when associated with forceful autoinflation – result in inner-ear barotrauma, producing a much more permanent effect.

External-ear barotrauma of descent

This condition is also known as 'reversed ear' (see page 74).

Middle-ear barotrauma of descent

This condition is also known as 'ear squeeze' (see page 75).

Case report/Figure 38.2 demonstrated in an objective manner that vertigo and nystagmus can be precipitated with the middle-ear pressure changes during descent, while case report/Figure 38.3

CASE REPORT 38.2

The patient, a certified diver, frequently developed dizziness during descent. She commonly experienced difficulty in equalizing the middle-ear spaces and frequently resorted to nasal decongestants. There were no symptoms suggestive of disorientation during ascent, and usually the sensation of dizziness reduced as she remained at a constant depth.

On examination, there was no conventional evidence of any abnormality in ear function; she had normal pure tone audiograms and a normal electronystagmogram (ENG) to positional and bithermal caloric stimuli.

Dysbaric ENGs (as described by Edmonds *et al.*, 1973) were performed with the diver sitting upright with the eyes closed in a compression chamber. ENG monitoring was continued while the patient was subjected to changes in pressure. The compression was at the rate of 9 metres/minute to a depth of 18 metres. The patient was then kept at the depth of 18 metres for 2 minutes and ascended at the same rate. Minor problems were encountered with middle-ear equalization during descent, but did not lead to cessation or delay of this descent.

The ENG results verified the subject's observation of vertigo associated with compression, which was relieved by maintenance of pressure and absent during decompression (see Fig. 38.2). The nystagmus 'saw tooth' pattern is obvious during and immediately after descent.

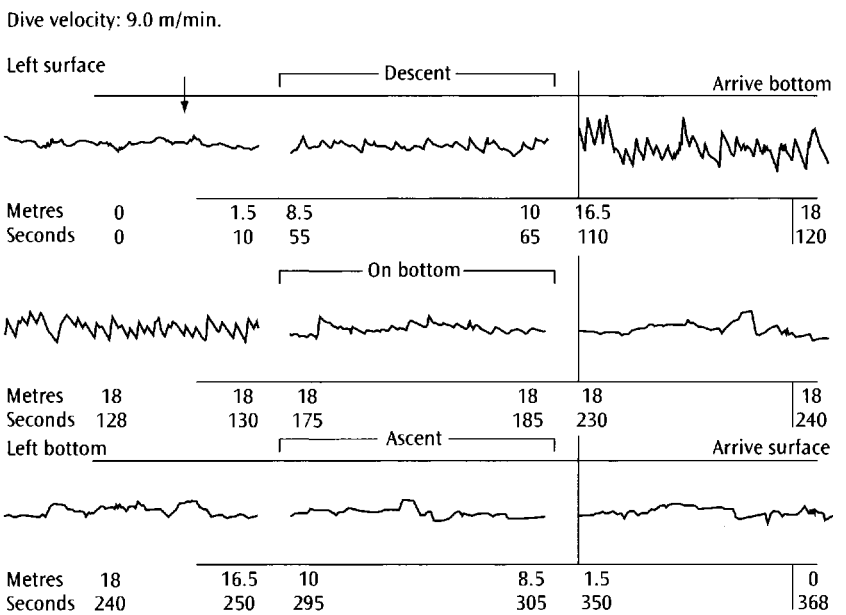

Figure 38.2
Electronystagmograms from Case report 38.2.

CASE REPORT 38.3

This subject also had normal hearing and vestibular function demonstrated before having difficulty in equalizing both middle-ear pressures during a recompression chamber descent. Because of the inability to achieve this middle-ear equalization, the compression was terminated at 4.5 metres. At that time the ENG monitoring (performed for another experiment) demonstrated severe nystagmus associated with the subjective complaint of vertigo. The diver was in the supine position. Vertigo persisted for many minutes after the initial middle-ear injury, and nystagmus was demonstrated in a progressively decreasing degree, for approximately 12 minutes. (see ENG in Fig. 38.3). The nystagmus 'saw-tooth' pattern was maximal between 3.0 and 4.5 metres during descent, less at 5 minutes, and almost gone at 12 minutes.

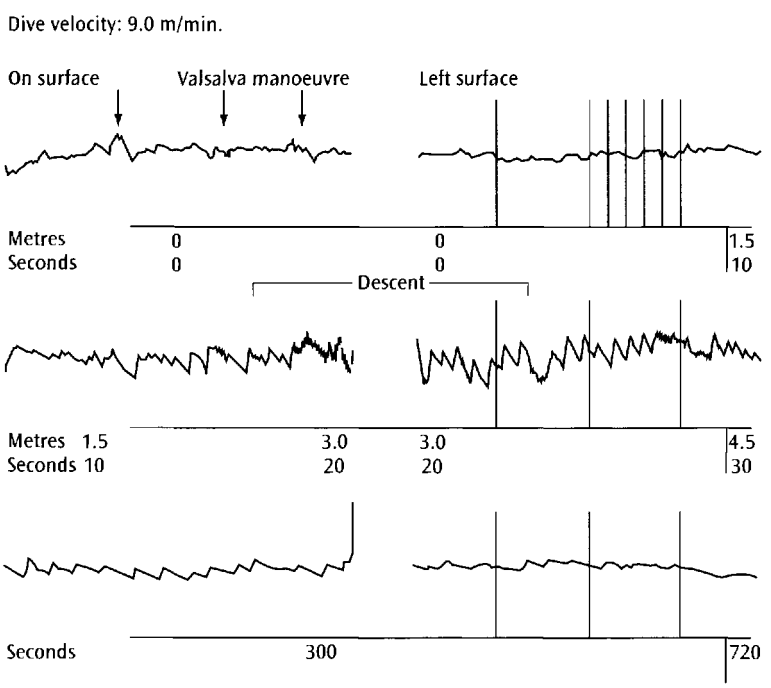

Figure 38.3
Electronystagmograms from Case report 38.3.

demonstrated that middle-ear barotrauma of descent can initiate vertigo and nystagmus, that this manifestation may continue, and that the condition need not be merely transitory.

Because there is no evidence of abnormal cochlear or vestibular function before the dives referred to above, there is no reason to believe that the vestibular response is due to an underlying vestibular inequality. In both cases a history of inability in equalizing the middle ear space, and presumed inequality of middle-ear pressure is the cause for the abnormal vestibular response.

These descriptions are thus similar in concept, although opposite in direction, to the alternobaric vertigo described by Lundgren and confirmed independently with ENGs by Edmonds (divers) and Tjernstrom (aviators). The nystagmus responses described in this and previous reports are inhibited

by eye opening, indicating the peripheral nature of the disorder. Persistent nystagmus implies more persistent barotraumatic pathology in the vestibule.

Dysbaric electronystagmograms supplement formal vestibular function tests in aviators and divers, and may be needed to demonstrate vestibular pathology. They are related to Hennebert's sign.

Middle-ear barotrauma of ascent

This condition is also known as 'alternobaric vertigo' (see page 83). The unequal release of gas from the middle-ear cavities, especially during the initial stages of ascent, results in a pressure difference between the two middle ears, and unequal stimuli to the vestibular systems. The nystagmus is toward the side of the block, with the diver's spinning sensation toward that ear with the higher middle-ear pressure.

The vestibular system is sensitive to pressure changes in the middle ear. Vertigo can be produced by a pressure increase of 60 cm H_2O in one middle ear. The range of effects is from a physiological disorder (Lundgren's alternobaric vertigo) to a pathological entity (Edmonds and Freeman's inner-ear barotrauma).

Forceful autoinflation

This condition is produced during 'equalising the ears'. Some subjects can produce vertigo merely by performing autoinflation, and it may be presumed that the middle ears are ventilated unequally, because of unequal eustachian tube patency. This is a clinical manifestation of Hennerbert's sign. Another proposed mechanism is an abnormally mobile stapes.

Decompression sickness (Chapter 12)

Although vertigo has often been reported among air scuba divers with this condition, there is considerable doubt about the frequency of the diagnosis when, as in many of the cases reported, there are no other manifestations of the disease. It is likely that many of the permanent vestibular damage cases originally reported as decompression sickness (DCS) were really due to inner-ear barotrauma. The delay of onset of symptoms is not necessarily a diagnostic feature favouring DCS, as it is not uncommon in cases with perilymph fistulae. Vertigo may occur without loss of hearing in both diseases. DCS may cause cerebellar manifestations ('staggers') as well as vestibular damage, and these are often confused.

Even allowing for the misdiagnoses, there are still some cases which presented with hearing loss and/or vertigo, probably due to DCS. This is especially so with deep diving, using helium as a breathing gas; in these cases it is necessary to institute recompression therapy to prevent permanent inner-ear damage.

The consequences of vertigo, such as near-drowning, vomiting, dehydration, electrolyte disturbances and distress, are more important in a patient who is already seriously ill with DCS. If incorrectly diagnosed during decompression (e.g. if the symptoms are attributed to seasickness), then further decompression may result in more damage to the vestibular apparatus.

Recompression therapy, if promptly instituted, should result in cure. Objective tests of vestibular function can and should be performed under hyperbaric conditions, when doubt exists regarding clinical management. These investigations include ENG and iced-water caloric tests, and are valuable in differential diagnosis, prognosis and response to treatment.

There are also long-term sequelae. With vestibular damage there is a likelihood that the diver may not be able to continue with his occupation, and that he may be restricted from other occupations such as flying or driving.

Miscellaneous conditions

Certain specific medical causes of vertigo are also occupational complications of diving. Migraine (see page 412) is a typical example, and it is recommended that patients with this disorder should limit their diving. Otitis media can also be classified both as an occupational disease of divers and as a cause of vertigo. Benign positional paroxysmal vertigo may be induced by the diver adopting a position which triggers this response.

Tullio phenomenon

The observation by Tullio early last century, that sound can stimulate the vestibular system, has received much attention from the 1930s onwards. Vertigo may be experienced, and nystagmus produced, when subjects are exposed to pure tones ranging from 200 to 2500 Hz at intensities from 120–160 dB. Dizziness, nausea and disturbances of postural equilibrium have been correlated with sound stimulation at intensities and frequencies lower than these.

In diving, the Tullio phenomenon is especially seen with compression chambers which do not have muffling systems over the air inlets and in which there is excessive enthusiasm to 'flush-through' the chamber with the compressed gas – producing very loud noises. It may also be observed in helmet divers, caisson workers and aircraft personnel.

UNEQUAL VESTIBULAR RESPONSES

This group includes subjects who, under most conditions, would be considered 'normal'. In these people, vertigo is the end result of unequal vestibular responses to equal stimuli, i.e. one vestibular apparatus being more sensitive than the other. Even small differences in the vestibular responses may produce some demonstrable dysfunction when exposed to the stimuli encountered with diving or hyperbaric environments.

In some of these cases the inequality can be demonstrated by conventional cochlear or vestibular function tests. Occasionally, a damaged vestibular apparatus on one side may have been the result of a previous diving accident, whereas in other cases non-diving aetiologies may be postulated. An example of the latter is the inner-ear damage which occurs due

CASE REPORT 38.4

A 35-year-old male was subjected to a recompression chamber dive to 9 metres (30 ft). There were no problems with middle-ear equalization during descent. While in the outer lock, and before equalizing the chambers, the subject used the intercom phone system, with the earpiece next to his right ear. The chamber pressures were equalized and the subject moved from the outer to the inner lock. A 'flush-through' was then performed. This procedure involves the replacement of a large quantity of compressed air in the chamber, exchanging the chamber air and removing carbon dioxide. In this particular case, it was performed by a chamber operator under training, and an excessive zeal was used in opening the valves, producing a great deal of noise. After approximately 20 seconds, the noise of the 'flush-through' was so intense that "it knocked me off balance". There was no actual change of pressure during this period and therefore no need to perform Valsalva manoeuvres or middle-ear equalization. The diver felt severely giddy and disorientated, and was unable to stand. His right ear was nearer the inflow of the gas, and when he attempted to use this ear to communicate through the telephone intercom, he was unable to hear the voice of the surface attendant. He was able to use his left ear for this. He noted that he was falling to the left when he finally got out of the chamber, and was feeling off balance through most of that day. He claimed that he was far more sensitive to noise, even though his audiograms had not changed. Impedance audiometry revealed a tympanic membrane far more mobile on the right side than the left. ENG demonstrated nystagmus on the vertical tracing in the positional test, even though the calorics tests appeared normal.

Clinical examination of the ear, nose and throat was normal, without any evidence of patulous eustachian tubes. The tympanic membranes were easily moved with middle-ear autoinflation, and there was no evidence of middle-ear barotrauma.

Diagnosis: Tullio phenomenon, presenting with vertigo and dysacusis.

to gunfire, especially when this is unilateral (see case report 38.5/Figure 38.4).

The fact that asymmetry cannot be demonstrated in many patients probably highlights the relative crudity of the investigations. As ENG or computerized positional tests become more precise, and with accurate and controlled stimuli, marginal asymmetry of vestibular function may be demonstrated.

Caloric

A very common syndrome of vertigo induced by diving seems explicable only by postulating a greater caloric response from one vestibular apparatus than from the other. There is usually no abnormality evident on otoscopic examination. There is no reason to postulate barotrauma, sensory deprivation, DCS,

Figure 38.4 *The first case (1972) in which a diver with vertigo during ascent was monitored with an electronystagmogram (ENG). (From Edmonds C.* et al. Otological aspects of diving. *© 1973 Australasian Medical Publishing Company, reproduced with permission.)*

CASE REPORT 38.5

After firing 50 rounds from a .22 rifle, this man developed tinnitus and slight hearing loss. When scuba diving, he developed vertigo and nausea during subsequent ascents, sometimes leading to vomiting. Some relief was obtained by stopping ascent and assuming a prone position.

Pure tone audiograms verified the slight hearing loss, but ENGs were normal, both positional and calorics. Exposure to compression in a chamber verified the development of vertigo during ascent (see Fig. 38.4).

Diagnosis: vertigo from unequal barotrauma effects because of inequality of inner ears.

inert gas narcosis, etc. Caloric vertigo of this type is most commonly experienced by divers who have descended without any difficulty in equalizing pressures in the middle-ear cavities, and have reached a level at which they then perform a horizontal swim. The vertigo normally comes on within 5 minutes of the commencement of the dive, and tends to recur when the diver attempts similar dives under similar conditions within the next few weeks. These cases have previously been given the nondescript term 'idiopathic vertigo of divers'.

The belief that this particular syndrome is due to unequal vestibular response to caloric stimulation is supported by:

- the time delay before vertigo is produced, unlike vertigo from barotrauma;
- the spatial orientation of the diver during the swim, suggestive of caloric-induced vertigo; and
- the tendency for this to recur during similar dives and without any otoscopic abnormalities present, refuting the likelihood of unequal stimulation.

Barotrauma

Even though the eustachian tubes may be patent and equal, ensuring symmetrical pressure changes in the middle-ear cavities, vertigo may result if one vestibular apparatus is relatively hypofunctional. Under these conditions the vertigo occurs during or immediately following the changes of pressure, i.e. descent or ascent.

Vertigo from barotrauma is more severe when the diver is in the upright position than in the almost horizontal 'caloric' position (see case report 38.5/ Figure 38.4).

Gas toxicity

This field has been little explored by otophysiologists. The difficulty of differentiating vertigo from dizziness, lightheadedness and disorientation makes any review of the historical diving literature almost valueless in this context. Because of the interference in cerebral function, the gas toxicities may result in serious disorientation whether or not vertigo is noted or nystagmus demonstrated.

Inert gas narcosis (see also Nitrogen narcosis, Chapter 15)

As divers descend beyond 30 metres while breathing air, they become progressively sedated and narcotic from the influence of nitrogen. Dizziness has been described by many divers under these conditions, but there is considerable doubt as to whether this proceeds to true vertigo. If this dizziness does represent a true vertigo in some cases, then like other symptoms of nitrogen narcosis, it should be quickly corrected by reducing the nitrogen pressures, i.e. with ascent.

Nystagmus seems to be accentuated by exposure to high nitrogen pressures, supporting the possibility of nitrogen narcosis as an accessory aetiological factor, in conjunction with the dysbaric and caloric stimuli to which divers are exposed.

High-pressure neurological syndrome

Vertigo, nausea and tremor are some of the symptoms reported with this syndrome. Nystagmus and vertigo are not now believed to be characteristic features. The effects are probably either in the subcortical or reticular activating system, and are aggravated by too rapid a compression, and are relieved within a few hours of reaching depth.

Oxygen toxicity

Vertigo is a documented symptom of this disorder when it affects the neurological system. Vertigo may be a warning symptom. It is also precipitated during the reduction from high oxygen pressures, i.e. an 'oxygen-off effect', as well as following oxygen convulsions. These situations are only likely when divers use oxygen, mixed gases or rebreathing equipment, when the safe limits for oxygen pressures and durations are exceeded. Nausea or vomiting are also associated with somewhat lower oxygen pressures (1–2 ATA), but whether these are related to vertigo is unknown at this stage.

Carbon dioxide toxicity

Disorientation is a characteristic feature of this toxicity, but vertigo is far less definite. It has been reported in association with vomiting by submariners who have become acclimatized to breathing high carbon dioxide pressures and then revert to breathing air or oxygen. This is known as the 'carbon dioxide-off effect'. A similar state occurs clinically in divers using rebreathing equipment with partially ineffective carbon dioxide absorbent systems, when they reduce their high carbon dioxide exposure, e.g. when they rest after an energetic swim.

Other gases

The effect of hypoxia, hypocapnia, carbon monoxide poisoning, etc. may well include vertigo, as this is a possible symptom with any factor which disturbs the state of consciousness. In these cases the effects are analogous to drug effects, although some – such as mefloquine – can specifically induce vertigo.

Motion sickness

Motion sickness is a complication of certain diving operations, for example on the diving boat, while decompressing on a platform or rope underwater, or swimming. One aetiological hypothesis is an excessive and unequal vestibular response to motion, with nausea, vomiting, syncope and vertigo.

Sensory deprivation

Sensory deprivation, especially when it involves those senses involved in spatial orientation, is likely to aggravate vertigo and to produce disorientation. Abnormalities of vestibular or cochlear function would not be expected in most of these cases. It is possible that sensory deprivation will serve to decrease the threshold for vertigo and nystagmus. This belief receives support from the techniques aimed at reducing extraneous stimuli, which are utilized during vestibular testing in otological laboratories. Deprivation produces disorientation.

> *Blue orb syndromes* *(see page 419).* *Frequently the diver overcomes the lack of sensory input by visual fixation on objects that are associated with him, such as a companion diver, a buddy or surface line, or equipment. The syndrome can be prevented by avoiding conditions mentioned above, and corrected by swimming to the surface, the seabed or along ledges.*

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39

Cardiac problems and sudden death

CHRIS LOWRY

INTRODUCTION

Sudden death has been observed and described since antiquity. In modern times there is little doubt that the immediate cause of many scuba fatalities is either a myocardial infarction or a cardiac dysrhythmia. This is increasing in frequency, possibly due to the increasing age of the diving population. On land, ventricular fibrillation is the commonest cause, followed by bradyarrthymias and torsade de pointes. In the water, the ratio is uncertain.

Sudden death may also ensue from hypoxia or other causes of unconsciousness, and these are discussed in Chapters 16 and 47.

Sudden cardiac death is natural death due to cardiac causes. The death is unforeseen, and heart disease may or may not be known to be present prior to the incident. Up to 21 per cent of deaths in scuba divers are attributed to 'cardiac disease'.[1] Unfortunately, this designation does not give a full understanding of the dynamic progress of events, but rather it belittles the complexities and interrelationships between the diver, his equipment and the environment.

Diving induces a series of stresses,[2] which may impact on cardiac function, via the conducting system, coronary blood supply or efficient muscular contraction. Environmental factors cause reduction of blood volume, tachycardia or bradycardia, hypertension and increased cardiac work.

Dysrhythmias, including various degrees of heart block, paroxysmal tachycardia and fibrillation, further reduce the efficiency of the heart's ability to supply an adequate blood flow, in the face of the heavy physiological demands imposed by the environment.

Exceeding the coronary arteries' ability to supply the myocardium with oxygen may result in **coronary insufficiency** and **myocardial ischaemia**, causing sudden death. Although this is possible with normal coronary arteries, it is much more likely when the diver has reached an age at which coronary pathology has developed. The majority of myocardial infarctions occur secondary to rupture of atheromatous plaque with less than 40 per cent coronary stenosis. Because these lesions are not haemodynamically significant the victim is often asymptomatic before the event. Half of all myocardial infarctions are associated with a triggering event, the triggers of plaque rupture perhaps being **extrinsic** or **intrinsic**. Extrinsic triggers induce a surge in sympathetic activity causing changes in heart rate, blood pressure and coronary blood flow. Intrinsic triggers induce a

hypercoagulable state or impair fibrinolytic activity, thus altering coagulation status.

The importance of triggering events is illustrated by the marked increase in myocardial infarction incidence during the Los Angeles earthquakes and the Scud missile attacks on Tel Aviv.

Eldridge[3] bases the following points on the investigations into sudden death in scuba diving. A substantial number of middle-aged males suddenly and inexplicably lost consciousness and died while scuba diving in cold water. The behaviour of the victims, ineffectiveness of resuscitation and the timing of the brain damage and death, suggest a cardiac dysrhythmia as the underlying cause. Most of the victims had either previous cardiovascular symptoms such as hypertension or dysrhythmia and/or showed significant coronary artery stenosis or pathology at autopsy. Eldridge's report concluded with a plea for physicians conducting diving medical examinations to be aware that minor cardiac irregularities may preface major problems to people diving in cold water.

After detailed assessment of the case histories, the following observations were made. The victim often appeared calm just before his final collapse. Some were unusually tired or resting, having previously exerted themselves, or were being towed at the time – suggesting some degree of exhaustion. Some acted as if they did not feel well before their final collapse. Some complained of difficulty in breathing only a few seconds before the collapse. Others under water signalled that they needed to buddy breathe, but rejected the offered regulator. Explanations for the dyspnoea include psychogenic hyperventilation, autonomic induced ventilatory stimulation and pulmonary oedema, the latter being demonstrated at autopsy. In all the cases there was an adequate air supply available, suggesting that their dyspnoea was not related to equipment problems. Some victims lost consciousness without giving any signal to their buddy, yet others requested help in a calm manner.

Most of the autopsies revealed moderate stenosis of a coronary artery. Some showed a 100 per cent blockage, and others had evidence of infarction. Victims who did not have substantial atheroma often had pre-existing hypertension. Pulmonary oedema to a degree frequently associated with left ventricular failure, was seen in some of the cases. A few had medical records of cardiac symptoms such as dysrhythmias, and one had had coronary bypass surgery.

Case report analyses suggested that the diagnosis of drowning was overemphasized, it being a consequence of the environment in which the disorder occurred, rather than an aetiological factor. It is very likely that these cases of sudden death are similar to others associated with swimming and cold-water immersion. It is probable that they are related to other sudden death syndromes in sports such as in squash, athletics, adultery, etc., although in these particular environments there is much more opportunity to desist and obtain relief from physical stress, and for subsequent resuscitation, than in the water.

The data were supported by the research of McDonough and his colleagues[4] showing strikingly higher frequencies of dysrhythmias when scuba diving. These included both supraventricular and ventricular premature contractions and dysrhythmias. Breath-holding and facial immersion produced other dysrhythmias, and was considered to best identify susceptible individuals.

Autonomic nerve control of the heart, either by overactivity or imbalance, is thought responsible for most of the dysrhythmias of diving. Many of the diving activities involve an increase in sympathetic tone on the heart; others induce parasympathetic effects. Some of the factors that stress the cardiovascular system in diving are:

- Exercise
- Psychological factors
- Cold
- Immersion
- Breath-hold diving
- Reflexes associated with diving.

These triggers are even more likely to provoke a cardiac incident in the presence of underlying cardiac disease, whether occult or manifest.

CARDIAC STRESS FACTORS

Exercise

Perhaps the most famous example of death due to exercise is that incorrectly attributed to the famous athlete Pheidippides (490 BC). This young Greek

soldier ran 26 miles from the battlefield of Marathon to the city of Athens, delivering news of victory over the Persians, and then dropped dead. The feat is commemorated by the present day marathon athletic event.

The increased parasympathetic (vagal) tone associated with fitness training, may make the fit individual more susceptible to a variety of atrial dysrhythmias. Alternately, fatigue is more likely in unfit individuals and has been shown to be a dangerous factor in those susceptible to dysrhythmias.

Increased cardiac output and heart rate do not produce blood pressure elevation on land, because of vasodilatation in the muscles and the skin. However, the latter is inhibited when the skin is exposed to cold water. The inclination to perform heavy work with elevated oxygen consumption is enhanced in the sea because of the more pleasant conditions, and the diver rarely feels overheated and uncomfortable. Vigorous exercise does not usually prevent – and may even intensify – the normal diving bradycardia.

The metabolic acidosis and hypercapnia associated with extreme exercise and the cardiorespiratory effects of diving, which hinder carbon dioxide elimination, will aggravate any tendency to cardiac dysrhythmia. Myocardial ischaemia and hypoxia, resulting from inadequate cardiac output and/or coronary narrowing, will complicate this condition and both will predispose to myocardial ischaemia and sudden death. Both clinically and on exercise provocation in the laboratory, dysrhythmias are often observed 5-10 minutes after the maximal effort, and are therefore more likely at the end of a dive.

Personality and psychological factors

> *"It is not the delicate, neurotic person who is prone to angina, but the robust, the vigorous in mind and body, the keen ambitious man, the indicator of whose engine is always set 'full speed ahead'".*
>
> *William Osler*

Anxiety and the sympathetic response may have a deleterious effect on cardiac function, especially if there is already cardiac pathology. This is appreciated by physicians, and is well typified by the demise of John Hunter – the eminent pathologist and surgeon who inoculated himself with syphilis, believing it to be gonorrhoea, while performing research for his treatise on venereal disease. When confronting a particularly disturbing managerial committee of the hospital, he clutched his heart, stated, "Gentlemen, you have killed me", and slumped dead over the table.

If the diver believes there is a threat to him, whether real or not, there will be an autonomic stress response. During this anxiety state a massive sympathetic discharge is present, with blood pressure reaching very high levels and causing excessive strain on the heart. Heart rates of up to 180 per minute can be produced and will reduce cardiac output, diminishing coronary blood flow proportionately.

The sympathetic autonomic response to stress can result in dysrhythmias, myocardial ischaemia and even sudden death in the presence of underlying cardiovascular disease.

Another form of psychic stress is the vagal response which produces exactly the opposite effects from the above, i.e. hypotension and bradycardia to the stage of cardiac standstill. This is analogous to the fainting or syncopal episodes of people who respond with an extremely exaggerated reaction to stress, e.g. fainting at the sight of blood, or on receipt of tragic news. The vagal parasympathetic response could cause an inappropriate cardiac inhibition in the aquatic environment, with syncope leading to drowning. One such influence is the diving response, which is augmented by exposure to cold, such as with cold water on the face, with perhaps concomitant induced breath-holding.

Panic disorder has been reported to produce cardiac ischaemia in the absence of atherosclerosis.[5] This state may develop insidiously in the apprehensive scuba diver. Nevertheless, although stress is the trigger, underlying coronary artery disease is usually the substrate.

Cold

Any patient with angina pectoris will attest to the deleterious effects of cold in aggravating their condition.

In the case reports on exercise stress precipitating cardiac death during scuba diving, a common factor was the association with cold water. Cold can produce a variety of cardiac insults, with various types of responses (see Chapter 28).

During cold-water immersion there is usually an increase in the sympathetic activity, as indicated by a rise in circulating norepinephrine (noradrenaline), concurrently with or just preceding the release of epinephrine (adrenaline) from the adrenal glands. The sympathetic activity will be responsible for the increase in heart rate, systolic blood pressure and ventilation. The deleterious effect on cardiac efficiency may be very significant. An increase in the diastolic blood pressure is also possible, but not invariable. It may be overridden by the adrenaline effect, which at physiological doses causes a drop in the diastolic pressure. The sympathetic response is observed to be greater in subjects who are physically unfit and who have not adapted to cold-water exposure.

The normal bradycardia of immersion is markedly enhanced in cold water. Breath-holding and cold immersion combine to produce such an intense bradycardia that unconsciousness and drowning may ensue – so-called hydrocution. Sudden death from a vagal parasympathetic reflex can follow inhalation of water into the nasopharynx and on the glottis. There is also thought to be a reflex following cutaneous stimulation from cold, producing coronary spasm or sudden death in people who are immersed in very cold water. Another cutaneous reflex induces considerable hyperventilation, sufficient to reduce carbon dioxide tension in the arterial blood to levels which have been associated with the ventricular fibrillation in both animals and man.

In general, the degree of the 'diving reflex' bradycardia is greater with lower water temperatures. With the development of hypothermia, the myocardium becomes hyperexcitable, and is susceptible to episodes of ventricular extrasystoles, tachycardia and fibrillation. This becomes more frequent in the late stages and during rewarming.

Peripheral vasoconstriction will result in central pooling of the blood, with diuresis and associated fluid and electrolyte loss.

Immersion

An immediate reflex from immersion causes a sudden and temporary increase in cardiac output and stroke volume, each by up to 100 per cent, before stabilizing at a lower level.

Immersion counters the effect of gravity thus expediting venous return from the vessels of the limbs (see Chapter 3). This redistribution of blood increases intrathoracic blood volume by up to 700 ml and right atrial filling pressure by 18 mmHg. The cardiac output, and thus the work of the heart, is increased by over 30 per cent. Cold water intensifies these effects, and may also subsequently lead to water and electrolyte loss from the body, and then an increased susceptibility to syncopal episodes. 'Negative pressure' breathing – a result of immersing the body but breathing through a snorkel, from the atmosphere – also increases the diuretic effect of immersion.

Reflexes associated with diving

Diving response (see Chapters 3 and 61)

In diving mammals, the diving response is associated with bradycardia and intense peripheral and selective visceral vasoconstriction. The reduced cardiac output is preferentially distributed to the heart and brain. This combination of effects results in a maintenance of normal arterial blood pressure, a reduction of heat loss and a conservation of oxygen for vital organs.

In man, the reflex is more rudimentary and undeveloped. Although there is a diving bradycardia, it is often complicated by the development of idioventricular foci producing ectopic beats. Electrocardiogram (ECG) abnormalities are frequent during or after the dive. T-wave inversion, premature ventricular excitation and atrial fibrillation, together with other irregularities and dysrhythmias are common. They reflect inhibition of vagal rhythms and interference with atrioventricular (AV) conduction. Hong *et al.*[6] showed an incidence of cardiac dysrhythmias in the Korean women divers, increasing from 43 per cent in summer (water temperature 27°C) to 72 per cent in winter (water temperature 10°C).

The drop in cardiac output noted in diving mammals is seen to only a slight degree in man. A proportionate drop in cardiac output does not compensate for the intense vasoconstriction of the peripheral vessels that results when man is immersed in cold water, and therefore there is a significant rise in arterial blood pressure, only partly attenuated by the accompanying bradycardia.

The diving response may well have some protective value in the drowning situation, and has indeed been used in the treatment of paroxysmal atrial tachycardia. Nevertheless, it is likely that it may contribute significantly to the otherwise inexplicable diving deaths.

Carotid sinus syndrome (see Chapter 42)

This disorder is frequently observed, although often not recognized, by divers whose wet suit or close-fitting dry suit applies pressure to the carotid bifurcation. The neck constriction is especially noted with wet suit tops, which are pulled over the head. Bradycardia and hypotension are reflexly produced resulting in a sensation of fainting, dizziness or even convulsions.

The extent of the relationship of the carotid sinus syndrome to sudden deaths in scuba divers is not yet clarified. Certainly the cardiovascular effects are dramatic and the disorder is not uncommon among divers who wear tight-fitting 'pullover' wet suits. It also increases in frequency and severity with advancing age. Occasionally, divers in great distress will be seen pulling the neck of the wet suit away from the throat, and sometimes this is done prior to the loss of consciousness. In only one of 100 well-documented deaths in the ANZ series,[1] was the carotid sinus reflex incriminated, but in others there were complaints of dyspnoea and tight-fitting wet suits, which could well be related to this syndrome.

Other cardiac reflexes

A variety of other reflexes influence dysrhythmias, some having the afferent stimuli from skin or mucosal areas (e.g. the pharynx) and others associated with breath-holding, Valsalva manoeuvres and exercise. A French electrocardiographic study[7] conducted in ten experienced breath-hold divers, reaching a maximum of 15 metres in 28°C water during repeated diving, showed the expected bradycardia but also dysrhythmias in six of ten divers. The atrial dysrhythmias were frequently multiple, and the ventricular dysrhythmias were bigeminal.

In persons with known coronary artery disease diving produces a much more powerful coronary vasoconstrictor response than the clinically used cold pressor test.

Hyperbaric exposure

Pacemaker automaticity, conduction and repolarization are all affected by hyperbaric exposure.

Experiments on subjects with prolonged exposure to hyperbaric air, between 1 and 40 metres of equivalent seawater depth, in a dry chamber, verified that the hyperbaric air caused an increase in parasympathetic tone of sufficient magnitude to induce cardiac dysrhythmias.[8] Hyperoxia is one cause of this, and nitrogen has been incriminated as a cause of beta-receptor blockade. Associated with the reduction in heart rate and the increase in QT interval in the ECG, asymptomatic supraventricular dysrhythmias (generally AV nodal escape rhythms) were seen in 10 per cent of the subjects. Oxygen at high partial pressures acts as a vasoconstrictor, increasing blood pressure and reducing the heart rate.

Electrolyte and acid–base changes

Many of the effects noted above will influence the cardiac contractility and efficiency. The influence of exercise on both pH and metabolic biochemical changes has been mentioned. The production of hypocapnia following cold-water stimulation and hyperventilation, diuresis and changes of blood volume from the effects of immersion, and the effects of saltwater aspiration, will all ultimately act towards altering the *milieu interieur*. These effects will be aggravated when superimposed on pathological changes of age and disease.

Acidosis, produced by the effects of exertion, breathing against a resistance (regulator and increased gas density) and carbon dioxide accumulation, reduces the ventricular fibrillation threshold and depresses myocardial contractility. Underwater

exertion, while breathing through scuba regulators, can produce end tidal carbon dioxide levels of above 70 mmHg.

The inter-relationships of many of the above factors may lead to either cardiac dysrhythmias or myocardial ischaemia. The dysrhythmia is made more likely by the occurrence of sympathetic and parasympathetic stimuli separately or combined, when there is a potentiation more than merely a summation of effects. The ischaemia may be due to diminished cardiac output, coronary occlusion or spasm, and increased metabolic demands of the myocardium.

Drugs

Certain drugs are likely to increase the possibility of cardiac events during diving, including:

- Alcohol
- Nicotine
- Caffeine
- Cocaine
- Sympathomimetics
- Beta-blockers
- Calcium channel blockers
- Pro-arrhythmic drugs.

Many of the anti-hypertensive drugs cause an interference in autonomic regulatory control of the heart rhythms, reduced exercise tolerance, an exertional syncope, syncope when climbing from the water and increased restriction of the airways (especially from beta-blockers).

As demonstrated with quinidine and its derivatives, **anti-dysrhythmic drugs** can become pro-dysrhythmic, under certain circumstances.[9] This is especially so with class I/III drugs, and the effect is not necessarily dose-related (except for sotalol). They may affect the original dysrhythmia by increasing its incidence, its frequency or its haemodynamic consequences. They may also induce new dysrhythmias due to their effect on re-entrant circuits, or **torsade de pointes** due to marked QT prolongation on repolarization. They may produce more bradycardia and increased ectopic beats, triggering the induction of spontaneous clinical re-entrant dysrhythmias.

The calcium channel and the beta-blockers have some characteristics in common. They both induce a bradycardia, aggravating the reflex and metabolic effects mentioned above. A ventricular rhythm breakthrough is then possible. In both cases the ventricular dysrhythmias are more likely if the diver has coincidental or occult coronary artery disease or conductive abnormalities.

Cocaine is infamous for its ability to cause ventricular dysrhythmias and sudden death, even in young, fit athletes.

PRE-EXISTING CARDIAC DISEASE

In younger divers, the presence of myocarditis (often associated with generalized infections) or undiagnosed hypertrophic cardiomyopathy may predispose to sudden death. Underlying cardiac disease, especially genetic disorders, may be completely asymptomatic and lead to sudden death in the apparently young and healthy. Inherited long QT syndrome was diagnosed in an apparently healthy swimmer,[10] and has been noted as a cause of paediatric drowning. Other drowning due to this and similar abnormalities may be undetected unless specific investigations are undertaken. Persons with Marfan syndrome are at risk of sudden death due to aortic dissection.

A study of sudden death in young competitive athletes revealed hypertrophic cardiomyopathy as the cause in 36 per cent, and coronary anomalies in 19 per cent. Ruptured aorta, myocarditis, dilated cardiomyopathy, mitral valve prolapse, premature atherosclerosis and aortic stenosis each had incidences of 3–5 per cent.[11]

Structural lesions found at post-mortem examination in cases of sudden cardiac death during occasional sporting activity are summarized in Table 39.1 Undiagnosed endocardial fibrosis was found at post-mortem examination in a 27-year-old scuba diver, illustrating the importance of pre-dive medical assessments and also the stresses of diving on a diseased heart.[14]

With increased age, there is an increased likelihood of pathology mainly associated with coronary artery disease and hypertension.

The risks of sudden death in apparently healthy men is markedly increased where routine ECGs have detected ST- or T-wave abnormalities, especially in

Table 39.1 *Cardiac lesions found after sudden death during sporting activity*

Younger group (aged <30 years)	Older group (aged >30 years)
• Hypertrophic cardiomyopathy	• Atherosclerotic coronary artery disease
• Arrhythmogenic right ventricular cardiomyopathy	• Hypertrophic cardiomyopathy
• Non-atherosclerotic coronary disease	• Bundle of His abnormalities
• Dilated cardiomyopathy	• Dilated cardiomyopathy
• Aortic stenosis	• Arrhythmogenic right ventricular cardiomopathy
• Atrial septal defect	• Obstructive cardiomyopathy
• Bundle of His abnormalities	

Adapted from Tabib et al.[12]

Table 39.2 *Characteristics of sudden deaths associated with strenuous physical exertion or sports in young persons*

Characteristic	Value
Age range (yr)	11–35
Prior symptoms (% of patients)†	26
Prior question of cardiac abnormality (% of patients)	26
Structural heart lesion (% of patients)	95
Cause (% of patients)	
Hypertrophic cardiomyopathy	23
Congenital coronary anomaly	16
Coronary artery disease	12
Myocarditis	9
Right ventricular cardiomyopathy	8
Idiopathic left ventricular hypertrophy	7
Mitral-valve prolapse	5
Conduction system disease	4
Aortic dissection	4
Dilated cardiomyopathy	1

†Chest pain, syncope, or palpitation.
From: Liberthson.[13]

association with ventricular ectopic beats or left ventricular hypertrophy.

The rise in heart rate is an integral part of the cardiovascular response to exercise. However, in the transition from rest to exercise, older individuals do not adapt as well, having a less adequate increase in both heart rate and cardiac output. Occasionally, tachycardia may be excessive and result in a decrease in efficiency by interfering with venous return and thereby reducing cardiac output. The reduction in diastolic coronary blood flow may precipitate myocardial ischaemia.

Some divers continue diving despite them having known cardiac disease, and these patients are likely to have increased risk of the sudden death syndrome. This is from both the disease entity aggravated by the dive and drugs being taken. Following myocardial infarction, the likelihood of dysrhythmias is increased. Underwater swimming has been shown to increase extrasystoles in these patients. Also, awareness of angina is rare underwater, compared to an equivalent land exercise, with similar ST depression. Persons with aortic stenosis are at particular risk of syncope on exercise.

The increased risk of infarction and dysrhythmias, following coronary by-pass surgery, angioplasty or stenting, is observed even on land. Skilled history-taking, including a search for predisposing factors such as family history, diabetes and hypertension, should help to identify those divers who are at risk.

The investigations in questionable cases include standard 12-lead ECG, which may reveal many of the conditions above such as hypertrophic obstructive cardiomyopathy, left ventricular hypertrophy, ischaemic or previous infarction or long QT syndromes. Maximal stress electrocardiography, with 24-hour Holter monitoring, and echocardiography may then follow. A computed tomography calcium score may prove to be the best screening test for coronary artery disease.

The chances of surviving a dysrhythmia or infarction are markedly diminished if the event occurs underwater. For assessment of cardiac fitness for diving, see Chapters 52, 53, 54 and 56.

BREATH-HOLD DIVING

The dysrhythmias identified with breath-hold diving are mainly those of alterations in respiratory

patterns, submersion, exercise and cold exposure, many of which may have implications for scuba diving.

Dysrhythmias are found even in common respiratory manoeuvres, such as deep inspiration, prolonged inspiration, breath-holding and release of breath-holding. The effects of breathing against resistances, 'skip' breathing and the changes in respiratory pattern that occurs with snorkel and scuba diving are presumably related, but are yet to be investigated.

Submersion (see above) increases the central blood volume, elevates arterial pressure and stroke volume.

The exercise of swimming fast underwater for 50 metres produces a heart rate of about 55 beats per minute. This requires an oxygen consumption of five to ten times the resting level, and would result in a rate of about 180 beats per minute on land!

The **bradycardia** of breath-hold diving is greatest when exercise is combined with cold-water exposure. There are a dozen reports of the bradycardia being 20 beats per minute or less, with half a dozen at 10 beats per minute, or less.

When **asphyxia** is superimposed on breath-holding apnoea, the stimulation of carotid body chemoreceptors is markedly enhanced and may lead to vagally mediated cardiac arrest.

PULMONARY OEDEMA

Immersion and cold increase the venous return to the heart (preload), with cold increasing the work of the heart (afterload) by vasoconstriction. The combination of immersion and cold exposure might therefore be expected to precipitate heart failure in those with impaired cardiac function.

Those cases with overt cardiac disease, especially with ischaemic heart disease, left heart failure from any cause or dysrhythmias as described above, are likely candidates for pulmonary oedema. However, there are reports of otherwise healthy individuals developing pulmonary oedema when swimming or diving.[15–17] Symptoms range from acute dyspnoea (with or without pink frothy sputum) to mild with haemoptysis. The cause is uncertain, but has been associated with cardiovascular changes associated with diving. Accentuated vasoconstrictor responses sufficient to lead to cardiac decompensation and pathological exercise-induced increases in blood flow have been postulated. Such people are at increased risk of recurrence and should probably not continue to dive. In Wilmshurst's series[17] of 11 such patients, nine demonstrated a pathological vasoconstriction and nine showed signs of cardiac decompensation when stimulated by cold. Follow-up many years later showed the development of hypertension in seven patients, Raynaud's phenomenon in one patient and atrial fibrillation in one patient. There were no cardiovascular events and/or deaths. The case histories showed a relationship between the episode of pulmonary oedema and exposure to cold. Further series suggest that the disorder may not be that rare, and that very cold water is not always associated.[18]

Additional mechanisms may be involved in swimmers and breath-hold divers, where a number of cases have been reported.[19] Head-out water immersion increases the cardiac preload because of the increased intrathoracic blood volume. Negative-pressure breathing is also increased because of the pressure differential between mouth and thorax. Deep breath-hold dives could reduce lung volume to below residual volume, which is compensated by a further increase in intrathoracic blood volume. The rise in transpulmonary capillary wall pressure leads to fluid shifting into the interstitial space and thus into the alveoli, leading to alveolar oedema and haemorrhage.

In some of the reported cases concurrent use of aspirin was thought to be contributory. Extreme exercise is also associated with pulmonary oedema in non-aquatic sports.

For a differential diagnosis of pulmonary oedema, see Chapter 42.

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40

Neurological disorders of diving

ROBYN WALKER

INTRODUCTION

Due to the extreme vulnerability of nerve cells to ischaemia, the nervous system is a primary target in diving accidents. Combined with the nervous system response to heightened environmental stimuli and its sensitivity to gas mixtures other than air, this may result in a plethora of effects related to diving.

Scuba diving may result in neurological injury, with both temporary and permanent impairment.[1,2] Many of these problems have been dealt with in previous or following chapters including cerebral arterial gas embolism (CAGE; see Chapter 6), neurological decompression sickness (DCS; Chapter 12), hypoxic encephalopathy (Chapter 16), nitrogen narcosis (Chapter 17), the high-pressure neurological syndrome (HPNS; Chapter 20) and neuropsychological disorders (Chapter 41).

Others include headaches in diving, brachial plexus lesions, scuba diver's thigh, epilepsy and other neuralgias and nerve lesions.

CEREBRAL ARTERIAL GAS EMBOLISM

Bubbles in the arterial circulation will travel largely to the brain (see Chapter 6) where they may lodge and cause infarction in the affected area, or alternately pass through the capillaries into the venous system and again travel back to the lungs. Bubbles damage the vascular endothelium as they pass through the blood vessels, and are thought to

activate the inflammatory cascade and impair the blood–brain barrier.

Serious symptoms, which develop immediately after an ascent, should usually be treated as air embolism. The manifestations are usually acute and may include:

- loss of consciousness;
- other neurological abnormalities such as confusion, aphasia, visual disturbances, paraesthesiae or sensory abnormalities, vertigo, convulsions, varying degrees of paresis;
- gas bubbles in retinal vessels;
- abnormal electroencephalograms (generalized slowing or flattening of waves) and abnormal brain scans, etc.

NEUROLOGICAL DECOMPRESSION SICKNESS

The nervous system is very frequently involved in decompression sickness (DCS) (see Chapter 12). Symptoms of brain involvement range from difficulty in concentration or thinking, mild confusion and impaired judgement through to frank confusional states and loss of consciousness. Spinal cord involvement occurs frequently and presents with bladder, motor and sensory disturbance.

Peripheral nerve involvement is also frequent, often only sensory and less frequently recognized. It may be more bilateral and widespread in nature than other neurological manifestations, but is sometimes patchy and only found by careful examination. It is important to differentiate this from the cerebral and spinal manifestations, which are more ominous.

SUBCLININCAL NEUROLOGICAL DAMAGE

Studies are available which provide evidence of chronic changes in the brains of asymptomatic divers who do not have a past history of DCS when compared with control subjects (see Chapter 44). There are also a number of anecdotal reports in the literature, which have generated the hypothesis that diving in the absence of a gross insult causes brain damage and dementia in divers. Reputable studies to support this hypothesis are lacking (see Chapter 41).

HYPOXIC ENCEPHALOPATHY

Hypoxia, most often seen in association with closed or semi-closed rebreather units, may occur as a result of exhaustion of the gas supply, failure of the diving equipment, inadequate flow rates, dilution and increased oxygen consumption (see Chapter 16). It also occurs in divers who survive a near-drowning. The clinical features are those of hypoxia from any cause and are not unique to divers.

NITROGEN NARCOSIS

Nitrogen narcosis refers to the clinical syndrome characterized by impairment of intellectual and neuromuscular performance and changes in mood and behaviour. The effects are progressive with increasing depth and reverse completely with ascent to the surface (see Chapter 17).

HIGH-PRESSURE NEUROLOGICAL SYNDROME (HPNS)

HPNS (see Chapter 20) occurs at depths greater than 200 metres (helium being used as the diluent gas), and is the current limitation to deep diving. Clinical features include:

- tremor;
- fasciculation;
- myoclonic jerks;
- psychomotor performance decrement;
- dizziness, nausea and vomiting; and
- impaired consciousness.

The onset of HPNS can be delayed by slowing the rate of compression and introducing a narcotic gas, e.g. nitrogen to the helium mixture.

NEUROPSYCHOLOGICAL DISORDERS

Because cerebral involvement is present in many of the major diving accidents, neuropsychological sequelae are relatively common. They may be evident from the time of the accident, or develop soon after. They may also be persistent and may be associated with abnormal electroencephalograms or neuropsychometric testing.

Anecdotal reports have been presented to suggest that diving *per se* is associated with, in the absence of clinical neurological disease, neuropsychological damage and possible dementia. These reports have not been substantiated, and are discussed at length in Chapter 41.

HEADACHE

This is a common symptom in diving medicine, but is not usually well documented. The following causes are not all inclusive, and the clinical details of each type of headache are to be found either in the appropriate sections of this book, or in general medical texts. The differential diagnosis will depend on a detailed clinical and diving history, a physical examination and laboratory investigation.

Anxiety (tension)

The psychological reaction, induced in susceptible novice divers exposed to a stressful underwater environment, may produce a typical tension headache.

Sinus barotrauma

Pain occurs during the diver's change of depth caused by the volume changes on the sinus gas spaces. Barotrauma of descent affecting the frontal sinus is the most common, and is often relieved by ascent. Ethmoidal sinus pain is referred to the intra-orbital area and maxillary sinus pain may be referred to the teeth. Sphenoidal sinus pain may be referred to the parieto-occipital area (see Chapter 8).

Sinus pathology

Mucocele or other sinus pathology can be produced by diving. Rupture of the cells in the ethmoidal sinus air cells can cause a sudden and explosive headache and result in a small haematoma or bruising below the glabella, at the root of the nose, following sinus barotrauma. A similar explosive headache can develop, often during ascent and following middle-ear barotrauma, with rupture of the mastoid air cells causing a generalized pain, localizing later to the mastoid region. Pneumocephalus can follow the sinus rupture (see Fig. 9.4). Computed tomography (CT) skull scans demonstrate these lesions with precision.

Infections

Infections in the mastoid cavities or sinuses usually cause pain 4–24 hours after the dive, and are commonly associated with a pre-existing upper respiratory tract infection and/or barotrauma. Other generalized infections, including *Naegleria* spp., are related to marine exposure (see Chapter 30 for more detail).

Cold

In some subjects, exposure to cold water may induce a throbbing pain particularly over the frontal area, but sometimes also including the occipital area. This is probably analogous to the head pain experienced by some people when eating cold food, such as ice cream. The onset maybe rapid after cold-water contact, or it may progressively increase in intensity with the duration of exposure. It usually remains for some minutes after the diver has left the water. Whether this is a migraine variant, a neurocirculatory reflex or merely due to an increase in muscular tone is not known. Prevention is by the use of a protective hood to ensure warmth.

Saltwater aspiration

Headache following aspiration of seawater usually follows a latent period of 30 minutes or more, is usually associated with myalgia and is aggravated by exercise and cold.

Mask tension

Inexperienced divers tend to adjust the facemask straps far too tightly, and this may result in a headache not dissimilar to that of a tight hat, misfitting spectacle frames, etc. due to direct local pressure effects. It is related to the duration of the dive, and clears in an hour or so. A similar disorder, called 'swim goggle' headache, may be related to migraine.

Gas toxicity

Specific gas toxicities sometimes cause a characteristic headache. The carbon-dioxide induced headache usually develops with a gradually increasing carbon dioxide tension, or follows a reduction in a sharply rising carbon dioxide tension, i.e. a carbon dioxide 'off effect'. The carbon dioxide-induced headache is throbbing in nature, lasts a few hours and is not relieved by analgesics or anti-migraine preparations. Headaches have also been described with oxygen toxicity, carbon monoxide toxicity and other gas contaminations.

Decompression sickness and pulmonary barotrauma

Headache is an ominous symptom in both neurological DCS and air embolism from pulmonary barotrauma. It is associated with intracerebral bubbles and/or raised intracranial pressure. Usually it arises within minutes of ascent, and is suggestive of bubbles of intravascular origin. Other neurological manifestations and a disturbance of conscious state are often associated. The headache may persist for a week or more, but is rapidly relieved by recompression therapy. Its recurrence is indicative of deterioration in the patient's neurological state and is also amenable to recompression or oxygen therapy. Pulmonary barotrauma and DCS are discussed in detail in Chapters 6 and 12.

Migraine

Traditionally migraine sufferers have been advised not to take up diving. Attacks are rarely induced by the diving environment, but may be of greatly increased severity when they do develop. The precipitation of the migraine attack may be due to any of the other headache-producing stimuli discussed in this section, e.g. cold, anxiety, oxygen or carbon dioxide tensions, intravascular bubbles, etc.

Once a migraine attack has commenced during diving, the patient is at considerable risk from the following: disturbance in neurological function, i.e. interference of consciousness, perception and motor activity; vertigo; psychological complications; and vomiting. A number of subjects have had their first migraine episode under water – although there is usually a positive family history to aid in diagnosis. This may lead to diagnostic confusion with cerebral decompression sickness and/or air embolism.

Prevention is best achieved by either not diving, or avoiding the specific provoking stimulus, e.g. use of a wet suit hood if cold is the precipitant, or limit of diving exposures to shallow no-decompression diving with generous safety margins. Safety techniques such as buddy diving are also very important for those migraine subjects who insist on diving.

Neuromuscular pain

Headaches produced by the environment or by locomotor stress may produce severe pains that are difficult to assess and diagnose. Many such patients give consistent accounts of headaches being induced by diving, and sometimes only by scuba diving, but with none of the specific features mentioned above. Some may be psychogenic or tension headaches, whereas others seem more vascular in nature.

One specific type, due to minor degrees of cervical spondylosis, may be confirmed by cervical spine X-rays. In these there may be loss of lordotic curvature, narrowing of intervertebral spaces and osteophytosis in the lateral views. Many divers who develop this disorder are in the older age bracket or have a history of head and neck trauma. They often swim under water with flexion of the lower cervical spine (to avoid the tank) and their upper cervical spine hyperextended (to view where they are going). This produces C1, C2 and C3 compression and distortion of the cervicocranial relationships, an unnatural posture aggravating

underlying disease, which may otherwise be asymptomatic.

The headache is usually occipital and may persist for many hours after the dive, and the area is often tender on palpation. Persistent occipital neuralgia can have a similar aetiology. Occasionally the pain is referred to the top and front of the head, possibly due to fibres of the trigeminal nerve passing down the cervical cord and being affected by damage to the upper cervical vertebrae.

Exertional headache

A sudden headache, which may be excruciating in intensity, may be precipitated by exertion and last for several hours.[3] It can be prevented by a gradual increase in physical activity and treated with non-steroidal anti-inflammatory drugs (NSAIDs).

Other causes

Many other obvious reasons may be incriminated in the aetiology of headache in divers. These include such diverse factors as alcohol overindulgence, head injury usually sustained during ascent, glare from the sun, and drugs such as vasodilators and calcium channel blockers.

Some causes of headache in diving:

- *Anxiety*
- *Sinus barotrauma, sinusitis and other pathology*
- *Cold exposure*
- *Saltwater aspiration*
- *Tight facemask straps*
- *Carbon dioxide and carbon monoxide toxicity*
- *Decompression sickness*
- *Pulmonary barotrauma*
- *Migraine*
- *Cervical spondylosis*
- *Drugs*
- *Exertion*

EPILEPSY

Epilepsy is a total contraindication to diving, but not infrequently the first epileptic convulsion develops under water. In some cases the cause may be obvious, such as oxygen toxicity (Chapter 17), cerebral arterial gas embolism (Chapter 6) or neurological DCS (Chapter 12).

A variety of medical causes also could be responsible, including previous idiopathic epilepsy, hypoglycaemia, cerebrovascular accidents, cerebral trauma or tumours. These are the easier cases to diagnose, and the management extends beyond the diving situation. The dilemma is the case without an established cause, and this is the most frequent situation in recreational diving. It appears as if diving lowers the seizure threshold. It is commonly seen in oxygen convulsions, where 'dry' exposures can be tolerated much longer than in-water exposures. It is possible that the reasons for non-specific convulsions are multifactorial and include:

- sensory deprivation;
- hyperventilation from positive-pressure demand valves;
- nitrogen narcosis (sedation);
- acidosis from carbon dioxide retention;
- anxiety; and/or
- misdiagnosis of hypoxic and carbon dioxide convulsions.

Some of these conditions have been used in general medicine to provoke convulsions.

Management of an epileptic attack under water poses unsolvable problems. The confusional state during the aura and post-ictally may cause unpredictable behaviour. The fear of pulmonary barotrauma during ascent, at least in the tonic phase, has to be weighed against the likelihood of drowning during the clonic and post-ictal phase, when the diver is unconscious. It is our experience that the diver is best served by maintaining the mouthpiece *in situ* and risking an ascent preferably in a face-down position until the surface is reached. This is certainly so once the clonic phase has begun.

Subsequent management includes the exclusion of other causes, appropriate investigations, exclusion from diving and the warning that subsequent episodes may occur on land, even years later.

BRACHIAL PLEXUS INJURY

This disorder was related mostly to the use of standard diving equipment (now used mainly as a tourist attraction as opposed to working dress), where the weight of the helmet is taken directly on the supraclavicular region. This may be from mishandling the helmet or by having inadequate or incorrectly placed padding over the area between the neck and shoulder. It is more likely to be caused out of the water, when the weight factor is greatest. The standby diver is thus more prone to this disorder.

The middle and lower cervical nerves are more likely to be involved, i.e. the fifth to seventh, and this may be either temporary or permanent. The minor cases present with paraesthesiae and numbness of the lateral aspect of the arm, forearm, thumb and adjacent fingers. Severe cases result in both motor and sensory damage over the affected nerve distribution. Rigid shoulder harnesses of scuba can also produce this.

SCUBA DIVER'S THIGH (MERALGIA PARAESTHETICA)

The lateral femoral cutaneous nerve is vulnerable to compression neuropathy by pregnancy, tight trousers, pelvic tilt, harnesses and low-positioned weight belts. It results in numbness over the upper thigh, anteriorly and laterally, but it usually clears up in a few months.

NEURALGIAS AND OTHER NERVE LESIONS

Chronic and sometimes severe pain, referable to either nerves, nerve roots or plexus, may follow neurological decompression sickness,[2] when myelin sheaths may be damaged. Spinal cord lesions may also be responsible for some of these cases.

Involvement of the second (optic) cranial nerve can follow DCS or oxygen toxicity. The eighth (auditory and vestibular) nerve can be damaged with both inner ear barotrauma and DCS (see Chapters 7 and 12).

Many of the fish poisons and marine toxins may produce a variably persistent bilateral peripheral neuropathy. Marine envenomations may produce adjoining localized neuropathies.

Involvement of the trigeminal and facial nerves may follow sinus or middle-ear barotraumas (see Chapters 7 and 8).

PARAESTHESIAE

Diving-induced paraesthesiae

The causes of diving-induced paraesthesiae include most of the causes of peripheral neuropathy referred to previously in this chapter. Specifically these would include localized or segmental paraesthesiae associated with DCS (usually unilateral or patchy or affecting both lower limbs or girdle), barotrauma-induced neuropathies affecting the fifth and seventh cranial nerves, and meralgia paraesthetica.

Paraesthesiae can be anxiety-induced, often associated with DCS, either actual or feared, and its subsequent treatment. This will often affect younger and possibly less experienced divers, who may have an increased trait anxiety level. It will present mainly as paraesthesiae affecting the hands, probably related to anxiety-induced hyperventilation, but usually not to a degree causing carpopedal spasm or loss of consciousness.

It is usually able to be ameliorated by rebreathing techniques (traditionally into a brown paper bag) but also by breathing carbogen (95 per cent oxygen, 5 per cent carbon dioxide) and by increasing the work of breathing – which occurs with recompression, sometimes giving a misleading impression of DCS 'cure'. It also responds to both reassurance and anxiolytics, although the latter is not currently fashionable.

Paraesthesiae may also be induced by a Raynaud's reaction to cold water exposure. This is specially seen in females and is associated with a cold, vasoconstrictive response, usually bilateral and affecting the hands which appear pale.

Other forms of cold-induced paraesthesiae occur in those predisposed because of pre-existing peripheral neuropathy or drugs. The carbonic anhydrase inhibitors typify the latter, either taken orally or as guttae (glaucoma treatment).

Some fish poisonings, especially ciguatera, will induce paraesthesiae and may be experienced during the diving vacation. Marine venoms, especially those of the coelenterates, but also with other marine stingings, can produce localized neuropathies with paraesthesiae.

Altitude-induced paraesthesiae

DCS paraesthesiae provocation or aggravation may be due to expansion of bubbles or gas nuclei affecting the cerebral, spinal or peripheral neurological system.

Paraesthesiae may occur during flight as a result of stimulation of previously damaged neural tissue due to altitude effects on respiration – especially hyperventilation associated with the anxiety of air travel (increased if there is a fear of DCS), causing hypocapnoea and mild alkalosis. This is complicated by the reduction of density of the environmental air. Hypoxia at altitude might also increase this. Relief may be obtained by anxiolytic medication, oxygen breathing and rebreathing systems, as described above. This disorder is commonly misinterpreted as a recurrence of DCS, and may be unnecessarily treated by recompression if an anxiety state continues once the diver has returned to sea level.

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41

Psychological and neuropsychological disorders

CARL EDMONDS

By far the most important psychological problem in diving, is **panic**. *It contributes to 39% of the recreational diving deaths (see Chapter 45).*

Other less catastrophic reactions include the psychoneuroses, i.e. personalized reactions to the environment and/or equipment.

Mild confusional states often accompany diving disorders. The post-traumatic stress disorder (PTSD) is encountered after serious accidents, as is symptomatic depression. Therapists attitudes and the therapeutic situation itself may contribute to post-treatment sequelae.

Brain damage, with its myriad of neuropsychological presentations, is due to some specific diving disorders (decompression sickness, cerebral arterial gas embolism, hypoxia, carbon monoxide toxicity), but is unlikely without such clinical incidents.

OVERVIEW

Psychiatric problems can be subdivided into those which are organic and those which are functional. The **functional disorders** are commonly referred to as psychological, neuroses or 'nervous illnesses', and are characterized by disturbances of feelings, attitudes and habits severe enough to impair the diver's life or to reduce efficiency. They are 'personalized' reactions to stress. The symptoms are usually those of anxiety and/or depression, but occasionally with hysterical or obsessional features.

Organic disorders are presumed to have a physical cause, e.g. damage to the brain from trauma, toxins, metabolic diseases or neuronal degeneration. They range from acute toxi-confusional states to dementia and are often associated with irritability, depression and cognitive changes.

BEHAVIOURAL CHARACTERISTICS

Psychological traits of divers

The relationship between personality and diving is complex.[1–4] Successful divers are characterized by an average or below-average neuroticism level. The psychological mechanism of denial, in which the subject refuses consciously to acknowledge the hazards, is thought to have adaptive value under some diving conditions. The combination allows professional divers to continue to work despite stress which would be disruptive to many 'normals'. Psychometric profiles show that intelligence is positively correlated with successful diving, as are emotional stability and self-sufficiency. Some aberrant behaviour of divers might be transitory, occur soon after diving, and be due to the organic disorders referred to below. It may also be due to constitutional factors.

Divers are, by nature and selection, **risk takers**, and this was even more so in past decades when the risks in diving were extreme. They also approach psychologists and questionnaires with a risk-taking style that may well produce variances between their results and those of 'normal' controls.

Because divers are risk takers, and physically active individuals, they are more likely to engage in activities that attract attention from law-enforcement authorities. In this way they are probably similar to Icelandic seamen, who also have a high incidence of accidents, violence, homicide and suicide.

Suicide has been shown to be responsible for 17 per cent of the deaths of professional divers in the UK. **Accidents** accounted for 48 per cent of their deaths (diving/drowning 28 per cent and non-diving 20 per cent), and the death ratio was significantly higher than expected after correction for age.

PSYCHOLOGICAL DISTURBANCES

Psychoneuroses[5,6] refer to the abnormal response of the individual to stress, but also normal responses to abnormal and excessive stress – when they may disrupt the diver's life.[7–10] Some psychological disturbances experienced during diving are well known, but poorly documented. Examples of such disturbances are outlined in the following sections.

Anxiety and panic

Anxiety and panic is often associated with hyperventilation, inducing paraesthesia, dsypnoea, tachycardia and other cardiac-type symptoms, disturbance of consciousness, carpopedal spasm and hyperreflexia, globus hystericus and abdominal symptoms (see Chapter 45 for a detailed account of the aetiology and importance of this subject). The hypocapnia, which causes many of the symptoms, is corrected by rebreathing and by hyperbaric exposure – and often leads to a false diagnosis of decompression sickness (DCS).

Phobic anxiety states

In susceptible people the normal anxiety induced by diving may be complicated by an overawareness of potential but definite dangers, with a resultant increase in anxiety. Apprehension, palpitations, increased rate of breathing, and epigastric sensations are symptoms of anxiety and interpreted as indicative of 'something wrong'. A vicious circle results, and the diver may then develop an actual **phobia** to being under water, or confronting marine animals.

CASE REPORT 41.1

AR, a 21-year-old diver with limited open water experience dived from a boat into clear water, without tidal currents. The water was 70 metres deep, although the diver was no deeper than 15 metres. Approximately 10 minutes after commencing the dive AR noted a feeling of fear of the deep blue water. His breathing became fast and he started to panic with an overwhelming desire to return to the safety of his boat. He ascended to the surface, to swim back to it. His anxiety remained until he saw the reef coming into his visual range, 5 metres below him. Once back on board he felt quite well.

Diagnosis: agoraphobic reaction.

Some candidates develop this before attempting a diving course, and realize that they would be apprehensive in such an environment. Other motivating factors may temporarily override this fear. In some cases there is a history of traumatic exposure to water (such as a near-drowning incident) which initiates the phobic state.

Cases of genuine **claustrophobia** prevent immersion or being confined in a recompression chamber. In the water, this syndrome may only present during times of diminished visibility (murky water, night diving, wearing a facemask, etc.) or prolonged exposure.

The **agoraphobic reaction** is also termed the **blue orb** or **blue dome syndrome**, from aviation medicine. It develops progressively, as the diver becomes more aware of his isolation and his lack of contact with people or objects. It may be aggravated by nitrogen narcosis, at depth, and sensory deprivation (see Chapter 38). The diver is usually alone, without physical or visual contact to diving craft or sea bed. The fear is one of isolation in the vastness and depth of the water.

If reassurance, in the form of a companion diver or visual fixation on familiar objects, is not available, then the diver may panic and ascend rapidly. Drowning, DCS or pulmonary barotrauma are possible complications. If the diver regains physical or visual contact with the sea bed, the diving boat or a companion diver (or even focuses on his own equipment), the symptoms usually abate.

All phobic anxiety states can be treated by desensitization or deconditioning techniques. They are prevented by avoiding the environmental circumstances which predispose them, and also by repeated diving exposures under supportive tuition. The use of sedatives is to be discouraged, but anxiolytics may be of value during the deconditioning process.

Somatiform disorders

This involves symptoms that were originally combined under the term **hysteria**. A common feature is a complaint that is somatic in nature, but with an origin that is psychological. The aetiology is described in psychological terms, but the symptom (frequently pain) is a means of eliciting care for an individual in distress – one who is otherwise unable to express this. As a general rule, this disorder pattern is established before the age of 30; otherwise the symptom is more likely to be based on a genuine organic disorder. For those clients who develop functional pain, one should always exclude a depressive background. In this situation treatment of the depression is integral to removal of the symptoms.

The classical production of **conversion** is a disorder consequent to trauma. An intolerable experience or feeling or memory is overcome by repressing that experience. Concurrently with this a somatic symptom arises. Most of these presenting symptoms are neurological in nature, but are functional in causation. There is thus a primary gain achieved by the patient. The secondary gain may be produced by the social environment surrounding the now invalid client.

The essential feature of **hypochondriasis** is a preoccupation with, and a fear of having, a serious disease – usually based on hypersensitivity to or misinterpretation of physical sensations, which are

used as evidence of illness. This is frequently seen after divers have been suspected of possibly having DCS, and treated accordingly. Some of these patients will continue to have recurrences of DCS-type symptoms months or years after the event, despite reassurances and evidence of their well-being.

In such cases, if it is thought necessary to investigate to exclude other causes, then the investigations should be performed as rapidly as possible so that the period of diagnostic uncertainty is reduced to a minimum. Reassurance should be as prompt as possible, as continuous interest and concern on the part of the physician can promote and prolong the disorder. It is for this reason, as well as many others including the post-traumatic stress disorder (PTSD), that unnecessary hyperbaric treatments should be avoided. These induce loss of both self-control and self-esteem.

The **Baron Von Munchausen syndrome** may be the basis of some false claims related to diving. A number of cases have presented with a diving history and clinical symptomatology suggestive of a diving disorder, usually DCS. The patient may move from one hyperbaric unit to another, for repetitive treatments – presumably either as an attention-seeking device or for the warmth and support that such a therapeutic unit may supply. These cases are the aquatic equivalent of the terrestrial Munchausen syndrome. There is a similar syndrome recorded in caisson workers.

CASE REPORT 41.2

During a flight to Sydney, Miss P.H., aged 30, developed episodes of breathlessness and unconsciousness with a convulsive state. Between episodes she explained to the stewardess that she had been diving to 50 metres for approximately 35 minutes, 2 hours prior to the flight.

On examination at a Sydney hospital she complained of pain on deep inspiration and a slight ache in the right knee that was exacerbated by movement. Over the next few hours she had three grand mal convulsions with epistaxis and periorbital petechiae. The pain in the right knee had become worse, and there was an overlying area of diminished sensation. A diagnosis of decompression sickness was made and treatment arranged at a nearby recompression chamber.

Prior to recompression she was conscious, rational, and gave a history of the dive in some detail. Apart from the area of diminished sensation over the right knee and decreased knee and ankle reflexes, there were no other neurological signs detected. She was placed in the recompression chamber and pressurized to 18 metres on 100 per cent oxygen initially, and improved symptomatically. During decompression she had three grand mal epileptic convulsions. Upon recovery she complained of worsening of her knee and chest symptoms. She was therefore pressurized to 50 metres, where she again showed improvement, but again subsequently deteriorated. Following a series of epileptiform seizures, she became unresponsive to vocal and painful stimuli for some time. Some hours later, after consciousness had returned, she complained of severe abdominal, chest, right hip and right knee pain.

At this stage investigations by telephone revealed that she had not, in fact, been diving. She had a long psychiatric history of hysterical symptoms, genuine epilepsy, and a recent interest in a *Skin Diver* article on decompression sickness. She stopped her anti-convulsant treatment about 24 hours before presenting for treatment. Because of the long duration of exposure to pressure of both the patient and the attendants, subsequent decompression proceeded at a slow rate. The chamber reached the 'surface' after approximately 48 hours.

Provisional diagnosis: an aquatic Baron Von Munchausen syndrome.

Illusions

Sensory deprivation, especially impaired diving visibility, is likely to aggravate the tendency to misinterpret stimuli. Anxiety associated with diving results in heightened suggestibility, e.g. terror on sighting unexpected objects, mistaking another diver for a shark, etc.

Post-traumatic stress disorder (PTSD)

General

As with motor vehicle accidents and other catastrophes (war, earthquakes, volcano eruptions, bush fires, mass shootings, murder, rape and home invasion), PTSD is but one of a number of possible psychological sequelae which may co-exist. The others are:

- dissociation (hysterical reaction);
- somatization (conversion of anxiety into a physical disorder);
- depression and bereavement (especially if loved ones have been involved);
- anxiety and hyperventilation;
- compensation neurosis; and
- affect dysregulation (a lack of self-regulation leading to difficulty in controlling anger, or producing self-destructive, suicidal or risk-taking behaviour).

PTSD was defined in 1980, but it was know previously as post-traumatic anxiety, shell-shock, battle fatigue, da Costa syndrome, etc. It is an anxiety disorder that develops in some divers exposed to a severe and usually life-threatening situation. In a lesser degree the PTSD could have survival value, ensuring that we refrain from situations which had previously been experienced as dangerous. It has been claimed that there are anatomical and pathological organic cerebral changes with PTSD, with specific changes in neuropsychological functioning. This is unlikely.

Aetiology

PTSD is more likely to develop in divers who normally have higher levels of anxiety (neuroticism), or when the stress has been particularly severe or prolonged. It is less likely to occur in non-neurotic divers who are trained in the emergency situation and who are more prepared for it.

The usual diving provocations are accidents which cause death of a diving companion or involve the diver in a near-death situation, usually with the threat of drowning. Other aggravating situations include the prolonged stress and trauma of medevac and hyperbaric therapies.

Prevention is achieved by the early application of rest, support and the comfort of one's companions. Unfortunately, in diving accidents – as in aviation and many others – there is a tendency to blame and discredit the victim, by instructors, peers and even therapists. It is a disorder encouraged and overdiagnosed by lawyers and litigonists.

Clinical features

The diver has intense fear that the event may recur, and is preoccupied with this. It might intrude as vivid memories – either dreams, nightmares or flashbacks. He responds by avoidance behaviour, i.e. avoiding things, situations or people that serve to remind him of the event. He may also appear to block out all emotion. There may be other general factors associated with the increased anxiety level; irritability, insomnia, various aches and pains, panic attacks, globus hystericus, hyperventilation, dyspnoea and/or choking sensation.

Treatment

First-aid after the accident should be supportive and reassuring, and yet still encouraging the patient to verbalize the experience. This may have some preventative value, although there is little to support the value of the conventional 'counselling de-brief'. Instead, the victim should be made to feel safe, allowed to rest and support provided. Repeated hyperbaric therapies tend to interfere with this.

The disorder is likely to be evident within a week or two of the event. The customary treatment is for psychological intervention in which the client is encouraged to remember the events in a non-stressful situation, but with the emotional memory expressed. Confidence is engendered and the disorder diminishes.

Unfortunately, PTSD is characterized by avoidance mechanisms, so that the victim tends to stay away from the therapeutic situation that will allow venting of the memories and the emotions. Other treatments that may be required include education about anxiety management, cognitive behavioural therapy and insight-orientated psychotherapy.

Medications may be of value, based on the symptomatology. Modern antidepressants (e.g. selective serotonin re-uptake inhibitors, SSRI) may stabilize the client, while tricyclic antidepressants may be of value if there is any major sleep disturbance. The anxiolytics are no longer fashionable.

Compensation (occupational) neurosis

This includes a range of presentations from malingering to the exaggeration of genuine symptoms for secondary gain. The lucrative occupation of professional diving and the litigious nature of many societies each encourage this disorder – which often bestows rewards to the adversarial consultants, both legal and medical. Frequently, the claimant becomes obsessed by the procedure, and the ultimate financial recompense is offset by a limitation in life style, a loss of self-esteem and withdrawal from the diving fraternity. Protraction of the legal proceedings has negative implications for all except the legal fraternity.

Drug use

During diving and diving medical examinations (see Chapters 53–59) the influences of drugs have to be considered, and this is especially so with recreational drugs and psychotropics. The use of illicit or recreational drugs may induce problems in their own right, such as with cocaine or Kava use. This causes problems with cardiac deaths, especially so in diving situations. Other problems such as the impairment of judgement and cognitive function, may have implications on the diving safety.

Various medical complications may also be related to drug usage, e.g. the withdrawal effects of nicotine, sedatives, anti-depressants, alcohol and narcotics. Other medical diseases such as systemic or hepatic infections may be related to illicit intravenous drug use.

PSYCHOSES

Psychosis would usually preclude a candidate from diving training. Certain psychoses may result in pathological delusions related to diving, a misuse of the diving environment or false claims related to diving. Diving does not cause psychoses.

Schizophrenia may result in a diver developing primary delusions centered on his diving activity, e.g. a paranoia toward sharks, resulting in a personal vendetta against them. The development of a complex delusion system regarding international undersea control, radioactivity, diving inventions, etc. have also been observed.

Affective disorders such as a cyclic, manic-depressive psychosis may be dangerous along both psychological axes. The grandiosity and self-assurance in a hypomanic state are as potentially dangerous as the suicidal inclinations during depression.

Suicide, although not well-recognized, is not a rare event among those who have access to the sea. With the more widespread attraction to this sport

CASE REPORT 41.3

F.R. refilled his scuba cylinder from the local dive shop, and hired a boat and diver to take him on a dive. He quietly read a science fiction book on the way out. He then entered the water, surfaced very soon and asked the boatman to hand him a bag he had bought. He was found the next morning by the water police still wearing his scuba equipment and with a 22-calibre bullet through his brain.

among a greater range of personalities, the incidence must increase. The aetiology of suicide is not different to that of the general population, but the means may differ considerably. Swimming into the oceanic horizon has a certain flamboyant appeal, and has been used by some prosaic souls. One diver completed the suicide formalities by documenting his intent to free ascent while breath-holding, and succeeded in bursting his lungs. Others use mundane methods, despite the exotic environment.

Unfortunately, **psychotropic drugs**, given for therapeutic reasons may cause problems. The side effects of psychotropic medications may include decreasing the epileptic seizure threshold, sedation, cardiac arrhythmias, reduced exercise capability and autonomic nervous system interference. These would increase the hazards associated with scuba diving, especially with inexperienced divers. The influence of drugs on the diving disorders such as stress reactions, dysbaric disease and gas narcoses are usually not well documented, but are potential sources of concern (see Chapter 43).

NEUROPSYCHOLOGICAL MANIFESTATIONS OF DIVING ACCIDENTS

Aetiology

The neurological insults from diving accidents may be many and varied,[11–15] and may summate. There are often neurological signs of cerebral or cerebellar disorder. Causes of permanent intellectual impairment from compressed-air diving include:

- neurological decompression sickness (DCS);
- cerebral arterial air embolism (CAGE) from pulmonary barotrauma;
- carbon monoxide toxicity; and
- severe hypoxia, usually from near-drowning.

Ill-defined and unquantified damage from other gas toxicities are possible, e.g. carbon dioxide, nitrogen, oxygen and contaminants. If extreme, HPNS and hypothermia have been implicated as causes of central nervous system (CNS) damage.

Organic brain syndromes

Delirium (toxic-confusional state)

This is, by definition, a fluctuating and transitory state although it can present briefly during other organic states. A disturbance of consciousness is the main characteristic, and this can be seen in many diving disorders, but especially hypoxia, carbon dioxide toxicity, hypothermia, cerebral DCS, CAGE, marine animal envenomations, nitrogen narcosis and HPNS.

The more florid delirium, with its extreme alertness, produces sleep disruption, disordered speech, perceptual abnormalities (illusions, hallucinations) and disturbances of affect (fear, delusions, paranoia) and disturbances of cognition (especially memory).

Although all these may be present in some of the more florid cases, e.g. those coming out of coma from a near-drowning accident, in other cases the disorder may be more subtle. All that may be evident is unaccustomed behaviour, either hyper- or hypo-activity, drowsiness, slight bewilderment or lapses of memory and judgement. The hyper-alert state may be evident only as an insomnia, and the hallucinations only as vivid dreams.

It is in such cases that the behaviour of the diver, based on his impaired judgement, may be inappropriate, i.e. he may deny symptoms or ignore them. He may insist on driving his vehicle to the recompression chamber. He may forget some of the symptoms he had complained of. Instead of gross confusion, there may be only irritability – which can present as intolerance to advice or treatment.

These patients must be treated gently and with encouragement, not with criticism and impatience, to maintain their cooperation. A common mistake made by hyperbaric clinicians is to confuse the behavioural abnormalities of an **acute organic brain syndrome** – as happens with both CAGE and DCS – with hysterical and psychopathic diagnoses. Thus, the obstreperous diver with an illogical reluctance to enter the recompression chamber, is the patient who may need it most. The best interpreters of apparent behaviour disorder are the diver's colleagues and family. They will recognize the behaviour as atypical, and thus indicate that it may be based on recent brain damage.

Most diving medical facilities will have simple clinical psychological testing procedures to record:

- orientation for time, place and person;
- attention and concentration (serial 7s, reversal of order, etc.);
- recent memory (three objects or words, recalled 3 minutes later);
- abstract thinking (proverbs, differences, etc.);
- speed of responses.

The mini mental state examination, once practised, is of considerable diagnostic value. Some groups even include formal psychological tests, e.g. Ravens Advanced Progressive Matrices, Digit Symbol, Koh's blocks, etc. Such tests may readily demonstrate an abnormality, even though it will not indicate the cause. The same test performed within minutes of an appropriate therapy, may demonstrate improvement.

Electroencephalograms (EEGs) and some of the recent cerebral scanning techniques may also indicate an anomaly.

One of the commonest causes of delirium in general medicine is a head injury, and this is not at all dissimilar to the intracerebral vascular trauma of arterial gas emboli, except that the pathology of the latter may develop more slowly with the emboli-induced vascular damage allowing a diffusion of blood components into the cerebral tissue, for hours after the initiating bubble has passed through the vasculature.

Sub-acute organic brain syndromes

Between the transitory delirium and the chronic dementia, there can be a variety of other syndromes, with characteristics of each of the two extreme disorders, in any variety of combinations. Cerebral involvement with the diseases mentioned above can produce organic psychiatric syndromes such as confusional states, depressive syndromes and symptomatic depressions which may last for months, often after all physical signs have cleared.

If **symptomatic depression** remains after hyperbaric therapy, the diver may develop a modification of personality problems, with anxiety, emotional lability, difficulty in coping, delayed insomnia (waking up in the early hours) and even suicide ideation. Small doses of tricyclic antidepressants (such as amitriptyline 25–75 mg nightly) may have a beneficial result in a week or two. An organically orientated psychiatrist should be consulted.

Superimposed on the organic brain syndromes there may be specific clinical features due to the anatomical sites affected by the disease.

Dementia and diving surveys

Dementia, by definition, involves permanent impairment of short- and long-term memory, with at least one of the following:

- impaired abstract thinking;
- impaired judgement (personal and social);
- impaired impulse control;
- regional cortical dysfunction; and/or
- personality change.

Any (or all) of these should be significant enough to interfere with work and social relationships. This disturbance of cognitive function must be demonstrated in the absence of any disturbance of consciousness (e.g. delirium).

These patients are less able to cope with or introduce new ideas, and there is a diminishing of thought, abstract concepts, judgement and insight (the 'dys-executive syndrome'). Affect changes, especially emotional lability, is characteristic. There can be a dominance of paranoid or depressive features, and the patient may attempt to cope with their limited abilities by obsessionality and orderliness.

Reports from the 1950s and 1970s claimed that divers and caisson workers who suffered severe neurological DCS were likely to sustain permanent brain damage. Unfortunately, they are often inappropriately quoted as evidence that diving, as such, is also a cause of dementia.

In 1959, **Rozsahegyi**[16] in Hungary, examined 100 subjects between 2.5 and 5 years after CNS DCS, and concluded that over 50 per cent of them had some form of psychological disorder. However, 75 per cent had neurological findings on clinical examination. Quiet men would frequently become irritable and uncontrolled after the injury, and pathologic drunkenness and alcohol intolerance were frequent. The neurological, electroencephalogram (EEG) and

psychiatric disturbances were organic in nature. Rozsahegyi concluded that a chronic, progressive encephalomyelopathy resulted from repeated decompressions and neurological DCS, though some of his cases improved over years of follow-up.

These observations prompted research on the relationship between the neurological sequelae of DCS and intellectual functioning. Between 1975 and 1977, three **Texas studies**[17–19] reported neurological and psychological problems, with psychometric tests, in a small number of divers who experienced DCS affecting the CNS.

A **Norwegian**[20] report in the early 1980s claimed neuropsychological damage after 'near-miss' diving accidents. These investigators compared nine divers with some controls, and there appeared to be a change in cognitive functioning; most divers reported an impaired memory capacity as the main problem. In addition, difficulty in concentration, irritability, alcoholism and aphasia were noted. These authors also used Rozsahegyi's series[16] of CNS DCS cases as evidence for the unsubstantiated hypothesis of a chronic progressive encephalomyopathy due to decompressions (without DCS).

Clinical DCS cases among recreational divers from Hawaii,[21] Australia[22] and Israel,[23] during the following decade, showed an increased proportion of neurological manifestations, compared to the predominance of joint involvement from the earlier Navy studies.

In a one-year follow-up of 25 recreational divers treated in **New Zealand** for DCS in 1987, Sutherland *et al.*[13] found that 74 per cent had some degree of morbidity, mostly in the form of personality changes. Impaired cognition was present in 48 per cent. Mood disorders were present in 56 per cent, and these mostly developed after their discharge from treatment.

Morris *et al.* (1991)[24] studied 292 professional divers and showed some impairment of cognitive function in divers who had suffered decompression illness. Impairment of memory and non-verbal reasoning in those without previous decompression illness, was predominantly related to age. There was no change in personality attributed to diving experience.

Unfortunately, this interesting study suffers from the absence of peer review. A large number of psychometric tests were performed, with results that were not very consistent. It appears that there is no obvious relationship between the quantity of diving and the neuropsychometric results, but there is a relationship between the presence of DCS and some types of cognitive impairment. There were serious questions[25] about the matching of the controls in this, and the earlier surveys. There was little consistency in the neuropsychological findings of the various studies on DCS cases.

An investigation by Gorman *et al.*[11] on recreational divers who had been treated for DCS by the Royal Australian Navy, demonstrated a large number of neurological, EEG and psychometric abnormalities during the subsequent weeks. This was so even with divers who had no obvious clinical neurological component to their DCS. There was no clinical or investigatory evidence, sought or implied in this series, to demonstrate long-term sequelae. Improvement was demonstrated, and the proportion of 48 per cent who had EEG changes the week after a DCS incident was reduced to 17 per cent one month after. This is close to the incidence in the general population.

Evoked responses may be abnormal at the time of such an incident, but are unlikely to persist unless clinical changes are gross.[26–28] EEGs and evoked cortical potentials in divers may be of clinical value, at least in the acute stages of DCS. Abnormalities in EEGs and neuropsychometric testing[12] are to be expected during illness, or if subjects are not fully alert and cooperative.

A review[29] on various effects of DCS on diving was made by Shields *et al.* in 1996. This was carried out on 31 divers who had DCS, 31 who did not and 31 controls. The following conclusions were drawn:

- Both diver groups had high levels of abnormally long latencies in evoked potential measurement (brainstem auditory evoked potential; BAEP, somatosensory evoked potentials; SSEP, SEP). The DCS-diver group had a 14-fold higher presence of abnormal P40 latencies (SEP - tibial) than the non-DCS divers, and a seven-fold higher presence of abnormal P40 latencies than non-diving controls.
- On psychometric assessment, the DCS-diver group had a statistically significant poorer logical memory performance than the non-diving control group on immediate recall and than the non-DCS-diver group, on delayed recall.

- Although 28 per cent of divers had HMPAO-SPECT scanning (see Chapter 12) results outside normal limits, the absence of an established baseline for the incidence of pattern variants in the standard population makes this observation difficult to interpret. The clinical significance is unclear and may only be answered by long-term study.
- The significance of these findings in terms of a diver's current state of health or quality of life is unclear, and more extensive studies are required to shed light on these aspects.

Recently, imaging techniques[30–33] have been used to demonstrate cerebral damage and perfusion abnormalities, after appropriate treatment has been instituted. This confirmed the presence of both anticipated and unexpected lesions. Nevertheless, definite spinal cord pathology in divers,[33] in excess of that expected from clinical examination, has been demonstrated by some pathologists. To a lesser degree the same may be claimed for cerebral vascular pathology, but a clinical concomitant has not yet been demonstrated.

The **MRC DCS Panel**[31] report in 1988, on the long-term health effects of diving, was edited by M.J. Halsey. It was stated that diver autopsies showed extensive histopathology of the brain. Dr Ian Calder stated that the pathology represents about ten years of aging. It is not clear from the MRC report (or to attendees), whether the pathologists were referring specifically to cases of DCS, deep and helium divers, or to conventional air-breathing divers. Reference was made to cases of serious DCS with the frontal cerebral lesions, causing neuropsychological problems of divers in Hawaii – apathy, depression and the socioeconomic difficulties. Some informative studies have been carried out, using neuropsychological techniques during or immediately after diving[34] and diving accidents,[15] showing acute neurological impairments.

The judgement of a brain-damaged patient, especially if he is in a powerful administrative position, may result in damage to other divers. The anecdotal observations of dementia in middle-aged professional divers, who had not adhered to established safety regulations and decompression procedures, are contentious and deserve investigation.

NEUROPSYCHOLOGICAL IMPAIRMENT FROM DIVING

An even more contentious belief has arisen regarding the possible dementia-producing effects of diving *per se*, without accidents sustaining clinical neurological disease.

Diving folklore

The folklore belief developed among many occupational diving groups that a dementia ('diver's dumbness' or the 'punch drunk' syndrome) was produced by prolonged compressed air diving. This presumption was supported by media reports of brain damage in divers during the 1980s. In the United Kingdom, anecdotal observations from the Royal Navy, as well as pilot studies from the University of Lancaster, have been widely quoted and have probably heightened the concern among divers. In a report on abalone divers in Australia,[35] it was stated that 30 per cent of the divers suffered chronic ear damage, 20 per cent had dysbaric osteonecrosis, and 10 per cent had brain damage, but no supporting evidence was submitted.

In a symposium in Norway[32] in 1983, there was apparent acceptance of the neuropsychological complications of diving with compressed air. However, no consensus was reached regarding any long-term neuropsychological complications of deeper diving – which has been investigated more fully.

Circumstantial evidence

A number of diverse reports have been used as circumstantial evidence for diving producing neuropsychological damage and, by implication and extrapolation, subsequent dementia.

In Sweden,[36] abnormal EEGs were noted in 3.5 per cent of free-ascent trainees, suggesting the presence of cerebral arterial gas emboli. Kwaitowski[37] investigated 150 professional Polish divers and found abnormal EEGs in 43 per cent, compared to 10 per cent in the normal population. Todnem *et al.* (1991: p. 128, ref. 33) also showed increased EEG abnormalities among divers (18 per cent) compared to

new divers (5 per cent), with slow wave and/or sharp potentials. However, these were not replicated by others.

Hallenbeck and Anderson[38] and Hills and James,[39] each demonstrated that 'silent' bubbles produced during decompression could damage the blood–brain barrier temporarily and produce extravasation of blood constituents and fluid into the cerebral tissue.

Calder[31,33] has reviewed neuropathology findings at autopsies on divers. These abnormalities were more extensive than would have been anticipated from the mild or treated DCS previously experienced by subjects. There was, however, more spinal than cerebral damage. There is also some doubt about how clinically free of symptoms and signs some of the cases were.[40]

Vascular changes in divers may be related to a higher than normal number of Unidentified Bright Objects (UBOs) in the MRI, but no correlation has yet been demonstrated between these and any neuropsychometric factors. As Hallenbeck[41] (1978) noted, when investigating the possibility of a 'diving encephalopathy', even clinically obvious transient ischaemic attacks with definite pathology, usually clear without sequelae.

Psychometric surveys: does diving cause dementia?

Most of the earlier studies[16–20] suffered from gross errors,[25] including:

- A heavy reliance on anecdotes.
- Ill-defined diagnoses (not differentiating DCS, CAGE, near-drowning and hypoxia, gas toxicities, inner-ear and lifestyle diseases).
- Statistics: controls were absent or inappropriate, and searches were made for multiple associations without adjusting the techniques applied. Other controls were not matched for previous IQ, age, alcohol consumption, head injury, etc.
- Selection: some were medicolegal cases or involved in compensation.
- Employing experimental and non-standardized psychometric and neurological investigations.
- Psychometric testings that are inappropriate or not internationally standardized.
- Support and misquotes from non-peer-reviewed articles or abstracts.
- Extrapolations and overstatements.

As this contentious issue has now been debated for almost half a century, it is no longer credible to rationalize current sloppy selection procedures and statistics or the basis that 'the topic needs to be discussed'. Nor is it constructive to search for smaller and smaller anomalies based on non-standardized investigations – no matter how modern or colourful.

Two conclusions can be made from the data available:

- Clinically evident brain damage, verified by neuropsychological testing and standardized neuroimaging techniques, to a degree consistent with a DSM4(R) definition of dementia, does occur from brain damage in diving accidents.
- There is no substantial evidence of an equivalent disorder (permanent, progressive brain damage or 'encephalopathy') from diving *per se*.

Two extensive surveys[7,8] on psychometric disability in US Navy divers demonstrated statistically significant – but opposite – results. Previous and subsequent psychometric examinations on divers revealed that divers' personality profiles, and their psychometric test results, are different to those of nondivers.[1–3] This difference needs to be appreciated when utilizing controls and drawing conclusions.

Becker,[14] in the US Navy, using conventional and reputable psychometric testing, could find no evidence in six subjects three years after a 1800-foot dive, to support his previous impression of impairment of cognitive function.

Curley[42] investigated 25 US Navy deep saturation divers with conventional psychometric testing. After a four-year longitudinal neuropsychological assessment at the Navy Experimental Diving Unit, no permanent residual defects could be detected in either cognitive or CNS functioning.

Australian 'excessive diver' surveys

In 1985, an investigation[43] was performed on Australian abalone divers because of their extremely aggressive diving procedures, excessive

diving exposure, the high prevalence of conventional occupational diseases, and the alleged presence of a punch drunk syndrome.[35,44] It was presumed that if damage was present, its specific nature would be more obvious in this group. Conversely, if this excessive diving group showed no evidence of intellectual impairment, the disorder would be an unlikely or uncommon complication in more conventional air-diving groups. An initial pilot survey[43] indicated that there may be such a problem. This excessive diving population[44] of 152 divers had, on average:

- been diving for over 16 years;
- spent 12 years in professional abalone diving;
- spent over 5 hours per day on compressed air (Hookah) for 105 days each year;
- reached just over 50 feet (15.25 metres) on a typical day; and
- claimed to have been 'bent' over four times, but probably did not recognize the less dramatic types of DCS.

Routinely, 58 per cent of the excessive divers employed a dive profile that required some time for decompression, but which was omitted. Sixty-nine instances of DCS were diagnosed and treated by recompression therapy in a recompression chamber. Of these, at least 39 were neurological in nature.

Multidisciplinary and special interest researchers investigated these excessive divers independently – using objective standardized psychological, neurological and electrophysiological tests. The purpose was to employ internationally accepted investigations, standardized for the Australian population, to indicate the existence or otherwise of brain damage.

Because of the contradictory findings between the Texan and Norwegian studies on the WAIS, this test was repeated by Edmonds and Coulton[45] on a much larger ($n = 67$) group of this 'excessive' diving population. A multiple regression analysis was made against all diving co-variants, and the 10 subtext scores (verbal IQ, performance IQ, total IQ) and deterioration index ('dementia score'), corrected for age. Apart from minor and clinically unimportant associations, the analysis showed no relationship between the type of diving and these measurements of intellectual functioning. Nor was there an abnormal profile or scatter in the divers' results, which could have implied brain damage. This investigation indicated that if neuropsychological changes were present, they would be of a more subtle nature than those detected by such conventional batteries.

Neurobehavioural researchers, who specialized in detection of minor abnormalities amongst occupational groups exposed to chemicals, heavy metals and toxins, also examined a group of excessive divers. Williamson *et al.* reported two such studies.[3,46] In the first study, they found that the divers did as well or better than the controls on some tests (reaction time, some memory and motor tests) and worse than controls on others (visual and short-term memory and some psychomotor learning skills). However, the way in which divers chose to complete their tests differed from the controls, in that they were more likely to take risks and substitute speed for accuracy. This difference in behaviour should be taken into account when interpreting neuropsychological assessments in other surveys. The second study focused on neuropsychological functioning and a number of diving-related variables, but the associations found were weak. What it did demonstrate was that the deficit in neurobehavioural tests were in those divers who consistently exposed themselves to gross decompression omissions, and so experienced DCS.

Another subgroup of 48 excessive divers were subjected to the more conventional psychometric tests by Andrews *et al.*[47] This study compared excessive divers to non-diving fishermen controls living in the same locality. These authors found that the differences were small, and the divers' scores were within normal limits for the general population. Andrews and colleagues also compared the 'abnormal low' performance members from both the divers and control groups, and found no evidence for a subset of divers with abnormal scores. The authors concluded that 'there was no evidence for the accumulation of sub clinical insults leading to a dementing process'.

Hjorth *et al.*[48] performed double-blind assessments on EEGs and carried out multiple evoked cortical potentials on 20 excessive divers. Apart from a couple of minor abnormalities in the EEGs, no significant findings were made. Visual evoked cortical potentials and upper- and lower-limb somatosensory evoked cortical potentials were all normal.

In the Australian 'excessive diver' cross-sectional survey, there was no evidence of a diving related dementia or evidence that diving *per se*, causes any cognitive impairment.

If divers who are exposed to excessive diving do not show clinical or insidious abnormalities from this, *per se*, then there is little likelihood of others, who are less exposed, being so affected.

UK and European reviews and surveys

A smaller Norwegian survey[49] of 'excessive' divers compared 20 construction divers (mean = >4000 dives) with age-matched controls and diving trainees. A variety of conventional neuropsychometric tests were performed, but the authors were unable to demonstrate any clear evidence of neuropsychological deficit due to the extensive diving. The only evident abnormality was a prolonged reaction time.

Two recent reviews[50,51] have been carried out in the UK. Unfortunately, they were not reported in the general medical literature, and presumably not exposed to peer review. Nevertheless, their considerations and conclusions are worthy of note, if only to demonstrate the current areas of contention. Evans and Shields[50] critically reviewed the literature of potential neurological long-term effects of diving, reassessing histopathology observations, diagnostic neurological imaging, psychometric testing, electrophysiological studies and retinal angiography. As regards the neuropathological studies, it was concluded that there was some cause for concern, but that further work was clearly needed – including assessment of human post-mortem material and animal studies to demonstrate that histopathological effects of hyperbaria could occur without signs of DCS.

As regards the diagnostic neurological imaging, CNS changes in divers presenting with acute DCS have been demonstrated, though the incidence of these in the healthy diver population has yet to be established. Investigations are continuing, particularly as regards cerebral perfusion and PET-mediated assessment of cerebral metabolism, with the potential of detecting subtle neurological changes, if such exist. The neuropsychological studies of diving are contradictory – with some recording no apparent deficits while others have found positive correlations between diving and poor test performance. Clearly, there is a need for a well-controlled longitudinal study of a cross-section of the diving community.

The findings from EEG studies have been disappointing, the evidence often being contradictory and the correlation between EEG abnormalities and clinical signs poor. Computer analysis, especially with brain electrical activity mapping (BEAN) is potentially of interest, but this technique is still in its infancy and controversy remains over its use and reliability.

Evoked potentials have been employed, including visual evoked potentials (VEP), BAEP and SSEP. As with EEG, measurement of evoked potential in divers has proved disappointing, both in the management of acute dysbaric illness and as a research tool in investigation of long-term sequelae. The field of electrophysiology does not appear to have lived up to its early promise in the identification of subclinical neurological lesions.

Retinal fluorescein angiography in asymptomatic divers has been employed to demonstrate neurological lesions.[33] At present the situation is unresolved, however, and further results are required to confirm original findings, the relevance of which in relation to diving is as yet unknown (see page 461).

Participants at the latest International Consensus Conference on long-term neurological damage in divers determined that it is not clear whether a problem exists. They reiterate the difficulty of drawing conclusions from a small number of papers repeatedly quoted, with a small number of cases and not well controlled.

Another excellent review of the long-term health effects of diving is presented by Elliott and Moon.[51] Since that publication, and also the Norwegian publication on *Long Term Health Effects of Diving*, of an international consensus conference at Godoysund, Norway, in 1993, there have been some additions to the available data on this subject. In general they have supported most of the conclusions prior to then.

A study from Finland,[52] evaluating the EEG and MRI investigations after diving and decompression incidents, supported the value of the EEG assessment, as a non-specific indicator of CNS damage, and its improvement with recompression therapy.

The MRI was not a particularly valuable investigation, and these workers were not able to verify evidence of increased CNS lesions in normal divers as compared to non-diving, healthy controls. Some of the divers treated for DCS had hyperintense lesions in the brain white matter.

Another well-controlled survey from Germany,[53] compared 59 military and commercial divers (with at least 500 hours of diving) with 48 control subjects matched for age, body mass index, alcohol and smoking history, but who had never dived. They could not find any increased prevalence of brain lesions in the divers, with a fluid-attenuated inversion recovery sequence and T1- and T2-weighted pre- and post-contrast MR images.

CONCLUSIONS ON NEUROPSYCHOLOGICAL EFFECTS

There is ample evidence that acute and temporary neurological insults are experienced by divers. These are not commonly translated into evidence of permanent brain damage or dementia, but if this does occur it is likely to be either minor or rare in compressed-air diving.

Brain damage and dementia can supervene if there is significant cerebral injury (severe hypoxia, carbon monoxide toxicity, CAGE or cerebral DCS). Any long-term effects of the HPNS have yet to be defined, either qualitatively or quantitatively.

Earlier studies, confirming the unquestioned concept that neurological DCS could cause permanent neuropsychological damage, had serious limitations in their diagnostic categories, statistical analysis and misuse of control groups. They were inappropriately used to imply a diving-induced brain damage without preceding clinical diseases. There is some relationship between excessive decompression stress, supporting the observations that DCS induces some neuropsychological changes. Otological, visual, spinal and peripheral neuropathies from DCS, oxygen toxicity and other diving disorders may also affect neurobehavioural function (and neuropsychological tests), without implying dementia.

The adverse effect of DCS and its treatment on the anxiety and self-esteem of the diver, together with attitudes of both peer and therapist groups, may well have psychological implications, such as symptomatic depression, a psychoneurotic reaction or post-traumatic stress syndrome. These effects, together with the physiological influences of sleep deprivation, hyperbaric therapy, drug administration and non-cerebral manifestations of DCS, may well complicate the interpretation of psychometric assessments performed soon after the incident.

Some divers tend to be more 'risk-taking' than non-divers, both in their life's activities and their approach to psychometric testing. More carefully controlled studies have failed to substantiate the hypothesis of an insidious development of an occult dementia, or even evidence of early 'neurological aging', in conventional divers.

There is insufficient evidence to conclude that long-term compressed-air diving exposure, apart from the well-documented neurological diseases of diving, causes dementia or any other clinically significant neuropsychological deficits.

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42

Miscellaneous disorders

CARL EDMONDS

INTRODUCTION

This chapter includes many less known or less appreciated disorders which have not been well defined. With future editions, some of these topics will warrant their own chapters.

CAROTID SINUS SYNDROME

Pressure may be exerted over the carotid sinus by tight-fitting wet suits, especially when they are of the 'pullover' variety without zippers. Most divers will be aware of the unpleasant sensation while wearing these, and will cut the neckline to release the pressure before other symptoms supervene. Others manually hold the collar open. In some cases, the sensation of confusion and disorientation may have a demonstrable nystagmus associated with it. Such cases may be verified by electronystagmography. Other mechanisms may be needed to explain some of these cases.

Some may experience a sensation of fainting and have the bradycardia and hypotension which result from pressure on the carotid sinus. This may contribute to some 'sudden death syndrome' cases (see Chapter 39). In all these cases it is necessary to **reduce the pressure around the neck.**

CAUSTIC COCKTAIL

This only occurs when rebreathing equipment is used. It is variously termed alkaline cocktail, 'proto cocktail' when Protosorb® is the carbon dioxide absorbent used, and 'soda cocktail' when Sodasorb® is used.

Exogenous or endogenous water, when mixed with alkaline carbon dioxide absorbent (usually sodium or lithium hydroxide), will produce an alkaline solution which may not remain in the absorbent canister. If it travels into the breathing tubing, it may be taken into the mouth and inhaled or swallowed. A severe inflammation, possibly with mucosal ulceration, can result. The extent of these injuries is related to the amount, concentration and distribution of the 'cocktail'.

Traditionally, **treatment** involves rinsing of the mouth with vinegar or other acidic mixture to neutralize the alkaline cocktail, though this therapy may itself be very painful because of the existing mucosal damage. Probably a rapid irrigation of the area with freshwater or seawater will expedite the removal of the irritant material, and reduce the symptoms and subsequent damage. Respiratory and gastric involvement is treated according to general medical principles.

COLD URTICARIA

This is a predominately localized IgE-dependent urticarial response to cold – whether it be cold water, ice, wind or volatile fluids. It is usually noticed on the exposed skin, but may also affect mucosa of the respiratory system (by breathing cold air) or in the mouth and gastrointestinal tract (by swallowing cold drinks). The symptoms may thus vary from skin rash, punctate erythema or urticaria, to nasal congestion, swelling of the lips and mouth, cough and dyspnoea, dysphagia and abdominal cramps. It can often be replicated by placing the hand and forearm in iced water for 5 minutes and observing the development of the skin manifestations over the next 5–10 minutes. Occasionally, it can be reproduced by an ice cube. (Plate 15).

Generalized **symptoms and signs** may develop, probably due to histamine release, and produce headache, flushing, hypotension, syncope and increased gastric secretion. In a highly sensitized patient, swimming in cold water may precipitate cardiovascular collapse and death.

Investigations may reveal cold-precipitated plasma proteins, such as cryoglobulin, cold haemolysins and agglutinin, cryofibrinogen, etc. There may be an elevated IgE level, and possibly eosinophilia. The stimulus to tissue damage and mast cell histamine release is the rate of decrease of temperature, more than the absolute temperature. The symptoms tend to develop after the return to normal temperatures, after the cold-induced vasoconstriction is corrected. The syndrome may develop spontaneously or after some illness or injury (one case followed a jellyfish sting). **Aquagenic urticaria** may be related, and has occurred in the same patient, though this may be seen with water of various temperatures (but perhaps carrying allergens).

The response to systematic antihistamines and topical steroids, is usually poor (although oral cyproheptadine has received some support). Desensitization by gradually increasing the severity of the stimulus from warm acceptable showers to cold-water swimming (under competent supervision) has worked in some cases. The condition tends to clear over some months or years, but may recur. It is sometimes familial.

DENTAL DISORDERS

Barotrauma (see Chapters 8 and 9) affecting the teeth, sinuses with pain referred to the teeth, and subcutaneous emphysema (also submucous) from dental procedures have already been mentioned.

Dental electrolysis may be experienced by divers who carry out electric welding. They notice a metallic taste in the mouth adjacent to amalgam fillings. The electrical field set up by the equipment causes metal in the filling to be released, producing both the metallic taste and the premature destruction of fillings.

Dental plates must be secure and not easily displaced either during buddy breathing, vomiting, resuscitation or otherwise. A candidate who exhales his plate while performing lung function tests is just as likely to inhale it while diving. Deaths have been caused by a loose dental plate. (see Fig. 51.1).

HYPERTHERMIA

The sea is commonly a heat extracting environment because of its high conductivity and specific heat, and low temperature. (see Chapter 27). Despite this, hyperthermia – which may develop in many ways – has claimed the lives of some divers.

Thermal protection suits, which effectively insulate the diver from low water temperatures, also help to retain his own heat output. Both wet suits and dry suits, worn before or after immersion, may produce hyperthermia and heat stroke in temperate climates. When these divers also wear their suits at tropical diving resorts, or perform exercise, the danger is increased. The suit may still be worn for mechanical protection, without realizing its thermal disadvantages. Armoured diving suits, such as the 1-atmosphere 'Jim', may predispose to hyperthermia because of inadequate ventilation.

Actively produced hyperthermia may result from wearing hot water suits or other heated suits, or by **breathing heated gases** of high thermal conductivity, such as helium. In deep diving operations this gas may need to be heated to reduce respiratory heat loss, but the operating range is small and it is easy to overstep the margins. A further complication is the heat produced by compression of the chamber gas to simulate descent.

Divers with hyperthermia may lose consciousness from postural hypotension when, in their vasodilated state, they are exposed to the effect of gravity – as they emerge from the water onto the dive boat or into the diving bell. Hyperthermia is a recognized complication of the treatment of hypothermia, by hot water immersion or by active core rewarming.

Prevention is achieved by avoiding the above circumstances. Treatment includes removal of the cause, applying cooling techniques, rehydration and electrolyte replacement.

MUSCULOSKELETAL PROBLEMS

The musculoskeletal problems of barotrauma (Chapter 9), decompression sickness (see Chapter 12), dysbaric osteonecrosis (see Chapter 14) and marine envenomations (see Chapter 32) have been described previously.

Compression (hyperbaric) arthralgia

With the advent of deep and helium diving, a syndrome of joint noises and sensations was recorded. The noises were described as cracking, creaking or popping, and the sensations varied from discomfort, to a dry and gritty feeling, to frank pain precipitated by movement.

The **symptoms** can appear as early in compression as 30 metres, but are more common at depths exceeding 100 metres. They are aggravated by a fast compression, but improve as the duration at depth continues. The divers show considerable individual susceptibility. The condition is more likely to be experienced in compression chambers than in water, when fast movements are limited and little mechanical load is placed on the joints.

The current explanation of this disorder involves a gas-induced osmosis interfering with joint lubrication and producing cavitation. As the subject is compressed, a relative imbalance is present between the concentration of inert gas in the blood and that in the synovial fluid, and articular cartilage, causing a water shift from the joint to the higher osmolarity blood. As equilibration of inert gas develops with a continuation of this exposure to pressure, the original fluid volumes become re-established.

Cramp

Divers seem particularly prone to muscle spasms, resulting in temporary pain and disability, and these may have disproportionately severe complications underwater. Cramp usually develops in muscles that are exposed to atypical exertion, e.g. physically unfit divers, the use of new fins, etc. Any damage to the neuromuscular system will predispose to muscle cramp. Although the most common sites are the calf of the leg and the sole of the foot, other muscle masses may be affected. These include the thighs (especially hamstrings), upper limbs and abdomen. Diagnosis is made by observation and palpation of the tight muscle mass.

The immediate **treatment** consists of a slow, passive extension of the cramped muscle, and then a return to safety. Prevention includes the maintenance of an adequate standard of physical fitness, constant diving exercise and practise, the fitting of comfortable equipment and fins, avoidance of dehydration and sweating, good nutrition and adequate thermal protection.

Decompression sickness

The musculoskeletal ('bends') pains of decompression sickness (DCS) have already been dealt with in Chapter 12. Such symptoms are thought to be due to tissue distortion from bubbles, and are relieved rapidly (within minutes) during hyperbaric exposure and less rapidly (hours) to inhalation of 100 per cent oxygen.

There are many less obvious and less well-defined musculoskeletal or arthralgic symptoms that may follow DCS and which are possibly attributable to subsequent tissue damage (in tendons, muscles, etc.) or even to early injury within the bones – which may or may not progress to dysbaric osteonecrosis. Such symptoms may improve somewhat with non-steroidal anti-inflammatory drugs such as paracetamol, ibuprofen or piroxicam, or cyclooxygenase (COX)-2 inhibitors. A technetium bone scan may be of value in excluding early and progressive osteoarthrosis pathology. The symptoms usually diminish and gradually disappear over weeks or months.

Occasionally, gas develops in the synovial joint cavities after long exposures (affecting slow tissues), producing loud crunching sounds for a day or two, and these are precipitated with movement (Fig. 42.1).

Lumbosacral lesions

Prolonged underwater swimming, in an abnormal hyperextended spinal position – such as is employed by shell fisherman scanning the seabed – can aggravate lumbosacral pathology. The positioning of the heavy weight belt around the waist may make this worse. Many divers so affected have replaced the weight belt with a much wider weight-containing corset. Details of cervical spine lesions have been outlined in Chapter 40 (see 'headaches').

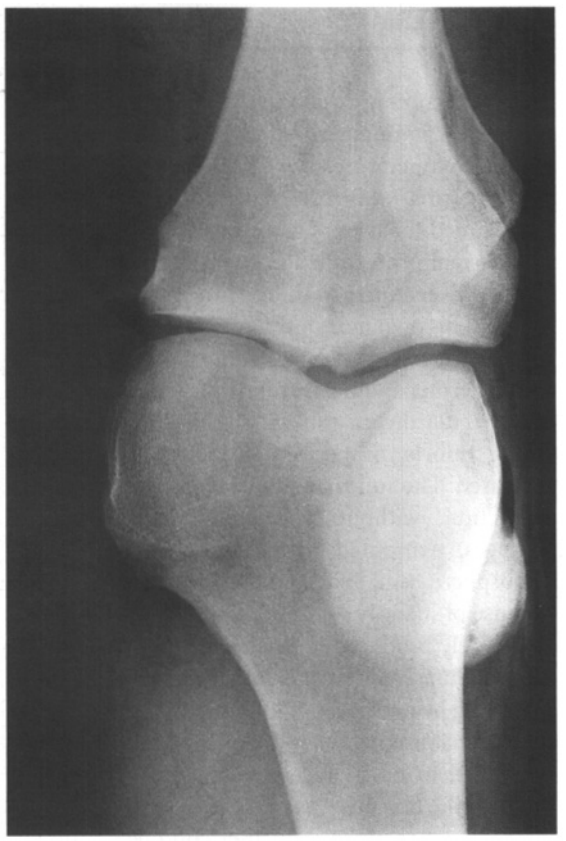

Figure 42.1 *Gas in knee joint, from decompression.*

Temporomandibular joint dysfunction

In the early stages of diving training a novice may experience apprehension in regard to the air supply. Consequently, he is likely to clamp his teeth hard onto the mouthpiece to such a degree as to cause a considerable temporomandibular joint stress with a resultant arthritis, the pain and tenderness being felt just anterior to the ear. Alternative or associated symptoms include trismus, restriction of the ability to open the mouth widely, 'clicking' of the joint and occasionally tinnitus and vertigo. The syndrome is readily relieved by education of the diver and encouraging him to relax.

Recurrent problems of temporomandibular arthritis and subluxation of this joint are also likely to be aggravated by diving. Apart from the above cause, which is unlikely in experienced divers, there are other stresses placed on the temporomandibular

Plate 13 *Blue-ringed octopus (Courtesy K. Gillet)*

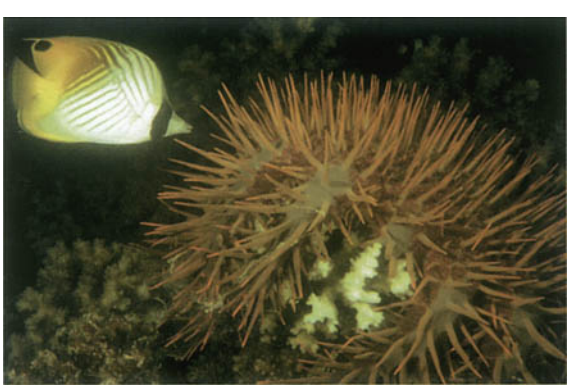

Plate 14 *Crown of Thorns (Courtesy R. Chesher)*

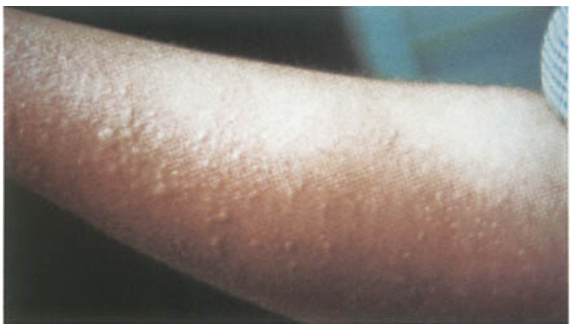

Plate 15 *Cold urticaria: histamine skin reaction developing 5-10 minutes after exposure to cold water. Mucosal surfaces were affected by drinking cold liquids. In this subject diving had to be suspended for 6 months. (see Chapter 42).*

Plate 16 *Bubbles developing between the hard contact lens and the cornea; these developed during decompression.*

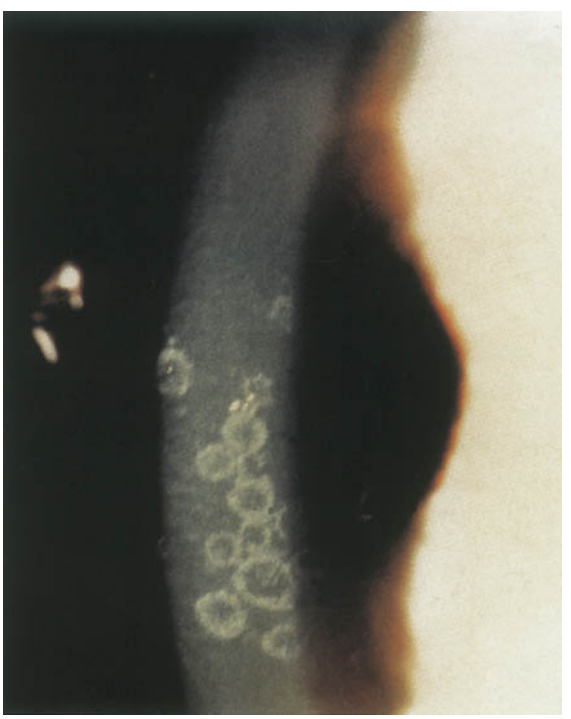

Plate 17 *Nummular patches of corneal epithelial edema, where the bubbles injure the cornea (see Chapter 42). (Photographs in Plates 16 and 17 courtesy Drs Mark E. Bradley and David Simon.)*

joint which are not normally experienced in non-divers. These include the use of mouthpieces not individually fashioned to the diver's oral and dental configuration, prolonged exposure to a chilling environment, and finally the use of equipment which tends to pull vertically or to one side of the diver's mouth. Most mouthpieces require the diver to hold his mouth open with the mandible protruded, tugging on the mouthpiece – an abnormal position. The symptoms are similar to those described above, but in the chronic cases, radiological evidence will demonstrate the extent of joint damage and dysfunction. The remedy is to avoid the provoking situations, i.e. use well-fitting mouthpieces or an oronasal mask, ensure there is no strain on the demand valve or its hose, and wear a well-fitting hood to avoid cold.

Tank carrier's elbow

In divers this has been described by Barr and Martin with biomechanical and electoromyogram studies. It occurs with overuse during dive trips when wrist pronation and dorsiflexion is incurred by lifting tanks by the valve or neck. For shorter subjects this is aggravated by the need to flex the elbow to keep the bottom of the tank off the ground (Fig. 42.2). This is a common entity, called 'tennis elbow', in other circumstances. There is frequently a weakness in grip. Ultrasound may reveal a thickening of the symptomatic common extensor tendon, or even a tear.

Figure 42.2 *Typical elbow position to produce epicondylitus.*

Prevention is by ensuring adequate muscular fitness, or by carrying tanks horizontally from straps. Treatment is by rest and strapping, but occasionally surgery is needed.

CASE REPORT 42.1

A middle-aged man, who was somewhat unfit, phoned three days after a multiple diving exposure complaining of decompression sickness. He had developed pain in the right elbow following the diving, and it had persisted, initially increasing but now constant. There was some weakness in wrist extension. Pressure with a sphygmomanometer cuff seemed to give relief, when he moved his hand and arm. A Table 6 recompression regime produced absolutely no improvement whatsoever.

On subsequent examination there was a local tenderness to pressure over the area of the lateral epicondryle. An alternative diagnosis became evident, and would have been obvious had an adequate history been taken initially.

In fact, he had spent a considerable amount of time during the diving holiday carrying scuba cylinders, and he had developed a lateral epicondylitis.

OCULAR DISORDERS

'Bubble eyes'

Some divers complain of gas bubbling from the inner canthus of the eye, the cause being an excessively patent nasolacrimal duct. This structure frequently has an imperfect valve, formed by the mucous membrane in the nasal cavity. The normal passage of tears from the eye and down the duct is not impeded. Unfortunately, the passage of air up the duct may be expedited when the diver increases nasopharyngeal pressure, such as during the Valsalva manoeuvre. This can be demonstrated by viewing the air bubbling out from the lacrimal canals during this manoeuvre. There is a possibility of spreading organisms from the nasopharynx and causing conjunctivitis.

Ocular damage from corneal lens

Recent work on the use of contact lenses during diving has shown that the soft corneal microlens is permeable to gas and nutrients. They are therefore safe, except for the likelihood of being lost during such diving techniques as facemask removal, followed by opening the eyes underwater.

Ophthalmologists claim that soft lenses shrink when exposed to freshwater, and may cling to the cornea and be temporarily difficult to remove. If exposed to seawater they swell and can float out of the eye. Others have observed that seawater can increase the adherence of soft contact lenses to become stuck to the cornea, though within the mask this should not cause a problem.

The smaller hard contact lenses are also not very secure in that they cover less of the cornea. Some are also less permeable and have the potential of causing problems during decompression (Plates 16 and 17).

Underneath the hard lenses, small bubbles develop during decompression, in the pre-corneal tear film. The bubbles coalesce and expand during decompression, and may damage the corneal tissue. Divers may be aware of a sensation of discomfort in the eyes, the appearance of halos with radiating spokes when looking at lights, and also a decreased visual acuity. In mild cases the symptoms last for only a few hours. The symptoms may be prevented by the use of a small 0.4-mm hole made in the centre of the hard lens, and this is then termed a 'fenestrated' hard lens. The hole serves as a channel through which the small amounts of tear fluid can pass, carrying the gas with it. These problems are only likely in deeper or longer dives, or in hyperbaric chambers.

The problem of loss of a lens during any diving operations makes these visual aids unacceptable for many professional divers. The increased likelihood of eye infections and the difficulty with eye toilet and lens disinfection procedures, as well as the blurred vision that sometimes accompanies the lens usage, makes them a hazard in remote areas and oil rigs.

Freshwater organisms, such as *Acantomoeba*, which can cause severe infections, corneal ulcerations and blindness, should not be a problem in seawater or inside facemasks.

Ocular fundus: lesions in divers

The ocular lesions of **barotrauma** and **hyperoxia** are described elsewhere (see Chapters 9 and 17).

Recently, retinal fluorescein angiography has demonstrated that the retinal capillary density at the fovea was low, and microaneurysm and small areas of capillary non-perfusion were seen, more often in divers than in non-divers (see Chapters 41 and 44). The prevalence of the fundus abnormality is related to the length of diving history, the changes being consistent with the obstruction of the retinal and choroidal circulations. This obstruction could be due either to intra vascular bubbles, formed during decompression, or to altered behaviour of blood constituents and blood vessels in hyperbaric exposures.

These lesions did not appear to have any influence on visual acuity, and there was an increased prevalence of such lesions in divers who had had decompression sickness (DCS) and this was significantly higher than in divers of equivalent experience but without a history of DCS. Nevertheless, even if the divers with DCS sickness were excluded, this did not abolish the correlation between diving experience and pigment epithelial changes.

The defects in retinal pigment epithelium are indistinguishable from those documented in eyes following choroidal ischaemia. It is possible, but there is no actual evidence, that these abnormalities may cause problems in later life. It is also necessary that these observations be verified by other workers, before preventative advice is considered (see page 461).

Swimmer's eyes (blurred vision)

Keratitis may cause blurred vision and 'rainbow' or 'halo' effects because of the corneal irritation from exposure to suspended particles and hypertonic saline (seawater), chlorine, ammonia and hypotonic water (swimming pools and freshwater). This is less likely with mask-wearing divers than with swimmers, but it is not uncommon when divers use chemical preparations to clean and demist the mask's glass face plate. Underwater welding causes ultraviolet keratopathy.

Apart from keratopathy, which is detected on slit lamp examination, other causes of blurred vision include the following:

- cerebral involvement with dysbaric disease;
- displaced contact lens;
- contact lens problems (see above);
- touching the eye after a transdermal hyoscine patch (SCOP);
- delayed delivery pilocarpine preparation with glaucoma;
- oedema and haemorrhages from facemask barotrauma;
- gas toxicities.

Trauma

Damage to the eyes can result from the facemask imploding, from inadequately strengthened glass during descent, or confronting a released anchor as the diver swims down the chain. Dramatic injuries have been caused by spear guns (see below).

It has been shown that divers with radial keratotomy (previously given for surgical treatment of myopia) may have a weakened cornea and be more susceptible to trauma – with rupture along the lines of the incision. Barotrauma effects in the facemask with descent, causing rupture of the eyeball, are a possibility, and for this reason such patients are usually advised not to dive with the usual (half) facemasks. Alternatives may include gas-filled contact lenses, facemasks with openings to the water, and full facemasks connected to the air supply.

Other disorders

These include oxygen toxicity (Chapter 17), decompression sickness (Chapter 12), mask barotrauma (Chapter 9) and mask burn (see below). Ocular aspects of medical selection for diving are outlined in Chapters 53 and 54.

PULMONARY OEDEMA AND DYSPNOEA

Diving diseases

Pulmonary oedema has been described in a variety of diving diseases, and in some it is a consequence of the other diving respiratory pathology, such as in:

- the drowning syndromes, including saltwater aspiration;
- pulmonary barotrauma;
- decompression sickness;
- underwater blast;
- some gas contaminants; and
- pulmonary oxygen toxicity.

Each of these conditions is discussed in its specific chapter.

In other instances pulmonary oedema has been described as the predominant manifestation from a diving exposure.[1] The pathophysiology explanations are often not completely convincing; nevertheless there are sufficient such cases to warrant concern. The various presumed aetiological types are as follows.

Pulmonary oedema of immersion

Immersion of the body, with the head above water, will have significant pulmonary effects.[2] Because of the hydrostatic pressure exerted by the water there is a redistribution of the blood into the large intrathoracic blood vessels and, to a lesser extent, into the

pulmonary microcirculation. It also greatly effects the structure and function of the thoracic cavity, resulting in the following changes:

- reduced vital capacity (approximately 5 per cent);
- reduced static lung compliance;
- increasing closing volumes leading to:
  - increase in functional air trapping;
  - increase in diffusing capacity;
  - increased flow resistance of airways;
  - altered ventilation, increasing in the apical regions; and
  - reduction in functional residual capacity.

Sometimes, dyspnoea occurs in otherwise healthy individuals during scuba diving. The aetiological stress factors which combine to induce this disorder include; increased cardiac loading (see Chapters 3, 39 and 61) due to the effect of water immersion, the respiratory changes noted above, and cold-induced peripheral vasoconstriction increasing the workload on the heart. It has also been claimed that some of these individuals subsequently develop hypertension.

The disorders occur while swimming or diving in shallow water, sometimes in the head-out position. The symptoms include dyspnoea, cough, haemoptysis and the expectoration of frothy sputum.

Cardiac disease

This condition may be related to pulmonary oedema of immersion, and some older divers, especially males, will develop dyspnoea 5–10 minutes after immersion (see Chapter 39). In these cases there is probably also a precipitation of coronary ischaemia or arrhythmias because of the effects of immersion.

The cardiovascular effects of immersion are complex, but can be summarized as follows:

- Increased stroke volume with increased cardiac output.
- Increased central blood volume, with blood passing from the periphery to the lungs due to the hydrostatic gradient.
- Extrasystoles during early phase of immersion (due to cardiac distension).
- The diving reflex, with facial immersion and trigeminal stimulation inducing bradycardia, a shunting of the blood to the heart/brain axis from the periphery and viscera, with peripheral vasoconstriction.
- The cardiac effects of cold exposure.
- Hyperbaric bradycardia, usually reduced by 10 beats per minute, and possibly related to increased oxygen partial pressure, increased gas density, increased hydrostatic pressure and a narcotic effect of inert gas.

Each or all of these may play a part in inducing mild pulmonary oedema and the respiratory symptom of dyspnoea. Subsequent investigation will demonstrate the coronary artery insufficiency or arrhythmia, and the problem will disappear when this is corrected.

Asthma provocation

The scuba situation is likely to induce asthma in those so predisposed. There are multiple provoking factors, including:

- exercise, especially if swimming against a significant tidal current;
- breathing against a resistance (the demand valve);
- breathing dense air (related to depth);
- breathing cold dry air (decompressed air);
- saltwater spray inhalation provocation; and
- psychological stress and hyperventilation.

Cold urticaria

This condition is described on page 434.

Deep-diving dyspnoea

Cold gas inhalation at depth makes all divers susceptible to dyspnoea at great depths, due to convective heat loss in the airways and the local response to this (see Chapter 68).

Other non-diving disorders may present as dyspnoea while diving, because of the exceptional physical demands of this activity, e.g., lung disease, drug effects from beta-blockers or irritants such as cannabis.

SKIN REACTIONS TO EQUIPMENT

A plethora of dermatological disorders may be found in diving medicine. Some of these, including decompression sickness and barotrauma are described fully in other chapters. However, there is a small group of illnesses which are apparently skin reactions to materials used in the equipment, or may be related to the direct effect of the equipment, without any specific diving medical illness. A few such disorders are described below, but many others are possible.

Contact dermatitis

Rubber may often have included within its composition an antioxidant or accelerator such as mercaptobenzothiazole. This acts as an irritant and, because of the minor insult to the skin, it may then sensitize the diver to further contact when an irritation with inflammation may be noted around the contact area. A similar disorder is seen among surgeons who wear rubber gloves. The three manifestations in diving include mask, mouthpiece and fin burn.

Mask burn

This varies from a red imprint of the mask skirt to a more generalized inflammation with vesicles, exudate, crusting, etc. (Plate 18). It may be several weeks before the mask can be worn again, and the condition will recur if a similar type of mask is used. To overcome this, silicone rubber masks may be made from Dow Corning medical grade silicone.

The treatment is either by soothing lotion, such as calamine, or more effectively by the use of a steroid preparation, applied regularly until relief is obtained.

Mouthpiece burn

This may present as a burning sensation on the lips, and is especially associated with hot drinks, fruit juices or spicy material. There may be an inflammation and vesiculation of the lips, tongue and pharynx. A similar product to that described above, using silicone to replace the rubber mouthpiece, may be obtained. For treatment, hydrocortisone linguets may be effective.

Fin or flipper burn

This is a very similar reaction to those described above, but may be mistaken for more common infections of the feet, and has even been mistreated as a fungal disease. The disease will of course continue while the fins or flippers are being used, and will not respond to fungicides. Wearing protective footwear, of a non-rubber nature under the fins, will prevent recurrence. The treatment is as described above for mask burn.

Angioneurotic oedema (dermatographia)

The effect of localized pressure on one part of the skin producing a histamine response, has been well recorded in other texts, and occurs in 5 per cent of the population. This is occasionally seen among divers when the ridges or seams of the wet suit push onto the skin. Under these conditions there are often stripes on the skin, with either erythema and/or oedema along these lines. It can be reproduced by firm local pressure.

The disorder can be confused with an allergy response to the resins and adhesives used in joining the seams. It can be prevented by either wearing wet suits which do not have such internal seams, or alternatively using undergarments which protect the skin from the localized pressure of the seams.

Allergic reactions

Unlike the above two complaints, there are rarely cases of allergy to either the wet suit material, the dyes used in it, or the adhesives used in its manufacture. Where this is the case there is a rash over the contact area.

Nappy rash (diaper rash)

The micturition that follows immersion, exposure to cold and emotional stress, may be performed during a normal dive. The urine usually has little effect, as it is diluted with water and gradually washed away with the pumping action of the diver's movements of the wet suit. Unfortunately if there is excess ammonia present, the diver may react with the equivalent of a

nappy rash of children. Sometimes the divers are unaware of the aetiology, and there is some embarrassment when this diagnosis is imparted to them.

With 'dry' suits, diapers are sometimes used for long-duration dives, producing a similar result.

Fin ulcers

Erosion of the hard edge of the fin, on the dorsum of the foot, may cause an ulcer. It is likely to occur with new or ill-fitting fins, and may be prevented by wearing booties as protection. Secondary infection by both marine and terrestrial organisms is common, and is aided by softening and maceration of the skin due to immersion.

The inflammation, which develops within a few hours of the trauma, may then prevent the subsequent use of fins, and endanger the diving operation. A prolonged period out of the water, and the administration of local antibiotics, may be required to achieve healing. The prompt use of these, such as neomycin, within an hour or two of the injury and the subsequent wearing of booties with fins, may avoid this situation. Chronic ulcers are mainly a problem in the tropics.

TRAUMA

Because divers use motorized boats as tenders, and because they use the same environment as other motor boats (speedboats, etc.), there is always a risk of boating injuries, such as lacerations from propellers. This risk is enhanced by any problem that the diver has in locating boats by either vision or hearing. Although the boats are readily heard underwater, the sound waves are more rapid, making directional assessment by binaural discrimination very difficult. Usually, by the time the diver sees a boat coming over him it is too late to take evasive action. All diving tenders should have guards over the propellors to reduce the hazard, and divers should employ floats with diving flags if they dive in boating waterways.

The lacerations inflicted by **propellers** tend to be parallel and linear along one aspect of the body or limb – unlike the concentric crescents on opposite sides, as seen in shark attack (q.v.). The treatment is along the same general lines as shark attack (Plate 19).

Head injury from ascending under the boat and hitting the hull, is not uncommon (thus the advised technique of holding the hand above the head during ascent), and also from injury as the diver swims alongside the hull (explaining the reason for holding an arm between the diver and the boat in that situation).

Other causes of trauma are numerous. **Weights** and scuba **cylinders** have taken their toll of broken toes and metatarsals, subungual haematoma. In Australia, **spear guns** account for as much morbidity and death as sharks. Power heads, carbon dioxide darts and other underwater weapons also cause a variety of injuries (Figs. 42.3 and 42.4).

Dam outlets and underwater siphons can trap divers, the outlet then becoming plugged with the diver's body. Exposure of the body to the full pressure gradient from the weight of water above

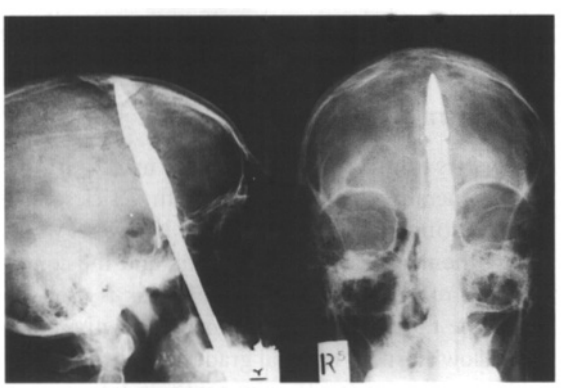

Figure 42.3 *Speargun injury: this 40-year-old male presented to a casualty department fully conscious and cooperative, with a 1-metre spear penetrating from underneath his chin to the coronal suture of his skull. The injury included a large palatal wound, penetration of the ethmoidal sinus and the sphenoidal region, severed left optic nerve and damage to the mesial cortex of the left frontal lobe. Craniotomy was performed and the cut spear was extracted through the craniotomy wound. Subsequent oronasal reconstructive surgery left the victim only with impaired vision and smell on the left side, and a functional left leucotomy – a contented customer and a happy diver?*

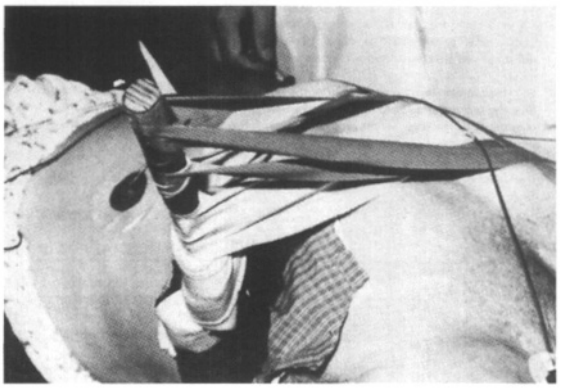

Figure 42.4 *Speargun injury: this spearfisherman stumbled on the way to the water, and caught an 80-kg amphibian – himself! The five spear prongs penetrated the pleura and pericardium on the left side. The 2-metre spear caused excessive leverage and pain when the victim laughed or breathed, and so the first-aid team wisely cut the shaft 20 cm from its tip. Cardiothoracic surgical intervention was successful. Note: more divers are killed or injured each year by spearguns than by sharks in Australian waters.*

leads to massive and grotesque injuries, and often also death.

Other forms of environmental trauma are described in Chapter 5.

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43

Drugs and diving

CHRIS LOWRY

INTRODUCTION

A drug may be considered as any agent which, when introduced into the body, produces a biochemical or physiological change. In this sense, oxygen, carbon dioxide and nitrogen or other inert gases under increased partial pressures may be considered as drugs, and are important in considering interactions with therapeutic agents more conventionally regarded as drugs.

The modern community has come to rely on drugs to overcome or prevent illness and cope with life, and the diving population is probably little different. Although many divers take drugs either routinely or intermittently, there is limited information of the effects of these under diving conditions.

Within the diving context, drugs should be considered in terms of their principal therapeutic effects, their possible side effects in the underwater environment, and their interaction with hyperbaria. The most important areas of concern in diving are the effects on the central nervous system, autonomic nervous system, and the cardiovascular and respiratory systems. Many studies have concentrated on the neurobehavioural effects of drugs under pressure, but other effects such as enhancement of cardiac arrhythmias or aggravation of oxygen toxicity may be equally important.

Other aquatic environmental influences, such as cold, sensory deprivation, spatial disorientation, reduced vision, reduced sound localization, vertigo and weightlessness, may also profoundly alter the behavioural effects of certain drugs.

Another largely unresearched area is the effect that drugs may have on the uptake and elimination of inert gas, thereby altering the propensity to decompression sickness (DCS). For example, dehydration has been associated with increased DCS, while certain drugs such as alcohol and diuretics may induce dehydration.

Immersion and pressure produce cardiovascular alterations (see Chapter 2) that affect the distribution of cardiac output. Reduced perfusion of the gut,

liver and kidney would theoretically impact on absorption, distribution, metabolism and excretion of drugs.

The significance in diving of certain side effects may not be immediately apparent. For example, the widely used drug aspirin has a profound effect on platelet function, causing an increased bleeding tendency. This effect may be crucial in, say, inner-ear barotrauma or severe DCS. Aspirin may also cause increased airway resistance in susceptible individuals. It is therefore important that all the effects of a drug be considered.

Some side effects of drugs that may be important in the underwater environment include:

- **Nervous system**: headache, dizziness, acute psychosis, tinnitus, tremor, incoordination, extrapyramidal syndromes, paraesthesiae and peripheral neuropathy.
- **Cardiovascular system**: tachycardia, bradycardia, arrhythmias, hypotension (postural), chest pain and oedema.
- **Blood**: anaemia, thrombocytopenia, neutropenia and disturbances of coagulation.
- **Alimentary system**: nausea, vomiting, abdominal cramps, heartburn, diarrhoea, altered liver function and liver failure.
- **Renal**: renal failure, electrolyte disturbances and disturbances of micturition.
- **Musculoskeletal**: myalgia, arthralgia and fatigue.
- **Skin and mucosa**: pruritus, rash, angioneurotic oedema and photosensitivity.
- Eye: glaucoma, photophobia, blurred vision and scintillation.

There are two possible attitudes to the use of drugs and diving. One advocates that drugs are useful to counteract minor problems such as eustachian tube dysfunction, seasickness, etc., and that these drugs will make diving easier and safer. The other view is that the diver should not be under the influence of any drugs because of possible side effects. The latter concept would require divers to stop taking some drugs days to weeks before diving. The more conservative physicians would include such drugs as alcohol, nicotine and caffeine in their recommendations.

As discussed in Chapter 53, a history of drug intake may give important clues as to the presence of otherwise undetected organic or psychological disorders.

As in pregnancy, the safe use of many drugs while diving has not been established.

DRUGS UNDER PRESSURE

Even if the effect of a drug at 1 ATA is well understood, it may be very different at hyperbaric pressures. Physiological influences, such as the increased hydrostatic pressure, varying pressures of oxygen and nitrogen or other diluent gases, may alter drug pharmacodynamics or pharmacokinetics. The functional result of drug–hyperbaria interaction may be negative (pressure reversal of anaesthesia), neutral, additive or synergistic. For example, the addition of some degree of nitrogen narcosis or elevated carbon dioxide may combine with a central nervous system depressant drug (such as alcohol, antihistamines or opiates) to produce an unexpectedly severe impairment of mental function, or even unconsciousness.

Rump *et al.* have reviewed the effects of hyperbaria and hyperoxia on the disposition of drugs.[1] The relatively few human and animal studies do not show alterations by hyperoxia of the pharmacokinetics of gentamicin, caffeine, lidocaine, pentobarbital or pethidine.

Other pharmacological studies in animals have not shown important changes at pressure equivalents of 200 metres seawater with morphine, pentobarbitone (pentobarbital), lignocaine, ethanol or digoxin.

Animal experiments have been carried out on a number of drugs, usually at very great pressures. Such information, while valuable in elucidating mechanisms of drug action and pressure interaction, is of limited value in the normal current diving range of pressures. Information in this area comes from a limited number of human and animal studies of the effects of drugs and pressure on behavioural and other physiological functions.

Walsh and Burch[2] studied the behavioural effects of some commonly used drugs at air pressures of 1.8, 3.6 and 5.4 ATA, and found that, at pressure, all drugs studied impaired learning (i.e. cognitive functioning). Among these drugs, diphenhydramine had the most effect, caffeine and dimenhydrinate varied widely with individual susceptibility, and aspirin and acetaminophen (paracetamol) had the least effect.

As an illustration of the difficulty in interpreting animal experiments, the same authors found that in rats, five to ten times 'normal' doses at pressures of 3, 5 and 7 ATA were required to show performance decrements with diphenhydramine and pseudoephedrine, yet no effect was seen with dimenhydrinate.

In animals, amphetamines exacerbate (not ameliorate, as expected) the behavioural effects of hyperbaric air. Indeed, hyperbaric air and amphetamines combine to produce behavioural changes not seen with either alone. Conversely, hyperbaric nitrogen can decrease the incidence of convulsions initiated by amphetamines.

A clear synergistic relationship between alcohol (2 ml/kg) and pressure (air at 4 and 6 ATA) in humans, in the processing of visual information, has been demonstrated.

One of the newer non-sedating antihistamines used for allergic rhinitis and nasal decongestion has been studied in man under hyperbaric conditions. Clemastine fumarate was found not to increase the sedative effects of nitrogen narcosis, nor to increase the development of cardiac arrhythmias.[3]

There are isolated anecdotal reports of therapeutic drugs producing unexpected effects on divers at pressure. The effects are usually on judgement, behaviour or level of consciousness and involve; paracetamol (32 ATA), dextropropoxyphene (4 ATA), hyoscine (scopolamine), oxymetazoline spray (5 ATA) and phenylbutazone.

Diabetics demonstrate reduced insulin requirements while undergoing hyperbaric oxygen therapy.

Some information comes from anecdotal reports of the use of drugs in hyperbaric or saturation diving operations. The fact that drugs have occasionally been used safely in such situations does not mean that they can be regarded as safe for diving.

ANAESTHESIA UNDER PRESSURE

Anaesthesia may be required for operative procedures during decompression and saturation diving operations. Decompression requirements may preclude removing the patient from the chamber.

In theory, **nitrogen** could be anaesthetic, but very great depths/pressures and a long period of decompression would be required. Other inert gases such as krypton and xenon produce anaesthesia at much lower pressures, but have not been used much in practice.

Nitrous oxide would require a partial pressure of greater than 800 mmHg for surgical anaesthesia,[4] but this pressure exposure is associated with signs of increased sympathetic activity such as tachycardia, hypertension and mydriasis. Clonus and opisthotonus may also develop, and a stable physiological state is difficult to maintain. Nitrous oxide use is also precluded by its high lipid solubility and tissue uptake, as well as the counter-diffusion problems (see Chapters 2 and 11) that increase the risk of subsequent DCS.

Volatile anaesthetics such as halothane and enflurane have been used to about 4 ATA pressure. These have the pharmacological advantage of reducing the cerebral vasoconstriction of hyperbaric oxygen. The newer volatile agents such as isoflurane and sevoflurane probably behave similarly, though little dose–response information is available at very great pressures, especially with regard to organ toxicity if higher doses are required to overcome pressure-reversal effects. Pressure reversal of anaesthesia at very great pressure (see Chapter 15) is not yet of practical significance.

Opiates appear to have similar effects under some saturation diving conditions as at the surface, but respiratory depression would be a possibility in hyperbaric air (due to narcotic effect of nitrogen) or at very great depth (due to increased gas density and increased work of breathing). Resultant carbon dioxide retention would enhance oxygen toxicity (see Chapter 17).

Neuromuscular blocking drugs are unaltered in effect at low (hyperbaric oxygen therapy) pressures, although they mask an oxygen convulsion.

The use of **regional** or **intravenous anaesthesia** is usually preferred to gaseous anaesthesia. **Regional**

anaesthetic techniques (with care being taken not to introduce gas) have theoretical support, and major procedures may be carried out. The minor effects of pressure-reversal are not relevant considering the relatively massive doses applied directly to nerves, but effects on toxicity and pharmacokinetics due to altered effects on blood flow and tissue binding may be. Conventional doses of lignocaine have been used up to 6 ATA. Ketamine, in combination with a benzodiazepine to prevent psychological phenomena, is a useful anaesthetic, and shows the least pressure-reversal effect of the intravenous agents studied.

Emergency drugs should not be withheld because of uncertainty about their effects in a hyperbaric environment.

PROPHYLAXIS AND THERAPY OF DIVING DISORDERS

Barotrauma

Divers seek therapy to allow them to enter the water when they might otherwise be unfit. The most common use of drugs is to prevent barotrauma of the middle ear or sinuses. Both topical and systemic vasoconstrictors are employed to shrink the nasopharyngeal mucosa to allow pressure equalization through the sinus ostia or eustachian tubes.

Some state that the widespread use of these drugs testifies to their safety, but problems may arise from this concept. Too often the drugs are used to overcome incorrect diving techniques, upper respiratory tract infections or allergies. Diving clinicians are aware of the problem of rebound congestion leading to either barotrauma of ascent or descent (see Chapters 7 and 8) later in the dive with topical vasoconstrictors.

Pseudoephedrine is used alone or as a common component of 'cold and flu' preparations. Side effects may include tachycardia, palpitations, hypertension, anxiety, tremor, vertigo, headache, insomnia, drowsiness and rarely hallucinations. Some individuals show a marked intolerance, however. For example, one diver suffered severe vertigo and unconsciousness in a dive to 45 metres having used an excessive amount of oxymetazoline spray before the dive.

Oxygen toxicity and nitrogen narcosis

Drugs have been sought to prevent or reduce the effects of raised partial pressures of these gases, but they remain in the research field and are of no practical value to the diver (see Chapters 15 and 17). No drug has been shown to overcome nitrogen narcosis in humans.

Diazepam has been used to prevent or treat the convulsions of oxygen toxicity (see Chapter 17). In addition, the roles of gamma-aminobutyric acid (GABA) analogues, superoxide dismutase and magnesium sulphate are under investigation.

It must be borne in mind that the use of drugs to prevent toxicity in lungs or brain may allow the development of toxicity in other organs.

High-pressure neurological syndrome (HPNS)

The addition of nitrogen or hydrogen to helium/oxygen to reduce the onset and severity of this syndrome is an interesting example of the interaction of pressure and a 'drug' (see Chapter 20). Drugs, which potentiate transmission at GABA synapses such as the anticonvulsant sodium valproate and flurazepam, protect against HPNS in small mammals.

Decompression sickness (DCS)

Prophylaxis

Researchers have considered the use of drugs to try to prevent or minimize this condition, and indeed drugs which inhibit platelet adhesiveness have been shown in animals to reduce the morbidity and mortality from DCS.

Post-decompression thrombocytopenia is well known in humans. It has been suggested that aspirin might prevent DCS by inhibiting platelet aggregation induced by intravascular bubbles. However, possible disastrous haemorrhagic effects, perhaps in

the inner ear, or barotrauma or spinal DCS, preclude its clinical use.

A number of other drugs have been suggested because of a possible influence on the blood–bubble interface. Alternatively, drugs might be used to reduce the release of tissue injury mediators from cells. Drugs such as isoprenaline (isoproterenol), which enhance cardiac output and cause general vasodilatation, have been shown to shorten the time to desaturate slower tissues in rats.

No drug has been proven safe and useful in preventing DCS in humans.

Therapy

Drugs used as an adjunct to the treatment of DCS are discussed in Chapter 13.

Drugs may be encountered in diving in four ways:

- *Prevention of disorders produced by the diving environment.*
- *Treatment of dysbaria-induced diseases.*
- *Treatment of concurrent diseases, acute or chronic.*
- *Drugs taken for 'recreational' or social purposes.*

THERAPY OF COINCIDENT ILLNESS

Quite apart from the unknown effect of many therapeutic drugs in the aquatic and/or hyperbaric environment, it is often the condition for which these drugs are taken that renders diving unwise (see Chapters 52, 53 and 54). For example, the use of a tetracycline antibiotic might be acceptable for chronic acne, but not for bronchitis; likewise, a thiazide diuretic may be acceptable for mild hypertension but not for congestive cardiac failure, and a calcium channel blocker may be tolerable for hypertension but not for paroxysmal supraventricular tachycardia.

Even if the medical condition for which the drugs are taken – and the possible interaction with pressure – are ignored, many drugs may themselves produce unwarranted risks. This is reflected in the denial of flying or driving licences to people who require certain therapeutic drugs. More stringent considerations should apply to the use of drugs in the subaquatic environment.

The **mode of administration** of drugs is relevant in certain situations. For example, drugs given in depot form may lead to localized scarring and be a nidus for bubble development. Certain implantable infusion pumps, especially those with a rigid casing, are designed to operate under positive internal pressure to deliver the drug and to prevent leakage of body fluids back into the pump. If the external pressure exceeds this, then not only is the drug not delivered, but body fluids will leak into the pump. Upon decompression, the increased pressure in the pump may suddenly dispense the contents into the tissues or blood with potentially disastrous consequences.

Some drugs or classes of drugs, which might be encountered in the diving population with possible associated risks in diving, are discussed. A number of these – the possible side effects of which the diver may be unaware – are available 'over the counter' (OTC) without a prescription:

- **Diuretics**: these may cause dehydration, or electrolyte loss, especially in a tropical environment, and this may increase the risk of decompression sickness. Potassium loss may predispose to arrhythmias.
- **Anti-arrhythmics**: under certain circumstances, these may provoke arrhythmias. Amiodarone produces photosensitivity and may impair vision. Beta-blockers and calcium channel blockers are also used as anti-arrhythmics.
- **Beta-blockers**: these are often prescribed for hypertension, but they also decrease the heart rate response to exercise and reduce exercise tolerance – effects that may be critical in an emergency situation. Bradycardia and other dysrhythmias may also be produced. Beta-blockers may also cause bronchoconstriction, especially in persons with a history of asthma. Divers may complain of cold hands or even a Raynaud's-like syndrome of the fingers on exposure to cold water. Topical beta-blockade for control of glaucoma

may be absorbed systemically and cause bronchospasm, bradycardia, arrhythmias or hypotension, and decreased stress response. Newer agents such as betaxolol are more specific and cause fewer cardiorespiratory effects than, for example, timolol drops. Divers using one of these specific eye drops should have no underlying cardiac disease and no abnormalities on cardiorespiratory assessment.

- **Peripheral vasodilators**: these include calcium channel blockers, and may produce orthostatic hypotension. This is not a problem underwater but it is at the moment of exit, due to loss of hydrostatic support. Dizziness or syncope may result.
- **Angiotensin-converting enzyme ('ACE') inhibitors**: these can produce a dry cough and airway mucosal swelling, which may be dangerous or diagnostically confusing in the diving/hyperbaric environment. There are also reports of bronchospasm. These side effects usually appear within the first two weeks of treatment. However, if these side effects do not develop, then they are probably the drugs of choice to treat hypertension in the diver. The newer angiotensin II receptor antagonist drugs, such as irbesartan and telmisartan, might not cause these problems.
- **Psychoactive drugs**: these include sedatives and tranquillizers, and produce varying degrees of drowsiness. The recipients are advised to avoid activities such as driving a car or operating machinery, where decreased alertness may be dangerous. Diving falls into this category. Potentiation of inert gas narcosis is also possible.
- **Phenothiazines**: these may produce extrapyramidal syndromes, and some which are used to treat nausea or motion sickness (e.g. prochlorperazine) may produce oculogyric crises.
- **Tricyclic antidepressants**: these are known to cause cardiac arrhythmias – an affect which may be exacerbated under conditions of increased sympathetic stimulation.
- **Antihistamines**: these are mainly used for their anti-allergy effects (urticaria, hayfever, allergic rhinitis, anticholinergic effects, drying effect with the 'common cold') or as anti-emetics (e.g. motion sickness). The main side effect important in diving is sedation. Newer agents, such as fexofenadine and loratadine, do not appear to cross the blood–brain barrier significantly, so drowsiness is less of a problem. The latter drugs are used for allergic rhinitis and not as anti-emetics. Many antihistamines are available OTC.
- **Anticholinergics**: these are used as antispasmodics for the gastrointestinal and urinary systems, but they cause dry mouth, blurred vision, photophobia and tachycardia. Decreased sweating may lead to heatstroke in the tropics. Divers may not be aware that anticholinergics such as atropine may be present in OTC anti-diarrhoea preparations.
- **Antibiotics**: these are probably safe for the diver, bearing in mind the indication and provided that there are no side effects. In the diving context, photosensitivity – especially to tetracyclines – may be relevant. Many antibiotics also cause nausea and vomiting.
- **Acyclovir**: this is used not only for HIV but also for oral and genital herpes. It may cause headaches, nausea and vomiting.
- **Anti-malarials**: these may be prescribed for divers visiting tropical areas. Divers and their medical advisers should be aware of the side effects of any antimalarials prescribed. Most antimalarial chemoprophylactic regimes must be commenced one to two weeks before departure, as this allows time for blood levels to reach a steady state. However, it also allows adverse reactions to occur, so that medications can be altered before departure. Apart from idiosyncratic adverse reactions a major concern among the widely prescribed antimalarials relates to **mefloquine** (Lariam), the common side effects of which include vertigo, visual disturbances, difficulty with coordination and psychosis. As the drug has a half-life of approximately three weeks, these effects can take some time to subside if they occur. Severe neurological symptoms have been reported two to three weeks after a single dose. Mefloquine has been withdrawn in some countries, as it can also cause bradycardia – an effect potentiated by the concurrent administration of beta-blockers. Safer alternatives for divers would include doxycycline, chloroquine and pyrimethamine (Maloprim).
- **Analgesics**: these should not be required in order to dive, although they may be appropriate in the recompression chamber. The problems of aspirin

have been discussed earlier. Paracetamol is probably safe. Non-steroidal anti-inflammatory drugs (NSAIDs), which may be available OTC, may precipitate bronchospasm and commonly cause gastric irritation and heartburn. The newer cyclooxygenase (COX) 2-specific inhibitors, which were introduced to avoid the gastric effects, have been associated with cardiac abnormalities and bronchospasm. Stronger analgesics reduce mental performance and may combine with inert gas narcosis to produce a more marked degree of central nervous system depression, as well as complicating the therapeutic assessment of pressure effects. Nausea is a common side effect.

- **Insulin and oral hypoglycaemics**: these may cause severe hypoglycaemia, with altered consciousness. The diving environment hinders the early recognition and self-treatment of insulin-induced hypoglycaemia (see Chapters 53, 54 and 57).
- **Thyroxine**: this may cause tachycardia, arrhythmias, tremor, excitability and headache. Hyperbaric oxygen toxicity is enhanced.
- **Steroids**: these may produce a wide variety of adverse reactions such as fluid and sodium retention and potassium loss, diabetes, peptic ulceration, avascular necrosis of femoral or humeral head (see also Chapter 14), thromboembolism, increased susceptibility to hyperbaric oxygen toxicity and infection. Anabolic steroids are widely abused for strength and body-building. Side effects include psychosis, aggression behaviour and sudden death.
- **Oral contraceptives**: because of their tendency to cause hypercoagulability of the blood, these were postulated to cause increased DCS, but this is probably not significant with the modern, low-dose agents (see Chapter 60). Some women experience migraine, nausea and increased mucosal congestion.
- **Bronchodilators**: these are contraindicated as the indication for taking them precludes diving. Nevertheless, some physicians have advocated their use before diving. This is imprudent, as theophylline and derivatives may produce cardiac arrhythmias and also allow bubble transfer through the pulmonary filter. Adrenergic drugs, even beta-2-selective drugs, taken orally (or more usually as inhaled aerosols) may have a cardiac stimulant effect. Aerosols, while producing marked improvement or prevention of symptoms, may still leave small regional microscopic areas of lung unaffected, and this may lead to localized air trapping.
- **Anti-neoplastic drugs**: bleomycin, which is used to treat testicular cancer and reticuloses, renders the patient especially sensitive to pulmonary oxygen toxicity.
- **Histamine H_2-receptor blockers**: these include cimetidine, ranitidine, famotidine and nizatidine, and are used to treat peptic ulceration and reflux oesophagitis. However, they may produce drowsiness and headache. Nizatidine has a lower incidence of such side effects. The **proton pump inhibitors** such as omeprazole and pantoprazole are used for similar indications, but do not appear to caused such problems.
- **Statins**: the plasma cholesterol-lowering statins represent another widely used class of drugs in modern society, but they do not appear to have any significant side effects.

RECREATIONAL DRUGS

These drugs, which may be either legal or illegal under various national legislations, are widely consumed (regularly or occasionally) for social, peer pressure or mood-altering qualities. They include tobacco, alcohol, marijuana, sedative-tranquillizers, hallucinogenics, cocaine and opiates (heroin, morphine, pethidine, etc.). These drugs may be taken singly or in combination. A Los Angeles coroner reported that 20 per cent of diving deaths in southern California were associated with the use of drugs.

Tobacco

The acute effects of nicotine include increased blood pressure and heart rate, and coronary vasoconstriction. The inhalation of tobacco smoke containing nicotine and tar causes increased bronchospasm, depressed cilial activity and increased mucus production in the bronchial mucosa. This may lead to intrapulmonary air trapping and increased pulmonary infection, and there is therefore an

increased possibility of ascent pulmonary barotrauma.

Carboxyhaemoglobin levels in smokers range from 5 per cent to 9 per cent. Significant psychomotor effects from exposure to this level of carbon monoxide have been reported.

Many studies of smoking and physical fitness show detrimental effects. Increased heart rate and decreased stroke volume are the opposite of the changes with aerobic training. Oxygen debt accumulation after exercise is greater among smokers.

The long-term use of tobacco may lead to chronic bronchitis and emphysema, with decreased exercise tolerance and eventually marked hypoxaemia. It may also lead to coronary artery disease and peripheral vascular disease. Reduced blood volume and decreased haematocrit also develop with long exposure to increased carboxyhaemoglobin.

Nasopharyngeal mucosal congestion may predispose to sinus and middle-ear barotrauma.

Alcohol

The acute effects of alcohol are well known, particularly its depressant effects on central nervous system functioning. Alcohol is associated with up to 80 per cent of all drowning episodes in adult males.

The detrimental effect on intellectual function, judgement and coordination is well known. The addition of hyperbaric air (nitrogen narcosis) has a synergistic action in impairing mental performance.[5]

Studies in pilots have shown significant performance decrements at blood alcohol levels of 0.04 per cent, which is a permissible level for driving in many countries. Visual tracking performance is reduced at 0.027 per cent. Alcohol also lowers blood glucose, with consequent performance impairment.

Other acute effects of alcohol that are relevant to diving include:

- increased risk of vomiting;
- peripheral vasodilatation increasing heat loss leading in turn to hypothermia;
- a diuretic effect causing dehydration and increasing the risk of decompression sickness; and/or
- dose-dependent impairment of left ventricular emptying at rest, probably acting directly on the myocardium.

The long-term over-use of alcohol is associated with damage to the liver, brain and heart as well as an increased risk of a number of other diseases.

Marijuana

Cannabis intoxication alters perception of the environment and impairs cognitive and psychomotor performance.[6,7] The 'mind-expanding' experience produces a sensation of heightened awareness, even for things that are not physically present. There may be feelings of euphoria, indifference, anxiety or even paranoia. Subjects may experience fears or 'hang-ups' of which they were not previously aware. Nitrogen narcosis can produce similar effects, and the notion of combining these effects is 'mind-boggling'. Impaired judgement and ignoring routine safety procedures while influenced by cannabis may be a major contributory factor in some diving accidents.[8]

Physiological effects include conjunctival injection, tachycardia, and increased oxygen consumption and heat loss with decreased shivering threshold. Hypothermia may thus develop insidiously. An acute toxic psychosis can also develop, with 'flashbacks', depersonalization and derealization. A high proportion of users occasionally experience 'bad trips'.

Regular use is associated with suppression of the immune system and respiratory disease. Heavy smoking can produce respiratory irritation and isolated uvulitis.

Adverse effects of cannabis on diving performance[6,7]

- *Slowed complex reaction time*
- *Space and time distortion*
- *Impaired coordination*
- *Impaired short-term memory*
- *Impaired attention, especially for multiple tasks*
- *Additive effects with alcohol and other drugs*
- *Impaired temperature control*

Caffeine

Caffeine is present in coffee and a variety of cola drinks. Moderate coffee drinking is not proven to be associated with increased risk of coronary heart disease and is probably safe in most healthy persons. However, acute consumption does produce a small increase in blood pressure and may induce cardiac arrhythmias or even lethal ventricular ectopic activity in certain susceptible individuals.

Caffeine withdrawal symptoms include headache and fatigue. Less often anxiety, nausea, vomiting and impaired psychomotor performance are seen.

Amphetamines

Methamphetamine, even in lower dose ranges, has been associated with erratic driving, risk taking, and increased accident rate. Higher doses cause tachycardia, dilated pupils, paranoia and aggressive behaviour. Withdrawal is characterized by fatigue, somnolence and depression. As in driving, methamphetamine is likely to produce symptoms that are inconsistent with safe diving.

Sedatives and tranquillizers

These drugs will also impair judgement and performance. Interaction with the diving environment produced the following extraordinary case (see Case report 43.1).

Cocaine

The most marked pharmacological effect of this useful topical local anaesthetic and vasoconstrictor drug is intense sympathetic stimulation both centrally and peripherally.

Symptoms, which are dose-related and rapid in onset, include changes in activity, mood, respiration and body temperature, blood pressure and cardiac rhythm. Morbidity and mortality are mainly associated with its cardiac toxicity producing myocardial infarction and cardiac arrhythmias in young people.[9] Sudden cardiac death syndrome in athletes taking cocaine is well known. Alcohol and cocaine are synergistic in producing cardiac damage. Cerebrovascular accidents, pneumomediastinum, rhabdomyolsis with renal failure and intestinal ischaemia have also been described. Death may also ensue from respiratory failure or convulsions.

CASE REPORT 43.1

DS, a 20-year-old man, dived to a depth of between 45 metres and 50 metres with companions, all of whom were deliberately under the influence of large oral doses of barbiturates, taken while on the way out to the dive site by boat. After an undetermined period under water, the patient lost consciousness at depth and was taken rapidly to the surface by his companion. On returning to shore, he was driven to his companions' apartment, where he remained unconscious, presumably sleeping off the drugs. On regaining semi-consciousness 48 hours later, he found that both legs were partially paralyzed and that he had paraesthesiae below the knees and in the palm of his left hand. He was dizzy and ataxic. He presented to hospital 60 hours after diving. A diagnosis of spinal decompression sickness was made with possible cerebral involvement from air embolism, although no firm evidence of pulmonary barotrauma was found.

Immediate management was by intravenous infusion of fluids, high doses of steroids with hyperbaric oxygen (USN Table 6). He improved rapidly with recompression.

(Courtesy of Dr I.P. Unsworth)

Cocaine inhalation ('snorting') produces intense mucosal ischaemia and may lead to infarction of nasal cartilage. Cocaine smoking has been reported to produce severe reactive airway disease.

Withdrawal symptoms are marked by severe depression.

Opiates

This class of drugs covers heroin and its derivatives, including morphine, papaveratum and synthetic opiates such as pethidine, fentanyl, dextromoramide and dextropropoxyphene. Although the analgesic effects of morphine have been shown to be little affected at pressure, the behavioural effects of these drugs with hyperbaric air have not been widely investigated. High doses of these drugs can produce respiratory depression, while lesser doses can produce alterations of mood and impair psychomotor performance. These effects may be altered in an unpredictable way by immersion and hyperbaria.

Other abused drugs with significant effects that may impact adversely on diving performance include **hallucinogenics** such as lysergic acid diethylamide (LSD), phencyclidine (PCP) and ketamine.

Performance-enhancing drugs are widespread in sport and may be encountered in certain diving populations. They are a mixed group, which include beta-blockers, amphetamines, anabolic steroids, hormones, diuretics and sympathomimetics.

The development of novel **methods of self-administration** of illicit substances has increased the incidence of pulmonary complications.[10] This may be due to the route of administration, the presence of contaminating foreign material or microbiological pathogens, or altered host immune response.

CONCLUSION

There is little information on the effects that drugs have on human physiological or psychological performance in the aquatic hyperbaric environment. Conversely, there is more information on the effect of certain drugs on animals under extreme hyperbaric conditions. This research is often designed as much to elucidate mechanisms of drug action and biochemical functions as to define the safe uses of drugs under pressure. Relatively few research data exist on the effect of drugs at conventional scuba depths, and more research is needed in this area.

In the meantime, a knowledge of both the diving environment and pharmacology – including side effects – should guide rational advice to divers.

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44

Long-term effects of diving

ROBYN WALKER AND CARL EDMONDS

Undisputed causes of diving-related residual sequelae include decompression sickness, cerebral arterial gas embolism, inner-ear and other barotrauma sequelae and dysbaric osteonecrosis. There is also some evidence suggesting that changes in bone, the central nervous system and the lung can be demonstrated in some divers who have not experienced a diving accident and who have no discernable clinical disease.

INTRODUCTION

With the improvement in prevention and treatment of diving accidents over the past few decades, attention has now been focused on the possible undesirable long-term health effects of diving. This interest has been encouraged by an increase in commercial diving activity (together with its workplace health and safety aspects), greater attention to worker's compensation and litigation, medical research funding and other financial grants, and finally to the proliferation of sophisticated high-technology techniques developed for medical investigation.

Much of the investigative efforts in this field have centered on the professional diver and yet, for the large numbers of recreational divers these concerns are also very real.

Long-term effects can be defined as an effect:

- outside the range of normal in an appropriately matched population;
- causally related to diving;
- persisting beyond the acute and rehabilitation phase of a diving accident;
- without non-diving pathology available to explain it; and/or
- producing a demonstrable reduction in the performance or quality of life of the diver.

There is no dispute that some diving accidents may result in permanent sequelae. Such diseases include:

- dysbaric osteonecrosis;
- neurological decompression sickness (DCS);
- pulmonary barotrauma with cerebral arterial gas embolism (CAGE);
- hypoxia from any cause;

- hearing loss and vestibular damage;
- other otological and sinus damage;
- many gas toxicities, including oxygen, carbon monoxide, carbon dioxide and HPNS; and
- some marine animal injuries.

A more difficult question to address is: Do long-term health effects occur in divers who have not suffered a specific diving accident?

AETIOLOGY

A variety of physiological and pathological changes have been postulated to produce a great variety of long-term health effects, not related to a specific diving accident. These non-exclusive aetiologies include:

- increased environmental pressure;
- increased gas partial pressures;
- oxygen toxicity;
- gas-induced osmosis;
- asymptomatic bubble development, with local tissue effects, blood bubble interaction (haematological effects), and blood–brain barrier disruption;
- barotrauma damage to surrounding tissues;
- asymptomatic lipid emboli; and
- adaptive effects of diving.

DYSBARIC OSTEONECROSIS

This significant and serious disorder has been reported in divers who have not necessarily suffered DCS. It is essentially a disease of divers exposed to environmental pressures for moderately long durations, and it is therefore more frequent in caisson workers, commercial and scientific divers and others who have prolonged exposures. It is fully discussed in Chapter 14.

NEUROLOGY

Barotraumas may permanently damage some cranial nerves (facial, infra orbital maxillary, cochlear, vestibular) (see Chapters 7, 8, 9, 37 and 38). (See Chapter 40 for a description of the various neurological diseases, and possible sequelae, induced by diving.)

A comparison of 156 divers with 100 non-diving controls[1] revealed that divers had a higher incidence of specific neurological symptoms and episodes of cerebral dysfunction, in non-diving situations. They had more general neurological symptoms and abnormal signs, the latter predominantly involving the distal spinal cord and peripheral nerves. There was an independent correlation between these abnormalities and diving exposure, DCS and age.

DCS does leave some individuals with permanent neurological symptoms and signs, the damage being typified by the classical case of spinal DCS with permanent paraplegia. Lesser manifestations may vary from the obvious and debilitating, to mild. Mild residual symptoms include paraesthesia and patchy areas of numbness from peripheral nerve involvement. Subjective manifestations such as short-term memory loss and difficulty in concentrating may require formal psychometric testing to identify their presence and quantify their degree.

The existence of incomplete resolution of neurological DCS as a long-term effect of diving is unquestioned. Whether or not subclinical and cumulative lesions can occur in divers who have never experienced clinical DCS, or whether they may develop after complete recovery is unknown. If they do occur, can they be avoided by conservative diving practises, and how important are they clinically?

Central neurological damage may be transient, or it may be permanent and cumulative. Electroencephalographic (EEG) changes after diving accidents or incidents may indicate damage, but the alteration in EEG behaviour is sometimes difficult to interpret. Computed tomography (CT) and magnetic resonance imaging (MRI) scans, as well as evoked cortical potentials may only reveal gross abnormalities and may miss small multifocal damage (see Chapters 40 and 41).

Investigators have turned to some of the newer radiological and scanning techniques in order to detect neurological effects of diving, though some of these techniques, in their own right, have yet to be adequately standardized.

^{99}Tc-HMPAO-SPECT scanning

Technetium-99 hexamethylpropyleneamine oxime-single photon emission computer tomography (^{99}Tc-HMPAO-SPECT) is a technique used to image regional blood flow. HMPAO is radiolabelled with technetium-99, and SPECT utilizes single gamma ray-emitting radiotracers, which are detected by gamma cameras to produce three-dimensional images. The ^{99}Tc-HMPAO is injected intravenously and diffuses across the blood–brain barrier. The complex then remains bound in the brain tissues for up to 8 hours, effectively producing a frozen picture of regional blood flow at the time of injection.

Adkisson *et al.*[2] first reported the use of this technique in diving accident victims in 1989. They studied 28 patients within one month of presentation with DCS (23 patients with neurological DCS, four with CAGE and one patient with a limb bend). These authors reported cerebral perfusion deficits in all cases of neurological DCS and CAGE, and a high degree of correlation between the clinical picture and the site of the perfusion deficit. The possibility of occult neurological damage was raised by the appearance of cerebral perfusion deficits in divers who showed clinical signs only of spinal cord involvement. The patient with a limb bend had a normal scan.

Subsequent investigations have raised doubts about the significance of these findings. A study by Hodgson *et al.*[3] in 1991 compared 10 divers with acute DCS to 10 divers who had been treated some three to five years earlier for DCS, 10 divers who had never experienced DCS, and 10 population controls. Although there was a trend towards a larger number of deficits in individuals with DCS, there was no statistical difference between the groups, and there was no apparent correlation between the sites of the perfusion deficits and the clinical presentation. There was also a higher than predicted number of positive scans in both the divers never treated for DCS and the non-diver controls. These findings therefore do not support the assumption that asymptomatic divers sustain neurological injury, as the same deficits were seen in individuals who had never dived. The significance of these deficits and the incidence in control populations is yet to be determined, however.

Evans *et al.*[4] reported the results of ^{99}Tc-HMPAO-SPECT scanning in 54 patients who presented to the Aberdeen Hyperbaric Unit with DCS. Of these patients, 62 per cent were reported to have abnormal scans, but there was no correlation between the site of the deficit and the clinical presentation. This study was then extended to include divers who had no history of DCS and non-divers.[5] The authors concluded that although the divers with abnormal scans in the DCS group tended to show a greater diving exposure, this pattern was not repeated in the 'diver' group. They could not conclude from these results therefore that greater diving exposure increases the likelihood of an abnormal ^{99}Tc-HMPAO-SPECT scan.

Divers with subclinical pathological deficits

Palmer *et al.*[6] reported the autopsy of a male sports diver who had recovered almost completely from an episode of spinal DCS, four years before death, but who was subsequently found to have extensive morphological changes in the posterior and lateral columns of the spinal cord. Palmer and colleagues[7] also have examined the spinal cords from eight professional and three amateur divers who died accidentally. All cases bar one had had a diving medical examination between 2 and 38 weeks before death, but none of the cases revealed a history of DCS and none had documented neurological abnormalities. Marchi-positive tract degeneration was found in the spinal cords of three professional divers, variously affecting the posterior, lateral and to a lesser extent the anterior columns. In one diver there was degeneration of afferent fibres within the posterior columns. These features were difficult to recognize in haematoxylin and eosin-stained sections, but were clearly shown by the Marchi technique. Marchi-positive material does not appear in degenerating myelinated fibres until seven to ten days after the initiating lesion, and does not appear intracellularly until some ten weeks after the lesion.

Palmer *et al.*[8] also examined the brains from 12 amateur and 13 professional divers, all but one of whom died accidentally. Only three of the 25 subjects had reported a previous episode of DCS.

Grossly distended, empty vessels were found in the brains of 15 out of 22 divers who died from diving accidents, these presumably being caused by gas bubbles (see Post-mortem decompression artifact, Chapter 51). The most striking long-term change observed in the brains was that of perivascular lacuna formation found in the cerebral and/or cerebellar white matter of three amateurs and of five professionals. In addition to lacuna formation, hyalinization of vessel walls was present in the brains of three amateurs and five professionals. Necrotic foci in the grey matter occurred in seven cases, and perivascular vacuolation of white matter occurred in seven cases. The authors proposed that these changes most likely arose from intravascular gas bubble formation producing a sudden distention and occlusion of small arterial vessels. With passage of the bubble, the vessel returned to its normal size, leaving a surrounding area of degenerated tissue within the lacunae. Hyalinization of the vessel wall was also thought to occur as a consequence of this rise in luminal pressure. In one previously asymptomatic professional diver, there was also unilateral necrosis of the head of the caudate nucleus.

The above pathological findings await independent verification, but they provided evidence of chronic changes (lacunae formation and hyalinization of vessel walls) in the brains of asymptomatic divers who did not have a past history of DCS when compared with control subjects. This may be relevant, as it is now generally accepted that bubbles are produced with all but the most innocuous dive profiles.

NEUROPSYCHOLOGY

Over the years a number of anecdotal reports in the literature, combined with an almost folklore belief among many occupational air-diving groups, have generated the hypothesis that diving in the absence of a gross insult causes brain damage and dementia in divers, though reputable studies to support this hypothesis are lacking (see Chapter 41).

BEHAVIOURAL FACTORS

Perhaps one of the most important basic findings, when comparing divers with non-divers, is the difference in their psychological and behavioural characteristics. A description of the psychological factors associated with the diving personality is provided in Chapter 41.

When it became evident that divers – and especially those who undertook excessive diving activities – perform differently in both psychological testing and behaviour, conclusions were often drawn as to the possible effects of diving on a 'normal' or 'average' personality. Not only did divers perform differently on the psychological tests, but they also differed in their mode of death. There was a preponderance of traumatic causes for death, and of suicides. The incidence of accidental deaths was higher, from drowning and diving accidents. Non-diving accidents (which included murders) comprised 20 per cent, and suicides 17 per cent, of the deaths of occupational divers.

There are a variety of explanations that need to be considered, before it is concluded that these statistics reflect the effect of diving. Rather, they may reflect the personality of divers. It is possible – and indeed likely – that divers are not identical to the 'normal' population. Indeed, they may well be attracted to this occupational activity by their love of adventure, risk-taking behaviour, physical activity, etc., and may well succeed in this occupation because of their reduced innate levels of anxiety (neuroticism, trait anxiety). This combination of factors would be expected to lead to situations in which their love of adventure becomes dangerous, or in which their propensity for physical activity may be inappropriate.

It is possible that the higher instance of violent death and suicide may be related to the lifestyle, which includes not only the diving activities, but also exposure to hazardous environments, possible drug and alcohol abuse and other unknown influences. Alternatively, it is also feasible that the neuropsychological sequelae associated with diving accidents, or some other form of cumulative brain damage, could lead to the behaviour referred to above.

HEARING LOSS AND VERTIGO

One of the most obvious long-term sequelae of diving is that of hearing loss. (This is fully described in Chapter 37, while the less appreciated permanent vestibular damage is described in Chapter 38). Because the hearing loss, from a variety of causes, is more easily recordable and perceived by the subject, it is more frequently documented. Permanent vestibular damage is likely to be compensated by adaptive neurological processes, which may result in the subject not being aware of any deficit. It may only be evident in provocative situations or with vestibular function testing.

SINUS DISEASE

Although rarely mentioned in this context, this is a common long-term sequelae of diving. It is usually a result of repeated sinus barotrauma (see Chapter 8), with the subsequent infections sometimes superimposed on the barotraumas.

The problem of chronic sinus disease is often a cumulative result of inappropriate diving activities, and especially when the divers persevere despite the evidence of barotrauma and infections. The reduction of the lumen of the sinus ostia or duct, results from chronic inflammation and gradual scarring. In these circumstances it is frequently the very experienced diver who will present at the latter part of his diving career with the development of sinus barotrauma interfering with descent.

Investigations on divers (e.g. MRI of the skull) will often coincidentally demonstrate the presence of chronic sinus disease.

OPHTHALMOLOGICAL EFFECTS

Because the eye develops as an extension of the forebrain it has long been recognized that the fundus may reflect changes occurring within the central nervous system. Lesions of the retina may be indicative of more widespread changes within the central nervous system. Such pathologies include oxygen toxicity, DCS and CAGE.

Polkinghorne *et al.*[9] used retinal fluoroscein angiography in 84 divers and 23 non-divers to determine whether blood vessel changes are common in the ocular fundi of divers. They proposed that such changes might provide an indication of vascular obstruction elsewhere, particularly in the central nervous system. Twelve of the divers had been diagnosed previously with DCS, nine neurological (none had visual symptoms) and three joint pain only. The authors reported that retinal capillary density at the fovea was low in divers, and microaneurysms and small areas of capillary non-perfusion were seen. The divers had significantly more abnormalities of the retinal pigment epithelium than the comparison group of non-divers. A positive correlation was found between the presence of fundus abnormality and length of diving history. No subject had any recorded visual loss as a consequence of diving.

These authors concluded that all changes were consistent with obstruction of the retinal and choroidal circulations, and that this obstruction was likely to be due to intravascular bubble formation during decompression, or to the altered behaviour of blood constituents and blood vessels under hyperbaric conditions.

Other investigators have not reproduced these findings, however. Holden *et al.*[10] performed fluoroscein angiography on 26 divers, who had used safe diving practices for at least ten years, and on seven controls. There was no significant difference in the incidence of macular abnormalities between these groups, which led the authors to suggest that adherence to safe diving practices might protect against the effects reported by Polkinghorne and colleagues.

Murrison *et al.*[11] examined and compared the retinal fluoroscein angiograms of 55 Royal Navy divers and 24 non-diver servicemen. No differences were found between divers and non-divers, and the prevalence of abnormalities was not correlated with diving experience.

It is difficult to interpret the diverse findings of the above three groups. Military diving is conservative and highly regulated, and therefore the proposal by Holden that safe diving may protect against lesions is credible. However, the lack of correlation of length of diving history in military divers with presence of lesions disputes the accumulative dose effect as proposed by Polkinghorne.

Prospective longitudinal studies of recreational, military and professional divers will be required before it is known if diving results in these ocular vascular changes.

PULMONARY FUNCTION

This contentious subject requires clarification before any confidence is placed in the current beliefs. It has long been believed that some divers are able to tolerate higher levels of carbon dioxide than non-divers. If this is correct, then it is uncertain whether such a physiological anomaly is based on natural selection for diving activities, or is a long-term adaptation from diving. Also, whether it is of value in diving (permitting the diver to not react to slight variations in carbon dioxide tensions) or hazardous (making the diver more susceptible to carbon dioxide toxicity, oxygen toxicity or DCS) is unknown.

As regards static lung volumes, it is generally accepted that divers have larger vital capacities than non-divers,[12] although there are some surveys in which this was not evident. One major longitudinal study demonstrated that there was an initial adaptive increase in lung volume in divers, followed by a progressive decline, possibly age-related.

It has also been shown that divers develop some degree of airflow obstruction, due to airway narrowing.[13] One aspect of this is an increase in hyperreactivity that develops, demonstrated by histamine provocation testing. It has been suggested that some of these effects may be related to a cumulative pulmonary oxygen toxicity or to repetitive pulmonary gaseous microembolisation. This condition has also been observed in other groups of athletes.

There has also been a slightly diminished pulmonary transfer capacity (TLCO) after deep dives, which tends to improve in the following few weeks but may not be totally reversible.[12] Indeed, there is an increased amount of evidence suggesting that lung function is altered by diving. Reed[12] reports that:

- the respiratory effects of single deep saturation dives have been shown to include a reduction in pulmonary diffusion capacity, increase in lung volumes and impairment of exercise performance. There may also be deleterious effects on indices of ventilatory capacity. These changes indicate a peripheral lung lesion affecting gas exchange.
- both cross-sectional and longitudinal studies relating pulmonary function to diving history have demonstrated loss of function associated with indices of diving exposure.

The underlying aetiology of these changes is uncertain. Proposed mechanisms include lung overdistension, hyperoxia, vascular bubbles, exposure to increased ambient pressure *per se*, and gas and particulate contaminants associated with compressors and chambers. The association with diving *per se* may not be causal.

OTHER EFFECTS

A variety of other possible long-term effects of diving have been recorded. Cardiac hypertrophy has been reported in professional divers and may have sinister implications. It is also possible that there are contributions to cardiac disease associated with either intravascular emboli affecting the coronary arteries, or damage to the primary endothelium from these intravascular bubbles. In 1986 a study of 1977 US Navy diving officers[14] demonstrated an increased number of hospital admissions for stress-related disorders and cardiovascular disorders, but this was especially so among the younger, less-experienced diving officers.

Arthritic disorders have also been postulated to have a higher incidence in divers than non-divers. One study[15] which investigated the morbidity and mortality among 11 517 US Navy divers found that there was an increased hospitalization for joint disorders among the 23- to 28-year-olds, without any obvious explanation.

An investigation of 328 US Navy divers[16] who had had DCS showed a higher rate of hospital admission in subsequent years for headache, musculoskeletal disorders, vascular disorders, respiratory diseases and alcohol and drug abuse. Nevertheless, these occurred in less than 25 per cent of the post-DCS population, and half of these continued to dive.

Many haematological and some vascular changes have been noted with various forms of decompression, but few continue for any length of time after

ascent. One study[17] illustrated an increased incidence of chromosomal aberrations in cultured T lymphocytes of divers. The health effects related to these isolated observations are unknown, but no permanent haematological or vascular changes have been demonstrated, apart from those referred to above.

Investigations suggesting either subfertility of divers or a preponderance of females in the divers' progeny have not been substantiated.

Long term effects of injuries from marine animal envenomations have been observed, both physical and neuropsychological in type. There are little or no documented investigations of these cases, despite the plethora of clinical evidence and case reports of osteomyelitis (and even amputation) induced by various marine envenomations. Long-term inflammatory responses from foreign body reactions are also well known (see Chapter 32). Very significant neuropsychological sequelae from some of the fish poisons (see Chapter 33) have also been documented. In all of these there is a specific temporal relationship, continuing from the time of the specific incident.

CONCLUSION

In 1993 an international consensus conference on the long-term health effects of diving was held in Norway. The stated objective of the meeting was to agree upon possible long-term health effects of diving based upon present knowledge and experience, though there was a strong bias towards presumed decompression effects. The participants summarized current knowledge[18] and produced the following consensus statement:

There is evidence that changes in bone, the CNS and the lung can be demonstrated in some divers who have not experienced a diving accident or other established environmental hazard.

The changes are, in most cases, minor and do not influence the diver's quality of life. However, the changes are of a nature that may influence the diver's future health. Nonetheless, the scientific evidence is limited, and future research is required to obtain adequate answers to the questions of long-term health effects of diving.

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Stress responses, panic and fatigue

CARL EDMONDS

INTRODUCTION

Although compressed-air diving has been possible for most of the 20th century, it only became popular following the development of scuba equipment during the late 1940s. At that time, only those who sought excitement and had a genuine love of the sea would embrace the sport of diving. Natural selection dictated that these would be aquatic people – with skills and personality suited to this and other water activities – the 'waterman' concept. Diving was merely an extension of this overall interest and ability.

In the 1960s these 'natural' divers exploited their talents commercially and became instructors. They required of their trainees a level of water skills often in excess of that possessed by most, and few trainees took up this challenge. Those who did were required to show personality characteristics that could tolerate extremes of physical discomfort, environmental hazards and inadequate equipment.

During the 1970s, with the availability of more user-friendly equipment, and the change in attitude away from the hunter/killer male chauvinistic approach, there was a movement towards the ecologically perceptive diver, equipped with a camera more often than a catch bag.

Thus, the tough male stereotype of the pre-1960s was supplanted by the sensitive and sociable diver of the post-1980s. The belief that diving should be available to all even resulted in a number of handicapped groups entering the scuba diving world. These included paraplegics, blind and deaf divers, as well as the introduction of 'friends of divers', i.e. children, members of the diver's family, other peer groups, etc.

Some of the people now undertaking scuba diving are probably more accident prone – or at least less able to cope with demanding situations than those of the earlier years. As a result, new types of disorders are arising apart from the traditional diseases, and the stress syndromes of diving are typical of these. Diving trainees of the new millennium represent the general population, with a wide range of physical, medical and psychological limitations.

The ocean environment can be unforgiving, and may not make allowance for the personality of the new divers. Also, the user friendly equipment may expedite diving but promote new difficulties and dependency.

PERSONALITY FACTORS

The early divers were somewhat like the early aviators in that they were adventure seekers, and often carried out their activities because of necessity. They included such groups as explorers, treasure hunters (salvage) and military divers. In contrast, and with the increasing sophistication of equipment and the greater complexities of deep diving, the commercial divers of this decade are far more careful, obsessional and conscientious than their forebears.

The personality characteristics required for a recreational diver, who can choose the dive conditions and vary the duration, are quite different to those of the professional diver who may have to remain isolated in the underwater environment and be able to use underwater habitats for days or weeks at a time. In this text we will summarize some of the observations that have been made on divers.

Traditional beliefs

A need for excitement, or environmental stimulus, is probably required for scuba diving. In 1955, Yarborough stated that the diver not only had to have an absence of physical defects, but should also possess a stable psyche and a phlegmatic personality. The possession of a temperament free of alarmist characteristic was essential. This requirement was later verified by the progressively increasing numbers of divers who died as a result of 'panic'.

In 1957, Dr. Harry Alvis stated that divers were not the most normal of normal people, and that a special type of personality was required. Sir Stanley Miles, in 1962, stated in the first sentence of his textbook on *Underwater Medicine* that 'It is most important, right at the outset, to realize that the problems of man's adaptation to a watery environment are primarily those of temperament'.

Bowen and Miller, in 1967, stressed the hazardous nature of diving activities. Danger ('and drowning') was always one breath away, and while cooperation was imperative for safety, the diver was necessarily a lonely person, dependent more on his own quick actions for his safety. Later, in 1969, Caille stated that of all divers, the greater physical and mental demands were made upon the military and naval personnel who fulfilled a combat role. There is only a small margin underwater for deviations from normal health, and no one could predict when – and in what circumstances – a candidate would be exposed to excessive stress (danger).

Research observations

Many psychometric studies have been performed on divers, but as each group has different operational requirements, the results do not have widespread relevance. In general, full psychometric assessments probably do not have a predictive value that is commensurate with the time and cost of the investigations.

In most of the diving training during the 1950s and 1960s, there was a consistent 50 per cent failure rate in professional diving courses. This selection meant that some sort of standards were being applied. In comparison, today there is little or no failure rate in many of the recreational diving courses now being held – suggesting that few or no standards are being applied, other than the ability to pay.

Ross, in 1950, conducted an investigation into Australian Navy divers and found that the major characteristic required appeared to be sufficient self-control to face threatening situations, without disabling anxiety. He also noted the importance of an 'adventuresome approach, diligence in performing work, self-reliance, tolerance of discomfort and an indifference to minor injuries and illnesses'. Physical attributes such as stamina, athletic fitness and an affinity for strenuous effort were very important.

In the training of the underwater demolition teams (UDT) from the US Navy, psychometric testing revealed that mechanical and arithmetical comprehension was more highly correlated with success than other characteristics, such as clerical ability. Psychological tests on some groups of divers, such as UDTs, also showed quite different traits to the tests performed on other diving groups. Nevertheless, there were some characteristics that continued to be present among most divers, and these included objectivity, low neuroticism (trait anxiety), aggression and self-sufficiency. Fear and anxiety were not acceptable characteristics.

In 1967, Edmonds carried out a prospective assessment of 500 diving candidates, who were undergoing a diving course that had a recognized 41 per cent pass rate. A statistical analysis of the results showed that the diver was a psychologically stable, medically and physically fit individual who was not overtly worried by diving hazards, and had both the desire and ability to perform in the water environment. In comparison to the unsuccessful candidates, the diver was usually more mature, motivated by love of water sports (but not by adventure or comradeship), was not fearful of the hazards likely to be encountered, physically fit, thick-set (low Cotton's Index of Build), a non-smoker and free of medical disorders, very capable of breath-holding and swimming, intelligent, self-sufficient, non-neurotic, simple and practical. Exceptional **physical fitness** – and especially aquatic fitness – were important characteristics for successful diving. A failure to complete a 200-metre swim in less than 5 minutes, without swimming aids, was an indicator of poor aquatic fitness. After three years of detailed investigation, Edmonds had merely confirmed the anecdotal views of Yarborough and Ross, expressed many years previously.

Comparisons of divers with non-divers in the US Navy revealed that divers had less hospitalization rates for stress-related disorders, but higher rates for environmentally induced disorders. The interpretation that they did more but thought less is possibly an oversimplification.

Different personality characteristics are required for different types of professional diving. In saturation diving, the divers must work together within a small enclosed area where an affinity and ability for teamwork and tolerance are needed. An abalone diver, who works in isolation for many hours each day, does not require such social skills (and often does not possess them). A Navy diver who detonates or defuses underwater explosives needs good mechanical aptitudes, while technical divers need increased conscientiousness and obsessionality because of the narrower margin for error.

Divers performed quite differently on psychometric testing than their non-diving controls. Professional abalone divers, even in the 1980s, tended to be risk-taking types, and this was demonstrated in their attitude to – and results in – psychometric tests. Similarly, the range of skills required for recreational divers varies with the type of diving. Nevertheless, self-reliance and a freedom from neuroticism (low trait anxiety) seems common among all diving groups.

Within any occupational group, because the requirements are the same, **males** and **females** will probably have similar personality profiles.

Morgan and his colleagues verified that **anxiety** (trait anxiety, neuroticism) was likely to predispose to panic responses. **Introverted** people are more concerned with the exercise demands of diving, becoming more susceptible to exhaustion and fatigue, than extroverts. He demonstrated that **panic** was a frequent problem, even among experienced divers, and was observed in 54 per cent of recreational divers (64 per cent in females, 50 per cent in males). Males tended to report this later – when the event became life-threatening – than females.

STRESS RESPONSES

Stress responses assist in the survival of the species, but are of value to the individual only if not excessive. The stress response acts through the autonomic nervous system, which prepares the animal for 'fight or flight'. The respiratory and circulatory systems are stimulated, and there are biochemical and haematological changes to support this hyper-alert state. The animal is then ready for action in a state of high physiological excitation.

The stress responses in diving can only be understood by having an appreciation of the complex interaction of man, his technology and the environment in which he uses it. The reason that such responses are ignored in most diving texts is that they are psychologically complex and ill-defined, and do not lend themselves readily to academic study or pathological scrutiny. They include three of the most common causes of accidents and deaths from scuba diving (see Chapter 46):

- **Panic**: a psychological stress response, related to anxiety.
- **Fatigue**: a physiological stress response to exertion.
- **Sudden death syndrome**: A pathological stress response of the heart (see Chapter 56).

The induction of panic in the anxious diver, fatigue in the physically unfit and cardiac death in the medically unfit is more appreciated when one examines specific equipment problems (see Chapter 4) and environmental demands (Chapter 5), and is cognizant of the diving and training techniques that influence these reactions.

Training in appropriate behaviour and learning how to recognize and reduce the development of stress disorders – how to avoid them and how to ameliorate them – is a major factor in promoting diving safety. Repetition of skills related to equipment and experience of different environments are the mainstay of instruction.

It is not uncommon for less experienced divers to indulge in a type of self-deceit that will cause them to risk injury rather than admit they are facing a new challenge for which they are ill-equipped or ill-prepared. And yet, 60 per cent of recreational divers indicated that they had experienced trouble and required assistance at least once in the previous three years. Stress situations are inevitable in diving.

PANIC

Panic is a psychological response to stress, and is probably the most common single cause of death in scuba diving. It is an extreme form of anxiety identified by unreasoning fear and loss of control. It is produced when an animal perceives or experiences a threat (the stressors) which is either real (environmental or physiological) or imaginary (psychological). The latter can be as effective a stimulus in producing the stress response, as the more obvious physical causes.

Personality factors

The psychological response of the diver to actual or perceived problems and hazards is based on his innate susceptibility (neuroticism). The diver must be aware of problems to be able to create an anxiety state of sufficient magnitude to be termed 'panic'. Under identical conditions, different divers will react in different ways, and those with a tendency to high anxiety are more likely to react with panic.

Neuroticism (trait anxiety) is an indication of the individual's tendency to break down under stress. It is mainly inherited, and can be assessed by various personality tests or by monitoring the physiological responses to stress. However, there are some divers who have specific anxieties (state anxiety) to aquatic threats, e.g. drowning, claustrophobia or sharks, even with normal neuroticism levels.

Panic commences as a loss of confidence. The diver then experiences a loss of control over the situation, thereby producing a vicious circle in which further loss of confidence is experienced. Inappropriate behaviour very rapidly takes over, with the diver reaching a state in which self-preservation is threatened. Panic was implicated as a significant factor in more than 80 per cent of scuba diving fatalities surveyed in Los Angeles County in 1970. It also contributed to at least 39 per cent of the deaths in an Australian survey of recreational diving fatalities, probably 37 per cent of recreational scuba drownings and 58 per cent of recreational scuba near-drownings.

Anxiety is often induced by certain diving tasks, especially those of mask clearing, buddy breathing, free-ascent training, open-ocean diving, diving alone, etc., and it is known that the autonomic nervous system responses are exaggerated before, during and after these experiences. As in all other diving techniques, repeated non-stressful diving training and experiences promote confidence and reduce the degree of state anxiety.

Specific fears or phobias associated with the diving and aquatic environments are also reduced by acquiring relevant experience. This is achieved by considerate and repetitive training under those environmental conditions. It is referred to by behavioural psychologists as 'desensitization', and it follows that a diver who is exposed to certain environmental conditions repeatedly, and with good tuition, in training and preparation for emergencies, is less likely to act irrationally or to panic when subsequently faced with those conditions.

Sensory deprivation is referred to as the **Blue Orb syndrome** (see Chapter 41). Both this, and excessive sensory overload, will act by disrupting psychological equilibrium and predispose to panic. It can be influenced in either direction, depending on the thought processes (cognitive and appraisal) of the victim. A beneficial influence of low neuroticism will

It takes as long to die from panic as it does to assess the situation rationally and initiate effective and corrective action.

CASE REPORT 45.1

(This is a composite report, with many unwitting contributors)
Nick was a recently qualified diver using hired equipment, and diving in an unfamiliar area. He borrowed a wet suit, but it was a bit tight around the chest, and restricted his breathing. He decided to overweight himself with two extra 1-kg lead weights, because he felt that he may have some difficulty with descent under ocean conditions.

It was his first open-ocean dive since his training, and there had been some question regarding whether the conditions were suitable for diving. Even before entry into the water he was not entirely happy with himself.

Initially the dive was uneventful, other than Nick being a little apprehensive regarding his ability to outlast his companions.

Note: one of the more serious marine hazards is the diver who aims to dive deeper or longer, utilizing less air than his companions – thereby setting up a competitive situation and placing everyone in jeopardy, including himself.

In this particular dive Nick was convinced that he was using more air than his companions. This tended to aggravate his apprehension, and he wondered whether he was running out of gas. There was unfortunately no way to confirm this, as he was not wearing a contents gauge on the scuba regulator, and he felt that he was probably a long way from either the boat or the shore.

A mixture of inexperience and misplaced pride prevented him from surfacing to clarify his position, and he noticed that he was becoming rather more anxious. His breathing rate increased and, as if to confirm his worst fears, he noticed a resistance in his regulator. He then concentrated on his breathing and noted that both it and his heart beat seemed to be fairly rapid. He considered the possibility of turning on his emergency reserve, and finally decided to do this, to see if it would have any effect on the respiratory difficulty. It did not.

A thought flashed through his mind that he was not enjoying this dive. He had spent a disproportionate amount of time either looking at his equipment, his buddy or the surface. He did not inform his companion that the reserve valve had been activated, hoping against hope that his companions would be equally short of gas. Another fear was that he was not making very much headway against the current, and he thought that he was probably still a long way from completing the dive. He was now becoming far more apprehensive and there was greater resistance from the demand valve. He decided to leave his weight belt on for the moment, arguing that perhaps he could last out a little longer. He was becoming very anxious, with increased respiration and a greatly increased breathing resistance.

In fact – and putting it in its simplest terms – he was not getting enough air. The situation was serious. He decided to surface, fast. During ascent, which was rapid but not rapid enough for Nick's peace of mind, he could see the surface, but despite previous assurances to the contrary he did not get more air as he ascended. He just reached the surface in time to wrench off his facemask and regulator, feeling about to 'blackout'. By expenditure of considerable effort he managed to keep his head above water for a few terrifyingly precious seconds before one of the small waves washed over him, with some water getting into his mouth and causing him to cough.

He struggled hard to maintain his head above water. Unfortunately he was becoming very fatigued, exhausted, and wondered how long he could keep this up. Then strength and determination seemed to recede.

His 'buddy' realized that Nick had disappeared, and after delayed reconnaissance of the area, he decided to ascend. By the time he reached the surface, Nick was no longer to be seen. His body was subsequently found within a few metres of where he had sunk (despite the tidal currents), with his weight belt still fastened, his buoyancy vest uninflated, ample air in his scuba cylinder – and at a distance so close to shore that he could well have swum under almost any conditions, using mask, snorkel and fins.

Autopsy diagnosis: Drowning.

True diagnosis: Death from panic.

be affected by such intangibles as volition, self-confidence, experience and aquatic skills.

The above report illustrates the effects of: equipment limitations, inexperience, negative buoyancy, incorrect trimming causing 'drag', overbreathing the regulator, egoism, failure to appreciate his limitations, anxiety leading to panic, inadequate weight ditching and compensator inflation, syncope of ascent, salt water aspiration, fatigue and inadequate companion diving practices. Drowning explains only the final result.

The sympathetic nervous system responses to fear will produce a terror-stricken facial appearance, pallor, dilated pupils, shallow rapid respirations, rapid jerky movements and irrational behaviour. The diver focuses excessive attention on either his equipment or the surface – all these can be observed by companion divers. The affected diver would add dyspnoea, palpitations and a sensation of panic to the symptom complex. The physiologist would include hypertension, increased cardiac output, arrhythmias, increased circulation to muscles, adrenal and steroid hormonal secretions and cerebral excitation. Fear alone, without the addition of any other stress, can cause death.

Surveys of divers indicate that most trainees consider that they are not adequately acquainted with the frequency and importance of anxiety symptoms induced by diving, or with their recognition and treatment. Addressing this problem requires both classroom discussion and repeated practical experiences and supervised training in skills to cope with both equipment and environments. The usual basic open-water certification course is inadequate for this, and explains the high over-representation of novice divers in the recreational diving fatality statistics (7 per cent of the fatalities occur on the first open-water dive and 23 per cent on an early one).

Morgan noted that terms such as panic, stress and fatality, rate little or no mention in diving courses, texts or manuals. However, there are many factors, and any one of them can lead to a panic situation. Some of these will be dealt with later, and are listed in Table 45.1. Basically, they all lead to a diver's inability to cope with his equipment, or his environment, and they tend to relate one to another. For example, on examining the first cause in each column, it will be evident that a diver who is swimming against a tidal current will be more likely to become fatigued if he has reduced his efficiency by being overweighted. In some cases one factor will predominate, whereas in others a combination will produce the same eventual result. There are some circumstances in which any and all divers would panic. The art lies in recognizing and avoiding these circumstances as much as possible and ensuring that your capabilities are not exceeded by the limitations of your equipment and the demands of the environment.

Respiratory control – encouraging divers to learn to breathe slowly and deeply through the regulator – has been advised by many diving instructors. It has some experimental backing both in reducing the resistance to breathing and as a technique to control the cyclical aggravation of anxiety and hyperventilation. Some divers have automatically adapted their respirations along these lines. In a near-panic

Table 45.1 *Contributors to panic*

Personal factors	Equipment problems	Environmental hazards
Fatigue	Buoyancy	Tidal currents
Physical unfitness/disabled	Snorkel	Entry/exit techniques
Previous medical disorders	Facemask	Cold
Seasickness and/or vomiting	Weight belt	Surf
Alcohol or drugs	Wetsuit	Kelp
Inexperience	Scuba cylinder	Caves, wrecks
Inadequate dive plan	Regulator	Ice and cold water
Techniques (buddy breathing)	Other equipment	Deep diving
Psychological characteristics	Reliance on equipment	Dangerous marine animals
Neuroticism and anxiety	Loss of equipment	Poor visibility
Sensory deprivation	Misuse of equipment	Explosives
Vertigo and/or disorientation	Entrapment – lines	Boat accidents
Diving accidents		

> *"A contented man is one who know his limitations".*
>
> *An old man in a pub*

situation, all should adopt this approach by reducing activity and controlling respiration.

'Know your limitations and dive within them' is a venerable admonition given to new divers. Every diver has certain limitations, and apprehension is felt when he perceives that these limitations are being exceeded. It is then that the first seeds of panic are sown.

FATIGUE

Fatigue is a common contributor to diving deaths (28 per cent) and accidents, either due to personal, equipment or environmental problems which impose excessive demands on physical effort and result in exhaustion.

Personal

Adequate physical fitness is essential in diving activities which, invariably, will sooner or later impose considerable physical demands. Fitness to undertake aquatic activities can be evaluated both by reference to past performance and present capabilities (swim speeds, breath-holding ability, etc.). Age is associated with a reduction in physical fitness. In some situations, even unfit divers are not at risk. In others, even very vigorous, fit divers will be sorely tried.

Personality factors are also important. Given the same physical fitness and exercise load, the extrovert diver has more ability to ignore the environmental and physical demands, whereas an introvert is likely to be aware of the development of fatigue earlier. A neurotic diver, or one with high trait anxiety, will be more susceptible to fatigue at an earlier time than the more stoic diver.

Many other factors may influence the diver's fitness, such as the use of alcohol or drugs, development of seasickness, medical disorders, diving accidents such as decompression sickness, vertigo or disorientation, saltwater aspiration or hypothermia.

Equipment

The scuba cylinder, buoyancy compensator and other equipment causes excessive drag, with normal swimming. Regulators limit the respiration, protective suits limit movement, and greater swimming effort is needed to overcome negative buoyancy. Even experienced divers without assistance, when supporting a 5-kg negative buoyancy (weights held

above the water), can remain on the surface for less than 10 minutes, before submerging (see Chapter 4).

Environment

Most tidal currents in excess of 1 knot, are beyond the capability of many divers for more than a few minutes. Cold exposure and hypothermia will aggravate fatigue (see Chapter 5).

ACKNOWLEDGEMENTS

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Why divers die: the facts and figures

CARL EDMONDS

BACKGROUND

The causes of diving deaths depend on the populations being investigated, the equipment used and the environments. Thus, oxygen rebreathing navy divers will have very different causes of death than air-breathing, surface-supplied shell divers or helium-breathing, deep oil rig divers.

The death rate of recreational divers, independently assessed by Monaghan as 16.7 per 100 000, was supported by Harpur's observations of 0.003 per cent deaths per dive in Tobermoray (assuming five to six dives per year this equates with 15–18 deaths per 100 000 divers). If one includes in the population all people who have been exposed to even one dive, the figure drops to between 5.8 and 6.5 per 100 000 divers. The diving industry has a history of inflating the alleged population at risk, so as to reduce the overall death rate to below 4. Using the population statistics described by Monaghan, there is every reason to believe that the death rate per dive has risen over recent years.

The USA Underwater Diving Fatalities Statistics were originally compiled almost single-handedly by John J. McAniff, Director of National Underwater Accident Data Centre (NUADC), University of Rhode Island. These records are of recreational divers and include US residents world wide and foreign nationals who die in US waters. They recorded 2853 fatalities over 26 years (1970–1997), i.e. 106 per year (range 67–114), these comprising 89 recreational divers per year. Over the past decade these data have been collected by DAN (Divers Alert Network) at Durham, North Carolina, USA.

> *Most accidents involve multiple factors that are mutually interacting.*
>
> *Mark Bradley, on Diving Fatalities*

In an Australia and New Zealand (ANZ) series, the deaths were less numerous, but with much more documentation and comprehensively catalogued. All factors were recorded which were likely to have contributed materially to the sequence of events which led to death, or which prevented action being taken to allow a successful rescue.

The Japanese statistics supplied by Mano and his colleagues are less detailed, but with almost 100 000 newly qualified divers per year they should soon be comparable with the USA numbers. The most recent information, from Ikeda and Ashida (2000) suggests a mortality rate of 17.5 per 100 000 – about 50 times more dangerous than driving a car.

The British Sub-aqua Association (BSAC) collect accident and death data in the UK. This is available on the Internet at http://www.bsac.com. The case reports are informative and the conclusions interesting, but there is insufficient detail to determine the causative factors. The BSAC, probably one of the more reputable diver training organizations, with reasonably accurate records, suggests a death rate amongst members reducing from over 20 per 100 000 in the year 2000.

The independent assessments of safety in recreational diving, from the USA, Australia and Japan all indicate that it is not a safe activity – despite the diving industry, who have described it as safer than driving a car, swimming or lawn bowls! The purpose of this chapter, and this text, is to make factual data available and make diving safer – not to support propaganda.

The NUADC detailed the diving activity, whereas the ANZ series defined the conditions which contributed to the deaths. The populations were very similar, probably because of similar socioeconomic conditions and affiliated diving instructor organizations. The figures were therefore complementary, and can be compared with the annual diving fatality surveys of DAN. The DAN statistics are sometimes not easy to interpret as the reporting and terminology has varied over the last few years; however, the details of the individual cases is greater than in the NUADC reports and is more instructive. The latest available reports (1998–1999) are supplied here for comparison. Unfortunately, only about half were subjected to an autopsy and an investigative report and a past medical history was rarely available.

Thus the NUADC has the advantage of large numbers, the ANZ of great detail, and the DAN is intermediate between them. Because the ANZ

series contains the most information on each diving fatality, the percentages given in the text refer to this series unless specified. For comparison, the DAN figures (as per cent) are given in squared parentheses.

This chapter mainly considers recreational scuba divers, and the statistics have been restricted to the 1980s and 1990s, as the instruction and equipment of earlier years would distort the lessons for today.

DIVING DATA

This gives an overview of the diver, the type of diving, the behaviour of the diver and observers.

Diver profile

	NUADC (1970+)	ANZ	DAN (1998)
Average age (years)	33.1	32.9	41*
First scuba dive (%)	5.4	8	6*
Under training (%)	9.0	5	15%
Multiple deaths (%)	9.8	4	–
Diving alone (%)	17.5	21	–
Male/female ratio	9:1	9:1	5:1
Age > 50 years (%)	9.7	8	23

*Value is an approximation

In most cases the accident came as a great surprise to all associates of the deceased, but in 9 per cent of cases the victim had been specifically advised by a diving medical specialist, and sometimes by a dive instructor, that they were unfit for scuba diving.

Activity

Two-thirds [69 per cent] of these divers were either recreational diving or amateur fishing (mainly for shellfish), 7 per cent were photographers, 5 per cent were cave diving, 3 per cent were wreck diving, 2 per cent were instructing, 2 per cent were night diving, and 1.5 per cent were ice diving (NUADC). In 14 per cent of cases the fatal dive was a repetitive one.

In the DAN surveys, 6–15 per cent were engaged in technical diving, but most of these were inadequately trained. In the preliminary report for the 2000 survey, 13 per cent were technical divers. In the future, DAN will allocate a separate category for mixed-gas technical divers.

Age

During the 1980s, there was an increase in NUADC fatalities of individuals over 50 years of age. The trend has continued, reaching 18 per cent of the fatalities during 1986 and 1987. It is not possible to determine whether this reflects an increased risk with age and/or more aged divers. The DAN data also show an increasing average age over the last decade, the age range being 12 to 71 years.

The age range in the ANZ series was 13 to 65 years, with majority between 21 and 35, and a definite increase around 46–50 years. The latter was related to the 'cardiac deaths' which had their peak in this age group. This bimodal distribution has subsequently shown up in other surveys.

Depths

The depths of the dive, the initiating problem and the unconsciousness (or death) are related. A small number in the ANZ and DAN surveys never descended at all, and over one-fourth first encountered their trouble on the surface (usually at the end of the dive). Despite the wide range of diving depths, at least half either died or lost consciousness on the surface.

In the Japanese series, two thirds of those who died were diving at less than 10 metres, and one-fourth never left the surface.

Duration

In 17 per cent of cases, the diver succumbed in the first 10 minutes of the dive. In 56 per cent the problem developed following an exhaustion of the air supply (either on reserve, low-on-air (LOA), or out-of-air OOA). In 8 per cent of cases it was intermediate between these times.

In the DAN data, the problem developed late in the dive, or after it had been terminated, in over 50 per cent of cases.

It would seem reasonable to conclude that, in planning a dive, accidents could be anticipated more often at the start or at the end.

Experience

In the NUADC series, 5.4 per cent died during their first dive ever with scuba, 3.9 per cent died in their first open-water dive, and 21.4 per cent in an early open-water dive. Almost one-third of the cases were early entrants into the sport:

- 8.7 per cent were under instruction at the time of the death.
- 33.3 per cent were considered to have 'some experience.
- 36 per cent were considered to be very experienced.

Experience

	NUADC	DAN	ANZ	Japan
First dive (%)	5.4	5-6	8	–
First open-water dive (%)	3.9	–	–	30
Under instruction (%)	8.7	14	5	–

The Japanese statistics show that nearly half of their deaths occur in novice divers (less than one years' experience) and 30 per cent happened during the first open-water dive.

Experience is relative, however. In the ANZ series, although 8 per cent died in their first dive and 5 per cent were under instruction, 49 per cent had enough experience to undertake the dive that killed them.

In the DAN surveys, the fatalities occurred frequently in the most experienced group (> 60 dives); 5.6 per cent were instructors; rescue divers and dive masters accounted for 1.8 per cent each. Nevertheless, when considering the degree of diving exposure, DAN surveys indicate that inexperienced divers are over-represented in the fatality database.

Responses

Once a problem had developed, even though the surface was sought in most cases, the weight belt was ditched by the victim in only 9 per cent of cases, and the buoyancy compensator (BC) was not inflated, either there or at depth in 48 per cent of cases.

The rescuer assisted:

- with the air supply (11 per cent)
- by ditching the victim's weights (12 per cent)
- by inflating the victim's BC (10 per cent)
- and assisting in rescue and first aid (23 per cent).

When the buddy remained with the victim, or eventually found him, there was usually an appropriate response. Rarely (1 per cent) did the rescuer become a victim.

Buddy breathing seemed to cause problems, especially during ascent. The NUADC had studied 24 cases of failed buddy breathing from a single regulator in 1946–1970, and stressed the following findings:

- Over half were attempted deeper than 20 metres.
- In no instance did the assisting buddy die and the victim survive (this should encourage would-be rescuers to share their air).
- In 29 per cent of cases the victim or buddy's facemask was displaced, considerably complicating an already difficult operation.
- In 12.5 per cent air embolism supervened. It is easy to imagine overinflation of the lungs occurring during the situation involving considerable victim anxiety, regulator sharing, purging and ascent.
- In 12.5 per cent the victim refused to return the mouthpiece and/or fought for it.

These figures supported the argument for an alternative or independent source of air for the victim.

OVERVIEW OF CONTRIBUTING FACTORS

The factors contributing to death were more comprehensively investigated in the ANZ survey. The number of factors increased with the detail available for the dive. A 'sole cause', such as a shark attack or an inexplicable burst lung, was a rarity, except in the divers who dived alone, when the records were probably incomplete. Each victim was recorded only once in each major category (medical disorders, equipment faults and environmental).

Major categories

	NUADC*	ANZ series
Medical disorders (%)	55.7	74
Equipment faults (%)	9.5	35
Environmental (%)	34.8	62

* The NUADC series, which had less information available on each death, did not use identical classifications, and only recorded one contributing factor – and then in only 73 per cent of cases. The ANZ series had much more detail and thus more identifiable contributing factors.

As well as these major categories, problems with certain diving techniques or activities were likely to have contributed to the final event. These are:

- Inadequate air supply, 56 per cent
- Buoyancy problems, 52 per cent
- Other equipment misuse, 35 per cent

MEDICAL CONTRIBUTIONS

Medical contributions to diving deaths include: psychological conditions (e.g. panic, fatigue); physiological conditions (e.g. vomiting, extreme physical unfitness); and pathological conditions (e.g. pulmonary barotrauma, cardiac disease). Unless specified otherwise, all figures given for the ANZ series in the following analyses refer to a percentage of the total fatalities.

Autopsy findings

Even though an understanding of the events is not obtainable by autopsy findings alone, they are indicative of the final event.

Cause of death

	ANZ	NUADC	DAN
Drowning (%)	86	74.2	52
Pulmonary barotrauma (%)	13	24.5	19
Cardiac (%)	12	9.1	27
Aspiration of vomitus (%)	6	<1	1
Trauma (%)	3	1.5	1
Asthma (%)	2	–	–
Marine animal injury (%)	1	–	–
Coincidental (%)	1	–	–
Decompression sickness (%)	–	–	1

ANZ survey

In assessing medical problems that contributed to the death, drowning was excluded as it represented only the inhospitable 'final act' after losing consciousness underwater. Saltwater aspiration, while the diver was still conscious, was in most cases overtaken and pathologically obscured by its logical extension, drowning (see Chapter 21).

The 'pre-existing' figures in Table 46.1 must be considered as underestimates of the true situation, as in only 49 per cent of cases was the past medical history able to be obtained.

Table 46.1 *Medical contributions to diving deaths (excluding drowning)*

	Pre-existing (%)	Fatal dive (%)
Panic	—	39
Fatigue	—	28
Vomiting	1	10
Nitrogen narcosis	—	9
Drugs	8*	7
Very physically unfit	4	4
Severe disability	3	3
Severe visual loss	3	3
Alcohol	—	2
Motion sickness	2	2
Gross obesity	8*	2
Carotid sinus reflex	—	1
Saltwater aspiration	—	37
Pulmonary barotrauma	—	13
Cardiac disease	3	12
Asthma	9	8
Respiratory disease	5	7
Hypothermia	—	3
Hypertension	8*	2
Ear problems	2	2
Diabetes	1	1
Others	—	1
Epilepsy	1	—
Decompression sickness	—	0
Contaminated air supply	—	0

*Not considered as contributing factors, but included because other related disorders coexisted. Otherwise, only the pre-existing disorders which were thought to infuence the death of the victim were included in the table. In 25% of the cases there was a pre-existing medical disqualification for scuba diving. This compares to an overall 'failure rate' of almost 10% during the 1980s and 1990s, among applicants for scuba diving, who were examined by specialists in diving medicine.

In this geographical area, patients who had insulin-dependent diabetes, epilepsy, cardiac disease or surgery and current asthmatics were not considered suitable for diving – both in the obligatory medical examination and in the signed declarations required by diving instructor organizations.

Stress responses, panic and fatigue

These subjective symptoms are 'soft' data that can only be presumed by a detailed description of the diving activities (see Chapter 45). Nevertheless, they occur frequently throughout the fatality case reports. To dismiss them because of the inability to demonstrate morbid pathology, would be to ignore two of the major purported contributory causes of diving deaths.

Shilling, in *The Physicians Guide to Diving Medicine* reported that "overexertion, fatigue, exhaustion, respiratory embarrassment, panic and resultant accident is the repeated sequence of events leading to a fatality". Likewise, Webster believed that physical exhaustion was the cause of death in over half the diving fatalities of earlier years. The stress syndromes can be based on psychological and physical unfitness, drug intake, medical problems, inadequate training and inexperience.

Panic (39 per cent)

Panic is an extension of the psychological stress reaction of anxiety, and the threat of death is a reasonable cause of anxiety. Under selected circumstances, anyone will panic. Difficulty in obtaining air is a very frequent cause, and the inhalation of water was associated with panic in 19 per cent of the deaths.

Panic occurred when unusual circumstances were present, such as greater than customary depth, compromised air supply, buoyancy problems, being left alone, poor visibility, strong water movement, unpleasant surface conditions (a long swim to the boat) or any equipment malfunction or perceived malfunction such as overbreathing a snorkel or regulator. The consequences include rapid ascents and inappropriate actions, such as abandoning a regulator, snorkel or mask, and a failure to respond appropriately, for example by ditching weights or inflating BCs. The high incidence of novice divers' deaths from panic in the Japanese studies were especially due to trying to cope with surface conditions and difficulty with middle-ear equalization during shallow dives.

Fatigue (28 per cent)

Fatigue is a physiological stress reaction to a muscular effort which was often underestimated by the victims. With sufficient physical demand anyone can become fatigued. Saltwater aspiration, panic and cardiac disease all occurred more frequently than would be expected in these cases.

Fatigue often developed when cold, being overweighted, or during an attempt to swim against a current. The latter is especially noticed when the victim swims against the equipment drag, if overweighted and with an inflated BC. It is more likely, but certainly not restricted to those who are physically unfit or disabled. Panic, water aspiration, cardiac disorder and asthma may be precipitated.

Vomiting (10 per cent)

After exclusion of those cases in which vomiting happened following removal of the victim from the water or as a terminal event, it either initiated or complicated the accident in 10 per cent of the cases. The more common causes included seasickness and saltwater aspiration. Both were associated with adverse sea conditions, and the aspiration with surface swimming, snorkeling and problems with the regulator 'leaking'. Others include ear barotraumas, gastrointestinal barotrauma, alcohol and food overindulgence.

Nitrogen narcosis (9 per cent)

Although this contributed to the death, it was never the sole or major cause (see Chapter 15). It was always produced by depths greater than 30 metres (100 feet), and aggravated by poor visibility, as in caves. It resulted in inappropriate behaviour such as rapid ascents or panic. It was also probably causal in interfering with buoyancy control, buddy breathing and rescue attempts.

Drugs (7 per cent)

Evidence of breathing gas contamination and drug intake was sought in most cases, but these were rarely contributors to the death. Alcohol, narcotics, cannabis and more recently, cocaine, were occasional contributors, but presumably most divers are aware of the dangers of combining these with diving. The relationship between alcohol and drowning is described elsewhere (see Chapter 21).

In only 7 per cent of deaths was it considered likely that drugs were a contributory factor. Prescribed drugs included drugs for asthma (in 9 per cent of deaths) and cardioactive drugs, such as beta-blockers (in 5 per cent). These diseases and their therapies are a cause of concern; indeed, deaths amongst these drug takers could be due either to the effects of the drug or the underlying disease.

In the latest DAN Report on Diving Fatalities (edition 2000), in the cases in which information was available:

- 25 per cent were taking cardiovascular drugs;
- 14 per cent were on decongestants;
- 12 per cent were on antihistamines;
- 11 per cent were on insulin;
- 7 per cent were on asthma medications; and
- 5 per cent were on antibiotics.

Analgesics, anti-seasick and anti-allergy drugs, and contraceptive drugs were each used in 4 per cent of cases.

Saltwater aspiration (37 per cent)

Saltwater aspiration (see Chapter 24) while still conscious was evident in a large number of cases, when full documentation was available. Others were demonstrated when tests were carried out on the equipment. This was usually an interim factor, following some other event such as using a snorkel in white water or an OOA situation – and leading to panic, fatigue, cardiac disease, asthma and drowning.

Problems with the regulator unexpectedly caused aspiration in 12 per cent, and buddy breathing was an occasional cause.

Pulmonary barotrauma (13 per cent) [19 per cent]

This was the major single pathologically demonstrable cause of death (excluding drowning) in younger and less experienced divers (see Chapter 6). In some cases the extensive pulmonary damage was obvious, but in others it was complicated by the effects of subsequent drowning.

The suddenness of these cases made other observations more difficult, though some associations were noted. These included narcosis, panic and lung diseases; other factors included excessive depth exposure, vomiting underwater, regulator failure and excessive buoyancy which resulted in rapid or emergency ascents.

Cardiac disease (12 per cent +) [16–27 per cent]

In accepting this diagnosis, very gross pathology or an excellent clinical description was required (see Chapter 39). If all autopsy and clinical diagnoses of cardiac disease were accepted, the incidence would have risen to 21 per cent in the ANZ survey. The higher DAN figure could be related to the greater age in the US divers.

Most of the divers who died of cardiac disease, were males aged 40–60 years. These divers tended to die quietly in the water, usually soon after entry or at the end of an otherwise uneventful dive, except possibly for exertion or aspiration of water. Most had a history of heart disease or hypertension requiring treatment.

With so many possible trigger factors (previous pathology, exertion, cold exposure, prescription drugs including beta-blockers, hypoxia from aspiration of sea water, etc.) for both myocardial ischaemia and arrhythmias, it would be difficult to incriminate one specific aggravating factor. Nonetheless, the fact that a diver is in the water at the time of a cardiac incident, makes survival less likely.

Asthma (8 per cent)

Although only 1–2 per cent of divers are current asthmatics (probably less where medical examinations

or questionnaires are required before diving; (see Chapter 55)), at least 9 per cent of the deaths were in asthmatics and in at least 8 per cent it was a contributing factor, i.e. in eight out of the 49 in whom the medical history was available.

Asthmatics, even more than others, had multiple contributions to death. The relative frequency of a compromised air supply, saltwater aspiration, panic and fatigue before drowning was evident. The cause of death was drowning in seven cases, and pulmonary barotrauma in two. Saltwater aspiration and the stress responses were present in five cases each, and six had a compromised air supply (LOA, OOA).

Respiratory disease (7 per cent)

Some divers had a respiratory disease other than asthma, and these included acute and chronic respiratory infections, pleural adhesions, cystic disease and other intrapulmonary pathology, contributing to the death.

DIVING TECHNIQUES

Certain diving procedures or techniques that involve human judgements were perceived as having an influence on diving deaths. These include; OOA situations, buddy diving, and buoyancy problems. These paralleled a subsequent survey on drowning deaths with scuba (see Chapter 25).

Air supply (56 per cent)

As 56 per cent of the problems developed after the air supply had reached reserve levels, LOA and OOA, it was concluded that the divers found it more difficult to handle problems under those conditions. This tallied with the observations on the number of 'surface' deaths, and the problems of coping with surface swimming conditions.

Most problems develop from the time the victim became aware that the air supply was compromised. Snorkeling on the surface was employed by divers attempting to conserve an air supply, and coincided with the development of problems in 8 per cent of cases.

A LOA situation was produced by using either a smaller cylinder than normal or by diving with less than the customary air pressure in 9 per cent of cases. In the case of small cylinders, not only is there less air supply than that available to the other divers, but when the LOA situation develops the amount of reserve air is much less than usual. In some of the cylinders, holding only 800 litres (28 ft^3), there are only a few breaths of air once the LOA situation is reached at depth.

Buddy diving

The buddy (companion) diving system, which has universal support among recreational diving groups and instructors, appeared to have more verbal than practical application. Many who claimed to be buddies were divers who only shared the same boat. In the ANZ series, 21 per cent dived solo from the start, 13 per cent separated voluntarily before any problem developed, 25 per cent separated voluntarily after a problem commenced, 20 per cent were separated by the problem, and only 14 per cent were not separated at all, i.e. correctly practiced buddy diving.

By far the most common reason for voluntary separation was that one diver (the subsequent victim) was OOA or LOA, with the buddy deciding to continue the dive alone. Occasionally, the buddy accompanied the victim to the surface and then left him to return alone. The problems that sometimes separated the buddies were; uncontrolled ascents, underwater and surface currents – sometimes sudden and unexpected.

Among the small numbers that were true buddy divers, there were some practices which seemed to detract from the buddy concept. In 15 per cent of cases there was not one buddy, but two or more. This led to confusion as to who exactly was responsible for whom. In 6 per cent of cases the victim was following the 'buddy'. Under this situation, any observation by the lead diver would have been fortuitous. To attract the lead diver's attention required energy, air and time-consuming behaviour on the part of the victim, who could rarely afford it. The experienced diver was invariably the one who took the lead, and

therefore had the luxury of a buddy observing him at all times.

In some cases there were groups of people being led on a dive. The procedure used was that the first diver to exhaust his air supply would inform the dive leader that he was 'on or near reserve'. The dive leader would then take time to determine who else was in or close to a LOA situation. These two divers were then buddied, to surface and return to base. Thus, the dive leader managed to select the two heaviest air consumers, and usually the two least experienced divers, and buddied them together into a situation in which either one was likely to develop a complete OOA situation during ascent, while performing a safety stop, or on the surface. This illogicality seemed to be an accepted practice in some 'resort' areas.

> *Over one-third of the victims were either diving alone or separated voluntarily before the problem developed. One-fourth separated voluntarily afterwards!*

In the NUADC series the dive buddy's activity was less verifiable than in the ANZ survey, but still only 25 per cent of the buddies claimed to stay with the victim. In the DAN database, separation from the buddy occurred in two-thirds of the fatalities.

Buoyancy (52 per cent)

Many of these problems also came under the 'equipment faults' category, but an appreciable number were clearly errors of judgement and were therefore included as difficulties in diving technique. Buoyancy problems contributed in 52 per cent of the deaths (47 per cent from negative buoyancy and 8 per cent from positive buoyancy).

The wet suits available for most of these decades required a weight compensation as follows: 1 kg weight for each 1 mm thickness, 1 kg extra for 'Long John' extensions and a hood, 1 kg for aluminium tanks, and 1–2 kg for individual variation in buoyancy.

In excess of this, the diver was overweighted and required extra effort, hyperventilation or reliance on the BC, to remain buoyant on the surface. Using this criteria it was found that 40 per cent of the divers who died were **overweighted** on the surface. At depth the problem of overweighting was compounded by the loss of buoyancy from the wet suit.

Apparently many divers have replaced the skills of buoyancy control with reliance on the BC. They are purposely overweighting 'to get down', and the BC is inflated to return to the surface. In these cases the BC is relied on not to trim buoyancy with depth, but to return the diver to the surface. Such a procedure introduces the potential for equipment failure and accidents.

The BC problems included:

- accidental inflation;
- confusion with use – some victims confused inflation with dump valves;
- overinflation during ascent – Boyle's law and the Polaris missile effect;
- inadequate and very slow inflation at depth – especially if LOA; and
- effort required to overcome drag when swimming underwater and on the surface.

The American Academy of Underwater Sciences, in a symposium in 1989, pointed out that of the cases requiring recompression therapy, half were related to loss of buoyancy control.

Weights

As in previous surveys it was found that very few of the victims – only 9 per cent – successfully ditched their own weight belts. In 40 per cent of cases this omission probably contributed to the victim's death. Failure to ditch the weights, when in difficulty, presumably reflects on training techniques.

EQUIPMENT CONTRIBUTIONS

(see Chapter 4)

The problems with air supply have been described above. The failure to ditch weight belts and many of the problems with buoyancy that were clearly errors of judgement, have been recorded above as technique or procedural faults.

In the ANZ survey on diving deaths, equipment faults contributed to 35 per cent of the deaths and equipment misuse to 35 per cent. There was an overlap between these. Equipment faults were defined as problems that could not have been reasonably attributed to action of the diver during that dive. It is not considered an equipment fault if the diver has injudiciously chosen the wrong equipment. This would be included under the 'misuse' category.

Faults (35 per cent); **misuse** (35 per cent)

These can be attributed as follows:

- Regulator 15 per cent
- Fins 13 per cent
- Buoyancy compensator 12 per cent
- Scuba cylinder 9 per cent
- Weight belt 6 per cent
- Harness 6 per cent
- Mask 5 per cent
- Protective suit 5 per cent
- Lines 4 per cent
- Gauges 2 per cent
- Snorkel ?

Regulator (15 per cent)

In 14 per cent of deaths there was a fault in the regulator, and 1 per cent it was misused. The laboratory testing of the regulators after the incident showed many minor discrepancies. The ones which contributed to the death were verified by either the case history or the in-water test in which the accident conditions were simulated. The contributing faults were:

- Saltwater inhalation 8 per cent
- Increase breathing resistance 4 per cent
- Catastrophic failure 2 per cent

Fins (13 per cent)

Flipper or fin loss were surprisingly frequent, and difficult to interpret. In 3 per cent of deaths there was definite misuse, in that the fins were either not worn or too loose. In another 10 per cent, one or both fins were unaccountably lost. In all these cases, other difficulties were present. The loss of a fin interferes with propulsion, and is as likely to be a result of panic or excessive leg movement, as it is a contributory factor to panic, fatigue or negative buoyancy.

Buoyancy compensator (12 per cent)

In 8 per cent of deaths there was a malfunction of the BC, and in 6 per cent there was a misuse of the BC. The main faults included failure to achieve buoyancy because of inflation mechanism malfunction, or failure to retain the air.

The misuse involved such actions as over-inflation, producing the 'Polaris missile' type ascent, or mistaking inflation mechanisms with dump valves. Sometimes the high-pressure hoses were not correctly connected, and in other cases there was difficulty with attempted ditching procedures.

Scuba cylinder (9 per cent)

In these cases there was rarely a fault in the equipment, but it was either inappropriately chosen or was misused in some other way. The causes were either: an initial low air fill in 3 per cent; a cylinder too small in 3 per cent; the cylinder valve not turned on in 2 per cent; and loss of the cylinder in 1 per cent.

Weight belt (6 per cent)

In 3 per cent of cases the weight belt was fouled, such as by being worn under other equipment and harness. In another 3 per cent it was unable to be released because of: entanglement with lines; the weights slipping onto the quick-release buckle; and the strap being too long and jamming the release on the belt.

Harness (6 per cent)

In 4 per cent of cases there was a fault in design or performance. Sometimes it was difficult to undo the harness, even for the would-be rescuers. Occasionally the tank fell through the harness. In 3 per cent

there was misuse in that the harness was placed over the weight belt.

Mask (5 per cent)

Broken straps, displacement and failure to achieve a watertight seal were the main problems associated with the facemask.

Protective suit (5 per cent)

In 4 per cent of cases the suit was considered to be so tight as to cause difficulties to the diver, including dyspnoea, panic, claustrophobia and fatigue. Tightness around the neck produced the carotid sinus syndrome.

Lines (4 per cent)

These resulted in entanglements, when misused.

Gauges (2 per cent)

Problems included not only incorrect information (air supply, depths), but explosive blow-off and snagging.

Snorkel

It was impossible to determine the number of problems with snorkels as these were not well-documented in scuba deaths, despite observations that deceased divers were having trouble snorkeling on the surface. Some were in difficulty because they either did not have a snorkel or were not skilled in its use. Others seemed to have difficulty with achieving sufficient air while swimming against a current or when overweighted on the surface.

ABSENCE OF EQUIPMENT

In many instances, not usually considered here as equipment misuse or failure, there were situations in which equipment should have been available and had it been so, may have resulted in a less serious outcome.

ENVIRONMENTAL CONTRIBUTIONS

(See Chapter 5)

These include both the natural hazards (e.g. tidal currents, sharks) as well as non-scuba-related man-made hazards (e.g. boats, dam outlets). Environmental problems contributed to 62 per cent of the diving deaths.

If the diver attempted to dive under conditions for which he was clearly untrained and inexperienced, then this is seen as an error of judgement. In 47 per cent there was either no experience of the type of diving environment being encountered, or inadequate experience to cope with that environment. In less than 50 per cent of cases was there considered to be sufficient training or experience for the planned dive.

General

About 66 per cent of sport diving fatalities in the USA occur in saltwater. As regards freshwater diving, diving in caves appears to be the dominant cause, although diving in dams, sinkholes and at altitude also incur specific hazards. Most divers are trained to dive in the more dense seawater, and problems with buoyancy are appreciably different in freshwater.

Such situations as freshwater diving, altitude exposure, etc. are not described here, as they themselves do not cause the death other than by influencing medical or equipment problems. In Australia and the tropics, most diving is in the ocean and freshwater hazards are less.

Environmental factors (62 per cent) contributing to diving deaths include:

- Excessive water movement 36 per cent
- Depth 12 per cent
- Poor visibility 6 per cent
- Cold 5 per cent
- Marine animals 5 per cent
- Caves 5 per cent

- Exit/entry problems 5 per cent
- Entanglement 4 per cent
- Boats 3 per cent
- Diving under a ledge or boat 3 per cent
- Night diving 2 per cent

Excessive water movements (36 per cent)

This is by far the major environmental problem contributing to diving deaths. In 15 per cent of cases the **tidal current** was too great for the diver to negotiate, while in 15 per cent there was rough surface conditions, often involving 'white water' – surf and surging water around rocks. One effect of white water is to reduce the diver's buoyancy and make surface swimming more strenuous. It also indicates turbulence and fast water movements, interfering with communication and visibility. In 3 per cent there was an **unexpected and sudden underwater surge** which put the divers into the difficulty. In 2 per cent it was a normal **to and fro surge** which caused the problem.

Because so many of these situations involved surface swimming, the effects of an exhausted air supply, overweighting, and excessive 'drag' of equipment, led to panic and fatigue, the sudden death syndrome, saltwater aspiration and aggravation of asthma.

Death in 2 per cent of cases was caused by being trapped and drawn into a **pressure outlet** (a valve) in a freshwater dam. It was often not appreciated that the outlets in dams, although not deep, produce a considerable pressure difference, and in both cases the diver was drawn into an outlet pipe. Although the flow of water was not great in either case, the pressure gradients were, once the body had obstructed the orifice. Similar pressure effects can occur in caves and near rocks if there are springs or currents.

Depth (12 per cent)

Sometimes the depth itself was excessive for that person. In 4 per cent it was considered a major cause, and in 8 per cent a probable one. In most of these it was the greatest depth to which that diver had dived.

Excessive depth produced the problems of more rapid air consumption (thus an inadequate air supply, OOA or LOA, at depth or during ascent), nitrogen narcosis, negative buoyancy, slower BC inflation and more reliance on BC integrity, less visibility, more difficult free ascents, panic, etc.

Caves (6 per cent)

These include caves in the ocean (especially along drop off and coral reefs) and in freshwater. Multiple fatalities are more common in cave diving. The specific problems included nitrogen narcosis, entanglement in lines, panic, loss of visibility due to the absence of light or silt (making the torches ineffectual), navigational difficulties, sudden catastrophic water surges, or equipment failure.

Poor visibility (6 per cent)

This was especially encountered in night, cave, white water and deep diving. It greatly increased the dangers of panic, disorientation and nitrogen narcosis.

Marine animal injury (5–8 per cent)

There is a tendency to belittle the hazards from marine animals, and with many such injuries the pathologist may not observe the lesion (e.g. from a cone shell, sea snake or blue-ringed octopus bite). In some cases the divers initiated the problem, but in an appreciable number the animal instigated the proceedings. Included among these are fatal shark bites, other bites from eels, octopus, cuttlefish, stings from fish, coelenterates, urchins, etc. The injury sometimes initiated the fatal sequence of events.

Entry and exit problems (5 per cent)

These were encountered during rock platform and boat diving, surf and night diving, ice, wreck and cave diving. It was usually complicated by excessive water movement (see above).

Cold environment (5 per cent)

The cold environment was considered to be a contributing factor in 5 per cent of deaths, because of

inadequate protection or excessive exposure. The effects included hypothermia, reduced dexterity and strength, fatigue and the sudden death syndrome. In other series the influence on equipment malfunction and decompression sickness have been noted.

Entanglement (4 per cent)

Entanglement from environmental hazards occurred in only 4 per cent of cases. In 3 per cent it involved lines used by divers, and in 1 per cent it involved kelp. Not included in this figure is the entanglement in the harnesses or diving equipment.

The specific difficulties with line entanglement were aggravated by poor visibility, incorrect choice of lines and allowing them to become slack. Other causes of entanglement, in other surveys, include fishing line, nets, anchor cables, underwater wrecks, and land falls in caves.

Trapped under ledge or boat (3 per cent)

This led to difficulty when the diver attempted to ascend to safety. In an emergency, there may be inadequate time or composure to swim back down to avoid an obstacle between the diver and the surface.

Boats (2–3 per cent)

Problems with tidal currents, causing the diver difficulty with finding and reaching the boat, often contributed to the deaths from fatigue, panic, cardiac disease, over-weighting, etc. These were not included in the 2–3 per cent of cases in which the boat was instrumental in causing physical damage to the diver.

Night diving (2 per cent)

This figure may be an underestimate because unless the night conditions very obviously contributed to the death, they were not recorded. The problems included vision loss, navigational difficulty, entanglement, panic, etc.

SUMMARY, CONCLUSIONS AND RECOMMENDATIONS

The purported low death rates among recreational divers during the early 1980s, were shown to be based on overly optimistic figures and creative statistical interpretations. So also was the alleged improvements in safety among scuba divers. The death rate of 15–18 per 100 000 now demonstrated has compelled the diving industry to review and appreciably modify its claims of safety.

The NUADC has conscientiously recorded the diving deaths in North America over many years, and DAN is now committed to continuing and enlarging this valuable activity. The ANZ series, supplemented by a recent survey of scuba drownings (see Chapter 25), documented the known contributing adverse factors. It differentiates medical disorders, diving techniques, equipment faults and misuse, and environmental factors. The ANZ cases demonstrate that although diving may be safe under many circumstances, when a number of adverse factors combine the diver is often unable to cope with the complexities of his equipment and environment.

Medical contributions

The fact that 9 per cent of divers who died were specifically told by diving experts that they were unfit to dive, suggests that sometimes good advice goes unheeded. At least 25 per cent of divers who died were medically unfit to undertake scuba diving, and should not have been diving.

A large number of asthmatics and hypertensives undergoing treatment, as well as patients suffering from cardiac disease, diabetes and epilepsy, were represented in the ANZ series. Their presence was difficult to comprehend, considering that the candidates were required to pass specialized medical standards for diving, as well as complete a screening questionnaire issued by the diving instructor organizations, before they were accepted for training. Clearly, the physicians and the instructors were not adequately applying these standards.

The failure of physicians to apply the medical standards, is due to ignorance of these standards and a failure on the part of the physician to appreciate

the problems of scuba equipment and the demands of the ocean environment. The reasons for the instructors not applying the standards is still conjecture, and may well be based on commercial factors, more than ethical standards.

In either case, the current system has not fully succeeded in selecting out the high-risk patients. Physicians and dive instructors are still confusing physical fitness (needed for many sports, including diving) and medical fitness (a freedom from medical diseases incompatible with safe diving). Both are required. Many deceased divers were said to have been very fit physically, despite having such medical diseases.

If drowning is excluded as only the final event in a sequence of adverse happenings, then the stress problems of panic and fatigue dominate the medical contributions. Because these do not feature in autopsies, they are not fully appreciated in some series. They are interwoven with faults in technique (or training), and with many equipment and environmental provocations.

The importance of these stress factors is contrasted with the great rarity of the 'high-profile' diving diseases of decompression sickness and gas contamination, which were absent in the ANZ series and less than 1 per cent of the NUADC series.

The importance of other major contributors that leave little or no evidence at autopsy, such as saltwater aspiration, nitrogen narcosis, vomiting and asthma, can only be comprehended by a detailed dive history. These do not show up as much in the USA series, because of the limitations of the data collection.

Current diving medical examinations and diver training need to be modified to reflect the relative importance of these contributing factors.

All recent series show reasonable agreement on the importance of pulmonary barotrauma and cardiac disease. The latter seems to be an increasing problem, as the age of divers increase. The importance of astute medical selection and then adequate training of divers is dominant in the prevention of these.

A DAN survey showed that established divers are a relatively healthy group. Of 2633 divers, with an average age of 40 years, 5.8 per cent had hypertension, 1.2 per cent had asthma (0.9 per cent took bronchodilators) and 1.1 per cent had diabetes. This was similar to UK and Australian data.

Diving techniques

These are more related to diver training than diver selection. The inexperienced and overconfident male was over-represented in both the USA and ANZ series. Diving well within the limitations of the diver, and the equipment, was not a well-practiced activity among these divers.

The majority who die do so after voluntarily inducing a **compromised air situation** (using up most or all the air available). They are then forced to surface to breath, or to conserve their emergency air supply. Returning with sufficient air, was not common.

The traditional admonition that **the surface is the danger area** for divers, was supported by the figures showing that at least half the cases lost consciousness and died there. Nevertheless, the surface was unavoidable in 56 per cent, as the diver was in a compromised situation as regards his air supply.

Surface problems were frequently aggravated by the decision not to ditch **weights**. This also contributed to many of the cases that developed at depth, where a failure to appreciate buoyancy factors resulted in excessive exertion being required. The training technique of older experienced instructors to require trainees to practice removal and replacement of the weight belt on each dive, could well be re-instituted. This practice alone would have prevented the deaths in which the belt was eventually found to be unreleasable. Instruction to unbuckle the weight belt and hold it at arm's length in demanding situations, was either not taught or not applied in any of these cases. Yet, had this been done and the situation deteriorated, the belt would have been dropped successfully and the diver made positively buoyant – assisting slow ascent and permitting surface swimming without being overweighted. If the situation had not deteriorated, the diver could have replaced his belt without penalty.

The extreme effort in swimming on the surface with scuba gear, heavy weights and an inflated buoyancy compensator, seemed not to be widely appreciated among this diving population. The technique of overweighting, 'to get down', and the subsequent strong reliance on the buoyancy compensator to ascend and remain on the surface, presumably makes instruction far easier. Failure to learn the skills of buoyancy control, without an overinflation/

overweight trade-off, is an expensive lesson not to learn. Dependency on equipment may well be related to the failure to ditch it in an emergency.

Buddy diving, as envisaged in the manuals, is in the minority in these cases. The majority of divers dived alone, and died alone. Even in the less detailed NUADC reports, less than one-half dived as a buddy pair, and only one-fourth claimed to have stayed together. The DAN data showed that two-thirds of the buddies had separated, while the ANZ series showed that:

- over one-third of the victims were either diving alone or separated voluntarily before the problem developed;
- one-fourth voluntarily separated after a problem developed;
- one-fifth were separated by the problem; and
- only one-seventh genuinely remained as buddy divers.

It seems as if the buddy concept, if used at all, was mainly employed when it was not needed. More buddies voluntarily separated from the victim at the start of problems (usually when LOA) than actually stayed together. Even when it is applied, the less experienced diver, or the one who will consume more air, is initially given the task of following the more experienced diver until he runs out of air – and is then sent to the surface to swim back alone! Or he is buddied with another LOA diver.

Traditionally, buddy diving was recommended and the need was self-evident because of the recognition that diving was a potentially hazardous activity. As diving is sometimes promoted as being a safe sport, perhaps the need for buddy diving is less appreciated. For uneventful dives this attitude is adequate. For others it is not. The observations in both the NUADC and ANZ fatality series, should emphasize the need for buddy diving. It involves the divers genuinely taking responsibility for each other for the whole time, until they return to shore or safety. **It needs to be taught, understood and practiced.**

The need for an adequate **independent emergency air source** is evident. Buddy breathing from a single regulator involves too many problems, and even octopus breathing was inadequate in many circumstances.

Equipment problems

There is a popular misconception that the equipment nowadays is so technically advanced that it is reliable. This is not so. The regulators that caused our scuba pioneers so much concern, were still common contributors to diving deaths.

The new equipment, such as power-inflated buoyancy compensators, seemed to cause as many problems as they solved – although perhaps this is as much due to inadequate training and excessive dependence, as to faults in equipment design.

Over the past decade, the use of mixed gases and rebreathing sets in technical diving has increased the scope for equipment faults and misuse.

Environmental hazards

Even with improved training and reliable equipment (not that this author concedes either factor), the environmental hazards are unlikely to change. Divers tend to overestimate their own abilities and to underestimate the power, unpredictability and capriciousness of the sea. The more aware diver has great respect for the ocean and its inhabitants. Training in one diving environment does not automatically transpose to others. Some such hazards are dealt with in Chapter 5.

High standards for scuba diving are as important for safe diving now as they ever were. They include:

- *physical fitness;*
- *a freedom from many medical diseases;*
- *training in accident prevention and management;*
- *an appreciation of the limitations of equipment; and*
- *a healthy respect for a potentially hazardous environment.*

Overview

Perhaps one of the most telling assessments on diving fatalities was made by Professor David

Elliott, when reviewing a text on Australian Diving Deaths 1972–1993. In reviewing the many fatality reports, he compares it to a Shakespearean tragedy:

> 'As you continue to read, you know that inevitably it will happen all over again. Why this repetitive pattern? How can so many fail to learn from the ultimate examples provided by others?'
>
> 'This is a sombre story . . . which does not get better as one moves from one year to the next. Each time . . . one expects some safety lesson has been learnt, that a stupid omission or short cut has now been rectified . . . and that divers have begun to recognise their own technical and physiological limitations'.
>
> 'Any early optimism about the ability of individual divers to learn from the errors of others, soon disappears . . . By describing the circumstances of each . . . fatality, an opportunity has been created to use the deaths to enhance health and safety'.
>
> 'Individual case histories might be suitable to classroom teaching, although that approach is not likely to be welcomed by those who wish to emphasise the safety of scuba diving. Nevertheless it would be a positive step (for information on diving fatalities) to become part of the official curriculum of all would-be instructors'.

Professor Elliott re-quotes the old adage, 'He who does not know history is doomed to repeated it'.

PROFESSIONAL DIVING

Professional diving fatality statistics vary with the type of diving. Oil rig divers of the Gulf of Mexico had a death rate of 2.49 per thousand per year, whereas those of the deeper and more treacherous North Sea reached 4.82 per thousand for the same period. Despite the careful medical selection and rigorous training in these groups:

- 6–7 per cent had a medical factor which contributed to the death;
- 19–28 per cent died from DCS or CAGE;
- 15–17 per cent showed poor judgement or panic;
- cold was a factor in up to 11 per cent of deaths;
- heavy seas were a factor in up to 15 per cent; and
- equipment problems contributed in 33–41 per cent.

Other full-time professional diving groups (abalone divers, commercial divers) have similar death rates, of 3–6 per thousand per year.

Some navies employ rebreathing equipment, and then the common causes of death are carbon dioxide or oxygen toxicity, or hypoxia. The US Navy diving fatality investigations suggested that:

- Inexperience contributed in 25 per cent of cases; and
- panic was an important factor in 30 per cent.

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47

Unconsciousness

CARL EDMONDS

INTRODUCTION

Normal brain function depends on a steady supply of oxygen and glucose to the organ. Impairment of cerebral circulation and/or a fall in blood oxygen or glucose will rapidly cause unconsciousness. Cerebral function may also be altered by toxic effects of gases, drugs and metabolites. Unconsciousness in a diver, during or after a dive, can result from many causes and must be treated as an emergency. Some of the causes require prompt action beyond simple first aid.

Drowning is the most likely result of unconsciousness in the water. It may be the final common pathway of a number of interactive situations related to environment, equipment, technique or physiology. Many accidents in the water are fatal, not because of the cause, but because there is no one to rescue the unconscious victim. In the water, unconsciousness from whatever cause may result in loss of a respirable medium and aspiration of water. This secondary effect may be the fatal one and may obscure the original cause of the 'blackout'.

Attempts should always be made to elucidate the specific factors leading to the unconsciousness. Otherwise it is likely to recur, but with more tragic results, in a subsequent and similar dive exposure.

This chapter summarizes the various causes of unconsciousness and relates them to the type of diving equipment used. Repeated reference to Figure 47.1 is recommended in the consideration of such cases. If you cannot comprehend this complex table, do not pass GO, do not collect $200. A more detailed discussion of the diagnosis and management of the specific diseases peculiar to diving will be found in the appropriate chapters.

CAUSES

A scheme showing the causes of unconsciousness in divers is shown in Figure 47.1.

Causes possible in all types of diving

Hypocapnia due to hyperventilation

Unconsciousness due to this cause is a theoretical possibility, and is self-correcting on land. It is observed in an anxiety or panic situation, and has been suggested as a cause of unconsciousness in divers. Hyperventilation can produce dizziness and altered consciousness, and the threshold for syncope is

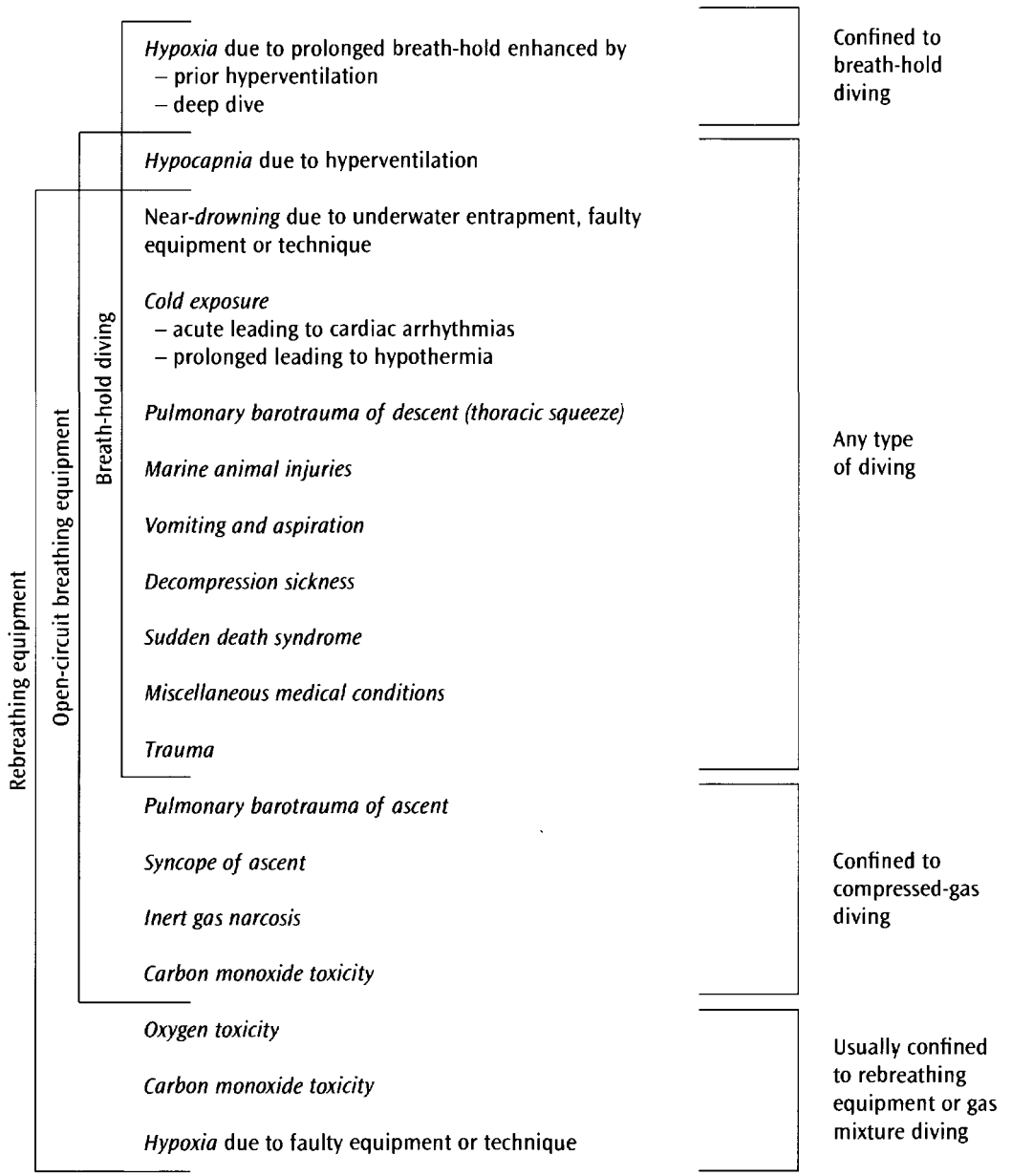

Figure 47.1 *Causes of unconsciousness in divers.*

reduced. The postulated mechanism involves cerebral vasoconstriction and reduced cerebral blood flow.

Hypocapnia is an uncommon event because it is countered by:

- Breath-holding.
- The resistance of the breathing apparatus and increased gas density at depth.
- Oxygen breathed at higher pressures depresses the respiratory response to carbon dioxide. It interferes with the transport of carbon dioxide in the blood and increases tissue carbon dioxide tension.

- With closed or semi-closed rebreathing apparatus, the resistance and dead space counteract hypocapnia.

If hyperventilation does cause unconsciousness during diving, it is only likely near the surface or with an excessive supply of gas.

Near-Drowning

This is one of the commonest causes of unconsciousness (and subsequent death) in divers (see Chapter 25), and it may follow saltwater aspiration (see Chapter 24). The analysis of the contributing factors is vital if recurrence is to be prevented (Chapter 25).

Cold

Sudden exposure to cold water may cause reflex inhalation. In some people cold water in the pharynx may stimulate the vagus nerve, causing reflex sinus bradycardia or even asystole. A prolonged exposure to cold sufficient to lower the core temperature to less than 30°C will cause unconsciousness, and the possibility of cardiac arrhythmias. A fall in core temperature to 33–35°C may lead to an insidious onset of confusion and decreased mental and physical function. In this hypothermic state the diver is likely to drown (see Chapters 27 and 28).

Cold urticaria causing unconsciousness is described in Chapter 42.

Pulmonary barotrauma of descent ('thoracic squeeze')

In the breath-hold diver, this is likely only when descent involves a pressure change ratio greater than that between his initial lung volume, or total lung capacity with maximum inspiration, and his residual volume (see Chapter 6). However, in the scuba diver, thoracic squeeze is unlikely except when the diver is grossly overweighted and sinks rapidly, or first loses consciousness from some other cause and then sinks. In both situations, loss of the air supply before the descent makes the diagnosis more tenable.

The diver supplied with gas from the surface is at greater risk. Thoracic squeeze is possible if the increase of gas pressure does not keep pace with the rate of descent, or if the gas pressure fails (e.g. severed gas line) in equipment not protected by a 'non-return' valve.

Marine animal injuries

Unconsciousness may result from the bite of the sea snake or blue-ringed octopus, the sting from coelenterates such as *Chironex*, or the injection of toxin from cone shells, fish or stingrays (see Chapter 32). The casual snorkeller is as likely to be a victim as the experienced scuba diver, more so if he is ignorant of these dangers. Accurate diagnosis depends on awareness, knowledge of geographical distribution of dangerous marine animals, the clinical features and, most important, identification of the animal.

Vomiting and aspiration

This may result from seasickness, aspiration and swallowing of seawater, gastrointestinal barotrauma, unequal vestibular caloric stimulation (e.g. perforated tympanic membrane) and middle- or inner-ear disorders. Causes unrelated to diving itself include gastroenteritis and overindulgence in food or alcohol.

Aspiration of vomitus from the mouth piece is a possibility in the inexperienced diver. Vomitus ejected into a demand valve may interfere with its function, or may damage the absorbent activity in rebreathing sets. If the diver removes his mouth piece to vomit, he may then aspirate seawater during the subsequent reflex inhalation. In severe cases, unconsciousness may result from respiratory obstruction and resultant hypoxia.

Decompression sickness

This is extremely unlikely in breath-hold divers, although it has been described in native pearl divers (Taravana) and submarine escape training instructors who have a very short surface interval between deep breath-hold dives (see Chapter 61).

Unconsciousness during, or immediately after, compressed-gas diving is occasionally due to cerebral or cardiovascular decompression sickness (see Chapter 12), or cerebral arterial gas embolism from pulmonary barotrauma of ascent (Chapter 6).

Miscellaneous medical conditions

The diver is as prone to the onset of sudden incapacitating illness as the equally fit, or unfit, land athlete. Diagnoses such as myocardial infarction, cardiac arrhythmia, syncope, cerebrovascular accident, diabetes with hypoglycaemia or hyperglycaemia, epilepsy, etc. should be considered. The metabolic disturbances of renal failure, liver failure or adrenal failure can affect cerebral function, but would be infrequent in divers.

Drugs, either legal or illicit, may depress central nervous system function, enhance carbon dioxide retention (e.g. opiates), affect cardiac performance (e.g. some antihypertensives), or alter blood sugar (insulin or oral hypoglycaemic agents) (see Chapter 43 and other text).

Trauma

A diver could be rendered unconscious by a blow to the head or chest. Underwater explosion is discussed in Chapter 34.

Diagnosis of cause of unconsciousness in a diver:

- *Type of equipment and breathing gases.*
- *Depth/duration, of this and other recent dives.*
- *Environmental factors.*
- *Clinical history and physical signs.*

Causes confined to breath-hold diving

The main cause, hyperventilation and breath-holding-induced hypoxia, is discussed in Chapters 16 and 61, as is hypoxia of ascent, together with the less frequent causes.

Causes usually confined to compressed-gas diving

Pulmonary barotrauma of ascent

Cerebral arterial gas embolism (CAGE) (see Chapter 6) must be the provisional diagnosis to be excluded in the diver who surfaces and rapidly becomes unconscious. Tension pneumothorax may also cause unconsciousness due to hypoxia or cardiovascular disturbance.

Syncope of ascent causing unconsciousness or altered consciousness may be due to partial breath-holding during ascent. This is thought to be due to an increase in intrathoracic pressure interfering with venous return and thus reducing cardiac output. Unless it proceeds to pulmonary barotrauma or drowning, it is rapidly self-correcting with the resumption of normal respiration.

Postural hypotension causing syncope may develop as the diver leaves the water due to loss of hydrostatic circulatory support. This is more likely in the dehydrated or peripherally vasodilated diver, from whatever cause. It is also aggravated by many drugs.

Inert gas narcosis

In deep dives, unconsciousness may result from the narcotic effects of nitrogen in the compressed air (see Chapter 15). An unconscious diver on the surface is not suffering from nitrogen narcosis, because the narcosis is reversible upon reduction of the environmental pressure. However, irrational behaviour or poor judgement due to narcosis may lead to drowning.

Unconsciousness due to narcosis is likely at a depth greater than 90 metres, breathing air, but variable degrees of impaired consciousness may be seen from 30 metres onwards. This is especially so if other factors contribute, for example carbon dioxide accumulation and oxygen pressures.

Carbon monoxide toxicity

Carbon monoxide toxicity is often proposed as a cause of unconsciousness in scuba divers (see Chapter 19). Diagnosis is aided by examining compressor facilities, and is confirmed by estimations of carbon monoxide in the gas supply and the victim's blood.

Causes more likely with technical diving and rebreathing equipment

Oxygen toxicity

The diver breathing gas mixtures containing high percentages of oxygen may induce central nervous

system oxygen toxicity if the partial pressure exceeds 1.6 ATA, especially with heavy exercise (see Chapters 17 and 63). Closed-circuit oxygen sets are particularly dangerous because of the complementary carbon dioxide effects.

Carbon dioxide toxicity

The most common cause of unconsciousness in divers using rebreathing equipment is a build-up of carbon dioxide (see Chapters 4, 18 and 63). The possibility exists that some compressed-air divers, under conditions of increased gas density at depth and heavy exercise, may retain carbon dioxide. Resultant carbon dioxide toxicity may impair consciousness, exacerbate oxygen toxicity and/or nitrogen narcosis.

Hypoxia due to faulty equipment or technique

This may be produced by insufficient oxygen in the inspired gas mixture, dilution, ascent, excess oxygen consumption or incorrect gas mixture for the set flow rate (see Chapters 4, 16 and 63). The manifestations are those of hypoxia from any cause. Provided that the hypoxia is rapidly corrected, prompt recovery can be expected.

> *A rapid recovery is to be expected in uncomplicated cases of transient hypoxia, carbon dioxide toxicity, syncope of ascent, oxygen toxicity – after the patient is removed from the offending environment.*

CONTRIBUTORY FACTORS

Several factors, although not direct causes, may contribute to the development of unconsciousness under water. The **inexperienced diver** is more likely to be involved in such an episode.

The role of **psychological factors** in causing unconsciousness has often been postulated. For example, the anxious diver may be more prone to cardiac arrhythmias related to sympathetic nervous system stimulation. Indeed, cardiac arrhythmias (see Chapter 39) have been postulated as a cause of otherwise unexplained collapse or death in the water.

A vigorous **Valsalva manoeuvre** may increase the likelihood of syncope due to reduced venous return, and hence decreased cardiac output. This is less likely in the water because the hydrostatic support reduces peripheral pooling of blood.

The **overheated diver** may develop heat exhaustion with mild confusion, but heat stroke with coma and/or convulsions is unlikely. Conversely, the diver exposed to cold may have a higher incidence of equipment (and absorbent) failure, dexterity reduction and cardiac reflexes.

A fall in plasma glucose may result from fasting or high physical activity. In the normal individual, the degree of **hypoglycaemia** is not enough to produce symptoms, though alcohol, by inhibiting gluconeogenesis, may contribute to significant hypoglycaemia in the fasted (longer than 12 hours) diver. Chronic alcohol abuse damages liver glycogen storage, and hypoglycaemia may develop after a shorter fast.

There are some cases of unconsciousness that are particularly difficult to explain, even incriminating a multi-factorial aetiology.

'SHALLOW WATER BLACKOUT'

This term was used initially by the British to refer to carbon dioxide toxicity with rebreathing equipment, whereas some Americans recently used it to describe unconsciousness following hyperventilation and breath-hold with free diving. The term has therefore created much diagnostic confusion and should be avoided. It also suggests (see Miles, 1957) that there is some mysterious cause of unconsciousness in divers and that there is a multifactorial aetiology.

Attempts to define specific causes of unconsciousness should always be made. Nomenclature should then follow this specific cause, e.g. syncope of ascent, dilution hypoxia, carbon dioxide toxicity, oxygen toxicity, etc.

MANAGEMENT

The first step in management of all cases is the establishment and maintenance of respiration and

circulation. The administration of 100 per cent oxygen at 1 ATA will do no harm, even in central nervous system oxygen toxicity, and will be of great benefit in most other cases. If breathing is not spontaneous, positive-pressure respiration will be required, despite the argument that it may be detrimental in cases of pulmonary barotrauma. External cardiac massage may also be necessary. Divers suspected of CAGE or DCS also require rapid recompression (see Chapters 6, 13 and 48–50).

The most common causes of unconsciousness in divers include:

- ***Breath-hold** – hyperventilation followed by breath-holding, leading to hypoxia.*
- ***Compressed-air equipment** – drowning due to faulty equipment or technique.*
- ***Rebreathing equipment** – carbon dioxide toxicity.*

Maintenance of adequate circulation and ventilation may be all that is required in cases of near-drowning, pulmonary barotrauma of descent and marine animal envenomations.

Cases of carbon dioxide toxicity, oxygen toxicity, nitrogen narcosis and temporary hypoxia from whatever cause, uncomplicated by aspiration of seawater, usually recover rapidly with return to normal atmosphere.

Miscellaneous medical conditions, hypothermia, aspiration of vomitus, trauma, etc. are managed along general medical principles. The assessment should encompass:

- a clinical and social history;
- a diving history and profile;
- physical examination and investigation;
- examination of the equipment and gas used; and
- and an appreciation of the environmental factors.

Valuable information, including the sequence of events leading to unconsciousness, may only be obtainable from the diver's companion or other observers.

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48

First aid and emergency treatment

ROBYN WALKER

> *"There is no such thing as an accident. What we call by name is the effect of some cause which we do not see."*
>
> *Voltaire*

GENERAL

This chapter deals with the immediate treatment of diving accidents. Specific diseases are discussed in greater detail elsewhere in this book, but an overview is presented here for easy reference.

Despite the best laid plans, diving accidents will occur. Frequently, they result from a chain reaction sequence originating from a failure to follow simple safeguard procedures. Prevention of injury is paramount, and individual divers have a responsibility to ensure that they are medically fit to dive, are appropriately trained on the equipment they are using, and plan their dive with due respect for the prevailing conditions (current, temperature, etc). Before entering the water, all divers should have discussed alternate exit routes, ensure that they are carrying an appropriate personal locator device (whistle, light, strobe, safety sausage, EPIRB), and have discussed emergency procedures with their diving buddy.

When a diving accident occurs, the dive organizer should be able to identify the symptoms and signs of this illness and implement an emergency action plan. That is, he should know the location of the nearest medical facility, the local contact numbers for emergency services, and also have a means of contacting them (radio or mobile telephone).

FIRST AID

There are two aspects to first aid, one being the rescue and the other the specific first aid treatment.

Rescue

In 70 per cent of the diving fatalities in one series, there was a period in excess of 15 minutes between the accident and the rescue. This is an unacceptable time for any patient to remain under water without functioning breathing equipment. Thus, in many cases, there is a progression from unconsciousness to death, which could have been prevented.

The practice of buddy diving is the single most important factor in rescue. It requires that each diver is responsible for the welfare and safety of his companion. It infers: a reliable method of communication between the divers; a practiced rescue technique; and a basic knowledge of resuscitation.

Whilst some authors and recreational dive training agencies advocate the use of in water expired air resuscitation (EAR) and external cardiac massage (ECM), there is insufficient evidence available to recommend the routine use of this practice. In water, EAR and ECM should never delay the retrieval of an injured diver to a stable platform or ashore. Resuscitation of the diver should follow the guidelines issued by the American Heart Association.

Specific treatment

All recreational divers should be encouraged to acquire first aid skills and maintain competence in airway management, EAR, ECM, haemorrhage control and delivery of 100 per cent oxygen. They should also be aware of warming techniques for hypothermia and methods of reducing marine animal envenomation, including pressure bandages and symptomatic relief.

Most importantly, divers should recognize their own limitations when dealing with diving emergencies, and seek expert medical advice promptly.

RESUSCITATION EQUIPMENT

Whether diving from shore or from a vessel there should be a proven and exercised system for retrieving, in a horizontal position, an unconscious diver from the water. All dive sites should be equipped with an oxygen delivery system capable of administering 100 per cent oxygen to conscious and unconscious victims. The volume of oxygen needed should be sufficient to supply two divers for the duration of the retrieval to the nearest medical facility.

Oxygen (100 per cent) should be administered continuously to all diving accident victims until specialized diving medical physician advice is received. (Oxygen delivery systems are described in Chapter 49.)

Previously, a head-down position was adopted in patients suspected of having cerebral arterial gas embolism in order to prevent re-embolization. Unfortunately, patients were transported (often for many hours) in this position, and this resulted in deteriorating cerebral function as a consequence of cerebral oedema. It is now generally advised that a horizontal posture is best for most diving accident victims. Divers who are suspected of having a cerebral artery gas embolism (CAGE) should not be allowed to sit or stand until advice has been obtained from a diving medical physician. Testing of balance or gait should ideally be left to the diving physician at the treating recompression chamber. In patients with an altered mental state or who are at risk of aspiration, the left lateral recovery position should be adopted. Divers with vertigo should be kept as still as possible and avoid straining manoeuvres likely to further damage their inner ear such as coughing, vomiting or performing a Valsalva.

Intravenous fluids should be administered to all seriously injured divers. A recommended regime is 1 litre as fast as possible, followed by 1 litre over 4 hours of normal saline alternating with Hartmann's solution. This regimen may need to be varied if clinical parameters such as urine output and blood pressure are low. Urinary catheterization may be required if urine output is low. Cerebral injury may be exacerbated by hyperglycaemia, and therefore glucose-containing solutions should be avoided unless the patient is hypoglycaemic.

Consideration may be given to the administration of suitable oral fluids as long as the patient is fully conscious and does not suffer from nausea and vomiting. The administration of oxygen will be interrupted if the patient is given oral fluids, and it is preferable for these interruptions to be kept to a minimum.

There is no indication for the use of aspirin or steroids in the first aid management of a diving accident victim with decompression sickness (DCS), and analgesics should only be prescribed after discussion with the treating hyperbaric specialist. Response of symptoms to recompression forms the basis of the selection of an appropriate treatment table, and analgesics may mask symptoms – making this decision more difficult than needed. Nitrous oxide (Entonox) must never be used in divers

following diving exposure, as it will diffuse into the bubbles aggravating the clinical effect.

LOCAL MEDICAL TREATMENT

The local physician has a role to play in the prevention of diving accidents by the appropriate medical screening of diving candidates and in the provision of advice on safe diving practices. The local physician will be responsible for primary care such as oxygen and intravenous fluid administration, correction of electrolyte disturbance and advanced life support.

In many cases the local physician can work as a liaison officer between the divers and a diving medicine specialist, especially when recompression therapy is required. The local physician will also be important in integrating the transport and medical evacuation needs, if these are required. The local physician should have available to him direct communication with a specialist in diving medicine, so that the appropriate measures can be instituted.

Prompt assessment and early consultation with a specialist diving medical physician is recommended if full recovery from disorders such as DCS and CAGE are to be achieved.

TRANSPORT

In cases of DCS and pulmonary barotrauma, recompression therapy is usually indicated. The need for prompt recompression in both CAGE and neurological DCS is universally accepted. If there is less than 5 minutes' delay in treatment of CAGE then, even though 5 per cent may die, the morbidity among the survivors is low. In contrast, if the delay exceeds 5 hours, mortality is 5–10 per cent and at least half the survivors are left with residual problems. In many circumstances the facilities may not be immediately available for formal recompression therapy. Thus, a decision must be made as to whether the patient is to be recompressed by descent in the water, transported to a recompression chamber, or have a recompression chamber transported to him.

Retrieval

The decision to transport an injured diver suspected of having DCS or pulmonary barotrauma should be made early, as it is likely that the longer the delay in recompression the worse the outcome. Whatever mode of transport is used, appropriate resuscitation such as oxygen and intravenous fluid administration must not be compromised.

The decision on mode of transfer should be made in conjunction with the receiving hyperbaric medical unit. Road transport may be preferable if long distances and ascent to altitude (mountain ranges) are avoided. The use of aircraft (fixed and rotary wing) poses a problem of additional altitude stress unless the aircraft cabin can be pressurized to 1 ATA.

A maximum altitude of 300 metres is considered reasonable when transporting an injured diver. There are circumstances, however, when a commercial aircraft (cabin pressure equivalent to altitude of 2400 metres) is the only available option, and the continuous administration of 100 per cent oxygen is very important in such a case.

A helicopter flight skimming the surface of the ocean adds little further altitude stress, but even limits of 300 metres over land and 600 metres at night or in bad weather may make road transfer a better option. Delays in specialized rescue assets reaching the victim should also be considered.

The severity and **duration** of the injury is also important; the diver who has had minor symptoms for over 24 hours is not likely to deteriorate, but the diver who surfaces with symptoms of spinal DCS is likely to progress, has a worse prognosis, and the need for medevac and emergency recompression is much greater.

Any gas-filled space will expand with ascent to altitude, so special care should be taken in patients suspected of having a pneumothorax.

It has been suggested that vibration characteristics of both rotary and fixed-wing aircraft may negatively affect the clinical condition of the injured diver; however, there are no research data available to quantify this clinical impression.

Portable recompression chambers

An alternative to transporting the injured diver to a hospital-based recompression chamber is to bring a small portable recompression chamber to the diver (Fig. 48.1). The logistics of such an operation, the specialized procedures for transporting the chamber, the lack of sophisticated patient monitoring equipment within the chamber, and ensuring that adequate supplies of air and oxygen are available makes this option less desirable.

If portable chambers are used they should be large enough to accommodate a medical attendant as well as the diver, and ideally they should have a transfer-under-pressure capability with other chambers. Once you commit a diver to a treatment in most of these small chambers you have no means of entering additional medical personnel until the therapeutic table has been completed. To surface the chamber mid-treatment exposes the medical attendant to DCS and also induces clinical deterioration in the patient.

In-water recompression

In an attempt to initiate early therapy, many divers have been recompressed on air in the water. Both mechanical and physiological problems are encountered. Requirements for the diver support include a sufficient supply of compressed air, tolerable weather conditions, a full facemask or helmet to prevent aspiration of water, adequate thermal protection, and constant attendance.

The problems often encountered with in-water air recompression therapy include seasickness, drowning, hypothermia, panic and the aggravation of the illness by subjecting the patient to a further uptake of inert gas in the tissues, compounding the problem on subsequent ascent. In many cases, the regime has to be terminated prematurely because of adverse weather conditions, equipment failure, sharks, physiological and finally psychological difficulties. Even when the treatment does give relief, it often needs to be supplemented with the conventional recompression therapy carried out in a chamber, soon after the patient has left the water.

A much more effective water recompression therapy uses 100 per cent oxygen, at a maximum of 9 metres, and is relatively short in duration. This regime, which has been particularly valuable in remote localities, is described in Chapter 13 and appendix C. It usually involves a 30 to 90-minute stay at 9 metres, and ascent at the rate of 12 minutes/metre. It has the advantage of not aggravating the disease by adding more inert gas, being of short duration, avoiding any problem of nitrogen narcosis, and being suited for shallow bays and off wharves, protected from the open sea.

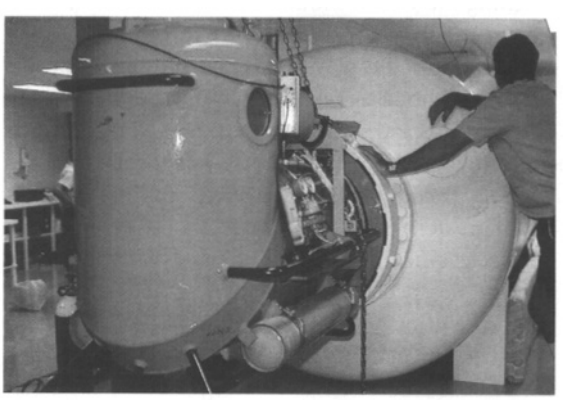

Figure 48.1 *Portable Duocom recompression chamber performing a transfer under pressure with a multiplaced chamber.*

DIVING MEDICAL KIT

Diving support boats should be equipped for likely accidents. No checklist can contain materials needed for all situations, but as a general guide, the following items should be considered:

- Airways for use in mouth-to-mouth respiration.
- Resuscitator with an oxygen supply and instructions regarding its use and fire risk (in drowning, pulmonary barotrauma, decompression sickness).
- Mechanical suction system, not reliant on scarce oxygen supplies.
- Topical antibiotic or antiseptic for skin injuries.
- Motion sickness tablets and injections.
- Dressings.
- Preparations to prevent and treat common otological problems, cold, sun or wind exposure.

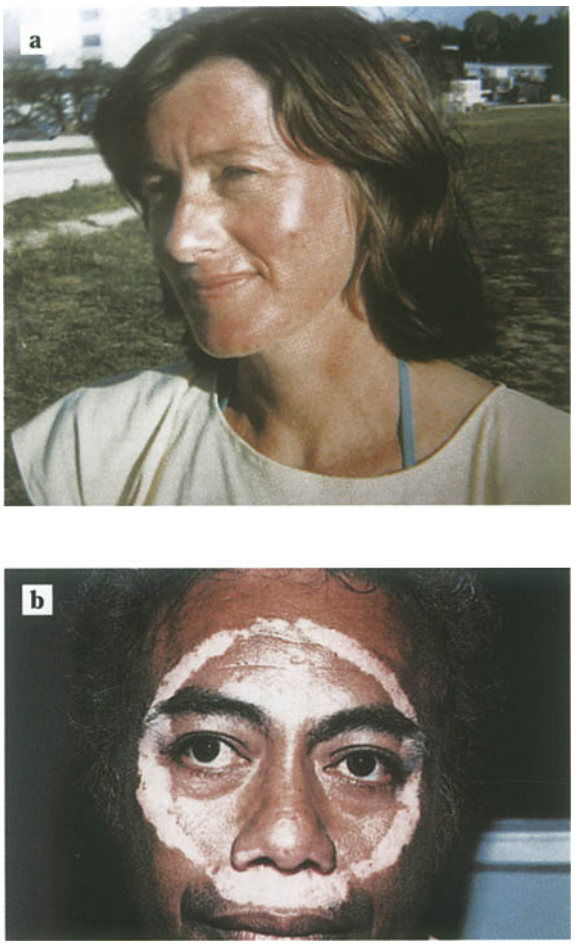

Plate 18 *Mask burn: (a) inflammation around the mask/skin contact. Mild and lasting only a few hours, this disorder leaves the conjunctiva unaffected. Compare Plate 2, showing mask barotrauma. (Courtesy Mrs R. Lowry.) (b) Using a full facemask, this islander experienced a severe reaction to the mask, with blistering, scarring and depigmentation. (Courtesy Dr C.W. Williams.)*

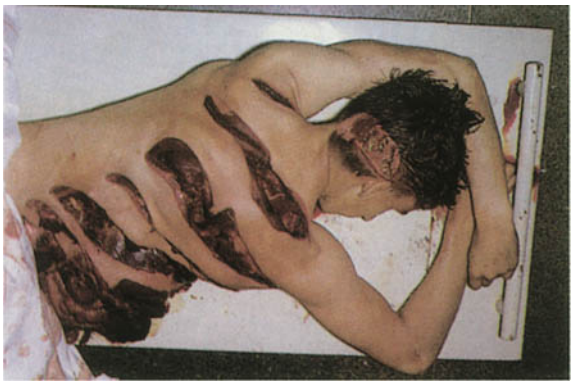

Plate 19 *Parallel lacerations from propeller injury. (Courtesy Dr W. Brighton, in Plueckhahn, V.D. (1991)* Ethics, Legal Medicine and Forensic Medicine, *2nd edition.)*

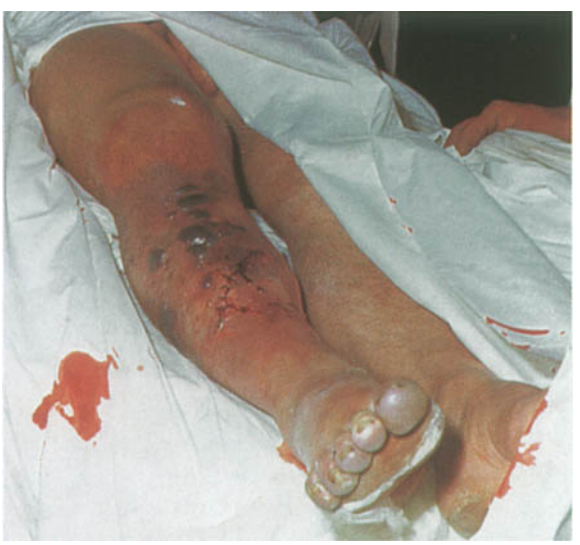

Plate 20 *Gas gangrene: swollen leg with pallor, hemorrhagic bullae and necrosis. The condition responded to hyperbaric oxygen therapy (see Chapter 49).*

Treatment for dangerous marine animal injury depends upon the fauna of the expected dive site, but may include:

- tourniquet (sharks, barracuda, eel bite) or pressure bandages (sea snake, blue-ringed octopus and some other venomous animals);
- local anaesthetic ointment and vinegar for box jellyfish;
- local anaesthetic injection without adrenaline (scorpion fish, stonefish, stingray, catfish); and.
- appropriate antivenoms (sea snake, box jellyfish, stonefish).

If DCS is possible, consider:

- a large oxygen cylinder for surface supply to helmet or full facemask with adapter to supply surface respirator if needed;
- an underwater recompression unit for remote areas;
- an intravenous infusion system (e.g. physiological saline);
- a urinary catheter;
- adequate compressor or air bank to support the diving attendants;
- a portable two-person recompression chamber if possible; and
- a medical kit required for recompression chamber.

Special circumstances will require supplementation of the above medical kits. In remote localities, diagnostic and therapeutic equipment ranging from a thermometer to a thoracotomy set may be appropriate. Eardrops to prevent and/or treat external ear infections are especially necessary in the tropics. Broad-spectrum antibiotics are also needed, though the use of these will depend on the clinical training of the attendants. A laryngoscope and endotracheal airway may be of use if trained medical assistance is available. It is important that diving support boats have radiocommunications to obtain expert medical advice on the treatment of difficult or unexpected problems. Frequently, medical or paramedical personnel are available coincidentally.

In an emergency, specialist diving medical advice can be obtained:

- USA
  Divers Alert Network (DAN)
  1-919-684-8111 or 1-919-684-4326
- within Australia
  Divers Emergency Service (DES)
  1800 088 200
  Divers Alert Network South East Asia Pacific (DAN SEAP) 61-3-98869166

(see appendix F for more information).

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49

Oxygen therapy

CARL EDMONDS

PHYSIOLOGY

Oxygen can be used, at least as a first aid, in almost all serious scuba diving accidents.

Breathing dry air, at 1 ATA results in an inspired oxygen pressure of approximately 160 mmHg. The oxygen pressure is diluted somewhat due to the 'dead space' of the respiratory passages, the continual absorption of oxygen from the lungs, and humidification, resulting in an alveolar oxygen pressure (P_{AO_2}) of approximately 100 mmHg. The arterial oxygen level (Pa_{O_2}) reaches about 673 mmHg, if 100 per cent oxygen is breathed. These and subsequent figures are approximations, and vary with many factors such as ventilation perfusion ratio, haemoglobin, pH, exercise and carbon dioxide (CO_2) levels.

Because of the difference between alveolar and venous pO_2, i.e. 100 and 40 mmHg, there is a 60-mmHg O_2 gradient diffusing toward the pulmonary blood. It is then carried in arterial blood, usually at a pressure of 100 mmHg to the tissues.

Normally, 97 per cent of the O_2 is transported in combination with haemoglobin in the red cells, with the remaining 3 per cent dissolved in plasma. One gram of haemoglobin can bind 1.34 ml O_2, and therefore 100 ml of blood can transport about 20 ml O_2 in combination with haemoglobin. As only 97.5 per cent of the haemoglobin is usually saturated, the O_2 carried in blood is usually 19.5 ml. In venous blood the saturation of the haemoglobin with O_2 is 75 per cent, with an O_2 pressure of 40 mmHg.

To maintain the alveolar O_2 tension, during exercise, the rate of ventilation increases. Thus, with a resting O_2 consumption of 200 ml/minute, the ventilation rate may be 5 litres/minute. At consumption of 1000 ml/minute the ventilation would rise to approximately 20 litres. Cardiac output also increases with exercise, up to six or seven-fold, transporting more O_2 to the tissues.

The O_2 in the tissues usually has an upper limit of about 40 mmHg, varying between 20 and 40 mmHg. In a compression chamber the O_2 pressure may rise 10-fold, but the tissue O_2 changes very little. The saturation of haemoglobin can rise only 3 per cent, as it is already mostly combined with O_2. The 3 per cent can be achieved at Pa_{O_2} levels of between 100 and 200 mmHg. Thus, increasing the inspired O_2 concentration or the total O_2 pressure does not greatly increase the O_2 transported by haemoglobin, but does influence that held in physical solution, in a linear relationship.

By the time the O_2 has passed from the alveolar blood (100 mmHg) through the tissues (20–40 mmHg), to the mitochondria within the cells, it may only be a few mmHg.

With the increasing pressures, the amount of O_2 dissolved in the plasma will increase as the P_{AO_2} increases. The latter will also increase disproportionately to the environmental pressure, because some of the other alveolar gases remain constant (CO_2 40 mmHg, water vapour 47 mmHg).

At 1 ATA, the O_2 carried in physical solution in the blood is 0.003 ml per cent per mmHg. Thus, breathing air the P_{AO_2} is 100 mmHg, and there is 0.3 ml O_2 per cent. Breathing 100 per cent oxygen increases the amount in physical solution to 2 ml O_2 per cent.

By the time a pressure of 3 ATA has been reached in the chamber, the subject would have 1.3 ml of O_2 per cent being carried in physical solution whilst breathing air. This increases to over 6 ml whilst breathing 100 per cent O_2. As the tissue requirements for O_2 is less than this, the full amount of O_2 required for tissue oxygenation can be carried in physical solution, with the haemoglobin remaining fully saturated. This will interfere with CO_2 transport from the tissues to the lungs.

In venous blood, approximately 20 per cent of the CO_2 is transported by haemoglobin. The excess CO_2 is transported by the bicarbonate buffering systems and in physical solution. The elevation of the cerebral venous pCO_2 is of the order of 5–6 mmHg when venous haemoglobin becomes 100 per cent saturated with O_2.

The tissue O_2 levels are determined by the arterial O_2 level and the tissue blood flow, but vary with its consumption.

PATHOPHYSIOLOGY

Pathophysiological effects underlay the development of some manifestations of O_2 toxicity (see Chapter 17). The effects of hyperoxia are as follows.

1 Inactivation of the haemoglobin transport of CO_2.
2 Ventilation depression due to suppressive effect on the carotid and aortic bodies.
3 Washout of nitrogen from the alveoli, with increased likelihood of atelectasis.
4 Cardiovascular effects of bradycardia producing a decreased cardiac output and decreased cerebral blood flow.
5 Vasoconstriction of peripheral vessels of most organs, with increased peripheral resistance. The vasoconstrictive effect of hyperbaric O_2 develops somewhere between 1.5 and 2 ATA. This can be seen as a regulatory mechanism to protect the healthy tissues from exposure to excessive O_2 pressures.
6 An acidosis, with inhibition of cell respiration and some enzymatic activity (sulphydryl group) and an increase in free radical production.
7 Angiogenesis and increase in fibroblastic activity is an effect not always desired, as in the retrolental fibroplasia causing blindness in premature infants treated with high O_2 concentrations. It is also hazardous with the production of pulmonary O_2 toxicity with long-duration administration.
8 Neurological O_2 toxicity (see Chapter 17).
9 In patients with chronic obstructive airways disease – and this is rare in the active diving population – the administration of O_2 may be detrimental because respiration can be dependent on hypoxic drive. Once this hypoxia is corrected, respiratory failure is possible.

Table 49.1 *Transport of oxygen in the blood*

Pressure (Breathing gas)	P_{O_2} (air)	P_{AO_2} (air)	P_{AO_2} (100% O_2)	Dissolved O_2(ml) (air)	Dissolved O_2 (ml) (100% O_2)
1 ATA	0.21	102	673	0.32	2.09
2 ATA	0.42	262	1433	0.81	4.44
3 ATA	0.63	422	2193	1.31	6.80

Note: the breathing gas is noted in parentheses.

METHODOLOGY

The needs of the O_2 delivery system shall vary according to the inspiratory requirements of the diver, i.e. whether he is breathing, and the degree of cardiovascular pathology.

Facemasks

There are loose-fitting facemasks or devices (e.g. nasal prongs) which simply supplement the diver's normal respiratory air with various amounts of O_2. The effect will vary with the type of mask or device, its fit on the diver, the flow rate of O_2 employed, and the respiratory minute volume of the diver. The flow rates are usually between 2 and 8 litres per minute; however, higher flow rates need to be employed with certain masks, especially if a high inspired O_2 concentration is to be achieved.

Nasal cannulas can produce an O_2 concentration around 30 per cent, with flows of up to 4 l/minute. The **Hudson mask** can deliver around 50 per cent O_2, with a flow rate of 8 l/minute. **Venturi masks** can provide O_2 concentrations up to about 40 per cent.

All facemasks, unless there is a functional expiratory valve system, and/or a high flow rate, will allow some CO_2 accumulation (dead space). None of these masks is adequate for treatment of diving casualties as the inspired O_2 concentration delivered is too low. However, one loose-fitting mask which is capable of providing a higher O_2 concentration is the non-rebreather mask. Such a mask is fitted with an O_2 reservoir and a series of one-way valves to minimize air entrapment and prevent expired breath from entering the reservoir. If a good seal is achieved and if an O_2 flow rate of 15 l/minute is employed, inspired O_2 concentrations of 90–95 per cent are possible, although probably seldom achieved in the field. The Divers Alert Network includes a mask in its O_2 units for use by breathing divers who cannot tolerate a demand valve or for use as a secondary delivery option.

Alternatively, masks can be **airtight**. These are usually made of PVC or silicone that is soft enough to mould to the diver's face. Such a mask will require an adequate flow of O_2 for normal ventilation, and an exhalation valve.

If a demand valve is combined with a double seal or an oronasal mask, then maximal O_2 inhalation, in both conscious and unconscious breathing patients, is possible. In addition, positive pressure can be used with a non-breathing victim if the demand valve is fitted with positive-pressure capability and the O_2 provider has appropriate skills. In each case it is vital that the attendant continually ensures that there is no air entry into whichever mask is used.

Especially in the case of divers who are used to **demand systems**, a mouthpiece may be substituted for the oronasal mask to provide O_2 to a conscious patient. When this is used, it is prudent to employ a nose-clip, otherwise some admission of air is likely through that orifice. Nevertheless, most divers are fully aware of how to block off their nose, and if necessary this can be done by donning their normal air-tight half-face-mask. Divers are used to breathing with this system, and may prefer it.

There are available other **plastic helmet systems**, and these are often employed in hyperbaric chambers. They use neck seals and either demand systems or high flow rates (usually well in excess of 10 l/minute).

The simplest and most effective way to provide near 100 per cent O_2 to a breathing diver is with a demand valve system. However, if the demand system with its positive pressure is not to be used – and some would argue against its use in certain circumstances such as pulmonary barotrauma – then the anaesthetic type **bag/mask** unit can be employed. In this case there is a high O_2 flow to a breathing bag which is then attached to the mask. Inhalation is from the bag, and exhalation through a one-way valve.

These are **open-circuit systems**, and will usually require large volumes of O_2. Very high O_2 concentrations can usually be achieved if a reservoir is used with a bag-valve-mask system and a flow rate of 15 l/minute or higher. Demand valves will require a minute volume of 5–10 l/minute, if relaxed and at rest.

To attempt to conserve O_2, by using a **re-breathing system**, CO_2 absorbent can be used in a semi-closed or closed-circuit system. In this situation a smaller O_2 supply can be used, e.g. 2–4 l/minute in a resting patient, with a small but constant and equivalent exhaust system to ensure that there is a no nitrogen build-up. The absorbent will remove the CO_2

produced. Problems with such systems include the greater maintenance involved, and the higher level of training required.

Auer supply a rebreathing set with superoxide powder which, when activated by CO_2, produces a larger quantity of O_2 in a re-breathing system. This is of considerable value when there are limitations of space or restrictions regarding transport of high-pressure O_2 cylinders. The disadvantage is high cost, however.

Assisted respiration

In the unconscious and non-breathing patient the ventilation can be taken over either manually (bag-valve-mask) or O_2-powered ventilators. The ventilators can also be employed to assist respiration when this is present but impaired. In the event of respiratory depression, or reduced compliance, it might be necessary to institute positive-pressure respiration to ensure that the airways are inflated and that adequate tissue oxygenation is achieved. A wide variety of respirators are available, both pressure-cycled and volume-cycled. Some are suitable for use in hyperbaric chambers.

O_2 can also be administered in the underwater situation (see Chapter 13), in recompression chambers (with the same variety of methods described above), and during Medivac (Chapter 48).

Hyperbaric O_2 therapy (HBO)

Hyperbaric O_2 therapy is often employed for a variety of non-diving therapies, including:

- carbon monoxide and cyanide poisoning;
- gas gangrene (Plate 20);
- chronic osteomyelitis;
- radiation injury;
- aerobic and anaerobic infections;
- crush injuries, grafts and other ischaemias; and
- diabetic and ischaemic ulcers.

It has been employed, on an experimental basis, for burns, severe head injury, Hansen's disease, some envenomations and acute myocardial infarction.

Oxygen supplementation and HBO have both been employed in order to achieve improved tissue oxygenation when this has been impaired, for any reason. Tissue oxygenation depends on a combination of the arterial O_2 pressure and the circulation to that tissue.

HBO can assist in relieving tissue hypoxia and oedema, and there is also evidence that it may reduce the disastrous effect of leucocyte adherence to damaged endothelium, which plays a pathological role in acute decompression illness. It also improves the elasticity of red blood cells and reduces platelet aggregation.

A single 45-minute exposure to an O_2 tension of 2.8 ATA will completely block the activation of leucocytes – a mechanism of central importance to tissue injury and endothelium damage. This effect lasts up to 10 hours.

Unfortunately, increased oxygenation is likely to produce vasoconstriction, at least in normal tissues. A 20 per cent flow reduction in limb circulation in man can result from 100 per cent O_2 breathing, but this probably does not occur in hypoxic tissues. **Angiogenesis** is accelerated in ischaemic wounds when extra O_2 is supplied.

In animal experiments, poor perfusion and decreased O_2 tension has been shown to increase the likelihood of infection. This is the basis of the development of gas gangrene, and HBO inhibits the production of clostridium alphatoxin, which destroys cell membranes and increases capillary permeability. It also acts directly on *Clostridium perfringens* and has an indirect effect by blocking the leucocyte destruction.

DROWNING SYNDROMES

Oxygen supplementation is of enormous value in overcoming the hypoxic effects, including encephalopathy, which often follow the ventilation–perfusion anomalies and blood shunting in the lungs of the near-drowned victim. Although the ventilation in these victims tend to increase in order to reduce the elevated CO_2 levels, this may not be adequate to overcome the hypoxia.

Supplementary O_2, and especially positive-pressure respiration, may reduce the degree of shunting and assist in the pulmonary gas exchanges.

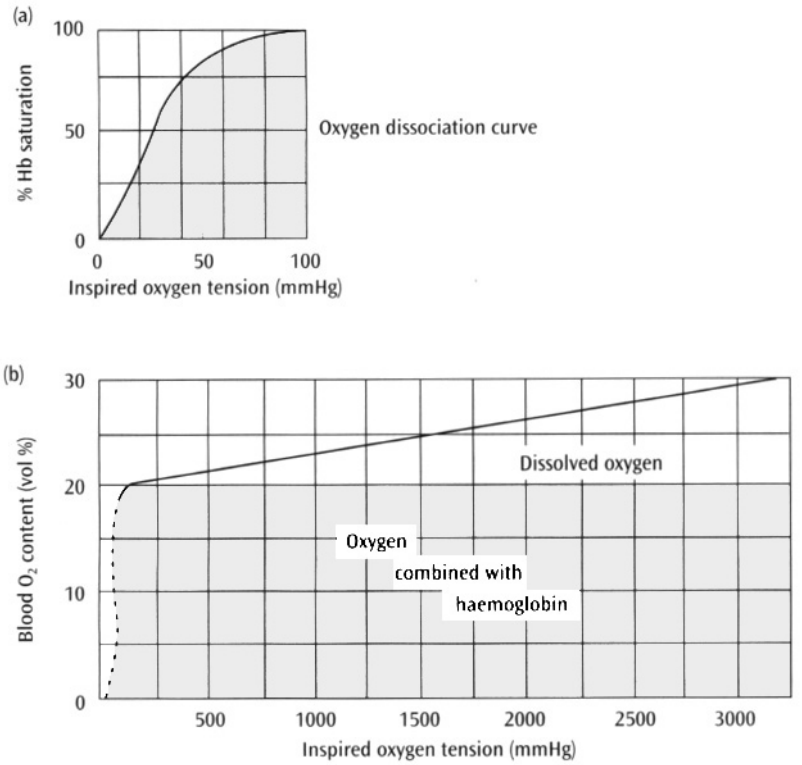

Figure 49.1 *Oxygen-carrying capacity of blood in (a) atmospheric and (b) hyperbaric oxygen environments. The curve in (a) represents the oxygen–haemoglobin dissociation curve.*

Indeed, one can gradually titrate and reduce the positive pressure of the assisted respirators, and subsequently the O_2 percentages, as the PaO_2 increases.

DECOMPRESSION

By removing the inert gas from the inspiratory gases, the inert gas pressure gradient is greater and therefore will result in a more rapid removal of this gas from tissues. This is the basis for which O_2 decompression is used to hasten the removal of nitrogen from tissues, more rapidly. This can be achieved by both supplementary O_2 at 1 ATA, and by increasing the depth or pressure. It is not so much the presence of O_2, but the absence of inert gas that is of value.

O_2 decompression is a well-established means of reducing decompression time. There are some theoretical difficulties, and it may well be that the increase in O_2 may also, in some ways, reduce the vascular perfusion of tissues, and thereby tend to reduce the nitrogen elimination.

Hyperoxia-induced vasoconstriction reduces tissue perfusion, resulting in a slowing of the nitrogen wash-out, causing -8.9 per cent and -16.9 per cent at 2 and 2.5 ATA respectively, compared with controls at 0.2 ATA. This effect is less obvious with helium.

BUBBLE DISEASE

Paul Bert first described the value of O_2 with decompression sickness, noting that the animals so affected did not deteriorate while breathing this gas. When the inert gas bubble phase has developed, for any reason within the body, it is important to remove the inert gas as rapidly as possible, so that the space will diminish and collapse. Unless this is achieved, then the tissue around the gas is likely to undergo histological changes which result in its stabilization – with the deposition of blood constituents around the bubble–tissue interface, or the bubble–blood interface.

The ways of reducing the size of the bubble include both pressurization (recompression) and

increased diffusion of inert gas by increasing the inert gas gradient from the bubble to the blood or tissue (see Figure 10.3).

The use of O_2 is relevant in both decompression sickness, in which there are gas bubbles within vessels or tissues, and also in some barotrauma accidents, such as pulmonary barotrauma (mediastinal emphysema, pneumopericardium, pneumoperitonium, air embolism, pneumothorax) and localized surgical emphysema from a variety of causes (see Chapters 6, 8 and 9). It might even be of some value in inner-ear barotrauma, where air has passed into the perilymph. Surface O_2 reduces clinical symptoms and the number of sequelae following treatment. There is some difference of opinion regarding the ideal pressure at which O_2 should be administered during recompression therapy, but usually the treatment pressures range from 2 to 2.8 ATA.

GAS TOXICITIES

Oxygen may be used in the form of nitrox to reduce or avoid the effects of nitrogen narcosis and decompression sickness. It may also be used to overcome or bypass some of the effects of carbon monoxide toxicity and the influence that this has, in a competitive manner, on O_2 transport.

DANGERS

These can be divided into logistic problems and those associated with fire and/or explosion. The **logistic difficulties** are similar to those with any compressed gas system, and similar – if not more stringent – care must be taken to ensure that there are adequate supplies of the gas to fulfill the various treatment regimes.

Accumulation of O_2, either from a cylinder or from the exhaust gases of a diver undergoing treatment, can result in a high O_2 concentration, with susceptibility to ignition. O_2 equipment must be specifically designed for this use, and as such there is an avoidance of the more flammable and corrosive materials.

Specifically designed O_2 regulators are employed to reduce the likelihood of spontaneous combustion, especially as the cylinder valve is turned on. O_2 regulators require special lubricants, because of the danger with high O_2 pressures, and must be maintained in a specialized manner, according to the manufacturer's instructions. Thus, the use of oils and greases is prohibited. Even the adhesive tape that divers often apply to protect the outlet and O-ring of the scuba cylinder, and to signify that it has been filled, should not be used. Cleanliness is vital in preparing and using O_2 systems.

When administering O_2, the equipment should be turned on, and shown to be functional before its use by the patient. In the case of a demand system, the cylinder is turned on and the valve purged before being offered to the diver. Although it is rare for the O_2 to be replaced by flame, this situation is worth avoiding.

O_2 cylinders for use in therapy require large volumes. The sizes, pressure ratings and colour-coding vary between countries. In Australia, the C size is 490 litres, D is 1640 litres, E is 4100 litres and G is 8280 litres. Even the smallest of these is not easily carried in some dive boats, where ideal stowage and stability may conflict with cleanliness, and produce other problems with boat handling and O_2 usage.

In some instances O_2 resuscitation devices are fitted with small cylinders for short-term emergencies – these are inadequate for O_2 therapy over long periods which require larger volumes. Specialized O_2 adaptors have been developed to supplement this inadequate supply with that from a larger cylinder, but these must be O_2- clean and compatible.

O_2 should only be used in areas which are well ventilated, and without any obvious source of ignition (e.g. cigarettes). In some instances (e.g. the confined cabins of aircraft) great care must be applied to ensure that the exhaled O_2 is ventilated overboard.

Attention must be given to the **storage** of the cylinders, ensuring that these are adequately shut following use, and with positive pressure still within the cylinders (to prevent egress of water). This is especially so with O_2, because of its increased corrosive properties. The remainder of the distribution system (regulators, hoses, etc.) should be stored after depressurization, as with scuba equipment.

Any O_2 cylinder less than half full should be replaced. The cylinder valve should not be left on,

because of the possibility of O_2 escaping and the fatigue it will cause to the O_2 regulator. The O_2 cylinder should not be allowed to become empty, but if for any reason it does then the valve should be turned off fully to prevent excess moisture and contaminants from entering.

Occasionally it may be necessary to use industrial O_2, when medical O_2 is not available. Because the industrial O_2 cylinder is frequently not evacuated and cleaned with each refill, contaminants may accumulate if the cylinder was left open. They are often used in situations in which there might be environmental and industrial pollutants. Despite this, testing of industrial O_2 by the suppliers does ensure that it is of an extremely high quality, and there should be no hesitation in employing it if it is the only available source. Before a medical O_2 cylinder is refilled, it is emptied, inspected and, if required, cleaned.

ACKNOWLEDGEMENTS

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Investigation of diving accidents: equipment

JOHN PENNEFATHER

INTRODUCTION

There are five areas in the investigation of diving accidents which require consideration. These are:

- Personal and past medical history.
- Environmental conditions.
- Dive profile and history.
- Medical examination.
- Diving equipment.

Only the last of these will be considered in detail in this chapter, as the others are discussed elsewhere in this book. Details of the diving disorders mentioned are also found in the appropriate chapters. This chapter is focused on the investigation of both deaths and non-fatal accidents.

Once an accident has happened, it is important that no interference occurs with the equipment, except to seal it and close the cylinders valves, in order to retain the breathing gas for analysis. In doing this it is important to note the readings on any gauges and the number of turns needed to close the valve(s). In particular one needs to be on guard against the well-intentioned but ignorant helper. In one accident involving a rebreathing set a 'helper' had decided that there was something wrong with the absorbent and he would have a look at it. In doing so he lost the opportunity to test the set for leaks and contaminated the gas samples with air.

PERSONAL HISTORY

Personal data may be relevant and may reveal the presence of diseases likely to predispose to a diving accident. The experience of the diver and his companions is of relevence, as accidents are more likely with inexperienced divers who are more likely to:

- misuse or fail to use equipment;
- use inappropriate equipment;
- mishandle and damage it;
- fail to undertake proper maintenance and repairs; and/or
- not recognize early malfunctions.

Any history of previous diving accidents is of interest, as problems experienced by divers with the same equipment may be repeated. Such disorders as salt-water aspiration, oxygen toxicity and carbon dioxide toxicity, may be due to equipment faults.

ENVIRONMENTAL CONDITIONS

The weather and water conditions at the time of the accident are of interest. During the dive there may be stress on equipment performance caused by swimming against currents. A diver who has to battle a 1-knot current frequently requires excess gas flow. The temperature of the water is also of importance, as it may cause malfunction in the diver (cold causing impaired manual dexterity and manipulation of equipment) and the equipment. Carbon dioxide absorbents are less effective under cold conditions; for example, with the temperature approaching freezing point regulators may freeze.

DIVE PROFILE AND HISTORY

Frequently, nowadays, dive computers will be able to be down-loaded to produce not only a detailed dive profile, but also documentation of other recent dives. This facility is especially valuable when diving fatalities occur without observers present. The depth is of importance in gas disorders, such as carbon dioxide and oxygen toxicity. The speed of ascent, if excessive, can contribute to dilution hypoxia as well as its association with barotrauma.

Activity during the dive is of importance, especially with rebreathing equipment. With strenuous exercise, the increased oxygen requirement may be in excess of that supplied by the flow rate and gas mixture chosen, causing hypoxia. With increased oxygen consumption there is also an increased carbon dioxide production, placing an added load on the absorbent system. Thus, the chance of both hypoxia and carbon dioxide toxicity are increased. With rebreathing sets if the set of primary interest has been flooded, the sets used by other divers in the group should be investigated because they may have the same defects.

With compressed air scuba there are also problems with increased exercise. First, there is an increased consumption of gas, and this may not be allowed for by a diver who is not noting the contents gauge. Second, there is the problem of an increased resistance to breathing, with a resultant dyspnoea and physical exhaustion. Panic and fatigue are then common sequelae.

With technical divers the possibility of confusing gas mixtures should be considered. Are the demand valves readily distinguishable? Is the gear likely to cause excessive drag?

Accidents are also likely to involve panic, so information of interest may not be remembered clearly. In the author's experience it is important first to let the witness tell their story without interruption, preferably without a third person present, and then for them to be questioned to clarify any points left unclear.

DIVING EQUIPMENT

A diving medical officer can help to minimize interference to the equipment used by the deceased. Other information of use may vary from information such as the absence of a fin or inadequate protective clothing in a diver exposed to cold water, and this should be noted. The correct authority for investigating the accident should be identified. In Australia there are several groups involved. For example, the water police have diving skills and knowledge of diving equipment, and normally the equipment is passed to them. In some circumstances it may be necessary to have a provable chain of custody so that unauthorized interference with the equipment is prevented. If the diver was employed, the workplace and safety inspectors will be involved to consider any possible fault by the employer. Fatalities will probably be considered by a coroner. A work-related accident, and accidents where there was possibly a criminal or negligent act causing a fatality, will probably be considered by a coroner. Other places have different investigatory organizations. A situation when the diver did not die may also be the subject of legal action in other courts. The possibility of a student or his/her relatives suing their dive school should also be considered.

The performance of other diving equipment or a cylinder containing air from the same source, used by other divers, may be informative. In fatal accidents, when the recovery of the body was delayed, or the set has flooded, the information will be scarce.

The sets used by companions may be a better guide to the cause than the set used by the diver of primary concern. It may also be the only information.

The following recommendations will require modification depending on the equipment used and the facilities available.

A **detailed external examination** will often be helpful. It may be necessary to consult the rescuer and any resuscitators to identify any item that may have been affected by the accident, or rescue. One should look for damage to protective clothing, the diving set and accessories. The facemask strap may be broken, and the buoyancy vest or counterlung may be flooded or damaged. The lugs of the mouthpiece may have been bitten during a convulsion, or there may be vomitus present on the equipment or on the mouthpiece. This could either be a cause of the accident, a complication aggravating the situation, or it may have occurred during resuscitation efforts.

Other observations may reflect both on the diver and on the rescue procedure. The absence of any equipment may be significant, and it should be determined – and noted – if this contributed to the incident, was caused by it or the rescue, or was coincidental. Points of possible significance include the ease with which the weight belt and other harnesses can be released, and whether this was done. The **protective suit**, its thickness and integrity, are relevant to both buoyancy and hypothermia. A depth gauge, watch and contents gauge on the gas cylinder will be worn by the diver who takes precautions against accidents. A knife is important to overcome entanglements in ropes and kelp. The state of inflation of the buoyancy vest may also be relevant.

The clothing may be cut from the body at the time of autopsy, but should be retained for assessment.

Investigation of air sets

With a compressed air-set, scuba or surface supply, before any connection is disturbed, it is often informative to attempt to inhale gas from the mouthpiece with the gas supply turned off. If air can be inhaled there is a leak in the system, and the set probably supplied the diver with a saltwater aerosol. This can cause saltwater aspiration or drowning, depending on the severity and duration of the leak. For reasons of hygiene it may be necessary to sponge the mouthpiece clean, but the demand valve should not be immersed or washed as this may dislodge the cause of the leak, if it is under the exhaust valve. The other causes of leaks that have been seen are perforations in the diaphragm and loose fittings that allow water entry. If there is likely to be air remaining in the cylinder it should be turned on, to check the reading on the contents gauge, and to look for leaks in all parts of the set.

The cylinder should then be disconnected and the air analysed for oxygen, carbon dioxide, carbon monoxide and water vapour; the oil content and odour should also be considered. It may be appropriate for this analysis to be performed by a qualified analytical chemist rather than a less well-equipped laboratory with inaccurate detector tubes. The chemist will normally have access to gas chromatography, which can be used to detect a wide range of substances in a small sample. With appropriate equipment it is even possible to analyse the gas in a cylinder that has been emptied, by withdrawing some of the residual gas with a syringe and injecting it into the gas chromatograph.

In some cases the cause of the accident is clear from the analysis. Low oxygen supports a diagnosis of hypoxia, while a high oxygen content in an air or nitrox mixture needs to be considered along with the depth reached, but may suggest oxygen toxicity. A high solids reading should lead to a microscopic examination of the solids. Some accidents have been caused by pollens triggering asthma.

The functioning of all parts of the equipment should be tested by a person who understands how it should perform, and should indicate the following:

- The demand valve gives a free, controlled release of gas. The purge button operates.
- Note if any reserve supply system was operated, and check that any low pressure warning system operates at the correct pressure.
- Vest inflation and dump, a faulty system can lead to either no buoyancy, or an uncontrolled ascent and pulmonary barotrauma.
- The accuracy of depth and cylinder contents gauges. Faulty contents gauge readings can lead to

unplanned gas exhaustion, while a faulty depth gauge can cause decompression sickness by the wrong table choice.

- Weight belt, release mechanism and amount of lead compared to minimum needed. Excess lead means an excessive amount of air in the buoyancy compensator, and possible loss of buoyancy control.
- Likely position in water if unconscious. Some vest/weight combinations help to keep a victim's head out of the water, but others may help to drown them.

Diving equipment

- *Facemask, snorkel, buoyancy vest, depth gauge, watch, contents gauge on cylinder, buddy line, knife, weights, etc. – presence, absence or condition.*
- *Protective clothing.*
- *Functioning demand valve and reducer.*
- *Cylinder condition, valve position (number of turns to close).*
- *Position of any reserve gas supply mechanism.*

In water, trials of the equipment may be conducted by experienced helpers (water police or navy divers), especially in cases where the cause of death is not clear. The trial should be performed by a person of approximately the same size as the deceased, using a similar dive profile (if deemed safe to do so), and with appropriate safety procedures. Valuable information regarding problems with equipment is sometimes obtained from these trials. For example, on one occasion a buoyancy vest caused a small person to float face-down if they relaxed in a manner to simulate loss of consciousness.

If there is access to the appropriate equipment, the dynamic performance of the gas supply system should be assessed at depth with a breathing machine, or person in a recompression chamber. This may demonstrate a gas supply failure that is not obvious in tests at 1 ATA. For example, in a case investigated by the author a restriction in the first-stage reducer was only obvious at simulated depth in a recompression chamber.

Investigation of mixed gas sets

For accidents involving the use of more complex equipment, such as a rebreathing set or technical diving equipment with multiple gas mixtures, the investigation can be more difficult. Often the critical question is what was the diver breathing at the time of the accident? If a rebreathing set has been sealed at the time of the accident, this can be determined by analysing the gas in the inhalation hose. Samples can be normally drawn from the hose with a fine needle and syringe, without causing a leak. Provided that the sample can be drawn soon after the dive, the carbon dioxide reading is often close to the correct reading even if the mouthpiece cock has not been shut. With a longer delay there will be a fall in carbon dioxide by diffusion to the absorbent even if the mouthpiece has been shut. Oxygen readings may remain helpful for weeks if the set was sealed promptly. In general, the more serious the accident the less likely that the set will be sealed promptly, and so the less helpful the gas analysis.

With a rebreathing set a **leak test** should be performed after the samples have been drawn and before any part of the set is dismantled. If there is gas remaining in the cylinder the gas can be turned on, the gauge read, and the leak test conducted before any part of the set is altered. This is normally conducted by filling the counterlung of the set and then immersing it in a tank of water. Compression of the counter lung, to raise the pressure in the set, will often reveal leaks that were not previously observed. If the gas supply is exhausted the set can be inflated via the mouthpiece.

With a demand set, where fresh gas flow is triggered by a negative pressure, a leak can sometimes be demonstrated by sucking the gas from the set with the cylinder turned off. A leak has been demonstrated if more gas can be sucked from the set a few minutes later. If the set leaks in these circumstances it is probable that water leaked into it during the dive. This is likely to be significant because a set with wet absorbent will have a reduced capacity to remove carbon dioxide and may give the user a caustic cocktail.

After leak testing, the set can be stripped and defects noted. **Valves** in the mouthpiece should be examined closely, as a perished valve can allow enough gas to escape scrubbing for carbon dioxide

toxicity to occur. On one occasion a small piece of grass stem in the valve seat caused the same problem. The packing of the canister also needs to be examined, as on occasion sets have been dived with no absorbent. More commonly there has been some water entry, and a carbon dioxide toxicity has occurred because of preferential flow through the dry absorbent.

In a semi-closed circuit set the flow of gas should be checked several times over a period equal to the dive of interest. During this time the set should be shaken and moved into several orientations that it could have been in during the dive. One puzzling hypoxia incident was caused by salt drying in the reducer housing and remaining as a dust that blocked the reducer jet after the set was shaken.

In any set the **composition of the gas** must be tested. Accidents where the wrong gas has been put in the cylinder are not rare. The effects of pressure on the performance of the reducer should be checked. In most cases this can be done with the maker's test equipment, but with some sets it may be necessary to conduct the test in a recompression chamber.

A test of the **performance of the absorbent** will be needed. This test can be done in some military laboratories, and sometimes by companies or departments which test other breathing equipment such as mine rescue apparatus. With this test it should be possible to establish if faulty absorbent or bad packing of the canister contributed to the incident. With the set connected to a breathing simulator, carbon dioxide is injected into the breathing loop of the set while it is cooled by immersing it in water. The absorbent should continue to absorb the carbon dioxide until the total of the dive time and the test time approximately equals the expected endurance of the set. If the results suggest that the absorbent was defective, the possibility of a faulty batch of absorbent should be considered and a fresh sample tested to prevent the absorbent causing another incident.

The testing of absorbent is an area where it can be helpful to test also the performance of the companion's set. In one case, the set used by the diver of primary interest was flooded and so it was not possible to test his absorbent. However, by testing the companion's set it was shown that the victim's absorbent was unlikely to have caused the incident.

RECOMMENDED READING

Most of the information provided in this chapter has been obtained from discussions with colleagues working for the Royal Australian Navy, the Royal Navy, the US Navy and Canadian Defence and Civil Institute of Environmental Medicine (DCIEM) in Canada.

The *Encyclopedia of Underwater Investigations* by Robert G. Teather (Best Publishing, 1994) is a useful general reference, but has less detail on the equipment aspects of accident investigation than has been provided above.

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Investigation of diving fatalities

CARL EDMONDS

Full fathom five thy father lies
of his bones are coral made.
Those are pearls that were his eyes:
Nothing of him that doth fade
But doth suffer a sea-change
Into something rich and strange

Shakespeare, The Tempest

INTRODUCTION

Once the body has been recovered, there are five separate areas which require assessment. These are:

- Personal and past medical history.
- Environmental conditions.
- Dive profile and history.
- Diving equipment.
- Autopsy investigation.

Some aspects are more relevant to the investigation by diving authorities, and are discussed in Chapters 4, 5, 25, 46 and 50. Details of the specific diving disorders are found in their appropriate chapters.

Once death has been certified, it is important that no further interference occurs with either the body, its clothing or the equipment, except for sealing the equipment, the closure of cylinder valves (recorded) to retain the breathing gas for analysis, and the taking of photographs.

As decompositional artifact often complicates the interpretation of autopsy findings, the body should be refrigerated as soon as possible, and transported to the mortuary for investigation.

RECOVERY OF BODY

Following a diving accident, information regarding the likely location of the diver is of relevance during the rescue phase, and subsequently during the body

recovery phase. In both instances the rescuer/recovery divers are frequently misled into searching for the victim 'down current' or 'out to sea' when the body is much closer to the site of the accident. The reasons for this include the increased weight of the victim with descent, entrapment, or snagging of the body or equipment on the sea bed.

A human body, when sinking to a depth of 5 metres or more, either without diving equipment or commencing with neutral buoyancy (weights and equipment versus buoyancy compensator and protective clothing) will develop a negative buoyancy. This is partly so because water replaces some of the gas spaces of the diver (especially the lungs), and partly due to the contraction of the gas spaces in the diver's body and the equipment, with descent. Submerged bodies weigh more than their surface weight. The apparent weight is increased with the depth of the water.

A series of relevant observations were made from diving accidents:

- If a rescue diver can effectively swim against the current, it is unlikely that a drowning victim will be far removed from the site at which the incident occurred.
- It should not be assumed that the body of the victim has drifted away from the 'last seen' point, until that area has been searched and found to be clear.
- A body, during its descent in relatively still water, will drift no more than a metre horizontally for every metre it descends vertically.
- If a marker buoy is dropped into the area where an observer has last seen the diver, the body is most likely to be found near that site. The marker buoy is very valuable in assisting others who may come to the site, after the original observers have left.
- The fatality usually follows quickly after the loss of buddy contact.
- The common exception to the above is when the diver has maintained positive buoyancy, and remained on the surface. Then the effects of tidal currents are very considerable, and the body may be found many kilometres distant from the site of the accident.

The tidal current movement is usually much less near the sea bed, where the body is found, than near the surface.

If the body is not in the vicinity of where the diver was last seen, then it is often able to be predicted by the last known activity of the diver, i.e. if his purpose was diving from point A to point B, the likelihood is that the body will be along that line. For **snorkellers**, the body is likely to be found in water in which the sea bed can be viewed from the surface – as snorkellers usually feel uneasy about swimming in water where this is not visible.

Apart from the search procedure, which is usually well known to rescue and police divers, other techniques are sometimes available for the location of the body. These include sonar systems and remotely operated underwater vehicles with video capability.

On discovering the body, if there is no likelihood of resuscitation, then observations should be made regarding its position, the presence of equipment in the surrounds, and any evidence of entanglement or tethering. If possible, **photographs** should be taken. The positioning of the body may explain the presence of 'travel abrasions', as often the forehead, nose, forearms, backs of the hands, knees and the top of the toes may be scraped along the sea bed with movement of the body.

The condition of the facemask, and any substance within it, should be noted.

Although the body may feel somewhat heavy initially, as it starts to ascend – from either the natural development of gas in the tissues, or because of the actions of the rescue/recovery team – then the ascent rate will increase rapidly according to Boyle's law. The body is usually able to be recovered by removal of the weights (preferable) or use of the buoyancy compensator. In either case note should be made of the conditions of this equipment prior to the rescuer's interference with it. If a buoyancy compensator is inflated, then care should be used to ensure that the ascent is not rapid, i.e. air may have to be released as it expands during the ascent.

PERSONAL AND PAST MEDICAL HISTORY

There are certain personal data which may be relevant in the assessment of the diving accident. First and foremost is the presence of diseases likely to predispose to the diving accident (see Chapters 46 and

52–54). Among recreational divers who died, at least 25 per cent were not medically fit for diving.

Other information which may be pertinent is the level of **experience** of the diver and his companions. Accidents are more likely with the inexperienced diver, and especially during his first 'open-water' sea dive.

The **history of previous diving accidents** should be sought. Many problems experienced by divers in the past are repeated under similar conditions. These include hypoxia from breath-holding after hyperventilation, panic with hyperventilation, saltwater aspiration, alternobaric vertigo, barotrauma and especially pulmonary barotrauma, nitrogen narcosis, syncope of ascent, decompression sickness (DCS), oxygen toxicity and carbon dioxide toxicity.

Alcohol and drug histories may also be very relevant, with both illicit and legal drugs contributing to accidents.

Personal and past medical history

- *Pre-existing diseases.*
- *Past diving experience and training.*
- *Any previous diving accidents.*
- *Drug and alcohol use.*

ENVIRONMENTAL CONDITIONS

Note should be made of the weather and water conditions prevailing at the time of the accident (see Chapter 5). Environmental factors contribute to 62 per cent of the deaths. Not only may the diver be exposed to an increased risk of injury during entry to or exit from the water, but also during the dive there may be exceptional stress in the form of impaired visibility, seasickness, tidal currents and cold.

A diver who has to battle a 1-knot current is exerting himself to a significant degree, consuming 2 litres or more of oxygen per minute, and exceeding the limitations of many divers and some equipment. It is virtually impossible for an unassisted diver to make headway against a current of 2 knots or more.

The **temperature** of the water will influence hypothermia, manual dexterity and DCS, as well as the functioning of various pieces of diving equipment (see Chapter 50).

Other environmental hazards include exposure to explosives, dangerous marine creatures, inability to surface (e.g. in caves, under ledges, in kelp, dam outlets, etc.), night diving, etc.

Environmental conditions

- *Wind and weather.*
- *Visibility impairment from sediment, 'white water', night diving, etc.*
- *Adverse water conditions, waves, rips, currents and 'white water'.*
- *Water temperature.*
- *Caves, ledges, kelp, fishing nets.*
- *Dangerous marine creatures.*

DIVE PROFILE AND HISTORY

In the predisposition to DCS, a history of recent dives is of importance. The history may be equally relevant in cases of pulmonary barotrauma, saltwater aspiration and hypothermia. The **log book** may indicate the type of diving performed, or accidents suffered, as may witnesses, relatives, dive buddies, dive leaders and decompression computers (which can be downloaded).

In cases of breath-hold diving, the depth and duration are relevant to hypoxia, as is the practise of prior hyperventilation.

With the use of diving equipment, the depth and duration of each dive are relevant to the development of DCS, especially if recommended stoppages have been omitted, or if the dive is in excess of that suggested in the tables, e.g. beyond the 'limiting line' in the Royal Navy tables, or in the extended exposure tables of the United States Navy.

The **depth** is of importance in nitrogen narcosis and its influence on the behaviour of the diver, and also in other gas toxicity disorders, such as carbon dioxide and oxygen.

The **speed of ascent** if excessive, can contribute to pulmonary barotrauma, DCS, or syncope of ascent. Some recreational divers have been timed to exceed 60 metres per minute in their routine ascents.

Exercise performed during the dive is of considerable importance, especially with rebreathing equipment. With strenuous exercise, the increased oxygen requirement may be in excess of that supplied by the flow rate for the particular gas mixture chosen. With increased oxygen consumption there is also an increased carbon dioxide production, placing an added load on the absorbent system. Thus, the incidence of both hypoxia and carbon dioxide toxicity are increased. Exercise also increases the likelihood of toxicity from high partial pressures of oxygen.

Even with scuba there are problems with increased exercise. First, there is an increased consumption of gas, and this may not be allowed for by the diver who is not wearing or observing a contents gauge to indicate his consumption. Second, there is the problem of an increased resistance to breathing with a resultant dyspnoea and physical exhaustion. Panic and fatigue are then common sequelae.

Dive profile and history

- *Recent dives. Log book. Dive computer.*
- *Depth/duration.*
- *Entry, exit.*
- *Speed of descent, ascent and stoppages.*
- *Pre-dive exercise, alcohol, drugs or food consumption*
- *Problems – seasickness, stress, exertion, illness, etc.*
- *Rescue, first aid and resuscitation attempts.*

Accidents are likely to involve panic and rapid ascent, and inadequate training will then become manifest. The out-of-air situation and the measures used to counteract it, are particular hazards, often resulting in one of the companions making a desperate ascent.

Probably the most neglected information relevant to any accident, and also to the interpretation of the autopsy findings, is that involving the **first aid** and **resuscitation** methods employed. Such activities as rescue procedures, reimmersion, recompression, intubation and respiratory assistance, cardiac massage and oxygen administration should be recorded in detail, as they influence subsequent treatment and autopsy findings.

DIVING EQUIPMENT

Investigation of the diving equipment, being essentially non-medical, was discussed in Chapters 4, 46 and 50.

Diving equipment

- *Facemask, snorkel, buoyancy vest, depth gauge, watch, decompression metre, contents gauge or console on cylinder.*
- *Buddy line, knife, weights, etc. – presence, absence or condition.*
- *Protective clothing.*
- *Functioning demand valve and reducer.*
- *Cylinder condition, valve position (number of turns to close).*
- *Correct gas mixture, pressure and flow measurements.*
- *Absence of contaminants.*
- *Diving profile recorder.*
- *Laboratory testing of equipment (regulators, buoyancy controllers, gauges, etc.).*
- *In-water replica trials.*

In-water trials of the equipment may be conducted by professional investigators (water police, Navy divers), in cases where the cause of death is not clear. The trial is performed by a professional of approximately the same physical stature as the deceased, using a similar dive profile (if deemed safe to do so), and with appropriate safety procedures. Valuable information regarding problems with equipment is often obtained from these trials.

GAS IN THE DIVING AUTOPSY

This is often detected as a swelling of the body, when the pathologist feels the body – or when he cuts into tissues and small gas pockets hiss out at him!

An erect chest and abdominal X-ray is adequate to verify gas in many tissues, but the ideal investigation involves a post-mortem computed tomography (CT) scan, which may demonstrate gas within the tissues and/or heart and blood vessels, post-mortem decompression gas artifact, pneumothorax, other lung lesions associated with pulmonary barotrauma, intracerebral gas, temporal bone, sinus and middle-ear pathology.

It is often stated that gas in the arterial system is due to embolism from pulmonary barotrauma, whereas gas in the venous system is derived from the tissues, and implies DCS. Unfortunately, this simplistic and theoretical explanation is not always valid in practise. In reality, some of the visible gas in a cerebral air gas embolism will be seen in the right atrium and pulmonary arteries, probably due to passing through to the venous system with trapping of gas in the pulmonary circulation.

There are three extraneous causes for the development of gas in the tissues and vascular system, none of which is specifically related to the diving accident ('dysbaric illness'). One involves putrefaction during the decomposition process; another involves the post-mortem decompression artefact; the third is introduced during resuscitation.

Dysbaric illness

Pulmonary barotrauma (PBT) will initially force gas into the pulmonary venous system and then the systemic arterial system, where most of its significant clinical affects are produced (see Chapter 6). With an active circulatory system, the gas passes through the arterial system and collects in the veins and right heart.

Gas also passes into the veins and to the right side of the heart in cases where death has been related to pulmonary interstitial emphysema. This is referred to as 'lymphatic air embolism', and is due to the bubbles moving into the lymphatic system of the lungs, and thence the thoracic duct, from where it passes into the venous system and the right heart.

Gas bubbles from **DCS**, (see Chapters 10–12) also enter the capillary system and pass into the veins. Here, they are likely to be further enlarged by ascent and the higher nitrogen load present in the venous blood and the immediately surrounding tissues. Some will then be trapped in the pulmonary arterial system. Others will pass through this, or a patent foramen ovale or other venoarterial anastomoses, into the arterial system.

Cardiopulmonary resuscitation – which maintains a circulation together with the bubbles – may also result in bubbles distributed through both arterial and venous systems. It also results in gas in the thorax, simulating the effects of pulmonary barotrauma.

Post-mortem decompression gas artefact

A post-mortem decompression gas artefact develops because the diver has absorbed inert gas into the body tissues during the dive. The 'fast' tissues may become more rapidly saturated with the shorter deep dives, and the 'slower' tissues with the shallower and longer dives. If death occurs while there is still an inert gas load in these tissues, i.e. during the dive or soon after it, then the perfusion of blood that is required to remove this gas is no longer available. The gas phase will then develop within the tissues, post mortem. This can occur within minutes, but reaches its maximum within the first few hours after death.

This gas can develop in all tissues and organs of the body, and is easily seen radiologically as gas strips along tissue planes normally unrelated to gas spaces, e.g. the thighs and joints. It is also evident in fluid-containing spaces such as the blood vessels and heart (see Fig. 51.3).

Decomposition and putrefaction

This is due to bacterial action which accelerates after death, due to the absence of the natural defences of the living body. These bacteria digest the soft tissues, reducing them to a fluid consistency. A by-product of this may be a complex mixture of foul-smelling gases that may develop within the various organs of

the body, but especially the gastrointestinal tract. The body then becomes distended and swollen. As a result, buoyancy may increase, and this will often bring the victim to the surface, days or weeks after the incident.

The speed of putrefaction depends on the environmental temperature, and when the water is near freezing, the body may be preserved indefinitely. In water at 20°C, it may only take two or three days, but in 30°C water, 12 hours may be sufficient. Putrefaction occurs more rapidly with obesity, is increased in polluted and stagnant water, and is affected by the time and contents of the last meal. Factors which inhibit putrefaction include saponification and the previous ingestion of antibiotics (often taken as antimalarials).

The chronology of decomposition will vary, but a typical sequence in 20°C water would be as follows:

- 12–24 hours: the skin changes colour from normal to light blue to almost green, initially in the lower body, around the pelvis and groin.
- 24–36 hours: discolouration becomes pronounced, and a marbled pattern develops on the skin. Hydrogen sulphide production causes a characteristic dark green or almost black discolouration, and the blood seeps from the vessels, giving the body a general purplish-black colour.
- 36–48 hours: face and body swell noticeably with the characteristic 'bloated' appearance. Where the skin is loose it may become dramatically swollen, such as around the lips, eyelids and scrotum. Crepitus may be noted.
- 60–72 hours: putrefaction is now widespread, even to the fingers and toes, and the entire body has now changed colour and the facial features are unrecognizable.
- 4–7 days: hair and nails become loose and are easily lost. The skin covering is damaged and also easily lost. Pockets of foul smelling gas form under the skin, escaping through tears. Malodorous coloured liquids may escape from the body's natural orifices.
- 2 weeks plus: the soft tissues will be reduced to a grey, greasy unrecognizable mass. Eventually only skeletal remains indicate the presence of the body.

The composition of the decompositional gas is different from the inert gas bubbles, having higher concentrations of carbon dioxide, hydrogen and/or methane. Decompositional gas tends to be first seen on X-ray in the hepatic/portal circulation, and therefore care should be taken when interpreting gas that is present only in the liver.

Resuscitation

Gas may be introduced into the body from intravascular infusions, active cardiopulmonary resuscitation and endotracheal intubation with positive-pressure respiration. These are all common with diving accidents. Intravascular air, pneumothorax and subcutaneous emphysema can all result from these non-diving activities.

SITE AND TIME OF THE AUTOPSY

To overcome the generalized and disruptive effects of the liberation of gas from the deceased diver who is brought to the surface, i.e. a post-mortem decompression artefact, it is often advantageous to perform the autopsy before the decompression. This is possible when the death has occurred in a recompression facility, where the pathologist can be transferred to the same pressure, and can perform the autopsy adequately under such conditions. This is only likely in saturation complexes, where the environment is such as to sustain life for an indefinite time. It is much more difficult under operational diving conditions where the pathologist himself may be exposed to the hazards of nitrogen narcosis and DCS. The former is likely to influence the pathologist's ability to perform the hyperbaric autopsy, and the latter his enthusiasm!

The results of the autopsy must be assessed in relation to the time between the diving accident and the diver's death, and also between the death and the time of autopsy. If there has been a substantial period of time between the accident, injury, drowning or DCS, and the time of death, then many of the blood gas and biochemical changes will have been nullified, even though the disease progresses to a fatal termination. Thus, the electrolyte changes of drowning and the gas changes associated with DCS may well be totally corrected. The pathological

lesions demonstrated hours or days later may reflect only the previous existence of the disease. A prolonged time between death and autopsy will nullify any electrolyte changes, and putrefaction influences both gas and alcohol production.

The widespread disruptive influence of 'post-mortem decompression artefact' have already been mentioned. This develops in divers who die under pressure, within minutes and hours after they are brought to the surface, or in divers who retain a nitrogen load and die soon after surfacing.

The results of **bacteriological investigations** will also be greatly influenced by the above factors. A time delay between the dive and death from encephalitis due to *Naegleria* is expected in divers who have exposed themselves to contaminated freshwater areas. Culture for organisms, such as marine *Vibrio sp.*, should also be performed in hypertonic saline. Unfortunately, many bacteriologically positive results may reflect contamination as opposed to actual aquatic infection. This is so with organisms such as *Clostridium tetani* and *C. welchii.*

AUTOPSY PROCEDURE

This requires specific pathological expertise and diving medical knowledge. Photographs, chest and soft-tissue thigh radiographs are taken first. A CT scan of the body, if available, and showing the pulmonary and cerebral vessels, is of greater value. The presence and distribution of gas in tissues, vessels and body spaces is relevant.

General

An autopsy on a diver who has died as a result of a diving accident should be undertaken by a pathologist who has had experience in these investigations. The presence of a diving physician is an advantage. Otherwise, the investigation may be incomplete and misinterpreted.

The deceased is examined prior to cleaning or removal of foreign bodies or clothing. Damage may result from either the cause of the accident, e.g. shark attack, propeller, or from the search and recovery, e.g. from a grappling hook or line.

Disruption of the skin and subcutaneous tissues is likely to result from post-mortem trauma (during the rescue and recovery), putrefaction and water-logging, causing its detachment during rescue, or by marine animals. A skin examination may show the duration of exposure (cutis anserine or 'goose flesh'), haemorrhagic or Tardieu spots of asphyxia, or signs of DCS (see Chapter 11). Trauma to the skin from marine animals may either be caused after death or be instrumental in the death. Small crustaceans and 'sea lice' may cause extensive loss of soft tissues in water, within a day or two of death. If there are marine animal lesions, these are usually easy to diagnose.

Shark bites cause multiple crescentic teeth marks, and tearing wounds illustrate the direction of the attack and the size of the animal. Teeth particles in the wound will assist in identifying the species of shark. Barracuda bites are clean-cut excisions. Post-mortem feeding by predators is common, and is indicated by the absence of haemorrhage in the tissues (i.e. circulation had ceased).

Coelenterate injuries may be recognizable by the number and distribution of the whip-like marks, which partly fade after death. An accurate identification of the type of coelenterate or jellyfish is possible by taking a skin scraping for examination of the nematocysts by a marine biologist. Nematocyst identification from jellyfish injury on the skin may remain possible for many days, even though the actual lesion may decrease in its florid appearance – not only between the injury and the time of death, but also post mortem. Immunoglobulins titres to coelenterate venoms may take several days to develop.

Fish spines can be demonstrated macroscopically and on histology. Cone shells may leave a tiny harpoon puncture, while blue-ringed octopus bite appears as a small angular nick or a single haemorrhagic bleb. The sea snake bite will show two or more fang marks with surrounding teeth impressions. Where an octopus has held a victim, multiple bruising from round sucker pads is seen along the extent of the tentacle contact. Squid and cuttlefish may leave similar circular marks and incisions.

The skin of the fingers may show the effects of immersion, i.e. pale and wrinkled with the so-called 'washerwoman's skin'. There may be lacerations from

where the diver has attempted to clutch barnacles, coral, etc. Injury inflicted by aquatic animals in the early post-mortem period is especially seen on protuberances such as the fingers, nose, lips and eyelids, if these are not protected by equipment.

There is often evidence of **vomitus**, which may be either causative or an important part of the sequence of events. In cases in which seawater has been aspirated there may be white, pink or blood-stained foam in the nose and mouth. Where the patient has been brought to the surface following a significant exposure at depth, there may also be distention of the gas spaces within the body resulting in a distended abdomen, faecal extrusions, etc.

External evidence of barotrauma is important in ascertaining the sequence of events. An unconscious diver, or one who has not been able to equalize his physiological or equipment gas spaces during submergence, will have evidence of barotrauma, as described in earlier chapters. Thus, there should be an examination for evidence of mask or face squeeze with haemorrhage into the conjunctiva, ear squeeze with haemorrhage or perforation of the tympanic membrane, and suit squeeze with long whip-like marks underneath the folds of the protective clothing. Total body squeeze may occur when a standard diving rig is being used. Middle-ear haemorrhage – long considered a sign pathognomonic of drowning – is merely evidence of descent while alive. It is verified by otoscopy.

Pulmonary barotrauma of ascent may be inferred by evidence of subcutaneous emphysema localized to the supraclavicular and cervical areas. If this finding is generalized over most of the body, then it is more likely to be due to the liberation of gas following death, i.e. 'post-mortem decompression artefact'. It should also not be confused with clinical DCS or putrefactive changes. Where the diver has suffered from DCS, there is often a reddish or cyanotic distention of the head, arms and upper trunk. The body will therefore have an appearance not unlike that seen in clinical cases of the superior vena cava syndrome.

Autopsy technique check list

External examination and photographs:
- *trauma*
- *barotrauma*
- *marine animal injuries*

Chest and soft-tissue thigh X-rays

Long-bone X-rays for dysbaric osteonecrosis

CT scan of head, neck and thorax
- *gas in vessels and heart*
- *gas in tissues, pleural space and mediastinum*
- *brain, paranasal sinuses and middle-ear pathology*

Demonstration of cerebral air embolism

Opening of thorax under water
- *pneumothorax*
- *mediastinal emphysema*
- *gas in chambers of heart or coronary vessels*
- *examination of lungs*

Abdomen
- *intravascular bubbles*
- *infarcts*

Bone – dysbaric osteonecrosis

Temporal bone
- *barotrauma*
- *decompression sickness*
- *haemorrhage*

Bacteriology, haematology, biochemistry, histology, toxicology

Radiology

Before any invasive technique is carried out, radiology should be performed with under-penetrated films to show evidence of gas within the tissues (see above). X-rays may be performed for bone lesions and dysbaric osteonecrosis in the shoulders, hips and knees. In the case of marine animal injuries, there may also be evidence of bone damage and foreign bodies, such as shark teeth and fish spines.

Upper body (head, neck and thorax) CT scanning is of value in detecting gas in the tissues, pleural

cavity, mediastinum, cerebral and other vessels, or pathology in the paranasal sinuses, middle ears and brain.

Brain

Gas in the cerebral circulation may be very relevant to the cause of death, or it may be an artefact. As a cause of death, it results from embolism consequent to PBT or (antemortem) DCS. Gas artefact may occur as a result of post-mortem decompression. Incision of the scalp and removal of the calvarium, or dissection of the neck, may introduce artefactual 'air bubbles' into the superficial cerebral veins and venous sinuses, but these are not of diagnostic importance.

The technical difficulties of opening the skull under water are far outweighed by the advantages. A technique to demonstrate air emboli is of paramount importance in the investigation of diving accidents.

The circle of Willis and its distribution arteries are the areas to be examined meticulously for evidence of the significant air bubbles. The customary technique is to perform the craniotomy and cervical incisions under water, with a constant flushing of the water, avoiding artefactual air being introduced during this or during dissection of the neck. If bubbles are found, they should be photographed *in situ*, or after clamping the carotid arteries and cutting proximal to the clamp, in order to obtain a better exposure.

Alternatively, the head may be submerged and suspended into a mortuary sink with the body resting on shelving. The scalp is first incised above the water, then reflected under water. The head is again elevated, and the skull cut with a vibrating saw, taking care not to damage the dura. The head is then submerged for a second time, the calvarium dislodged, the dura incised, and the brain removed under water. The brain may then be placed directly into a formalin container beneath the water; the formalin and brain then remain when the sink is drained. No air enters the cerebral circulation during the removal of calvarium and brain, and any gas bubbles present when the brain is examined after fixation would have been present at the time of death. Alternatively, a C.T. scan is easier.

Sections of the brain should include the 'watershed' area of the frontal cortex 2.5 cm from the mid-line.

Spinal cord

Cord removal may be indicated, and examined after fixation. Ante-mortem bubble formation may produce microscopic haemorrhages and infarcts, which are most easily recognized in the spinal cord. After fixation a transverse section of the spinal cord should be taken from each segment and parasagittal longitudinal sections between each transverse section. Air may enter the spinal venous plexus during removal of brain, abdominal and thoracic organs, so that finding gas bubbles in this area in the final stages of the autopsy may not be significant. Some neuropathologists use the *Marchi stain* to demonstrate less obvious lesions.

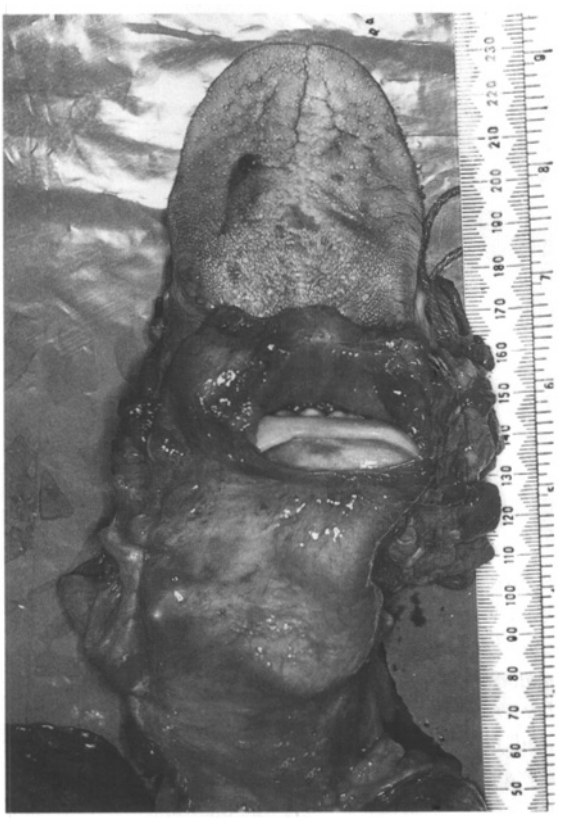

Figure 51.1 *Dental plate swallowed during amateur dive.*

Temporal bones

In diving accidents, and in diving pathology, the temporal bone and its associated middle-ear and mastoid air-cell cavities are of special importance. Not only will haemorrhage occur in this area, due to barotrauma of descent, but also there are specific disorders causing disorientation which may be instrumental in the diving accident. These include such injuries as tympanic membrane perforation, middle-ear barotrauma of descent and inner-ear disturbances either from haemorrhage or round window rupture. It is thus important to record the

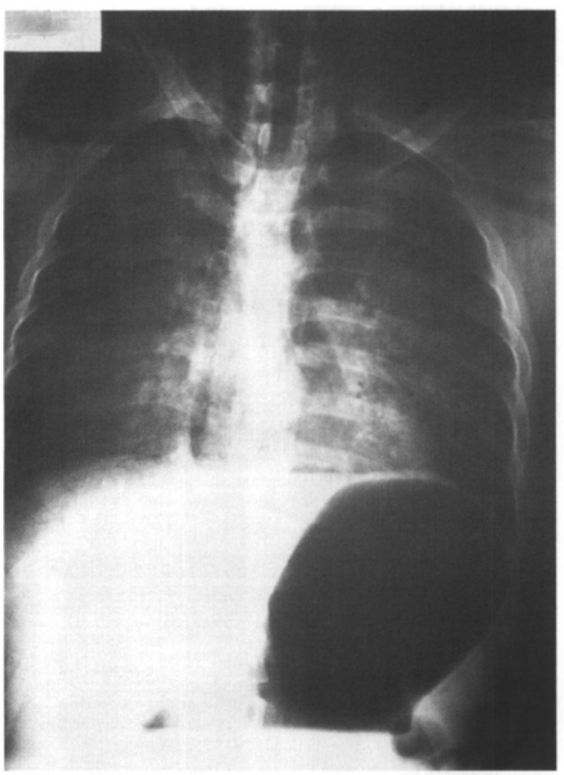

Figure 51.2 *Post-mortem decompression artefact – chest X-ray. A large amount of gas is seen within the heart chambers and the left pulmonary artery, as well as in the major vessels of the neck and the subclavian vessels bilaterally. Subcutaneous emphysema is seen in the chest wall. The gaseous distension of the gastric fundus is well demonstrated. These characteristics are commonly found in divers and caisson workers who die while under pressure and who are then brought to the surface.*

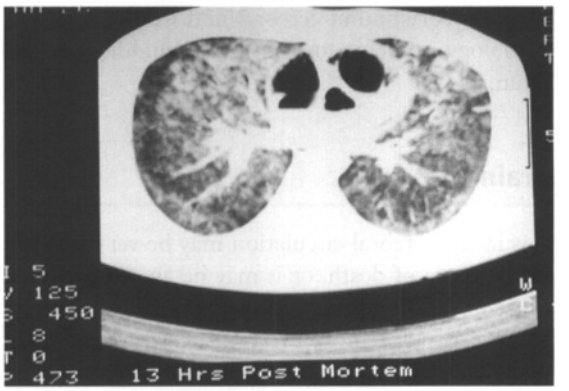

Figure 51.3 *Computed tomography (CT) scan of the chest. This is an axial CT scan through the heart. On these 'lung settings', diffuse changes are seen throughout both lung fields, particularly anteriorly. The gas within the heart chambers is well visualized.*

Figure 51.4 *Tracking of intravascular bubbles in acute DCS.*

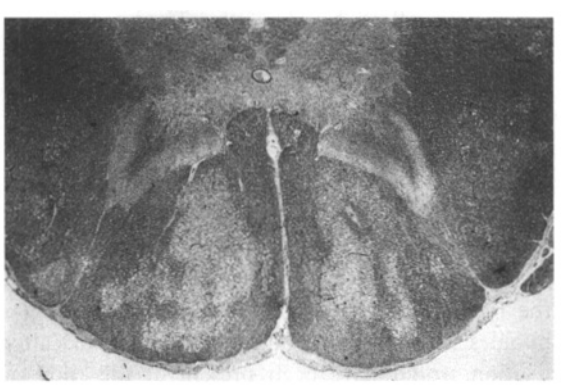

Figure 51.5 *Cord at T8 in acute DCS – 30 hours.*

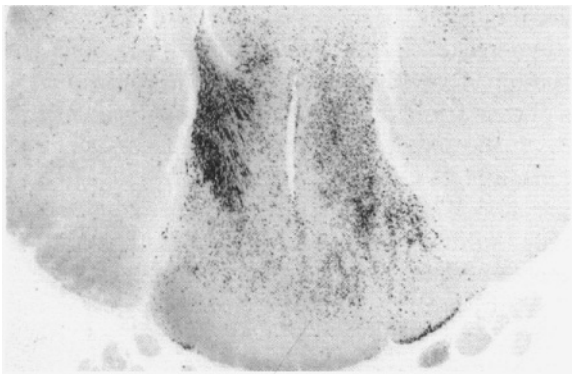

Figure 51.6 *Marchi stain of cord in 'fit diver'. No history of DCS but could have been between 10 and 90 days after a 'silent' incident.*

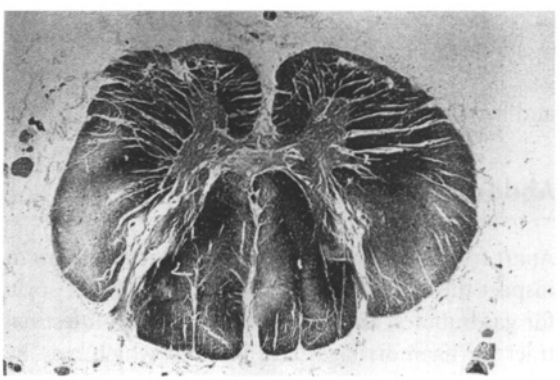

Figure 51.7 *T8 in 'recovered bends'.*

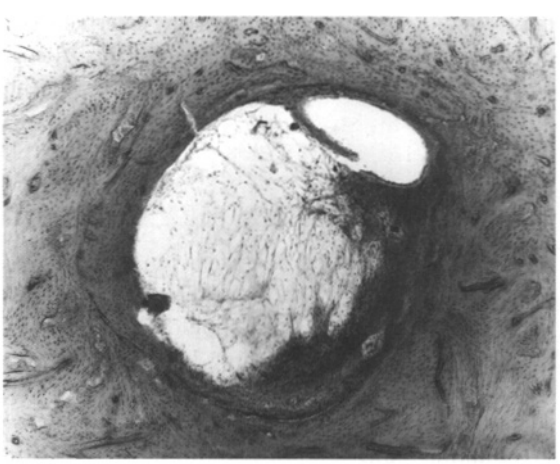

Figure 51.8 *Temporal bone. Round window fistula.*

appearance of the tympanic membrane on otoscopy, the presence of any fluid within the middle-ear cavity, and finally a histological examination of the temporal bones, ear and mastoid cavities, performed by a specialist pathologist.

Cardiothoracic systems

Various techniques have been established for demonstrating a gas aetiology for death, with X-ray and/or CT scans before autopsy being most useful in this respect. The further investigation can be either of the following.

Some pathologists elevate the chest with a block under the shoulder and carefully open the chest between the major neck vessels. The pericardial sac is opened, after which blood (? and gas) is aspirated from the superior point in each of the four chambers, measured and possibly analysed. Unfortunately, not all aspirations are performed with an 'air-tight' technique.

Gas collections can be aspirated, preferably under water or through a water seal. The chest can be opened beneath water in the bath or a water seal, so that small amounts of gas in the pleural cavities may be detected. Similarly, the pericardium can be opened and gas sought in the pericardial space and visceral pericardial vessels. The heart is removed, and each chamber opened under water. Unless the procedure is carried out under water, intracardiac gas can be missed.

Gas can be demonstrated without the necessity of having the whole body immersed, by immersing only the heart and lungs underwater. This is achieved by performing a neck incision extending down the midline from the sternal notch to the midpoint between the xiphisternum and umbilicus, and then to each of the anterosuperior iliac spines. The skin and the muscles over the chest and the skin over the abdomen are then widely reflected by undercutting, and the trough between the body and the skin filled with water. The heart cavities can then be opened, noting the presence of any abnormal gas collection. Some pathologists may not relish performing any procedures under water, as the impaired visibility may increase the risk of personal injury and infections.

If there is doubt about drowning, **blood samples** should be taken from the vena cava and the pulmonary veins. A routine blood sample should always be taken, either from subclavian vessels or later from a femoral vein. Blood can be taken from the right and left ventricles for whole-blood chloride measurements – while this technique is controversial in unrescuscitated saltwater drowning, it can produce positive results.

In PBT the voluminous lungs may bulge out to the thoracic cavity, and may have interstitial emphysema, subpleural blebs and mediastinal emphysema. Mediastinal tissues and the soft tissues of the neck should be examined for interstitial emphysema. Rupture of alveolar walls may be associated with widespread intra-alveolar haemorrhages. Interstitial emphysema recognized in sections of lung and acute intra-acinar emphysema may be seen in cases of PBT. Haemorrhage, varying in intensity from lobule to lobule, may be also found when PBT has occurred.

PBT may be identified at post-mortem examination by demonstrating an air leak from the lungs to the pleura, interstitial tissue or blood vessels. The lungs are inflated using compressed air or oxygen and a cuffed endotracheal tube, to a pressure of 2.5 kPa (25 cm H_2O), producing slight over-inflation. With the tube clamped, the lungs are then submerged beneath water, using a coarse metal grill to produce complete submersion. Using an inverted water-filled glass measuring cylinder, some estimate should be made of the volume of air escaping per minute into veins or from pleura. The tube is then removed, the lungs separated, weighed, reinflated at similar pressure with buffered formalin, and retained for examination after fixation.

The pathological cause (e.g. pleural scarring, evidence of chronic asthma) of the PBT may also be detected. Acute effects of asthma, although of great importance clinically, will not be evident pathologically within 24 hours. Mucous secretion and eosinophils will usually be present in longer-lasting disease, and basement membrane thickening with chronic asthma. The apex of the lung is a common site for unrecognized bullae which may predispose to PBT.

The appearance of the lungs may be characteristic for certain diving diseases. In drowning, the lungs are usually heavier than normal, with oedema and haemorrhage in the lung tissue and frothy red sputum in the airways. In freshwater drowning, if sufficient time has elapsed between rescue and death, the lungs may appear 'dry'. Evidence of marine foreign bodies, diatoms, etc. should match those of the area where death occurred. However, this verifies only the site of immersion before death – not the cause of death.

With severe DCS the lungs may be engorged and distended with blood and intravascular gas. These effects may be complicated by the addition of disseminated intravascular coagulation.

The presence of a patent foramen ovale may indicate the reason for death from 'paradoxical' emboli derived from decompression in the venous system and bypassing the pulmonary filter to end up in the brain.

In cases of underwater blast, the tissues with a gas/fluid interface are most affected, with shredding and haemorrhages along the airways.

Abdomen

Apart from the above observations, it is necessary to inspect the inferior vena cava and descending aorta for gas bubbles, and to examine the gastrointestinal tract for haemorrhages and infarcts, which may be related to either emboli or DCS. In cases of underwater blast, most of the haemorrhages occur between a gas and fluid interface, and the abdomen and gastrointestinal tract are especially involved. Under these conditions, there are seldom any external signs of violence on the skin or subcutaneous tissue, despite severe haemorrhages occuring within the bowel itself.

Histology

Routine blocks should be taken from the brain, spinal cord, heart and abdominal viscera. After reinflation and fixation, sections should be taken from the pleural surface of each lung lobe, and sections also across each segmental bronchus, including adjacent lung parenchyma.

Temporal bone studies and those for dysbaric osteonecrosis are specialized investigations, and best

referred to a specialist pathologist. They may indicate recent or past diving accidents.

Samples should be taken and kept, even if the pathologist does not intend to examine them, himself. They will be valuable in future investigations

OTHER LABORATORY EXAMINATIONS

Urine samples may be tested for drugs and fibrin degradation products (FDP). Evidence for antemortem intravascular blood coagulation and fibrinolysis may occur when there has been intravascular bubble formation. A blood sample should be examined routinely for alcohol, carbon monoxide and other drugs. Blood from the vena cava and pulmonary veins may be centrifuged, the serum separated and examined for haemolysis, and the osmolarity of each sample measured.

It is necessary to test for alcohol and drugs in blood and tissues, as contributors to the diver's death. The alcohol level must be correlated with the post-mortem interval to assess the putrefactive neoformation of this substance. Other biochemical investigations which may be relevant include serum electrolytes, performed on both the right and left side of the heart, serum haemoglobin and haptoglobin levels, and especially carboxyhaemoglobin estimation. Various assays may become possible to determine marine animal toxins, with some immunoglobulin analyses already available from specialized laboratories if death has been delayed.

Histological examination of the blood, the tissues, kidney and the bone marrow, as well as the material in the respiratory and gastrointestinal tracts, is necessary for the identification of diatoms and aquatic organisms. This information should be compared with similar investigations on a sample of water taken from areas in which the diver could have died.

CONCLUSIONS

There needs to be a close integration of investigations performed by the investigating (police) divers, the diving physician and the pathologist, to obtain information and then to assess it. There is little information on the pathology and autopsy techniques associated with diving accidents.

In most countries there are no diving units that are analogous to those which investigate aircraft accidents. Thus, the usual practice is for the investigation of the diving accident to be performed by a police inspector who is moderately ignorant in the technical aspects of diving equipment, a local clinician who has no training in diving medicine, and a pathologist who is overworked and not amenable to varying his standardized and venerable techniques. The result is often either a mistaken diagnosis of DCS, air embolism or drowning, without an explanation of the causative sequence of events. There is therefore a loss of valuable information, and a failure to learn from the mistakes of the past.

Adequate investigation that will result in useful information requires close cooperation between the divers, the diving physician, the police and the pathologist.

ACKNOWLEDGEMENTS

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52

Medical standards for snorkel divers

CARL EDMONDS

INTRODUCTION

All prospective divers should first become adept at snorkelling. This expedites the swimming to and from the dive site, and also extends the distances that a surface swimmer may achieve with greater ease. Snorkelling acts as an emergency activity to be used when more complex diving equipment fails, allowing the diver to remain on the surface, breathing relatively easily. Snorkelling is much more widespread, less expensive, less complex and a less regulated activity, but this does not mean that it is always safe.

Problems with basic equipment are dealt with in Chapter 4, the ocean environment in Chapter 5, specific medical problems in Chapter 61, and general problems elsewhere. Because many snorkellers are children, Chapter 58 may also be relevant.

Like scuba diving, there are reasonable standards of physical and medical fitness that should be applied, but these are usually ascertained by completing a basic questionnaire. Even those who 'fail' the questionnaire can still snorkel under more restricted conditions and with supervision.

Ensuring that the potential snorkeller is capable of physical exertion and possesses aquatic skills sufficient to survive, is axiomatic. Also there should be no increased likelihood of impairment of consciousness – which is far more dangerous at sea than in terrestrial environments. If the snorkeller intends to submerse himself, then the increased risks of barotraumas and breath-holding become relevant.

A typical questionnaire, presented in *Diving Medicine for Scuba Divers* was designed for schoolchildren who wished to experience snorkelling, but is now used by commercial dive boat operators who take tourists to the Great Barrier Reef.

MEDICAL CHECK LIST FOR SNORKELLERS

A typical check list might be as follows:

Have you ever had:

1	Any cardiovascular disease? (heart, blood pressure, blood, etc.)	Yes	No
2	Any lung disease? (asthma, wheezing, pneumothorax, TB, etc.)	Yes	No
3	Any fits, epilepsy, convulsions or blackouts?	Yes	No
4	Any serious disease? (such as diabetes)	Yes	No
5	Serious ear, sinus or eye disease?	Yes	No
6	Any neurological or psychiatric disease?	Yes	No

Over the last month have you had any:

7	Operations, illnesses, treatment?	Yes	No
8	Drugs or medications?	Yes	No

9 If female, are you pregnant?	Yes	No
Can you:		
10 Swim 500 metres without aids?	No	Yes
11 Swim 200 metres in 5 minutes or less?	No	Yes
12 Equalize your ears when diving or flying?	No	Yes

Name:

Date of birth:

Address:

Signature: (If under 16 years, guardian to sign)

Note: If the candidate indicates an answer in the left-hand column, then further investigation or action (limitation) is required before snorkelling is considered as safe.

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53

Medical standards for recreational divers

ROBYN WALKER

INTRODUCTION

To certify that a person is fit to be taught diving ideally implies that he is physically fit, medically healthy and psychologically stable. Humans are clearly not adapted naturally to dive; if they were they would possess gills. Yet recreational scuba diving is one of the fastest growing leisure sports, and medical practitioners are required by their patients to be capable of furnishing an opinion as to their medical fitness to dive. This chapter provides the medical practitioner with a basic synopsis of recreational diving medical standards.

Many physicians limit their consultation to the exclusion of diseases rather than performing a comprehensive assessment based upon knowledge of the different aquatic environments, the myriad of diving equipment and diving gas mixtures available for use and the skills required for the diver's safety.

Why have medical standards? The imposition of medical fitness standards for diving in some part has arisen in response to a perceived risk of serious injury associated with the activity. In many countries, regulations were first developed to protect professional divers and their employers, and this practise of risk management resulted in only the very fit being allowed to enter the profession.

With the advent of a boom in recreational diving, some countries have followed the pathway of mandatory diving medical examinations for recreational divers. This is a prescriptive approach aimed at minimizing the risk accepted by the diving instructor agency, and the candidate has little opportunity to participate in the risk assessment. Other countries have no mandatory requirement for recreational dive medicals; the candidate therefore assumes most of the risk, but is denied the opportunity to discuss this risk with an informed medical practitioner.

Medical standards for recreational diving candidates leave greater room for flexibility than professional diving standards, and are harder to define in absolute terms. The recreational diver has the choice of depth and duration, can avoid adverse environmental conditions, and can usually avoid strenuous exercise. It is possible to consider voluntary restrictions both on the exposure to different diving environments and their diving practices (depth limits, equipment used, etc.).

Whichever system is in place, there are four important questions to consider when assessing fitness to dive:[1]

1 Does the condition or disease affect the personal in-water safety of the diver?
2 Does the disease or condition affect the safety of the other divers who might have to lend assistance?
3 Will diving exacerbate the condition or disease?
4 Will diving result in any long-term sequelae in the presence of the condition or disease?

Different countries have different approaches to recreational diving medical requirements. In the USA, where the Professional Association of Dive Instructors (PADI) is the largest recreational diving instructional agency, no mandatory medical examination is required. PADI issues to each trainee a scuba diving medical statement that informs the new diver of some of the potential risks involved in scuba diving, along with a medical questionnaire.

If any of the questions are answered in the positive, the candidate must seek the advice of a physician before continuing with the training. If all questions are answered in the negative, then no further medical review is required. A disadvantage with this approach is that the trainees may not be truthful as they are aware a positive answer could disqualify them from diving. There is also no clinical data recorded and no mechanism for reviewing the medical status of the divers as they age, or if they suffer from a new medical condition.

In the UK, the British Sub Aqua Club (BSAC) is the dominant force in recreational diving. Under their rules scuba instruction must not commence until the candidate holds a valid certificate of fitness to dive. The medical is generally valid (unless otherwise endorsed by the medical practitioner) for five years up until the age of 40 years, for three years between the ages of 40 and 50 years; an annual medical is required thereafter. A questionnaire is administered and the examination is in accordance with a specified format.

Standards

Australia have produced an Australian Standard AS4005.1 which details the medical criteria to be addressed by individuals who wish to undertake recreational scuba diving. The medical should be conducted by a medical practitioner who has done an approved course of training for medically examining candidates for recreational dive training. The medical examination is only required once before undertaking scuba training, and there is no requirement for follow-up medicals as the diver ages or suffers from a new medical condition. There is, however an option to recommend when such a medical should be performed, on the diving medical certificate.

The physician should clarify, on the medical certificate whether the candidate is medically fit to dive (physician's decision and responsibility) or whether merely advice regarding risk analysis has been given (thus becoming the candidate's responsibility).

In an ideal society, physicians can make recommendations, but not demand obedience from their patients. People do have a right to dive, as long as they do not involve or endanger others. If some candidates decide 'to dive', it is possible that they will ignore the advice and prove the physician incorrect. Diving candidates also have the right to seek alternative opinions from other diving physicians, and sometimes medical colleagues do not share these hard lines. The candidate seeks advice, and if this is given accurately and in detail, he will be more likely to agree with the physician.

A great deal of diving medical practice involves reviewing divers after their local physician has told them that they cannot dive again. Alternately, a great deal of time and effort is required to convince a candidate with a potentially life-threatening disability not to dive if he has been 'passed' by another doctor.

Medical regulations must be explained rationally. Stating that diving should not be allowed while

taking drugs epitomizes the opposite attitude. There is no evidence available to suggest that anyone who is taking allopurinol for gout should not dive. The same situation applies to antibiotics for acne, or even chloroquine for malaria prophylaxis.

Medical fitness to dive recreationally should be related to the environmental situation. In some circumstances, no one is fit to dive; in other circumstances, diving is safe for most people. It is often possible to tailor the type of diving to the person being examined.

> *Medical standards come in black and white; people come in shades of grey.*

In many cases, it can be stated that the recreational diver is fit in accordance with specific standards such as those produced by the Australian Standards Association, the National Oceanographic and Atmospheric Administration (NOAA) etc. or the United Kingdom Diving Medical Guidelines. In other cases, it has to be stated that the candidate is fit to dive, but under specific conditions and with specific recommended limitations. These limitations may be due to such conditions as physical disability, obesity, migraine, age, otological problems and visual impairment. Concessions can be more often made for the experienced diver, as compared to the novice.

The following discussion on various aspects of a diving medical evaluation is loosely based on a history and examination form devised by the authors to fill their needs (Figs. 53.1–53.3).

PHYSICAL FITNESS

This subject is still a contentious one. Often we are faced with diving candidates who have demonstrated their physical prowess by excelling in other sports, but who are then informed that they are not medically fit to dive. Thus a champion swimmer, who has active asthma, may well feel that his physical fitness qualifies him for scuba diving. This illustrates a major maxim. Even though a candidate can be very physically fit in one sport, e.g. marathon running, this proves only his fitness for that sport; it does not imply fitness for diving.

Aquatic conditions can change dramatically within a short period of time. The dive may have commenced in good weather, flat seas and no current, yet, it is possible to surface 40 minutes later some 100 metres away from the boat in stormy conditions with a 2-metre swell and over 1 knot of current. The prospective dive candidate must have sufficient reserves to cope with such conditions, and be able to render assistance to his buddy if required. Diving can involve unpredictable and extreme workloads in the unplanned emergency situations produced by currents, tides and changing weather conditions.

Some idea of the candidate's fitness may be assessed by inquiring into his normal daily physical and sporting activities. Those who can swim 200 metres without fins in 5 minutes or less are far more likely to succeed in diving than those who cannot achieve this relatively undemanding standard. It also follows that the dive instructor agency must take some responsibility for ensuring all dive candidates can demonstrate adequate in-water skills before progressing to open-water dives.

> *Physical fitness does not imply fitness for scuba diving.*

PSYCHOLOGICAL AND PSYCHIATRIC CONSIDERATIONS

A full psychiatric assessment is impossible to perform in the limited time of a routine diving medical examination, but some clues can be gleaned. A personality characterized by a tendency to introversion, neuroticism and global mood disturbance is more likely to panic (37 per cent of recreational diving deaths are due to this diver error). Useful insight may be gained by direct questions regarding the motivation to dive, a history of claustrophobia, hydrophobia and previous water sports. In order to respond appropriately during a diving emergency, the candidate will need to display emotional maturity and stability.

DIVING MEDICAL CENTRE

(MEDICAL HISTORY) TO BE FILLED IN BY CANDIDATE

1. SURNAME OTHER NAMES | 2. DATE OF BIRTH

3. ADDRESS PHONE | 4. SEX: MALE/FEMALE

5. MARITAL STATUS: SINGLE ☐ DIVORCED ☐ MARRIED ☐

6. DIVING SCHOOL:

7. OCCUPATION:

8. HAVE YOU ANY DISEASE OR DISABILITY AT PRESENT? NO ☐ YES ☐ NAME OF CONDITION:

9. ARE YOU TAKING ANY TABLETS, MEDICINES OR OTHER DRUGS? NO ☐ YES ☐ TYPE OF DRUG:

HAVE YOU EVER HAD OR DO YOU NOW HAVE ANY OF THE FOLLOWING:

	NO	YES	NOTES ON HISTORY
10. DO YOU WEAR GLASSES			
11. DO YOU WEAR CONTACT LENSES			
12. ANY HEART DISEASE OR BLOOD DISORDER			
13. HIGH BLOOD PRESSURE			
14. ABNORMAL SHORTNESS OF BREATH			
15. BRONCHITIS OR PNEUMONIA			
16. PLEURISY OR SEVERE CHEST PAINS			
17. COUGHING UP BLOOD			
18. T.B. (CONSUMPTION)			
19. CHRONIC OR PERSISTENT COUGH			
20. PNEUMOTHORAX (COLLAPSED LUNG)			

	NO	YES	NOTES ON HISTORY
21. ASTHMA OR WHEEZING			
22. OTHER CHEST COMPLAINT OR CHEST INJURY OR OPERATION ON CHEST, LUNGS OR HEART			
23. HAY FEVER			
24. SINUSITIS			
25. ANY OTHER NOSE OR THROAT TROUBLE			
26. DEAFNESS OR RINGING NOISES IN EAR			
27. DISCHARGING EARS OR OTHER INFECTION			
28. OPERATIONS ON EARS			
29. EYE OR VISUAL PROBLEMS			
30. DENTAL PROCEDURES (OVER LAST MONTH)			
31. FAINTING, BLACKOUTS, FITS OR EPILEPSY			
32. SEVERE HEADACHES OR MIGRAINE			
33. SLEEPWALKING OR SLEEP DISTURBANCES			
34. SEVERE DEPRESSION			
35. CLAUSTROPHOBIA			
36. ANY PSYCHIATRIC ILLNESS			
37. KIDNEY OR BLADDER DISEASE			
38. DIABETES			
39. INDIGESTION OR PEPTIC ULCER			
40. VOMITING BLOOD OR RECTAL BLEEDING			
41. RECURRENT VOMITING OR DIARRHOEA			
42. JAUNDICE OR HEPATITIS			
43. MALARIA OR OTHER TROPICAL DISEASE			
44. SEVERE LOSS OF WEIGHT			
45. VENEREAL DISEASE			
46. AIDS OR HIV-POSITIVE			
47. HERNIA, RUPTURE OR PILES			
48. ANY SKIN DISEASE			
49. ANY REACTION TO DRUGS OR MEDICINES			
50. ANY OTHER ALLERGIES			

	NO	YES	NOTES ON HISTORY
51. UNCONSCIOUSNESS			
52. CONCUSSION OR HEAD INJURY			
53. ANY MAJOR JOINT OR BACK INJURY			
54. ANY FRACTURES (BROKEN BONES)			
55. ANY PARALYSIS OR MUSCULAR WEAKNESS			
56. DENTURES			
57. MOTION SICKNESS (CAR, PLANE, SEA)			
58. DO YOU SMOKE			
59. APPROX. NUMBER OF CIGARETTES A DAY			
60. HAVE YOU EVER BEEN REJECTED FOR INSURANCE			
61. HAVE YOU EVER BEEN UNABLE TO WORK FOR MEDICAL REASONS			
62. HAVE YOU EVER BEEN ON A PENSION			
63. HAVE YOU ANY EAR PROBLEMS OR DISABILITY WHEN FLYING IN AN AIRCRAFT			
64. HAVE YOU ANY INCAPACITY DURING PERIODS			FEMALES ONLY
65. ARE YOU NOW PREGNANT			
66. HAVE YOU BEEN IN HOSPITAL FOR ANY REASON			
67. HAVE YOU HAD ANY OPERATIONS			
68. HAVE YOU HAD ANY OTHER ILLNESS OR INJURY NOT MENTIONED IN THIS LIST			
69. APPROX DATE OF LAST CHEST X-RAY	NORMAL/ABNORMAL		

Additional history (taken by physician).

Family history of cardiac disease.

Personal history of lipid/cholesterol abnormality.

Routine exercise exposure.

Figure 53.1 *Medical history.*

DIVING MEDICAL HISTORY (TO BE COMPLETED BY CANDIDATE)

1. CAN YOU SNORKEL (FREE DIVE)	
2. APPROX. DATE OF FIRST SCUBA DIVE	
3. APPROX. NUMBER OF SCUBA DIVES	
4. GREATEST DEPTH OF ANY DIVE	
5. LONGEST DURATION OF ANY DIVE	

HAVE YOU EVER HAD ANY OF THE FOLLOWING

DISORDERS, DURING OR AFTER DIVING (DIVERS ONLY)

	NO	YES	
6. SEVERE EAR SQUEEZE			
7. RUPTURE OF EARDRUM			
8. DEAFNESS			
9. GIDDINESS OR DIZZINESS			
10. SEVERE SINUS SQUEEZE			
11. SEVERE LUNG SQUEEZE			
12. RUPTURED LUNG (BURST LUNG)			
13. EMPHYSEMA			
14. PNEUMOTHORAX			
15. AIR EMBOLISM			
16. NITROGEN NARCOSIS			
17. DECOMPRESSION SICKNESS (BENDS)			
18. NEAR-DROWNING			
19. SEVERE MARINE ANIMAL INJURY			
20. OXYGEN TOXICITY			
21. CARBON DIOXIDE TOXICITY			
22. DYSBARIC OSTEONECROSIS (BONES)			
23. ANY OTHER DIVING ACCIDENTS			

I CERTIFY THAT THE ABOVE INFORMATION IS TRUE AND COMPLETE TO THE BEST OF MY KNOWLEDGE

SIGNED ..... DATE .....

Figure 53.2 *Diving medical history.*

MEDICAL EXAMINATION TO BE COMPLETED BY MEDICAL PRACTITIONER

1. PHYSIQUE	2. HEIGHT	3. WEIGHT	4. VISION	5. B/P	6. URINALYSIS
GOOD	ins	lbs	R6/ CORR 6/		ALBUMEN
AVERAGE	cm	kg	L6/ CORR 6/		GLUCOSE
POOR					

7. CHEST X-RAY, IF INDICATED	8. RESPIRATORY FUNCTION TEST	COMMENTS:

DATE..... VITAL CAPACITY..... F. E. V. $_{1.0}$.....

PLACE..... RESULT..... PERCENTAGE.....

9. AUDIOMETRY

FREQUENCY (Hz)	250	500	1000	2000	4000	6000	8000	COMMENTS:
LOSS IN dB (R.E.)								
LOSS IN dB (L.E.)								

CLINICAL EXAMINATION	NORMAL	ABNORMAL	NOTES ON ABNORMALITIES
10. NEUROLOGICAL SYSTEM			
11. CARDIOVASCULAR SYSTEM			
12. ECG (IF INDICATED)			
13. ENT SYSTEM			
14. EUSTACHIAN TUBE FUNCTION			
15. SHARPENED ROMBERG SCORE			
16. RESPIRATORY SYSTEM			
(AUSCULTATION WITH HYPERVENTILATION)			
17. MUSCULOSKELETAL			
18. ENDOCRINE SYSTEM			
19. PERIPHERAL VASCULAR/LYMPHATIC			
20. OTHER ABNORMALITIES			

FITNESS TO DIVE:

	YES. PROFESSIONAL DIVING		YES. RECREATIONAL DIVING
	YES (QUALIFIED). RESTRICTIONS:		
	TEMP. UNFIT. REASON:		
	PERM. UNFIT. REASON:		

SIGNATURE OF PHYSICIAN..... NAME..... DATE.....

ADDRESS.....

INVESTIGATIONS REQUIRED.....

NEXT MEDICAL ASSESSMENT DATE.....

Figure 53.3 *Physical examination.*

The diving instructor is in a better position to assess the candidate's psychological suitability for diving during the period of instruction.

Freedom from gross psychiatric disorders is essential. There should be no evidence of: anxiety states, depression, claustrophobia or agoraphobia, psychoses or any organic cerebral syndromes (see Chapter 40). A history of antidepressant, tranquilizer or other psychotropic drug intake is important both as an indication of psychopathology and because of possible interactions with diving (see Chapter 43). The candidate with a history of alcohol or other drug abuse should be assessed critically.

AGE (See Chapter 58)

As a general rule, it is recommended that diving in excess of 9 metres should not be allowed prior to a child reaching osteogenic maturity, i.e. when the epiphyses have fused. This is a very common statement, but there is very little evidence that decompression sickness has really influenced bone growth in young animals. Nevertheless, the possibility makes for hesitation, when recommending that children be allowed to dive in excess of this depth.

Children, although they may be trained in diving techniques, often do not have the physical strength or psychological stability to cope with the occasional unexpected hazards of diving. A child exposed to scuba training must be totally and completely under the control of a competent adult diver, of instructor standard. A salutary warning about allowing children to dive is given by reference to the age range in the fatality statistics. So far the youngest child to die while scuba diving was aged 7, but many were between the ages of 10 and 14 years.

No upper age limit applies provided that the candidate meets all medical standards. Although physiological age is more important than chronological age, for divers over 40 years it is recommended that regular re-examinations be carried out in order to detect medical abnormalities, which may interfere with efficiency and safety in the diving environment. Electrocardiographic (ECG) examinations during maximal exercise may be recommended as part of this medical, especially if cardiac risk factors are present. Some[2] have suggested that the ability to perform exercise up to 13 metabolic equivalents (METS) be considered a reasonable minimum for engaging in diving activities from a performance-criteria point of view. The means by which this is tested is at the discretion of the physician.

With increasing age, allowance must be made for a more conservative approach to diving activity, as well as to restricting the decompression schedules. This author arbitrarily recommends that older divers reduce their allowable bottom time by 10 per cent for each decade after the age of 30 years.

WEIGHT

Weight should be less than 20 per cent above the average ideal weight for age, height and build. Obesity is undesirable because it may increase decompression sickness (DCS), even though it may reduce the likelihood of hypothermia. For sport diving, it is permissible to allow diving with obesity that would not be accepted in professional or military diving. This is achieved by imposing an added safety margin in calculating the allowable duration of the dive. With divers who are obese, the bottom time is reduced, depending on the degree of obesity.

One arbitrary standard, used for many years at the Australian Diving Medical Centres, has been to reduce the allowable bottom time for the dive according to the percentage that the candidate's weight exceeds that expected for height and build. Appropriate tables or an index such as the body mass index (weight in kg/height in metres^2)(BMI) can be applied. Thus, if the BMI is exceeded by one-third, the permissible no-decompression bottom time for a given depth is reduced by one-third, but the longer duration is used for decompression calculations. An 18-metre dive allowing 60 minutes in the tables would allow an actual bottom time of only 40 minutes in this example.

OCCUPATION

The candidate's occupation may give some indication of his physical fitness, but may also be important in

increasing the relevance of diving hazards, e.g. aviators or air crew should be advised of the flying restrictions imposed after diving. Sonar operators and musicians may not wish to be exposed to the possible otological complications of diving, which may prejudice their professional life.

DRUGS

A thorough drug history is important in that it may give a clue to the presence and/or severity of otherwise undetected but significant diseases, such as hypertension, cardiac arrhythmia, epilepsy, asthma or psychosis. Also, the effects of drugs may influence diver safety and predispose to diving diseases. Both therapeutic and 'recreational' drugs should be considered. These possibilities are discussed more fully in Chapter 43.

CARDIOVASCULAR SYSTEM (see Chapter 56)

The existence of serious cardiovascular disease disqualifies the candidate from diving because of the risk of sudden collapse or decreased exercise tolerance. These diseases are responsible for 12–23 per cent of the deaths in recreational scuba diving. Any abnormality should be investigated fully prior to the issue of a fit-to-dive certificate.

The maximal stress exercise ECG is a valuable addition to the medical examination of all divers over the age of 45 years, and those younger where significant coronary risk factors are present. It is important to remember that significant coronary artery disease may be present despite a normal physical examination.

RESPIRATORY SYSTEM

Respiratory disease is the major cause for disqualification of diving candidates. Divers must not only be able to tolerate severe physical exertion, which requires good respiratory function, but also be able to tolerate rapid changes in lung volumes and pressures with equal compliance throughout the lung.

Until recently a history of **spontaneous pneumothorax**, because of the high incidence of recurrence and the implications of other basic pulmonary pathology, precluded diving. Spontaneous pneumothorax is predominantly a disease of young males, with the peak incidence being between 16 and 25 years with a second peak in patients over 40 with pre-existing lung disease. The over-40s with pre-existing lung disease are usually disqualified on the basis of their respiratory status. Primary spontaneous pneumothoraces are believed to be due to the rupture of apical, subpleural blebs that have probably filled with alveolar air dissecting from splits in local small airways.[3] Approximately 50 per cent will have a recurrence, usually on the ipsilateral side. However, recurrences are infrequent after an interval of two years.[3] It is not considered familial and is not fatal in most cases, except when its recurrence is provoked by such activities as scuba diving, with transformation into a tension pneumothorax.

Some diving physicians now believe that if no recurrence of the spontaneous pneumothorax occurs within this two-year period, candidates can be permitted to commence or resume diving. Others express concern with this approach as this presupposes that the cysts somehow disappear after these two years. The lack of credible autopsy data to support either argument is a consequence of these individuals being excluded from diving in the past. These authors believe that the history of a spontaneous pneumothorax precludes diving.

Some diving physicians also take the view that a history of **traumatic pneumothorax** does not necessarily disqualify a candidate from diving, provided that lung function has returned to normal and the chest X-ray is normal. Others maintain that pleural adhesions may form during the healing phase and increase the risk of pulmonary barotrauma (PBT). Current screening techniques do not reveal all such adhesions, but high-resolution computed tomography (CT) scans or spiral CT scans may demonstrate those not visible on chest X-ray. Again, there is a lack of credible data to support either view, but case reports do indicate that pleural tears occur at the sites of adhesions. A detailed explanation of the potential risks must be given to the candidate if no scarring can be demonstrated and they elect to dive.

Surgical pleurodesis to prevent recurrence does not render the patient fit to dive, as small blebs or bullae are still present, and air embolism is not prevented by this operation. In one case hemiplegia from cerebral arterial gas embolism developed in the first dive after pleurodesis. The resultant adhesions and decreased distensibility of the lung from pleurodesis or pleurectomy may also predispose to other PBT.

A history of **asthma** (see Chapter 55) is particularly important, because its persistence will result in increased pulmonary airway resistance and also may require the use of adrenergic drugs. Neither is acceptable in any diving operations, whether recreational or professional.

Cystic lung lesions, chronic bronchitis, chronic obstructive pulmonary disease and emphysema, and active or chronic respiratory infections, such as tuberculosis, histoplasmosis, mycotic infections and their sequelae, are all contraindications to diving. Sarcoidosis is also a contraindication because the pulmonary involvement is usually greater than suspected clinically, and the mucous membranes are also often involved.

Penetrating chest injuries and chest surgery should be considered as disqualifying because the scar formed in the lung tissue during healing may lead to tethering of the pleura or air trapping. It is of interest that the intensive care literature reports increased PBT in the opposite lung when one lung has scars or decreased compliance. Also, systemic gas embolism has been reported with the use of positive end-expiratory pressure (PEEP).

The history of respiratory disorders is complemented by the physical examination and simple respiratory function tests. High-pitched expiratory rhonchi, which may only be elicited during hyperventilation, indicate airway obstruction and preclude diving prior to further investigation. Thus, auscultation should be performed during hyperventilation through a wide-open mouth.

Most standards for divers require that the forced vital capacity (FVC) should be more than 4 litres in males, 3 litres in females, and the forced expiratory volume in 1 second (FEV_1) should not be less than 80 per cent of the predicted values based on population norms for age, height and sex. Forced expiratory flow (FEF) and mid-expiratory flow (MEF) measurements have been found more useful by some investigators, whilst others believe the peak expiratory flow rate (PEFR) should be equal to two times the predicted FEV_1. Since the introduction of respiratory standards, there has been an apparent reduction in the incidence of PBT.

Whenever it is decided to allow a candidate to continue diving, despite not meeting respiratory standards as described above, it is essential that the diver be made aware of the increased danger to himself, and the likelihood of serious consequences during rapid ascents. As a result of the morbidity and mortality from PBT, we are less lenient in dealing with minor degrees of respiratory impairment than with other abnormalities in the diving medical examination. A burst lung may not allow for adequate resuscitation, whereas if a diver trainee ruptures an eardrum, or even loses hearing in one ear, or suffers dental barotrauma, then these events are not usually life-threatening.

It would seem reasonable, if a person was perfectly normal in every other respect, but had reduced spirometry, to apply restrictions to his diving activities. These may include:

1 Slower than normal ascent. (This can be achieved by leaving the bottom earlier – perhaps coming up at the slower rate of 8 metres/minute, instead of 18 metres/minute.)
2 No 'free' or assisted swimming ascent or buoyant ascent practice. These are especially dangerous because of the involuntary tendency of the trainee to inhale deeply prior to the ascent, and therefore increase the likelihood of pulmonary barotrauma. In the event of a genuine emergency free ascent, it is more common for the diver to commence the ascent without a large lung volume because he is no longer able to inhale, either deep or shallow!
3 Restricted depth: it could also be suggested that the diver does not exceed 30 metres, as beyond this the likelihood of exhausting the air supply is greater due to both the narcotic effect reducing his alertness and the increased consumption of air at greater depth.

The full-plate posteroanterior chest X-ray may be taken in inspiration and expiration, in an attempt to demonstrate air trapping. Combined with a lateral

view, the yield may be increased but it certainly does not exclude significant degrees of air trapping. As for other aspects of the respiratory assessment, the X-ray must be considered in conjunction with the history, the clinical signs and respiratory function tests. Tomograms may be required to clarify doubtful radiological lesions.

The actual benefit of a chest X-ray is debatable. If there is no history of respiratory disorders, with a normal physical examination and acceptable lung function tests, the return from radiography is low. More conservative physicians would still require an X-ray to exclude cysts, bullae, fibrotic lesions and other abnormalities.

There is no need for routine follow-up annual chest X-rays unless there is a clinical indication.

In cases where the clinical history is equivocal or spirometry yields unexpected or borderline results, further sophisticated pulmonary function testing may be valuable, such as saline and histamine bronchial provocation testing, chest CT scanning, static lung volumes, compliance testing, CO diffusion coefficient and CO_2 tolerance. These tests may also be of value in further assessing the veteran diver (e.g. after PBT).

The most common dilemma is to establish whether bronchial hyperreactivity is present in the diving candidate. We have found pulmonary function testing before and after both histamine and ultrasonically nebulized hypertonic saline inhalation to be very useful (as well as being a dramatic demonstration to the previously doubting candidate). Others have used mannitol, distilled water, methacholine, hypercapnic hyperventilation and exercise provocation.

A 20 per cent reduction in peak expiratory flow rate or forced expiratory volume after 15 ml 4.5 per cent saline or after 4.0 mg/ml histamine would verify clinically significant asthma, and a reduction of 10 per cent would be considered sufficient to advise against scuba diving. Neither would indicate that treatment is required, however. Auscultation with hyperventilation often demonstrates the expiratory restriction more than spirometry – especially if it is in a localized area. Sometimes the positive response (increasing FEV_1 by 10 per cent) to inhaled salbutamol is used to support the asthma diagnosis.

UPPER RESPIRATORY TRACT

Disorders of this system comprise the largest cause of morbidity in divers. A history or physical sign of chronic or recurrent pharyngitis, tonsillitis or sinusitis may lead to problems with diving. Allergic rhinitis may affect sinus aeration or eustachian tube function, as well as arousing the suspicion of bronchial hyper-reactivity. Acute disorders of the ears, nose or throat may temporarily disqualify the diving candidate.

Sinus and nasal **polyps** may produce obstructions during ascent or descent, resulting in barotrauma. A deviated nasal septum may also result in abnormal airflow and nasal mucosa, influencing patency of sinus ostia and the eustachian tubes. Whenever obstruction or restriction of the upper respiratory airways occurs, barotrauma is likely. If infection is present, it may be aggravated and spread by diving.

A break in the skin or mucosal lining of gas-filled spaces is a danger in diving, allowing access of gas into the subcutaneous body tissues, resulting in barotrauma or surgical emphysema. This break is commonly produced by trauma (nasal injuries, dental extractions, etc.).

The larynx is vital in protecting the airways, and any significant pathology in this region precludes diving.

THE EAR

Outer ear

A diver is rendered temporarily unfit by the presence of acute or chronic otitis externa (see Chapter 29). The diver must be able to clear his ears, and the examining doctor must be convinced that the tympanic membrane is mobile. Cerumen does not need to be removed unless its presence has resulted in a complete blockage of the external canal. Inspection should be made for the presence of exostoses, and these should not be of such size as to block the external auditory canal, or lead to occlusion by superimposed cerumen or infection.

Middle ear

A healthy tympanic membrane, intact and mobile, is a prerequisite for diving. Evidence of otitis media, however mild, should preclude diving until fully recovered. Chronic otitis media or cholesteatoma should be cured before diving. Candidates with a tympanic membrane perforation or ventilation tubes should not dive. Obviously it would be unwise to submit a tympanic membrane, which has been weakened by a thin atrophic scar, to pressure changes involved in diving. However, a healed perforation, which left the tympanic membrane normal in strength and mobility, would be quite acceptable. A tympanoplasty is not necessarily a contraindication to diving, if healing has been completed. A retracted and immobile tympanic membrane is unacceptable.

Otosclerosis surgery, with the use of an ossicle prosthesis, or stapedectomy, predisposes to spontaneous or provoked oval window rupture and therefore precludes diving. Patients who have undergone extensive mastoid surgery may experience a strong caloric response and severe vertigo.

Observation of tympanic membrane movement is an essential part of the medical examination.

The eustachian tube must function normally; i.e. autoinflation must be accomplished voluntarily and without excessive force. The tympanic membrane is observed through the otoscope to move outward as the subject performs the Valsalva manoeuvre. It should be noted that the ability to autoinflate at any one point in time does not exclude the possibility of eustachian tube obstruction at another time. The function of the eustachian tube is dependent upon normal nasal function, and this requires careful assessment, especially in candidates with such conditions as allergic or vasomotor rhinitis, cleft palate or bifid uvula (see Chapter 35).

Inner ear

Cochlear function

Ideally, divers should have normal cochlear and vestibular function, but moderate changes in auditory acuity may be acceptable. Loss of cochlear function may be associated with loss of vestibular function. If the vestibular portions of the inner ear respond to stimuli unequally, then vertigo might result, especially when visual fixation is poor, as frequently occurs in diving (see Chapter 38).

Threshold hearing for divers should ideally be 20 dB or less at the frequencies between 500 and 4000 Hz using audiometres calibrated to ISO standard. Frequencies should extend to 6000 and 8000 cycles/second, in both initial baseline and annual examinations, even though these may be affected by noise damage. They are also the frequencies most commonly affected by diving.

Some individuals may have 'normal' ears, which do not withstand trauma as well as others. A previously damaged inner ear is more susceptible to further damage.

Audiometry is part of the routine diving medical examination.

All divers should have an audiogram after any episode of middle-ear barotrauma in order to exclude an additional but clinically important inner-ear barotrauma.

Vestibular function

It is wise to exclude from diving those individuals whose vestibular function is not normal and equal on each side. Divers who have inner-ear damage from DCS or barotrauma may fall into this category.

The sharpened **Romberg test** may detect vestibular abnormality. A history of Meniere's disease or other chronic vestibular disorders should bar diving.

Variations in the sharpened Romberg test have evolved. For more reliable and standardized testing, as in DCS treatment assessment, the best of four,

Procedure for sharpened (tandem) Romberg test[4]

1 *Wear flat shoes, or bare feet, on a solid flat surface.*
2 *Stand heel to toe, with arms crossed over the chest.*
3 *Eyes are closed and the candidate attempts to remain upright for 60 seconds. He may have up to four attempts.*
4 *Scoring is as follows:*

1st attempt = duration in seconds × 4 ____
2nd attempt = duration in seconds ×3 ____
3rd attempt = duration in seconds × 2 ____
4th attempt = duration in seconds × 1 ____
Total */240*

Example: *If the diver was able to stand for only 15 seconds on each attempt, the score would be (15×4) + (15×3) + (15×2) + (15×1) = 150/240.*
Scores tend to be very low, implying vestibulocerebellar damage, or very high in normals.

1-minute attempts may be employed. In the more expedite diving medical examinations the ability to stand for more than 15 seconds may be accepted as relative normality.

THE EYE

Good vision is needed under water to avoid dangerous situations and, after surfacing, when the diver may have to identify other divers, landmarks, floats, boats, etc. Distant vision should not be less than 6/18 for both eyes, and 6/24 for the worse eye (corrected or uncorrected).

Presbyopia may lead to problems reading gauges and watches. A convex lens added to the facemask should correct hypermetropia. Myopia is the most common deficiency requiring correction. The use of a corrected lens in the facemask or soft contact lenses is of value in reducing the danger of reduced visual acuity. Due to the risk of damage to the cornea by bubbles developing underneath the lens, hard contact lenses, unless fenestrated, should not be worn when diving.

In **ocular surgery**, the cornea is slow to heal. In radical keratotomy, the cornea never attains full strength if the operation is successful. Mask squeeze or blunt trauma may lead to corneal rupture, usually along the surgical incisions. Patients who have undergone radial keratotomy are usually advised to avoid contact sports thereafter. Ophthalmic surgeons may be unaware of the pressure imbalance that develops between the mask and the eye surface, especially in novice divers. Experienced divers might be permitted to continue diving with special precautions to avoid pressure imbalance in the facemask. Newer procedures such as excimer laser keratotomy do not involve such deep incisions, and generally divers undergoing these procedures will be fit to dive once full recovery from the procedure has occurred.

Similar considerations apply to the person who has undergone corneal grafting. In addition, the risk of corneal abrasive trauma must be considered because the cornea has no sensory nerve supply for at least three months postoperatively and then recovers only partially.

Glaucoma and its treatment involve complex interactions with diving, and most of the drugs commonly used for this have contraindications. Surgery, especially in producing a very low intraocular pressure, may also be a problem.

Hollow ocular prostheses would be unacceptable during diving and should be removed before descent.

Ocular problems are discussed further in Chapter 42.

NERVOUS SYSTEM (see Chapter 40)

Many neurological abnormalities will add danger to the diver, as well as complicating the management of neurological disorders due to diving, such as cerebral or spinal DCS, air embolus from PBT, oxygen toxicity, etc.

Cerebrovascular disease, such as intracranial aneurysm and arteriovenous malformation, carries

the risk of sudden death or coma. Subjects with a history of any form of intracranial haemorrhage should be discouraged from diving, even if there are no apparent sequelae. Patients who have undergone craniotomy are at an increased risk of epilepsy (see below).

Migraine occurs commonly in the general population, but few see a doctor and even fewer are disabled, although many take drugs that may be relevant. In diving, migraine may be precipitated by: elevated arterial carbon dioxide tension (see Chapter 18); cold water exposure; psychological stress; glare; intra-arterial bubbles (DCS, cerebral arterial gas embolism); and possibly increased oxygen pressure (hyperbaric oxygen therapy). Those with a history of migraine should either be advised against diving or advised of depth/duration limits to reduce the diagnostic dilemma of possible cerebral arterial gas embolus or neurological DCS. Such advice might be a maximum depth of 18 metres, duration well within no decompression limits, a slow ascent rate and avoidance of cold exposure to the head. Migraine sufferers with neurological features should not dive at all, and those with a history of associated vomiting are also at greater risk.

Epilepsy is an absolute contraindication to diving. Apart from the risk of drowning, there is the risk of PBT if the diver is returned to the surface during the tonic phase. A diver's safety may be impaired under water by the sudden development of confusional states (aura or post-ictal), sensory disturbance, nausea, vertigo or severe headache. Vomiting under water can be disastrous. Epilepsy, which may be defined as two or more definite seizures, occurs in 0.5 per cent of the population. Salient points in history include the facts that 34 per cent of childhood epileptics recur after withdrawal of medication, 50 per cent of people who have had one seizure will have another; and 'controlled' epileptics are 2.5 times more likely to be involved in motor vehicle accidents. These should not be confused with the febrile convulsions of infants, which do not have the same ominous sequelae. In the diving environment, glare, sensory deprivation, narcosis, stress and hyperventilation may be possible 'triggering' factors. In fact, some divers have their first epileptic attack under water.

A history of **head injury** may be important because of both the risk of seizure and the effect on cognitive functioning. In assuming the severity of such an injury, the following points indicate a high risk of post-traumatic epilepsy within six months:

- Duration of loss of consciousness greater than 10 minutes.
- Seizure in the immediate post-trauma period.
- Neurological deficit (e.g. post-traumatic amnesia for 24 hours, transient hemiparesis).
- Disruption of cortex (depressed skull fracture, intracranial haematoma).

Where there is no loss of consciousness (i.e. subconcussive), there are generally no sequelae, but post-traumatic amnesia indicates a concussive injury, which should be carefully assessed. A normal electroencephalogram in the immediate post-trauma period does not preclude the development of post-traumatic epilepsy. Candidates with any of the above features should be free of seizures, should not require drug therapy and should be functioning normally in their daily lives. Those with a history of penetrating head injury (e.g. missile), post-traumatic amnesia greater than 24 hours, depressed skull fracture or acute haematoma should not dive for at least five years because of the risk of later post-traumatic epilepsy.

Spinal cord injury or disease is important because, in addition to the limitation exposed by the primary neurological deficit, there may also be an increased vulnerability to spinal DCS and/or further injury. The vulnerability may be due to the exhaustion of spinal cord redundancy, e.g. in myelopathies due to DCS or poliomyelitis. Candidates with trauma or disease of the cord should not dive, even if there is no clinically detectable residual deficit.

Some **neurological handicaps** (such as traumatic paraplegia, muscular dystrophy, multiple sclerosis) may be 'accepted' for special diving circumstances, such as the specialized handicapped diver programme (see Chapter 64). As well as the obvious limitations, the candidates may also exhibit poor heat and cold tolerance.

A more common problem is the candidate with a history of herniated intervertebral disc. If asymptomatic and with no neurological signs, restricted diving may be permitted. Diving may also be considered reasonable four to six months after

successful, uncomplicated surgery, provided that there is no residual deficit.

Spinal stenosis due to degenerative joint disease renders the cord more vulnerable to damage from any cause.

Peripheral neuropathy may cause diagnostic confusion in assessing possible DCS, and sensory loss may lead to trauma and delayed wound healing. The underlying cause usually precludes diving, however.

ENDOCRINE SYSTEM

Diabetes mellitus (see Chapter 57) should be regarded as a disqualifying condition for a number of reasons. The most obvious is the risk of hypoglycaemic coma in an insulin-dependent diabetic or even in those taking oral hypoglycaemic drugs. The early warning signs of such a reaction may be masked by the underwater environment, which interferes with taking remedial action.

Despite the foregoing, there are grounds for allowing a diet-controlled diabetic without complications to dive after an exploration of risk factors and limitations to avoid the above potential problems.

Thyroid disorders may be missed clinically but are important because of the profound effects that they may have on cardiac and neurological function, if untreated. With good control of hyper- or hypothyroidism, verified by thyroid function tests, recreational diving may be undertaken. Significant goiter would preclude diving until corrected because of possible airway compression.

Other severe endocrine disorders, such as Addison's disease, Cushing's syndrome, hyperparathyroidism, etc., are also unacceptable in the diving environment.

GASTROINTESTINAL SYSTEM

The **mouth** should be inspected for signs of dental caries and periodontal disease severe enough to produce fragile or loose teeth that could be inhaled, or might cause difficulty retaining the scuba mouthpiece. **Full or partial dentures** should be assessed for stability in maintaining the mouthpiece.

Abdominal wall hernias (inguinal, femoral, umbilical, and incisional) which potentially contain bowel may cause problems with the variation in gas volumes during changes of depth. As well as restricting the diver's physical capabilities, there is the potential for incarceration incurred by lifting heavy scuba equipment. Candidates may be passed fit after successful surgical repair. The presence of a **hiatus hernia** may lead to underwater reflux or regurgitation, especially with the head-down position of descent. During ascent, reflux is also possible, as is gastrointestinal barotrauma due to expansion of gas in the stomach (see Chapter 9). This is especially so with the paraoesophageal hernia. After surgical repair of hiatus hernia, the candidate should be able to eructate, indicating the ability of gastric gas to escape during ascent. Otherwise, gastric rupture is possible.

A history of **peptic ulcer** may be important because of the potential risk of perforation or bleeding. A history of gastric surgery may have led to a 'dumping syndrome' with the risk of hypoglycaemia. Pyloric obstruction may also cause stomach overexpansion during ascent.

Inflammatory bowel diseases, such as Crohn's disease or ulcerative colitis, are not necessarily a bar to recreational diving during periods when the condition is in remission, and drug therapy is not required. The possibility of flare-up at a remote diving location should be borne in mind, however. Ileostomy or colostomy (except the 'continent' ileostomy type) should present no great problem with diving.

MISCELLANEOUS

Space prevents discussion of all possible disorders that diving candidates may present to the examining physician. An experience of diving and a sound knowledge of diving medicine are vital in arriving at a sensible assessment of fitness to dive.

Bleeding disorders, such as haemophilia and Von Willebrand's disease, are grounds to reject the recreational diver, because of the risk of trauma

and the unknown effect of expanding intravascular or tissue bubbles. Dysbaric pathology, especially in the brain, spinal cord or inner ear, may be extended by haemorrhage.

A sickle-cell assessment may be needed because of the exposure to potentially hypoxic conditions. A sickle-cell crisis may be catastrophic in remote localities, or under hyperbaric conditions. It will also complicate the management of serious decompression sickness cases, and is another cause of aseptic necrosis of bone (see Chapter 14). The candidate with sickle-cell disease should be advised not to dive. Those with asymptomatic sickle-cell trait may also be at risk with severe hypoxia or with local tissue hypoxia secondary to DCS. If diving is undertaken, the diver should be advised to keep shallow (18 metres maximum) and well within decompression limits.

Polycythaemia, which causes a cellular circulatory overload, may lead to occlusive vascular phenomena and is thus disqualifying. Candidates with leukaemia in remission would require careful assessment.

Acquired immune deficiency syndrome (AIDS) should disqualify candidates because of risk both to the individual and to potential rescuers. Problems for rescuers include the sharing of equipment, such as demand valves, and reluctance to undertake cardiopulmonary resuscitation. The AIDS sufferer has a greatly increased risk of infection, particularly of the lung and brain. Recent reports describe cerebral dysfunction (cognitive deficits) and other neurological disturbances (ataxia, confusion, aseptic meningitis and hemiplegia) in otherwise previously asymptomatic HIV seropositive patients. On this basis, asymptomatic subjects (even with normal immune function) should be regarded as unfit for diving. Other reports have suggested that passage of the AIDS virus into the brain may be aggravated by the disruption to the blood–brain barrier, which may occur with normal decompression.

A **musculoskeletal problem** of any severity will limit the diver's physical capabilities, and complicate DCS assessment. A history of back injury or recurrent back pain is a strong contraindication to professional diving, although the recreational diver may be considered after appropriate advice, provided that there is no neurological deficit.

Pregnancy is a contraindication to diving, and is discussed further in Chapter 60.

Motion **sickness** is a dangerous disorder to have while diving from boats or in rough water. Vomiting under water is a problem, especially if the diver vomits into his regulator. The psychological manifestations of motion sickness may also result in injudicious decisions, e.g. to surface without completing adequate decompression stops (see Chapter 26).

Smoking is detrimental, because of its specific effect on upper and lower respiratory function increasing the chance of ear, sinus and pulmonary barotrauma. There is also an increased risk of coronary artery and peripheral vascular disease. Other recreational or social drugs also need to be assessed (see Chapter 43).

Any acute illness is usually a temporary bar to diving.

DIVING HISTORY

Knowledge of previous hypobaric (aviation), hyperbaric and aquatic accidents may be invaluable in assessing the likelihood of potential future problems. Specifically, a history of barotrauma, DCS, dysbaric osteonecrosis, nitrogen narcosis, gas toxicities, and unconsciousness or near-drowning should be sought.

Divers who have suffered minor (type 1) DCS should undergo a thorough follow-up (see Chapter 13) and, in any case, should not dive for at least four weeks following the episode. If there are no sequelae, diving may be recommenced, but a more conservative diving profile is recommended.

Neurological sequelae after DCS are not uncommon and infer a subsequent predisposition to more accidents of a similar nature. Divers with any persisting neurological deficit after DCS should not dive again. Some would argue that divers who had DCS with any neurological feature – no matter how rapidly and completely resolved – should be advised against further diving.

Divers who have had PBT are usually regarded as permanently unfit for further diving (see Chapter 6).

CONDITIONAL CERTIFICATES

To the uninformed, restricting a potential diver with a medical condition to shallow water may offer a level of protection. However, as the greatest proportionate pressure changes occur in the first 10 metres of water, the diver is at greatest risk of PBT close to the surface. Conditional certificates limiting a diver to less than 18 metres will not be accepted by the recreational diving agencies, as in all basic courses students must complete an 18-metre dive.

This does not prevent the diving physician from discussing safety strategies with the diver; e.g. additional safety stops and limiting bottom times in individuals with risk factors for DCS.

Table 53.1 *Some important medical contraindications to diving**

Risk of sudden death
- Asthma (CAGE)
- Coronary artery disease
- Intracranial aneurysm or atrioventricular (AV) malformation
- Other cerebrovascular disease
- Cardiac arrhythmia ± drugs
- Severe hypertension
- Congestive heart failure
- Causes of vomiting

Impaired consciousness
- Drugs
- Epilepsy
- Diabetes
- Cardiac arrhythmia
- Transient ischaemic attacks
- Unexplained syncope

Impaired judgement
- Drugs
- Psychosis
- Severe anxiety
- Severe depression
- Claustrophobia

Risk of disorientation
- Tympanic membrane perforation
- Inner-ear disease or surgery
- Uncorrected poor visual acuity

Impaired mobility
- Spinal column/cord disease or injury
- Neuromuscular disease (neuropathy, myopathy)
- Obesity
- Poor physical fitness
- Pregnancy

Risk of barotrauma
- Asthma
- Spontaneous pneumothorax
- Pulmonary cysts, fibrosis, scars, bronchitis, chronic airway disease, etc.
- Blocked eustachian tubes or sinus ostia
- Acute or chronic respiratory infections
- Radial keratotomy

Risk of decompression sickness
- Obesity
- Acute physical injury
- Spinal cord disease or injury
- Intracardiac shunts (ASD, VSD and foramen ovale)
- Extremes of age
- Pregnancy

* This list includes some common absolute and relative contraindications. It is no way complete, nor does the order signify importance.
ASD, atrial septal defect; VSD, ventricular septal defect.

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54

Medical standards for commercial divers

ROBYN WALKER

INTRODUCTION

The commercial or professional diver encompasses a broad range of divers including the military diver, the offshore oilfield saturation diver, the recreational diving instructor, the photojournalist, the scientific diver and the police diver. Whilst their scope of diving varies markedly, what they do have in common is that they are employed and receive financial gain from their diving.

All commercial divers should receive appropriate training in order for them to carry out their task, be capable of dealing with emergencies involving themselves or their buddy, and be able to operate in the face of demanding environmental conditions. The diver who is paid to get the job done, does not have the luxury of deciding when he will dive. The professional diver is often only one part of a team. He must be able to replace, and be replaced by, other members as the task demands.

Most deviations from the norm make for complications. A diver who is less physically strong than the others will sooner or later be exposed to conditions which he will be unable to handle. The professional diver must be able to perform all the diving tasks expected of the team as a whole, and not have any specific restrictions or limitations imposed on him or his diving practice.

When assessing a candidate's fitness for commercial diving the doctor is usually constrained by governmental regulations or by commercially agreed standards (Table 54.1). The doctor has to consider the position of the employer as well as the diver, and has less scope for including the potential candidate in a discussion of risk factors. The doctor must also consider any legal aspects/compensation liabilities that may arise in the future.

In most industrialized countries workplace regulations dictate that the employer has a duty of care to ensure that all diving carried out is as safe as is reasonably practicable. The diver also has a responsibility to declare if his fitness to dive is compromised for any reason.

Anti-discrimination laws now require statistically sound scientific-based evidence to support the exclusion of a worker from a particular occupation on medical grounds. It is therefore important that the

Table 54.1 *Diving regulations*

UK	Health & Safety Executive UK Diving Regulations (1998)
France	Hyperbaric Work – Health and Safety Act (1990)
Norway	Manned Underwater Operations (Norwegian Petroleum Directorate 1990)
USA	Association of Diving Contractor Standards (1994)
Australia/ New Zealand	AS/NZS 2299.1-1999

medical practitioner who undertakes commercial diving medical examinations has an in-depth knowledge of the particular workplace and environmental conditions under which the diver will operate.

In general, the minimum standards for commercial divers are similar to those that apply to recreational divers and are outlined in Chapter 53. This chapter will deal with the additional specific fitness requirements for commercial divers. Because these medicals are required at regular intervals, usually annually, the original or baseline data – both clinical and investigatory – allows for comparison with the current assessment and the detection of adverse health effects.

PHYSICAL FITNESS

Physical fitness standards are often contentious and have effectively excluded women from the realms of military diving. Professional diving is arduous and involves heavy equipment – one of the rebreather units used by many of the world's navies weighs 29 kg on land when fully charged. Not many women are capable of lifting and carrying this type of load on land, and neither can some men! Fitness tests have long been detailed in terms of aerobic performance (i.e. a 2.4-km run in under 10 minutes) and tests of upper body strength (push-ups or chin-ups). However, these types of test give no indication of an individual's ability to do a specific occupational task.

Fitness tests should be performance-based, reflect the nature of the activity, and be job-specific. For example, if the job requires the diver to climb into a small boat wearing a 30-kg set, the test should be exactly that. Swimming ability should be assessed as opposed to running speed. Professional divers also need to have sufficient physical reserves in order to respond to changing environmental conditions and to respond to emergencies.

AGE

Most authorities agree on 18 years as the minimum age for entry into the commercial diving world. This is also realistic when considering the time required for the prerequisite training for bell divers or the age limits in place for enlisting in the military or police forces. It is unlikely that a scientific diver will have the necessary educational qualifications at less than 18 years of age. These general limits on entering the workforce exist to ensure the potential employee has sufficient emotional and physical maturity to accept the responsibility of the job.

There is no upper limit of age for professional divers, but like recreational divers the risks of cardiovascular disease with aging must be considered. Physical fitness declines with age, along with the ability to recover from injury. The medical practitioner must make some assessment of the aging diver's pulmonary and cardiovascular reserves, and also his ability to continue to perform all required tasks. As the diver ages, his experience may be used to more benefit in a diving supervisory role.

SKIN

Saturation divers may spend days to weeks at depth, living in a habitat at or close to their working depth. These habitats are often hot and humid and can be an incubator for bacterial skin infections. Divers often share equipment such as wetsuits and regulators. Wet skin can promote the spread of bacteria normally confined to intertriginous zones. Common fungal infections such as tinea pedis require treatment to prevent the entire diving team from becoming infected. *Molluscum contagiosum*, because of its high infectivity, requires treatment before a diver is declared fit to dive. Herpes simplex is relatively common in the general community and will not harm

the diver; however, if oral lesions are so widespread and painful that holding a mouthpiece becomes difficult then the diver should be made temporarily unfit to dive. Herpes zoster infections will make the diver unfit to dive until he is no longer infectious and the pain and lesions have settled.

Allergic contact dermatoses occur in divers. Antioxidants and glues used in the manufacture of neoprene wetsuits and drilling muds have been implicated, and require assessment by a dermatologist. As all future exposure to the allergen must be avoided this can have serious effects on the diver's future.

Aquagenic urticaria is an absolute contraindication to a professional diving career.

VISION

The commercial diver often works in such low visibility environments that he uses his hands to 'see'. However, the diver must be able to read gauges, watches and decompression tables. Contact lenses may be used to correct vision, although hard lenses should be avoided due to the risk of corneal ulceration secondary to the formation of bubbles beneath the lens. This risk is reduced if fenestrations are drilled through the lenses.

Colour vision is not essential to diving, although there will be some exceptions, e.g. military explosive ordnance divers who require normal colour vision to distinguish between the red and the green wire!

EAR, NOSE AND THROAT

Exostoses are particularly common in subjects who spend extended time in cold seawater. The diver is rendered temporarily medically unfit by the presence of acute or chronic otitis externa, which is particularly troublesome in chamber operations (see 'Skin' above).

Round window fistula secondary to inner-ear barotrauma (see Chapter 7) has in the past resulted in an automatic disqualification from diving. There is no doubt that individuals who have persistent labyrinthine symptoms such as vertigo, dizziness and loss of balance should not resume diving. However, in divers who have made a full recovery (with either conservative or surgical management) there are some diving physicians who would consider them fit to dive. This requires careful consideration based upon the audiological outcome, and detailed and informed advice must be provided to the diver. Forceful autoinflation must be avoided at all times.

Hearing

The commercial diver must be able to communicate and understand verbal instructions. Most commercial diving standards require an annual audiogram covering the frequency range from 250 to 8000 Hz.

It had been considered for many years that professional divers suffered from an accelerated form of hearing loss. However, many of these studies failed to take into account the diver's exposure to workplace noise in addition to his diving exposure. For example, naval divers were exposed to gunfire and engine room machinery noise. With advances in the management of industrial noise exposure the diver should not be at increased risk of hearing loss from these extraneous causes.

Diving exposure may result in hearing loss as a consequence of inner-ear barotrauma and inner-ear decompression sickness. There is also some experimental evidence of cochlear degeneration in animals that have been subjected to accepted compression/decompression schedules. Therefore, the appearance of hearing loss (particularly a unilateral loss) should be evaluated carefully. At the time of the annual medical it is also appropriate to provide specific advice to the diver on the importance of using appropriate hearing protection in designated high-noise areas.

NERVOUS SYSTEM

A detailed examination of the nervous system is mandatory, and should be normal. The presence of any neurological disease is grounds for rejection, especially progressive, relapsing or intermittent conditions. The exception to this rule will be any

well-documented, non-progressive abnormality such as a patch of anaesthetic skin, provided that generalized disease is excluded.

RESPIRATORY AND CARDIOVASCULAR SYSTEMS

The minimum standards for the professional diver are the same as those for the recreational diver, and are discussed in Chapter 53. There is no requirement for an annual chest X-ray in the commercial diving population. However, a chest X-ray may be indicated in divers who have suffered an intercurrent respiratory illness, a pulmonary injury, or as follow-up of a previously documented abnormality.

GASTROINTESTINAL SYSTEM

The commercial diver is often required to work in remote areas, in the absence of qualified medical support. A perforated peptic ulcer is a medical emergency at the best of times, but is a life-threatening emergency on an isolated diving platform or during a saturation dive. Similarly, treatment of an acute exacerbation of inflammatory bowel disease may cost a company dearly if a saturation dive has to be aborted on medical grounds. Serious consideration must be given to the continued fitness to dive of a commercial diver with chronic gastrointestinal disease.

DIABETES

Diabetes requiring medication is usually considered a contraindication to commercial diving.

SCREENING FOR DYSBARIC OSTEONECROSIS

In recent years most diving medical standards have required all professional divers to undergo annual long-bone X-rays as a screening test for dysbaric osteonecrosis (see Chapter 14). This is no longer required as the incidence of the disease is low, the diver is exposed to a significant radiation dose, and the yield of positive results is low. It is recommended that long-bone screening be carried out on only those divers who are considered to be at increased risk of dysbaric osteonecrosis. These baseline X-rays should be performed once on entry to the profession and be retained for at least the career lifetime of the diver.

If dysbaric osteonecrosis is suspected, then a further series should be taken, guided by the clinical presentation. The films must be comparable, and it is recommended they be performed in accordance with the procedural guidelines laid down by the UK Medical Research Council Decompression Sickness Registry. Alternatively, magnetic resonance imaging is both more informative and carries less risk.

RETURN TO DIVING AFTER ILLNESS OR INJURY

A careful assessment of dive fitness is required in any diver who suffers an illness or injury. It is important to consider whether or not the condition impacts on dive safety, and whether the diver can continue to perform his job. Workplace legislation requires employers to undertake reasonable and practical steps to provide a safe working environment for all. The diver who suffers a traumatic amputation of one or more fingers may still be able to handle heavy and bulky underwater tools, act as a standby diver and tend lifelines. If the individual can still demonstrate his proficiency in all these tasks, then continued diving may be possible.

The diving medical practitioner must understand the specific job requirements of particular diving operations in order to be able to provide informed judgements on diving fitness. The diving supervisor can assist the medical practitioner by delineating exact job descriptions. It is equally important to assist the diver who is permanently unfit to dive to understand and accept the reasoning behind the decision.

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55

Asthma

CARL EDMONDS

Expiratory spirometry: abbreviations and definitions

PEF	= *Peak expiratory flow*
VC or FVC	= *(Forced) Vital capacity*
FEV_1	= *Forced expiratory volume over 1 second*
FEF_{25-75}, *MMEF*	= *Maximum mid-expiratory flow*
MEF_{75}, FEF_{25}	= *Maximum expiratory flow at 75 per cent of vital capacity*
MEF_{50}, FEF_{50}	= *Maximum expiratory flow at 50 per cent of vital capacity*
MEF_{25}, FEF_{75}	= *Maximum expiratory flow at 25 per cent of vital capacity*

PATHOPHYSIOLOGY

Wheezing as a child [1]

Wheeze is common in infants, its prevalence in the first year of life perhaps being as high as 60 per cent (USA) and as high as 32 per cent in the first five years of life (UK). Wheeze is due to a restriction of air flow, and is more frequent in infants because they have proportionately smaller airways. The three commonest causes are:

- **Recurrent wheeze**: this is related to abnormal airway mechanics, maternal smoking during pregnancy, and genetic factors. Male infants are affected more often, with an increased risk of developing chronic obstructive pulmonary disease after the age of 60 years.
- **Episodic viral wheeze**: this is is unrelated to lung function at birth, and there is normal bronchial hyperresponsiveness. Most children are asymptomatic by the age of 10 years.
- **Asthma**: the risk is increasingly related to atopy and bronchial hyperreactive response as age increases. Both genetic and environmental factors are present, with peak prevalence between five and ten years of age.

Other causes of wheeze include abnormalities of the tracheobronchial tree, cystic fibrosis, inhaled foreign bodies and chronic lung disease of prematurity.

Often, wheezing children are inappropriately labelled as asthmatic. Even so, only 25 per cent of

children with asthma at the age of seven years will have asthma in adult life. This makes the automatic extrapolation from childhood asthma to adult asthma a dubious one.

Asthma as an adult[2–4]

Clinical asthma is estimated to occur in 4–7 per cent of the USA population, but respiratory symptoms are much more common. Asthma is due to a hyperresponsiveness of airways to stimuli which do not affect normal subjects. The obstruction is due to smooth muscle contraction, airway inflammation and oedema, mucus hypersecretion and impaction. Airway lability and episodic airway obstruction are hallmarks of asthma.

Recent studies indicate that airway inflammation is present even in asymptomatic asthmatic patients. Eosinophilia and neutrophils are present in the airways and lumen.

The functional consequence of airway narrowing in asthma is a slowing of the rate of expiratory airflow in both small and large airways, and an increase in expiratory effort. There is a propensity for small intrapulmonary airways to close prematurely at normal lung volume.

The increase in total lung capacity and residual volume as the asthma progresses, requires the asthmatic to hyperventilate to maintain alveolar ventilation and patency, with increased work of breathing. Compliance is reduced with increasing resistance to air flow, reducing alveolar ventilation and making the lungs stiff. Increasing ventilation with exercise increases these effects and results in dyspnoea at a lower exercise level than in non-asthmatics.

Although some subjects can estimate the degree of the obstruction, this varies. Others may not be able to detect even considerable changes in airway function. Therefore, reliance on symptoms to assess the clinical status of subjects with asthma is not advisable.

There is a classification for asthmatics. In **mild intermittent asthma**, the patient is usually asymptomatic with normal or near-normal (within 80 per cent) pulmonary function, and the treatment of choice includes inhaled corticosteroids and intermittent short-acting beta-receptor agonists. Other categories are **mild persistent asthma**, **moderate persistent asthma** and **severe persistent asthma**.

With the increase in clinical severity, the lung function tests are progressively impaired, even between episodes. With this increasing severity the FEV_1 is more reliable than the PEF, while the FEF_{25-75} is an even more accurate measure of this small airway obstruction. The PEF correlates mainly with flow in larger airways.

Over many years persistent airway inflammation may cause fixed airway narrowing with a reduced response to bronchodilating agents. In older adults, although typical asthma symptoms also occur, many only notice a reduced exercise tolerance and intermittent chest tightness. They frequently comment that they do not have asthma because they do not 'wheeze'. They progressively modify their lifestyle to accommodate this loss of pulmonary efficiency. The FEV_1 reduces by an average of 38 ml per year in asthmatics compared with 22 ml in normals.

Asthma is **diagnosed** by both clinical features and lung function assessments, neither of which is adequate on its own.

The underlying causes for asthma are not known, but a combination of genetic factors (especially atopy) and allergen exposure is known to influence sensitization and airway inflammation. Allergen exposure results in increased airway inflammation, inducing prolonged increase in bronchial responsiveness and abnormal lung function. While allergens initiate and sustain airway inflammation, irritants may also trigger this airway narrowing. **Sensitizers** include allergens (house mite, moulds, dust, and animal dander – especially that of cats and dogs which live inside). Chemicals include timber preparations, formaldehyde, paint fumes, perfumes, varnishes containing PVC, glutaraldehyde, latex and fumes from soldering. **Trigger factors** include exercise, mouth-breathing, food (metabisulphite, monosodium glutamate), medications (aspirin, NSAIDs, beta-blockers) and irritants (pressurized aerosols, fire smoke, environmental tobacco smoke, soap powders, air-conditioning, solvents, household and hobby glues).

ASTHMA IN DIVERS [3–14]

Respiratory disease is the major cause for the medical disqualification of diving candidates. Divers must be able not only reliably to undertake severe physical exertion, which implies good respiratory function, but also to tolerate rapid changes in lung volumes and pressures with equal and normal compliance throughout the lung. Any local airway restriction, for example fibrosis or cysts, may result in pulmonary barotrauma (PBT), with a tearing of lung tissue and subsequent complications, including air embolism (see Chapter 6).

A history of asthma is important, because its recurrence will result in increased pulmonary airway resistance (predisposing to PBT and impaired respiratory performance) and also may require the use of adrenergic drugs. Neither is acceptable in diving operations, recreational or professional.

At present, many organizations that train recreational divers, as well as the US Navy and commercial diving operators, will not accept subjects having a history of asthma beyond 12 years of age, for training or employment. This is based on the belief that they may develop airway obstruction under conditions associated with diving. It is reasoned that airway obstruction developing under water would predispose to PBT because of the impaired ability to empty the lungs during rapid ascent. The risk may be aggravated by: the greater inspiratory reserve volumes used to keep their airways open; interference with the elastic properties of the lung; greater resistance to exhalation; and the occasional association of cystic changes.[3–5]

Some clinicians[6,7] have documented the association of asthma with diving accidents, including PBT with minimal provocation.

Hyperventilation at depth causes divers to consume their air supply more rapidly. The rapid shallow pattern of ventilation in asthmatics further increases the dead space/ventilation ratio, decreases alveolar ventilation, and increases resistance and the work of breathing the denser gas mixtures inspired at depth.

Dyspnoea and reduced ventilatory capacity due to asthma may lead to rapid uncontrolled ascent, panic, fatigue and drowning.

CASE REPORT 55.1

AB, aged 43, was a very experienced diver who previously had asthma as a child, and who still had high-pitched rhonchi on auscultation, during hyperventilation.

A professor of respiratory medicine informed AB that his lungs had quite adequate function for scuba diving, and that the lung function tests were normal. This advice was refuted by members of a Diving Medical Centre, but academic brilliance won out.

At a depth of 18 metres while exploring a wreck, he suddenly became aware, as he floated up over the deck, of a pain in the left side of his chest. He then attempted to ascend, but took over half an hour to reach the surface. During this time there was a continual pain in the chest, aggravated if he tried to ascend more rapidly. With extreme courage, and commendable control over his breathing gas consumption, he reached the surface although in great discomfort. He was then given oxygen and transferred to hospital.

The clinical and X-ray evidence verified the diagnosis of left pneumothorax, and a thoracentesis was performed. He returned to the professor of respiratory medicine, to be reassured that on statistical grounds it was unlikely to happen again. The Diving Medical Centre physicians assured him that not only would it happen again but that, with the lung damage and the surgical treatment received, it was *more likely* to happen again and that it should not have been allowed to happen in the first place. He decided, this time, to take our advice.

Diagnosis: pneumothorax with minimal provocation, asthma.

CASE REPORT 55.2

NZ, aged 20, had been certified fit to dive despite an asthma history. Prior to the dive there were no symptoms, but he still took a salbutamol inhalation.

In his first deep dive, after 8 minutes at 30 metres, he took 23 minutes to ascend to 15 metres. A burning pain in his chest then caused him to make a rapid ascent. He was pulled out semi-conscious and apnoeic. He had four grand mal seizures and was given oxygen. There were no neurological defects, other than disorientation, on examination. After 6 hours, during which time he had another three seizures, he was recompressed to 18 metres on O_2 and treated with anticonvulsants. There was no evidence of pneumothorax, and he was eventually treated on an air table at 50 metres, having continued to convulse while on O_2 at 18 metres. He survived, but has subsequently stopped scuba diving.

Diagnosis: asthma, pulmonary barotrauma, CAGE.

Summarized from *SPUMS Journal* (1982) **12**(3), 17–18, courtesy of Dr David Clinton-Baker

CASE REPORT 55.3

A male aged 46 years had a lifelong history of asthma, and was a certified diver. He made an uneventful dive to 9 metres for 30 minutes and surfaced, apart from the group. On the surface he began to struggle and those who assisted him stated that he was noticeably wheezing. By the time he was brought into the boat he was in cardiopulmonary arrest, and resuscitation efforts were unsuccessful. The forensic pathologist described the death as "status asthmaticus". His usual treatment included theophylline and salbutamol.

Courtesy of DAN[19], Record No. 1196.

CASE REPORT 55.4

DMcM, aged 23, was a very fit and courageous athlete, who had mild asthma and was advised against scuba diving. Unfortunately his father, who was a professor of medicine, succumbed to family pressure and wrote a fit-for-diving certificate.

This patient suffered two episodes of a very similar nature. In neither case had he had any evidence of active asthma prior to the dive, and with the second episode he had actually taken a salbutamol spray prior to the dive. They were in similar sites, at depths less than 10 metres. On the first occasion, after 30 minutes he had developed dyspnoea and attempted to return to shore. He had informed his buddy that he was returning to get a salbutamol spray, but he appeared panicky and inhaled seawater. He was then rescued in a comatose state and eventually recovered after a week in intensive care.

The second episode was of a very similar nature, except that he did not recover. The autopsy revealed evidence of drowning, with mild asthma.

Diagnosis: asthma, panic, near-drowning.

The combination of a number of trigger factors in the scuba environment may explain the increased precipitation of asthma while scuba diving. These factors include:

- Exertion.
- Hypertonic saline (seawater) inhalation.
- Breathing cold, dry hyperbaric air.
- Increased inspiratory effort, from regulator resistance and increased gas density.
- Hyperventilation or increased respiration.
- Stress.

The exertion with diving is especially likely when swimming against a strong current, or during rescue attempts while towing a companion. Excessive drag and resistance is caused by scuba equipment and an inflated buoyancy compensator.

Seawater is a hypertonic saline solution, which provokes bronchospasm in some asthmatics. It is sometimes used as an asthma provocation test akin to histamine, methacholine or exercise challenge.

Scuba divers typically breath air with a low relative humidity, higher density and lower temperature than at the surface. Cooling of the airways results from warming air to body temperature and the heat of vaporization needed for humidification of inspired air. The extent of airway cooling depends on the level of ventilation (usually increased), temperature of the air (decreased with depressurization), and the reduced humidity of the inspired air (during compression, water is extracted). Breathing through the mouthpiece of the demand valve circumvents the normal warming and humidification of breathing through the nose.

The asthmatic already suffers from increased resistance to breathing, but this is aggravated by the resistance from the regulator, increased when the cylinder pressure is low, the increased density with depth and the hyperventilation with asthma and exercise.

Many of the diving accidents in asthmatics have been preceded by use of a 'preventative puffer'. The recommendation that an asthmatic takes a bronchodilator before diving has little to commend it, as it ignores the following:

- The asthma-inducing stressors of diving compete effectively with the anti-asthma medications.
- The aerosol bronchodilator is more likely to reach and relieve proximal, as opposed to distal, areas of airway resistance.

CASE REPORT 55.5

DW, age 20, was a fit young diver who carried out 30 scuba dives to a maximum of 30 metres, without incident, prior to being examined by an experienced diving physician. There was a past history of asthma for which he had used steroid inhalers. On examination there was no evidence of bronchospasm and the FEV_1/VC was 3.9/4.5 without bronchodilators. The chest X-ray was normal. He was advised that he would be medically fit to dive providing he was free of asthma and that he had taken an inhalation of Berotec (fenoterol) prior to each dive.

After undertaking in-water rescue and resuscitation exercises, to a maximum of 5 metres, he developed dyspnoea on the surface. He informed the instructor that he was suffering from asthma and was towed 30 metres back to shore. By then he was cyanosed with wheezing on inspiration and expiration. He then lost consciousness and required expired-air resuscitation (by two novice divers but experienced internists). He suffered a grand mal seizure and then gradually improved following oxygen inhalation. He responded to treatment of his asthma, over the next few days, with aminophylline.

There was no evidence of CAGE, and the seizure was considered to be due to cerebral hypoxia. In retrospect, a history of a mild asthmatic episode four days previously was elicited. It was later ascertained that the asthma, which developed on the surface, followed the aspiration of sea water, exertion and exposure to cold.

Diagnosis: asthma, near-drowning.

CASE REPORT 55.6

JJM, age unknown. This reply, from a physician, was published in the *British Medical Journal* in response to an oft-quoted recommendation that asthmatics could be allowed to scuba dive.

"I have extremely mild asthma, which manifests perhaps once every three years for a brief time during a respiratory tract infection. As I did not encounter any asthmatic symptoms during strenuous high-altitude mountaineering I thought it would be reasonable to try scuba diving. I learnt to dive in a warm shallow swimming pool and experienced no difficulties during this or my first sea dive. During my first deep sea dive, however, I had an extremely severe and sudden attack of bronchospasm at a depth of 30 metres. I barely made it to the surface, where my obvious distress and lack of speech caused my partner to inflate my life jacket, thus compromising my respiration further. It was a frightening experience and I have not dived since".

Reproduced courtesy of *British Medical Journal*[9]

- The aerosol may be effective in allowing the person to descend while breathing relatively normally, but is less effective at the end of the dive when the dive stressors have been influential.
- Most sympathomimetic drugs have cardiac arrhythmogenic effects, aggravating an appreciable hazard that already exists in the diving environment (see Chapter 39).
- Sympathomimetics cause pulmonary vasodilation and reduce the beneficial effect of the pulmonary filtering of venous gas emboli, thereby increasing the risk of arterial bubbles and severe decompression sickness.

CASE REPORT 55.7

FG, male aged 42 years. This diver completed his course with some difficulty, being physically unfit, somewhat obese, mildly diabetic (not insulin-dependent) and not an aquatic person. He also had complained of difficulty with breathing produced by exertion on land, and had lung function tests suggestive of mild asthma.

He took a 'puffer' before his first open water dive – to 15 metres for 18 minutes. This was followed by a sudden and unexplained death, attributed to a cardiac arrhythmia.

Explanation: the use of 'sympathomimetic' drugs, such as the Ventolin (salbutamol) 'puffer', many decongestants and stimulants, are thought to increase the likelihood of ventricular fibrillation – the Sudden Death Syndrome of scuba diving. This is now one of the commonest causes of death in recreational diving.

CASE REPORT 55.8

MB, aged 25 years, was a very fit, mildly asthmatic, sportsman. He had been diving for four months when he went to 18 metres for 20 minutes. Without an obvious reason, he performed a rapid ascent, developing dyspnoea and confusion on the surface and left-sided hemiplegia within a few minutes. He was taken by helicopter to the Navy recompression chamber. He was initially compressed to 18 metres on oxygen, but as he did not regain consciousness he was then taken to 50 metres.

After a 3-day vigil in which the patient was subjected to vigorous attempts to treat him, he died, still under pressure. During that time he was treated conscientiously for his asthma, which was evident on auscultation, impaired ventilation and CAGE. He was given steroids and anticonvulsants (for his repeated epileptic episodes), with measures to counter possible cerebral and pulmonary oedema, maintenance of his electrolyte and pH levels. It was a therapeutic quandary and nightmare.

The autopsy revealed mild cerebral oedema, congestion of the meningeal vessels and ischaemic cell damage in the right frontal lobe and the right thalamus. There was a tear on the posterior section of the upper lobe of the right lung, with intra-alveolar haemorrhages and rupture of alveolar septae. The basal membranes were thickened and muscles showed hypertrophy, consistent with asthma.

Diagnosis: asthma, pulmonary barotrauma, CAGE.

Courtesy of Royal Australian Navy Submarine and Underwater Medicine Unit.

CASE REPORT 55.9

WD, aged 33 years, was a qualified diver for four years, despite being a known, but very mild, asthmatic. He was classified as fit by a doctor who claimed experience in diving medicine. The doctor also gave a prescription for salbutamol, and advised him to take it prior to diving. He followed this advice. He even had a pocket included in his wet suit to hold the inhaler.

He descended to 9 metres for 20 minutes, prior to an ascent to take his bearings. On returning to his companion, he appeared distressed and then did a further rapid ascent to the surface. There, he appeared to be confused and removed the regulator from his mouth. He inhaled some seawater and then lost consciousness and had an epileptic convulsion.

He was rescued by his companion, and within 30 minutes reached the navy recompression chamber by helicopter. He was comatose with brainstem spasms and with bronchospasm and a very inadequate air entry bilaterally. He was compressed to 18 metres on oxygen. Despite endotracheal intubation and 100 per cent oxygen at 18 metres, with positive-pressure respiration, the Pao_2 level remained at 50–70 mmHg. The $Paco_2$ levels were usually above 100 mmHg and the pH remained below 7.0.

Mainly because of the death of an almost identical asthmatic diver, just previously, after a descent to a much greater depth, it was decided to surface this patient over a period of approximately 5 hours, while attempting to maintain as high an oxygen pressure as possible. The problem was in the combination of diagnoses, including cerebral gas embolism (the initial incident), asthma (as detected by the significant bronchospasm) and near-drowning (caused during the surface difficulties and rescue of the patient).

A decision to go deeper, to overcome the effects of the air embolism, would be complicated by prejudicing the Pao_2 level. The greater depth and increased density of the gases would probably interfere with adequate ventilation, CO_2 exchange and acidosis. Aminophylline could cause arterialization of pulmonary emboli. The coincidental hypothermia (33–35°C) was not considered a definite problem, and could even be advantageous – if it was not for the effect that sympathomimetics, required for asthma, could have on cardiac arrhythmias. Steroids were given for rather indefinite but multiple reasons (asthma, cerebral damage, near-drowning).

Initially the chest X-rays verified gross pulmonary oedema, consistent with the combined effects of asthma and near-drowning. Subsequent chest X-rays revealed a persistent right lower-lobe opacity, clearing up over the next month.

With attention to the respiratory status, the brain damage, fluid and electrolyte status, the patient gradually improved over the next few weeks and regained consciousness. The result was a severely brain-damaged young man, continually incapacitated by myoclonic spasms, almost certainly post-hypoxic but possibly contributed to by CAGE. There was a residual dysarthria, a left hemiparesis, an ataxic gait and myoclonic jerks. The EEG was consistent with hypoxia and the CT scan was normal.

He wished to continue scuba diving, and we considered referring him back to the clinician who had originally classified him as 'fit'.

Diagnosis: asthma, CAGE, near-drowning.

Treatment

Great difficulty is experienced in treating asthmatic scuba divers who are rescued and survive until recompression. They may suffer a difficult clinical complex comprising deep unconsciousness from near-drowning and probable cerebral gas embolism, respiratory impairment from near-drowning and asthma.

The problems of combining respiratory support and recompression therapy (restricted to 18 metres because of the requirement for 100 per cent oxygen in the presence of arterial hypoxia), the dangers from arrhythmias, and the possible arterialization of trapped pulmonary air emboli with sympathomimetic drugs, all make these cases a nightmare. Deeper recompression, even though the patient is not responding, may well be a death sentence, and ultimate decompression is daunting with an arterial oxygen tension less than 50 mmHg! Some successes are doubtful achievements with residual hypoxic brain damage.

Occasional episodes of otherwise inexplicable and serious acute decompression illness have been observed. Even accepting that the case reports are incomplete (especially as regards previous medical history), it seems evident from the DAN data that the risk of arterial gas embolism and severe decompression sickness are significantly greater in current asthmatics, with an odds ratio of 4.16. The exact degree of increased risk is a matter of controversy.

Other risks have been observed, and unexplained unconsciousness at depth is one of these.

CASE REPORT 55.10

WS, a very fit dive instructor experienced two episodes of unconsciousness under similar conditions, about one year apart. They both were associated with diving between 30–50 metres depth, non-stressful and requiring little exertion. They both occurred 10 or more minutes after reaching the sea bed, and there were no problems during descent, and specifically no difficulty with middle-ear autoinflation.

Other divers on the same dives used similar scuba equipment and gases from the same compressor (WS's own dive shop) and experienced no difficulty.

The first episode resulted in a sensation of imminent loss of consciousness, to a severe degree and resulted in him ditching his weight belt and ascending, with help. With the ascent he regained his normal state of awareness. On his second episode he totally lost consciousness and

was brought to the surface by one of the companion divers. He was fully conscious and alert within a few minutes of surfacing. Following this dive he was aware of a dull headache.

The only contributory factors that could be ascertained were as follows: He was renowned for consuming extremely small quantities of air and he did admit to employing 'skip breathing' in the earlier part of his diving career – although such a voluntary decision was not made over the recent years. He was also an asthmatic of moderate degree. He had not taken any anti-asthma medication prior to the dives. He then sold his diving practice, and refrained from diving activities.

Provisional diagnosis: a combined carbon dioxide/nitrogen narcosis effect. ?asthma contribution.

Whatever the explanation, remember the maxim that any diving accident not explained and not prevented, will recur under similar conditions.

There is no specific disadvantage in the use of inhaled steroids, taken in moderation, and the hope is that some of the new anti-asthmatic drugs now under development will totally block the hyperreactive and bronchoconstrictive effects of asthma. When this is achieved then the specific provoking factors associated with scuba diving may be blocked and, if no lung damage has occurred, the optimistic hopes of safe diving for asthmatics may be realized.

CONTROVERSY

Controversy currently exists regarding the medical fitness required for asthmatics to scuba dive. This arises historically because of a variety of different mind sets employed in the diving medical community, but they can usually be recognized as follows:

1 A rigid approach whereby all asthmatics and previous asthmatics are automatically unfit, based on both the beliefs and experiences of diving physicians of the distant past, often in the military. The reasons given include the hazards of diving with asthma causing medical complications. It is probably also related to the non-voluntary nature of diving activities in the military, and the unacceptability of having a diver not wishing to dive, because of a self-perceived illness. It is for this same reason that most medical problems which are intermittent and which could incapacitate divers, are deemed unacceptable to the military.
2 Diving physicians with long 'hands-on' involvement in diving activities. These physicians have taken detailed histories of divers encountering problems because of asthma, as well as being involved in the treatment of divers with asthma in recompression chambers. They have described the aggravating and precipitant trigger factors of asthma during diving, and the consequences to the diver. It is from these observations that the case reports, described above, are selected.
3 Investigation of diving organizations. Unfortunately, there is a wide range of such investigations, varying from the fatality statistics based on newspaper articles to the more extensive – but still incomplete – analyses of diving fatality investigations (see Chapter 46).
4 The avante garde physician, often desperate to obtain or retain a reputation in a fringe field of medicine, preferably one not requiring academic qualifications or critical peer review.
5 The academic, who can come from any of the previous groups, searching for a *cause celebre.*
6 A diving physician, genuinely searching for an answer from the available but inadequate data.

POPULATION STATISTICS

It has been estimated that 4–7 per cent of the population has clinical asthma. However, the incidence in Australia and New Zealand is higher,[2] and is increasing in children; there is less evidence of such an increase in adults, however. In one symposium[13] on asthma and diving, Professor Des Gorman referred to an 'infamous UK epidemiological survey which I

believe is a role model of how not to do such studies'. Unfortunately most of the other population 'statistics' available are no better, do not warrant any extrapolations, and probably would not pass adequate peer review.

The incidence of asthma in divers is not known. Self selecting surveys – such as those asking a large population about a disease X – will automatically select disproportionately more responses from people with disease X. Such surveys often also suffer from a very low percentage response rate, and are virtually valueless or misleading in determining prevalence figures. A number of such surveys have been performed.

Questionnaires sent out in a skin diving magazine with a response rate of less than 5 per cent, indicated that 3.3 per cent of the respondents were current asthmatics.[14] A UK prevalence of asthma in about 4 per cent (31/813) of divers, was deduced from a survey of selected diving physicians,[15] though details were not available to identify whether these were historical (childhood) or current asthmatics.

Surveys with a higher percentage respondence among divers still have the disadvantage of screening a survival population (e.g. those who have not died or retired from diving because of the illness under question or from prudence).

Some authors[3,4,10] assume, against all logic and even their own statistics,[14] that the diving population (trainees or experienced) has the same prevalence of asthma as the normal population. One would expect that many asthmatics would not wish to even contemplate the possibility of scuba diving, because of increased respiratory difficulties. One review[10] which is frequently quoted, claims a prevalence of asthma in divers as 5–8 per cent, but this is not evident from the references cited.

Most of the current statistics are contaminated by selection of the data, or not discriminating between a past history of asthma and current asthma. Frequently these reports of population statistics are in the form of abstracts (no detailed data available), or unaccompanied by details of the questionnaire supplied.

A more comprehensive UK survey[16] was also less biased as it did not focus on asthma, and analysed 2240 responses (21 per cent); this indicated an asthma prevalence of 1.7 per cent amongst divers.

A DAN survey[11] indicated that 13/696 respondents were currently asthmatic (defined as having an attack within one year or using bronchodilators). This equates with a 1.9 per cent incidence, but there was also a 5.3 per cent incidence of respondents with a past history of asthma.

An analysis of the DAN membership survey,[17] in a 1989 report by Wachholz indicated that only 1.2 per cent of the 2633 respondents were active asthmatics, and 3.9 per cent had a history of asthma. The DAN survey had a 11 per cent response and was multifactorial, avoiding specific self-selection of respondents for asthma.

In Australia, where pre-diving medical examinations are mandatory for scuba training, 68 of 2051 (3.3 per cent) admitted to a history of asthma – some of which were childhood. Only 1.4 per cent gave a history of asthma since childhood, but among those who gave a history of childhood asthma only, 50 per cent were ultimately considered fit to dive. Parker's survey[18] verified not only the sometimes innocuous nature of the asthma history, but also that the incidence of asthma in diver trainees did not reflect the normal adult population.

DEATH STATISTICS

Again, these are not comprehensive. Although some authors quote the large US fatality statistics, based originally on University of Rhode Island studies, the compilation of these data in fact allowed hardly any conclusions regarding such contributing factors as asthma. As most of the information was obtained from newspapers, the likelihood of a documented medical history was close to nil. However, this has not prevented some authors from quoting these figures, ignoring the statistical adage that absence of evidence is not evidence of absence.

A more recent collection of US fatalities from the DAN[19] database are more reliable, as at least some attempt is made to obtain a past medical history. This was admitted to be not very successful, however. In this survey four of the 60 cases were recorded as having asthma (see Case report 55.3). During the last year of the fatality statistics from DAN (1998), of the deaths on which information was available, 7 per cent were taking medication for asthma.

One series of 100 consecutive recreational diving deaths was recorded in Australia,[20] and an active attempt was made to obtain the full medical history of all the divers, together with an assessment of the various factors contributing to the diving death. It revealed that eight (of the 49 cases in which the medical history was available) had asthma as a likely contribution to the death. One other case clearly had asthma, but because it had never been diagnosed as such, was recorded merely as a 'respiratory disease contribution'. This would suggest that a minimum of 9 per cent of the deaths had asthma as a contributing factor in this series. In 51 of the cases the past medical history was unavailable, and was not forthcoming for a variety of reasons, including medico-legal. Thus, the true incidence of the contribution of asthma to diving deaths could be well in excess of the 9 per cent.

In comparing the Australian data on diving deaths, with the presumed incidence of active asthma in the diving population (see above), it was concluded that the asthma cases were more highly represented, than they should have been by chance alone. This is also consistent with the individual clinical cases demonstrating the existence and effect of the asthma. The death statistics highlight the difficulty in obtaining precise information, and also the time-consuming nature of this approach.

A microcosm of the problem is evident in the New Zealand reports. Those who denigrate the contribution of asthma to diving deaths, quote a figure of 1 out of 11,[21] but those who are more impressed with its relevance quote 5 out of 20.[22] The difference in the statistics may reflect the thoroughness with which the medical history is sought.

Insisting on autopsy data to implicate asthma, as has been suggested by some, is illogical. The first pathological sign of an acute episode of asthma, is eosinophyllic infiltration of the mucosal lining of the lungs.[23] This usually takes some 24 hours to develop, and so an acute attack initiated during diving would therefore not likely be detected at autopsy. Evidence of chronic asthma effects, albeit indicative of the disease, do not necessarily imply that this has contributed to the death.

Prospective surveys of asthmatics who do dive, have been under way now for more than 10 years.[24] Apart from the obvious selection, i.e. these are 'survival' statistics, this approach could produce valuable information, both negative and positive, if the researchers are fully informed by the divers, impartial, and adhere to conventional statistical guidelines.

INVESTIGATORY TESTS

These can be subdivided into the basic expiratory spirometry, performed in most clinicians' offices, flow–volume curves and more sophisticated pulmonary function tests as performed in lung function laboratories, bronchial-provocation tests, chest X-rays and scanning techniques.

Lung function tests

Expiratory spirometry[2,3,13,25–27] is now performed readily and relatively cheaply by the use of either the traditional Vitalograph or more sophisticated digital expiratory spirometers that not only record the volume and the flow data, but also convert this into a percentage of the predicted level according to the age/sex/height of the diver.

The use of the PEF meter has now been relegated from the clinician's office to the patient. Repeated measurements of the PEF, showing not only the actual flow rate but its variation, can indicate the need for treatment. It should not be used as a single test diagnostic instrument.

The Vitalograph, which was very popular in the 1970s, measured the FEV, usually over a 1-second time period (FEV_1), and the vital capacity (VC), i.e. the maximum amount of air able to be exhaled. The ratio of these two, the FEV_1/VC was an estimate of overall lung efficiency, and if it was more than 2 standard deviation (SD) below the mean, then it was considered abnormal.

Previously, a reduction in the FEV_1/VC ratio to <75 per cent was thought to indicate a probable degree of airway obstruction, and this is usually so. However, elite athletes with extremely high VCs could, because of the latter, reduce the FEV_1/VC percentage level. Thus, this ratio should be treated with caution to ensure that a low figure is not due to the large lungs seen in these athletes.

Investigations of submariners[28] exposed to free ascent training, compared the results of the FEV_1/VC measurements with the incidence of PBT. The investigators concluded that there was not a relationship between a low FEV_1/VC ratio and this disorder, but there was an association with a low VC. As this report has been used to discredit the value of the FEV_1/VC ratio as a criterion for selecting those predisposed to PBT, it should be remembered that all these candidates were very carefully selected beforehand, to exclude lung disease and pathology. Thus, it is a survey of demonstrably normal healthy submariners. These candidates not only had no medical history or physical examination suggestive of lung disease, but were also subjected to much more sophisticated lung function tests, including provocative testing with exercise and lung volumes with helium dilution and gas transfer analyses, when required, to ensure that normality. It is not surprising, in this study, that a reduced FEV_1/VC ratio had a poor predictive value for PBT, as all the cases that would have been identified by this ratio were already screened out, or were verified to have no pathology.

As regards the relationship with low VC, Harries[13] noted that "anything that might affect someone's vital capacity, forced or otherwise, would tend to make it small rather than large", and therefore the occasional candidate with lung pathology that did manage to slip through the selection net, might have been picked up by the lower VC.

In an unselected population, such as those wishing to undertake recreational diving activities, the FEV_1/VC measurements and ratio should still be performed, to detect the occasional trainee with lung pathology.

The **mid-expiratory flows**, as measured by the FEF_{25-75} or the MMEF, provide an excellent indication of obstruction to airflow. The MEF_{75}, MEF_{50} and MEF_{25} respectively give measurements of obstruction to the large, medium and smaller airways. A value 2 SD below the predicted, would be considered abnormal.

An obstruction to airflow would be expected to indicate a propensity to PBT, and an investigation of pulmonary function in divers who developed this condition[29] demonstrated reduced compliance at maximum inspiratory pressures. This, and hyperinflation, is characteristic of asthmatics.

A more recent and valuable survey performed by German diving clinicians[27] supported this concept. These workers analysed 15 consecutive cases of PBT, investigating clinical, radiological and lung function information. The data were then compared with data from 15 cases of decompression sickness. A comparison of the pre-dive lung function between the groups showed a significantly lower MEF_{50} and MEF_{25} in the PBT patients ($P < 0.05$ and $P < 0.02$, respectively). Of the 15 cases of PBT, two had a past history of asthma. As all the divers had passed medical examinations for fitness, and a number were professional divers, this group would have to be considered as selectively excluding some with previous lung damage, or asthmatics.

These investigations support the use of lung function tests in the selection of divers to reduce the risk of PBT.

In assessing divers to ensure that their lung function is normal, a reduction of the expiratory spirometry levels below 2 SD (or at the 5th percentile level) have been recorded by Neuman[3,13] as follows: FVC below 75 per cent of predicted, FEV_1 below 80 per cent, FEV_1/VC below 85 per cent, and FEF_{25-75} below 65 per cent. Above these predicted levels, the results could be considered normal.

Bronchial provocation and dilator tests

Some tests[23,26,30] provoke asthma in those so inclined, by inducing hyperresponsive activity of the airways.

Pharmacological stimuli – methacholine and histamine – act directly on the airways to induce bronchial hyperreactivity.

Physical stimuli induce airways drying and changes in osmolarity by means of exercise and hyperventilation. As the major effect is in the evaporation from the airways, the dryness of the inhaled air and the degree of ventilation are the major determinants. Hypertonic saline inhalation has a similar result. Exercise inducing a (dry air) ventilation of about 20 times the total volume, for 6–8 minutes, will produce a 10 per cent drop in the FEV_1 in asthmatics, 10–20 minutes later. Isocapnoeic hyperventilation with cold dry air has a similar effect, and is a less unpleasant investigation. Non-isotonic aerosol

sprays, such as hypertonic saline, provoke a similar response in asthmatics.

Recently, mannitol has been demonstrated to be a stable and predictable provoking substance which can be used in consulting rooms, with good reliability.

The reduction in flow rates using these provocation tests is detected by serial measuring of the FEV_1 or MMEF, although originally the PEF was used.

The chemical challenges were found to be more sensitive than the physical stimuli, and had more false-positive results.

The **hyperventilation provocation** with dry air has some face validity for scuba, as the increased density of the breathed gas with increased depth, together with the increased ventilation with exercise and anxiety, will have a comparable effect in dehydrating the airways and triggering asthma.

In the air-conditioned clinic, breathing cold dry air, **exercise-induced asthma** can be verified by repeating expiratory spirometry tests while the candidate undergoes a reasonably strenuous exercise such as bicycling at 900 kpm/minute for 5–6 minutes. This is equivalent to a 1-knot swim, which most divers encounter during diving. Subsequent detection of rhonchi on auscultation (more obvious with hyperventilation) or a progressive reduction in lung function (MMEF, MEF_{50}, MEF_{25}) should provide sufficient indication that the candidate is hyperresponsive and should not proceed with a diving course.

Another clinical test for asthma is by using a rapid-action beta-2 agonist such as salbutamol and observing a change in expiratory spirometry 15 minutes later. An increase in FEV_1 by 15 per cent is considered positive.

CONSENSUS ATTEMPTS

A **Medical Seminars Inc.** symposium on 'Are asthmatics fit to dive?' was held in 1995.[13] This was an open symposium, and perusal of some of the unsubstantiated claims in the transcript of the seminar led to doubts regarding the expertise available – in both diving and respiratory medicine. A concluding statement was not regarded as a consensus, but more as a basis for possible future agreement:

1 The problems for an asthmatic recreational diver may include:
   – a greater risk of PBT or embolism;
   – a greater risk of decompression sickness;
   – limited exercise capability under water;
   – the drugs may reduce the effectiveness of pulmonary filtration.
2 Current policies may be effective in reducing apparent asthma-related incidents, but paradoxically they increase the hazard for asthmatics, by discouraging medical assessment and advice.
3 Although asthma is an absolute or relative contraindication according to many guidelines, determined individuals evade the medical scrutiny.
4 A history of childhood asthma alone is not significant.
5 Hyperreactivity can be stimulated best by evaluation of submaximal exercise. It may be triggered by histamine or methacholine.
6 Ventilatory capacity is best assessed by exercise capacity.
7 The diving candidate with some asthmatic history is assessed first by demonstrating normal pulmonary function at rest (FVC, mid-expiratory flow, FEV_1, FEF_{25-75}) and then again after exercise.
8 Experienced and healthy divers who acquire adult-onset asthma can still dive safely if they have no abnormal pulmonary mechanics before and after exercise.
9 Chronic asthma, if quiescent and with normal lung function, is acceptable even if corticosteroids are required.
10 Acute asthma is an obvious immediate contraindication, but recreational diving can resume when pulmonary function (FVC, expiratory flow, FEV_1, FEF_{25-75}) has returned to baseline.
11 Providing asthma manifestations are completely controlled and pulmonary function is normal (as no. 7), then the slightly increased risk for an asthmatic individual is not supported by sufficient data to exclude them.
12 The competency of medical assessment is enhanced if the doctor applies an understanding of diving medicine.

The Thoracic Society of Australia and New Zealand[31] conducted a symposium (1993) on the respiratory fitness to dive, so that diving candidates can be advised on the risks associated with respiratory disease, particularly asthma. This was a closed symposium, and restricted to thoracic physicians, occupational and diving medicine physicians. A summary of its conclusions and recommendations are as follows:

1 Abnormalities in the respiratory system place the intending diver at an increased risk of drowning, PBT, and potentially fatal arterial gas embolisms.
2 Spirometric tests before and after bronchodilation should be performed on all intending divers. If there is an increase in the FEV_1 of more than 15 per cent after bronchodilation, the physician should inform the subject that he/she may be at increased risk as a diver. Bronchial-provocation testing should then be performed. If the FEV_1 and FVC are more than 2 standard deviations below the predicted value, or an FEV_1/FVC ratio is less than 75 per cent of the normal value, he/she should be alerted to the increased risks of PBT. Many good athletes have a low FEV_1/FVC ratio because of the exceptionally high FVC. In this case, the maximal flow–volume curve has a normal shape, and it would be inappropriate to suggest that these people would be unfit to dive.
3 Divers with a history of current asthma should be advised not to dive. Spirometric tests including flow–volume curves are useful diagnostically, but more sophisticated tests are not warranted when the history is self-evident.
4 With a past history of asthma, within the previous five years, the candidate should be advised not to dive. Bronchial provocation tests may help to demonstrate the airway hyperresponsiveness. In the intending diver a history of asthma symptoms and/or treatment, should suffice to preclude diving.
5 With a history of asthma in the past, but normal spirometric tests and no asthma symptoms or medication required in the last five years, bronchial provocation testing is performed. If this is abnormal, then probably subjects should not be passed fit to dive, even though this is based on theoretical grounds. Otherwise they are presumed to be fit.

In 1991, the Diving Medical Centres of Australia[7] met to determine policy on asthmatics applying to undergo diving training. The following protocol was evolved from this, with some modifications based on the recommendations of Neuman[13] in 1995.

The history of respiratory disorders is complemented by the physical examination and simple respiratory function tests. High-pitched expiratory rhonchi, which may only be elicited during hyperventilation, indicate airway obstruction and preclude diving prior to further investigation. This auscultation should be performed with the subject hyperventilating through a wide-open mouth and larynx.

In assessing the diving candidate with a past history of 'wheeze' or asthma and a normal chest X-ray, the following protocol may be of value:

- History of asthma over the last 5 years — Fail
- Requirement for bronchodilators over the last 5 years — Fail
- Respiratory rhonchi or other abnormalities on auscultation — Fail
- High-pitched expiratory rhonchi on hyperventilation — Fail
- High-pitched expiratory rhonchi 5–10 minutes after exercise stress — Fail
- VC <75 per cent of predicted, FEV_1 <80 per cent of predicted — Fail
- FEV_1/VC <85 per cent of predicted value — Fail
- Expiratory flow rates of <65 per cent of predicted value (basic spirometry MMEF, $MEF_{75,50,25}$) — Fail
- Asthma provocation producing >10 per cent reduction of expiratory flow rates after either conventional histamine, hypertonic saline, exercise, hypercapnia with or without dehumidified cold ventilation — Fail
- If all the above are clear, and chest X-ray is normal permit limited diving to a maximum depth of 18 metres without free ascent practice — Pass

The alternative expiratory spirometry levels, proposed by Neuman (see below) vary slightly from the above, but are fully acceptable to these authors.

CONCLUSION

There is little consensus on which, if any, asthmatics are safe to dive. If there was a past history of asthma, but no clinical or laboratory evidence of it or its sequelae over the last five years, most physicians would accept the candidate as fit. The increased likelihood of asthma recurring in a past asthmatic may indicate more frequent periodical medical checks and monitoring.

Most argue that the respiratory impairment with asthma is unacceptable, with the known but unpredictable and excessive environmental demands on pulmonary function during diving.

Some physicians require only that the candidate appears to have normal lung function (clinical and spirometry) at the time of the examination. This seems illogical in that:

- Asthma is an episodic disease.
- The provoking factors are present in the scuba diving environment, not the consulting room.
- Aerosol treatments pre dive have uneven distribution and effect, with sympathomimetics contraindicated (see Chapter 43).

This problem may be solved in the near future, however, as pharmacological development trials with systemic drugs may soon be shown to have a total or near-total blockage of airway hyperreactivity. If this is demonstrated in the asthmatic diving candidate under provoking conditions equivalent to those of the diving environment, then this whole subject can be reassessed more favourably. Until then, some physicians rely on dubious statistics and arguments to assure asthmatics of their safety for scuba diving.

The conservative, and the author is of this group, are strongly influenced by the clinical experiences of asthmatic divers who have not survived or who withdraw from diving because of their asthma (see Case reports, above).

Physicians who advise asthmatics not to dive, frequently support the candidates' snorkelling and swimming interests as these are usually carried out in a non-asthma-provoking environment – breathing 100 per cent humidified air and not exposed to PBT.

Immersion itself is thought to increase the risks of impaired exercise capacity of divers, and this may be related to the reduction of maximum ventilation incurred by increased airways resistance.

Table 55.1 *Approximate lower limits of normal at the fifth percentile level*[13]

Parameter	Percent of predicted
VC	Below 75
FRC	Below 70 or above 130
RV	Below 65 or above 135
TLC	Below 80 or above 120
FEV_1	Below 80
FEV_1:FVC	Below 85
FEF_{25-75}	Below 65

From Clausen J. Pulmonary function testing. In: Bordow RA, Moser KM, eds. *Manual of Clinical Problems in Pulmonary Medicine*, 2nd edition. Boston: Little, Brown & Company, 1985.

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Cardiac disease

ROBYN WALKER

INTRODUCTION

The existence of significant cardiovascular disease largely excludes a candidate from being declared fit to dive. Diving is an unpredictable sport, and a diver must have sufficient cardiovascular reserve to cope with the physical stresses of for example, swimming against a strong current or providing assistance to a buddy who is in trouble. Psychological stresses such as panic and anxiety also place demands on the cardiovascular system.

The existence of serious cardiovascular disease disqualifies the candidate from diving because of the risk of sudden collapse or decreased exercise tolerance (see Chapter 39). These diseases are responsible for 12 to 27 per cent of the deaths in recreational scuba diving, and over half those in divers over the age of 40.

Any congenital abnormality that interferes with the exercise capacity of the individual, induces arrhythmia or reduces the cardiac response to exercise, precludes diving activity. This would be the case with such disorders as coarctation of the aorta.

HYPERTENSION

In the past, acceptable standards for blood pressure for divers have varied. It is generally accepted that the young initial trainee (in their early 20s) diver's blood pressure should not exceed 140 mmHg systolic and 90 mmHg diastolic, with some flexibility for the older recreational candidate. Australian Standard AS 4005.1-1992 (describing the medical criteria to be used when assessing a recreational diving candidate within Australia) states that the resting blood pressure should not exceed 150/95 mmHg. It is perhaps more appropriate to suggest that the blood pressure should be within the normal range for the age of the candidate.

In assessing the hypertensive diver, the following should be considered: the aetiology (e.g. coarctation, endocrine disorders, idiopathic), severity (end-organ damage), drug therapy and the increased risk of coronary artery disease and stroke.

It is unreasonable to condone diving if there are end-organ manifestations of chronic hypertension, such as retinal changes, left ventricular hypertrophy or dysfunction, or abnormal renal function. These candidates usually have sustained blood pressure elevation, with diastolic values above 100–110 mmHg.

These individuals should be advised not to dive, and should be referred for diagnosis and treatment.

Antihypertensive drugs alter the capability of the cardiovascular system to respond to stress, or exercise. Unless the blood pressure is controlled by diet, weight loss, exercise plus perhaps, a thiazide diuretic, moderate and severe hypertensives should be advised against diving. Thiazide diuretics do not appear to compromise the circulation and with chronic therapy the cardiac output remains normal, the peripheral resistance decreases, plasma renin activity decreases and there is a persistent deficit in extracellular water and plasma volume. Associated hypokalaemia must be treated.

Beta-blockers are associated with reduced exercise tolerance and autonomic nervous system blockade, and the added danger of cardiac deaths if diving is carried out while on these drugs (see Chapter 39). Beta-blockers may also have other side effects, such as bronchospasm. Calcium-channel blockers may have negative inotropic effects and may be arrhythmogenic. Side effects of angiotensin-converting enzyme (ACE) inhibitors include hypotension, dizziness and bronchospasm. The persistent cough which develops in approximately 1–3 per cent of patients taking ACE inhibitors may represent increased bronchial hyperreactivity, which is undesirable in divers.

For the mild hypertensive and the labile hypertensive, individual judgement must be exercised in each case, possibly permitting restricted recreational diving.

The maximal stress exercise electrocardiogram (ECG) is useful in evaluating patients with borderline hypertension. The stress simulates the physical activity required in diving, and the recording of the blood pressure during the test will allow an assessment as to how the individual will respond to some of the diving stresses. The hypertensive individual will show a marked elevation in the systolic pressure, and a small rise in the diastolic. The normal individual shows a rise in systolic to a lesser degree, and a slight fall in diastolic pressure. The stresses associated with diving other than exercise may aggravate this hypertensive response. Individuals with labile hypertension and marked elevations of systolic blood pressure during exercise must be considered with some reservation for diving.

For patients whose hypertension is not associated with end-organ damage, who are adequately controlled by drugs (without any evidence of the above side effects), and who have no excessive risk factors (see Table 56.1), diving may be permitted.

ISCHAEMIC HEART DISEASE

Coronary artery disease is a potentially lethal, but often asymptomatic, disease that may be present from the mid-30s onwards and needs to be detected prior to medical approval for diving. With coronary artery disease, the myocardium is susceptible to ischaemia whenever there is an excessive load placed upon it, whether from physical activity or stress. The latter may produce an elevation in blood pressure, and can be initiated by such factors as cold, anxiety and real or imagined threats. In these individuals, physical activity or elevation of the blood pressure may be required before the disease becomes manifest, and this may be particularly hazardous in the underwater environment.

Significant coronary artery disease may be present despite normal physical examination. Risk factors for coronary disease, such as smoking, hypertension, family history, raised serum cholesterol or triglycerides, should be sought (see Chapter 53, p. 542). If in doubt, a resting and maximal exercise electrocardiogram (ECG) should be performed, and this should not show evidence of abnormal arrhythmias or ST depression.

Myocardial ischaemia may have several manifestations. Symptoms of chest pain or discomfort, dyspnoea or syncope on exercise should be actively sought. Angina pectoris associated with exercise may or may not be associated with a history of myocardial infarction, and is likely to proceed to this condition in the event of severe exercise. A large infarction may result in sudden death.

The resting ECG shows abnormalities only when there is interference with the conduction system, or when significant damage has already been caused to the cardiac tissue. A maximal stress test produces demands comparable to that which is likely to be experienced in the diving environment, and may be appropriate in individuals over the age of 45 years.

Table 56.1 *Risk factors for cardiac disease*

Risk factors for cardiovascular disease
Used for risk stratification
- Levels of systolic and diastolic blood pressure (grades 1–3)
- Men aged >55 years
- Women aged >65 years
- Smoking
- Total cholestrol >6.5 mmol/l (250 mg/dl)
- Family history of premature cardiovascular disease

Other factors adversely affecting prognosis
- Reduced HDL cholesterol
- Raised LDL cholesterol
- Microalbuminuria in diabetes
- Impaired glucose tolerance
- Obesity
- Sedentary lifestyle
- Raised fibrinogen
- High-risk socioeconomic group
- High-risk ethnic group
- High-risk geographic region

Heart disease
- Myocardial infarction
- Angina
- Coronary revascularization
- Congestive heart failure

Renal disease
- Diabetic nephropathy
- Renal failure (plasma creatinine concentration >2.0 mg/dl)

Vascular disease
- Dissecting aneurysm
- Symptomatic arterial disease

Advanced hypertensive retinopathy
- Haemorrhages or exudates
- Papilloedema

Target organ damage
- Left ventricular hypertrophy (electrocardiogram, echocardiogram or radiogram)
- Proteinuria and/or slight elevation of plasma creatinine concentration (1.2–2.0 mg/dl)
- Ultrasound or radiological evidence of atherosclerotic plaque (carotid, iliac and femoral arteries, aorta)
- Generalized or focal narrowing of the retinal arteries

Associated clinical conditions
Cerebrovascular disease
- Ischaemic stroke
- Cerebral haemorrhage
- Transient ischaemic attack

During this stress test there may be several important findings:

- The blood pressure response may be abnormal.
- The cardiac rhythm may show exercise-induced abnormalities, with conduction disorders, premature ventricular or atrial beats.
- The ECG may suggest the presence of myocardial ischaemia, with ST segment or T-wave changes.

Although occasional false-positive results with such examinations may be found, it would be unrealistic to subject such a person to any form of diving without ensuring that there was no demonstrable coronary artery disease. Those with a genuinely positive stress test should be advised against diving.

An occasional false-positive result to the exercise ECG is seen when there is ventricular hypertrophy associated with extreme athletic activity. Other false positives may be noted in other causes of left ventricular hypertrophy or the use of digitalis derivatives, although in both these cases there are adequate reasons for exclusion from diving. Beta-blocking drugs may render the exercise ECG unreliable. Occasional premature ventricular and atrial beats are found in the ECGs of many asymptomatic individuals with no known heart disease, but these do not preclude diving. However, if the arrhythmia becomes predominant with exercise, it may have far more significance.

Serum cholesterols and triglycerides, serum cardiac enzyme levels, glucose estimations and other biochemical tests have been suggested by various authorities for professional diving, and may also be useful in assessing the older recreational diver – especially if other coronary heart disease risk factors exist.

Some physicians allow experienced divers who have had a myocardial infarction to return to non-strenuous recreational diving after a year or more, if there were no sequelae and a normal exercise stress ECG without evidence of ischaemia or arrhythmia

on exercising to at least 13 metabolic equivalents (METS). Other physicians believe that this is poor advice because this disease has a high incidence of recurrence and increased propensity to arrhythmias; still others would require further investigations (e.g. echocardiography or even coronary angiography). The reader is reminded that it is virtually impossible to make early diver activity non-stressful or non-strenuous.

An experienced diver who has undergone coronary artery revascularization and has a normal stress test causes problems because he has an increased risk of myocardial infarction, and arrhythmias are more likely. Also, not all obstructions may be overcome and the re-stenosis rate is high. With internal mammary artery grafting, the pleural cavity is usually invaded leading to the probability of adhesions, thus also precluding diving.

PERIPHERAL VASCULAR DISEASE

Peripheral vascular disease would limit aquatic exercise capability, delay wound healing and perhaps also delay the elimination of inert gas during decompression. Cold water may precipitate Raynaud's phenomenon in susceptible individuals. Peripheral vascular disease has a strong association with concomitant coronary artery disease, cerebrovascular disease and diabetes, though these all require investigation in their own right when assessing a candidate's fitness to dive.

Varicose veins are hydrostatically supported in the water and the main problems would be trauma causing haemorrhage, or infection if skin changes are present. These are preventable with protective clothing.

HEART FAILURE

Individuals with significant left ventricular dysfunction are at increased risk of developing pulmonary oedema when diving. This is aggravated by the increased venous return associated with immersion and the cardiovascular stresses associated with physical activity and psychological stress. Heart failure should be considered a contraindication to diving.

VALVULAR DISORDERS

Valvular heart disease is usually a contraindication to diving. In many cases the abnormal valve will produce a chronic overload on the work of the heart, and this may be aggravated by the effects of stress with increased cardiac demand. Valvular regurgitation, as well as obstruction to forward flow, must influence the capacity of the heart to respond with an appropriate cardiac output to stress and exercise. Turbulence may also increase gas phase separation.

Some authorities feel that the asymptomatic patient with mild aortic or mitral valve regurgitation, which does not limit exercise tolerance, may be passed for recreational diving. The prospect that turbulence across the valve may increase gas phase separation has not been investigated. Nevertheless, patients with aortic or mitral stenotic lesions should never dive because of the reduced fixed output and the likelihood of central blood shifts precipitating pulmonary oedema. Aortic stenosis is also associated with syncope on exertion.

Patients with **prosthetic valves** and/or who are taking **anticoagulant drugs** should also be excluded. Anticoagulated patients are at serious risk of bleeding into the ear with middle- or inner-ear barotrauma and into the spinal cord with neurological decompression sickness (DCS). Coral cuts and minor trauma may also place the anticoagulated patient at risk of uncontrollable haemorrhage.

Mitral valve prolapse is found in about 7–10 per cent of the general population (females: males, 2:1), with clinical evidence (systolic auscultatory click) in one in seven. In some patients mitral valve prolapse is associated with arrhythmias, chest pain and sudden death. The prolapse itself may be of little consequence, but associated arrhythmias are relevant. Their presence may require Holter monitoring for detection. The totally asymptomatic diver with a normal ECG (normal Q-T interval), no arrhythmias and no redundant valve leaflets on echocardiography may be permitted to dive.

ARRHYTHMIAS

Arrhythmias, whether due to coronary artery or other disease, are another cause of sudden death. Especially ominous are the cardiac dysrhythmias resulting in ventricular tachycardia and a reduction of cardiac output. These individuals are in danger of sudden death with any severe stress or exercise. The stress may be either environmentally or emotionally precipitated.

Patients with paroxysmal atrial or supraventricular tachycardia are susceptible to cardiac symptoms that cannot be tolerated in scuba diving (see Chapter 39). Although the diving reflex is one of the treatments for supraventricular tachycardia, it can also precipitate it.

Wolff–Parkinson–White syndrome, with the short P-R interval, is associated with paroxysmal atrial tachycardia, and these individuals can rapidly move into a shock state, with severe hypotension. It should be noted that when left-sided accessory pathways are ablated, an interatrial transseptal puncture is often used, which may create a right-to-left shunt and predispose the individual to an increased risk of DCS.

Sinus bradycardia, with a rate of less than 60 per minute, must also be investigated. The bradycardia associated with athletes will respond to exercise with an appropriate increase in rate, and this differentiates it from the more serious bradycardias due to ischaemia or conduction defects affecting the sinoatrial node, which respond inadequately or inappropriately to exercise. Common causes of bradycardia in a general population are drugs such as beta-blockers.

Conduction defects need to be assessed. Isolated right bundle-branch block in an asymptomatic individual with no other heart disease evident is acceptable, although a small percentage have associated atrial septal defect, and this may need to be excluded. The left bundle-branch block is also occasionally found in the normal population, but is more likely to be associated with coronary artery disease. If, following adequate cardiac assessment, there is no evidence to believe that the left bundle-branch block is other than benign, then the individual may be allowed to dive.

First-degree atrioventricular (AV) block is sometimes a normal finding, but if there is no evidence either in history or examination for cardiac disease, and if there is a normal ECG response to exercise, the individual should be allowed to undertake diving.

Second-degree AV block is much less likely to be of benign nature and usually excludes diving activities. It is sometimes a normal finding in athletic young adults with a high vagal tone; during sleep or at rest, it is vagally induced. The second-degree block of Mobitz type 2 is especially ominous and often precedes the onset of complete heart block. If the conduction abnormality disappears with a mild degree of exercise, the heart rate response is appropriate and there is no underlying disease or drug causing the rhythm disturbance, then the individual can be approved for diving. Complete AV block is a contraindication, because of the inability of the heart to respond appropriately to exercise stress. Prolongation of the QT interval as a result of electrolyte disturbance, antiarrhythmic drugs, or idiopathic in nature, may be associated with ventricular tachyarrhythmias and sudden death and excludes diving activities.

PACEMAKERS

The presence of a pacemaker usually implies the presence of an underlying cardiac disease which in itself will disqualify the patient from diving. Pacemakers vary in the ability to withstand pressure, and in rate-responsive pacemakers the rate response is often lost at depth, only to return on surfacing as long as the pacemaker has not been taken so deep as to cause permanent damage.[1] There are considerable differences in the depths to which pacemakers have been tested: Vitatron Collection II and Vita pacemakers are tested to a pressure equal to 11 metres of seawater (msw); Medtronic recommend that Thera, Prodigy and Elite II pacemakers should not be exposed to pressures greater than 30 msw and the Intermedic Dash 292-03 has been tested to 60 msw.[1] It is advised when considering the fitness to dive of a patient with an implantable pacemaker, the pacemaker manufacturer be consulted for individual information. There is also the possibility of scar tissue associated with the pacemaker increasing bubble formation.

INTRACARDIAC SHUNTS

Congenital heart disease should be carefully assessed. Atrial or ventricular septal defect, even though diagnosed as having left-to-right shunts, is usually bidirectional with a small right-to-left flow during diastole. This could allow bubbles returning to the right heart from the periphery to pass into the left heart and thus the systemic circulation, bypassing the normal pulmonary filter.

In approximately 33 per cent of the population the foramen ovale does not seal at birth, and during changes of intrathoracic or atrial pressure (e.g. with a Valsalva manoeuvre) blood from the right atrium may pass directly to the left atrium through a patent foramen ovale (PFO). This could result in bubbles that have formed during decompression passing directly to the left heart and arterial circulation, bypassing the pulmonary filter. Wilmshurst *et al.*[2] and Moon *et al.*[3] believe that the presence of a PFO seems to be a risk factor for the development of DCS, and in particular DCS occurring early (within 30 minutes of exiting the water) and with symptoms and signs out of proportion to the dive stress. Cross *et al.*[4] however reported 26 divers (10 professional divers logging 650 dives over 13 years, 16 recreational divers logging 236 dives in an average of 7.5 years) with an easily demonstrable foramen ovale who have never experienced DCS.

Whilst one-third of the general population has a PFO, one-third of divers do not experience DCS. Bove[5] has estimated the presence of a PFO produces a 2.5-fold increase in the odds ratio for developing serious DCS in recreational, Navy and commercial divers. However, the statistical interpretations have been questioned and as the incidence of serious DCS in these populations is 2.28/10 000 dives, the risk of developing DCS in the presence of a PFO remains small. Both Moon[3] and Bove[5] recommend against the routine screening by echocardiography of potential divers.

Balestra *et al.*[6] cautioned divers to refrain from strenuous arm, leg or abdominal exercise (e.g. dive cylinder handling or boarding the boat fully geared up) after diving as these activities have been demonstrated to cause sufficient changes in intrathoracic pressure which may promote the shunting of blood through a PFO.

Wilmshurst *et al.*[7] have described a transcatheter occlusion of a PFO in two professional divers who have experienced neurological DCS and who have since returned to unrestricted commercial diving activities.

A consensus opinion on the approach to a PFO has not been reached. However, it is the author's belief that screening for PFO in prospective divers (recreational, military or commercial) is unwarranted. Testing for PFO post episode of DCS also poses ethical considerations, as the test requires the injection of microbubbles which, in the presence of a right-to left shunt, may enter the arterial circulation with initiation or progression of the bubble injury.

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Insulin-dependent diabetes mellitus

Part A: The 'Pros' – 'CHRIS EDGE* AND PHIL BRYSON†
Part B: The 'Cons' – CARL EDMONDS, CHRIS LOWRY AND ROBYN WALKER

PART A: THE 'PROS' – Edge and Bryson

INTRODUCTION

The current recommendation of the large majority of diving medicine physicians is to disqualify any individual with insulin-dependent diabetes mellitus (IDDM) from participating in sport diving.[1] This opinion is echoed and expanded to include people with non-insulin-dependent diabetes mellitus (NIDDM) by the current Recreational Scuba Training Council (RSTC) medical form (Revision 7/90), used by the largest sport diver training agency. In the section 'Guidelines for recreational scuba diver's physical examination', the form states that sport diving is absolutely contraindicated in 'diabetics on insulin therapy or oral anti-hypoglycaemia medications'.

In Australia and New Zealand, the South Pacific Underwater Medicine Society (SPUMS) published a statement in 1992[2] opposing diving for all but the diet-controlled diabetic diver. A paper in the same journal in 1994[3] put forward the case for allowing diabetic divers to dive under certain carefully controlled circumstances, and this generated both assenting[4] and opposing[5] views.

Before 1975, the situation in the United Kingdom was somewhat different. Until that year, certain diabetic persons, whether insulin or non-insulin-dependent, were allowed to dive provided that their

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diving physicians passed them as medically fit. In 1975, a diabetic diver who was diving on a wreck in British waters to 30 metres ascended normally within the no-stop times according to the BSAC/RNPL 1972 tables. On the surface, the diver signalled 'OK' to the boat and appeared to be fit and well according to his buddies, but it was then observed that he was having difficulty in swimming to the boat. He had to be dragged on board where he collapsed. Unfortunately, his problems were put down entirely to him having diabetes mellitus (DM) and a diagnosis of decompression illness (DCI) was not considered. He was not recompressed for many hours, by which time he had become permanently paralysed from the waist down. Later, he committed suicide as a result of his depression at being confined to a wheelchair. The medical committee of the British Sub-Aqua Club (BSAC) of the time then banned all newly diagnosed diabetic persons from diving with the BSAC – a ban that was later extended to most of the existing diabetic divers already diving with the club. However, in 1992, as a result of studies by Moon[6–8] and Wilmshurst[9,10] on patent foramen ovale (PFO) in divers, the case was re-examined. The diabetic diver's symptoms, which had appeared within minutes of surfacing from the dive, suggested strongly that a PFO might be found in the diver. As fortune would have it, the diver's heart had been well preserved, and a large PFO was indeed found. Expert opinion was given that a cerebral arterial gas embolism due to bubbles passing through the PFO was a much more likely cause of the problems suffered by the diver.

In addition to this case, Dr Claire Eno, a diabetic diver, had conducted in 1987 a questionnaire survey on the UK diving population. The results showed that, despite the ban on diabetic divers, a small number of them were still diving covertly within the BSAC. The divers who responded indicated that they were all managing to control their diabetes so that they could dive safely and enjoyably. This survey was biased in many ways, not least because it was only those diabetic divers who were diving safely who were likely to reply, but it did give a useful indication that it might be possible for some diabetic divers to dive safely.

After careful consideration of the whole problem of diabetic persons learning to dive, it was decided by the UK diving associations [the BSAC, the Sub-Aqua Association (SAA) and the Scottish Sub-Aqua Club (SSAC)] that certain well-controlled diabetic people would be allowed to dive. Guidelines were drawn up and published[11] (see also Appendix A), and a system of data collection was started. These data would be used to determine whether diabetic divers had more incidents either in or out of the water than normal healthy divers.

A conference held in Edinburgh in 1994 discussed the medical assessment of fitness to dive. It was clear that the majority of diving physicians present believed that diabetic persons should not be allowed to dive.[12–15] It is interesting to note that there were no data put forward at the conference to support this view. At this meeting, notice was given of the data collection on diabetic divers by Edge and colleagues.[16] Dear and colleagues from the Divers Alert Network (DAN) also announced[17] a similar plan at the Undersea and Hyperbaric Medical Society conference that year.

PATHOPHYSIOLOGY

In DM, the fundamental defect is in insulin secretion and/or action. In the classical young-onset form of the disease (type 1), there is near-total insulin deficiency, with inevitable widespread metabolic changes. Almost invariably, this means that the diabetic person must inject variable amounts of insulin at regular intervals to counteract the deficit, thus giving rise to the term IDDM.

In the type 2 form of DM, there is resistance to secreted insulin, and this leads to hyperglycaemia and hyperinsulinaemia resulting in obesity, hypertension and dyslipidaemia. When this form of DM is treated by controlling the diet alone, or by means of diet control and oral medication, a diabetic person is said to have NIDDM.

PHARMACOLOGY

The first preparations of insulin were impure and short-acting, but later preparations were purified to improve efficacy and tolerance. Their form was altered to lengthen the duration of action, analogues

were synthesized, and insulins with the human amino acid sequence were manufactured. Most insulin used in the UK is pure 'human' insulin, manufactured using recombinant DNA technology, although highly purified pork and beef preparations are still available. Several preparations have been developed, including quick, intermediate, and long-acting forms. Quick-acting (soluble) insulins are absorbed after subcutaneous injection at a rate which is a function of the time for dissociation of the hexameric pharmaceutical insulin to single (monomeric) units. This is necessary before absorption into the circulation can occur. The longer-acting preparations are intended to supply basal insulin to control blood glucose concentration between meals, but absorption still varies greatly and there is no homeostatic mechanism, so glucose concentrations fluctuate widely in many patients.

The most common oral medications used to treat NIDDM are the sulphonylureas and the biguanides. The **sulphonylureas** appear to act in at least two ways: first, they sensitize the cell to the stimulatory effect of glucose (and perhaps other agents) on insulin secretion. This causes a left-shift in the dose–response curve of insulin secretion against glucose concentration, which may persist for years. They also appear to 'up-regulate' the insulin receptors in peripheral tissues, increasing their number and so magnifying the effect of the available insulin. The **biguanides** act by reducing the efficiency of ion exchange across membranes, with resulting decreased cellular efficiency, so that both hepatic gluconeogenesis and the yield of chemical energy from peripheral utilization of glucose are decreased, with a resulting fall in blood glucose.

In the last few years, a new class of agents, known as peroxisome proliferator activated receptor (PPAR) gamma agonists has been developed. These compounds are also called thiazolidinediones or glitazones. The **PPAR gamma agonists** are thought to enhance the actions of insulin, thereby increasing insulin-dependent glucose disposal and reducing hepatic glucose output. The result is better glycaemic control by improving insulin sensitivity at sites of insulin resistance, namely fat, liver and skeletal muscle. Importantly, at the 1999 American Diabetes Association Conference, evidence was presented that the glitazones decrease the number of hypoglycaemic episodes experienced by patients compared to a group of patients on sulphonamides. Hypoglycaemia symptoms were reported in 12 per cent of glibenclamide-treated patients compared with less than 2 per cent of those taking 8 mg of rosiglitazone per day. It is probable that many more people who wish to scuba-dive will be taking this class of drug, either as a sole anti-diabetic agent or in combination with other medication, but further research will be necessary before the pharmacological profile of these drugs is known when diving.

There are theoretical concerns about the absorbtion and utilization of insulin and, to a lesser extent, the oral hypoglycaemic and biguanide agents when the subject is either under pressure and/or exposed to a higher partial pressure of oxygen. One particular concern that has been expressed is the effect of pressure on a depot injection of long-acting insulin. The studies of diabetic subjects in the hyperbaric chamber and in open water would indicate that from a practical viewpoint this does not appear to be a problem.

ACUTE COMPLICATIONS OF DM

In diabetic people taking insulin or, much less commonly a sulphonylurea or glitazone, there is the potential for blood glucose to fall quickly over a period of a few minutes. Such an acute situation may occur if an otherwise healthy diabetic person:

- is given an overdose of insulin, sulphonylurea or glitazone or is taking a long-acting sulphonylurea (e.g. chlorpropamide) without sufficient glucose intake to correct for this;
- does not have a sufficient food intake for the normal dose of medication that has been prescribed;
- The peripheral tissues metabolize larger quantities of glucose, as may happen during exercise or shivering. Scuba diving involves much muscular activity, particularly if the diver is forced to swim against a current or to rescue a dive buddy. In colder waters the potential for shivering, even in a drysuit, is great – especially if the diver has to remain in the water for a long period of time before being picked up by the boat cover.

The signs and symptoms of hypoglycaemia that may occur in the diver include:

- **Adrenergic symptoms:** sweating, tremor, and palpitations; and
- **Neuroglycopaenic symptoms:** poor concentration on the task in hand, double vision, tingling around the mouth, fits and coma.

Some of the symptoms may mimic other medical conditions brought about by diving. For example, tingling around the mouth or the occurrence of a fit might be thought to be due to hyperoxia in the diver, especially as the use of hyperoxic gas mixtures is gaining in popularity among the sport-diving fraternity. Other symptoms may be confused with DCI or vice-versa, as in the case sited in the Introduction. Those who argue against allowing diabetic persons to dive all cite the fact that episodes of hypoglycaemia are unpredictable and that the hypoglycaemic diver may therefore drown, as an argument for their decision.

CHRONIC COMPLICATIONS OF DM

The chronic complications of DM are all considered to be due to the development of diabetes-specific microvascular problems. These problems do not occur without long-standing hyperglycaemia. Subjects with IDDM or NIDDM (including the diet-controlled diabetic subjects that some diving organizations allow to dive) are susceptible to microvascular complications, although subjects with NIDDM are older at presentation and may die from macrovascular disease before microvascular disease is advanced. However, it is worth noting that over 40 per cent of subjects with IDDM survive for more than 40 years, and half of these never develop significant microvascular complications.

There are two chronic complications of DM that are of particular concern to scuba divers: diabetic neuropathy (particularly autonomic neuropathy) and cardiovascular disease.

Diabetic neuropathy

The presence of a diabetic neuropathy in a diabetic diver can cause a number of symptoms that may be confused with DCI. Sensory changes in the distal limbs are commonly found in neurological DCI. It is important that these are not confused with the sensory changes that can occur in DM, which commonly affects the autonomic nervous system, with up to 40 per cent of diabetic patients demonstrating some autonomic abnormality.[18] Autonomic symptoms are uncommon in young diabetic patients, but more abnormalities are found in the older patients and in those with DM for more than 20 years. The prevalence of autonomic abnormalities (defined as abnormal tests of autonomic function, usually based on cardiovascular tests, in the absence of clinical symptoms) is similar in IDDM and NIDDM patients, suggesting that the metabolic consequences of hyperglycaemia rather than the type of DM lead to autonomic nerve damage.[19,20] Such dysfunction may cause a number of problems, including sudden death, loss of any warning of impending hypoglycaemia, abnormal sweating, postural hypotension and heart rate abnormalities. All of these problems could lead to a diabetic diver having to be rescued in the water, with possible grave consequences for any diving buddy as well as the diabetic diver.

Cardiovascular disease

In patients with DM, cardiovascular disease causes an increase in mortality as compared to the non-diabetic population, and there is a large excess of cardiovascular deaths in patients with proteinuria. This increased risk of death is shared by those with impaired glucose tolerance. Symptoms of angina pectoris may be masked in diabetic patients by autonomic neuropathy, and breathlessness may be the only sign of silent reversible ischaemia.

STUDIES IN DM AND SCUBA DIVING

The well-controlled IDDM diver, when not diving, is almost invariably hyperinsulinaemic. Thus, during exercise, the increased peripheral glucose uptake is not compensated for in the normal way by an increase in the hepatic release of glucose, and so hypoglycaemia occurs. The poorly controlled IDDM diver, when not diving, may be relatively

hypoinsulinaemic, and exercise may aggravate hyperglycaemia and ketonaemia. The effect of exercise on IDDM and NIDDM subjects has been extensively researched; for details, the reader is referred to reviews[21–23] and the references contained therein.

The most common causes of hypoglycaemia in divers taking insulin or a sulphonylurea drug are unpunctual meals and unaccustomed exercise. Both situations are avoidable. Education of the diver and adherence to the published guidelines (see Appendices) are essential to try to ensure that the diver is as safe as possible when diving in open water.

Before conducting any trials on diabetic scuba divers, it is necessary to ensure the safety of the divers should any episodes of DCI and/or hypo- or hyperglycaemia arise in the study group. During any hyperbaric oxygen therapy that might be necessary to treat an episode of DCI, there is a risk that diabetic patients may become hyper- or hypoglycaemic. Springer[24] has reported that whole-blood glucose concentration decreased by an average of 2.8 mmol/l in 25 insulin-dependent diabetic patients with long-term complications of DM after repeated hyperbaric oxygen therapies. Therefore, part of the process of ensuring the divers' safety is to check that any blood glucometers that might be used during the hyperbaric treatment of DCI in the diabetic diver group are able to record accurately the blood glucose level in a hyper- or hypobaric environment. Several groups have reported such studies.[24–31] These studies, with the exception of one,[31] have found that the altered partial pressure of oxygen in the environment affects the glucometer under pressure such that the glucometer gives a falsely high reading when whole blood glucose is low. Clearly, this is a problem when measuring glucose concentrations in diabetic patients in the hyperbaric chamber, and due allowance must be made for this.

Edge *et al.*[32] conducted a trial in which well-controlled (defined as no hypoglycaemic episodes within the last 12 months, HbA1c <9.0 per cent, and none of the long-term complications of diabetes) diabetic divers with IDDM were exercised in a hyperbaric chamber at 375 kPa (equivalent to a depth of 27 metres of seawater) for 16 minutes. The exercise was designed to simulate a dive under arduous conditions. The diabetic divers also carried out a control dive performing the same exercise in the hyperbaric chamber at atmospheric pressure (101 kPa). A group of normal age- and sex-matched control divers undertook the same two dives. Both groups of divers were allowed to have a standard breakfast before diving, and the diabetic divers injected themselves with the dose of insulin that they would normally take before a dive. The results of the study produced two important findings:

1 Despite being exercised hard for 16 minutes at 375 kPa, it was over 4 hours before the first of the diabetic divers' serum glucose measurements were less than 5 mmol/l, at which point the diabetic diver would in practise require a further intake of carbohydrate.
2 The blood glucose concentration in the diabetic divers at 375 kPa decreased slower than when the diabetic divers were at 101 kPa, in contrast to the results of Springer.[24] The possible reasons for the discrepancy are discussed in the paper.

At the same time, Lerch *et al.*[33] examined glucose control in a group of seven divers with IDDM compared to a group of seven age- and sex-matched normal control divers during a diving vacation in Papua New Guinea. The subject inclusion and exclusion criteria were defined and were broadly similar to those used in the chamber study above. On the first and second days, one dive per day was undertaken. On the third, fourth, and fifth days two dives were performed and on the sixth day, three dives were conducted. Before entering the water, diabetic subjects were instructed to aim for a minimum blood glucose of 9 mmol/l and a maximum of 12.3 mmol/l. The amount of insulin that each diabetic subject required on the subsequent days was adjusted on an individual basis considering the development of the blood glucose levels during the previous day of diving. The important findings from this study were:

1 The daily dose of insulin, both short- and long-acting, had to be reduced due to higher energy consumption during the dives as well as the need for higher blood glucose levels before diving and for the prevention of post-exercise hypoglycaemia.
2 In addition to the decreased insulin requirements, the daily carbohydrate intake rose significantly

during the study. This rise appeared to be due to a rise in the daily amount of exercise while diving.

3 No emergency glucose was required during the trial. However, this is not surprising as the divers were under constant medical surveillance throughout the trial.

Those diving physicians working in the UK are still trying to determine how Lerch managed to raise the money to conduct a study in such a wonderful environment. Clearly there are further lessons to be learnt here!

The Divers Alert Network (DAN) has been collecting data from diabetic divers. Uguccioni *et al.*[34] studied 16 divers with IDDM for 131 dives. The diabetic subjects all had HbA1c values of less than 9 per cent and were allowed to regulate their own diving. In this study, the fall in blood glucose was very similar for both single and repetitive dives, being of the order of 2.3–3.1 mmol/l, but with a wide variation. Interestingly, nine episodes of hypoglycaemia were reported, of which the nature and exact causal relationship to the diving were not reported, but none of which was said to be related to diving. Additionally, it is not reported in what form the hypoglycaemic episodes manifested themselves.

The studies reported lend support to the idea that diabetic people may be able to dive safely, provided that certain conditions are met. However, all the studies can be criticised from the point of view that the diabetic divers were closely monitored throughout the studies. Furthermore, with the exception of the study carried out in the hyperbaric chamber,[33] the diabetic divers all entered the water with a blood glucose level as low as 4.4 mmol/l, but which was stable or rising pre-dive. The studies are not able to answer the question as to whether a diabetic diver, diving in open water away from medical facilities, can dive safely. Nonetheless they do permit a detailed examination of the blood glucose and other hormonal profiles of divers during a typical diving period.

The answer to the question of whether diabetic divers may dive safely in open water away from medical facilities may come from the long-term study being carried out in the UK by the Diving Diseases Research Centre (DDRC),[16] from which interim observations have been published.[35] In November 1999, the study had collected data from 230 diabetic divers (190 males, 40 females). The age range of the divers is 19 to 69 years, with 10.4 per cent having NIDDM. The total number of dives logged by the divers is 5348, with one diabetic diver logging more than 1200 dives. Over this period, 83 divers have ceased diving for a variety of reasons, but none of them has reported having problems associated with diabetes while diving. The deepest dive recorded in the series is to 40 metres. Eleven respondents had episodes of hypoglycaemia in the past year. Manual investigation of these records, including telephoning the divers, established that seven of the hypoglycaemic attacks were not in any way diving-related. Three subjects had diving incidents, and one subject had a hypoglycaemic attack underwater, which was successfully managed.

During the seven years in which data have been collected, two diving deaths have been reported in the NIDDM group. One suffered a myocardial infarction underwater and was dead on arrival at the surface. The cause of the other death is unknown. The mortality rate in the study group may not be different to that in the normal population, however.

The long-term study also provides some evidence that divers can use glucose paste when underwater to avoid a hypoglycaemic attack. This technique is used by most diabetic divers, and avoids the need to remove the mouthpiece of the demand valve from the mouth. The nozzle of the tube of glucose paste can be placed between the mouthpiece and the corner of the mouth, and glucose paste then inserted into the mouth. This is a common technique used by technical divers to drink during long decompression stops underwater.

CAN DIABETIC DIVERS BECOME SCUBA INSTRUCTORS?

As divers who were diabetic when they started their recreational scuba diving training progress through the personal training levels, it is inevitable that a few will wish to develop their instructional skills. Currently, the position of all diver-training agencies throughout the world is that no diabetic diver can become an instructor. However, within the BSAC,

there are diabetic divers who had trained as instructors to the highest levels prior to them becoming diabetic. Should these divers retain their instructor status, or should they continue to dive but not be allowed to teach?

The diving instructor has a general duty of care to the person or persons he/she is instructing. All recreational diving agencies take this duty of care seriously, with compulsory courses and examinations for the various levels of instructor grading that they offer. For the diabetic diving instructor, there is an added level to this duty of care, which is that he/she must not become hypoglycaemic at any stage during a dive, thereby necessitating rescue either by the diver under instruction or by another diver or member of the surface cover. For a novice diver under instruction the responsibility for rescuing the instructor may be too great, depending upon the skill of the novice and the level at which they have competence to deal with the situation. The chance of a hypoglycaemic attack occurring in the instructor will be greater if he/she is a relatively inexperienced diver, and as the duration of the dive becomes longer.

Within the BSAC, there are known to be three National Instructors (the highest level of instructor qualification within the organization) and one Advanced Instructor who are diabetic and have continued to teach diving at all levels. No reports have been received of these instructors requiring to be rescued, or of them having hypoglycaemic attacks underwater.

At present, the BSAC, the SAA and the SSAC are considering recommendations proposed by the United Kingdom Sport Diving Medical Committee (UKSDMC) that recreational scuba diving instructors who are diabetic should have to provide evidence that they have been able to avoid disabling episodes of hypoglycaemia, both on-shore and in the water for a continuous period of not less than two years. This is a compromise between two views:

1 If a diabetic diver has proved that he/she can dive safely throughout their training, then they should be allowed to instruct, provided he/she is able to attain the necessary skill level.
2 No diabetic diver should ever be allowed to instruct because of the increased risk in the water as a result of being diabetic.

At a time when legislation concerning the rights of disabled persons to be employed has been passed, or is under consideration by many countries, evidence that a disabled person cannot perform a particular job of work has to be justified. It would be unsafe to draw any firm conclusions concerning the fitness of diabetic divers to become instructors from the small numbers of diabetic diving instructors in the industry. However, it would be true to say that there is no actual evidence that a well-controlled, responsible diabetic diver could not be employed effectively as a diving instructor and some evidence that those diabetic divers who are instructors perform at least as well as their non-diabetic colleagues.

CONCLUSIONS

The main reason advanced by diving physicians for not allowing IDDM and NIDDM divers controlled with medication to dive is that there is a risk (the level of which is undefined) of the diver becoming hypoglycaemic while diving, and thereby drowning. Sufficient evidence has been gathered from the studies under medically supervised conditions and the study of diabetic divers in a normal diving environment that, in well-controlled and well-educated diabetic subjects who follow the guidelines, this risk is minimal. The risks of experiencing mild hypoglycaemic attacks underwater are small, but are not negligible. However, the problems arising as a result of a hypoglycaemic attack while diving are decreased when the diabetic diver carries glucose paste in a form which can be ingested underwater. Furthermore, the risk of hypoglycaemic attacks while diving can be further decreased by ensuring that the blood glucose is measured immediately before going diving and again immediately after returning to the boat or shore (whichever is first). The advent of personal, portable, calibratable glucometers has made these measurements much less of a problem than they were a few years ago.

A bigger problem would appear to be the risk of sudden death underwater as a result of an increased tendency for the development of coronary atheromata in the diabetic diver. This risk is higher in those diabetic divers who have NIDDM, whether taking

medication or diet-controlled only. However, there are insufficient data to be able to say whether the risk of sudden death underwater is increased over that present in the normal population, and it must be remembered that 'absence of evidence is not evidence of absence'.

The studies have not shown whether the diabetic diver is increasing his/her risk of developing long-term complications of DM as a result of diving, or whether the severity of any complications that do develop is worse as a result of diving.

In light of the evidence presented above, it is believed that a ban on all diabetic persons diving is unsupportable. If a diabetic person conforms to the guidelines presented, there appear to be no significantly increased risks to that person's health and well-being over that of a normal diver. However, diabetic divers must continue to dive with a degree of caution that is over and above that required by their non-diabetic counterparts. It is known that various diabetic divers will not wish to follow the guidelines, and that is of course their right, but they are not allowed to dive within the club system in the UK. Whether a diabetic diver can go on to become an instructor is more debatable, but at present there is no evidence that a well-motivated, well-controlled individual should not be allowed to do so.

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APPENDIX A: MEDICAL GUIDELINES FOR THE DIABETIC DIVER

1 The medication regime of the diabetic diver should not have altered significantly during the course of the last year.
2 No episodes of hypoglycaemia requiring hospital admission, treatment with glucagon, or help from another person should have occurred in the diabetic diver during the last year.
3 The diabetic diver should not have required hospitalization for any condition related to the diabetes during the last year.
4 The level of control of diabetes should have been satisfactory during the last year as measured by HbA1c levels, which should always be below 9 per cent.
5 There should be no evidence of retinopathy, neuropathy or vascular disease caused by the diabetes.
6 No evidence of microalbuminuria or proteinuria should be present.
7 The physician in charge of the diver's diabetes should confirm that he/she considers the diver both mentally and physically capable of undertaking the demands of sport diving.

APPENDIX B: ADVICE TO THE DIABETIC DIVER

Pre-dive

The diving diabetic should be as fit and mentally prepared to dive as his/her non-diabetic buddy. The diabetic diver should be especially careful with regard to being adequately hydrated, as there is some evidence that the level of hydration affects the chances of experiencing decompression illness. The Dive Marshall should be aware that the diver is diabetic, and should also be informed of the profile of the dive (plan the dive, dive the plan). The diabetic diver's buddy should be a person who is either:

- A regular diving partner and who is familiar with the diabetic person and the problems he/she is likely to experience.
- A trained medic or paramedic who is familiar with the problems of diabetes.

The diabetic diver should carry the following in his/her dive kit:

- Oral glucose tablets or a tube of glucose paste.
- Emergency intramuscular injection of glucagon.
- Glucose oxidase sticks together with the necessary glucometer kit and CLEAR instructions for use of such a kit.

It is essential that there is at least one person in the dive party of the diabetic diver who is able to use and administer the glucose tablets and intramuscular injection of glucagon.

A diabetic diver should probably dive no deeper than 30 metres until a considerable experience of diving and its associated problems have been gathered by the UK Sport Diving Medical Committee. He/she should remain well within the tables or have no more than 2 minutes no-stop time left on a dive computer.

He/she should not dive with another diabetic diver as a buddy.

Safety equipment must be carried, e.g. marker buoy, flag, flares, etc. Long-term build-up of nitrogen in the tissues must be avoided by ensuring that no more than three consecutive days' diving are undertaken, with no more than two dives to be done each day.

It would seem sensible for the diabetic diver to ensure that he/she has a slightly high blood sugar level before the dive by consuming glucose in whatever form takes their preference.

Post dive

On arrival back at the boat (or on shore if a shore-dive) the diabetic diver should check their glucose level and, if necessary, correct it in the appropriate manner. Any adverse symptoms or signs should immediately be reported either to their diving buddy or to the Dive Marshall and should not be passed off as merely 'part of diving'.

It is important to realize that the symptoms of low blood sugar may mimic those of neurological decompression illness or a gas embolism and vice-versa, e.g. confusion, unconsciousness, fits. In this situation, give first aid therapy to the casualty as if he/she had both conditions. Give oxygen therapy and treat for possible low blood sugar.

Treatment of a possible low blood sugar attack

In the event of there being an incident in the water or on the boat, the diabetic diver should be brought to the boat or shore as soon as possible. The blood glucose should be measured using the equipment in the diabetic emergency kit if this can be swiftly performed. Oral glucose should be administered to the diver with low blood sugar if conscious; otherwise, an intramuscular injection of glucagon (1 mg) should be given. Medical attention and recompression facilities should be sought as soon as possible.

PART B: THE 'CONS' – Edmonds, Lowry and Walker

Problems are likely to be encountered when type 1 or IDDM patients dive, and these are outlined in the following sections.

HYPOGLYCAEMIC EPISODES

In well-controlled IDDM, hypoglycaemia is likely to occur during extreme or prolonged exercise,[1–3] unless the insulin dose is reduced or adequate carbohydrates are ingested. The required modification will depend on the degree of exercise, and will vary for each patient. It is the cause of up to 7 per cent of IDDM-related deaths.

Exercise can cause hypoglycaemia due to an increase in muscle glucose uptake during the activity, and for up to 24 hours afterwards. This has implications both for single and repetitive dives.

The adrenergic symptoms of hypoglycaemia include nausea and anxiety. The neuroglycopaenic symptoms include disturbed mentation, behavioural anomalies, headache, paraesthesia, hypothermia, blurred vision, other neurological manifestations, coma and seizures. In the diving environment this translates to increased risks from anxiety (panic), drowning, pulmonary barotrauma during ascent and diagnostic confusion with diving disorders.

The incidence[4] of severe hypoglycaemic episodes requiring assistance, for each 100 patient years, varies from 62 in intensively treated groups to 19 in the conventionally treated groups. Of the severe hypoglycaemic episodes that occurred in the waking hours, 36 per cent were not accompanied by warning symptoms. The ability to recognize hypoglycaemic symptoms becomes less over time.

Hyperbaric exposures of diabetics undergoing oxygen therapy often resulted in an unstable blood sugar control, inducing hypoglycaemia.[5–8] This could be related to the production of plasma glucagon or increased sensitivity to insulin. Kluger's observations[5] were that hypoglycaemia can be rapid and unpredictable, with blood sugars falling from 12 mmol/l to less than 2 mmol/l in the space of 1 hour. The clinical hypoglycaemia manifesting as confusion, agitation and loss of consciousness is real, and often occurs in previously well-controlled diabetics.[6]

The surveys on diving diabetics[9] showed some variation in the insulin requirements of IDDM divers. They had to reduce their short-acting insulin by between 30 and 70 per cent for each dive, and their long-acting insulin up to 50 per cent to avoid hypoglycaemia. The reduction was greater with more dives per day and more diving days. Additionally, they had to increase their daily carbohydrate up to 200 per cent, and their fluid intake levels.

HYPERGLYCAEMIC (ACIDOTIC) REACTIONS

One way of avoiding hypoglycaemia is by reducing insulin intake and consuming carbohydrates, but this increases the possibility of hyperglycaemic states.[1–3]

Features of diabetic ketoacidosis include abdominal pain, vomiting, increased respiration, dehydration, hypotension, collapse, altered consciousness (sometimes leading to diagnostic confusion with drunkenness) and coma. In the diving environment this relates to risks with anxiety (panic), drowning, dyspnoea (over-breathing the regulator), errors in judgement, and diagnostic confusion with diving disorders.

Intense exercise and/or overeating in under-insulinized diabetics (glucose >14 mmol/l) can disproportionately raise the hyperglycaemic and acidotic level in IDDM, [2,3] and is contraindicated. Such levels have been noted in IDDM divers.[9] Similar cases have been described in the diving literature,[10–12] leading to increased respiratory resistance from the equipment, dyspnoea and panic. Others have been observed, but not reported.

LONG-TERM COMPLICATIONS OF IDDM

The major complications are due to a vasculopathy and may produce neuropathy, nephropathy, retinopathy, cardiovascular and cerebrovascular disease. Some of the neuropathies may resolve, while others regress. Probably the major complication is that of an increased incidence of cardiac disease[1] presenting as orthostatic hypotension, arrhythmias and/or ischaemia.

In diabetic patients, coronary heart disease is often asymptomatic; thus, an exercise stress ECG before embarking on an exercise program is recommended for all IDDM patients older than 35 years of age, or for younger patients with other diabetic complications.

All current proponents of IDDM divers exclude those with such long-term complications, and this is especially so with neuropathy and cardiac disease.

DIAGNOSTIC CONFUSION WITH DECOMPRESSION DISORDERS

The problem of treating a case of decompression sickness (DCS) in a diabetic has been referred to in the literature.[12,13] The difficulty in diagnosis was highlighted in a high-profile case[14] in the mid-1970s when a diabetic, undertaking a no-stop decompression dive according to the BSAC/RNPL 1972 tables, completed a normal ascent, but needed to be helped-out of the water, and then collapsed. The confusion between a diabetic induced state (hypoglycaemia or ketoacidosis) resulted in a delay in recompression, permanent paraplegia, and subsequent depression and suicide.

If an unconscious diver is at risk of DCS/CAGE, hypoglycaemia or hyperglycaemia, the confusion between them must increase the danger from all three life-threatening conditions. Less urgent, but still worrying, is the diagnostic confusion between resolving or permanent neuropathies induced by both diseases.

DIVING DISORDERS AGGRAVATED BY IDDM

The concern that IDDM may increase the likelihood of DCS has been implied in the above case report, and addressed by the diving restrictions suggested earlier by Edge and Bryson. Normal decompression requires normal vascularity and circulation.

Other diving problems may be aggravated by, or confused with, diabetes. These include hypothermia, narcosis, seasickness, oxygen toxicity, anxiety and panic, infections, cardiac disease, and possibly others. Seasickness and anxiety will also reduce the efficiency of carbohydrate absorption.

Aseptic osteonecrosis of the humeral head was reported in one IDDM diver following approximately 100 dives, performed in accordance with the US Navy tables and to a maximum depth of 30 metres. Repetitive dives also conformed to these tables. Causes of aseptic bone necrosis other than IDDM and dysbaria were excluded.[15] This disease is extremely rare from such diving exposures, in normal divers.

CONTROVERSY

Any handicapped individual can be subjected to diving with a relatively low risk, if the diving conditions can be meticulously controlled, supervised with adequate safety precautions, and include specialized procedures for rescue and first aid.

An analogous situation exists for the under-aged diver, whereby a diving 'experience' is able to be enjoyed, while the child is otherwise medically fit, under the full control of a diving instructor – extending from dive planning to an environmental assessment and then total dive supervision.

These live-aboard controlled conditions are not present in most diving situations. Diving boats, at least in the tropics, with the effects of seasickness, high temperatures, variable catering, unhygienic conditions, and a physically demanding and unpredictable diving environment, must increase enormously the IDDM problems of both hypoglycaemic attacks and hyperglycaemic (ketoacidotic) reactions.

Diving in the USA, Australia and much of the world, is much less regimented than the well-disciplined BSAC (club diving) of the UK, or of the Caribbean Camp DAVI with its specialized diabetic instructor, physician and nurse. In many dive sites, especially in the Indo-Pacific, the conditions are often not predictable and are sometimes very demanding and dangerous. The physical stresses of diving and its risks are not equitable with other sports. Unlike the athlete on the track or the tennis court, the ocean does not always permit the diver to take 'time off'. Thus, we are in some agreement with Dr Kelly Hill,[12] when he noted that any certification should be based on a handicapped scuba model, not an open-water certification.

In 1995, when the YMCA introduced a protocol for training divers with IDDM, Dr Hill pointed out some inherent difficulties in the protocol:

- The mandate that the diabetic diver must have an 'informed dive buddy' was unrealistic among the diving population, who knew little about diabetes, glucose testing, differential diagnosis or treatment of IDDM.
- The recommended time/frequency limitations, being between 20–30 minutes, may cause problems with group diving, particularly drift diving, and this – together with the restriction of two dives per day – would likely not be observed.
- There seemed no restriction as regards other certifications being given, e.g. junior or advanced qualifications. Juvenile diabetics were less compliant concerning diabetes management, and less likely to be stable. Increased risks were likely in deep, wreck, ice or cave diving – and leadership certifications would be of concern.
- Modifying or omitting prescribed medications, in order to undertake diving activities, were to be devised with the assistance of the diabetic's physician. This physician would usually have little or no experience in diving physiology or medicine.
- No prevention is 100 per cent successful, and the treatment of these diabetic complications during the dive would require extreme knowledge and skill.
- The difficulty in diagnosis if the diver becomes symptomatic or unconscious.
- The significant problem of hyperglycaemia in patients who have reduced their insulin dosage to prepare for a presumed or possible excess effort, is more than a theoretical concern.

Conflicting opinions on IDDM divers are that the diabetic is at no more risk than normal, or that very significant precautions have to be taken when type 1 diabetics dive. We agree with the latter.

If diabetic divers are treated as 'normals', this implies receiving a normal dive certification and being classified and treated as a diver, without specific limitations.

If diabetics were at no more risk of accidents while diving than normal, as inferred by some 'accident statistics', then there is no reason why they should follow the otherwise very reasonable precautions proposed by Edge and Bryson. There would be no reason why diabetics should not undertake all diving protocols, dive together, or be classified as fit for occupational diving or qualified as dive instructors.

It is our opinion, and this may not differ greatly from that of Edge and Bryson, that diabetic divers are a very specialized group. They require specialized and individual training by very knowledgeable dive instructors, working with conscientious and disciplined clients. Once training has been completed, it does not imply that these divers equate with the normal certified diver, regarding risk exposure or routine precautions.

One addition that we would make to their recommendations is to suggest that if glucose is required as a supplement during a dive, then this is a potential emergency and an indication that the dive should be terminated, with control passing to the diver's companion. The diabetic should then avoid excessive or unnecessary exercise.

The authors of Part A of this chapter are astute physicians, but are also enthusiasts. The material described is somewhat contentious and some reservations must be held regarding conclusions that others could draw. A critical review of some of the propositions are appropriate to maintain a balanced approach.

From Part A, one could get the impression that hypoglycaemic episodes have not been reported in association with diving, except for one case. This is also often deduced from a *SPUMS Journal* article[14] where it was stated that 'there have been no reported incidents involving diabetics or their dive buddies (October, 1993)'.

Elsewhere,[16] Dr Edge has made reference to the UK survey, between November 1991 and March 1994, stating that there were two reported incidents of hypoglycaemia noted in the water, on the surface, amongst 'more than 80 diabetic divers'.

Of the 206 IDDM divers in the UK survey, quoted previously, the incidence of hypoglycaemic attacks was reported as 11/206, i.e. five per 100 diabetic years (of which seven were 'not in any way dive-related'). This presumably includes **all** episodes of hypoglycaemia, and compares very favourably with the rates of **severe** hypoglycaemic episodes per 100 years among IDDM patients (requiring assistance and treatment) as recorded in an extensive, multicentre-survey of 1441 IDDM patients in 1993.[4] One would have to presume that the subjects in the UK divers

trial were either extraordinarily well controlled, or that the reporting was inadequate.

There is likely to be considerable bias in retrospective mail or telephone surveys on 'successful' populations who respond to soliciting requests (see Chapters 41 and 55).

In the previously described UK experiments, ostensibly demonstrating the effect of exercise, it is stated that the exercise load was 'equivalent to an arduous swim'. Perusal of the original report[17] does not support this. In fact, 6 minutes of 'work' spread over a 16-minute period might possibly be considered demanding by some indolent divers, but it would certainly not be equivalent to a near-maximum oxygen uptake exertion induced by a long swim, against a current more than 1 knot or while rescuing a companion diver.

To our knowledge, no reports to date have subjected diabetic divers to an arduous exercise task. In the Camp DAVI series the divers were also subjected to minimal exercise stress. From this very competently supervised training camp, the most recent reports [18–20] covers 33 IDDM divers, performing 423 dives. The environmental stress level was minimal. The training was carried out in the gentle, warm waters of the British Virgin Islands, not known for significant tidal currents or difficult conditions. Some of the divers were experienced, some inexperienced, and all stayed well clear of decompression requirements. Although 13 had hypoglycaemic episodes, these did not occur during the dives.

Even though protagonists quote these figures, some other observations must be noted. As the DAVI group are presumably not suggesting that diving prevents hypoglycaemia, one can only congratulate them on their good fortune that the 13 instances were on land. The mean blood glucose levels dropped 2.46 mmol/l, with a range of a 19.7 rise to a 15.8 mmol/l drop. The lowest blood glucose immediately post dive was 2.3 mmol/l. These figures do not engender confidence in the diabetics' safety under more demanding underwater circumstances.

Also, in a previous report,[21] (rarely referred to now) by the same investigators, among 50 DAVI divers, 4 per cent experienced hypoglycaemic episodes underwater (two episodes per 50 diver vacation weeks).

In a survey from DAN,[22] admittedly from a successful 79 IDDM divers who considered that they were controlling their diabetes well, 55 per cent claimed to have experienced hypoglycaemic episodes while exercising, and 15 per cent while diving. It would be informative to find the relative durations of land exercise and diving. Post-dive low blood glucose was reported in 20 per cent. They concluded that 'hypoglycaemic episodes [in IDDM] are common . . .with diving'.

In the last completed year of fatality statistics from DAN (1998),[23] of the cases on which information was available, 11 per cent were taking insulin. One cannot help but believe that insulin-takers are over-represented in these diving fatalities.

The work of a German group, which was also carried out during a six-day tropical island dive holiday on seven IDDM divers,[24] demonstrated worrying hyperglycaemic levels (up to 14.6 mmol/l). They did notice an increased mean air consumption of the diabetic subjects, but attributed this to the greater experience in the non-diabetic controls – as opposed to a worsening of the diabetic state. They demonstrated that it is possible to avoid hypoglycaemia, but at the risk of considerable hyperglycaemia.

Although the specific range of blood sugar (glucose) levels were not always available from the above surveys, those that were given seemed to vary from potentially hypoglycaemic to clinically hyperglycaemic (ketoacidotic) levels. The results available do not reassure doubting physicians that diabetes-related accidents are unlikely.

References made to data on diabetic divers are not particularly convincing. There have really only been three major groups involved in this field. Two have exploited the opportunity of diving holidays in the tropics to generate their 'reports', most of the information is not in the form of conventional medical research papers, and indeed the majority of the 'references' are actually abstracts – the full text of which was not, and never has been, made available. This makes critical peer review particularly difficult.

Investigations on the skills of IDDM drivers have revealed impaired performance on a driving simulator at glucose levels of between 4.0 and 3.4 mmol/l, yet the diabetics did not stop their driving or treat themselves by ingesting sugars until their level was as low as 2.8 mmol/l.

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Age and diving

CARL EDMONDS

PROFESSIONAL DIVING

The age range for the initial training of professional naval divers varies from 17 to 20 years at one extreme, to 35 at the other. In adulthood, age is of limited value in success prediction for achieving diving skills, but the most satisfactory age is in the early twenties. Most professional diving schools prefer to select divers aged 22 to 29 years. Beyond this range the candidate will have a more limited life as a working diver, whereas under the age of 22 years they are usually not experienced enough in their basic work skills, and also perhaps are not as reliable or mature. Failure rates in training professional diving candidates below the age of 19 years makes this practice commercially unprofitable (see Edmonds, 1972).

The incidence of decompression sickness (DCS) (which is very relevant to deep or commercial diving) doubles in divers of 28 years, compared to those of 18 years. The incidence of death from cardiac disease is much greater after the age of 45–50 years, when it is second only to drowning as the cause of death. It has been shown that dysbaric osteonecrosis incidence is related to age (but this may also be due to greater hyperbaric exposure with age).

Beyond the age of 35 years, apart from an appreciable increase in susceptibility to some diving illnesses, there is a probable reluctance to persevere with adverse environmental and social conditions. There is also an increased incidence of general medical problems. In an Australian survey, the incidence of medical disorders causing failure to comply strictly with the Australian Standards for Professional Divers was 45 per cent in the over-thirty-fives. This was compared to a 20 per cent incidence in the candidates in their twenties, and illustrates the high medical standards required (but not always achieved), as well as the adverse effects of age.

THE AGED DIVER

In recreational diving, there is no upper age limit. Especially with experienced divers, they can often modify their diving activities to take into account the limitations imposed by aging.

There is also a tendency for much older people, and especially those associated with yachting, to take up diving as part of their marine lifestyle. Thus we now have people commencing diving who have retired from their normal occupation, and although diving is often a valuable contribution to the quality of life of these people, this places an added burden on the medical examiner.

There are still the same hazards as mentioned above, but because the diving activities can be tailored to the individual, greater tolerance can be allowed in recreational than professional diving. Nevertheless, recreational diving often does not have the same logistical support and safety procedures that are employed by professionals.

With increasing age, there is increasing infirmity and the increased use of medications, both of which can reduce diving safety. Of special note is the prevalence of:

- arthritis and musculoskeletal disorders;
- cardiovascular disorders
- diabetes/obesity;
- cerebrovascular disease and dementias;
- ocular problems; and
- neoplasia.

The reduction in physical fitness, involving both cardiac and respiratory function as well as muscular strength, is likely to restrict considerably the environmental demands that can be met safely. Reduced physiological reserves attributed to aging may also be related to genetically determined disorders or disuse. Preconceived attitudes may reduce the expectations of performance, leading to disuse.

Physical fitness

Maximum oxygen consumption reduces approximately 1 per cent per year of life. In sedentary individuals, the decline is greater in early adulthood and the curve is less steep in later life, but with regular exercise this decline can be halved. The decline is due to both cardiorespiratory and musculoskeletal factors.

Swimming without fins, at a speed of about 30 metres per minute (a 1-knot current speed), requires a metabolic equivalent unit (MET) of 10.0. (One MET unit is defined as consumption of 3.5 ml O_2/kg body weight per minute.)

Cardiovascular

There is an increase in arterial stiffness with aging, leading to an increase in blood pressure and left ventricular hypertrophy. The exercise-induced rise in heart rate declines by 3.2 per cent per decade of life.

Respiratory

There is a decrease in elasticity of the lung parenchyma, and increase in fibrous tissue and increased resistance to airflow, with age. The vital capacity reduces by 30–50 per cent, and there is an increase of residual volume by 40–50 per cent, by the age of 70 years. This produces breathlessness at lesser work loads.

Muscle

Maximum strength is achieved by the third decade, levels off until about the age of 60 years and then declines by 10–15 per cent per decade.

Listing the physiological decrements associated with age ignores the considerable individual variation, and so our assessment must be performance based in each case.

If the aged diver is able to continue to achieve a standard of physical fitness commensurate with diving safety, then the multiple theoretical and practical decrements should not be held against him. Thus, irrespective of age, the diver (either trainee or experienced) should still be able to perform a 1-km swim in less than 30 minutes, or a 200-metre swim in less than 5 minutes, unaided by equipment. If the diver, aged or otherwise, is unable to achieve such a basic standard of physical fitness and aquatic skill, then much diving activity would be unacceptably hazardous.

Diving disorders

Certain diving illnesses are more likely to develop with age. The most important of these is cardiac disease, which is now probably the most common genuine cause of death in recreational diving (drowning only reflects the medium in which the accident occurs). The strenuous exercise required to swim against tidal currents and to rescue other divers, is such that a subclinical obstruction to a coronary artery (80 per cent or greater) is likely to be converted into a clinical case report of the sudden death syndrome.

Although physiological age is more important than chronological age, for divers aged over 40 years it is recommended that regular re-examinations be carried out in order to detect medical abnormalities which may interfere with efficiency and safety in the diving environment. Electrocardiographic examinations during maximal exercise may be recommended as part of each five-year medical, after this age, especially if cardiac risk factors are present.

DCS is also far more common with increasing age, and becomes a considerable handicap beyond the age of 40 years. With increasing age, a more conservative allowance must be made – restricting the decompression schedules. These authors arbitrarily recommend that older divers reduce their allowable bottom time by 10 per cent for each decade after the age of 30 years.

One of the commonest problems with the aging diver, is **presbyopia**. This interferes with the diver reading his meters, gauges and camera settings. It can be overcome by the use of a convex lens, stuck onto the lower rim of the face mask.

Increased **sensitivity to cold** can be partly countered by limiting diving to warmer waters, or by the use of thermal protection clothing.

There are suggestions that repetitive and excessive diving may produce some form of cerebral impairment which increase the effects of aging, but there is little evidence to support such a concept. Indeed, the anecdotal evidence for such a situation is countered by similar anecdotal evidence implying that old divers are an exciting, innovative and socially active group (see Chapter 41).

There are positive aspects to age, including the accumulated knowledge both of diving and other related activities, more prudence and care in dive planning and the choice of diving sites, and possibly more mature judgement. The aged diver is less likely to be swayed by social, peer, financial or ego pressures.

Many divers have continued diving into their eighties, and the social value of diving should not be underestimated. In Australia, there is even a Sub-Aquatic Geriatric Association (SAGA), to which this author has received (junior) honorary membership.

CHILDREN

There is a common belief that children should not exceed 9 metres depth until their bones have reached osteogenic maturity, i.e. when the epiphyses have fused. There is very little evidence that DCS or dysbaric osteonecrosis has influenced bone growth in young animals; nevertheless, that possibility leads to some hesitation in contemplating diving in excess of this depth, because of our limited knowledge.

So far, the youngest child to die scuba diving was aged seven years, but there have been many accidents and deaths between the ages of 10 and 15 years.

The most prudent advice to parents is to encourage children to acquire the aquatic and snorkelling skills at this age, to consolidate the basic capabilities needed later for scuba training.

Official recommendations

The Recreational Scuba Training Council (RSTC) in the USA has made 15 years the minimum age for certification of sport divers. This may be reviewed.

The South Pacific Underwater Medicine Society committee on medical standards for recreational diving met in 1990 and considered this subject. They recommended a minimum age of 16 years for scuba diver training, the decision being based purely on safety factors.

The Australian Standards reduced the recommended age to 14 years, to comply with the diving instructor agencies' requirements. The Australian Standard 4005.1 of 1992 stated that the selection criteria required that the trainee shall comply with the following:

- Be at least 14 years of age, but persons younger than this may in some cases be eligible to train for conditional certification which allows the young person to dive with a certified diver, with the consent of parents or guardians.

Under the medical section of the Australian Standards AS 4005.1 it is stated "Children under the age of 16 shall only be medically examined after consultation by the doctor with the parent or guardian to establish the child's physical and psychological maturity. Between the ages of 16 and 18 it is preferable to consult with the parent or guardian before medically examining the child".

The Australian Surf Life Saving Association, the judgement of which is not influenced by commercial factors but which is very committed to children's involvement, will not allow active life-saving responsibilities until the age of 16 years, and even then only under the supervision of a patrol leader, 18 years or older.

Attitudes

The physicians, who are probably more concerned with safety than some instructors, recommend a minimum age of 16 years, with parental informed consent and approval necessary between the age of 16 and 18 years.

Unfortunately, the Australian Standards were not prepared by diving physicians, and although there was a representative present, he was greatly outnumbered by the industry and diving training organization representatives. These groups have different agendas, and different motivations to the physicians. There was no one present with pediatric and psychological training.

If one looks at the Australian Standards document, it is implied that even they have some concern regarding the child's safety, until the age 18 years. Off-loading the responsibility to parents who have no practical knowledge of the risks of scuba diving was a reprehensible act.

I am surprised to find that they have accepted an under 14-year-old diver to "dive with a certified diver". This is inadequate, as it allows one young 'conditionally qualified' youth to dive with another diver who may be equally inexperienced.

By analogy, would you allow a 12 to 14-year-old child to:

- fly an airplane?
- drive a motor vehicle?
- take out a financial loan?
- be legally responsible for decisions made?
- make medical and health judgements?
- make life-threatening decisions for himself and others?

If one agreed that 12- to 14-year-old children should be restricted in this way, then it would be interesting to compare this attitude with that of a similarly aged child undertaking scuba diving.

Psychological maturity

This is the main reason why we would prefer children not to be given diving certification. Indeed, certification implies that the diver can make informed judgement as regards dive planning, environmental conditions, equipment usage, and the interrelationships of all these. For a dive to be safe, this judgement is sometimes essential. It is related to maturation and experience, not just intelligence.

A child may have difficulty handling the intellectual content of the diving course, but he will have difficulty with its application (abstract thought).

Unfortunately, children do not have the same appreciation of mortality (death) and the implications of morbidity (disease or accidents) as an adult.

Children are more **immature** – that is what makes a child. They tend to be more immediate in their gratification needs, and have a shorter attention span. They are also not as good at long-term planning as adults. Unfortunately, sometimes the long-term planning will not be needed if the child dies or suffers significant damage.

Materialistic factors also come into play. Children are less likely to abort a dive if they have already committed themselves financially or logistically. Judgement comes with age. Older people see death more clearly.

Psychological reactions are also different in children who react with behaviour that, in adults, would be abnormal. They are far more likely to display anxiety or hysterical reactions, and the control of these is part of the maturing process. The appropriate response to a life-threatening situation, or even one that is perceived to be life-threatening, is not to burst into tears. Unfortunately, this is a child's natural reaction, and it is often very successful in obtaining assistance. But tears are not easily seen through a facemask, and in any case, they tend simply to add to the large ocean environment. They do not have the same power under water as they do on land, when the parent(s) is (are) watching.

Children's reactions are certainly rapid, but not always appropriate for long-term health and safety.

Endurance and perseverance are characteristics which develop with age and which take over when panic and tantrums have been controlled. Imagination is an endearing characteristic, but makes kids susceptible to fear and terror.

Children are **dependent**: they mature slowly to become independent, and act responsibly. Thus they are more likely to rely on the statements and decisions of others, as opposed to deciding what they themselves are capable of doing. This might be

acceptable on a trip to the zoo, but it is not good when diving in the open ocean, where divers must be self-reliant, recognize their own limitations, and act accordingly. Divers are responsible for the safety and rescue of their companions. Would you really want a 12-year-old child to be responsible for your safety, or your own child's safety?

Children are suggestible and very easily impressed. They can be intimidated directly by their parents, and also by the encouragement and enthusiasm that their parents may give them.

Thus, the child might well continue an activity such as scuba diving, to please mum or dad, to impress their parents and peers and to gain attention, but these are not good motivations for scuba diving. Kids are very easily intimidated, and for the sake of the child, I would prefer to see an indifferent parental reaction than an enthusiastic one.

Physical maturity

The requirement for good-fitting equipment implies the need to upgrade regularly during the growing years.

There is also the likely problem, sooner or later, of the child having to swim against unexpected tidal currents to return to safety. Some children may have this physical ability, but not have the psychological endurance in such an emergency. Others will have neither. A small child might also have great difficulty in coping with the rescue of a larger 'buddy'.

With physical immaturity, there is also the problem of increased dangers from certain diving medical disorders, including hypothermia, gas toxicities, susceptibility to marine venoms and barotraumas.

Medical aspects

The reason that children get 'glue ears' is that their eustachian tubes are narrower and smaller, and so are their sinus ostia and respiratory airways. Children's upper and lower respiratory passages are narrower by comparison to the air cavities associated with them, and that is the reason why children have more trouble with barotraumas in aviation, as well as diving exposure.

Some diseases, such as asthma, are more likely to occur in young children than in late adolescence, when the airways have grown relative to the lung volumes. That is why children sometimes seem to 'grow out' of asthma.

Many have questioned the safety of exposing children to conditions such as pulmonary barotrauma and arterial gas embolism (one cause of acute decompression illness), especially in children where there is still growth of organs, i.e. where a bubble can do more damage than it would in a full-sized adult.

Such tissues that could be so affected include the brain, inner ear, bone and coronary artery. The worry here is that, for the same degree of bubble development, there might be a much greater ultimate damage.

Another possible factor increasing the risk of neurological DCS is the patency of the foramen ovale (see Chapter 11). It may close late in childhood and thus have an increased incidence in childhood. This is another reason to limit the dive exposure – to minimize the DCS incidence.

When should children dive?

It is our opinion that a child under the age of 16 years should only have 'dive experiences' under the following, moderately safe, conditions:

1 When they want to, without parental or peer pressure.
2 When they are medically fit to do so.
3 That there is a maximum dive depth of 9 metres, to prevent some of the problems referred to above. The 9-metre depth will certainly not prevent a child from developing pulmonary barotrauma, cerebral arterial gas embolism or any of the other respiratory tract barotraumas. It will, however, usually prevent decompression sickness.
4 That they are trained and taken by a qualified instructor, and are under the personal and total control of that instructor (i.e. not three or four trainees together). A buddy line between the child and the instructor is prudent, to prevent uncontrolled ascents.
5 That after this acquaintance dive, all other dives are only to be carried out in calm and good environmental conditions, and with the same controls

as referred to in points 1–4, with an experienced diver of instructor standard taking absolute control.

Giving a certificate to dive to children under the age of 16 years, other than one which stipulates diving under the above very special conditions is, in my opinion, irresponsible.

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Diver selection

ROBYN WALKER

INTRODUCTION

If the medical profession believed totally the recreational dive industry propaganda there would be no need for diving medical standards or medical examinations. Advertisements suggest that everyone can be taught to scuba dive, and furthermore that scuba diving is a fun, safe and easy recreational activity. But what makes a good diver – and more importantly, are there particular individuals who should not dive?

Diving can be a physically demanding and arduous sport, yet little research is available on what essential criteria a candidate should possess in order to become a good diver. Reports of diver selection criteria are mainly anecdotal, and while psychometric studies have been performed, each group has different operational requirements and therefore the results should not be generalized. Most studies infer that the diver should have a psychologically stable or even phlegmatic personality, be able to endure physical and emotional stress, and be free of all serious disease. In appreciating the importance of selection criteria, it is necessary to consider the qualities required for both professional and recreational divers (see also Chapter 45).

THE PROFESSIONAL DIVER

There are relatively few data in the scientific literature on diver selection, but most of what is available deals with professional Navy divers. These divers are subjected to an arduous physical fitness program, and operate under extreme conditions. They are commonly seen as the elite of the Navy and – anecdotally – have egos to match.

Biersner[1] found successful military divers to have a greater incidence of conduct-disorder behaviours (truancy, arrests, etc.) than their non-diving counterparts. He also found that a significant proportion of divers demonstrated antisocial personality traits,[2] and that divers were more likely to be individualists, unsympathetic and aggressive. Others[3] have found a positive relationship between mechanical aptitude and success as a diver.

Beckman *et al.*[4] evaluated Navy divers (who had successfully passed their diving course) and found they were best described as optimistic, independent, self-serving, analytical, and tending toward social aggressiveness. This personality style appears to most closely resemble the non-pathological antisocial personality. Beckman also goes on to say this personality style may be quite adaptive considering the unique nature of their employment. Yet an antisocial

personality creates havoc in team-building, team activities and discipline-orientated organizations.

Whilst most navies require their diving course recruits to undergo a formal psychological screening test, they do not specifically deny entry to a candidate who is somewhat introspective and pessimistic. The screening tests are used to exclude those with increased anxiety and depression levels as well as gross psychopathology. Generally, the best candidates are conscientious, are open to experience (and this generally reflects intelligence levels), are low on neuroticism, and are socially adjusted. Teamwork is most important along with adjustment to military life and authority.

Professional divers must be able to operate under conditions of stress, and do not have the luxury of only diving when they feel like it. Aquadro[5] states that tendencies which mitigate against the ability to withstand stress are: below average intelligence; tendencies to claustrophobia; unhealthy motivations; history of past personal ineffectiveness; difficulties in interpersonal relations; and lack of adaptability.

However, despite the above, it is not possible to select only successful candidates prior to the commencement of the diving course, and professional diving courses still have failure rates of up to 30 per cent. Candidates often say they feel uncomfortable, or it was not what they expected. Previously unrecognized claustrophobia may become evident. Poor visibility, strong currents, hard work, heavy equipment, cold and obvious risk does not equate with the often glamorous, but misconceived, perception of diving.

THE RECREATIONAL DIVER

Recreational divers still require a level of physical and psychological fitness to undertake sports diving. It is argued they can pick and choose when they wish to dive and under what environmental conditions; however, a certain level of intelligence and knowledge of water conditions are required to exercise this judgement. Even then, the ocean is not always predictable.

Motivation for undertaking a diving course should be considered, and perhaps the most important selection criteria should be that the individual is a willing volunteer. Spousal pressure can be enormous. It is most important to identify the scared, reluctant candidate whose only reason for undertaking the course is their partner's insistence. These candidates are at risk of being bullied by their partner, concealing their true anxieties and ending up as a diving statistic. This individual may not feel able to refuse their partner's request; however, if the doctor declares the individual unfit to dive this is often acceptable to the dominant partner.

It may be appropriate to advise couples not to form a buddy pair during their training course. The male partner is often more aggressive, may put his partner's equipment together and generally take charge. This often has the effect of making the female partner feel inadequate for not understanding the activity, but she then is too embarrassed to ask questions. The couple may not always dive together, and both individuals must have confidence in their own individual ability and their ability to take charge in the event of an accident.

All diving candidates must feel comfortable in the water, and in one study poor swimming ability was a major factor in diving course failure. Any recreational activity is usually undertaken for the pleasure it provides. The candidate who is uncomfortable in the water, is a poor swimmer and is physically unfit is an accident waiting to happen.

The commercial recreational dive instructor agencies tend to downplay the risks of scuba diving. However, scuba diving is a high-risk sport and there is evidence that supports panic behaviour as one of the major influencing factors for injuries and fatalities. Individuals with raised anxiety trait levels, in comparison with divers in general, are more likely to experience symptoms of state anxiety and panic episodes when confronted with various stresses while diving.[6] This group of individuals should be discouraged from diving.

Whilst the ideal diving candidate has not been identified, intelligence, adaptability, physical fitness, water skills, low anxiety trait levels, strong motivation and social adjustment have all been recognized as contributing to the production of a good diver.

The diving medical examination (see Chapters 53 and 54) outlines the prerequisite medical standards. The diving medical physician does not have the time

or experience to conduct an in-depth psychological assessment on all diving candidates. The diving instructor is perhaps in a much better position to assess the candidate during the practical evolutions of the course, and should have the ability to detect anxiety or discomfort and discuss this with the student.

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Female divers

ROBYN WALKER

INTRODUCTION

The era of scuba diving being considered a male dominated macho sport is long past. Females account for some 35 per cent[1] of all recreational diving certifications issued,[1] and are employed worldwide in recreational, commercial and in certain specific military diving operations. Whilst anti-discrimination laws and equal opportunity legislation has paved the way, there are important physiological differences between the sexes, and these should be considered when assessing fitness to dive.

HISTORY OF WOMEN IN DIVING

With a history going back some 2000 years, the original shell divers of Asia were both male and female. This changed approximately 400–500 years ago, some say because of the better tolerance to cold exhibited by females, whereas others attribute it to the folklore, that diving affects male virility.

The Ama of Korea and Japan have been famous for their diving capability. They have dominated the breath-hold diving scene, and these women adapted well to their diving activities by increasing their basal metabolic rate, at least during the colder months, up to 30 per cent above normal. This meant that they utilized more food to produce heat and energy to allow them to endure the cold water. To conserve heat they developed increased body tissue insulation, about 10 per cent above normal, a reduction in their blood flow to the skin (30 per cent less than normal) and an ability to tolerate a lower water temperature before shivering developed.

In Western society there were both cultural and legal restrictions on the aquatic activities of females. In the early part of the twentieth century it was customary to have the women swimmers bedecked in a full blouse plus skirt, long dark-coloured hose, rubber bathing slippers and a bathing hat. Presumably only a very competent swimmer would attempt to cope in the ocean with those restrictions!

In the 1940s, Simone Cousteau joined her illustrious husband, Jacques Cousteau, in using and becoming adept with the scuba apparatus, called the Aqualung.

Lottie Haas proved her expertise at diving and underwater photography, despite discouragement

from her husband, the famous Hans Haas. She inspired others with her success in both roles and in her autobiography *Girl on the Ocean Floor.*

In Australia, Valerie Taylor and Eva Cropp led a group of very enterprising and capable women who captured the admiration of the public with their skills and abilities at handling marine animals, immortalized in film and television productions, while Dee Scarr demonstrated the same skills in the Caribbean.

Some female scientists have excited the diving world. Eugenie Clark became known as the 'shark lady', because of her brilliant work in this field, while Sylvia Earle, in 1969, led the first all-woman team of aquanauts in the Tektite 2 habitat experiments. They stayed underwater for two weeks in the Tektite habitat and were acclaimed for their diving and scientific professionalism.

Kati Garner was the first woman to graduate from the US Navy Diving School, which she did in 1973, and many hundreds have since successfully followed her.

Perhaps the most important contribution of women to diving has been in the instructional area, however. Many male instructors used the training period as an ego trip for themselves, denigrating the trainees' apprehensions, and issuing stories to demonstrate the instructor's prowess. With the advent of women instructors there has been a removal of the old 'bravado' image of the instructor. Instead of a glib and depreciating response to questions, the diving trainee is now far more likely to be listened to, have their question considered, and answered in a non-point-scoring way.

About one in three of the current trainees are women, while the ratio of females in the diving death statistics is 1 in 10. Women divers are less likely to die than male divers, but to make the figures more meaningful the diving frequency of the sexes is needed.

ANATOMICAL DIFFERENCES

Compared to males, females typically have a lower threshold for peak sports performance, and generally can produce less power or speed and have a lower work capacity and less stamina. Despite fitness training, females possess a higher percentage of body fat. A 20-year-old sedentary female has approximately 25 per cent body fat, a trained female 10–15 per cent and a trained male 7–10 per cent. Of total body mass, trained males have relatively more muscle (40 per cent) compared with comparatively fit women (23 per cent).[2] This means that a female may find it more difficult to swim against a strong current, and tire more easily than her male buddy.

The female diver may also experience difficulty in lifting and carrying her dive equipment. A fully charged standard dive cylinder can weigh up to 17 kg on land, while the mixed gas rebreather set used by many of the world's navies is 29 kg when fully charged. The buoyancy upon entry into the water alleviates this load, however.

Because women are smaller, and because their lungs are smaller, they require less oxygen and produce less carbon dioxide. Their respiratory minute volume is less, and they need to carry less compressed air. This means that they may not require as large a scuba cylinder and have a lighter load to carry.

Females are able to conserve energy more efficiently than males, and their increased body fat provides better insulation from heat loss and increased buoyancy. Females have a lower basal metabolic rate reducing their caloric need, and also have fewer sweat glands so that sweating begins at a higher core temperature, conserving heat. However, the converse is true, so that a female's susceptibility to heat stress is increased. Overall, women are more susceptible to thermal stress than men due to an increased conductive heat loss due to their slightly higher surface area to volume ratio, and to their smaller muscle mass and less metabolically active tissue to generate heat during activity.[2]

The ability to cope with cold water exposure is an adaptation, i.e. it can be acquired by practice. It is unfortunate that many women seem to be far more sensitive to cold water, probably because of the lack of previous exposures. The Korean Ama proved that females are able to adapt, and it is noteworthy that since 1976 with the introduction of wetsuits these adaptations have been lost.[3] The likelihood in our culture is, however, that the females will expose themselves less to cold water, and therefore be more

sensitive during the diving course. This can be corrected by appropriate training in the aquatic environment prior to the course.

The effect of these physiological differences for recreational divers is relatively minor. Upon entering the sport of diving the average female will be unable to match the performance of her male counterpart, but with training she will soon adapt. Sports divers should plan their dives to avoid conditions requiring sustained maximal aerobic endurance (e.g. swim into the current at the start of the dive and use the current to aid your return at the end of the dive), and should learn to dive within their own physical limitations. Modern diving equipment with properly fitting wetsuits or dry suits should ameliorate the cold disadvantage, and if overheated on the boat the female diver can remedy this by immersion. For the professional female diver individual characteristics and capabilities are more important than alleged gender inadequacies.

MENSTRUATION

Whether a woman dives during her menstrual period, depends on how she feels. Over the three to five days, she is likely to lose between 50 and 150 ml of blood and cellular debris. This is an insignificant amount physiologically, but there has been a great deal of fear that even this small amount may attract sharks. There is no support for this belief, and in fact female divers experience a much lower incidence of shark attack than males. One hypothesis to explain this is that the haemolised blood associated with the menses may act as a shark deterrent, and not the opposite.

For most healthy active women, changes associated with their menstrual cycle are negligible and cause minimal interruption to their lives. Although the menstrual cycle may have some psychological and physiological effects, examination of the statistics in the Olympic Games during the 1970s showed no significant performance decrement at any specific stage of the menstrual cycle. Brown[4] compiled a small survey on female divers and found that 89.9 per cent of females dive while menstruating without any physical or psychological problems. Of those who did not dive, 4.8 per cent were menopausal or had not had the opportunity to dive while menstruating.

Another disorder which sometimes has a marked increase in incidence during the menstrual cycle is that of **migraine**. If divers suffer from this, especially in the premenstrual period, it is worthwhile either avoiding diving or ensuring that they do not do anything that can aggravate or precipitate a migraine-type syndrome (see page 412).

At premenstrual and menstrual periods, there may be a congestion of the mucosal membranes, possibly associated with the oedema and fluid retention. When this happens it may be more difficult to equalize the middle ears (because of eustachian tube swelling), and also predispose towards sinus barotrauma.

It has been conjectured that the fluid retention and oedema that develops before and during the menstrual period could, in theory, increase the possibility of decompression sickness (DCS). A study[5] examining the role of menstrual history in the development of altitude DCS in United States Air Force (USAF) personnel undertaking hypobaric exposures demonstrated a clear correlation between the incidence of DCS and time since the start of the last menstrual period. A higher number of subjects developed DCS earlier in the menstrual cycle (0–4 days). This study concluded that women were at higher risk of developing altitude DCS during menses, with the risk decreasing linearly as the time since the last menstrual period increases. The study did not address the likely mechanisms for the findings, nor its application to female divers. Prospective human studies assessing the relationship between stage of the menstrual cycle and DCS are not available. A retrospective analysis of female DCS cases presenting between 1984 and 1996 at the Diving Diseases Research Centre[6] suggested that women may be at greater risk of DCS during the early phase of the menstrual cycle. However, only 26 records out of a possible 73 were suitable for analysis.

Although there are no data to support this, it may be prudent for female divers to add some safety margin to their decompression staging requirements, during the week before, and while menstruating. Susceptible females would be prudent to avoid or modify their diving if they have psychological or physiological problems during this time, such as

anxiety, tension, depression, malaise and muscle cramps, nausea and vomiting, or a propensity to sea-sickness.

ORAL CONTRACEPTION

Oral contraceptive pills are known to be associated with an increased risk of thromboembolism as they accelerate blood clotting, increase platelet aggregation and are associated with an increased blood concentration of some clotting factors.[7] There is however, no evidence to suggest that oral contraceptives alter a female's susceptibility to DCS and the risk of falling pregnant is far greater.

DECOMPRESSION SICKNESS

There has been a common perception that women have an increased susceptibility to DCS, perhaps due to their increased body fat percentage. However, reputable studies to support this perception are conflicting. Bassett[8] looked at the incidence of altitude DCS in the USAF from 1966 to 1977. Of these 104 cases, 32 (31 per cent) occurred in women. Statistically females were four times more susceptible to altitude DCS than males, had more cutaneous symptoms, had more rapid onset of bends pain and had more recurrences and more lasting effects of DCS compared to males exposed to the same altitude exposures. These women also gave a history of vascular or migraine headaches. The application of these results to hyperbaric exposures is uncertain.

Bangasser[9] conducted a retrospective study of 649 female divers by questionnaire comparing the reported incidence of DCS in female versus male diving instructors. Her results suggested a 3.3-fold greater incidence of DCS in females. However, there were several major weaknesses associated with this study – it was retrospective, no controlled criteria existed to determine if DCS actually occurred, and the diagnostic evaluations were based solely on the respondents' replies. It is unlikely that divers incapacitated by accidents or those suffering from fatalities were accounted for.

Zwingelberg *et al.*[10] compared females with males in a review of DCS in deep diving operations. The study was in two parts, the first comparing females to males in a general review of DCS incidence on deep dives, and, a second female–male matched analysis on deep diving involving females. The authors concluded that women divers are at no greater risk of developing DCS under similar bounce dive exposures, but cautioned against the extrapolation of the results to all dive exposures. The dives in these exposures were of short duration and thus may not reflect repetitive, computer based, multi-level, prolonged and/or technical diving.

A retrospective survey[11] looking at men and women in diving showed males had a higher rate of DCS per 1000 dives than females. Although males and females dived the same depths, males dived more aggressive profiles. These authors concluded that females are not more susceptible to DCS than males, and the effects of gender differences are completely masked by the differences in diving profiles.

DCS has been associated with altered clotting activity, specifically increased platelet aggregation. Markham *et al.*[12] studied the behaviour of washed platelets during different phases of the menstrual cycle and the differences in aggregation of male and female washed platelets in response to decompression stress and arachidonic acid. Their study did not support the possibility that platelets aggregate differently during the menstrual cycle; however, it did support the existence of a sex difference in platelet aggregation that is not altered by external factors, e.g. decompression stress. Compared to men, females do have increased sensitivity to platelet aggregation, but the importance of this in relation to the development of DCS in females is uncertain.

No evidence exists in the literature of a difference in susceptibility to pulmonary barotrauma/cerebral arterial gas embolism between the sexes, and the incidence of patent foramen ovale is equal.

PREGNANCY

Diving during pregnancy is a controversial subject, and questions of whether diving increases the risk of foetal abnormality or what is the incidence of foetal

DCS, remain unanswered. Medical ethics prevents us from performing prospective studies; hence, much of the available data is derived from animal studies and by extrapolation of our knowledge of other situations that may influence the health of the mother and foetus.

Although only 35 per cent of the current diving population are women, they are mostly of childbearing age, and many are such enthusiasts that a nine-month interruption is not appreciated. For those whose career involves diving, the nine months' interruption is sometimes very disruptive.

There may be a conflict between personal liberty and a safe conservative attitude. It is likely that those who insist upon the former, will continue to dive no matter what advice is given. It is also likely that the latter will not dive, because it could never be proven to be 100 per cent safe.

Potential problems of the pregnant diver

These are outlined as defined by Lanphier (1987).[13]

Maternal factors

These include:

- Morning sickness and motion sickness.
- Reduced respiratory function.
- Circulatory competition with placenta.
- Altered sympathetic response.
- Reduced fitness and endurance; unusual fatigue.
- Size: fit of suit, harness, etc.; clumsiness leading to injury.
- Effects of lifting heavy weights.
- Increased fat and fluid – ? increased susceptibility to DCS.
- Mucous membrane swelling – difficulty in equalizing middle-ear and sinus spaces.

Foetal factors

General foetal factors include:

- Hypoxia from various mishaps.
- Hyperoxia – closure of ductus? haemoglobin breakdown? consumptive coagulopathy?
- Exercise hyperglycaemia; post-exercise hypoglycaemia.
- Exercise hyperthermia.
- Physical injury.
- Leaking membrane – infection.
- Marine animal envenomation – direct or indirect damage.
- Decompression – bubbles – altered placental flow.

Early foetal factors include:

- Malformation related to maternal DCS.
- Teratogenic effects of pressure ?oxygen ?nitrogen, ?dive-related medications ?bubble formation ?other.
- Recompression treatment – exceptional exposure to O_2 and N_2.
- Decompression – bubbles – birth defects.

Late foetal factors include:

- Prematurity (Ama diving).
- Decompression – stillbirth.

Perhaps the greatest concern of any mother is that some behaviour/activity in which she indulges may harm the foetus. Many women may dive unaware that they may be pregnant (i.e. before their first missed period), and it is this early stage when vital organogenesis is occurring.

Maternal factors

During the first trimester, and especially between the sixth and twelfth weeks, there is a variable but definite increased incidence of nausea, vomiting, gastric reflux and a propensity to seasickness. These contribute to diving accidents and deaths (see Chapter 46).

From the fourth month onwards there tends to be fluid retention and mucosal swelling, thereby making the middle-ear and sinus equalization process more difficult, and predisposing to barotrauma.

During pregnancy there is a progressive interference of respiratory function. The tidal volume increases at the expense of the expiratory reserve volume, and there is also a progressive difficulty with the oxygenation of the blood flow through the lungs, and an increase in the resistance to airway flow. The latter may increase up to 50 per cent, and is probably due to the effect of progesterone aggravating bronchoconstriction. The results of this respiratory

impairment may be to reduce the woman's ability to cope with strenuous activity, and perhaps to increase the likelihood of pulmonary barotrauma.

Uterine blood flow may be compromised during periods of increased exercise and increased sympathetic activity, both of which occur during diving. Erratic blood shifts in flow dynamics are more likely to compromise uterine blood flow than a gradual increase or sustained aerobic activity.

The possibility of DCS during pregnancy may be increased by the increased blood flow, fluid retention, total body fat and blood clotting mechanisms.

The change in shape of a pregnant woman may have unfortunate implications. The wetsuit must be altered, to ensure that there is not an increased abdominal tension, which will push the diaphragm even further up into the thoracic cavity, and aggravate the respiratory difficulties. The weight gain and the change in posture results in the woman being more unbalanced. Weight belts are difficult to position, and exits from the water are more difficult.

During the last three months there is a number of pregnant females who 'leak' through the membranes of the foetus into the vagina, without being aware of it. They presume that this slight discharge is normal, but unfortunately should seawater gain entry to the womb, it would carry the danger of infection and/or premature labour. This is verifiable by testing with litmus, as the normal vaginal secretion is slightly acid whereas the foetal amniotic fluid is slightly alkaline. The possibility of uterine infections after the birth of the child must also be considered.

Effects on the foetus

The effects of bubble formation on the foetus have not been completely elucidated, with conflicting results in animal experiments. Current opinion[14] is that bubbles are less likely to form in the foetus than in the mother, but foetal bubbles have been found in the absence of maternal DCS. That is, the absence of DCS symptoms in the mother does not exclude formation of bubbles in the foetus. Bubbles in the foetus are likely to be more ominous than in the mother due to differences in foetal anatomy and physiology.

The lungs in an adult act as an effective bubble filter, whilst in the foetus most of the blood bypasses the lungs by passing through the ductus arteriosus and patent foramen ovale. Therefore, any bubbles in the foetus may pass directly to the cerebral circulation as a cerebral arterial gas embolism. Animal studies have shown an increased rate of foetal loss when the mother is exposed to a decompression insult. The impression is; the closer to term the greater the risk.

Investigation of the Ama, the free-diving females of Korea, suggested that both hypoxia and bubble production under certain conditions, were potential problems for the foetus. The breath-holding Ama divers who dive up until a few days before childbirth, have a 44.6 per cent incidence of prematurity with an infant of less than 2.5 kg (compared to 15.8 per cent in the non-diving females from the same district).[15] The rate of stillbirth however, was lower among the Ama.

Bangasser[16], in 1977, followed up a group of women who were pregnant when they were diving. Approximately 72 women were questioned, and it was found that more than one-third stopped during the first trimester (when they found out they were pregnant), more than one-third stopped in the second trimester mainly due to the increased size, but 20 per cent continued diving. Most were very seasoned and competent divers. The deepest dive was 55 metres, and five decompression dives were performed. All babies were normal, but there were some complications, including one premature birth, one septic abortion, two miscarriages and two caesarean sections.

Bolton[17] conducted a retrospective survey of 208 female divers of whom 136 dived in pregnancy. The average depth of the dive was 13 metres, and 24 females dived to 30 metres during the first trimester. The frequency of birth defects was significantly greater in pregnancies during which females dived (5.5 per cent compared to 0 per cent), but this was still within the range of the normal population. The data were also not analysed with reference to maternal age – a known factor contributing to birth defects.

Turner and Unsworth[18] reported the case of a mother who dived 20 times in 15 days, during the end of the first trimester. Most dives were 18 metres or less, but three were to 30 metres, and one was to 33 metres. There were no diving accidents, although there was one episode of rapid ascent. The only medication used was pseudoephedrine on two or three occasions. The foetus had unusual malforma-

tions, and the embryopathic timetable would suggest that the damage was done around day 40–45. The woman had dived between days 40–55. The abnormalities in the foetus were a unilateral ptosis (drooping eyelid), a small tongue, micrognathia (a small lower jaw) and a short neck. The penis was adherent to the scrotum, and the fingers were in fixed flexion with webbing between them, and the thumb was abnormal. The hip joints were dysplastic and had a reduced range of movement; one hip was dislocated. There were flexion deformities of the knees and other abnormalities of the feet. Arthrogyposis was present and presumed to result from either muscle disease or abnormalities of the cells forming the anterior root ganglion, so the same embryopathic timetable may be applicable.

A Scandinavian study by Bakkevig *et al.*[19] was made on 100 pregnancies in divers, 34 in which diving was continued and 66 in which it was not. The diving exposures were associated with five birth defects, and the non-diving with one. The incidence of infant anomalies was thus 15 per cent in the diving group and 1.5 per cent in the non-diving divers. None of the divers had DCS, and the incidence of other pregnancy-related problems were the same in each group.

A potential problem for the foetus is **oxygen toxicity**. Diving on compressed air exposes the foetus to an increased partial pressure of oxygen, and the mainstay of treatment regimes for diving accidents is recompression on 100 per cent oxygen. In the foetus, ductus arteriosus blood flow decreases dramatically when the oxygen tension in the pulmonary circulation increases. The foetal pulmonary bed is hypersensitive to oxygen tension and responds with vasodilatation when the oxygen tension rises. There is consequently a shift from a foetal to a neonatal blood flow pattern.[1] This shift will reverse when the oxygen tension falls, but it is unknown as to whether this has long-term sequelae for the foetus. Hyperbaric oxygen therapy (HBO) in pregnancy has been used liberally by the Russians, although the outcomes of such therapy are not well reported. In the western world HBO has been used in the treatment of carbon monoxide poisoning in pregnant women, without reported complication.

There are no reports in the diving literature of air embolism affecting a pregnant diver. Taylor[2] reported 15 cases of air embolism from sexual encounters, all of which occurred in young women in their second or third trimester where air was forcibly blown into the vagina. In 12 of the 15 cases there was maternal and foetal death. One patient was treated with HBO for 39 hours with resultant moderate neurological defects in the mother and a stillborn infant. It was concluded that air embolism of the uteroplacental bed appears lethal.

Marine envenomation carries undefined foetal toxic effects, and specific antitoxins may also hold risks for the foetus.

Post partum

There are no contraindications to women diving while breast feeding; however, it is generally recommended that women do not dive until six weeks post partum to avoid intrauterine infection.

Recommendations

In 1978 the Undersea Medical Society held a workshop on this subject and it was recommended that, until further studies were made, women who are or may be pregnant should be discouraged from diving. The conclusion, promulgated by Lanphier in 1983 and supported in the Societies' symposium on Woman In Diving in 1986, was as follows:

- Diving can increase the incidence of birth defects.
- Foetal resistance to bubble formation (DCS) is offset by the dire consequences of this.
- Maternal DCS late in pregnancy entails a higher risk of stillbirth; the risk may be increased by recompression.

There are insufficient hard data available to say, unequivocally, that diving will produce danger to the foetus, but unfortunately there is also enough evidence to suggest that this could well be so. A nine-month respite from diving seems a small price to pay for a healthy child. Alternately, a birth defect, with the possibility that it was caused by diving, would be a heavy burden, outweighing any benefit that diving in pregnancy could possibly confer.

These authors respect and commend Lanphier's conclusions, therefore.

MAMMARY IMPLANTS

Vann *et al.*[20] exposed mammary implants to various simulated dive profiles, followed by altitude exposures to simulate aircraft travel. The implants were observed for bubble formation and volume changes. Minimal volume changes occurred after each dive, although numerous bubbles formed which reached their maximal size in 3 hours. When the implants were exposed to high altitude following a dive, significant volume changes occurred. The volume changes were least for saline and greatest for gel saline implants. The authors concluded that bubble formation in breast implants might occur after shallow saturation diving, but it is unlikely to result in tissue damage. However, prolonged deep saturation diving followed immediately by flying in an unpressurized aircraft at 30 000 feet should be avoided as the resultant bubble formation may be of sufficient magnitude for tissue trauma to occur.

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Breath-hold diving

CARL EDMONDS

INTRODUCTION

Breath-hold diving is also called 'snorkel diving' and 'free diving'.

Traditional breath-hold divers include: the female shell divers of Japan (Ama) and Korea (Hae-Nyo); the sea-men (Katsugi) of Japan; sponge divers of Greece; pearl divers of the Tuamotu archipelago and Bahrain, and the underwater warriors of the Persian, Xerxes, in the fifth century BC. More recently, the abalone and paua divers of the USA and New Zealand, spear fishermen worldwide and submarine escape tank operators of USA, Europe and Australia, illustrate the diversity and dangers of this type of diving.

The numbers of professional breath-hold divers of Korea and Japan have remained steady at about 20 000. The abalone and paua divers have remained fairly constant, probably only a few hundred, because of the limited supply of this natural resource. The pearl divers of the Tuamotu archipelago, the Middle East and the Torres Strait, as well as the sponge divers of Greece, no longer have a viable industry. Compressed-air diving, including scuba and hooker, have dominated the occupational activities associated in the past with breath-hold diving.

The recreational snorkellers of Australia are now a major part of the tourist industry of the Great Barrier Reef. Similar explosions of this population are seen in the Caribbean, Indo-Pacific Islands and the Mediterranean. Indeed, recreational snorkelling has become one of the most widely embraced sports of the latter part of the twentieth century and thus the risks associated with this type of diving, are becoming better appreciated.

A small group of adventurers have extended the depths, as well as the techniques and parameters of deep breath-hold diving. Sometimes the descents and/or ascents are assisted by weights, floats, etc., and sometimes the breathing gases or techniques are modified. However, with this complexity comes added risks which are beyond the scope of this text.

FATALITY STATISTICS

When a recent series of 60 snorkelling deaths was compared to a previous series of 132, it was seen that there has been a change in the demography of the sport in that divers of the later series were significantly older, and a higher proportion were females. The three major causes of death were drowning (45

per cent), cardiac (30 per cent) and hypoxia from hyperventilation and/or ascent producing drowning (20 per cent). Other causes included deaths from marine animals and trauma. The characteristics of fatality groupings are different.

Tourists were over-represented in both the drowning and the cardiac groups, with inexperience, medical and physical unfitness, equipment and environmental factors contributing to the deaths occurring in these two groups.

The **drowning cases** usually occurred in situations in which supervision was inadequate and therefore rescue and resuscitation were delayed. In contrast, the **cardiac cases** often died very quietly in calm, still water, the deaths frequently being predictable from the divers' cardiovascular history.

The deaths in younger, fitter, experienced divers were more related to the production of hypoxia after hyperventilation and breath-holding, often occurring during ascent and associated with spear fishing or underwater endurance attempts.

MARINE ENVIRONMENTS

Like all other divers, breath-hold divers are susceptible to the hazards of the marine environment. These include injuries from marine animals, infections and envenomations. They include exposure to water temperatures less than thermo-neutral (35°C) as well as the various drowning syndromes, including saltwater aspiration and near-drowning. Motion sickness is a common problem, as is trauma (ocean currents, rocks, boats, etc.) and entrapment.

EQUIPMENT PROBLEMS

These hazards include a variety of problems due to the equipment being worn by the free diver, i.e. mask, snorkel, fins, etc., together with the problems from catch bags, spear guns, floats, boats, etc. They are no different in principle to those encountered by scuba divers, but the disadvantage for the free diver is that a supply of air is not available to him. Results of this increased danger include entrapment and entanglement in lines (floats, spears, etc.). Some of the modern monofilament fishing lines cannot be broken manually or even cut by a knife (scissors are effective). A scuba diver has much more time to cope with such difficulties.

The commonest equipment problems include flooding of the facemask and restriction to snorkel breathing with exertion. The reduction in maximum voluntary ventilation and increase in the work of breathing produces dyspnoea when the respiratory demands are great.

DYSBARIC DISEASES

Barotrauma

Barotrauma of descent is more common in free divers than scuba divers, because of the rushed nature of the activity. Free divers have so little time that they have to descend more rapidly, often without much attention to the various symptoms that may be caused by barotrauma. They also experience more ascents and descents, producing more barotrauma.

Following barotrauma of descent, there is often an associated **barotrauma of ascent**. This is especially seen with otological, sinus, dental and gastrointestinal barotraumas. Ascent cannot be delayed, or even slowed, and so the manifestations cannot be diminished – as with scuba. (see Chapters 7, 8 and 9).

Pulmonary barotrauma of descent (lung squeeze) is almost entirely confined to breath-hold divers (Chapter 6).

Decompression sickness

Decompression sickness (DCS) has also been postulated as a sequel of intensive free diving. Cross described an illness called Taravana (*tara* = to fall, *vana* = crazily), in the pearl divers of the Tuamotu archipelago. The dives were to 30–40 metres, lasting 1.5–2.5 minutes each, over a 7-hour period. The illness, which was characterized by vertigo, nausea, paresis, unconsciousness and death, could be due to DCS in some of the cases. Perusal of the report would indicate that many of the cases could have been due to a variety of other disorders, e.g.

inner-ear barotrauma, saltwater aspiration, near-drowning causing hypoxic encephalopathy and drowning.

The reason that DCS can develop with breath-hold diving is that the nitrogen pressure in the lungs increases with depth, and with the greater depths there is a greater nitrogen partial pressure, with nitrogen diffusing from the lungs into the bloodstream and thence to the tissues. If the surface interval is inadequate to eliminate this nitrogen, then it will accumulate with repeated dives throughout the day.

Paulev, a Danish submarine escape tank diver in the Norwegian Navy, performed 60 breath-hold dives to 20 metres in 5 hours, each lasting about 2.5 minutes with surface intervals of less than 2 minutes. He developed symptoms consistent with DCS. Other submarine escape instructors have suffered similar problems, in both Norway and Australia. Unfortunately, many of these cases were also exposed to compressed-air breathing – either in chambers or air pockets in the escape tanks.

HYPOXIC BLACKOUT

This problem, which encompasses the contributions of hyperventilation then breath-hold, exertion and the hypoxia of ascent, is fully described in Chapter 16. Despite these classical causes of hypoxia, probably the most common cause is the aspiration of saltwater, resulting in near-drowning and drowning states (see Chapters 21–24).

CARDIAC DISORDERS

Human breath-hold divers produce a dramatic bradycardia from the diving reflex. It reaches its maximum effect in 20–30 seconds, usually to two-thirds of the pre-dive level, but sometimes to less than 10 beats per minute in experienced divers. It bears a linear relationship to the water temperature below 15°C, and non-linear above that. The bradycardia might well permit other arrhythmias to develop. The arterial blood pressure seems to increase with the diving reflex in humans, and the diving response is augmented by fear.

In humans, unlike most of the diving mammals, free diving is associated with significant cardiac arrhythmias. These can occur even with common respiratory manoeuvres such as deep inspiration, prolonged inspiration, breath-holding, release of breath-holding and Valsalva.

In a study of Korean women divers the incidence of cardiac arrhythmias was 43 per cent in the summer (water temperature 27°C) as compared with 72 per cent in the winter (water temperature 10°C).

There is a high frequency of arrhythmias in association with immersion breath-holding, even without diving.

The head-out immersion position increases the workload on the heart, because of the negative pressure effect (the intrapulmonary pressure remains at 1 ATA, whilst a negative pressure, needed to inhale, is approximately -20 cm H_2O). There is a reduction in the functional residual capacity of the lungs, an increased work of breathing and an increase in the intrathoracic blood volume, with a corresponding dilatation of the heart, and especially of the right atrium. The immersion diuresis and associated loss of sodium may perpetuate cardiac problems.

With very deep breath-hold divers, when the peripheral circulation replaces some of the residual volume of the lungs – due to the effect of Boyle's law contracting the total lung volume to less than the residual – over 1 litre of extra blood can fill the pulmonary circuit and the heart. The distension of the right atrium may be a major cause of arrhythmias taking over from sinus rhythm.

The relatively high incidence of cardiac deaths during snorkelling (and scuba diving) activities may be partly related to the above findings, and partly due to the excessive workload experienced by novice snorkellers, in attempting to overcome the stressful influences of panic, adverse tidal currents and negative buoyancy.

PULMONARY DISORDERS

The most common lung disease is the aspiration of seawater, producing either the drowning syndromes (see Chapters 21–24) or provoking asthma in those so inclined. The changes in lung volumes with the

head-out position have been described above, with the pooling of blood in the thorax, reducing respiratory capability.

Pulmonary oedema has been described in association with immersion, as have other causes of dyspnoea, including coronary artery disease, cardiac arrhythmia and cold-induced hypertension (see Chapter 42).

GASTROINTESTINAL PROBLEMS

The pressure gradients associated with the head-out immersion position, commonly experienced in free divers between dives, causes an increased gastroesophageal pressure gradient, from 6 mmHg in air to 16 mmHg during immersion. This predisposes to gastric reflux in those who are susceptible to it, and who have an inadequate oesophageal sphincter. This also increases the tendency to **vomiting**, which itself can be aggravated by other factors such as alcohol intake, seasickness, otological barotrauma and gastrointestinal barotrauma.

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Technical diving

JOHN PENNEFATHER

INTRODUCTION

There is little agreement on most aspects of technical diving, and even its definition attracts controversy. In its widest usage it is diving outside the normal range of air diving. For this chapter it is considered to include diving with any gas mixture other than air, with decompression in the water. Some would also note that it is largely an amateur-organized approach to diving. This is only partly correct as most of the methods come from, and are used by, professional organizations. Others would not include oxygen/nitrogen mixture (nitrox) diving in the definition.

A constant feature is that technical diving involves manipulation of the gas mixture for a reduction in decompression requirements. This can be with a single gas mix, or a series of mixtures. Because the dive is more complex than a no-stops air dive there may be an increase in risk in technical diving. Any diver involved in it needs to be aware of these risks, and should evolve methods of minimizing or managing them.

The only possible exception to this is that, as far as possible, the risk should not be imposed on others. This creates an area of conflict in Australian law if a paid employee is expected to participate. In some ways the paid technical diver is like a flying instructor or mountain guide. His employment involves risks, and possibly the law should accept this.

HISTORY

Hamilton[1] has provided an outline of points he considers important in the development of technical diving. It could be argued that technical diving started with the development of the first rebreathing set in 1879. Other possible starting points are the development of oxygen decompression or the use of mixed gas diving sets. An appropriate beginning is 1970 when NOAA, a civilian component of the US Government, launched the use of oxygen/nitrogen mixtures with more oxygen than the 20–21 per cent in air. This has been termed nitrox or enriched-air diving, and NOAA did this to increase the productivity of their divers. By 1986 Rutkowski, who was associated with NOAA, had launched an association to promote and regulate the amateur use of nitrox.

The initial response from 'the establishment' was largely to condemn and ban the use of amateur nitrox diving because of the perceived risks. In less than 10 years this situation had been reversed, and nitrox diving was accepted and taught by most of the diving instruction organizations.

This reversal had several causes. Most important, the customers recognized that nitrox diving had benefits for them. The opposition of the establishment was shown to be largely unjustified. Also, some diving opinion makers realized that the nitrox diving enthusiasts needed guidance, not condemnation, and provided it. Reports appeared with findings that supported the use of nitrox diving. Ornhagen and Hamilton[2] reported a rigorous set of Swedish trials, and the favourable results of a US Navy study, including an input from divers who had been trained in the technique and asked to evaluate it for US Navy use.[3]

The use of mixed gas diving with changes in mixtures can be traced back at least to the dives of Keller in the 1960s, these included a 307-metre (1000-feet) dive. Zetterstrom could also be considered a technical diver with his pioneering hydrogen mixture dives. Since about 1980 dives with closed- and open-circuit systems have pushed the depth and time boundaries, though not always safely. In 1994, Exley became one of several leading proponents to die during a dive.

One of the more impressive set of technical dives was the expedition to the *Lusitania*, where 120 dives were made for a total bottom time of about 40 hours.[4] These dives were to depths of up to 93 metres in the highly tidal North Atlantic. A couple of years later divers reached the wreck of the *Britannic* in 119 metres of water.[5] Both these series of dives were conducted without injury – feats that could be matched by few navies! Deep diving companies could dive these wrecks safely with saturation methods, but they would need a large ship and support staff rather than a small launch.

It is noted that there was criticism of the *Lusitania* dives from within the technical diving fraternity. The main critic came to technical diving from commercial diving, and seemed to not accept that the expedition was self-funded and could not afford the back-up he would have liked.[6]

Because of the greater risks, the more complex forms of technical diving have attracted condemnation and this has slowed the development of the sport. To some extent it has been the province of the thrill-seeker, but is also being used by workers with a need to reach greater depths. Many of the leading diving experts are now accepting the use of rebreathing sets for technical diving.

Often technical diving victims died doing something that they would consider careless if done by another. Two Australian leaders in the field probably died by choosing the wrong gas mixture from the range they carried.

NITROX (ENRICHED-AIR) TECHNICAL DIVING

Nitrox diving entails the use of an oxygen-rich mixture to reduce the nitrogen content of the gas. A BSAC review summarized their nitrox findings as follows:[7]

Advantages of nitrox	
Extended no-stop times	Valid
Reduced decompression	Valid
Reduced residual nitrogen	Valid
Reduced risk of DCS	Valid
Reduced narcosis	Not valid
Improved cellular integrity	Not valid
Reduced subclinical DCS	Not valid
Reduced gas consumption	Not valid
Disadvantages of nitrox	
Risk of oxygen toxicity	Valid
Gas mixing/handling/testing	Valid
Equipment compatibility	Valid
Difficulties in hyperbaric treatment	Not valid

The effect of increasing the concentration of oxygen on decompression can be considered in an example. An air dive to 30 metres (4 ATA) has the diver breathing 0.8 ATA of oxygen and 3.2 ATA of nitrogen. If he dived to the same depth on 40 per cent oxygen he is breathing 1.6 ATA of oxygen and 2.4 ATA of nitrogen. An air diver gets 2.4 ATA of nitrogen in a dive to 3 ATA, or 20 metres (2.4 ATA of nitrogen and 0.6 ATA of oxygen). It is generally, but not universally, accepted that nitrogen is the gas that dictates the need for decompression, so the diver using 40 per cent oxygen could use an air schedule for 20 metres instead of 30 metres. In this case the equivalent air depth (EAD – the depth where the diver gets the same nitrogen pressure as he would for an air dive) is said to be 20 metres.

This difference may not seem much, but consider the nitrox or air choice for a diver using the DCIEM

schedules to do two dives to 24 metres in a day and not wishing to do decompression stops. Air or 36 per cent oxygen mixture are available. If the dives are separated by 4 hours, the total bottom time for the two dives increases from 47 minutes to 81 minutes by using nitrox – a significant gain if one is paying for a holiday or the diver is being paid to do a task.

To conduct a nitrox dive the diver with air diving equipment needs a cylinder filled with the appropriate mix and a demand valve system cleaned to the appropriate standard. For training, he needs a short session to learn how to use modified decompression tables. The training should also stress the dangers of exceeding the safe depth for the mixtures.

The gas supplier needs a modified air compressor instillation, a supply of oxygen and an analyser to confirm the contents of the filled cylinders. The supplier also needs to be aware of an increased fire risk with gas mixing, though this has not been found to be a major problem if appropriate precautions are taken.

OPEN-CIRCUIT TRIMIX DIVING

Open-circuit trimix diving is the method used by most technical divers to go deeper than the depth allowed by air or nitrox. Again, it involves using formulated gas mixtures to the diver's advantage. Trimix consists of a mix of oxygen, helium and nitrogen. Various methods have been used to describe mixtures, but one short-hand is to refer to trimix by two figures, the percentage of oxygen and helium in that order, with the understanding that the balance is nitrogen. Thus, trimix 10/40 will be 10 per cent oxygen, 40 per cent helium and 50 per cent nitrogen.

The three components are controlled to give a compromise between conflicting factors. Increased oxygen reduces the amount of inert gas absorbed, as its use is limited by oxygen toxicity. Extra helium reduces nitrogen narcosis but increases heat loss and cost of the mix. Extra nitrogen reduces cost and heat loss but causes narcosis, and increases the inert gas load.

For the dives on the *Britannic* mentioned above,[5] the divers used a bottom mix of 9/57 and breathed this on ascent to 60 metres. There they changed to 17/19, then to 40 per cent oxygen nitrox at 30 metres. At 15 metres they took off the 17/19 cylinder and took a cylinder of 70 per cent nitrox from the decompression staging line. At 3 metres they breathed oxygen from another supply to complete their decompression with a break of 5 minutes breathing nitrox after every 25 minutes, to reduce the risk of oxygen toxicity. The bottom mix was carried in back-mounted cylinders, and the other mixes in side-mounted cylinders.

This bottom mix gave them a maximum oxygen pressure of 1.16 ATA which is conservative compared to that used by many commercial and military diving groups. The use of progressively richer oxygen mixtures during ascent gives an increased diffusion gradient that favours the loss of inert gas.

In another series, dives of various mixtures including trimix dives were reported. It used observations from over 2000 dives to 70–100 metres of seawater (msw). Decompression sickness (DCS) was seen with other gases, but none in 860 trimix dives.[8]

This type of dive provides more opportunity for things to go wrong than does a single gas mixture. Choosing the wrong gas mixture can be fatal, as can failing to find the stage with the gases needed to complete the decompression. These risks can be reduced to what the exponents regard as an acceptable level by training and preferably a well-developed buddy system, whereby the divers can check each gas change for each other before making it.

As an example of preparation and training, some of the divers on the *Lusitania* had nearly 50 training dives to develop techniques and choose equipment before going to the ship. Because of the large gas volumes used, these divers have to compensate for greater than normal buoyancy changes. A dry suit can be used as an additional buoyancy control device.

The remaining important problem is the selection of **decompression tables**. The more successful tables are based on computer programs that have been evaluated against available military and commercial diving results. The mathematical processes are then used to prepare tables for the proposed depth and mixtures. They can suggest gas mixtures that meet chosen safety limits, such as maximum oxygen and nitrogen pressure and how many

mixtures are to be used. This allows the diver to conduct a series of simulated dives before choosing a schedule. A safety margin can also be selected and applied to reduce risk of DCS.

REBREATHING SETS AND TECHNICAL DIVING

Technical divers are also using semi-closed and closed-circuit mixed gas sets, as described in Chapter 4. They are being used for shallow dives, where the long endurance for the size is being welcomed. For deep dives the saving in gas costs is important; the other gain is the reduction in the volume of gas that must be carried.

The functioning of these sets will not be described again here. The problems of oxygen and carbon dioxide toxicity, hypoxia and caustic cocktail are inherent and occur in a technical dive, and have been outlined in earlier chapters. The inherent risks of this diving cannot be avoided, but the more responsible suppliers insist on a thorough (and costly) training course to minimize them.

A RESPONSE TO COMMON CRITICISMS OF TECHNICAL DIVING

Some of the more conservative members of the diving medical profession continue to produce criticisms of technical diving that are largely unfounded. Others have produced a more thoughtful response, suggesting that nitrox diving requires extra training and technical diving that pushes the limits is not for the majority of recreational divers.[9] This summary of the issues raised is not a defence of the more extreme forms of mixed gas technical diving.

As an example of the medical criticism, it has been claimed that oxygen contributes to the formation of gas bubbles and so should be included with the inert gases in calculating decompression. The evidence for this claim is highly suspect. It includes Donald,[10] who created DCS associated with adding oxygen to air in a series of goat dives in a chamber. However, in the next paragraphs he states that it was a consequence of the very high oxygen pressure used, and he knows of no case to suggest that the EAD formula had not worked in the accepted range of oxygen pressures.

Thalmann[11] has been quoted as saying that oxygen should be included in calculating EAD. My reading of the paper is that he includes tissue oxygen as a parameter in calculating tissue gas tensions. Thalmann was developing tables for equipment that keeps oxygen pressure constant, so inspired oxygen pressure is unlikely to be a variable studied. He does not include any mention of inspired oxygen, neither does he question the validity of the EAD assumption in the discussion.

Possibly the best study of the problem is by Weathersby *et al.*,[12] where 477 controlled dives were reported. In the first paragraph of the discussion the authors state, 'The results support the traditional view of O_2 having no effect (on decompression), but conclusions are limited by the variability of human DCS'. In their enthusiasm to support their cause, the opponents of nitrox diving seize on a later comment, that there is a small statistical chance that oxygen could be up to 40 per cent as potent as nitrogen, conveniently neglecting the comment that oxygen may be protective against DCS.

There are other works that could be cited as proof that oxygen is involved in decompression.[13,14] These workers compressed rats to high pressures on a variety of gas mixtures. In some cases the oxygen pressure was well outside the physiological range. In all the trials the rate of pressure reduction was extreme, up to 1 ATA/second. This will facilitate the formation of bubbles in the arterial supply because the blood will be over-saturated when it reaches the capillaries, so the findings could be predicted and are not relevant to discussion of normal diving.

If there was a significant flaw in the EAD assumptions then it should have been detected in the thousands of nitrox dives carried out to date.

There is debate on what should be the maximum accepted oxygen pressure. At the tail of the risk curve there will be occasional incidents unless a low value is assumed, and there may be a case for using full face-masks to minimize the consequences. It is noted that the *Britannic* divers used 1.16 ATA of oxygen at maximum depth, and never more than 1.6 ATA during decompression. The US Navy uses up to 2.5 ATA during decompression from deep dives, so the

technical divers may be able to teach the professionals about safety in relation to oxygen pressure!

In the early years of nitrox diving some thought it should give a reduction in narcosis. This was because there was a reduction in the nitrogen pressure. However, it is debatable if this occurs, because oxygen seems to have narcotic properties in some trials. Given that most nitrox diving is conducted shallower than 40 metres, narcosis is not a major problem. For deeper dives the technical diver goes to trimix. It is possible that the problem was caused by the amateurs believing the professionals – when they called a disease 'nitrogen narcosis', the amateurs assumed that it was caused by nitrogen.

Of course there are people making silly mistakes in technical diving. For example, in one dive a participant convulsed while breathing oxygen at a partial pressure of 1.26 ATA, but he later admitted a history of seizures and that he was on anti-convulsant medication. It is considered that this is evidence of the need for more stringent medical examination – not that 1.26 ATA is dangerous.

There has been criticism of the use of rebreathing sets by amateurs. In part these may be justified, but the risks are inherent in the set – they are not a result of amateur status. In military circles, the number of deaths that occur while using rebreathers is probably in the region of one per several thousand diver-years, so most would accept that this is not a high-risk activity. The amateur has the advantage that he does not have to dive if he does not feel like it, and is not rushed by his supervisors. These are significant causes of service diving accidents. It is a matter for the diver and his training organization to be confident that he is properly trained.[15,16] It is noted that some significant advances in rebreather design have been made by amateur users.

A problem for any diver without access to research data is that manufacturers tend to exaggerate the performance of their sets. Some endurance figures quoted are either the designer's dream or were obtained with a resting diver in warm water. Failure rates may be underestimated or not available. However, there may be a role for the teaching groups to publish product reports with realistic performance figures.

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Technical diving problems

CARL EDMONDS

The more conservative authors of this text would prefer that normal recreational scuba divers would not get involved with this extension of 'the diving envelope'. Nevertheless, technical diving is both a fact and a growth industry. It is another challenge, with extra risks and rewards. This chapter is an overview of a type of diving which is more complicated, expensive and hazardous (see also Chapters 4, 16–18, 46 and 62).

Propaganda on the relative merits of technical diving are usually based on its potential to reduce the risk of decompression sickness DCS. Most of the diving accidents and deaths that occur in recreational scuba diving, are not due to DCS. Indeed, the major causes include the hazards of the ocean environment, the stress responses on the individual, equipment failure or misuse and some diving practices which are potentially dangerous, including exhaustion of the air supply, buoyancy problems and failure to follow buddy diving practices (see Chapter 46).

The risk of serious DCS is small for recreational scuba divers. Thus, when some influential divers purport to reduce this incidence by complex techniques, while at the same time increasing the hazards from the more common diving problems, one must question the motivation.

Technical diving should be restricted to experienced divers who are somewhat obsessional in nature, mechanically skilled and extremely competent, and who have completed specialized training courses in this subject.

DEFINITIONS

Decompression diving
Deep diving (>30–40 metres)
Not compressed air
Gases = *Oxygen*
Nitrox (Oxygen-enriched air),
Heliox,
Trimix (± argon, hydrogen, etc.)
Re-breathing equipment

Technical diving refers to diving in excess of the usual range for recreational scuba divers. This may involve an extension of duration at any depth, the depth itself (in excess of 30–40 metres), changing the gas mixtures to be used, or using different types of

diving equipment. All these fall into the realm of technical diving.

It is important, when discussing technical diving, to specify which type, as the risk varies from little or no additional risk (compared with recreational diving) to an extremely high one, such as with mixed gas rebreathing equipment.

Decompression and deep diving using only compressed air have added risks, and have been discussed in previous chapters.

The risks increase as the gas mixture deviates from normal air and with increased complexity of the equipment.

Diving on 32 per cent oxygen (O_2), 68 per cent nitrogen (N_2) instead of air in a normal scuba cylinder, to a maximum of 40 metres on a no-decompression conventional air profile, could possibly incur slightly less risk than a recreational scuba air dive to the same depth.

THE TECHNICAL DIVER

Very experienced (500+ dives)
'Technical' (toys) orientation
High IQ, Obsessional, Studious
+ risk-taking behaviour
with Reduced safety margin re Death.
++ Money and ++ Time
Commercial interests

The technical diver is – or should be – a very experienced scuba diver, having logged at least 500 dives before entering this new field. Such a diver is usually male, and oriented towards technical toys. He is of a high intelligence but with a large ego, frequently obsessional in his attention to detail, often studious and attracted to risk-taking behaviour with a reduced safety margin as regards death.

He will apply considerable funds and time to his project. Often this has commercial implications, and he may well be involved in wreck salvage, equipment manufacture, marketing and sales, diver training, or related commercial activities.

The diver attempts to select the theoretically ideal gas mixture for the ascent and descent (travel mixes), the bottom (bottom mix) and the decompression staging (usually O_2).

GENERAL PROBLEMS

1 *Equipment complexity and cost*
2 *Dive profile variations (depth/duration)*
3 *Physiological questions*
EAD/O_2/CO_2/Inert gas transfer
4 *Environments extended, with more hazards*
5 *Complex dive techniques*
6 *Accident and rescue implications*

Technical diving involves more complex equipment for producing, supplying and delivering the various gases, other than air. With an increase in the complexity of the equipment there is an associated increase in the likelihood of human and equipment error at all three stages.

The handling of mixtures with higher than normal O_2 percentages implies greater risk of fire and explosion.

When various gas mixtures are being breathed, the decompression profile of the dive may become very complex. Decompression regimes are often unproven, and inadequate factual information is available regarding the physiological interactions of the gases.

There is considerable doubt regarding many of the physiological assumptions on which technical diving is performed. It is claimed that the equivalent air depth (EAD) can be used to determine the influence of the gas mixture on the diver, and this has been related to both nitrogen narcosis and decompression sickness (DCS). There is, in fact, no convincing evidence that this EAD is appropriate to either.

There are also the physiological implications of breathing O_2 at varying partial pressures, as well as the often increased carbon dioxide (CO_2) retention

with both high O_2 and deep diving. The use of gas mixtures is also likely to influence the transfer of inert gases in many ways – far more complex than can be deduced from a simplistic formula. Anyone who doubts this should peruse the seminal texts on such topics as nitrogen narcosis and the counter-diffusion of gases.

The main purpose of technical diving is to extend the environments into which diving is performed. This usually means an increase in the hazards associated with such environments. The exception is a reduction of the nitrogen narcosis of deep diving, by the use of helium (He). Most of the other problems with deep diving are aggravated. Not only can the depth or duration of the dive be extended, but so can the actual diving terrain. This is the reason why many wreck divers and cave divers have embraced this activity.

Problems develop from:

- mixing and transport of gas;
- handling it at the dive site;
- analysing the gas and confirming that it is the one appropriate for the dive to be performed; and
- selection of appropriate gases during the dive.

Multiple gas mixtures require different or additional cylinders together with the various attachments; manifolds, O-rings, contents gauges, high-pressure hoses and regulators. The result is that the mixed-gas diver often wears a large amount of equipment, that is both extremely complex and bewildering – especially when other environmental risks develop during the dive. The likelihood of equipment problems has been compounded. Other related difficulties include buoyancy variations and sometimes the need for a full facemask, so that drowning is less likely and rescue becomes more possible.

Because of the different equipment and gases, and the extension of the environments, the techniques for accident management and rescue have to be altered to take into account the specific problems. With each variation from the conventional scuba system there is a price to pay, and a modification of the first-aid and treatment procedures.

Financially there are increased initial capital outlays, operating and maintenance costs.

INCREASED OXYGEN PRESSURE

1 *O_2 toxicity*
2 *Effect on recompression therapy*
3 *N_2 narcosis and DCS*
4 *Mixing and handling danger*
5 *Hypoxic mixtures*
6 *CO_2 build-up*
7 *Equipment changes*

There is little concern about O_2 toxicity with recreational scuba diving in the no-decompression range. Neurological and respiratory O_2 toxicity are virtually impossible. Also, the amount of O_2 inhaled is unlikely significantly to influence any recompression treatments that may be needed for decompression accidents. Neither statement can be applied to technical diving.

The dangers of **hyperoxia** have not yet been fully defined. Many other organs are affected by it, and small increases in pressure are now incriminated as a contributor to abortion in early pregnancy. Visual changes have been noted in 1.3–1.6 ATA O_2-breathing divers.

It had been assumed that O_2, by virtue of its replacement of N_2, would to some degree reduce the severity of nitrogen narcosis and DCS. Although this is possibly so in theory, the inadequate experimental evidence available would suggest that O_2 actually contributes to nitrogen narcosis and some DCS.

The handling of gas mixtures, where O_2 or other gases are added to air, can produce problems; O_2 also increases the risk of fire and explosion.

Inadequate mixing can result in O_2 pressures being higher or lower than intended, and this has implications regarding the safe dive profile. Higher O_2 levels are also likely to produce a 'build-up' of CO_2 transport in the blood. This has further implications in that it potentially increases O_2 toxicity, nitrogen narcosis and possibly also DCS.

OXYGEN-ENRICHED AIR OR NITROX (EANx)

Most of the technical diving now performed involves the use of O_2 /N_2 mixtures in which the O_2

concentration is greater than that of compressed air. Under these conditions it is very important to specify exactly how much O_2 is being used. Such phrases as 40/60 or 60/40 are not only confusing but often misleading. In Europe 40/60 is more likely to imply 40 per cent O_2, whereas in the USA it is more likely to imply 40 per cent N_2. For safety, the O_2 percentage should be stipulated.

The actual percentages used in technical diving do vary with different countries and establishments, but NOAA in the USA have chosen 36 per cent O_2 and 32 per cent O_2 as their two major mixes. **These should not be referred to as Nitrox 1 or Nitrox 2, as this could also be misleading.**

Any EANx diving has a safe depth range less than air, due to O_2 toxicity.

The O_2 pressures that are considered acceptable vary with different authorities, and in many cases there is a confusion between the neurological O_2 toxicity (which can result in nausea, vomiting, seizures, etc.) and respiratory O_2 toxicity, which tends to occur only with prolonged diver exposure. Also, many of the pressures being quoted in the literature refer to the O_2 pressures observed with rebreathing equipment, when the CO_2 levels have not been measured – complicating considerably the actual cause of symptoms. Most of the work carried out during the second World War and soon after, failed to measure the CO_2 levels, and therefore their conclusions regarding safe O_2 limits, are open to question if applied to open-circuit equipment.

NOAA states that the maximum O_2 pressure acceptable is 1.6 ATA, while the National Undersea Research Centre in North Carolina recommends 1.45 ATA, the Swedish authorities have recommended 1.4 ATA, and Dr Richard Vann of the *Divers Alert Network* has suggested 1.2 ATA. The US Navy allow a greater value, but relates it to the duration of the exposures.

The claimed advantages of EANx diving include a probable reduction in DCS incidence, and a possibility of reduced nitrogen narcosis.

On a theoretical basis, presuming N_2 pressure as the sole cause of nitrogen narcosis, a 20 per cent O_2 mixture (air) at 23 metres could be replaced with a 36 per cent O_2 at a depth of 30 metres, to give an equivalent 'narcotic effect'. Experimental verification for belief in this theory has been sought, but it was unable to be verified (see Linnarsson, *et al.*, 1990; Bennett, 1970).

Although O_2 is used as a treatment to replace N_2, when the latter has caused DCS, it has also been contentiously incriminated as a cause in its own right (see Donald, 1992; Weathersby *et al.*, 1987) or as a contributor (Thalmann, 1986) to this disease.

A common claim is made that there is less post-dive fatigue with EANx than there is with air, but this has not yet been verified.

Low-risk nitrox diving

Nitrox (EANx) replace air. Same equipment (same profile as air dive). Range 15–40 metres

Advantages

1 *Less DCS*
2 *? Less N_2 narcosis*
3 *? Less Post-dive fatigue*

Disadvantages

1 *Gas mixing problems*
2 *Less max depth (O_2 toxicity)*
3 *? Deterioration of dive equipment*
4 *? More CO_2 retention*

It is possible to use EANx to obtain possible advantages, with relatively few disadvantages, under certain conditions. In this type of technical diving, the nitrox mixture, usually 32 per cent or 36 per cent O_2, replaces air, but the same equipment is used and the same air decompression profiles applied, within the 15 to 40-metre range.

It has been claimed there is deterioration in the dive equipment by using high O_2 mixtures, but this has not been verified.

It is likely, because of the higher O_2 levels inhaled, that there will be a concomitant degree of CO_2 retention, based on the common and competitive pathways for the transfer of these gases.

Higher-risk nitrox diving

Nitox (EANx) replaces air – larger cylinders (profile as for EAD) – range 15–40 metres

Advantages

1 *Increased duration of no-decompression dive*
   or Less decompression stops
   or Greater duration/depth of dive for same decompression
2 *Decompression value – if 'air' stops are followed (less N_2)*

Disadvantages

1 *Gas mixing, handling and correct usage*
2 *Max depth limited (O_2 toxicity)*
3 *?Increased risk of DCS (O_2 effect, untested algorithm)*
4 *?Alteration of DCS and recompression therapy,*
5 *?Dysbaric osteonecrosis (Slow tissues affected by longer dives)*

In this type of diving (EANx) the profile of the dive is altered to make allowance for the high O_2, lower N_2 levels, based on the EAD or similar calculations. Thus, the diver is likely to increase the duration of his no-decompression dive, reduce the decompression stops required, or increase the duration or depth of the dive for the same decompression time commitment. Whether this calculation is justifiable under all conditions, has yet to be demonstrated.

Probably the only definite advantage of this kind of diving occurs if 'air' stops are followed during decompression, while using EANx.

There is a possibility of an increased risk of DCS, due to the effects of O_2 contributing to this disorder, or because of the use of untested algorithms used in commercial nitrox decompression profiles. The 'bent' diver is also more likely to have had a high O_2 dose, contributing to respiratory damage during the recompression therapy, than his air-breathing colleague.

There may well be an alteration in the type of DCS sustained with this form of diving because of the increase duration that it frequently entails. Thus, the deeper tissues are more likely to be affected, and this should be considered during the subsequent recompression therapies, and also the possible increased susceptibility to dysbaric osteonecrosis. The reason for proposing this is that the dives, being longer, will more likely influence the 'slower tissues'.

High-risk helium diving

He = Less dense, less soluble
Faster diffusion, heat conductivity

Advantages

1 *Less narcosis* *Greater depth*
2 *Less breathing resistance* *Greater depth*
3 *Less decompression (for longer dives)*

Disadvantages

1 *Deeper diving*
2 *More decompression (for short dives)*
3 *Heat loss (environment, ?respiration)*
4 *Voice distortion*
5 *Hypoxic accidents*
6 *High-pressure neurological syndrome*

There are significant differences in the way the body handles helium to the way it handles nitrogen. Both are inert gases, but He is much less dense and is also less soluble in some tissues than N_2. It does, however, have a much greater speed of diffusion and also conducts heat more rapidly.

The real advantage compared to N_2 is that it does decrease the incidence of nitrogen narcosis. For dives in excess of 30–40 metres, the risks of nitrogen narcosis can be proportionately decreased as He replaces N_2. It thus tends to permit dives at greater depths. An additional factor is the reduction in breathing resistance due to its decreased density and other factors, also allowing dives to greater depths.

The effects on decompression likelihood are more complicated. Helium is probably likely to produce less decompression requirement for the longer dives, but may well require more decompression for shorter dives. Specific DCS syndromes may develop, especially those affecting the inner ear. Many of the

He and trimix decompression tables are less well validated than the air tables.

The main problem is that the divers are diving deeper with He and trimix than with compressed air, and therefore are exposed to all the associated problems of depth (other than nitrogen narcosis and breathing resistance). Barotrauma and DCS risks are aggravated. The environmental difficulties associated with depth include poor visibility, buoyancy problems, excess gas consumption, stress factors and the increase risks and difficulties with first aid, rescue and resuscitation.

There is also a greater conductive heat loss from He, even though there is some question regarding the respiratory heat loss. Heliox feels colder to breathe, and in a He environment the heat is lost more rapidly. Increased depth aggravates heat loss.

Voice distortion can produce communication problems. At greater depths the high-pressure neurological syndrome (HPNS) also becomes relevant.

The difficulties with mixing gases, referred to above are also present with He and are complicated by the different compressibility of He, as well as the risk of ascending with low O_2 pressures – which are commonly used with deep He diving.

Comparison with the commercial deep divers is noteworthy. These experts usually require a surface supply of gas, full facemasks, communication systems, a standby diver, a wet bell and a recompression chamber on site. Experience has demonstrated the need for these in the past. The less trained amateurs often have no such requirements.

Very high-risk, rebreathers or circuit sets

Advantages
Silent, Economical, ± Non-magnetic

Disadvantages
1 CO_2 and O_2 toxicity
2 Dilution hypoxia
3 Caustic cocktail

O_2 Rebreathers, Depth limit 8–9 metres
Constant flow pO_2 = Flow versus consumption
O_2 Monitors = FAILURE. DCS?

Rebreathing equipment has been in use for more than a century, serving real needs but causing many deaths and cases of unconsciousness. Despite the recent electronic advances, the essential problems of rebreathing equipment remain. It is very much a high-risk strategy to be employed for specific reasons, by professionals.

The value of rebreathing equipment is that it produces fewer bubbles, and is therefore more silent. This is of use both in clandestine operations and for marine photography. It is more economical on gas, as the gas is recycled through the diving equipment, in a 'circuit'. It can also be constructed with low-magnetic materials, which is useful if one is working near magnetic mines.

The disadvantage that is inherent in all types of rebreathers is the failure of the CO_2 absorbent system to work effectively under all diving conditions. This may occur for many reasons, but includes inappropriate canister design. In fact, there has been little genuine improvement in CO_2-absorbent canisters over the last thirty years – and they were inadequate then!

Also, the absorbent itself is not always reliable. It may vary in efficiency, and each absorbent batch should be tested. This is not feasible for the individual diver, however. The handling and storing of absorbent may also result in deterioration in efficiency, as will the degree and type of wetting that may occur.

When diving in seawater, this can enter the system, causing a great reduction in efficiency. The absorbent itself, when combined with CO_2, produces water as a by-product, which can also influence the efficiency.

The CO_2 absorbent must be packed correctly into the canister – a skilful process that requires training as the density of packing influences the efficiency. Lower temperatures also reduce the efficiency of the absorbent.

Often absorbent canisters will work very well at a moderate work load with smaller tidal volumes, but when exertion is required, these volumes will increase and the absorbent canister will frequently fail – especially if it has been in use for a considerable time.

The manufacturers' claims regarding the safe duration of CO_2 absorption in their diving equipment are

usually exaggerations, and do not apply to emergency situations where the diver is exerting himself maximally (such as when swimming against a current, or trying to rescue and tow a companion – even on the surface).

Any rebreathing set can produce a dilution hypoxia. Even those that use high or 100 per cent O_2 cylinders can occasionally cause this – usually because of an incorrect technique in 'clearing the set' (and the lungs) of the inert gas. It can also occur if there is a small amount of inert gas in the cylinder, and especially so when there is a considerable amount of N_2 or He, such as with nitrox, heliox or trimix diving. It may be induced by an incorrect mix, a leak from or obstruction to the inflow, or loss of cylinder pressure.

Sometimes the hypoxia will only be noticed during ascent. There the lower O_2 percentage at depth is acceptable, but translates to a dangerously low O_2 partial pressure nearer the surface.

When water gets into the rebreathing set, it may collect some of the alkali from the absorbent and then may enter the diver's mouth and lungs. This may cause severe burning, and is referred to as a 'caustic cocktail'.

Rebreathers require specialized diving protocols when rescue and resuscitation are needed. It is not simply a matter of removing one mouthpiece and replacing it with another. Companion diver drill needs to be tailored for each type of rebreather.

The problems of gas mixing and handling, as described above, also relate to this equipment:

- O_2 rebreathers are closed-circuit sets, used to a maximum depth of about 8–9 metres, are usually restricted to naval warfare, and have resulted in many cases of unconsciousness and death. Occasionally, photographers will use this equipment, but they would be considered unwise to do so.
- Some rebreather sets have a constant flow of nitrox, heliox or trimix gas. They are usually semi-closed circuit sets. With these the O_2 level in the breathing bag or inspiratory tube may vary according to two major factors. The first is the flow of O_2 into the set, and the second is the amount lost from the set. The inspiratory O_2 range can be a variable quantity, and make the equipment less safe. The influential factors include:
  - the volume and mixture of the incoming gas;
  - the energy utilized in metabolism; and
  - the gas released as bubbles (with ascents).
- The interaction between the input and output of O_2 will result in a variable O_2 percentage, and ascent or descent will determine the O_2 pressure. These sets are especially likely to cause dilution hypoxia and hypoxia of ascent. As hypoxia usually produces no warning prior to unconsciousness, the use of constant flow rebreathing sets in recreational diving is considered unwise. Close attention to the cylinder pressure, ensuring an adequate inflow of gas, and a replacement with fresh gas prior to ascent (a 'flush-thru') is essential.
- Some closed-circuit rebreathing sets use solenoids to measure the O_2 pressures during the dive, and a feed-back system adds O_2 or a diluent gas (N_2, He, mixtures) as required – to ensure that the O_2 partial pressure remains within a certain range. This equipment is extremely expensive, often not reliable, and should only be used by those with considerable faith in technology.

Because of the much greater risk of unconsciousness and subsequent drowning with rebreathing sets, a full facemask is usually required.

CONCLUSIONS

Perhaps the most important point about technical diving is to realize that the majority of the diving deaths that occur among recreational divers occur for reasons which will be aggravated by the use of more complex equipment, and in more hazardous environments. Technical diving is therefore, by its very nature, likely to have greater risks than normal recreational diving, other factors being constant.

The margin for error in this type of diving is appreciably less, and therefore it should only be employed by divers with extensive experience, detailed training and meticulous attention to the equipment and its use.

The advocates of technical diving tend to lay great stress on aspects of safety, which are relatively less important in recreational scuba. They stress the importance of DCS, and the physiological advantages of O_2, but gloss over the more frequent causes

of diving deaths, such as exhaustion of gas supply, buoyancy problems and stress responses. They also tend to ignore the areas in which the 'technical advances' have been meagre, e.g. the efficiency of CO_2 absorbents, in preference to sophisticated high-tech O_2 sensors and theoretical decompression algorithms.

However, there must be a reason why the Diver Alert Network database on diving deaths has, since the year 2000, allocated a special subsection to technical diving.

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Handicapped divers

ROBYN WALKER

INTRODUCTION

The medical assessment of a fully able or disabled diver is identical. Informed assessment of the disabilty, the effect of the disabilty on the independence of the diver in the water, and the ability of the disabled diver to render assistance to their dive buddy is mandatory. Not all disabled divers will be successful in attaining an open-water certification; however, under certain conditions the disabled diver will still be able to enjoy a supervised 'diving experience'.

DEFINITION

A **handicapped** individual can be defined as an individual with a disadvantage, resulting from impairment or disability that limits fulfillment of a normal role. Impairment describes the loss or abnormality of physiological, psychological or anatomical structure or function, whilst **disability** is defined as the restriction or lack of ability to perform in a normal manner or range, resulting from an impairment.

It has long been recognized that rehabilitation programs should address both the physical and psychological needs of the handicapped individual. The mastery of a recreational sporting activity usually associated with the physically fit goes a long way to improving self-esteem and sense of competence. Can a handicapped individual successfully learn to scuba dive? The answer is yes, of course; but this does not necessarily mean they can become fully certified independent divers. Both the single amputee and the high-level quadriplegic are classified as handicapped, yet the degree of their handicaps and their ability to undertake a scuba diving course differ greatly.

TRAINING OPTIONS

Worldwide there are two different approaches to the question of teaching handicapped divers to scuba dive. The first approach is to offer no special training for the disabled and require them to undertake the standard open-water course like everyone else. Most of the entry-level courses do not teach buddy rescue skills, a problem for many disabled divers, and many handicapped individuals will be able to successfully complete the course. A perceived difficulty with this approach is that the disabled diver may require more intensive instruction and assistance than is available on the standard weekend courses.

The second approach is one of providing an independent training and certifying agency for the

handicapped, e.g. the Handicapped Scuba Association (HSA). The HSA founded in 1981 offers different levels of certification based on degrees of functionality in the water:

- **Level 1**: has successfully met all standards, can take care of himself or herself, help another diver in distress, and perform a rescue. These divers are certified to dive with another certified buddy.
- **Level 2**: has demonstrated ability to handle emergency situations, and in general, care for himself or herself while scuba diving. This diver is unable to help another diver in distress. This diver should dive with two able-bodied certified dive buddies.
- **Level 3**: has demonstrated ability to safely dive, but needs considerable assistance and would not be able to respond to an emergency, such as self-rescue. This diver must dive with two able-bodied, certified buddies, one of whom is trained in scuba rescue.

Most instructor agencies would agree that training a severely disabled person is staff- and time-intensive with at least initially a one-to-one student–instructor ratio and constant attention to safety. The British Sub Aqua Club[1] believes that the integrated approach with main stream divers is more beneficial in the long term.

MEDICAL FITNESS

Disabled divers require the same medical examination as non-disabled divers. In Australia, Australian Standard AS4005.1-1992 details the minimum requirements for the training and certification of recreational divers and describes in detail the medical criteria to be used. The standard requires that 'a full examination of the central nervous system must show normal function, but localized minor abnormalities such as patches of anaesthesia are allowed, provided that generalized nervous system disease can be excluded'; effectively ruling out the paraplegic or victim of childhood polio. Yet, experience has shown that some such individuals can become proficient divers.

Fleming and Melamed[2] reported in 1974 the results of a training course for six severely disabled paraplegics and double-leg amputees. They concluded 'that self-contained diving training is an excellent rehabilitatory activity for disabled people with the following limitations: no paraplegic should dive in the sea with a lesion above T5, no paraplegic whose injury was caused by the bends should dive at all and no disabled diver should undertake decompression dives'. These were based on their practical experiences.

Patient A, paraplegic (T4) after a gunshot wound, was permitted to dive in the pool only. The other five candidates successfully completed an open-water dive. Patient A performed most of the requirements correctly in the pool, but short bursts of intense exercise caused a difficulty in breathing, as his diaphragm was his sole muscle of respiration. This proved critical, whereas the pupil with a T6 lesion was considered safe.

Fleming *et al.* believe that this loss of respiratory reserve due to the loss of the normal muscles of respiration, should require careful assessment of individuals with lesions above T8. They also point out to individuals with partial spinal lesions, from whatever cause, that there is a possibility that diving might make the lesion complete.

Williamson *et al.*[3] report on the medical and psychological aspects in the selection and training of disabled persons for scuba diving. Sixteen individuals with a wide range of disabilities underwent a formal scuba-diving course. Subjects' disabilities ranged from brainstem injury, congenital deafness, congenital blindness, paraplegia to bilateral amputees. Nine of the 16 candidates completed the open-water training program. The medical assessment differed significantly from the norm in that:

- no selection of applicants occurred on the basis of previous athletic achievement, or independence of mobility;
- no preconceptions existed that full, unrestricted diving certification of the 'successful' candidates was a necessary end-point of their training; and
- the group included a range of medical disabilities hitherto regarded at least relative contraindications to diving, such as brainstem damage, myelitis, impaired bladder and bowel control, and blindness.

One of the candidates (poliomyelitis, L2) developed symptoms of spinal decompression sickness (DCS). He had completed eight open-water dives over a period of three days, all within the limits of the United States Navy diving tables. He required five recompression chamber treatments and was left with residual paraesthesia in the left leg and an area of enlarged numbness on the inner aspect of the left thigh. In view of this case, the authors acknowledge the reservations of others with regard to the increased hazards in individuals with partial spinal cord lesions.

Follow-up psychological test results revealed an improvement on the subject's physical-self-concept subscale that was considered the result of their diving experience. The authors also reported an initially higher incidence of ear problems and coral cuts, and found that incomplete bladder and bowel control were not a handicap.

Both studies reflect the need for the medical assessment of disabled divers to be performed by experienced diving physicians with knowledge of diving training and the individual's disability. Particular hazards to consider include the risk of trauma and coral cuts in the paraplegic with loss of sensory input, and also the risk of pressure ulcers from sitting for prolonged periods on the boat. The loss of muscle control may result in the paraplegic's legs floating; this can be counteracted by the use of leg weights. The lower-limb amputee has no use for fins, but in those with use of their arms, webbed gloves may help forward propulsion. The double lower-limb amputee may also experience difficulty in maintaining positional control when swimming. The loss of upper-limb function in some ways is more serious, as this causes problems with buoyancy control and mask clearing. Stroke victims with weak facial muscles may have difficulty holding the regulator within the mouth.

Perhaps the greatest difficulty to be faced by disabled divers is gaining access to dive boats, pools and dive shops. Wheelchair access to these facilities is often limited, and the disabled diver may well need assistance to carry their equipment. Fear of falling or injury while transferring to a dive boat or in transit to the dive site should not be underestimated. Consideration should also be given to the safety of the disabled diver in the event of a boating accident that could result in the boat sinking.

Blind divers will need assistance with navigation. The profoundly deaf will have difficulty in responding to an emergency recall alarm, but are superior at communicating with each other underwater, by hand signals.

In assessing fitness to dive, the medical practitioner should always consider whether the underlying medical condition will predispose the individual to a diving-related illness such as pulmonary barotrauma and DCS. Madorsky and Madorsky[4] argue that as reduced circulation in paralyzed limbs may increase the time it takes for nitrogen to escape from the tissues, it is recommended that dive tables be used extremely conservatively. Williamson *et al.*[3] recommend that a specially modified set of decompression tables be used by disabled divers, based on the assumption that disabled divers are predisposed to the development of DCS because of their pre-existing pathology. It seems reasonable advice to suggest that disabled divers introduce a 'safety' factor when planning their dives.

It is important that disabled divers with neurological conditions have their impairment fully documented, and that their condition is stable before they learn to dive. The individual with progressive multiple sclerosis poses major diagnostic difficulties if they develop neurological symptoms after diving: are the symptoms the result of neurological DCS or a progression of their disease? The below-knee amputee with a discrete well-demarcated area of numbness on their stump secondary to nerve resection is far easier to assess.

Novice divers are more likely to suffer from pulmonary barotrauma and resultant cerebral arterial gas embolism. Non-disabled divers find buoyancy control difficult to master, let alone the disabled diver with reduced upper-limb control. This may increase the risk of pulmonary barotrauma and neurological impairment in an already handicapped individual.

LEGAL IMPLICATIONS

The Americans with Disabilities Act became effective in July 1992 in the USA and is applicable to businesses that employ 15 or more persons. This Act

prohibits employers from discriminating against qualified persons with disabilities and charges them with the responsibility of making reasonable accommodation to allow and assist the worker to do the job. Reasonable accommodation may not be required if it creates undue hardship for the employer or, there is a reasonable medical judgement that there is a high probability of substantial harm to the disabled individual. This reasonable medical judgement must be based on sound scientific evidence and established data.

In the UK, Australia and New Zealand the test of forseeability is applied when considering fitness for an occupation. If it is reasonably foreseen that, because of a certain disease or disorder, injury could be sustained to either the person or others, then this exposure should be avoided if at all possible.

The emphasis should be on the candidate's ability in determining what is safe to undertake, not so much on the disability.

SUMMARY

The medical assessment of a disabled diver should not be different from that of a fully able person. Consideration should be given to the nature of their handicap, the effect of that handicap on the disabled diver's ability to operate independently in the water and, on their ability to provide assistance to their buddy. The examining doctor must fully understand the aetiology of the disorder and be competent to assess whether a diving-related illness such as DCS could result in progression of their symptoms and signs.

Many disabled divers will not be able to fulfill the certification requirements demanded by mainstream training agencies; however, with appropriate supervision and training they may be able to undertake a 'diving experience' with resultant positive psychological benefits.

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APPENDIX: CONTACT ORGANIZATIONS

Handicapped Scuba Association International, 1104 El Prado, San Clemente, California 92672, USA. Internet address: http://ourworld.compuserve.com/homepages/hsahdq/index.htm

International Association for Handicapped Diving. Internet address: http://www.iahd.org/index

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Occupational groups

CARL EDMONDS AND JOHN PENNEFATHER

INTRODUCTION

This chapter deals with the specific problems encountered by various occupational groups. Here, we summarize the type of diving performed by various occupational groups, and only mention in passing some of the physiological effects and illnesses which have been ascribed to them. These are more fully described elsewhere in this text.

Although there have been a few symposia devoted to these occupational groups, we are still awaiting a concise and readable text, describing the activities of both the traditional and the more recent diving occupations.

Many of the traditional occupational diving groups have become extinct. The **Greek sponge** divers of Symi and Kalymnos have inspired many authors. In Symi, one diver in three was either dead or crippled from neurological decompression sickness (DCS), before he had reached marriageable age. Others were disabled at a later age from dysbaric osteonecrosis.

Shell (pearl) divers from the *Tuamotus*, suffered Taravana disease – causing both death and disability. This was attributed to the extreme breath-hold diving exploits of these native fishermen. The dives were to 30–40 metres, lasting around 2–5 minutes each, over a 7-hour period. The disease probably encompassed a variety of illnesses, including otological barotraumas, the drowning syndromes and possibly cerebral DCS.

This chapter deals with the specialized diving groups other than those referred to elsewhere in the text, such as technical divers (see Chapters 62 and 63), deep and saturation divers (Chapters 67 and 68) and submariners (Chapter 70).

BREATH-HOLD DIVING

This form of diving is readily available to all, without relying on complex equipment or facilities. It is employed by primitive peoples and in remote areas, frequently permitting diving to depths of 20 metres or so.

The major causes of death from this activity include drowning, with or without initial hypoxia from hyperventilation or ascent, cardiac problems and environmental hazards (hypothermia, marine animal injuries, ocean trauma and entrapment) (see Chapter 61). Because of the multiple and rapid ascents and descents, **barotraumas** are particularly frequent.

The **Ama** are the traditional breath-hold divers of Japan and Korea, whose activities have been

recorded for over 2000 years. They dive repeatedly to depths as great as 18–20 metres, throughout the day, gathering shells, urchins and sea grasses. They would dive throughout the year, and the interesting observations have centered on their adaptation to cold exposure, with various physiological modifications. They also were noted to have an increased incidence of cardiac arrhythmias (especially during the winter months) and suffer some effects on foetal development.

Submarine escape tank trainers tend to dive to considerable depths, often 20–30 metres, in order to escort their submariners-under-training to the surface, and ensure that an appropriate ascent rate and exhalation are achieved by these trainees. Because these divers are very experienced and well-trained, barotraumas are not particularly frequent. Also, because the water is usually fresh, purified and warmed, many of the hazards of ocean diving are avoided. Unfortunately, the absorption of nitrogen with each dive does permit the occasional development of DCS, requiring recompression therapy.

Spear fishermen and other hunters tend to be less disciplined, and more frequently hyperventilate before their dive. Because of the competitive nature of this activity, the diver is likely to exceed reasonable depths and durations. Under these conditions the hypoxia which may develop with prolonged breath-holding associated with exercise, as well as the hypoxia on ascent, is a serious consequence. The loss of consciousness during the ascent, with subsequent drowning has been observed too frequently. These divers tend to be high-risk takers, although often very competent regarding their aquatic skills. Other major problems include barotraumas of descent, and marine animal injuries associated with hunting.

SURFACE SUPPLY DIVING

This equipment, using compressors or large storage cylinders, allows for prolonged durations at all depths. The tethering of the surface supply increases the possibility of entanglement. Carbon monoxide toxicity, as well as hydrocarbon (lipoid) pneumonia and saltwater aspiration, are consequences of the reliance on surface supply compressors and low-pressure lines respectively.

The **Australian abalone divers**, together with many others throughout the world, use a surface supply system because of their need to remain under water for longer periods, harvesting the shells. The sea conditions are often treacherous, and the work strenuous. The depths are usually less than 30 metres, and often less than 20 metres. However, with the shallower depths there is usually a greater effect of the sea state, with dangerous surge, and white water or rock formations. Marine and boating hazards are major contributors to accidents.

The diving is excessive, often 4–6 hours per day for over 100 days per year, and it is for this reason that the incidence of DCS and dysbaric osteonecrosis are increased. The former is related to the excessive nitrogen load absorbed during these periods at significant depths and with inadequate decompression staging, and the latter more related to the duration under water causing a nitrogen load in the slower tissues. Treatments of DCS tend to be somewhat individualistic, varying from deep air to shallow and surface oxygen regimes, applied in an arbitrary manner.

Because of the equipment used, compressor problems (including carbon monoxide toxicity) are not infrequent.

Pearl divers spend about the same time underwater as the abalone divers. However, they are much more regimented because they work as a team, usually being exposed to similar depths and total durations, suspended from a drifting boat while collecting shells before surfacing and then performing more 'drifts'. The Australian pearl divers were exposed to various depths which influence greatly the diving risks. Dives of 10–14 metres averaged 8.3 drifts per day for a total of 455 minutes and a DCS incidence of <0.2 per cent. Dives of 25–34 metres averaged 4.7 drifts per day, for a total of 285 minutes and a DCS incidence of 2.2 per cent. Dives of 45–54 metres averaged 4.4 drifts per day for a total of 152 minutes and a DCS incidence of 13.6 per cent.

The problems are similar to those of abalone diving, being mainly DCS and dysbaric necrosis. The former is often considerably reduced by the use of underwater oxygen at 9 metres depth, both in decompressions and in the treatment of DCS. This

prompt use of oxygen, at the earliest sign of any DCS symptom, allowed these divers to achieve excellent results with minimal treatment times.

Shallow construction/maintenance divers are mostly ship and harbour divers, who often use surface supply equipment because it allows them a greater duration under water. Most of their maintenance and construction work is in relatively shallow depths, often less than 10 metres. These divers are less exposed to DCS and dysbaric osteonecrosis than the shell divers. To avoid a complete loss of gas supply if the surface system fails, the diver should carry a reserve scuba cylinder on his back.

A rigid helmet gives an air space for clear communications and protection from head injury. It will also reduce heat loss and decreases the risk of drowning should the diver lose consciousness. A light or welding shade can also be fitted that permits communication with the surface and other divers – or even video/TV monitoring for surface control.

Because the diver will often work at a fixed site, it is common to wear heavy boots and extra weights. With this he may have to be lowered to the job and then hauled up. It is important that the weights be releasable so that he can ditch them in an emergency.

The rules for attendants, rescue (standby) divers and on-site recompression chambers vary with local laws, depths and profiles. Common sense requires a chamber if there is significant decompression time. Surface decompression, possibly on oxygen, may be a more acceptable and comfortable alternative to in-water decompression.

Because these divers work in harbours and around ships, one of the major dangers is trauma from marine craft and underwater equipment (explosives, oxy-welding, high pressure and suction appliances).

SCUBA DIVING

This equpment also uses compressed air, but permits greater mobility than surface supply – at the cost of reduced duration underwater, per cylinder.

Scientific divers often gather data and collect marine specimens in relatively shallow water, but for long periods of time. Unfortunately, scientific divers frequently are more skilled in their academic discipline, than in diving and so decisions may be made based more on the needs of the former, than the limitations of the latter. They often have an unrealistic faith in technology, such as decompression meters, and will utilize their verbal and intellectual skills to override the diving experience of others. It is for this reason that scientific divers will not infrequently place themselves at far greater risk than others.

Dive instructors are at increased risk of dysbaric illnesses, by virtue of their multiple dives, and often rapid ascents. The latter may be necessary in certain training procedures, such as ditch and recovery, whereas conservative dive profiles may be disrupted because of the sometimes unpredictable behaviour of trainees. The extra skills possessed by the group, in order to cope with both their own diving exposure and that of an inexperienced trainee, are often sorely tested. Both the death rate of dive instructors while diving and the incidence of DCS, is a source of concern. The responsibility and psychological stress imposed on many instructors is considerable.

Fish farm divers, as seen in Australia (tuna, salmon, pearl shell), Norway (salmon) and the United Kingdom (salmon) are becoming more commonplace. With tuna, the fish are captured at sea and then fattened and harvested within very large pens. Tuna divers have the added risks entailed in escorting the catch in ocean cages, for days or weeks on their way back from the exposed ocean to sheltered bays. There pens are used to feed, grow and value-add the fish for the lucrative Japanese sushimi market. Usually scuba is used in the open sea/towing phase of the diving, with scuba or surface supply for the diving at the actual farm (pen). Exposure to high seas, boating, underwater construction equipment, high-pressure gas and sunction pumps make this a high-risk occupation.

Salmon farm divers also work with fish being grown in pens, but are usually in sheltered waterways. The pens are frequently 10–15 metres deep, and may hold as many as 5000 fish. The pen is usually surrounded by a protective heavier mesh, to deter predators, and the whole structure is tethered by mooring lines, often extending to much greater depths. The whole structure has to be cleaned and maintained by the divers, who use either scuba or surface supply breathing apparatus.

Although the conditions vary considerably, the task of these divers is usually to dive repeatedly to depths of 10–20 metres, often for a couple of hours each day, maintaining, cleaning and unsnagging nets and lines, pontoon and jetty maintenance, fish feeding, removing dead fish, and ensuring protection from other predators, such as seals and sharks. Multiple ascents and descents increase the likelihood of dysbaric illness, both DCS and pulmonary barotrauma. The former is aggravated by deeper, repetitive and multi-day diving. The presence of under-water lines increase the likelihood of entanglements, and predators may also cause problems.

Among fish farm divers there have been a number of case reports of DCS, ostensibly from shallow water diving. On close inspection of these reports, in most cases the depths have either been in excess of 10 metres, or the diver has also undertaken deeper dives in conjuction with the shallower ones.

The Miskito Indians of Honduras and Nicaragua have relied heavily on lobster fishing to exist, since the early 1960s. The free diving changed to scuba in the late 1970s, and the results of inadequate diver training, and the absence of reasonable medical and recompression facilities in these remote areas, soon became apparent. Of the 5000 divers, most are employed on lobster boats which put to sea for periods of about two weeks. The divers would descend to depths as great as 27–33 metres, nine to 20 times a day. This extreme repetitive and multi-day diving exposure resulted in most of the divers being affected by DCS, with a high incidence of neurological manifestations.

The few recompression chambers available were distant from the diving area, resulting in an average of five to seven days delay in treatment. Between 1991 and 1997 there were over 200 divers treated in the local chamber, with 70 per cent having either paraplegia or quadriplegia as their presenting symptom. Despite the extreme delay, 65 per cent responded with good or excellent outcomes. However, many of the others were left with significant residual symptoms, often being paraplegic and requiring urinary catheterization for the remainder of their life – which was frequently terminated by renal infections, bed sores, etc.

Other common problems encountered by this group included carbon monoxide toxicity (from incorrect positioning of the compressor intake), shark attack, air embolism and equipment failure. Recent attempts to reduce the mortality and morbidity of this group have centered on a more appropriate management of the marine resource (ensuring a greater supply of lobsters and thus obviating the need to dive deeper), reduction of alcohol and drug usage, disability recompense and earlier treatments (surface oxygen, underwater oxygen and possibly even portable chambers).

Unfortunately, the Miskito Indians are typical of many other poverty-stricken indigenous communities who have had to extend their diving activities in order to utilize marine products, and have then had to extend their diving activities deeper and longer as the marine resource becomes more scarce.

DEEP AND SATURATION DIVING

Deep commercial divers

Non-divers and many recreational divers consider that the life and work of the professional diver is one of glamour and excitement. This is not accurate – it is often a tedious job with occasional catastrophic hazards. Usually it is cold, wet and boring, but reasonably well paid.

People only pay a diver to perform a job that cannot be done in a less expensive manner. The most efficient diving firms will survive, but this creates high expectations and more stress on the workers. Some commercial divers derive little pleasure from diving and do not dive recreationally.

Almost every job that is required for surface construction is also conducted underwater. With steel work for example, the diver can cut with flame cutters and mechanical saws, drill, rivet, use gas and electric welding, bolt with normal and explosive bolts, and apply protective coatings. The equivalent range of skills in concreting, carpentry or surveying can also be applied. Often a tradesman with a skill will be trained as a diver, rather than teaching a diver a new skill.

Oil rig divers are not only exposed to extremely hazardous environmental conditions on the oil rigs at sea, but also to techniques and equipment that have small safety margins. The risks of DCS and

dysbaric osteonecrosis are reflections of the large inert gas loading and also the lack of knowledge regarding dive exposures on the edge of our decompression experience. Deaths among the 1960–1970 'oil boom' divers in the North Sea were excessive, but were reduced considerably when the deep bounce diving, often to 100–200 metres, gradually became replaced by saturation exposures.

For deep water tasks the diver will generally be kept at elevated pressure in a deck-mounted compression chamber and commute to his work site in a submersible chamber. He will normally operate with another diver, with one in the bell as attendant and the other at the work site. With a change-over in roles the pair may work an 8-hour shift. The attendant's role is as a communicator, tool passer, hose attendant and possibly gas recycler.

In deep water the diver will normally be breathing an oxygen/helium mixture from a system similar to that used in shallow water. Because of the cost of gas there will probably be a return pipe system so that exhaled gas is collected, purified and reused. The recycling from the diver will generally be via the bell to reduce the length of hose. He will also need a source of heat to keep him warm; this may be by hot water from the surface via the bell, or from water heated in the bell. On-diver electrical heating is a logical alternative but is less common than hot water. Thermal problems are common. The bell will be supplied with gas, power, communication and hot water from the surface, and return gas back to the surface for processing and reuse. The use of different breathing gases, and the complexities associated with delivering these at great depths and for long durations, increase the likelihood of gas toxicities, hypoxia, DCS and dysbaric osteonecrosis (see chapters 67 and 68).

The period between work shifts and for decompression back to atmosphere is spent in a chamber mounted on the support ship or rig platform. It is equipped with inclusions such as bunks, table, shower and flush toilet. A communication system including TV or video is now commonplace.

Even in the relative comfort of saturation systems and decompression chambers, there are the continued problems of gas toxicities, contaminants and fire. In addition, in pressure chambers the high humidity and enclosed environment increase the likelihood of skin and ear infections. The latter can be particularly troublesome, and prophylactic measures are often indicated.

Caisson workers

Although not strictly applicable to a diving text, the experiences of caisson workers are of interest. These men are exposed to relatively shallow 'depths', usually less than 20 metres, but occasionally in the 20–30-metre range. The work shifts last from 4 hours upwards, and the slow compressions and decompressions make barotraumas much less likely. The major dysbaric problems are those of DCS, more frequently of the non-neurological type, and dysbaric osteonecrosis. Because of the long durations affecting the 'slow' tissues, this latter disease is particularly prevalent and appears not to have been eliminated by the otherwise more conservative schedules imposed over the past few decades.

The high humidity and sometimes unhygienic conditions of the caissons, compression and decompression locks, may increase the likelihood of otological and dermatological infective disorders.

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Diving in contaminated water

JOHN PENNEFATHER

INTRODUCTION

There is no need for the average amateur diver to enter contaminated water, but circumstances occur where a professional diver must do so. Often a medical officer with an interest in diving becomes involved as medical adviser to a professional diving squad. From this, because of their training in toxicology, they are asked for advice on diving in contaminated water. It should be noted that this diving is a highly specialized occupation, and the need to seek advice for a job normally indicates that the divers lack the appropriate training or equipment. This chapter does not make the reader an expert on the subject. It aims to indicate the problems that should be solved. It is a summary of some of the points expounded in Barsky's text on the subject.[1]

The first question to answer is that is the dive is really necessary? Barsky mentions a case where police dived in a sewage pond to recover a body. They found the body and the task could be regarded as a success, but it was not necessary as the plant had the capacity to drain the pond. If this could not be done a search could have been conducted by dragging the pond with nets. The man was seen to slip and fall in, so there was no suggestion of foul play. The job was unnecessary and exposed the divers to a high-risk microbiological environment with inadequate protection.

If the dive is needed, then the following points should to be answered. What is the nature of the hazard(s)? Can the diver be protected against them? Can the diver be recovered if he becomes incapacitated? Can he and his equipment be safely decontaminated?

HAZARDS

In general terms, hazards fall into four groups: chemical; biological; radioactive; and thermal. Guidance on dealing with them should be sought from the local hazardous materials disposal organization. They may not understand diving, but they will have the expertise to protect the attendants and the know-how to decontaminate them.

Chemical hazards can be caused by the need to dive in a processing plant, or as a result of an accident when chemicals have been spilt. The first may be simpler as the composition of the materials will be known.

Biological hazards are generally caused by bacterial and/or viral contamination of the water to be dived. Normally, a laboratory can identify and quantify the bacterial contaminants, which may include *Aeromonas, Klebsiella* and *Salmonella.* Also of concern are amoeba such as *Naegleria fowleri* (see Chapter 30).

Diving in **radioactive water** may be needed for two reasons. First, a carrier can have an accident and containers need to be salvaged from water. This is often an over-rated danger as most containers are damage-resistant. Second, in some countries, diving may be needed in nuclear reactors, but this should be left to the expertise of a large company experienced in the work. In some cases a remote-controlled vehicle can do the job, but in others a gas return line to the surface is needed to prevent gas pockets forming in the reactor pipes. Sometimes a diver in a 1-atmosphere rigid suit can do the work; such a suit provides better shielding, but in all cases there will be some exposure to radiation.

Thermal hazards are dealt with in Chapter 27. Heat stress can be a problem if a diver has to stand in the sun while their dry suit is decontaminated.

Diving in contaminated water requires good planning and calm implementation. There may be a claustrophobic element that even some experienced men cannot tolerate because an escape option may not be available. Previous experience in confined space work is a good predictor of success in this field.

DIVING EQUIPMENT

The equipment used should be matched against the hazard and diving required. As a minimum, a second air supply system is needed so that the diver can return on it if required. He also needs protection from the substance he is diving in, and this normally requires a dry suit and helmet, which reduces skin and eye contact with the hazard. It can be in a free flow design such as a standard rig; this has the benefit that any helmet leak is likely to be outwards and the helmet to suit join is well-sealed.

A demand system such as the Superlite helmet can be used, but this can be problematical because each inhalation normally creates a negative pressure in the helmet. This can cause the exhaust valve to leak a small amount of liquid into the demand valve. One practice is to fit two exhaust valves in series to prevent leakage. There is redundancy of the system when a bailout supply can be used if the air supply fails. The free flow can also be used to supply gas in the unlikely event that the demand valve fails. The other major advantage is that most commercial divers are familiar with such helmets.

A dry suit can be connected to gloves so that the diver's hands are protected from contact with the toxic material. A possible problem with chemical pollution is that the suit must be resistant to attack from it, and no suit material is resistant to all likely chemicals. A smooth-surfaced dry suit will also be easier to decontaminate than a fibre surface, as well as being easier to patch repair any leaks that occur.

Before entering contaminated water the system should be tested for leaks. This may involve a small pond like a play pool. It may also be part of the decontamination routine.

DECONTAMINATION

It must be possible to clean the diver and dispose of the washing liquid and other cleaning material safely. In many cases a hose, a brush and a platform so that the washings go back into the polluted liquid is adequate. Special care needs to be given to cleaning joins in the suit assembly. The helmet join is of concern because when it is opened, any drips often fall inside the suit. A planned method of recovering and cleaning down a disabled diver is needed.

Consideration should be given to the protection of the rest of the dive team, especially those persons responsible for cleaning the diver. In a civic matter, such as a police rescue squad, those responsible for cleaning up after land contamination may be available for this task, but they will need to be trained in the handling of the diver and his equipment.

Trials and exercises of the techniques before an emergency are a useful method of examining the problems and testing solutions, and represent a valuable insurance.

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Deep and saturation diving

JOHN PENNEFATHER

HISTORY

During the history of civilization, man has made a continuing effort to dive to greater depths (see Chapter 1). Early in the ninteenth century the dive depth was limited by the capacity of the air pumps, but when these were improved then depth became limited by decompression sickness (DCS). Another problem was that the limited air supply caused carbon dioxide accumulation in the helmet of the standard diver.

When decompression tables were introduced and compressors improved, man found that he could reach depths of about 70–80 metres before being seriously affected by nitrogen narcosis and not being capable of useful work.

Problems

The introduction of helium as a diluting gas allowed the depth limit to be extended, and in 1937 an American diver reached 128 metres (420 ft). However, dives to these depths required a long decompression, even for a short time at the working depths.

For example, a 70-metre air dive would require 100–150 minutes of decompression for a bottom time of 30 minutes. For the same bottom time at 100 metres, while breathing a mixture of 16 per cent oxygen in helium, nearly 3 hours of decompression are required (about 100 minutes of this is spent breathing oxygen, with a risk of oxygen toxicity). Both these dives would involve a significant risk of DCS.

Deep diving is associated with a more rapid consumption of the gas supply, increased respiratory resistance, thermal difficulties, voice distortion, sensory deprivation, inadequate information about decompression schedules, and equipment problems. Other difficulties include a much greater risk of dysbaric osteonecrosis, inner-ear disorders and a greater than usual danger from coincidental medical disorders.

Solutions

Three ways have been developed to cope with this problem of excessive decompression times. One is to avoid the excess pressure by operating from a submersible vehicle or a pressure resistant suit, at atmospheric pressure.

Hannes Keller demonstrated that decompression times could be reduced by **changing gas mixtures**

in order to capitalize on maximum gas tension gradients. In 1962 his experiments culminated in a dive to 305 metres for 5 minutes bottom time, though the divers still required 270 minutes of decompression. Keller's companion died, and this approach did not become popular with commercial divers because of the short bottom time and the risk of severe DCS. Ironically, the approach is being revived with the more serious forms of technical diving.

The total decompression requirements for a long task can be reduced by allowing the diver(s) to stay at depth until the task is finished, and then decompress slowly in a chamber. Thus, only one decompression is required. This procedure – called **saturation diving** – was first demonstrated by Dr George Bond of the United States Navy, who was exploiting a suggestion made by Behnke in 1942, as a method of increasing the duration of exposure in caisson workers. Although these men were instrumental in applying the concept of saturation diving, they were predated by Dr Cunningham (Kansas City, USA in 1927) who used air under pressure for several days, followed by a slow decompression, as a form of hyperbaric therapy.

The main value of saturation diving is that a diver needs the same decompression time for a dive lasting either two days or two months. Once the body has equilibrated with the gases in the environment at any pressure, it will not take up any more gas, so the amount of decompression will not increase.

Bond tested this concept with animals and then with men in compression chambers. In 1964 he had a group of four men in a cylindrical underwater house for nine days at a depth of nearly 60 metres (Sea Lab I). Other early dives based on underwater houses, or habitats, were conducted by Link and Cousteau.

Instead of permanent underwater habitats, saturation dives can be conducted by the use of transportable chambers. The first commercial work in saturation diving was conducted by Westinghouse Inc. The men lived in a pressurized chamber on the surface called a deck **decompression** chamber (DDC) and were lowered to work in a capsule called a **submersible decompression chamber** (SDC), often called a 'bell'. This allowed their transfer in the chamber to the working depth without any alteration in pressure. In 1965 this procedure was used for a series of dives to repair a dam in the USA. Four men at a time were pressurized, and they performed 800 hours of work in 12 weeks. With surface diving the men could have performed only about 160 hours of work during the same period.

The main need for saturation diving is in the offshore oil industry, which relies on saturation divers to carry out many tasks underwater. These include observations, welding joins in pipes, cleaning, antifouling and repairing damaged components. Military saturation dives have been conducted for a variety of purposes, but most have been for the recovery of valuable, dangerous or strategic items from the sea bed.

The recovery of the components from crashed aircraft is a common task, and salvage divers and treasure hunters have also used this technique. The recovery of a large amount of gold from the wreck of *HMS Edinburgh* in over 200 metres of water is one example. Oceanographers often find the submersible decompression chambers too restrictive, and have found fixed **underwater habitats**, with direct access to the surrounding terrain, to be a useful alternative as these allow direct observation and prolonged data collection. At the end of their period on the seabed they use a SDC to transfer to a DDC for decompression.

SATURATION DIVE PROCEDURES

Many skills are needed to conduct a saturation dive. The facilities include a diving tender, compression chambers, large quantities of compressed gas, and technical staff. There are also many logistic problems in navigation and seamanship, necessary to support such an operation. The details are beyond the scope of this book, but the aim of the remainder of this chapter is to outline the more important aspects of conducting a dive using a DDC and SDC. First, the biomedical and habitability problems in conducting a saturation dive are considered, after which the conduct of a dive is outlined. More common medical problems are mentioned in Chapter 68.

THE ENVIRONMENT REQUIRED

The area occupied by the divers needs to have a controlled atmosphere as the loss of either pressure or temperature control can cause the death of divers. The environmental maintenance systems need to be reliable and to be supplemented by alternative systems, and power failure must be allowed for in particular. Evacuation proceedings to be used if the ship or oil rig has to be abandoned, need also to be considered.

Contamination of the atmosphere also needs to be considered. Gas purity standards are discussed in Chapter 19, but more stringent standards are needed for deep saturation dives. Prevention of contamination, with monitoring systems to measure and control oxygen and carbon dioxide levels, are needed. Alternative breathing gases must be supplied direct to the divers for use if the chamber atmosphere becomes contaminated, although for a deep dive this can involve enormous reserves of gases being required.

Within large chamber complexes it may be possible to transfer the divers to another chamber if contamination occurs. Most groups conducting saturation dives have their own procedures and prescribed limits for certain contaminants. The limits specified reflect the attitudes of various authorities, as well as the operational performance of each diving system. Examples of such recommendations are:

- **Oxygen:** partial pressures of 0.2–0.5 ATA have been used, but the most common is about 0.4 ATA. This gives a safety margin between risks of hypoxia and pulmonary oxygen toxicity. Recently there has been a tendency to increase the oxygen pressure to about 0.5 ATA during decompression as this results in fewer cases of DCS. Because of the fire risk associated with high oxygen concentrations, the chamber oxygen is generally kept below 21 per cent during about the last 10 metres of ascent. The reduction of oxygen pressure may require a slowing of the ascent rate. The alternative is to use periods of breathing high-oxygen mixtures, from a mask with an overboard dump system.
- **Carbon dioxide:** this is kept below a limit of 0.005 ATA, equivalent to 0.5 per cent at 1 ATA. For shorter periods a higher limit, 0.015–0.02 ATA is tolerable. A high carbon dioxide level will lead to hypercapnoea and a reduced work capacity.
- **Diluting gas:** This may be nitrogen for shallow dives, but for deeper dives helium, hydrogen and nitrogen mixtures with oxygen have been used. Each mixture requires a different decompression schedule. Addition of extra nitrogen to an oxygen/helium mixture is often used to reduce the high-pressure neurological syndrome (HPNS), but this increases the risks from DCS. If it cannot be avoided, it may be necessary to use a slower decompression than the standard oxygen/helium schedule. **Hydrogen** is being trialed as diluting gas, particularly by the French, who find it to be like nitrogen in reducing some of the HPNS symptoms. They have added hydrogen to the helium/oxygen mixture at depth and used it in the breathing mixture for dives. If used in the DDC atmosphere, hydrogen must be removed during ascent so that there is none left when the oxygen concentration is high enough for there to be a risk of combustion. At depth, the oxygen is diluted by so much inert gas that a hydrogen fire will not burn.
- **Trace contaminants:** various authorities specify arbitrary limits for a variety of possible contaminants. The limits are extrapolations from occupational health advisory groups, adjusted for depth. Because the designated contaminant limits are not comprehensive, medical staff need to consider the toxicology of many preparations they prescribe or wish to use, at depth. For example, solvents may pose a problem. Mercury thermometers or sphygmomanometers should be avoided because of the possible generation of mercury vapour and the possibility of the chamber being condemned because of amalgam formation. Other common sources of toxic products include paints, welding, refrigeration leaks and cooking fumes. Activated carbon or molecular sieve compounds may be needed to control contamination.
- **Temperatures:** the high thermal conductivity of helium and hydrogen mixtures requires an increase in the optimal working temperature, and narrows the range for thermal comfort. This may increase from about 25°C to 33°C, with greater depths. Deviations from this range

can cause hypothermia or hyperthermia, and need to be avoided. Hyperthermia may occur rapidly and is not easily detected at an early stage.

- **Humidity:** humidity levels need less strict control than the other parameters. When using soda lime to remove carbon dioxide, a relative humidity (RH) of over 75 per cent gives better performance. Other absorbents may require different optimal percentages. A higher RH also reduces the risk of static sparks, a possible source of ignition in the fire risk zone. The problem with accepting a high RH is the increased risk from certain bacterial and fungal infections, which can be a problem in saturation dives – so the range chosen will be a compromise. With water from wet gear and showering, it is often difficult to keep humidity down to the 60–75 per cent RH range recommended.
- **Fire hazards:** If the oxygen pressure is kept at 0.4 ATA there will be a fire hazard in the chamber at depths shallower than about 50 metres. This is called the 'fire risk zone'. To reduce the hazard, precautions are needed to limit the amount of combustible material. At greater depths there will be insufficient oxygen concentrations present, to support combustion.

THE ENVIRONMENT CONTROL SYSTEM

Processing and analytical equipment is needed to provide the required environmental conditions. Typically, the chamber gas is drawn through scrubbers to remove carbon dioxide, then past a cold-water radiator to condense excess water vapour, and then past a heater coil to rewarm the gas to provide a comfortable temperature. Other chemicals absorb the trace contaminants and odours. The scrubbing and temperature control units are normally in the chambers, but in the case of the DDC they can be outside the chamber with pipes connecting the purification system to the chamber. If the equipment is backed-up and can be isolated from the chamber it can be serviced during a dive. If it is in the chamber, then the divers must do any servicing, although siting it inside the chamber does simplify the piping design.

Gas analysis equipment is needed to keep a check on the composition of the atmosphere. As a minimum the following measurements are needed:

- **Oxygen**, to show when oxygen should be added to replace metabolic consumption.
- **Carbon dioxide**, to show that the scrubbers are working; if there is a rise in reading it may indicate that the absorbent is exhausted.
- **Temperature and humidity** are needed to show that the divers are in a comfortable thermal environment.

These parameters should be measured continuously from each compartment. Other monitoring may be less frequent; for example, trace contaminants will build up slowly and so daily measurements may be adequate.

It is also normal to have TV monitoring of the divers in each compartment so that the supervisors can watch for problems developing, as well as perhaps advising on any problems that do occur.

OTHER HABITABILITY REQUIREMENTS

The divers will need toilet and washing facilities; these are normally provided in a small outer lock. This allows the space to be depressurized for cleaning without depressurizing the rest of the chamber. Eating and sleeping spaces are provided in the inner compartment, or compartments. To minimize boredom it is normal to provide amusements such as access to TV and music.

Above all, consideration needs to be given to things that can go wrong. This can range from a power or equipment failure to the ship carrying the DDC sinking, and answers to most problems have been evolved. In some systems the occupants of the chamber move to a chamber built into a lifeboat that is connected to the DDC. If the vessel sinks or catches fire, the DDC occupants then move into the chamber on the lifeboat which is then launched with a surface crew to operate the chamber.

Gas will be used by several processes, and the diver in the water will probably be on open circuit. Any equipment failure in the gas purification

systems or contamination of the chamber will require the divers in the DDC to use the built-in breathing system (BIBS). This gas is normally collected, purified and compressed back into cylinders for reuse.

THE SATURATION DIVE

The compression phase

It is common to commence an oxygen/helium saturation dive by compressing the subjects to about 2 ATA on air, which allows the chamber seals and oxygen sensors to be checked, without wasting helium. At that stage the divers and operators can test systems before continuing the compression with helium. The other alternative is to compress with helium and then oxygen to create an atmosphere with 0.4 ATA of helium, without creating a dangerous oxygen-rich mixture.

The compression can continue until the desired depth is reached, or a depth at which problems of compression arthralgia or HPNS can be expected. From that depth, compression is slowed and pauses are introduced.

Several problems of a mechanical nature can occur during compression. In chambers with poor gas mixing, the lighter diluting gases can float over the heavier nitrogen and oxygen, giving a hypoxic layer and an oxygen-rich layer. This can be avoided at the design stage by injecting fresh gas where there is good gas circulation. Sensors that are influenced by pressure can give misleading indications of gas concentrations, and these should be avoided by selecting more suitable sensors. A rapid compression can cause overheating and hyperthermia.

At maximum depth

Once the chamber reaches working depth the divers will start to use the SDC. In normal operation, two divers enter the SDC from the DDC and are lowered to the work site. One remains in the chamber, and the other enters the water from a door in the bottom of the SDC and goes to work in diving gear. They are both supplied with gas from the surface that is fed to them, but the diver also receives gas and hot water from a hose that leads back into the SDC. Electrical power for lighting, heating and any tools also comes from the surface. TV cameras and voice communications allow the surface crew to monitor and assist in the work by lowering tools and operating cranes and hoists as needed.

The man in the chamber is there to assist the man in the water and rescue him if he gets into difficulties. The normal work pattern is an 8-hour shift, with the diver and the SDC operator swapping about half-way through the shift.

When pressurized, there is a depth range through which the men can move without risking DCS. It is normal for the depths to be chosen so that the sea bottom is the lower limit of this depth range and DDC depth at the upper limit of the range. A saturated diver can move through a range of depths from 100 metres to 129 metres and return, without concern about DCS.

The diver commonly uses modified demand equipment such as a Superlite helmet, which will be fitted with a gas heater so that the inhaled gas is warmed. The diver will also be heated, probably by a hot-water suit. A back-mounted cylinder of gas will be used to get back to the SDC if there is a gas supply failure. Because of the gas density the cylinder will have a very short endurance; a cylinder that lasts 60 minutes will last less than 2 minutes at 300 metres.

The decompression phase

The decompression phase is long because the rate of ascent is in turn set by the rate of gas elimination from the slowest tissues. The United States Navy schedules take about 12 days to decompress men from 300 metres pressure. Boredom of both the divers and chamber operators, with subsequent loss of concentration, may develop during the protracted decompression from a saturation dive.

A saturation dive is an expensive complex operation, but it can be conducted with safety provided that everybody knows and does their job. What can go wrong is considered in Chapter 68.

RECOMMENDED READING

Davis, R.H. (1962) *Deep Diving and Submarine Operations.* Seventh edition. Surrey, UK: Siebe Gorman & Company, Ltd.

Haux, G. (1982) *Subsea Manned Engineering.* London: Baillière Tindall.

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U.S. Navy Diving Manual (1966) Chapter 12, Saturation Diving.

68

Deep and saturation diving problems

JOHN PENNEFATHER

DIVER SELECTION

It has been said that a saturation diver is more remote from full medical care than an astronaut on the moon. For this reason, care needs to be exercised with selecting a diver who may be developing a medical problem. In the commercial world this is more difficult because the cash benefits of a dive may lead to denial of symptoms.

Apart from normal medical fitness and fitness to dive, two areas need special consideration in relation to the selection of saturation divers compared to normal divers. Psychological and dermatological problems are common in saturation diving. Not all divers have the stable, phlegmatic personality needed for saturation diving. The chamber operators and other support staff need to be aware of the stress on the divers and make allowances for this. However, it is also important to not select divers who lack the stoic stable temperament that is needed.

MEDICAL PROBLEMS

Because of the risk of infection the diver should be free of acne, bacterial and fungal conditions. Any skin infection is likely to aggravated in the warm, humid, oxygen-rich environment of the saturation complex. The environment is similar to an incubator with the diver as the culture medium, and for this reason a proactive policy is needed to avoid infection. Aluminium acetate ear-drops after each dive and shower, and routinely during the day, are often used to prevent otitis externa. The risk of cross-infection is reduced by having separate containers for each diver, or even for each ear. The risk of skin infection is reduced by daily showers and the use of medicated soap, followed by clean clothing. Preventative measures are also needed to avoid tinea of the feet or groin. Maintaining a high standard of cleanliness in the pressure chambers and of diving gear also helps to prevent serious infection. The US Navy has controlled the ear problem by a policy where drop use is observed and timed so that the drops are instilled for a set time in a controlled manner throughout the dive.

Problems during descent

Apart from aural barotrauma, the main problems to be expected during descent are compression arthralgia and the high-pressure neurological syndrome (HPNS) (see Chapters 20 and 40). They may

be minimized by using an established compression regime, and are treated by suspending compression until symptoms abate. HPNS has been reduced in deep dives by the use of nitrogen and/or hydrogen as part of the gas mix.

Problems at depth

Once working depth is reached, the activity for which the dive is conducted can start. In a commercial saturation dive this will normally involve submersible decompression chamber (SDC) excursions. The common problems encountered at depth are related to the diving tasks. These include burns from the hot water used for heating the in-water diver. The other problem is diver **dehydration**, which has two main causes – heating the diver by hot water induces sweating, while the use of dry gas in the breathing system also consumes water. This can result in significant weight loss, with more than 5 per cent loss of body weight having been reported in some cases.

Hypothermia will occur quickly if the heating fails, either in the water, the bell or the habitat. **Breathlessness**, associated with exertion, is a common consequence of the density of the breathing gases at depth, and can lead to hypercapnoea. The development of pulmonary oedema due to breathing cold gas was discussed in Chapter 27.

Industrial accidents or diving equipment problems can lead to the attendant having to bring an unconscious diver back into the SDC. One method is partially to flood the chamber so that the diver floats up into it. The attendant then shuts the hatch and blows the water out. Another method is to bring the diver back to the hatch and then winch him up into the bell. Whichever method is adopted, it should be rehearsed during training.

Because of the isolation and risk of injury it is normal for the divers to have good first-aid training. Decompression sickness (DCS) is more likely during the decompression phase of a saturation dive, but excursions from the saturation depth can also cause DCS if the safe depth excursion range is exceeded.

Situations in which a diver breathes one gas while he is surrounded by another can cause isobaric counter diffusion (see Chapter 11), an unusual type of bubble-related disease. This should not occur with the normal choice of gases, however.

Problems during decompression

Boredom of the divers and chamber operators, with subsequent loss of concentration, may develop during the protracted decompression from a saturation dive. If the decompression schedule used is a proven one, then DCS should be uncommon. If it does occur it will usually be mild, develop close to the surface or after surfacing, and should respond to a pressure increase of 30 metres or less, with periods of breathing oxygen-rich mixtures, at an oxygen pressure of 1.5–2.5 ATA. Six periods of 20–30 minutes with 5-minute breaks are commonly used. Decompression can be continued after a 'hold' at the increased pressure, and often a reduced ascent rate is used. Cases that appear after surfacing are usually treated on a conventional oxygen therapy table.

Uncontrolled and rapid decompression is likely to result in a severe type of DCS, such as when the habitat diver becomes buoyant and ascends to the surface, or when the pressurization within the decompression chamber is reduced too rapidly. Under these conditions the treatment may need to be vigorous, with a rapid return to the previous pressure.

RESCUE

Several circumstances can occur where a diver or divers need rescuing or evacuation, and these are outlined below.

Rescue of divers trapped in a bell

Failure of the lifting wires connecting an SDC and its surface support can lead to divers being trapped on the sea bed. The other potential problem is a winch failure or fouling of the bell or cable. These incidents are rare and are usually preventable, but rescue procedures should be available nonetheless. These should include emergency environmental control and salvage methods. Some designs of SDC

can sever connections and drop ballast to improve their chance of making an emergency ascent, after which another crane (if available) on the platform can lift the SDC back onto the deck decompression chamber (DDC) connection.

The provision of a rebreathing system can protect the divers from carbon dioxide accumulation in an isolated SDC. It may also give thermal support by reclaiming the heat in the exhaled gas and from carbon dioxide reacting with its absorbent. The outer surface of the rebreather should be insulated to trap this heat. A supplementary oxygen and mixed-gas supply may be needed, together with a means of monitoring the gas pressures.

Thermal protection may be active or passive. An active system incorporates a heating unit, usually an electrical or hot water supply from the surface. In deep dives and in cold water, active heating within the bell is used for a normal dive. Passive protection relies on insulation to protect the diver from excessive heat loss. SDC insulation, warm clothes and a rebreathing system will all help to keep the crew of a trapped SDC alive until they can be rescued.

These precautions need to be linked to a **rescue procedure**. For example, British regulations require each SDC to be fitted with an acoustic beacon to help rescuers locate it. Divers from a second SDC may be able to free the trapped bell, or release its ballast weights. The other possible method is for the divers to transfer to the second bell, but freeing the trapped bell would normally be the preferred option.

Evacuation of divers from a DDC

There are several circumstances that may necessitate the evacuation of divers from a DDC. For example, it may be necessary to leave the diving platform because of fire, explosion or collision damage. Other factors may require abandoning the deck chamber – it may become uninhabitable due to contamination, and oil field diver operators are often well prepared for such incidents.

Hyperbaric lifeboats have been designed to allow divers to escape in a pressurized chamber. This consists of a lifeboat with a chamber in its hull. A connection to the deck chamber allows the divers to transfer under pressure to the lifeboat. The boat would then proceed to another chamber where the divers would be transferred to a less restricted facility. The lifeboat itself has all the potential hazards associated with both watercraft and decompression chambers, however.

Treatment of ill and injured divers

A saturation diver at depth is one of the most isolated people on earth, as in an emergency situation it may take several days for a doctor to be transported and pressurized to reach him. An accident or illness may make it desirable to move a diver to a more appropriate facility, while he remains under pressure.

Hyperbaric rescue chambers have been developed to allow the evacuation of a sick or injured diver to a chamber connected to hospital facilities. The diver is moved into a portable chamber, called the 'transfer chamber', and taken ashore by helicopter. The procedure is reversed to transfer the diver to a hospital-based chamber. The North Sea field system allows the transfer of a diver at a pressure of up to 23 ATA, but this system is complex, takes time to set into operation, and is weather-dependent.

Medical care on site is the common approach to caring for sick or injured divers. To expedite this, several divers in each group need comprehensive paramedic training. The Royal Navy training, for example, includes suturing, catheterization and insertion of intravenous infusions. People with this type of training can then initiate treatment and obey expert advice without delay.

Use of drugs under pressure is a field of pharmacology where experience is very limited, notably because drugs may have different effects under high pressures (see Chapter 43).

69

Hyperbaric equipment

JOHN PENNEFATHER

INTRODUCTION

There are two main types of human occupied pressure vessels used in medicine and research. These are:

- **Hyperbaric chambers**, where the ambient pressure can be increased from 1 ATA to many times atmospheric pressure depending on the structural capabilities of the vessel concerned. These chambers are used for diving, and hyperbaric medicine and research, and are built to withstand an explosive force.
- **Hypobaric chambers**, where the ambient pressure can be decreased from 1 ATA to sub-atmospheric pressure levels. These chambers are used for aviation, space medicine and research, and are built to resist an implosive force.

Some chambers are capable of performing both functions, but these are not common and are mostly found in research centres. This chapter is concerned with the features of hyperbaric chambers that are of concern to the medically orientated users.

The hyperbaric chamber is subject to various nomenclature depending on its primary use; terms used for chambers used in diving include the following:

- The **recompression chamber** (RCC), where the major use of the hyperbaric chamber is for compressing a caisson worker or diver, usually as part of a therapeutic regime. It may also be used in the training of divers, so that they may experience the effects of hyperbaria.
- The **decompression chamber**, where the major use is to decompress a subject already exposed to increased pressure.
- A **submersible decompression chamber** (SDC) which is used to transport divers under pressure to and from the working depth.
- The SDC usually allows transfer under pressure to a surface or **deck decompression chamber** (DDC); this allows for the definitive decompression under controlled conditions. The diving bell, which is open at the bottom and exposed to ambient sea pressure, was the forerunner of the SDC.

Only the hyperbaric or recompression chamber is discussed in this section, as this is the chamber of importance to diving medicine. It consists of a strengthened vessel or hull which can be pressurized

by compressed gas. The gas may be supplied from gas cylinders or direct from a compressor. Decompression is achieved by allowing the gas to escape.

Since the 1920s, numerous hyperbaric chambers have been designed for the treatment of divers and caisson workers. Earlier chambers were used for hyperbaric medicine and study of the effects of pressure change.[1] However, with the progress of hyperbaric and diving medicine, chambers are becoming more complex, and their design now requires the involvement of personnel ranging from the specialist diving physician and the diver to the design and construction engineer.

RECOMPRESSION CHAMBER CLASSIFICATION

The initial consideration in designing a hyperbaric chamber concerns its expected use. There are several main types of recompression chambers, viz:[2]

- Large multicompartment chambers used for research as well as treatment of divers and caisson workers and capable of compression to many atmospheres (greater than 5 ATA). These may incorporate a wet chamber where immersed and pressurized environments can be created for training, research and equipment testing activities.
- Large multicompartment chambers capable only of low pressures (2–4 ATA) used for treatment with hyperbaric oxygen.
- Portable high-pressure multi-man chambers for treating divers and caisson workers.
- Portable one-man, high- or low-pressure chambers used for the surface decompression of divers. Similar chambers are widely used in hyperbaric medicine units. These sometimes have to be used for treatment of divers and caisson workers requiring recompression therapy.

The low-pressure units may be inadequate for the treatment of an emergency such as cerebral air embolism which may arise during their use. Any properly designed high-pressure complex should be capable of treatment of both diving accidents and hyperbaric medicine patients.

Fashionable jargon is often used to describe the numbers of places available in chambers. Thus 'multi-place' and 'mono-place' are meant to indicate the numbers of patients that can be accommodated, but these terms may also infer fixed and portable chambers, respectively. The implication is not always accurate, and the numbers of places available in a chamber may also need to be modified, depending on the clinical circumstances.

A basic choice in chamber design is between scrubbing the chamber atmosphere to remove carbon dioxide and flushing with fresh air. For a chamber that is expected to be routinely pressurized to more than 20 metres the cost and noise of air flushing may make a scrubbing system preferable, but this introduces the need for more complex monitoring equipment. For this reason an engineer who is an expert in the field should be consulted.

In most countries and organizations there will be one or more codes that any chamber should comply with. In any country it will include the pressure vessel code. In a ship-mounted chamber it will normally be to the code of the organization that classifies the ship, e.g. Lloyd's, American Bureau of Shipping or DNV.

LARGE MULTICOMPARTMENT RECOMPRESSION CHAMBER

This is a high-pressure chamber capable of accommodating several persons in each lock. It should be suitable for treating divers, caisson workers and patients requiring hyperbaric oxygen.

The recompression chamber may be composed of the following elements, the principles of which have medical implications.[3] The finer technical details will not be discussed.

Hull

This should consist of two or more interconnecting chambers or locks, some of which can be separately compressed or decompressed. The number of compartments depends on the use of the unit, but the usual system consists of a large inner lock and a smaller outer lock. The inner lock is used for therapy and the outer lock for transfer of personnel. Useful information on all aspects of chamber design is provided by Jain.[2] The inner lock should also have a

medical lock to allow transfer of items such as food, excreta, drugs and equipment.

If constructed as a cylinder, the chamber diameter should be sufficient for an adult to stand erect in both locks, especially the inner lock where most treatments are conducted. This requires an inner diameter of at least 2 metres. The length of the inner lock should be over 3 metres in order to accommodate a prone patient on a stretcher and allow free movement of attendants around the patient.

A cylindrical shape has been the normal form for the hull until the last few years. Recently, Fink Engineering in Melbourne, Australia have developed a more square-section box form with rounded joins along the edges. This has been the common shape for most new large chambers installed in Australian hospitals. This provides better external space utilization when the chamber must be fitted into a building. In the interior the users appreciate the more spacious form with headroom being available over beds fitted along the walls. Larger doors can be fitted to facilitate access. A chamber of this type is shown in Figure 69.1.

Pressurization capabilities of such a chamber will depend on the anticipated requirements, but should be as great as possible to allow for all eventualities. It is likely that a pressure capability of 100 metres will suffice for nearly all cases.

Pressure-sealed doors are needed between the outer lock and the area outside the chamber and for the opening into the inner lock. Doorway dimensions and locations should allow easy entry and removal of persons and stretchers. **Observation ports** should be fitted to both or all locks so that the entire compartment can be easily observed. All interior surfaces should be painted with light, easily cleaned, unreflective, fire-retardant paint. The coating should minimize static electricity.

Furniture and fittings

Comfortable seating should be available for the anticipated number of occupants because decompression may be prolonged. Fold-away seating allows maximum use of space for different requirements. Both locks should have metal storage shelves or lockers for storage of medical instruments, etc.

Lighting of both locks can be either direct interior pressure-sealed and fire-safe lights, through-hull fibreoptic lighting, or external lights separated from ceiling ports by heat filters or air-flow cooling systems. Both locks should have shadow-free lighting.

Facilities should exist for one or two stretchers or trolleys to be used, at least in the inner lock. The trolley should be 1 metre high with adjustable head and foot elevations, and fitted with antistatic straps and

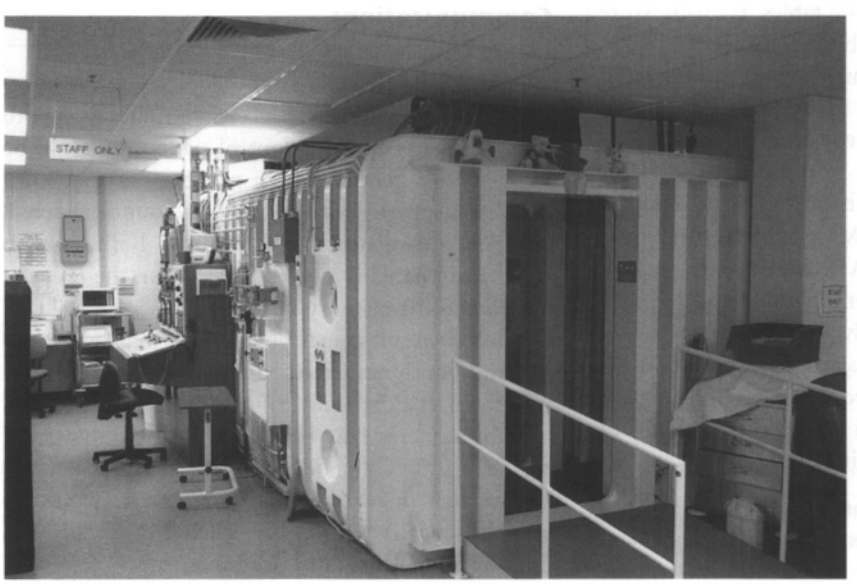

Figure 69.1 *Large, multicompartment recompression chamber. (Photograph courtesy of Cowan Manufacturing.)*

lockable wheels. Due to the confined chamber space and door design the trolley system may not be appropriate. In such cases removable rails may be utilized to facilitate moving a stretcher into the inner lock.

Chamber plumbing

Intake openings into both locks should be muffled to keep sound levels below 90 dB at maximum compression rate. The inlets should be distant from the outlets to promote gas circulation, and not adjacent to personnel or movable objects. Outlets should be placed in inaccessible positions on the ceilings and covered by grids. Outside the chamber the pipe openings should be removed from the control panel to avoid a noise hazard. They should not be near heating or electrical apparatus due to the danger of fire from possible high oxygen partial pressures in the exhaust gases.

Equalization valves may be needed between locks to allow doors to be opened and the transfer of people between compartments.

Breathing gas supply

Pressurization of both locks should be under the control of the panel supervisor. The chamber should be capable of a pressurization rate at least equal to that of diving and therapeutic tables. High-pressure air should be ducted into both locks from a large storage bank after having passed through a filtration bank from an electrically driven compressor. A standby diesel or gasoline compressor may be needed.

Provision for breathing helium/oxygen or other gas mixtures is required. Most tables require the patient to breathe oxygen. A second storage bank of oxygen/nitrogen and oxygen/helium mixtures may also be needed for more advanced tables and allow the attendant to breathe gas if the chamber atmosphere becomes contaminated.

Two systems of oxygen or mixture supply to patients and attendants are in common use, viz:

1 A reducer, flow meter, and an oronasal mask and bag. As an alternative, this may supply a hood surrounding the patient's head.
2 A demand valve and regulator analogous to scuba.

Exhaled oxygen-rich gas should be fed into an overboard dump system to prevent high oxygen levels developing in the chamber.

The breathing gas supply should be useable as an emergency air supply. It may be called a built-in breathing system (BIBS), and should be fitted for all personnel in both compartments. These consist of demand valves connected by short high-pressure hoses to spring-loaded bayonet mountings on a bulkhead. All hoses should be fire-resistant. A breathing gas supply should be available that bypasses the main supply, coming from separate emergency bottles. This system will ensure an air supply and prevent toxic fumes caused by burning contents from overcoming the chamber occupants.

While this text was being printed contamination was found in the breathing gas supply lines on two Australian recompression chambers. It seems that this was caused by lubricants and particles from the pipe walls. It is not yet known how widespread the problem is. It is also not yet known if recleaning will be needed in other systems or how often pipe systems should be stripped for cleaning. Readers should consult an engineer with experience in gas systems if they are concerned that the problem may apply to their chamber.

Communications

Communication facilities should include an intercom or two-way transmit/receiver unit between both compartments and the control panel. It should be fitted with a helium speech processor if a helium-rich atmosphere is to be used. A sound-powered, electrically insulated telephone from both locks to the control panel is a useful back-up in the event of a failure in the primary system.

Closed-circuit television coverage may be needed so that all occupants can be monitored from the control panel. If electrically safe, or in a pressure housing, the cameras may be located inside the locks. If not electrically safe, they can be located at an observation port on the outside. An auxiliary back-up electrical power source is needed for compressor, lighting and all ancillary equipment.

Transfer under pressure

If transfer to or from another chamber is needed, the outer opening of the outer lock should be fitted with a system enabling transfer under pressure from a portable chamber using a transfer sleeve. Such manoeuvres may require a sling and gantry or rails to support the mobile chamber. A stable system is needed to avoid pivoting at the connection between the chambers. There has been a general standardization on the NATO size connection.

Fire

Sheffield and Desautels[4] report that fires in 35 hyperbaric chambers have claimed 77 lives. Other incidents have involved experimental animals. The severity of these fires is indicated by the fact that in this series there were few survivors, and none of the survivors was in a chamber which had an oxygen-rich atmosphere. Most recent fires have been caused by occupants taking dangerous material into the chamber.

Any fire has three prerequisites: a source to ignite it; fuel; and oxygen. Fire can be prevented by removing any of these elements. In a chamber the hair on the diver's skin can provide fuel to spread the fire, and if the oxygen partial pressure is greater than about 1 ATA then hairless skin will burn. Oxygen must be available for the diver to breathe and is often present at high partial pressures – so two elements of fire are always present.

Further aggravating factors are the increased ease of ignition and combustibility that exist in some hyperbaric conditions. Substances that do not burn in air can burn if the oxygen partial pressure is elevated, and this even includes some substances treated with fire-retarding chemicals. Even static sparks generated by clothing could cause a fire as a result of the increased ease of ignition.

An apparent contradiction to these facts is that it is impossible to sustain a fire in some of the gas mixtures used in deep diving because of the low oxygen concentration.[5,6] This cannot be used as an excuse to avoid precautions against fire because in these conditions there may be pockets of gas that can sustain combustion, and periods of high fire risk will be encountered during compression and decompression.

The safety of the chamber occupants depends on everyone involved being aware of the fire hazards, and their continuing vigilance to prevent any action that could increase the risk of fire. Risks can be minimized and fire-fighting systems installed.

Both locks should be fitted with fire-fighting facilities using water as the extinguishing medium. A system of spray nozzles should be capable of wetting all surfaces thoroughly with a fine spray or mist within 2–3 seconds of the onset of any fire. Continuous spraying requires a compensatory air loss to avoid an increase in pressure as the locks flood to half their height. Activation of this system can occur automatically via ultra-violet, infra-red and/or carbon dioxide sensors. Manual switches should be placed inside the locks and on the control panel. A secondary system of hand-operated hoses fitted with spray nozzles should be fitted in each lock so that an attendant can direct water on to any source of fire or heat.

An oxygen elimination or exhaust dump system is a mandatory requirement for modern chambers, when the subjects breathe from masks. This enables expired gases containing elevated oxygen and carbon dioxide pressures to be exhausted to the exterior. Techniques used include extraction from a reservoir bag using a Venturi system connected to the exhaust, or non-return valves.

Any padding allowed on stretcher or seats is best constructed of material which is not only fire-resistant or non-combustible but which will not, if exposed to extreme temperatures, produce toxic fumes. The same applies to all clothing and bedclothes within the chamber, and this should be specially treated to minimize the risk of static electricity build-up. High partial pressures and concentrations of oxygen increase the fire risk in a hyperbaric chamber.

Instrumentation and operation

Emphasis should be placed on obtaining rugged reliable equipment in preference to sophisticated but delicate instruments.

Control panels should always be fitted with easily

read instruments, and must be located in a manner that prevents confusion as to which lock they refer. Colour codes are useful, and each lock should have different-coloured instruments.

Pressure/depth gauges should be sited in both locks and on the control panel. Two gauges may be coupled, one covering the low pressure range and the other the high pressures. An independent pressure transducer connected to a chart recorder or logger is useful in providing a permanent dive profile record.

Ambient oxygen concentrations should be measured in both locks and displayed on gauges on the control panel. A warning system can be incorporated, activated by deviation of oxygen concentration outside pre-set values. A similar system can be used to warn of a dangerous rise in carbon dioxide. **Clocks** should be fitted so that elapsed time and individual compression/decompression times are easily recorded. **Flow meters** that measure the ventilation rates on the exhaust lines from both locks allow adequate ventilation rates without air wastage and help minimize noise. Temperature and humidity should be controlled and monitored.

Electrodiagnostic monitoring should be possible in each lock. Physiological parameters which may need recording include the electrocardiogram, the electroencephalogram and the electronystagmogram.

A broad guide to the suitable design for a large treatment chamber has been discussed. Ideally, such facilities should be situated near major hospital complexes to ensure optimal medical control as well as expert maintenance. Ground level locations are preferable because of transfer under pressure requirements with mobile chambers. In view of the high noise levels associated with their function, soundproofing is also desirable.

PORTABLE RECOMPRESSION CHAMBER[7]

These treatment chambers should be capable of being transported by air, and therefore size and weight problems are important. The gas pressurization systems accompanying such units must be included in size and weight allowances.

Most of these chambers are designed to accommodate two or three persons in the single or main lock. Apparatus is best kept to a minimum, providing only the essentials. These chambers are principally used for a diver suffering from decompression sickness or air embolism. Once compression is achieved, the patient can be transferred while still under pressure for subsequent therapeutic decompression. Because of the possible complications in such a transfer the US Navy and Royal Australian Navy have purchased small chambers that house a patient and attendant. If needed, a second chamber can be connected to it as an outer lock or the chamber can be transferred if this is needed. A pair of these chambers is shown in Figure 69.2.

The minimum requirements for these chambers are as follows. The **hull** should be of appropriate

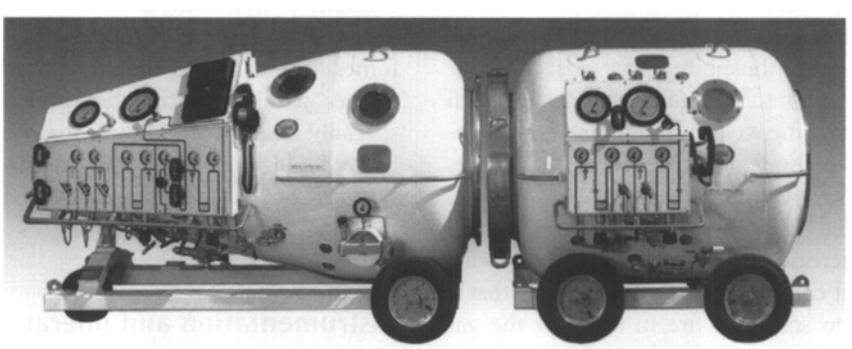

Figure 69.2 *Portable, lock-on, two-man recompression chambers. A side view of the chamber system built by Cowan Manufacturing for the American and Australian Navies. The cone-shaped treatment chamber is shown next to the cylindrical transfer chamber. When the two chambers are joined they can be operated as a two-compartment chamber. (Photograph courtesy of Cowan Manufacturing.)*

weight and dimensions to be loaded onto the transport aircraft. A maximum size would be about 2–3 metres in length, 1–1.5 metres in diameter, and 600 kg in weight. Trolley or pallet mounting of the unit will assist with later transport. The **door** surround should have a transfer under pressure flange compatible with other systems. A **medical lock** is valuable, as are observation ports. Adequate **lighting** and **communication** should be incorporated. A roll-in stretcher is of considerable advantage.

Soundproofing of inlets and protection of exhaust outlets similar to the large recompression chambers is essential. **Temperature control** may be needed in extreme climates and with rebreathing systems. Oxygen or other breathing gas should be supplied independently of the chamber gas. An **oxygen elimination system** is advantageous. Rebreathing systems with carbon dioxide scrubbers may be used to reduce the air consumption.

Compressed air supplies and compression facilities should be sufficient to provide for maximum pressurization and adequate ventilation of the chamber (main lock) for two occupants for a period during which transportation will occur.

Control panels for these types of chambers are usually located on the hull structure and include clocks, pressure gauges, communication facilities, flow metres, control valves and ancillary aids. Electrophysiological monitoring connection plugs are also advisable.

MEDICAL SUPPLIES

An emergency kit comprising examination and treatment instruments, drugs and dressings, should be available on site for both small and large recompression chambers. Some instruments and equipment which may be required in the chamber include:

- Stethoscope
- Aneroid sphygmomanometer
- Percussion hammer, pin, tuning forks
- Urinary catheter, introducer, collecting bag
- Sterile syringes and needles
- Sterile intravenous cannulas and catheters
- Intravenous transfusion sets, 'cut-down' and suture sets
- Intravenous fluids, e.g. Hartmann's solution, normal saline, plasma expanders
- Thoracic trocar and cannula with sterile plastic tubing and underwater drain system or
- Heimlich valve
- Endotracheal tubes and connections
- Adhesive tape
- Antiseptic solution and swabs
- Sterile dressings
- Automatic ventilator and connections
- Ophthalmoscope, otoscope, laryngoscope (potential fire ignition sources and not to be taken into the chamber unless needed)
- Drugs which should be immediately available include; frusemide; isoprenaline; lignocaine; aminophylline; phenytoin; diazepam; morphine; pethidine; prochlorperazine; promethazine; atropine; epinephrine (adrenalin); thiopentone; suxamethonium; a non-depolarizing muscle relaxant.

CONCLUSIONS

This chapter has been included to familiarize readers with some of the requirements of recompression chambers so that, if possible, new designs will be improved by the collaboration of doctor and engineer. One-man chambers have not been discussed as these are only used for treatment in the most dire circumstances. If fitted with transfer under pressure facilities, there is a chance of transporting the patient to a better unit; if not, then the patient is committed to whatever treatment is available. Unfortunately, should an emergency arise within the recompression chamber no help is possible directly to the occupant, and it is for this reason that treatment in a one-man chamber should be avoided if possible.

Despite the excellent facilities that are available in most large chamber complexes, the majority of diving accidents are treated under primitive conditions in remote localities. Flexibility and improvisation are necessary and valuable qualities in the diving physician.

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Submarine medicine

ROBYN WALKER

INTRODUCTION

Submarines are the 'silent enemy', operating in often hostile waters. The nature of the environment in which they operate offers a great advantage in warfare, but if disaster befalls the boat this environment can quickly become a tomb. This chapter introduces the reader to this hostile environment, and the medical aspects of submarine escape and rescue.

The first recorded submarine military success belongs to the *Seadiver*, a hand-propelled submarine built by Wilhelm Bauer, a Bavarian artilleryman. In 1850 the Danish fleet blockaded the port of Kiel and the very appearance of the *Seadiver* was enough to scatter the Danes in panic. By 1851 the Danes had regrouped and the *Seadiver* attempted to repeat her success. Unfortunately, she sank to the bottom in 18 metres of water and Bauer and his two crewmen working from first principles devised and carried out the first successful submarine compartment (or rush) escape. This method, albeit with modification and refinement, is still taught today. Although submarines became more sophisticated it needed the success of the German U-boats during the First World War to spark the interest of many nations.

The experience of most nations' involvement with submarines is exemplified by that of Australia. Australian submarine operations date back to the First World War. The AE1 was commissioned in 1913, and was lost with all hands on approximately 14th September 1914 off New Britain. The submarine failed to return from patrol and the cause of its loss remains unknown, no trace of the vessel ever having been found. The AE2 was commissioned in June 1913 and was lost as a result of enemy action in the Sea of Mamora on 30th April 1915. The AE2 was the first allied warship to penetrate the Dardenelles, and saw five days of action in these waters before being sunk by enemy fire, although the entire crew survived. It has been reported that the AE2 has been found off Turkey and that salvage operations are being planned. Australia became an active diesel submarine nation in the 1960s with the purchase of Royal Navy Oberon Class boats, and during the 1990s the RAN commissioned the new Collins Class submarines.

The world's first nuclear powered submarine, the *USS Nautilus* was launched by the United States Navy (USN) in 1954. The development of the nuclear propulsion plant was a result of the combined efforts of Navy, government and contract engineers led by a Captain Hyman G. Rickover. Nuclear power enabled a submarine to operate as a true submersible, capable of staying submerged for a prolonged period.

The USN submarine force of today comprises multi-mission nuclear Attack Class submarines (SSN) and Ballistic Missile submarines (SSBN). The Attack class submarines are designed to seek and destroy enemy submarines and surface ships, to collect intelligence and for Special Forces delivery to anti-ship and strike warfare. The SSBNs are armed with long-range strategic missiles which have no pre-set targets but they can be rapidly targeted using secure and constant at-sea communications links. Their sole role is to provide strategic deterrence, and this class of submarine provides the USA's most survivable and enduring nuclear strike capability.

THE SUBMARINE ENVIRONMENT

Submarine patrols, where the boat remains submerged and undetected, may last for 14 days for conventionally powered and up to 90 days for nuclear-powered boats. Environmental conditions onboard submarines are constantly improving, and maintaining a respirable atmosphere is one of the most important tasks for the crew. Even so, it is said that a submariner could always be detected by their lingering aroma of diesel. Some continue to conserve shower water even when ashore!

The submarine operates at 1 atmosphere pressure. When submerged, the shipboard air may be exchanged with the external environment via the 'snorkel' (an air pipe to the surface). The 'snorting' time required to replenish the boat with clean air free of contaminants is directly proportional to the internal volume of the boat and inversely proportional to the ventilation flow rate. If the rate of air leaving the boat is greater than the flow of fresh gas into the boat, a partial vacuum can be drawn. When snorting, oxygen is replenished and contaminants such as carbon dioxide, carbon monoxide, hydrocarbons, refrigeration gases and bacteriological aerosols are eliminated.

If for operational reasons 'snorting' is not possible, oxygen can be generated by burning oxygen candles, from high-pressure or liquid oxygen stores, or via an oxygen generator which produces oxygen through the electrolysis of water. Carbon dioxide is absorbed by soda lime or lithium hydroxide scrubbers (a non-reversible process and therefore large stores are required), or by passing the submarine air through a regenerative scrubber. Monethanolamine absorbs carbon dioxide when cold and releases it when heated. The liberated carbon dioxide is then dumped overboard. Hydrogen and carbon monoxide are removed through a catalytic oxidative process and aerosols and vapours are separated from the atmosphere by filters and adsorption onto activated charcoal.

Monitoring systems provide a constant read-out of principal atmospheric constituents (e.g. oxygen, carbon dioxide and some fluorocarbons) and activate an alarm if abnormal readings are detected. For substances not measured routinely, other gas detection methods are available, e.g. detection tubes. On nuclear submarines radiation levels are routinely monitored at multiple locations, both inside and outside of engineering spaces. The crew wear personal dosimeters which provides individual exposure information for radiation health surveillance, and portable radiation monitoring equipment is available for the detection of contaminants and for use in the case of accidental exposure or system failure.

SUBSUNK

There have been over 170 recorded peacetime submarine sinkings worldwide since 1900, and no less than 10 between 1985 and 1995. It is said that the most likely scenario for a submarine accident will be at times of transit through ports, channels and fishing grounds, with collision and grounding the most likely mechanism. The basic underlying premise for survival is that once a submarine becomes disabled at least one compartment remains intact, or can be secured long enough for survivors to decide upon

and carry out a course of action. This may involve either escape or rescue.

If a submarine is disabled and sinks, the means by which the crew member is evacuated back to the surface is dependent on a number of factors including:

- the internal pressure of the submarine;
- the internal atmosphere of the submarine;
- the weather conditions; and
- whether or not rescue forces on the surface are available.

There are two methods of escaping from a disabled submarine. One is where the survivors leave the submarine through an escape hatch and make an ascent to the surface. This ascent may be done through the escape tower (SET), where the submariner spends the least time exposed to ambient environmental pressure. The other is from a flooded compartment that is in direct contact with the outside environment. This is known as 'compartment escape', and is less desirable due to the longer period that the individuals are exposed to raised ambient pressure.

The escapers wear a specially designed submarine escape immersion suit (SEIS). A SET escape has been proven to be possible from a depth of at least 180 metres, whilst a compartment escape is only thought to be survivable from a depth of 60 metres.

Rescue involves the use of a submersible to transport the survivors to the surface, where if required subsequent decompression can be undertaken. The depth of the submarine and the operating capability of the rescue craft generally limit rescue. The simplest form of rescue is by salvaging the submarine. Although not applicable in most circumstances, there have been some very successful salvage operations whereby the boat is floated or mechanically towed to the surface – allowing the submariners to evacuate.

The range of medical conditions seen in survivors from a submarine accident will vary depending on whether or not they have escaped or have been rescued.

SUBMARINE ESCAPE

Survivors who have escaped from a disabled submarine are likely to suffer from:

- decompression sickness (DCS);
- barotrauma – pulmonary, ear, sinus, gastric;
- gas toxicities – chlorine, carbon monoxide, carbon dioxide;
- hypothermia;
- near-drowning; and
- traumatic injuries and burns.

Decompression sickness

The internal pressure of the submarine is likely to rise with damage to the pressure hull of the submarine (ingress of seawater, ruptured high-pressure air lines, etc). The elevated partial pressure of nitrogen in the air complicates escape – first as the survivors may experience nitrogen narcosis, and second as their tissues become saturated. It has been shown that a survivor saturated in air can make a direct ascent from 1.7 ATA (equivalent to a depth of 7 metres) to the surface without developing symptoms and signs of DCS. For survivors saturated at deeper depths the risk of life-threatening DCS increases proportionately, and on-site recompression facilities must be available to handle these casualties. First-aid measures include the administration of 100 per cent oxygen and intravenous fluids.

Pulmonary barotrauma

The survivors wear a specially designed SEIS during an escape. This suit provides an air-filled space surrounding the head and supplying in-built buoyancy that assists their passage to the surface. The submariners are taught to breathe in and out normally as they make their ascent; however, panic may override their training or, if the hood ruptures and they find their face in water, the survivor may breath-hold. Pulmonary barotrauma (pulmonary tissue damage, pneumomediastinum, pneumothorax and cerebral arterial gas embolism) can all be expected in survivors as they surface, and urgent recompression may be life-saving.

Other barotraumas

The SET rapidly pressurizes the escaper from whatever the internal pressure of the submarine is to that

of the external environment. For example, if the submarine is resting on the bottom at a depth of 180 metres of sea water (internal atmosphere of 1 ATA), after entering the SET it will take approximately 20 seconds for the escaper to be pressurized to 180 metres of sea-water (msw) and the hatch to open. This rapid pressurization rate may take the escapers by surprise, and middle- and inner-ear barotrauma are likely.

During a live training exercise a British escaper suffered a ruptured stomach during an escape from 150 msw in open water due to the rapid expansion of stomach gas.

Gas toxicities

Chlorine gas may be liberated from the submarine's main batteries if flooding occurs. Delayed bronchospasm and pulmonary oedema may result from intense, brief exposures to chlorine that may also predispose the individual to pulmonary barotrauma during the escape.

If there has been a fire onboard the crew may be exposed to raised levels of carbon monoxide with resulting central nervous system (CNS) depression.

Carbon dioxide toxicity is a major threat, and rising levels may well force the crew to escape before the rescue forces have arrived. Headache and hyperventilation are early warning signs.

Hypothermia and near-drowning

All survivors should be wearing a SEIS which permit survival for a minimum of 6 hours in sub-Arctic water, but this period can exceed 24 hours with water temperatures such as can be expected in temperate climates. Problems with localized frostbite can occur on exposed areas such as the hands and face. Dehydration and seasickness, with time, will contribute to the effects of exposure. In adverse sea conditions it is likely that survivors – particularly those who may be suffering other injuries – may well aspirate seawater.

Traumatic injury

Significant injury occurring in the submarine prior to the escape is likely to prevent the individual being able successfully to enter and operate the SET. Unconsciousness or major limb fracture will preclude escape, but where there is a will to survive individuals can make superhuman efforts, and some with injuries such as lacerations, upper-limb fractures and burns can well be expected on the surface. Survivors are likely to experience acute psychological stress reactions that may make initial assessment difficult, however.

SUBMARINE RESCUE

Once a submarine sinks beyond 180 msw the only option normally available to the crew is to await rescue. Staying alive until the rescue forces arrive – which may take several days – becomes the primary focus of the crew. Regular monitoring of the internal atmosphere, including the pressure, radiation levels, oxygen levels and carbon dioxide levels becomes essential. The medical problems experienced by the survivors will be somewhat different to those experienced by survivors who make a successful escape. Likely medical problems include:

- hypoxia;
- hyperoxia;
- gas toxicities;
- trauma and burns;
- hypothermia;
- DCS; and
- radiation injury.

Pulmonary barotrauma should not complicate rescue as the ascent is controlled. However, the added complications of pulmonary gas toxicity, lack of recompression facilities and the need to treat other injuries sustained in the accident ensure that the management of such a rescue requires the involvement of experienced underwater medicine physicians.

Hypoxia

People will not lose consciousness from hypoxia if breathing an oxygen partial pressure of 0.10 ATA or greater. As oxygen partial pressures fall from 0.16 ATA, progressive symptoms of hypoxia develop,

including increased respiratory rate, clouded thought processes, decreased awareness of surroundings and finally, unconsciousness. The crew should be monitoring the internal atmosphere and burning oxygen candles in order to alleviate this problem. As the internal pressure of the submarine rises the percentage of atmospheric oxygen needed to maintain an adequate partial pressure falls.

Hyperoxia

Oxygen is toxic in high concentrations. Indeed, partial pressures of oxygen above 2 ATA can lead to CNS toxicity with grand mal seizures and breathing oxygen at lower partial pressures, but above 0.6 ATA for prolonged periods will lead to symptoms of pulmonary oxygen toxicity. The rapidity of onset of the relative symptoms increases as the partial pressure of oxygen is increased. Symptoms of pulmonary oxygen toxicity include chest tightness, cough, chest pain, shortness of breath and a fall in vital capacity. The severity of oxygen toxicity is important when selecting a decompression schedule.

Gas toxicities, burns and trauma

Injuries due to trauma, burns, and gas toxicities will be similar to those seen in survivors who have completed an escape.

Hypothermia

At depth, the ocean temperatures may only be 5° C, and without power, the submarine cools to the temperature of the surrounding water. Consequently, survivors may quickly develop hypothermia.

Decompression sickness

Most scenarios which lead to the disabling of a submarine will involve some internal pressure increase in the submarine. This may be a result of flooding, high-pressure air leaks, salvage air and use of emergency breathing systems. If this increase in pressure is maintained for a sufficiently long period, the survivors will have accumulated a decompression obligation. Ideally, rescue vehicles will be capable of transferring under pressure (TUP) – that is, the rescue bell is pressurized to the same pressure as that in the submarine, and the survivors are transferred at that pressure to the surface, where the rescue vehicle is mated to a recompression chamber. The survivors then transfer to the recompression chamber and undergo staged decompression. This process should therefore prevent the occurrence of DCS, but saturation decompression schedules may be required and in the worst case up to 72 hours or so of decompression may be needed. Logistically this is difficult to manage when submarine crew sizes may be as high as 180. As the rescue process may take several days to extract the entire crew it is likely that the pressure will continue to rise in the submarine. This means that the last survivors will have earned a greater decompression obligation than those rescued first.

Radiation injury

If a nuclear submarine suffers a major reactor accident, the survivors may suffer from acute radiation injury. Radiation injury *per se* does not imply that the patient is a health hazard to medical staff, and treatment of life-threatening injuries takes precedence over decontamination procedures. Simple decontamination procedures include removing outer clothing and shoes, washing with soap and tepid water, and irrigating open wounds and covering with a sterile dressing.

SUBMARINE RESCUE VEHICLES

Rescue vehicles have been constructed by many nations including the United States, United Kingdom, Sweden, Australia and Italy. However, many of these are coming to the end of their working lives and replacements are being sought. The United States Deep Submergence Rescue Vehicle (DSRV) is aircraft-transportable but requires a mother submarine (MOSUB) to transport it to the site of a disabled submarine. The DSRV can mate with specially modified MOSUBS and can transfer survivors under

pressure to the bow compartment pressurized up to 2 ATA. The distinct disadvantage of this system is the time it takes to transport the vehicle to the site, and the likelihood of significant DCS if the survivors are saturated at pressures greater than 2 ATA.

The British LR5 is not air-transportable, and this therefore limits its range of operations to the UK area. It again is capable of transferring survivors to a MOSUB at 2 ATA, but currently has no TUP ability with the surface.

The Australian Submarine Escape & Rescue Service (SERS) is the newest system and comprises:

- surface recompression facilities for a maximum of 72 people;
- a rescue submersible (*Remora*) capable of operating in waters down to the crush depth of Australian submarines;
- a transfer under pressure facility (up to 5 ATA) with the surface recompression facility; and
- an extension of life support capability (a means of resupplying the stricken submarine with oxygen candles, soda lime absorbent, food, water, medical supplies, etc.) to provide time for the rescue service to be transported to the site.

The SERS has been exercised with TUP of crew from a Collins class submarine, and there is also a capability to transfer injured personnel from the submarine to the *Remora* and then to the recompression chamber facility using a harness/pulley system.

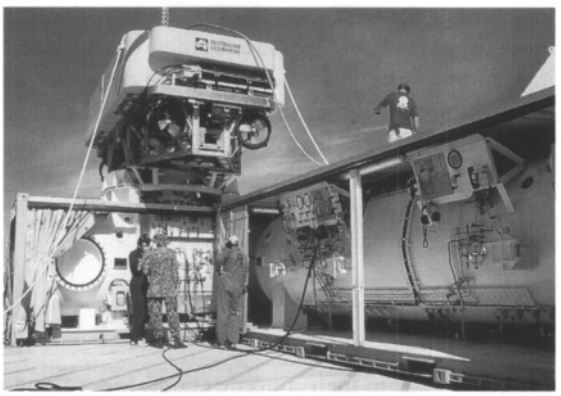

Figure 70.1 *The Royal Australian Navy submarine rescue vehicle* Remora *mating to a deck decompression chamber complex. (Photograph courtesy of the Royal Australian Navy Photographic Unit.)*

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Appendices

Caution: The reader is advised to consult the original source of any table used. The authors have no way of transferring any changes to the reader or of correcting any errors discovered after publication. Also, some tables have been condensed and information omitted that may be important to a user.

APPENDIX A1: BRITISH SUB-AQUA CLUB (BSAC) DECOMPRESSION TABLES

The complete version of the 1988 BSAC tables consists of seven tables for diving in the altitiude range from sea level to 250 metres. The first three are printed here with the permission of the BSAC and Dr Tom Hennessy who developed them. It should be noted that that the tables are a copyright document. The information presented here is to assist the reader in assessing if a diver has followed the tables. The complete tables are available in a waterproof booklet from the BSAC (16 Upper Woburn Place, London).

For his first dive, the diver uses Table A; the table is entered on the left-hand side with the depth, the deepest depth reached during the dive. The diver then looks across to find the dive time. This is the time from leaving the surface to reaching 6 metres on the return to the surface (or reaching 9 metres if a 9-metre decompression stop is required). The time used should be the next longer tabulated if the exact time of the dive is not listed.

If the dive is to the left of the line that seperates No-stop from Decompression stops the diver can surface at a rate of 15 metres/minute to 6 metres and 6 metres/minute for the last 6 metres.

If the dive is on the decompression side of the table then the decompression stop(s) listed below the time are to be taken. The diver may then ascend to the surface at the stipulated rate.

At the foot of each table is a series of letters in a row entitled surfacing code. This is an estimate of the nitrogen load at the end of the dive. To allow for any nitrogen remaining from previous dives the diver uses the Surface Interval Table (page 674) and enters on the line that starts with his surfacing code. For example if his surfacing code was F he goes to line F. The surface interval till the next dive is found and another letter found called the current tissue code. For example, a diver who surfaced in group F and then spends between 90 minutes and 4 hours on the surface is in group C. This means he should use Table C for the next dive.

For example, a diver wishes to make two dives to 18 metres with a surface interval of 2 hours. How long can each dive

be without having to make decompression stops? What decompression stops are needed if the dive time for the second dive is to be 50 minutes?

Table A is used for the first dive. It will be found that the longest time allowed before decompression is required is 51 minutes. It should be noted that the diver has to be back at 6 metres by that time. To get there it would be necessary to leave the bottom 50 minutes after leaving the surface. On returning to the surface the diver is in surface code F. In the surface interval table for a diver that surfaces in group F it will be found that the diver is in group C after 2 hours.

Table C is used for the second dive. It will be found that the second dive can be for no longer than 15 minutes if decompression stops are to be avoided. If the dive time for the second dive is to be 50 minutes the decompression required is 21 minutes at 6 metres.

The complete BSAC tables also contain altitude tables and rules for flying after diving.

The USN tables cover depths and times beyond the limits of sensible scuba diving. They are presented because there may be a call to use them for longer dives, for example when a diver is trapped underwater and a relay of tanks keeps them alive until they can be freed.

For many divers the No-decompression limits table (Table 2) is all they need to consult. They enter the table knowing the depth they intend to dive to (rounded up to the next depth listed), and can read off the maximum allowed duration of the intended dive. The time (bottom time) is measured from leaving the surface until leaving the bottom. The tables assumes that the diver will descend at a rate of 60 ft per minute or slower.

In this table the first column is the depth, and the second table is the maximum bottom time allowed before the diver needs to pause at nominated depths during ascent. On this table a diver diving to 85 ft uses the 90 ft table and has a maximum bottom time of 30 minutes. At the end of this time the table makers expect him to ascend at a rate of 30 ft per minute to the surface. He is not allowed to prolong his ascent. The other entries in this table are used in the process used to modify the decompression of a second (and later) dive(s) to allow for the nitrogen absorbed during previous dive(s).

If he needs to spend more time at a depth than allowed for in this table, the diver will need to stop at nominated depths during the ascent. In this case the standard air decompression tables are used. If the diver in the last paragraph wanted to dive to 90 ft for 60 minutes he would take 2 minutes 40 seconds to ascend to 10 feet, stop there for 25 minutes then ascend to the surface, taking another 20 seconds. The total ascent takes 28 minutes, and the diver is in repetitive dive group M.

For decompressing from a second and subsequent dives a procedure called a 'repetitive dive calculation' must be undertaken. The letters are used as an indication of the amount of nitrogen in the diver's body. In the tables, these are listed across the page for the No decompression dives. Examination of this table shows that the diver moves through the alphabet as he spends more time at a depth, and at deeper depths he moves through the alphabet more quickly. Thus our diver in the first example spending 30 minutes at 90 ft was a group H diver, If he had ascended after 10 minutes he would have been a group C diver.

To use the letters, the first step is to allow for the nitrogen that is lost from the body during the time the diver spends on the surface. The residual nitrogen is used for this purpose. It is entered at the appropriate letter on the diagonal slope on the top left of the table. The table is entered on the row with the letter related to the last dive. The next step is to move along the row until the intended time to be spent on the surface is reached. This time is called the 'surface interval'. In this table the figures are in hours and minutes. From the intended surface interval time the user reads down the table and can select a new repetitive group. These move back toward the front of the alphabet as more time is spent on the surface.

Below the new letters in this table are figures called the 'residual nitrogen times'. They are listed against the depth of the next dive.

For example, a diver finishes the first dive as a group H diver, waits on the surface for 3 hours, and becomes a group D diver. If the next dive is to 60 feet, a group D diver has a residual nitrogen time of 24 minutes. This can be used in two ways. If it is intended that the second dive should not require decompression stops, then the maximum permitted time for a 60-foot dive of 60 minutes needs to be reduced by 24 minutes to allow for the residual nitrogen. So the maximum bottom time is 36 minutes. If the next dive is for 40 minutes, the residual nitrogen time is added and the second dive is considered to be 40 + 24 minutes = 64 minutes, and this time can be used in the standard air decompression tables to find that the diver needs to stop at 10 feet for 2 minutes and is a group K diver for the start of the next surface interval if there is to be a third dive.

Table 1 *US Navy standard air decompression*

Depth (feet)	Bottom time (min)	Time to first stop (min:s)	Decompression stops (feet)					Total ascent (min:s)	Repetitive group
			50	40	30	20	10		
40	200	-	-	-	-	-	0	1:20	(*)
	210	1:00	-	-	-	-	2	3:20	N
	230	1:00	-	-	-	-	7	8:20	N
	250	1:00	-	-	-	-	11	12:20	O
	270	1:00	-	-	-	-	15	16:20	O
	300	1:00	-	-	-	-	19	20:20	Z
50	100	-	-	-	-	-	0	1:40	(*)
	110	1:20	-	-	-	-	3	4:40	L
	120	1:20	-	-	-	-	5	6:40	M
	140	1:20	-	-	-	-	10	11:40	M
	160	1:20	-	-	-	-	21	22:40	N
	180	1:20	-	-	-	-	29	30:40	O
	200	1:20	-	-	-	-	35	36:40	O
	220	1:20	-	-	-	-	40	41:40	Z
	240	1:20	-	-	-	-	47	48:40	Z
60	60	-	-	-	-	-	0	2:00	(*)
	70	1:40	-	-	-	-	2	4:00	K
	80	1:40	-	-	-	-	7	9:00	L
	100	1:40	-	-	-	-	14	16:00	M
	120	1:40	-	-	-	-	26	28:00	N
	140	1:40	-	-	-	-	39	41:00	O
	160	1:40	-	-	-	-	48	50:00	Z
	180	1:40	-	-	-	-	56	58:00	Z
	200	1:20	-	-	-	1	69	72:00	Z
70	50	-	-	-	-	-	0	2:20	(*)
	60	2:00	-	-	-	-	8	10:20	K
	70	2:00	-	-	-	-	14	16:20	L
	80	2:00	-	-	-	-	18	20:20	M
	90	2:00	-	-	-	-	23	25:20	N
	100	2:00	-	-	-	-	33	35:20	N
	110	1:40	-	-	-	2	41	45:20	O
	120	1:40	-	-	-	4	47	53:20	O
	130	1:40	-	-	-	6	52	60:20	O
	140	1:40	-	-	-	8	56	66:20	Z
	150	1:40	-	-	-	9	61	72:20	Z
	160	1:40	-	-	-	13	72	87:20	Z
	170	1:40	-	-	-	19	79	100:20	Z
80	40	-	-	-	-	-	0	2:40	(*)
	50	2:20	-	-	-	-	10	12:40	K
	60	2:20	-	-	-	-	17	19:40	L
	70	2:20	-	-	-	-	23	25:40	M
	80	2:00	-	-	-	2	31	35:40	N
	90	2:00	-	-	-	7	39	48:40	N
	100	2:00	-	-	-	11	46	59:40	O

Table 1 *continued*

Depth (feet)	Bottom time (min)	Time to first stop (min:s)	Decompression stops (feet) 50	40	30	20	10	Total ascent (min:s)	Repetitive group
	110	2:00	-	-	-	13	53	68:40	O
	120	2:00	-	-	-	17	56	75:40	Z
	130	2:00	-	-	-	19	63	84:40	Z
	140	2:00	-	-	-	26	69	97:40	Z
	150	2:00	-	-	-	32	77	111:40	Z
90	30	-	-	-	-		0	3:00	(*)
	40	2:40	-	-	-	-	7	10:00	J
	50	2:40	-	-	-	-	18	21:00	L
	60	2:40	-	-	-	-	25	28:00	M
	70	2:20	-	-	-	7	30	40:00	N
	80	2:20	-	-	-	13	40	56:00	N
	90	2:20	-	-	-	18	48	69:00	O
	100	2:20	-	-	-	21	54	78:00	Z
	110	2:20	-	-	-	24	61	88:00	Z
	120	2:20	-	-	-	32	68	103:00	Z
	130	2:00	-	-	5	36	74	118:00	Z
100	25	-	-	-	-	-	0	3:20	(*)
	30	3:00	-	-	-	-	3	6:20	I
	40	3:00	-	-	-	-	15	18:20	K
	50	2:40	-	-	-	2	24	29:20	L
	60	2:40	-	-	-	9	28	40:20	N
	70	2:40	-	-	-	17	39	59:20	O
	80	2:40	-	-	-	23	48	74:20	O
	90	2:20	-	-	3	23	57	86:20	Z
	100	2:20	-	-	7	23	66	99:20	Z
	110	2:20	-	-	10	34	72	119:20	Z
	120	2:20	-	-	12	41	78	134:20	Z
110	20	-	-	-	-	-	0	3:40	(*)
	25	3:20	-	-	-	-	3	6:40	H
	30	3:20	-	-	-	-	7	10:40	J
	40	3:00	-	-	-	2	21	26:40	L
	50	3:00	-	-	-	8	26	37:40	M
	60	3:00	-	-	-	18	36	57:40	N
	70	2:40	-	-	1	23	48	75:40	O
	80	2:40	-	-	7	23	57	90:40	Z
	90	2:40	-	-	12	30	64	109:40	Z
	100	2:40	-	-	15	37	72	127:40	Z
120	15	-	-	-	-	-	0	4:00	(*)
	20	3:40	-	-	-	-	2	6:00	H
	25	3:40	-	-	-	-	6	10:00	I
	30	3:40	-	-	-	-	14	18:00	J
	40	3:20	-	-	-	5	25	34:00	L
	50	3:20	-	-	-	15	31	50:00	N
	60	3:00	-	-	2	22	45	73:00	O

Table 1 *continued*

Depth (feet)	Bottom time (min)	Time to first stop (min:s)	Decompression stops (feet)					Total ascent (min:s)	Repetitive group
			50	40	30	20	10		
	70	3:00	-	-	9	23	55	91:00	O
	80	3:00	-	-	15	27	63	109:00	Z
	90	3:00	-	-	19	37	74	134:00	Z
	100	3:00	-	-	23	45	80	152:00	Z
130	10	-	-	-	-	-	0	4:20	(*)
	15	4:00	-	-	-	-	1	5:20	F
	20	4:00	-	-	-	-	4	8:20	H
	25	4:00	-	-	-	-	10	14:20	J
	30	3:40	-	-	-	3	18	25:20	M
	40	3:40	-	-	-	10	25	39:20	N
	50	3:20	-	-	3	21	37	65:20	O
	60	3:20	-	-	9	23	52	88:20	Z
	70	3:20	-	-	16	24	61	105:20	Z
	80	3:00	-	3	19	35	72	133:20	Z
	90	3:00	-	8	19	45	80	156:20	Z
140	10	-	-	-	-	-	0	4:40	(*)
	15	4:20	-	-	-	-	2	6:40	G
	20	4:20	-	-	-	-	6	10:40	I
	25	4:00	-	-	-	2	14	20:40	J
	30	4:00	-	-	-	5	21	30:40	K
	40	3:40	-	-	2	16	26	48:40	N
	50	3:40	-	-	6	24	44	78:40	O
	60	3:40	-	-	16	23	56	99:40	Z
	70	3:20	-	4	19	32	68	127:40	Z
	80	3:20	-	10	23	41	79	157:40	Z
150	5	-	-	-	-	-	0	5:00	C
	10	4:40	-	-	-	-	1	6:00	E
	15	4:40	-	-	-	-	3	8:00	G
	20	4:20	-	-	-	2	7	14:00	H
	25	4:20	-	-	-	4	17	26:00	K
	30	4:20	-	-		8	24	37:00	L
	40	4:00	-	-	5	19	33	62:00	N
	50	4:00	-	-	12	23	51	91:00	O
	60	3:40	-	3	19	26	62	115:00	Z
	70	3:40	-	11	19	39	75	149:00	Z
	80	3:20	1	17	19	50	84	176:00	Z
160	5	-	-	-	-	-	0	5:20	D
	10	5:00	-	-	-	-	1	6:20	F
	15	4:40	-	-	-	1	4	10:20	H
	20	4:40	-	-	-	3	11	19:20	J
	25	4:40	-	-	-	7	20	32:20	K
	30	4:20	-	-	2	11	25	43:20	M
	40	4:20	-	-	7	23	39	74:20	N
	50	4:00	-	2	16	23	55	101:20	Z
	60	4:00	-	9	19	33	69	135:20	Z

Table 1 *continued*

Depth (feet)	Bottom time (min)	Time to first stop (min:s)	Decompression stops (feet)					Total ascent (min:s)	Repetitive group
			50	40	30	20	10		
170	5	-	-	-	-	-	0	5:40	D
	10	5:20	-	-	-	-	2	7:40	F
	15	5:00	-	-	-	2	5	12:40	H
	20	5:00	-	-	-	4	15	24:40	J
	25	4:40	-	-	2	7	23	37:40	L
	30	4:40	-	-	4	13	26	48:40	M
	40	4:20	-	1	10	23	45	84:40	O
	50	4:20	-	5	18	23	61	112:40	Z
	60	4:00	2	15	22	37	74	155:40	Z
180	5	-	-	-	-	-	0	6:00	D
	10	5:40	-	-	-	-	3	9:00	F
	15	5:20	-	-	-	3	6	15:00	I
	20	5:00	-	-	1	5	17	29:00	K
	25	5:00	-	-	3	10	24	43:00	L
	30	5:00	-	-	6	17	27	56:00	N
	40	4:40	-	3	14	23	50	96:00	O
	50	4:20	2	9	19	30	65	131:00	Z
	60	4:20	5	16	19	44	81	171:00	Z
190	5	-	-	-	-	-	0	6:20	D
	10	5:40	-	-	-	1	3	10:20	G
	15	5:40	-	-	-	4	7	17:20	I
	20	5:20	-	-	2	6	20	34:20	K
	25	5:20	-	-	5	11	25	47:20	M
	30	5:00	-	1	8	19	32	66:20	N
	40	5:00	-	8	14	23	55	106:20	O

* See table 2 for repetitive groups in no-decompression dives.

Table 2 *No-decompression limits and repetitive group designation table for no-decompression air dives*

Depth (feet)	No-decompression limits (min)	Repetitive groups (air dives)														
		A	B	C	D	E	F	G	H	I	J	K	L	M	N	O
10	-	60	120	210	300	-	-	-	-	-	-	-	-	-	-	-
15	-	35	70	110	160	225	350	-	-	-	-	-	-	-	-	-
20	-	25	50	75	100	135	180	240	325	-	-	-	-	-	-	-
25	-	20	35	55	75	100	125	160	195	245	315	-	-	-	-	-
30	-	15	30	45	60	75	95	120	145	170	205	250	310	-	-	-
35	310	5	15	25	40	50	60	80	100	120	140	160	190	220	270	310
40	200	5	15	25	30	40	50	70	80	100	110	130	150	170	200	-
50	100	-	10	15	25	30	40	50	60	70	80	90	100	-	-	-
60	60	-	10	15	20	25	30	40	50	55	60	-	-	-	-	-
70	50	-	5	10	15	20	30	35	40	45	50	-	-	-	-	-
80	40	-	5	10	15	20	25	30	35	40	-	-	-	-	-	-
90	30	-	5	10	12	15	20	25	30	-	-	-	-	-	-	-
100	25	-	5	7	10	15	20	22	25	-	-	-	-	-	-	-
110	20	-	-	5	10	13	15	20	-	-	-	-	-	-	-	-
120	15	-	-	5	10	12	15	-	-	-	-	-	-	-	-	-
130	10	-	-	5	8	10	-	-	-	-	-	-	-	-	-	-
140	10	-	-	5	7	10	-	-	-	-	-	-	-	-	-	-
150	5	-	-	5	-	-	-	-	-	-	-	-	-	-	-	-
160	5	-	-	-	5	-	-	-	-	-	-	-	-	-	-	-
170	5	-	-	-	5	-	-	-	-	-	-	-	-	-	-	-
180	5	-	-	-	5	-	-	-	-	-	-	-	-	-	-	-
190	5	-	-	-	5	-	-	-	-	-	-	-	-	-	-	-

Instructions for use (Table 2)

No-decompression limits

This column shows at various depths greater than 30 feet the allowable diving times (in minutes) which permit surfacing directly at 60 feet/min with no decompression stops. Longer exposure times require the use of the Standard Air Decompression Table.

Repetitive group designation table

The tabulated exposure times (or bottom times) are in minutes. The times at the various depths in each vertical column are the maximum exposures during which a diver will remain within the group listed at the head of the column.

To find the repetitive group designation at surfacing for dives involving exposures up to and including the no-decompression limits: enter the table on the *exact* or *next greater depth* than that to which exposed and select the listed exposure time *exact* or *next greater* than the actual exposure time. The repetitive group designation is indicated by the letter at the head of the vertical column where the selected exposure time is listed.

Example: a dive was to 32 feet for 45 minutes. Enter the table along the 35-foot-depth line since it is next greater than 32 feet. The table shows that since group D is left after 40 minutes' exposure and group E after 50 minutes, group E (at the head of the column where the 50-minute exposure is listed) is the proper selection.

Exposure times for depths less than 40 feet are listed only up to approximately 5 hours since this is considered to be beyond field requirements for this table.

Table 3 *Surface interval credit table for air decompression dives [repetitive group at the end of the surface interval (air dive)]*

New group designation

Repetitive group at the beginning of the surface interval from previous dive	Z	O	N	M	L	K	J	I	H	G	F	E	D	C	B	A
Z	0:10 0:22	0:23 0:34	0:35 0:48	0:49 1:02	1:03 1:18	1:19 1:36	1:37 1:55	1:56 2:17	2:18 2:42	2:43 3:10	3:11 3:45	3:46 4:29	4:30 5:27	5:28 6:56	6:57 10:05	10:06 12:00
O		0:10 0:23	0:24 0:36	0:37 0:51	0:52 1:07	1:08 1:24	1:25 1:43	1:44 2:04	2:05 2:29	2:30 2:59	3:00 3:33	3:34 4:17	4:18 5:16	5:17 6:44	6:45 9:54	9:55 12:00
N			0:10 0:24	0:25 0:39	0:40 0:54	0:55 1:11	1:12 1:30	1:31 1:53	1:54 2:18	2:19 2:47	2:48 3:22	3:23 4:04	4:05 5:03	5:04 6:32	6:33 9:43	9:44 12:00
M				0:10 0:25	0:26 0:42	0:43 0:59	1:00 1:18	1:19 1:39	1:40 2:05	2:06 2:34	2:35 3:08	3:09 3:52	3:53 4:49	4:50 6:18	6:19 9:28	9:29 12:00
L					0:10 0:26	0:27 0:45	0:46 1:04	1:05 1:25	1:26 1:49	1:50 2:19	2:20 2:53	2:54 3:36	3:37 4:35	4:36 6:02	6:03 9:12	9:13 12:00
K						0:10 0:28	0:29 0:49	0:50 1:11	1:12 1:35	1:36 2:03	2:04 2:38	2:39 3:21	3:22 4:19	4:20 5:48	5:49 8:58	8:59 12:00
J							0:10 0:31	0:32 0:54	0:55 1:19	1:20 1:47	1:48 2:20	2:21 3:04	3:05 4:02	4:03 5:40	5:41 8:40	8:41 12:00
I								0:10 0:33	0:34 0:59	1:00 1:29	1:30 2:02	2:03 2:44	2:45 3:43	3:44 5:12	5:13 8:21	8:22 12:00
H									0:10 0:36	0:37 1:06	1:07 1:41	1:42 2:23	2:24 3:20	3:21 4:49	4:50 7:59	8:00 12:00
G										0:10 0:40	0:41 1:15	1:16 1:59	2:00 2:58	2:59 4:25	4:26 7:35	7:36 12:00
F											0:10 0:45	0:46 1:29	1:30 2:28	2:29 3:57	3:58 7:05	7:06 12:00
E												0:10 0:54	0:55 1:57	1:58 3:22	3:23 6:32	6:33 12:00
D													0:10 1:09	1:10 2:38	2:39 5:48	5:49 12:00
C														0:10 1:39	1:40 2:49	2:50 12:00
B															0:10 2:10	2:11 12:00
A																0:10 12:00

ENTER HERE

Instructions for use (Table 3)

Surface interval time in the table is in *hours* and *minutes* (7:59 means 7 hours and 59 minutes.) The surface interval must be at least 10 minutes.

Find the *repetitive group designation letter* (from the previous dive schedule) on the diagonal slope. Enter the table horizontally to select the surface interval time that is exactly between the actual surface interval times shown. The repetitive group designation for the *end* of the surface interval is at the head of the vertical column where the selected surface interval time is listed. For example, a previous

dive was to 110 feet for 30 minutes. The diver remains on the surface 1 hour and 30 minutes and wishes to find the new repetitive group designation. The repetitive group from the last column of the 110/30 schedule in the Standard Air Decompression Tables is 'J'. Enter the surface interval credit table along the horizontal line labelled 'J'. The 1-hour-30-minute surface interval lies between the times 1:20 and 1:47. Therefore, the diver has lost sufficient inert gas to place him in group 'G' (at the head of the vertical column selected).

Note: dives following surface intervals of more than 12 hours are not considered repetitive dives. Actual bottom times in the Standard Air Decompression Tables may be used in computing decompression for such dives.

Table 4 *Repetitive dive timetable for air dives*

Residual nitrogen times (minutes)

Repetitive groups	Repetitive dive depth (ft) (air dives)															
	40	50	60	70	80	90	100	110	120	130	140	150	160	170	180	190
A	7	6	5	4	4	3	3	3	3	3	2	2	2	2	2	2
B	17	13	11	9	8	7	7	6	6	6	5	5	4	4	4	4
C	25	21	17	15	13	11	10	10	9	8	7	7	6	6	6	6
D	37	29	24	20	18	16	14	13	12	11	10	9	8	8	8	8
E	49	38	30	26	23	20	18	16	15	13	12	12	11	10	10	10
F	61	47	36	31	28	24	22	20	18	16	15	14	13	13	12	11
G	73	56	44	37	32	29	26	24	21	19	18	17	16	15	14	13
H	87	66	52	43	38	33	30	27	25	22	20	19	18	17	16	15
I	101	76	61	50	43	38	34	31	28	25	23	22	20	19	18	17
J	116	87	70	57	48	43	38	34	32	28	26	24	23	22	20	19
K	138	99	79	64	54	47	43	38	35	31	29	27	26	24	22	21
L	161	111	88	72	61	53	48	42	39	35	32	30	28	26	25	24
M	187	124	97	80	68	58	52	47	43	38	35	32	31	29	27	26
N	213	142	107	87	73	64	57	51	46	40	38	35	33	31	29	28
O	241	160	117	96	80	70	62	55	50	44	40	38	36	34	31	30
Z	257	169	122	100	84	73	64	57	52	46	42	40	37	35	32	31

Instructions for use (Table 4)

The bottom times listed in this table are called 'residual nitrogen times' and are the times a diver is to consider he has *already* spent on the bottom when he *starts* a repetitive dive to a specific depth. They are in minutes.

Enter the table horizontally with the repetitive group designation from the Surface Interval Credit Table. The time in each vertical column is the number of minutes that would be required (at the depth listed at the head of the column) to saturate to the particular group.

Example: the final group designation from the Surface Interval Credit Table, on the basis of a previous dive and surface interval, is 'H'. To plan a dive to 110 feet, determine the residual nitrogen time for this depth required by the repetitive group designation: enter this table along the horizontal line labelled 'H'. The table shows that one must *start* a dive to 110 feet as though he had already been on the bottom for 27 minutes. This information can then be applied to the Standard Air Decompression Table or No-decompression Table in a number of ways:

1 Assuming a diver is going to finish a job and take whatever decompression is required, he must add 27 minutes to his actual bottom time and be prepared to take decompression according to the 110-foot schedules for the sum or equivalent single dive time.
2 Assuming one wishes to make a quick inspection dive for the minimum decompression, he will decompress according to the 110/30 schedule for a dive of 3 minutes or less (27 + 3 = 30). For a dive of over 3 minutes but less than 13, he will decompress according to the 110/40 schedule (27 + 13 = 40).
3 Assuming that one does not want to exceed the 110/50 schedule and the amount of decompression it requires,

he will have to start ascent before 23 minutes of actual bottom time (50 − 27 = 23).

4 Assuming that a diver has air for approximately 45 minutes bottom time and decompression stops, the possible dives can be computed: a dive of 13 minutes will require 23 minutes of decompression (110/40 schedule), for a total submerged time of 36 minutes. A dive of 13 to 23 minutes will require 34 minutes of decompression (110/50 schedule), for a total submerged time of 47 to 57 minutes. Therefore, to be safe, the diver will have to start ascent before 13 minutes or a standby air source will have to be provided.

APPENDIX A3: NOAA MODIFIED AIR TABLES

NOAA NO-DECOMPRESSION AIR TABLE

WARNING:
EVEN STRICT COMPLIANCE WITH THESE CHARTS WILL NOT GUARANTEE AVOIDANCE OF DECOMPRESSION SICKNESS. CONSERVATIVE USAGE IS STRONGLY RECOMMENDED

CHART 1 - DIVE TIMES WITH END-OF-DIVE GROUP LETTER

ESDT RESIDUAL NITROGEN TIME
+ABT ACTUAL BOTTOM TIME
RNT EQUIVALENT SINGLE DIVE TIME

(USE ESDT TO DETERMINE END-OF-DIVE LETTER GROUP)

DEPTH MSW	DEPTH FEET	(00) MAXIMUM NO-STOP TIME													
12	40	5	15	25	30	40	50	70	80	100	110	130	150	170	(170)
15	50		10	15	25	30	40	50	60	70	80	90	(100)	120 5	160 21
18	60		10	15	20	25	30	40	50	55	(60)	70 2	80 7	100 14	
22	70		5	10	15	20	30	35	40	45	(50)	60 8	70 14	80 18	
25	80		5	10	15	20	25	30	35	(40)		50 10	60 17	70 23	
28	90		5	10	12	15	20	25	(30)		40 7		50 18	60 25	
31	100		5	7	10	15	20	22	(25)	30 3		40 15			
34	110			5	10	13	15	(20)	25 3		30 17				
37	120			5	10	12	(15)		20 2	25 6	30 14				
40	130			5	8	(10)	15 1		20 4		25 10				

msw	fsw	GROUP LETTER
12	40	A
15	50	B
18	60	C
22	70	D
25	80	E
28	90	F
31	100	G
34	110	H
37	120	I
40	130	J
		K
		L
		M
		N

A	B	C	D	E	F	G	H	I	J	K	L	M	N
12:00 0:10	12:00 3:21	12:00 4:50	12:00 5:49	12:00 6:35	12:00 7:06	12:00 7:36	12:00 8:00	12:00 8:22	12:00 8:51	12:00 8:59	12:00 9:13	12:00 9:29	12:00 9:44
	3:20 0:10	4:49 1:40	5:48 2:39	6:34 3:25	7:05 3:58	7:35 4:26	7:59 4:50	8:21 5:13	8:50 5:41	8:58 5:49	9:12 6:03	9:28 6:19	9:43 6:33
		1:39 0:10	2:38 1:10	3:24 1:58	3:57 2:29	4:25 2:59	4:49 3:21	5:12 3:44	5:40 4:03	5:48 4:20	6:02 4:36	6:18 4:50	6:32 5:04
			1:09 0:10	1:57 0:55	2:28 1:30	2:58 2:00	3:20 2:24	3:43 2:45	4:02 3:05	4:19 3:22	4:35 3:37	4:49 3:53	5:03 4:05
				0:54 0:10	1:29 0:46	1:59 1:16	2:23 1:42	2:44 2:03	3:04 2:21	3:21 2:39	3:36 2:54	3:52 3:09	4:04 3:23
					0:45 0:10	1:15 0:41	1:41 1:07	2:02 1:30	2:20 1:48	2:38 2:04	2:53 2:20	3:08 2:35	3:22 2:48
						0:40 0:10	1:06 0:37	1:29 1:00	1:47 1:20	2:03 1:36	2:19 1:50	2:34 2:06	2:47 2:19
							0:36 0:10	0:59 0:34	1:19 0:55	1:35 1:12	1:49 1:26	2:05 1:36	2:18 1:54
								0:33 0:10	0:54 0:32	1:11 0:50	1:25 1:05	1:35 1:19	1:53 1:31
									0:31 0:10	0:49 0:29	1:04 0:46	1:18 1:00	1:30 1:12
										0:28 0:10	0:45 0:27	0:59 0:43	1:11 0:55
											0:26 0:10	0:42 0:26	0:54 0:40
												0:25 0:10	0:39 0:25
													0:24 0:10

CHART 3 - REPETITIVE DIVE TIME

BLACK NUMBERS (TOP) ARE RESIDUAL NITROGEN TIMES (RNT)
GREY NUMBERS (BOTTOM) ARE ADJUSTED NON-STOP REPETITIVE DIVE TIMES
ACTUAL DIVE TIME SHOULD NOT EXCEED THIS NUMBER

msw	12	15	18	22	25	28	31	34	37	40
fsw	40	50	60	70	80	90	100	110	120	130
A	7 193	6 94	5 55	4 46	4 36	3 27	3 22	3 17	13 12	3 7
B	17 183	13 87	11 49	9 41	8 32	7 23	7 18	6 14	6 9	6 4
C	25 175	21 79	17 43	15 35	13 27	11 19	10 15	10 10	9 6	8 2
D	37 163	29 71	24 36	20 30	18 22	16 14	14 11	13 7	12 3	
E	49 151	38 62	30 30	26 24	23 17	20 10	18 7	16 4		
F	61 139	47 53	36 24	31 19	28 12	24 6	22 3			
G	73 127	56 44	44 16	37 13	32 8	29 1				
H	87 113	66 34	52 8	43 7	38 2					
I	101 99	76 24								
J	116 84	87 13								
K	138 62	99 1								
L	161 39									
M	187 13									
N										

CHART 2 - SURFACE INTERVAL TIME

The NOAA version of the US Navy table is an example of how they can be condensed to a form that allows their use by a diver who is uncertain of the likely depth or duration when he enters the water.

APPENDIX A4: THE DCIEM SPORT DIVING TABLES

DCIEM sport diving tables

A: *AIR DECOMPRESSION*

Depth	No-decompression bottom times (minutes)				Decompression required bottom times			
20' 6 m	30 A 60 B 90 C 120 D	150 E 180 F 240 G 300 H	360 I 420 J 480 K 600 L	720 M ∞				
30' 9 m	30 A 45 B 60 C 90 D	100 E 120 F 150 G 180 H	190 I 210 J 240 K 270 L	300 M	360	400		
40' 12 m	22 A 30 B 40 C	60 D 70 E 80 F	90 G 120 H 130 I	150 J	160 K 170 L	180 M 190	200	215
50' 15 m	18 A 25 B	30 C 40 D	50 E 60 F	75 G	85 H 95 I	105 J 115 K	124 L	132 M
60' 18 m	14 A 20 B	25 C 30 D	40 E	50 F	60 G	70 H 80 I	85 J	92 K
Decompression stops in minutes			**at 10' 3 m**		**5**	**10**	**15**	**20**
70' 21 m	12 A 15 B	20 C	25 D	35 E	40 F	50 G	60 H 63 I	66 J
80' 24 m	10 A 13 B	15 C	20 D	25 E	29 F	35 G	48 H	52 I
90' 27 m	9 A	12 B	15 C	20 D	23 E	27 F	35 G	40 H 43 I
100' 30 m	7 A	10 B	12 C	15 D	18 D	21 E	25 F 29 G	36 H
110' 33 m		6 A	10 B	12 C	15 D	18 E	22 F	26 G 30 H
120' 36 m		6 A	8 B	10 C	12 D	15 E	19 F	25 G
130' 39 m			5 A	8 B	10 C	13 D	16 F	21 G
140' 42 m			5 A	7 B	9 C	11 D	14 F	18 G
150' 45 m			4 A	6 B	8 C	10 D	12 E	15 F
Decompression stops in minutes			**at 20' 6 m**		-	-	**5**	**10**
			AT 10' 3 m		**5**	**10**	**10**	**10**

- ASCENT RATE is 60' (18 m) plus or minus 10' (3 m) per minute
  NO-DECOMPRESSION LIMITS are given for first dives
- DECOMPRESSION STOPS are taken at mid-chest level for the times indicated at the specified stop depths

→ Table B for Minimum Surface Intervals and Repetitive Factors
→ Table C for Repetitive Dive No-Decompression Limits
→ Table D for Depth Corrections required at Altitudes above 1000' (300 m)

B: *SURFACE INTERVALS*

Rep. group	0:15 0:29	0:30 0:59	1:00 1:29	1:30 1:59	2:00 2:59	3:00 3:59	4:00 5:59	6:00 8:59	9:00 11:59	12:00 14:59	15:00 18:00
A	1.4	1.2	1.1	1.1	1.1	1.1	1.1	1.1	1.0	1.0	1.0
B	1.5	1.3	1.2	1.2	1.2	1.1	1.1	1.1	1.1	1.0	1.0
C	1.6	1.4	1.3	1.2	1.2	1.2	1.1	1.1	1.1	1.0	1.0
D	1.8	1.5	1.4	1.3	1.3	1.2	1.2	1.1	1.1	1.0	1.0
E	1.9	1.6	1.5	1.4	1.3	1.3	1.2	1.2	1.1	1.1	1.0
F	2.0	1.7	1.6	1.5	1.4	1.3	1.3	1.2	1.1	1.1	1.0
G	-	1.9	1.7	1.6	1.5	1.4	1.3	1.2	1.1	1.1	1.0
H	-	-	1.9	1.7	1.6	1.5	1.4	1.3	1.1	1.1	1.1
I	-	-	2.0	1.8	1.7	1.5	1.4	1.3	1.1	1.1	1.1
J	-	-	-	1.9	1.8	1.6	1.5	1.3	1.2	1.1	1.1
K	-	-	-	2.0	1.9	1.7	1.5	1.3	1.2	1.1	1.1
L	-	-	-	-	2.0	1.7	1.6	1.4	1.2	1.1	1.1
M	-	-	-	-	-	1.8	1.6	1.4	1.2	1.1	1.1

Repetitive factors (RF) given for surface intervals (h:min)

The complete Canadian Defence and Civil Institute of Environmental Medicine, Toronto, Canada (DCIEM) decompression publication contains tables for in-water and surface decompression with the diver breathing oxygen as well as a wider selection of depths and times for in-water decompression with the diver breathing air. The information in this appendix is from a short version of the tables for the sports diver marketed by UDT Inc. and is presented with the permission of DCIEM and UDT. The tables, in a waterproof format, and an instruction book are available from UDT, 2691 Viscount Way, Richmond, BC, Canada. The complete DCIEM tables should be consulted for the other tables.

Table A: Air decompression

This gives no-decompression limits, repetitive group letters and decompression stops for dives that require stops. The group letters move from A through to M as the diver spends more time at depth. For example, for a dive to 60 feet (18 metres) for less than 14 minutes the diver is in group A and at the maximum dive without stops (50 minutes) he is group F. If he remains at depth for longer, he crosses onto the right-hand side of the tables and must make the decompression stops listed further down the tables. Note that there are two sets of decompression stops: the 3 metre (10 feet) stops for shallower dives and the schedules for dives deeper than 18 metres; these may require stops at 6 metres (20 feet).

Table B: Surface intervals

This is used to allow for the elimination of nitrogen during intervals on the surface. The user enters the table on the line corresponding to his repetitive dive group at the end of the last dive and finds the number that is in the column headed by his surface interval. This factor can then be transferred to Table C to find the no-decompression stops limit for a second dive. For example, a diver surfaces in group F and spends 2 hours 15 minutes on the surface. What is the longest dive he can make to 18 metres without stops? He enters Table B on row F. At the intersection of this row with the column from 2 hours to 2 hours 59 minutes he finds 1.4. This is transferred to Table C. At the intersection of 1.4 and 18 metres is the no-stops limit for the second dive, 29 minutes.

The factor from Table B can also be used to calculate stops. If the task in the second dive considered above took 50 minutes the diver multiplies this by the factor obtained from the surface interval table and uses this time in Table A to get the decompression time. In this case the $1.4 \times 50 = 70$, so the stop(s) for the second dive are for a 70-minute dive to 18 metres

C: *REPETITIVE DIVING*

Depth	1.1	1.2	1.3	1.4	1.5	1.6	1.7	1.8	1.9	2.0
30' *9m*	272	250	230	214	200	187	176	166	157	150
40' *12m*	136	125	115	107	100	93	88	83	78	75
50' *15m*	60	55	50	45	41	38	36	34	32	31
60' *18m*	40	35	31	29	27	26	24	23	22	21
70' *21m*	30	25	21	19	18	17	16	15	14	13
80' *24m*	20	18	16	15	14	13	12	12	11	11
90' *27m*	16	14	12	11	11	10	9	9	8	8
100' *30m*	13	11	10	9	9	8	8	7	7	7
110' *33m*	10	9	8	8	7	7	6	6	6	6
120' *36m*	8	7	7	6	6	6	5	5	5	5
130' *39m*	7	6	6	5	5	5	4	4	4	4
140' *42m*	6	5	5	5	4	4	4	3	3	3
150' *45m*	5	5	4	4	4	3	3	3	3	3

Repetitive factors no-D limits given in minutes according to depth and RF

(10 minutes at 3 metres). The resulting repetitive group letter (H) can be transferred to Tables B and C for any later dive. The diver can use Table A without allowance for a previous dive when he has been on the surface for enough time for the factor to fall to 1.0.

The depth, as in other tables, is the maximum depth reached during the dive. The bottom time is the time from leaving the surface till beginning the ascent to the stop or surface. The ascent rate should be 18±3 metres/minute (60±10 feet/minute). The stops times are the times to be spent at the nominated depths.

Table D

This is used to convert the depth of a dive at altitude to an effective depth that can be used in Table A and to find the correct depth for any decompression stops. The table is entered on the left-hand column in the row with the actual depth of the dive. A correction to be added to the depth obtained is obtained from the intersection of this row with the column that includes the altitude of the water surface at the dive site. For example, a diver intends to conduct a 50-foot dive in water where the surface is at 4500 feet. What is the effective depth to be used for decompression? At the intersection of the actual depth 50 feet row and the 4000-5000 feet column is found 10 feet. This is to be added, so the diver should decompress for a 60-foot dive. The foot of the column shows that any stops to be taken should be at 18 and 9 feet instead of 20 and 10 feet. A further increase of one depth should be added if the diver has been at a lower altitude less than 12 hours before the dive. This corrects for the additional nitrogen remaining in the diver's body.

For a diver to fly after a no-decompression-stops dive the DCIEM table instructions require that he should wait till his repetitive factor in Table B has decreased to 1. He should have a minimum surface interval of 24 hours after a decompression dive.*

The complete instructions for the tables have much information that has been omitted here. This includes rules for omitted decompression and a multilevel dive decompression procedure as well as rules for adjustments for multiple repetitive dives.

* Flying after diving guidelines have recently been the subject of a workshop organized by the Undersea and Hyperbaric Medicine Society. It is reported (Sheffield, PJ, Abstract 20, Supplement to Undersea Biomedical Research, Vol 17, 1990) that the consensus was for more stringent rules than the DCIEM rules.

The workshop suggested a wait for 12 hours for divers who had less than 2 hours diving (surface to surface) in the last 2 days. Divers should wait at least 24 hours before flying after multiday unlimited dives. A delay of at least 24 hours, and preferably 48 hours, was suggested after any dives requiring decompression stops. It is not known if DCIEM will change their rules in line with these guidelines.

D: *DEPTH CORRECTIONS*

Actual depth		1000' → 1999		2000' → 2999		3000' → 3999		4000' → 4999		5000' → 5999		6000' → 6999		7000' → 7999		8000' → 10 000	
		300m → 599		600m → 899		900m → 1199		1200m → 1499		1500m → 1799		1800m → 2099		2100m → 2399		2400m → 3000	
↓	↓																
30'	*9m*	10	*3*	10	*3*	10	*3*	10	*3*	10	*3*	10	*3*	20	*6*	20	*6*
40'	*12m*	10	*3*	10	*3*	10	*3*	10	*3*	10	*3*	20	*6*	20	*6*	20	*6*
50'	*15m*	10	*3*	10	*3*	10	*3*	10	*3*	20	*6*	20	*6*	20	*6*	20	*6*
60'	*18m*	10	*3*	10	*3*	10	*3*	20	*6*	20	*6*	20	*6*	20	*6*	30	*9*
70'	*21m*	10	*3*	10	*3*	10	*3*	20	*6*	20	*6*	20	*6*	30	*9*	30	*9*
80'	*24m*	10	*3*	10	*3*	20	*6*	20	*6*	20	*6*	30	*9*	30	*9*	40	*12*
90'	*27m*	10	*3*	10	*3*	20	*6*	20	*6*	20	*6*	30	*9*	30	*9*	40	*12*
100'	*30m*	10	*3*	10	*3*	20	*6*	20	*6*	30	*9*	30	*9*	30	*9*	40	*12*
110'	*33m*	10	*3*	20	*6*	20	*6*	20	*6*	30	*9*	30	*9*	40	*12*		
120'	*36m*	10	*3*	20	*6*	20	*6*	30	*9*	30	*9*	30	*9*				
130'	*39m*	10	*3*	20	*6*	20	*6*										
140'	*42m*	10	*3*														
Add depth correction to actual depth of altitude dive																	
10'	*3m*	10	*3.0*	10	*3.0*	9	*3.0*	9	*3.0*	9	*3.0*	8	*2.5*	8	*2.5*	8	*2.5*
20'	*6m*	20	*6.0*	19	*6.0*	18	*5.5*	18	*5.5*	17	*5.0*	16	*15.0*	16	*5.0*	15	*4.5*
Actual decompression stop depths (feet/*metres*) at altitude																	

APPENDIX B1: US NAVY RECOMPRESSION THERAPY TABLES

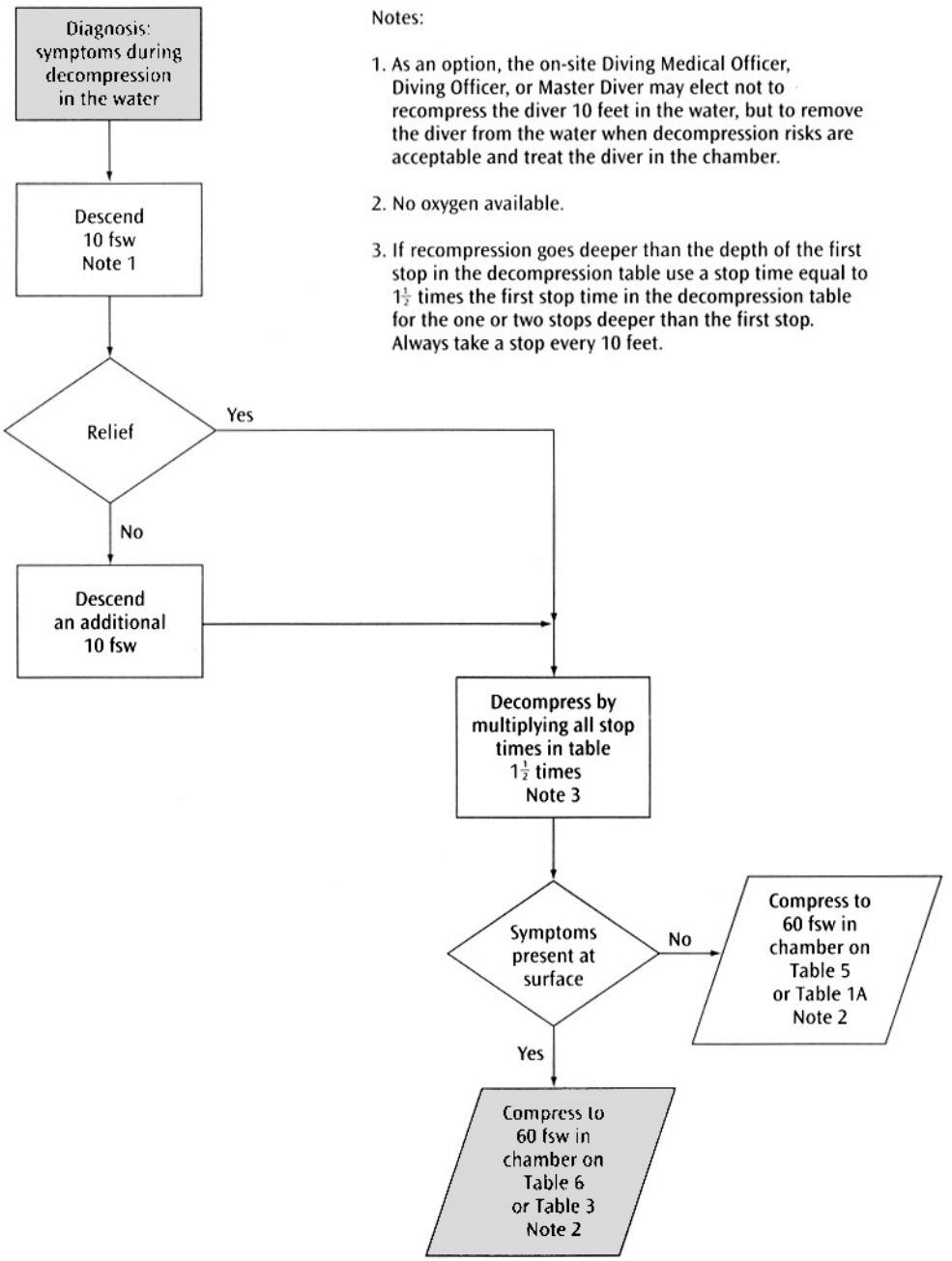
To guide its users in the selection of a decompression therapy table the *US Navy Diving Manual* contains four flow charts. These are reproduced below. After following the appropriate chart the reader is guided to selecting an appropriate table. These are also presented below.

It should be noted that it has been necessary to omit much of the text that contains advice on selecting and using the tables. Any person using these tables, or any other therapy table, should have the complete original document. This presentation is offered as a teaching aid, not as an official guide.

Treatment Table 5: Oxygen treatment of type I decompression sickness

1 Treatment of type I decompression sickness when symptoms are relieved within 10 minutes at 60 feet and a complete neurological exam is normal.
2 Descent rate - 25 feet/min.
3 Ascent rate - 1 feet/min. Do not compensate for slower ascent rates. Compensate for faster rates by halting the ascent.
4 Time at 60 feet begins on arrival at 60 feet.
5 If oxygen breathing must be interrupted, allow 15 minutes after the reaction has entirely subsided and resume schedule at point of interruption.
6 If oxygen breathing must be interrupted at 60 feet, switch to Treatment Table 6 upon arrival at the 30-foot stop.
7 Tender breathes air throughout unless he has had a hyperbaric exposure within the past 12 hours, in which case he breathes oxygen at 30 feet in accordance with paragraph 8.13.5.7.

Depth (feet)	Time (minutes)	Breathing media	Total elapsed time (h:min)
60	20	Oxygen	0:20
60	5	Air	0:25
60	20	Oxygen	0:45
60–30	30	Oxygen	1:15
30	5	Air	1:20
30	20	Oxygen	1:40
30	5	Air	1:45
30–0	30	Oxygen	2:15

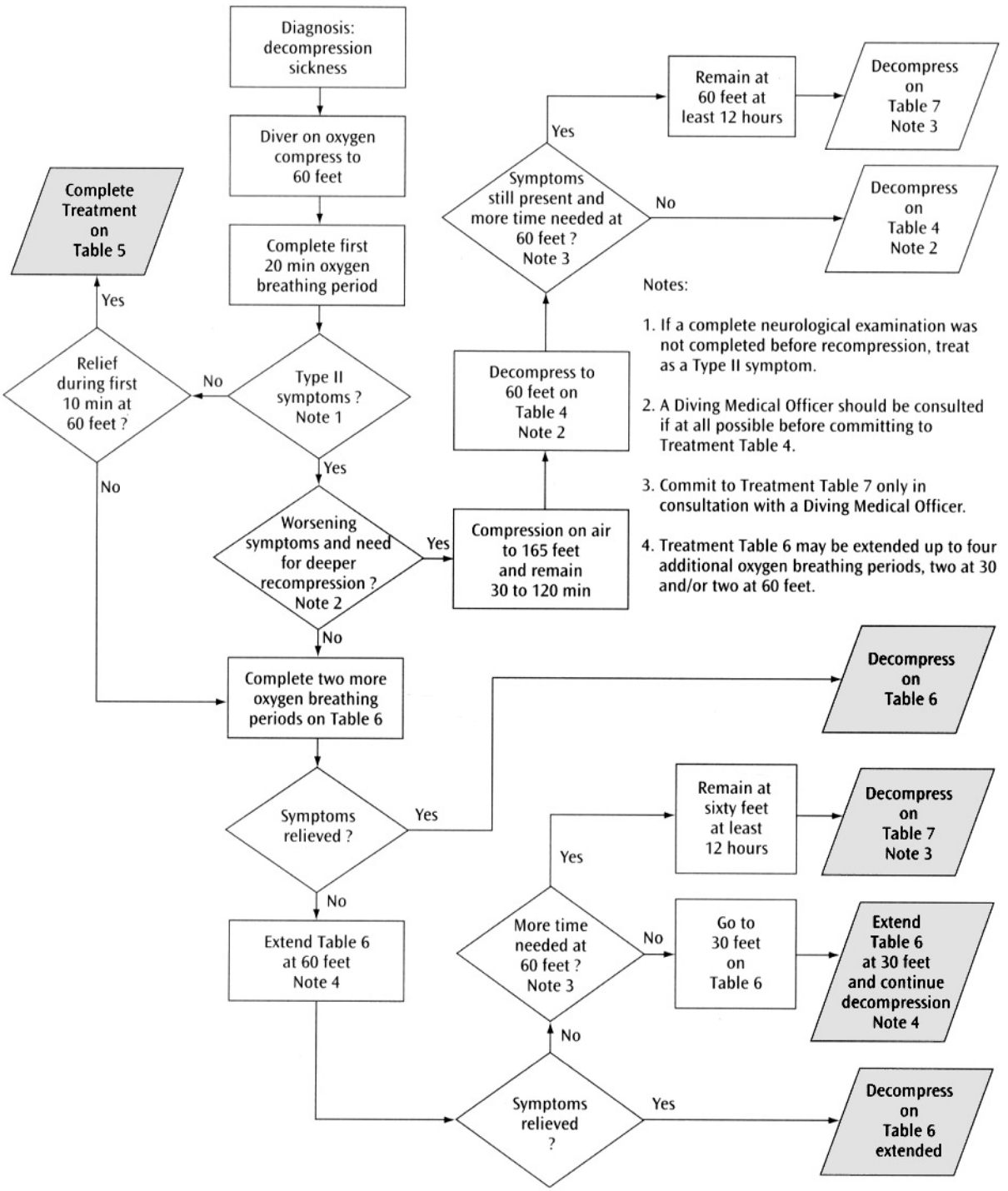


Notes:

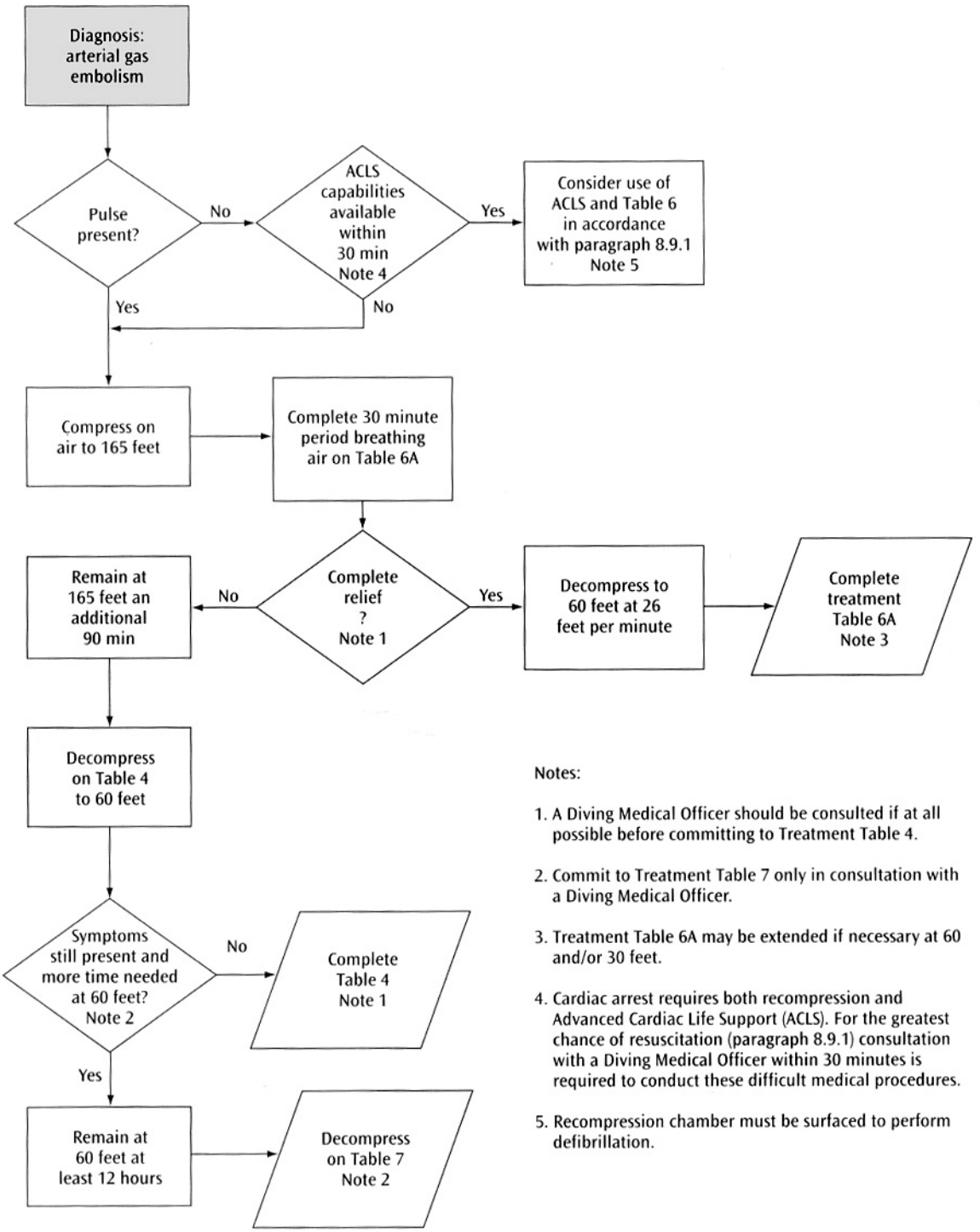
1. As an option, the on-site Diving Medical Officer, Diving Officer, or Master Diver may elect not to recompress the diver 10 feet in the water, but to remove the diver from the water when decompression risks are acceptable and treat the diver in the chamber.

2. No oxygen available.

3. If recompression goes deeper than the depth of the first stop in the decompression table use a stop time equal to $1\frac{1}{2}$ times the first stop time in the decompression table for the one or two stops deeper than the first stop. Always take a stop every 10 feet.



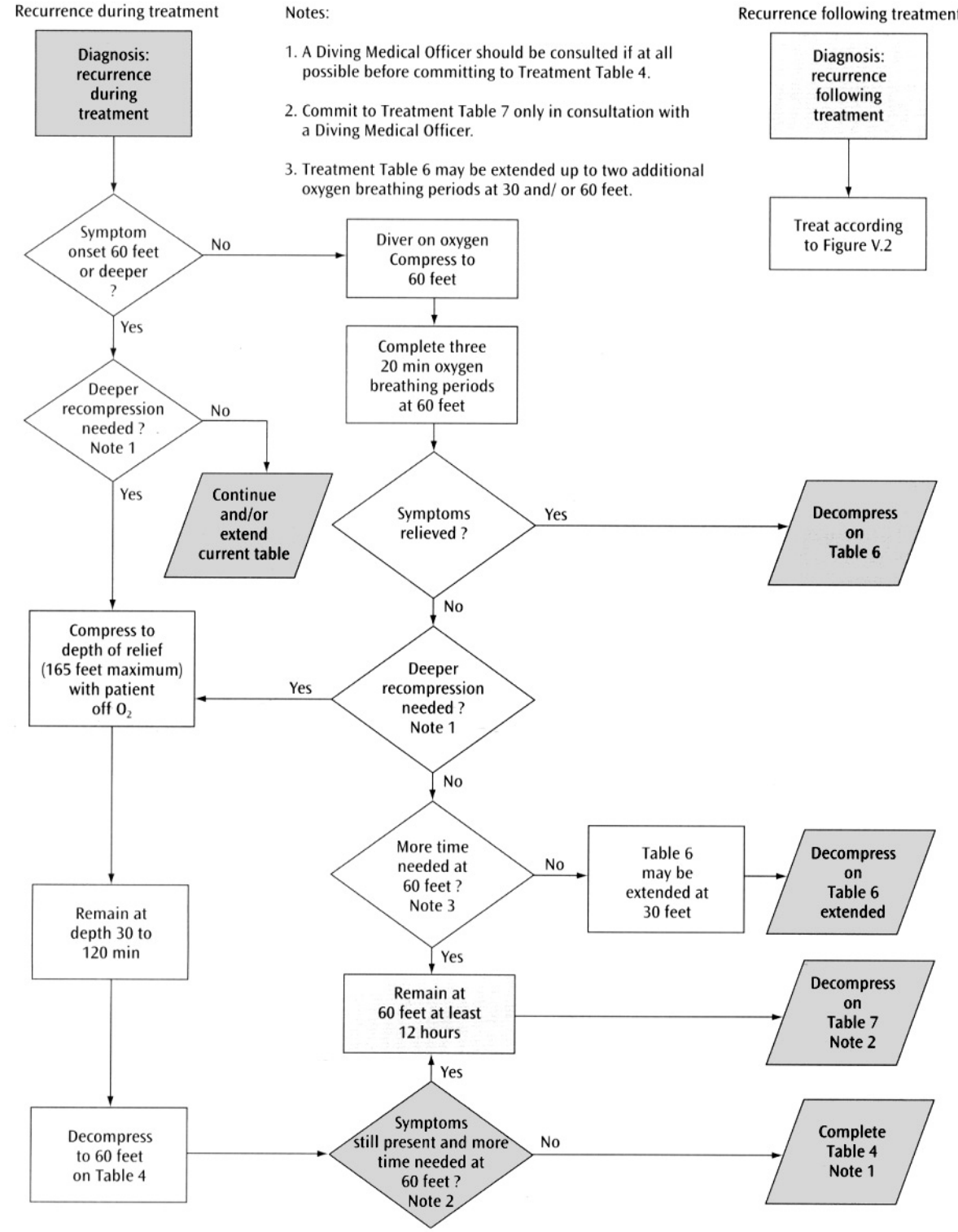
Decompression sickness treatment from diving or altitude exposures



Notes:

1. A Diving Medical Officer should be consulted if at all possible before committing to Treatment Table 4.
2. Commit to Treatment Table 7 only in consultation with a Diving Medical Officer.
3. Treatment Table 6A may be extended if necessary at 60 and/or 30 feet.
4. Cardiac arrest requires both recompression and Advanced Cardiac Life Support (ACLS). For the greatest chance of resuscitation (paragraph 8.9.1) consultation with a Diving Medical Officer within 30 minutes is required to conduct these difficult medical procedures.
5. Recompression chamber must be surfaced to perform defibrillation.

Treatment of arterial gas embolism



Treatment of symptom recurrence

Treatment Table 6: Oxygen treatment of type II decompression sickness

1 Treatment of type II or type I decompression sickness when symptoms are not relieved within 10 minutes at 60 feet.
2 Descent rate - 25 feet/min.
3 Ascent rate - 1 foot/min. Do not compensate for slower ascent rates. Compensate for faster rates by halting the ascent.
4 Time at 60 feet begins on arrival at 60 feet.
5 If oxygen breathing must be interrupted, allow 15 minutes after the reaction has entirely subsided and resume schedule at point of interruption.
6 Tender breathes air throughout unless the tender has had a hyperbaric exposure within the last 12 hours, in which case oxygen is breathed at 30 feet in accordance with paragraph 8.13.5.7.
7 Table 6 can be lengthened up to two additional 25-minute periods at 60 feet (20 minutes on oxygen and 5 minutes on air), or up to two additional 75-minute periods at 30 feet (15 minutes on air and 60 minutes on oxygen), or both. If Table 6 is extended only once at either 60 or 30 feet, the tender breathes oxygen during the ascent from 30 feet to the surface. If more than one extension is done, the tender begins oxygen breathing for the last hour at 30 feet and during ascent to the surface.

Depth (feet)	Time (minutes)	Breathing media	Total elapsed time (h:min)
60	20	Oxygen	0:20
60	5	Air	0:25
60	20	Oxygen	0:45
60	5	Air	0:50
60	20	Oxygen	1:10
60	5	Air	1:15
60–30	30	Oxygen	1:45
30	15	Air	2:00
30	60	Oxygen	3:00
30	15	Air	3:15
30	60	Oxygen	4:15
30–0	30	Oxygen	4:45

Treatment Table 6A: Initial air and oxygen treatment of arterial gas embolism

1 Treatment of arterial gas embolism where complete relief is obtained within 30 min at 165 feet. Use also when unable to determine whether symptoms are caused by gas embolism or severe decompression sickness.
2 Descent rate - as fast as possible
3 Ascent rate - 1 foot/min. Do not compensate for slower ascent rates. Compensate for faster rates by halting the ascent.
4 Time at 165 feet - includes time from the surface.
5 If oxygen breathing must be interrupted, allow 15 minutes after the reaction has entirely subsided and resume schedule at point of interruption.
6 Tender breathes oxygen during ascent from 30 feet to the surface unless the tender has had a hyperbaric exposure within the last 12 hours, in which case oxygen is breathed at 30 feet in accordance with paragraph 8.13.5.7.
7 Table 6A can be lengthened up to two additional 25-minute periods at 60 feet (20 minutes on oxygen and 5 minutes on air), or up to two additional 75-minute periods at 30 feet (60 minutes on oxygen and 15 minutes on air), or both. If Table 6A is extended either at 60 or 30 feet, the tender breathes oxygen during the last 90 minutes of the treatment; 60 minutes at 30 feet and 30 minutes during ascent to the surface.
8 If complete relief is not obtained within 30 minutes at 165 feet, switch to Table 4. Consult with a Diving Medical Officer before switching if possible.

Depth (feet)	Time (minutes)	Breathing media	Total elapsed time (h:min)
165	30	Air	0:30
165–60	4	Air	0:34
60	20	Oxygen	0:54
60	5	Air	0:59
60	20	Oxygen	1:19
60	5	Air	1:24
60	20	Oxygen	1:44
60	5	Air	1:49
60–30	30	Oxygen	2:19
30	15	Air	2:34
30	60	Oxygen	3:34
30	15	Air	3:49
30	60	Oxygen	4:49
30–0	30	Oxygen	5:19

Treatment Table 4: Air or air and oxygen treatment of type II decompression sickness or arterial gas embolism

1 Treatment of worsening symptoms during the first 20-minute oxygen breathing period at 60 feet on Table 6, or when symptoms are not relieved within 30 minutes at 165 feet using air treatment Table 3 or 6A.
2 Descent rate - as fast as possible.
3 Ascent rate - 1 minute between stops.
4 Time at 165 feet - includes time from the surface.
5 If only air is available, decompress on air. If oxygen is available, patient begins oxygen breathing upon arrival at 60 feet with appropriate air breaks. Both tender and patient breathe oxygen beginning 2 hours before leaving 30 feet.
6 Ensure life support considerations can be met before committing to a Table 4. Internal chamber temperature should be below 85°F.
7 If oxygen breathing is interrupted, no compensatory lengthening of the table is required.
8 If switching from a Treatment Table 6A or 3 at 165 feet, stay the full 2 hours at 165 feet before decompressing.

Depth (feet)	Time	Breathing media	Total elapsed time (h:min)
165	½ –2 h	Air	2:00
140	½ h	Air	2:31
120	½ h	Air	3:02
100	½ h	Air	3:33
80	½ h	Air	4:04
60	6 h	Air	10:05
50	6 h	Air	16:06
40	6 h	Air	22:07
30	12 h	Oxygen/Air	34:08
20	2 h	Oxygen/Air	36:09
10	2 h	Oxygen/Air	38:10
0	1 min	Oxygen	38:11

Treatment Table 7: Oxygen/air treatment of unresolved or life-threatening symptoms of decompression sickness or arterial gas embolism

1 Used for treatment of unresolved and life-threatening symptoms after initial treatment on Table 6 or 4.
2 Use only under the direction of or in consultation with a Diving Medical Officer.
3 Table begins upon arrival at 60 feet. Arrival at 60 feet is accomplished by initial treatment on Table 6, 6A or 4. If initial treatment has progressed to a depth shallower than 60 feet, compress to 60 feet at 25 feet/min to begin Table 7.
4 Maximum duration at 60 feet is unlimited. Remain at 60 feet a minimum of 12 hours unless overriding circumstances dictate earlier decompression.
5 Patient begins oxygen breathing periods at 60 feet. Tender need breathe only chamber atmosphere throughout. If oxygen breathing is interrupted, no lengthening of the table is required.
6 Minimum chamber O_2 concentrations is 19%. Maximum CO_2 concentrations is 1.5% SEV (11.4 mmHg). Maximum chamber internal temperature is 85°F.
7 Decompression starts with a 2 foot upward excursion from 60 to 58 feet. Decompress with stops every 2 feet for times shown in profile below. Ascent time between stops is approximately 30 seconds. Stop time begins with ascent from deeper to next shallower step. Stop at 4 feet for 4 hours and then ascend to the surface at 1 foot/min.
8 Ensure chamber life support requirements can be met before committing to Treatment Table 7.
9 Chapter 8 of the *USN Manual* emphasizes that Table 7 is a table to be considered as 'a heroic measure for the treatment of life-threatening decompression sickness'. It is normally invoked at the end of the time at 60 feet on another table, e.g. Table 6, 6A or 4. The decision to change to Table 7 should only be made if the condition of the patient is such as to expect 'that marked residual impairment or loss of life may result if the currently prescribed decompression from 60 feet is undertaken'.
10 The manual suggests that the decision to commence ascent after the 12 hours at 60 should be made based on the patient's condition. A patient who has shown signs of improvement may benefit from further time at 60 feet; a patient who has not shown signs of improvement probably will not.
11 Periods of breathing oxygen should be used if the patient can stand it. Because of the variable times

that the patient may have spent on oxygen it is not possible to stipulate what these should be.

12 Further guidance on the use of this table is contained in Chapter 8 of the *USN Diving Manual*. This should be consulted, and it would be prudent to seek the advice of the USN (Medical Research Institute or Experimental Diving Unit) before using this table.

Depth (feet)	Time (hours) from leaving 60 feet	Ascent rate (feet/hour)	Steps to get ascent rate
60	A minimum of 12 hours hold at 60 feet		
60	0	3	2 feet every 40 min
40	6	2	2 feet every 60 min
20	16	1	2 feet every 120 min
4	32	-	(4 hours hold and then surface 36 hours after leaving bottom)

Air Treatment Table 1A: Air treatment of type I decompression sickness - 100-foot treatment

1 Treatment of type I decompression sickness when oxygen is unavailable and pain is relieved at a depth less than 66 feet.
2 Descent rate - 25 feet/min.
3 Ascent rate - 1 minute between stops.
4 Time at 100 feet - includes time from the surface.
5 If the piping configuration of the chamber does not allow it to return to atmospheric pressure from the 10-foot stop in the 1 minute specified, disregard the additional time required.

Depth (feet)	Time (minutes)	Breathing media	Total elapsed time (h:min)
100	30	Air	0:30
80	12	Air	0:43
60	30	Air	1:14
50	30	Air	1:45
40	30	Air	2:16
30	60	Air	3:17
20	60	Air	4:18
10	120	Air	6:19
0	1	Air	6:20

Air Treatment Table 2A: Air treatment of type I decompression sickness - 165-foot treatment

1 Treatment of type I decompression sickness when oxygen is unavailable and pain is relieved at a depth greater than 66 feet.
2 Descent rate - 25 feet/min.
3 Ascent rate - 1 minute between stops.
4 Time at 165 feet - includes time from the surface.

Depth (feet)	Time (minutes)	Breathing media	Total elapsed time (h:min)
165	30	Air	0:30
140	12	Air	0:43
120	12	Air	0:56
100	12	Air	1:09
80	12	Air	1:22
60	30	Air	1:53
50	30	Air	2:24
40	30	Air	2:55
30	120	Air	4:56
20	120	Air	6:57
10	240	Air	10:58
0	1	Air	10:59

Air Treatment Table 3: Air treatment of type II decompression sickness or arterial gas embolism

1 Treatment of type II symptoms or arterial gas embolism when oxygen is unavailable and symptoms are relieved within 30 minutes at 165 feet.
2 Descent rate - as rapidly as tolerated.
3 Ascent rate - 1 minute between stops.
4 Time at 165 feet - includes time from the surface.

Depth (feet)	Time (minutes)	Breathing media	Total elapsed time (h:min)
165	30	Air	0:30
140	12	Air	0:43
120	12	Air	0:56
100	12	Air	1:09
80	12	Air	1:22
60	30	Air	1:53
50	30	Air	2:24
40	30	Air	2:55
30	720	Air	14:56
20	120	Air	16:57
10	120	Air	18:58
0	1	Air	18:59

APPENDIX B2: US NAVY SATURATION THERAPY PROCEDURES AND TABLES

Decompression sickness during saturation diving may result from excursion ascents or may be associated with the Standard Saturation Decompression. In the US Navy, decompression sickness manifesting during saturation decompression is common and has been characterized by musculoskeletal pain alone. The onset is usually gradual and generally occurs while the diver is still under pressure. However, decompression sickness resulting from excursion ascents may be more severe and may involve the cardiorespiratory system, the central nervous system and the organs of special sense.

Serious decompression sickness resulting from an excursion ascent should be treated by immediate recompression at 30 feet per minute to at least the depth from which the excursion ascent originated. If there is not complete relief at that depth, recompression should continue deeper until relief is accomplished.

Decompression sickness manifested only as musculoskeletal pain and occuring during Standard Saturation Decompression should be treated by recompression in increments of 10 feet at 5 feet per minute until distinct improvement is indicated by the diver. In most instances, improvement continues to complete resolution of the symptoms. Recompression of more than 30 feet is usually not necessary and causes increasing pain in some cases.

During recompression and at treatment depth, a treatment mixture may be given by mask to provide an oxygen partial pressure of 1.5-2.5 atmospheres. Pure oxygen may be used at treatment depths of 60 feet or less. The mask treatment should be interrupted every 20 minutes with 5 minutes of breathing the chamber atmosphere.

A stricken diver should remain at the treatment depth for a minimum of 12 hours in serious decompression sickness and a minimum of 2 hours in pain-only decompression sickness. The Standard Saturation Decompression Schedule can then resume from the treatment depth. However, excursion ascents must not be perfomed.

Treatment gas mixtures

For treatment use, the following gas mixtures, having a range of oxygen partial pressure from 1.5 to 2.5 atmospheres (pure oxygen is used to the depth of 60 feet), should be available:

Depth	Mix
0–60	100% O_2
60–100	40/60
100–200	64/36
200–350	79/21
350–600	87/13
600–1000	92/8
1000–1600	95/5

APPENDIX C1: COMEX THERAPY TABLES (1986)

Type I DCS

Recompress to 12 metres on O_2

If relief within 15 mins
Use Cx12

If no relief
Use Cx18

Type 2 DCS

Recompress to 30 metres on Heliox 50/50
Use Cx30

Cx12

Depth (metres)	Duration (minutes)	Gas mixtures			Elapsed time
		Patient	Attendant	Chamber	
12	120	Oxygen: four sessions (25 mins on, 5 min off)	Ambient	Heliox 20/80 or	2 hours
12→0	30	Oxygen	Oxygen	air	2 h 30 min

Cx18

Depth (metres)	Duration (minutes)	Gas mixtures			Elapsed time
		Patient	Attendant	Chamber	
18	90	Oxygen: three sessions (25 min on, 5 min off)	Ambient	Heliox 20/80 or air	1 h 30 min
18→12	30	Oxygen: one session (25 min on, 5 min off)	Ambient		2 hours
12	150	Oxygen: five sessions (25 min on, 5 min off)	90 min ambient Then O_2, two sessions (25 min on, 5 min off)		4 h 30 min
12→0	30	Oxygen	Oxygen		5 hours

Cx30

Depth (metres)	Duration (minutes)	Gas mixtures			Elapsed time
		Patient	Attendant	Chamber	
30	60	Heliox 50/50	Ambient	Heliox 20/80 or air	1 hour
30→24	30	Heliox 50/50: one session (25 min on, 5 min off)	Ambient		1 h 30 min
24	30	Heliox 50/50: one session (25 min on, 5 min off)	Ambient		2 hours
24→18	30	Heliox 50/50: one session (25 min on, 5 min off)	Ambient		2 h 30 min
18	60	Oxygen: two sessions (25 min on, 5 min off)	Ambient		3 h 30 min
18→12	30	Oxygen: one session (25 min on, 5 min off)	Ambient		4 hours
12	180	Oxygen: six sessions (25 mins on, 5 min off)	Oxygen: six sessions (25 min on, 5 min off)		7 hours
12→0	30	Oxygen	Oxygen		7 h 30 min

For failures, recurrences or worsening of symptoms, and for other than simple cases of DCS, refer to *Comex Medical Book*, 1986, for other procedures and tables.

APPENDIX C2: AUSTRALIAN UNDERWATER OXYGEN TABLE

Notes

1 This technique may be useful in treating cases of decompression sickness in localities remote from recompression facilities. It may also be of use while suitable transport to such a centre is being arranged.
2 In planning, it should be realised that the therapy may take up to 3 hours. The risk of cold, immersion and other environmental factors should be balanced against the beneficial effects. The diver must be accompanied by an attendant.

Equipment

The following equipment is essential before attempting this form of treatment:

1 Full face mask with demand valve and surface supply system *or* helmet with free flow.
2 Adequate supply of 100 per cent oxygen for patient, and air for attendant.
3 Wet suit for thermal protection.
4 Shot with at least 10 metres of rope (a seat or harness may be rigged to the shot).
5 Some form of communication system between patient, attendant and surface.

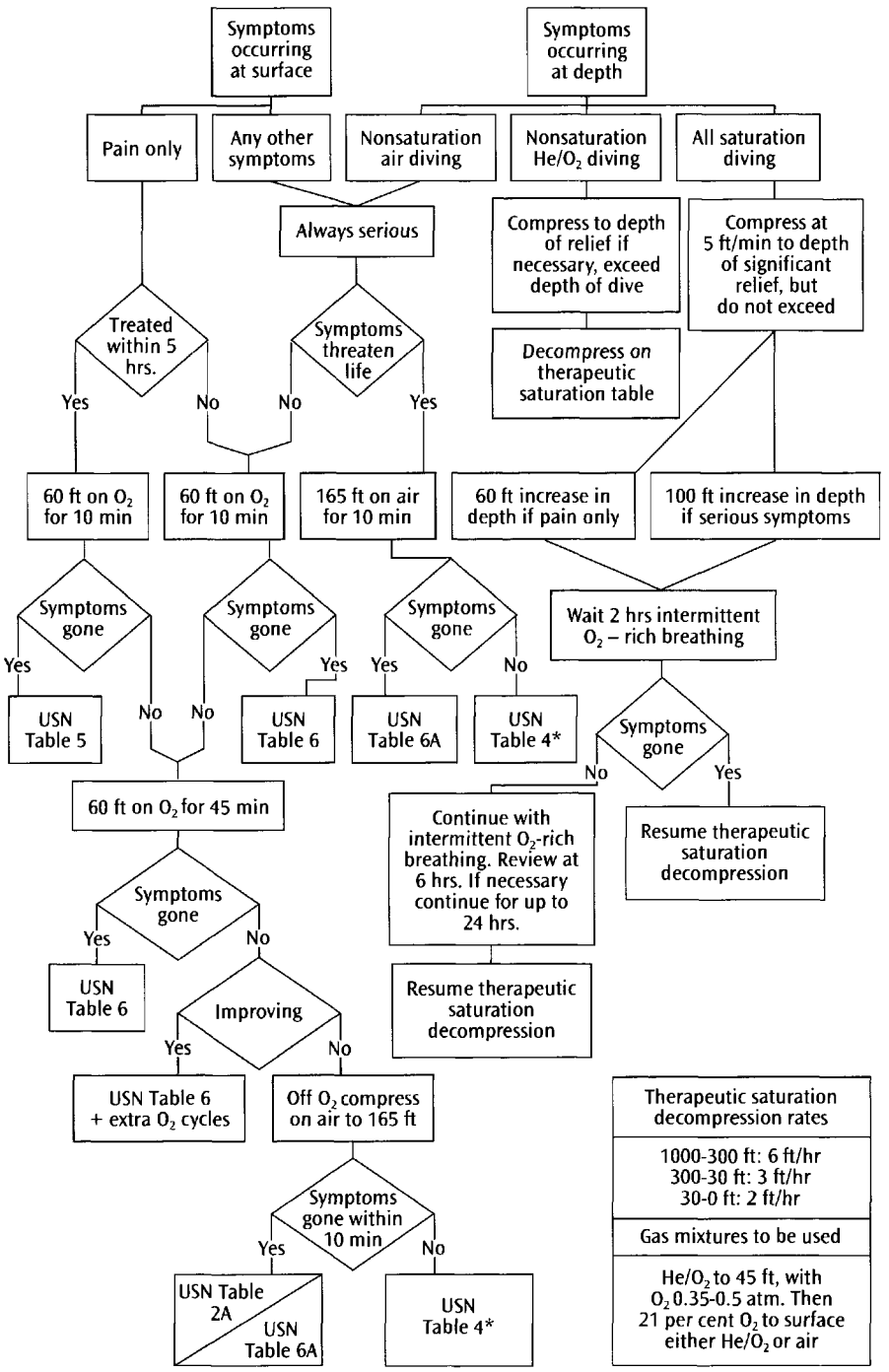
Method

1 The patient is lowered on the shot rope to 9 metres, breathing 100 per cent oxygen.
2 Ascent is commenced after 30 minutes in mild cases, or 60 minutes in severe cases, if improvement has occurred. These times may be extended to 60 minutes and 90 minutes respectively if there is no improvement.
3 Ascent is at the rate of 1 metre every 12 minutes.
4 If symptoms recur remain at depth a further 30 minutes before continuing ascent.
5 If oxygen supply is exhausted, return to the surface, rather than breathe air.
6 After surfacing the patient should be given one hour on oxygen, one hour off, for a further 12 hours.

Table Aust 9 (RAN 82): Short oxygen table

Depth (metres)	Elapsed time		Rate of ascent
	Mild	Serious	
9	0030–0100	0100–0130	
8	0042–0112	0112–0142	
7	0054–0124	0124–0154	12 minutes
6	0106–0136	0136–0206	per metre
5	0118–0148	0148–0218	(4 min/foot)
4	0130–0200	0200–0230	
3	0142–0212	0212–0242	
2	0154–0224	0224–0254	
1	0206–0236	0236–0306	

APPENDIX C3: DUKE UNIVERSITY FLOW CHART



*Always use the oxygen version of USN Table 4, for both patient and attendant(s).

APPENDIX D: DIVING MEDICAL LIBRARY

Classics

La Pression Barometric (Barometric Pressure) by Paul Bert (1878). The Hitchcock translation. Ohio: Columbus Book Company, 1943.
Caisson Sickness and the Physiology of Work in Compressed Air (1912) by Sir Leonard Hill, London: Edward Arnold.
Respiration (1935) Haldane, J.S. and Priestley, J.G. Oxford: Clarendon Press.
Decompression Sickness (1951) edited by Fulton, J.F. London: W.B. Saunders.
Deep Diving and Submarine Operations, 6th edition (1955) by Davis, R.H. London: Siebe, Gorman and Co. Ltd.
Key Documents of the Biomedical Aspects of Deep Sea Diving, Vols 1–5 (1983) A selection from the world's literature 1608–1982. Bethesda, MD: Undersea Medical Society.
Physiology of Breath-Hold Diving and The Ama Of Japan (1965) Rahn, H. and Yokoyama, T. (eds). Washington, DC: National Academy of Sciences.

Current medical texts

Diving and Subaquatic Medicine, 4th edition (2002) by Edmonds, C., Lowry, C., Pennefather, J. and Walker, R. London: Arnold.
Diving Medicine, 3rd edition (1997) Bove, A.A. and Davis, J.C. W.B. Saunders, Philadelphia. ISBN 0-7216605-6-8
Case Histories of Diving and Hyperbaric Accidents (1988) Waite, C.L. (ed.). Bethesda, MD: Undersea and Hyperbaric Medical Society.
Medical Examination of Sports Scuba Divers, 3rd edition (1998) Bove, A.A. (ed.). Surrey, UK: Medical Seminars.
The Sports Diving Medical (1994) by Parker, J. Carnegie, Victoria: J.L. Publications. ISBN 0-9590306-8-9.
Diving Physiology in Plain English (reprinted 1999) Bookspan, J. Bethesda, MD: UHMS Publication. ISBN 0-9304061-3-3.
Diving Medicine for Scuba Divers, 2nd edition (1997) Edmonds, C., McKenzie, B. and Thomas, R. Carnegie, Victoria: J.L. Publications.
UHMS Workshops. Published regularly on topics of interest. See UHMS Society in Appendix F.

First aid

The DAN Emergency Handbook (1995) Lippman, J. and Bugg, S. Carnegie, Victoria: J.L. Publications.
Scuba Diving First Aid (1995) National Safety Council. Published by Jones and Bartlett Pub Inc.
Field Guide for the Diver Medic, 2nd edition (1992) Daugherty, C.G. Austin, TX: Coastal Aquatic Publications.

Specialist texts

The Physiology and Medicine of Diving, 4th edition (1993) Bennett, P.B. and Elliott, D.H. (eds). London: W.B. Saunders. ISBN 0-7020-241-0-4.
Medical Assessment of Fitness to Dive (1995), Elliott, D.H. (ed.). Surrey, UK: Biomedical Seminars. ISBN 0-9525162-0-9.
Long Term Health Effects of Diving (1994), Hope, A., Lund, T., Elliott, D.H., Halsey, M.J. and Wiig, H. (eds). Norwegian Underwater Technology Center.
The Lung at Depth (1999), Lundgren, C.E.G. and Miller, J.N. (eds). New York: Marcel Dekker, Inc.
Stress and Performance in Diving (1987) Bachrach, A.J. and Engstrom, G.H. San Pedro, CA: Best Publishing. ISBN 0-941332-06-3.
Dangerous Marine Creatures (1995) Edmonds, C. Flagstaff, AZ: Best Publishing. ISBN 0-941332-39-X.
Venomous and Poisonous Marine Animals (1996) Williamson, J.A., Fenner, P.J., Burnett, J. and Rifkin, J.F. Sydney: University of NSW Press. ISBN 0-86840-279-6.
Venomous Creatures of Australia (1994), Sutherland, S. Melbourne: Oxford University Press. ISBN 0-19553700 9.
Hyperbaric Medicine Practice, 2nd edition (1999) Kindwall, E.P. Flagstaff, AZ: Best Publishing. ISBN 0-941332-78-0.
Hyperbaric Facility Safety: A Practical Guide (1999) Workman, W.T. Flagstaff AZ: Best Publishing.

Journals

Undersea Biomedical Research. Undersea and Hyperbaric Medical Society. ISSN 0093-5387.
Journal of Hyperbaric Medicine (first published as *Hyperbaric Oxygen Review*), Undersea and Hyperbaric Medical Society. ISSN 00884-1225.
Undersea and Hyperbaric Medicine. Undersea and Hyperbaric Medical Society. ISSN 1066-2936.
Pressure. Undersea and Hyperbaric Medical Society. ISSN 0889-0242.
Aviation, Space and Environmental Medicine. Aerospace Medical Association. ISSN 0095-6562.
Journal of Applied Physiology. American Physiological Society.
EUBS (European Undersea Biomedical Society) Newsletter.
SPUMS Journal. South Pacific Underwater Medicine Society. ISSN 0813-1988.

Indexes

A Bibliographic Sourcebook of Compressed Air, Diving and Submarine Medicine, Hoff, E.C. and Greenbaum, L.J., 3 volumes covering the period up to 1961. Washington, DC: Department of the Navy.
An Annotated Bibliography of Diving and Submarine Medicine (1971) Shilling, C.W. and Werts, M.F., covering the period 1962–69. New York: Gordon and Breach.
Underwater Medicine and Related Sciences: A guide to the literature. Schilling, C.W. *et al.* covering the period 1970–1981.
Vol. 1 (1973) – IFI/Plenum, New York covering the period 1970–71.
Vol. 2 (1975) – IFI/Plenum, New York covering the period 1972–73.
Vol. 3 (1977) – Undersea Medical Society, Bethesda, MD covering the period 1974–75.
Vol. 4 (1979) – Undersea Medical Society, Bethesda, MD covering the period 1976–77.
Vol. 5 (1981) – Undersea Medical Society, Bethesda, MD covering the period 1978–79.
Vol. 6 (1983) – Undersea Medical Society, Bethesda, MD covering the period 1980–81.
Underwater and Hyperbaric Medicine: Abstracts from the Literature. Undersea and Hyperbaric Medical Society (first published 1986). ISSN 0886-3474.

Diving manuals

US Navy NAVSEA

Royal Navy BR2806. HMSO, London, UK.

NOAA (National Oceanographic and Atmospheric Administration), US Government Printing Office, Washington, USA.

BSAC (British Sub-Aqua Club), London, UK.

APPENDIX E: DIVING MEDICAL TRAINING

The Royal Navy, the United States Navy and the Royal Australian Navy have regular courses for their officers; on occasion places are made available to members of other Navies. The British and Australian courses have, on occasion been offered to civilian physicians who can demonstrate a need to attend.

Other courses are offered by the societies mentioned in Appendix F. The Undersea and Hyperbaric Medical Society conducts an advanced training course in conjunction with the National Oceanic and Atmospheric Administration (NOAA). The American Board of Preventive Medicine (ABPM) offers a certifying examination in Undersea and Hyperbaric Medicine. Some hospitals with chambers offer courses, often combined with training in hyperbaric medicine. Introductory courses in diving medicine are also available which concentrate on fitness to dive and diving accident management. The courses are normally advertised in the Journal or newsletter of the appropriate Diving Medical Society.

APPENDIX F: DIVING MEDICAL ORGANIZATIONS, CONTACTS AND INTERNET

Divers alert network

(http://www.diversalertnetwork.org/)

This organization is based in the USA, but has extensions in Europe, Japan, South-East Asia Pacific and Southern Africa. It collates data on diving accidents and analyses this information in regular reports. It also supplies emergency and routine information to divers, diving physicians and others. It distributes diving safety texts and runs courses in diving medicine. It also assists in insurance and emergency transport for divers.

Address: DAN, The Peter B. Bennett Center, 6 West Colony Place, Durham, North Carolina 27705, USA. For non-emergency medical questions, Tel: +1-919-684-2948

Professional societies

THE UNDERSEA AND HYPERBARIC MEDICAL SOCIETY (http:www.uhms.org/)

10531 Metropolitan Avenue, Kensington, Maryland 20895, USA is probably the most important single organization. Their journals, workshop reports, newsletters and abstracts are required reading for an interested physician. It has a worldwide membership. The UHMS holds an Annual Scientific Meeting and publishes the abstracts from these meetings. The UHMS also hosts workshops on specialized topics and publishes these as reports.

More local societies include:

THE SOUTH PACIFIC UNDERWATER MEDICINE SOCIETY (SPUMS) (http:www.spums.org.au)

c/o Australian & New Zealand College of Anesthetists, 630 St Kilda Rd, Melbourne, Victoria, 3004 Australia. Membership mainly from Australia, New Zealand, South-East Asia, but with increasing membership from Europe and the USA. SPUMS holds an Annual Scientific Meeting and the presented papers are published in the *SPUMS Journal*. This is a very practical journal providing clinical diving medicine advice for medical practitioners.

THE EUROPEAN UNDERSEA BIOMEDICAL SOCIETY (EUBS) (http://www.eubs.org/)

EUBS holds an annual scientific meeting and publishes a regular newsletter.

SOCIETE DE PHYSIOLOGIE ET DE MEDECINE SUBAQUATIQUES ET HYPERBARES DE LANGUE FRANCAISE (MEDSUBHYP)

(http://www.azurweb.com/medsubhyp/introus.htm)

Interesting diving medicine Internet sites

Best Publishing – Diving and Hyperbaric Medicine Books: http:/www.diveweb.com/best/index.shtml
Contact Sources for Diving Medicine Online and Ernest S. Campbell MD:
http://www.gulftel.com/~scubadoc/contact.htm
Diving and Hyperbaric Medicine on the Web:
http://azurweb.com/medsubhyp/links.htm
Diving Medicine and Research:
http://www.diverlink.com/medicine.htm
Doc's Diving medicine Home Page:
http://faculty.washington.edu/ekay/
British SubAqua Club Medical Services:
http://www.bsac.com/membserv/medical/medsection.htm

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