MENDING THE BENDS

ASSESSMENT, MANAGEMENT, AND RECOMPRESSION THERAPY

DAVID MERRITT, MD, FS, DMO

BEST PUBLISHING COMPANY

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FLIGHT SURGEON, DIVING MEDICAL OFFICER

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Semper Gumby (Forever Flexible),

David Merritt, M.D., FS, DMO
(Flight Surgeon, Diving Medical Officer)

CHAPTER 1

DIVING PHYSICS

INTRODUCTION

There are a number of different environmental factors, that, unique to the diving community, all interact simultaneously with the diver. These environmental factors include pressure, buoyancy, water temperature, water viscosity, the behavior of sound waves and light waves, and reduced visual and proprioceptual cues.

Pressure is the most important environmental factor affecting the diver. Pressure is defined as the amount of force applied per unit area. Hydrostatic pressure is the force resulting from the weight of water over the object. Absolute pressure is the force that results from the sum of the mass of the water over an object, plus the weight of the atmospheric pressure over the water. Pressure continually increases with the depth of the diver.

Ways to measure pressure:

- Atmospheres (atm or ATA)
- Pounds per square inch (psi)
- Kilograms per square centimeter (kg/ cm^2)
- Millimeters of mercury (mmHg)
- Torr
- Centimeters of water (cmH_2O)
- Feet seawater (fsw)
- Meters of seawater (msw)
- Bars (b)
- Newtons per square meter (Pascal)

GAS LAWS ASSOCIATED WITH PRESSURE

Boyle's Law

For any gas at a constant temperature, the volume of the gas will vary inversely with the pressure.

$$P \times V = K \text{ or } P_1V_1 = P_2V_2.$$

In plain English: ***For any gas at a constant temperature, the volume of gas will vary inversely with the pressure.***

Charles' Law

For any gas at a constant pressure, the volume of the gas will vary directly with the absolute temperature.

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

In plain English: ***If you heat a gas it will expand, if you cool it, it will lose volume, providing it has the space to do so.***

Gay-Lussac's Law

For any gas at a constant volume, the pressure of the gas will vary directly as the absolute temperature.

$$P_1/T_1 = P_2/T_2$$

In plain English: ***If you heat a gas confined within a space, the pressure that gas exerts on the wall of that space will increase. Likewise, if you cool that same gas the pressure will decrease.***

Dalton's Law

The total pressure exerted by a mixture of gases is equal to the sum of the pressures of each of

the different gases making up the mixture, with each gas acting as if it alone was present and occupied the total volume.

$$P_{Total} = PPA + PPB + PPC \text{ etc.}$$

In plain English: *As you dive deeper while still breathing the same percentages of gases in the mixture, the amount of each gas you inhale increases as you dive deeper.*

Henry's Law

The amount of any given gas that will dissolve in a liquid at a given temperature is a function of the partial pressure of that gas in contact with the liquid and the solubility coefficient of the gas in the particular liquid. In plain English: *Because a large percentage of the human body is water, the law simply states that as one dives deeper and deeper, more gas will dissolve in the body tissues and that upon ascent, the dissolved gas must be released.*

BUOYANCY

Buoyancy is the body's ability to displace water. Any object placed in water will do one of two things: float or sink. This is dependent on the density of the object in relation to the density of the solution it is being immersed in. Buoyancy is best described by Archimedes Principle which states "*Any object wholly or partly immersed in a fluid is buoyed up by a force equal to the weight of the fluid displaced by the object.*" Divers tend to be more buoyant in salt water than fresh water due to the increased density of sea water. Without proper buoyancy, a diver must increase the amount of energy that he will exert on a dive to remain neutral. This will both increase the rate at which he consumes his air supply and increase the rate at which he will absorb nitrogen, and possibly increase his risk of decompression sickness.

Factors that affect buoyancy include:

- Body Composition (including amount of body fat)
- Salinity of water (Fresh water weighs 62.4 pounds per cubic foot, sea water weighs 64 pounds per cubic foot)
- Divers' lung volume
- Volume of air in the wet suit (that changes with depth)
- The change in gas volume in the breathing container

WATER TEMPERATURE

Thermal conductivity in water is 25 times greater than air. This will account for a significant loss of body temperature unless there is adequate thermal protection. Heat can be lost by the diver in a number of ways. Conductive heat loss occurs by direct molecule-to-molecule contact with subsequent heat transfer. In this manner, the body warms the thin layer of water that surrounds the diver, and through continuation of conductive forces, the heat continues to move away from the diver's body, subsequently taking more heat away from the diver. And if the water around the diver is moving, heat is lost via convection. Heat can also be lost by radiation. Radiation heat loss is the result of the emission of infrared waves from bare skin. Radiation heat loss is negligible when compared to the heat lost by convection and conduction. The final way that divers lose heat is by evaporative heat loss, from both the skin and the lungs. While cold water divers tend to not sweat very much, the air source that they are breathing from is very dry. As a result, exhaled air has a high degree of moisture contributed by the lungs. While this may seem inconsequential, it takes only 2.5 ml of evaporative water to cool the surface it evaporated from 2°F. Also, some breathing mixtures (primarily helium) can contribute significantly to hypothermia, even to the point of inducing clinical hypothermia.

WATER VISCOSITY

The increased viscosity of water compared to that of air significantly increases the energy requirement for any physical needs of the diver. Even simple physical skills require an increased workload on the diver. This increased workload on the muscles also affects the movement (and absorption) of gases throughout the body.

SOUND WAVES

Sound moves at a much greater speed (4,700 ft./sec vs. 1,090 ft./sec) through water than through air. This means the direction location ability associated with sound is lost. The effect of water on the tympanic membrane causes it to lose elasticity and compliance, which causes the loss of air conduction of sound (which is a much more sensitive method of hearing). The threshold of hearing is raised 40-75dB in water. Bone becomes the principle mode of hearing.

LIGHT WAVES

Light, like sound, behaves differently under water. The refraction index is 1.3 times that of air, making objects appear 25% larger and closer. The water-to-cornea interface causes an additional loss of refraction, resulting in a hyperopia of about 40 diopters. Tunnel vision further restricts vision. Additionally, there is a loss of both light transmission and color, further hampering the senses of the diver. An additional drawback is that the diver's eye must accommodate to the loss of ambient light, as the diver descends. The effect is no different than when you must accommodate to the loss of light as the sun sets, or going into a dark room. This is worsened by the fact that divers tend to descend at a faster rate than light accommodation can occur.

PHYSICAL PROPERTIES OF GASES WHEN DIVING

There are several properties that adversely affect our ability to adapt to an undersea environment. These are density, viscosity, solubility, diffusibility, and thermal conductivity. The diffusion of one gas through another is an important problem that relates to diving. Oxygen, in an air mixture, must diffuse through the alveolar gas to arrive at the air-blood interface. The ability of one gas to diffuse through another directly affects the exchange of oxygen and carbon dioxide. Three elements should be noted: 1) The diffusion rate of a gas through the gas phase is approximately five orders of magnitude greater than the diffusion rate through a liquid phase; 2) The diffusion coefficient increases with temperature; and 3) The diffusion coefficient is inversely related to the barometric pressure. At 1,000 fsw (31.3 ATA) the diffusion coefficient would be reduced 31-fold. This should be considered as an indication of intrapulmonary gas diffusion problems that might be seen at depth.

GASES USED IN DIVING

Oxygen (O_2)

Oxygen is the most important of all gases. Atmospheric air is about 21% oxygen. It is colorless, odorless, tasteless, and readily combines with other elements. Water is about 89% oxygen by weight. Of all the gases we breathe, only oxygen is actually used by the body. The remaining 79% serves to dilute and carry the oxygen. Oxygen at mixtures over 100% (which can happen when breathing mixture under pressure) can be dangerous; subsequently, patients breathing pure oxygen at depth must be monitored for oxygen toxicity.

Nitrogen (N_2)

Also colorless, odorless, and tasteless, and is a component of all living organisms. It does not support combustion or life, and does not combine easily with other elements. It is essentially a carrier for oxygen. In the diving environment, it has some significant disadvantages. The two principal disadvantages are nitrogen narcosis (which limits the depth to which air can be safely used), and decompression sickness (which limits the duration for which air can be safely used).

Helium (He)

Also colorless, odorless, and tasteless, but is totally inert (it exists as a single atom in its free state). When used instead of nitrogen in an air mixture, it eliminates a lot of the problems associated with nitrogen. Most importantly, it has a much lower solubility in water and fat than nitrogen. Since most people on the dive boats these days tend to be comprised mostly of those two substrates (plus hot air), there is a significantly lower amount of absorbed gas that has to be released from the

tissues on the ascent to the surface. This fact has given rise to significantly fewer incidents of decompression sickness, especially when used in extreme depth dives. Numerous scientific articles by Philip B. James have attested to this. But this gas does incorporate its own unique set of disadvantages. Primarily, its distortion of speech makes communication very difficult if not impossible, and its extremely high thermal conductivity can cause a rapid loss of body heat through the respiratory tract, which can lead to hypothermia.

Hydrogen (H_2)
Hydrogen has been used in experimental diving (in place of nitrogen for the same reasons as helium), but because of its violently explosive nature when used in the presence of more than 5.3% oxygen, it never really caught on.

Neon (Ne)
It does not have the problems associated with nitrogen, nor does it create problems with speech like helium. It is also thermally stable, and is not prone to explosion. It is not readily available.

Argon (Ar)
Like neon, it was used briefly, and had the potential to be a great gas for diving, except for one disadvantage. As soon as it was breathed under pressure, it would induce unconsciousness. Dive boat operators felt this would not be viewed as favorable.

Carbon Dioxide (CO_2)
This is a natural by-product of cellular respiration, and is generally of no concern to SCUBA divers. However, in a hyperbaric chamber, or closed-circuit breathing, it can easily become toxic and fatal.

Carbon Monoxide (CO)
This is a by-product of carbon combustion, and is very toxic in a hyperbaric environment. Special attention must be made to prevent any contamination by this gas. The greatest danger with this gas is that it is less toxic with depth. Its lethal effects are greatest as the diver ascends.

CHAPTER 2

MECHANICAL EFFECTS OF HYDROSTATIC PRESSURE

INTRODUCTION

The clinical name for the mechanical effect of pressure is barotrauma. Barotrauma during descent is called a squeeze. Barotrauma of ascent is called reverse squeeze. A squeeze is the result of the change in ambient pressures to air-filled spaces in the body during changes in the water column. Divers have descended to depths of deeper than 1,000 fsw (445 psi) in the open sea, and deeper than 2,250 fsw (1,001.3 psi) in experimental situations with no problems associated with barotrauma. Yet frequently, when divers complain of a squeeze, it occurs in the shallowest part of the dive. This is because the greatest pressure changes occur in the first 33 fsw. There is a 50% pressure change in the first 33 fsw. Even if you could descended to 30,000 fsw, you would never experience another change this great.

There are 5 conditions that must be met for barotrauma to occur:

- There must be a gas-filled space.
- That space must have rigid walls.
- The space must be enclosed. If the space was allowed to vent or equilibrate, injury would not occur.
- The space must be vascularized, and have a membrane lining at least one wall of the space. The membrane, once traumatized by the pressure changes, will allow blood to enter the space, helping to equilibrate the pressure change.
- There must be a change in ambient pressure (Boyle's Law).

The primary symptom in barotrauma is pain. This is the most frequent symptom. It is by no means the most severe. Other more serious symptoms will be discussed later. In all patients who present with a squeeze, no matter where it is or how small it may seem, these patients are at increased risk for DCS (decompression sickness) and AGE (arterial gas embolism). While the yield is low, and it is an unlikely diagnosis, it must be considered in your differential and ruled out.

TYPES OF BAROTRAUMA

Middle Ear Squeeze

This is the most common type of barotrauma. As the diver descends through the water column, the volume of gas in the middle ear chamber is decreased (Boyle's Law). This can be equalized by the diver blowing air through their eustachian tube. Failure to equalize will result in negative pressure inside the tympanic membrane (TM), causing the TM to retract sharply. This induces an immediate and sharp pain, usually when the pressure differential reaches about 60 mm/hg (as little as 4 fsw). If descent continues, one of two things will happen: either the blood vessels on the inside to the TM will eventually tear, allowing blood to enter the middle ear, and the blood then will equalize the pressure; or the TM will burst. Rupture will generally occur if the pressure differential is greater than 100 mm/hg (as little as 5 fsw). Rupture will generally relieve the symptom of pain. However, if diving in cold

water, perforation of the TM is generally followed by vertigo. Initial symptoms usually occur between 11-20 fsw.

Reverse Middle Ear Squeeze

If the eustachian tube (which connects the throat with the middle ear, allowing for pressure equalization) becomes blocked on ascent, pressure in the middle ear increases, causing the TM to bulge outward. If the overpressure continues, the TM may rupture, causing symptoms similar to a simple ear squeeze with perforation. However, symptoms of vertigo or nausea and vomiting on ascent must first be presumed to be the result of an AGE (arterial gas embolism).

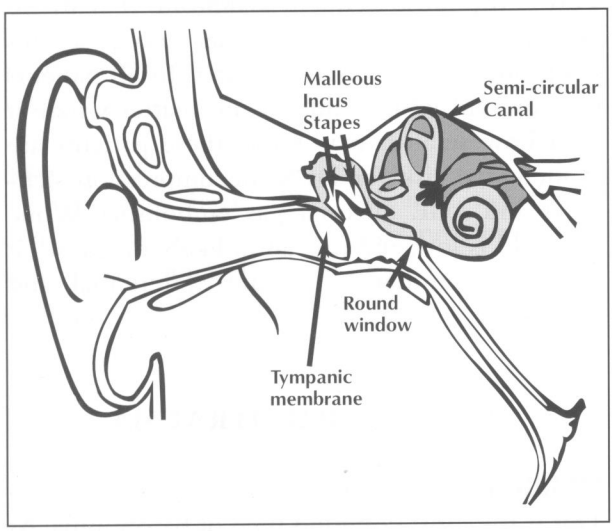


Figure 1 – Ear

Sinus Squeeze

The sinuses are also prone to barotrauma, if the ostium of the sinus becomes obstructed. Unlike the middle ear, there is no way to equalize your sinuses once they begin to experience a squeeze. Usually once a sinus squeeze begins, the only way to get relief is to ascend. While ascending a few feet may alleviate the pain, and a slower descent may allow continuation of the dive, failure of the pain to resolve should terminate the dive. Frequently, when the diver surfaces, he may notice a bloody nasal discharge. Physical exam may reveal a sinus that will not transilluminate. It is usually self-limiting.

Reverse Sinus Squeeze (Sinus Overpressure)

This occurs when gas is trapped inside the sinus, with the symptoms occurring during ascent. This can occur when edema, polyps, or some other pathology can act as a check valve, preventing the escape of gas from the sinus. Rarely, the trapped gas can extravasate into surrounding tissue, resulting in subcutaneous emphysema. While self-limiting, this should be treated with antibiotics.

Face or Body Squeeze

Any part of a diver's equipment that has an air/body interface can be prone to a squeeze. A mask squeeze can be prevented by exhaling through the nose. If the diver is wearing goggles, he cannot equalize the pressure. This can have significant effects on the eyes. Divers with a mask are at similar risk if they do not equilibrate by nasal exhalation. Divers with a history of radial keratotomy are particularly at risk. When wearing a dry suit, air can become trapped by a fold in the suit. Normally, this will only cause mild discomfort. If, however, this occurs on ascent, and enough of a vertical change occurs in the water column before the trapped air is released, an explosive decompression of the tissue distal to the trapping site results, causing catastrophic gas embolization.

Overexpansion of Stomach and Intestine

Persons who consume flatulogenic foods prior to diving will have no problem getting to depth. However, on ascent, the gases that formed at depth will not only continue to form, but will also begin to expand (Boyle's Law again). This may cause the diver to stop his ascent and expel this gas (the infamous double bubble). Failure to properly vent can cause perforation and gas embolization. Better to vent, than to get bent. Caution should be taken when in an oxygen-enriched chamber, as the mixture of oxygen and mystery gas can be explosive if exposed to a spark.

Alternobaric Facial Palsy

A rare complication can be the exposure of the facial nerve to middle ear pressure as it traverses the temporal bone. If the middle ear is pressurized

during ascent, the vascular supply of the Facial Nerve (CN VII) can be compromised, resulting in an ischemic neuropraxia. The complete clinical picture includes Bell's Palsy, nausea,vertigo, and ataxia. These symptoms are easily confused with AGE. Once the middle ear equilibrates, the symptoms resolve in a matter of minutes. This problem is also associated with flying after diving, and can be associated with high-altitude flights in an unpressurized aircraft or an aircraft suffering an explosive decompression.

Tooth Squeeze
This can be caused by a small pocket of air trapped below a faulty filling, or under a tooth as a result of decay. These can develop pain either on descent or on ascent, which may ultimately cause the filling to become dislodged.

Alternobaric Vertigo
This is usually the result of middle ear overpressurization during ascent. In this instance, the patient will often feel a pressure in his inner ear, followed by a potentially incapacitating vertigo. The vertigo may last for several minutes. Relief is generally abrupt, often associated with a sound that is audible to the patient like air leaking from his inner ear (which it is). This will completely resolve if the diver stops his ascent and descends a few feet. Usually, a short stop at depth followed by a slow ascent will prevent a recurrence.

Round or Oval Window Rupture
At the far end of the cavity that is covered on one side by the tympanic membrane is a membrane called the oval window and the round window. Just as the malleous is attached to the tympanic membrane, the stapes is attached to the oval window, ultimately transmitting vibrations to the cochlea. A sudden pressure change in the inner ear (most likely on ascent), can cause rupture of the round window. The symptoms will be similar to alternobaric vertigo, but will not spontaneously resolve. They may also be accompanied by a roaring tinnitus (ringing of the ears). This can lead to permanent hearing loss and chronic vertigo. Differentiation of these symptoms from an AGE are difficult at best. If it was a short, shallow dive, it would seem unlikely to be an AGE; however, recompression therapy will not further hurt a round window rupture. So if you are not completely sure of the cause of the symptoms, recompress. Persistent symptoms at depth will confirm your diagnosis.

PULMONARY OVERINFLATION SYNDROMES

POIS are a group of barotrauma-related diseases caused by the expansion of gas trapped in the lung during ascent, or overpressurization of the lung with subsequent overexpansion and rupture of the alveoli. The two main causes are 1) excessive pressure inside the lung caused by positive pressure, and 2) failure of the gas to escape from the lungs via non-pathologic routes.

Pulmonary overinflation can result from the diver either voluntarily or involuntarily failing to exhale during ascent. Involuntary causes include asthma, thick pulmonary secretions, and coughing or sneezing during ascent.

The clinical manifestations of POIS depend on the location of the free air. This can be either soft tissue or vascular.

Arterial Gas Embolism
AGE is the most immediate life-threatening emergency to face a diver (other than sharks and being Evan's dive buddy). On ascent, air expands in the alveoli, and if not properly vented, the alveoli will expand, forcing the gas directly into the capillaries surrounding the alveoli. The gas forms bubbles that are carried into the systemic circulation, where they will eventually embolize when the arteries/arterioles become too narrow to allow the bubble to proceed. This will cause anoxia (a lack of oxygen) to the tissues distal to the embolis. If this tissue happens to be CNS (central nervous system) or cardiac, the results can be disastrous. Delay in treatment can leave a patient with permanent sequelae. If these patients are not immediately recompressed, the outcome is frequently fatal.

Mediastinal and Subcutaneous Emphysema

Mediastinal emphysema occurs when gas has been forced through traumatized lung tissue into the mediastinum. This emphysema can extend up into the neck, face, and axilla. Airway compromise is always a concern, as is latent infection and compression of blood supply. In addition, if the emphysema occurs at depth, you still have to get the patient to the surface, causing further expansion of the bubbles.

Pneumothorax

Pneumothorax is the result of air entering the space between the lungs and the chest wall. The depth that it occurs and the size of the air pocket will determine the treatment required and the seriousness of the insult. If a patient with an untreated pneumothorax is recompressed, it is almost guaranteed that the pneumothorax will maintain its size at depth, making intervention (a chest tube or needle thoracotomy) a necessity prior to ascending to the surface.

Tension Pneumothorax

This is the same as a pneumothorax, except that the air in the pleural space forces the lung to push into the opposite side of the thorax, further compromising the ability to breathe.

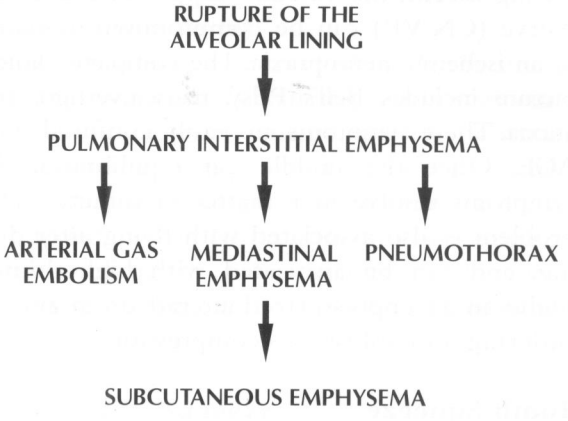


Figure 2 – Pulmonary Overinflation Syndromes

CHAPTER 3

AIR DIVING

INTRODUCTION

The function of under water breathing devices is twofold: first, they must supply an acceptable respiratory gas to the diver, and second, they must supply this gas at a pressure equal to the surrounding environment. In an open-circuit system, the compressed gas is stored in a cylinder, either on the surface or on the diver's back. The trick comes in getting the high-pressure gas into low-pressure lungs.

OPEN CIRCUIT DEMAND UNDER WATER BREATHING APPARATUS

By looking at the below illustration, we can see how high pressure is made breathable for the diver. Cylinder pressure at P1 enters the high-pressure chamber which contains a valve stem and seat held closed against the high-pressure orifice by a lightweight sealing spring (S1). The valve seat is connected through this orifice to a heavy diaphragm and spring assembly (S2). On one side of the diaphragm is the intermediate pressure chamber (P2), and on the other side is

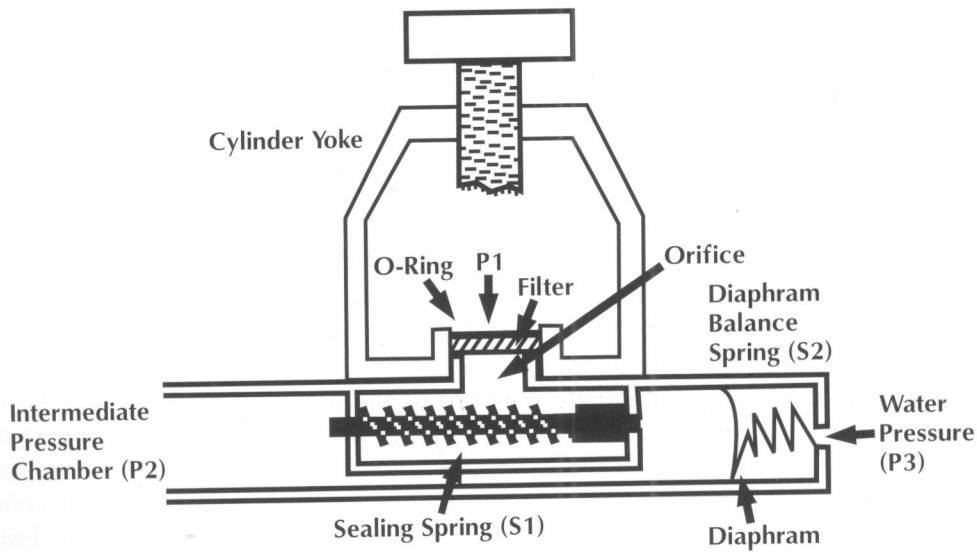


Figure 1 – First Stage Regulator

water pressure (P3) plus the pressure due to the spring (S2). When the intermediate chamber pressure falls due to inhalation, the diaphragm is deflected inward, opening the valve and allowing high-pressure air from the cylinder to the intermediate chamber. The intermediate chamber then leads to the second stage.

The second stage works similarly to the first stage, in that when the diver inhales, a spring holding a valve shut against the intermediate pressure chamber is compressed, allowing air to enter the mouthpiece and be inhaled by the diver. It is then exhaled directly into the water via an exhaust port. Simultaneous to when the diver stops inhaling, the valve to the intermediate pressure chamber closes.

MANUFACTURE, FILTRATION, AND TESTING OF DIVERS' AIR

The air used for diving must meet stringent standards. The requirements set forth by FED SPEC BB-A-1034 are as follows:

CONSTITUENT	FED SPEC
Oxygen (O_2) (percent by volume)	20-22%
Carbon Dioxide (CO_2) (by volume)	500ppm (max)
Carbon Monoxide (CO) (by volume)	10ppm (max)
Total Hydrodrocarbons (as CH_4) (by volume)	25ppm (max)
Oil, Mist, Particulates (weight/volume)(max)	.005mg/L
Odor (not pronounced or objectionable)	
Separated Water	none
Total Water (weight/volume) (max)	0.02mg/L
Nitrogen Dioxide (NO_2) (by volume)	2.5ppm (max)
Nitrous Oxide (N_2O) (by volume)	2.0ppm (max)
Sulfur Dioxide (SO_2) (by volume)	2.5ppm (max)
Halogenated Compounds (by volume)	
Refrigerants	2.0ppm (max)
Solvents	0.2ppm (max)
Acetylene (C_2H_2)	0.1ppm (max)
Ethylene (C_2H_4)	0.4ppm (max)

CHAPTER 4

TOXIC EFFECTS OF INHALED GASES

INTRODUCTION

Oxygen, like any other drug available to today's physician, can be both lifesaving and detrimental, depending on how it is used. Damage to the lungs from oxygen was noted as early as the turn of the century, while seizures related to oxygen were documented in the 1930s. In a dry chamber, oxygen can usually be tolerated up to 2 ATA (oxygen treatments at 60 fsw are 2.8 ATA).

Susceptibility to oxygen toxicity varies from person to person, as well as from episode to episode. An oxygen seizure does not mean that an individual will always have a seizure every time they encounter the same partial pressure of oxygen. The mixing of oxygen with inert gases does not affect the likelihood of having a seizure. It makes no difference whether the oxygen is mixed with nitrogen or helium; once you reach sufficient partial pressure, you will suffer the effects of oxygen toxicity.

Statistically, the likelihood of encountering a seizure during oxygen therapy is 1.3 times per 10,000 exposures. There are several factors that can affect an individual's tolerance to CNS oxygen toxicity. The most important consideration to the diving population and individuals in hyperbaric therapy is to make sure that the mixture that the individual is breathing does not have an elevated level of CO_2. Carbon dioxide will significantly lower an individual's seizure threshold.

The most common symptoms noted prior to seizing include, but are not limited to, muscular twitching (frequently around the mouth and eyes), nausea (with or without vomiting), vertigo, visual disturbances, tinnitus, air hunger or shortness of breath, anxiety, confusion, irritability, fatigue, or loss of coordination. The most common symptoms can be remembered by the following mnemonic:

CONVENTID

CON-vulsions
V-Vision
E-Ears (tinnitus)
N-Nausea
T-Twitching
I-Irritability
D-Dizziness

These symptoms do not always occur. Sometimes the first symptom will be seizure activity. If a seizure occurs in the chamber, the treatment is to simply remove the oxygen. The seizure is usually self-limiting, and while it can be quite alarming to the individual witnessing it, tends to be benign. The most critical element that needs tending during a seizure is that the patient must not be allowed to move to a shallower area while seizing. While an individual is seizing, he or she is not able to exhale, and is at risk of pulmonary overinflation syndromes. If a tender suffers a

seizure, then another tender will need to be placed in the chamber to monitor the situation and to help get that tender to the surface. It is also important to evaluate any individual for trauma after suffering a seizure.

Once a patient or tender has a seizure, they may continue with further treatments. However, precautions should be taken to minimize the risk of future seizures by comparing their history with the above list, and to ensure that they receive air at breaks when scheduled or breathe oxygen at a lower partial pressure.

FACTORS AFFECTING THE DEVELOPMENT OF OXYGEN TOXICITY

Things That Make Seizures More Likely

- Adrenocortical Hormones
- CO_2 Inhalation
- Amphetamines
- Hyperthermia
- Insulin
- Oral Hypoglycemic Agents
- Paraquat
- Hyperthyroidism
- Vitamin E Deficiency
- Disulfiram (Antabuse™)

Things That Make Seizures Less Likely

- Acclimatization to Hypoxia
- Adrenergic Blocking Agents
- Antioxidants
- GABA
- Ganglionic Blocking Agents
- Glutathione
- Hypothyroidism
- Reserpine
- Vitamin E
- Disulfiram (Antabuse™)
- Succinate
- Hypothermia

*It should be noted that disulfiram has the ability to block both pulmonary and central nervous system toxicity. The drug probably acts in competition with enzymes containing SH bonds for free radical oxygen molecules and thereby exerts a protective influence; however, its protective activity is limited to the first hyperbaric oxygen treatment. On subsequent treatments, disulfiram inhibits the formation of super oxide dismutase (SOD), which is the body's major protection against oxygen toxicity. While a single exposure to hyperbaric oxygen would illicit protective benefits from the drug, repeat exposures would most likely invite complications of increased risk of both pulmonary and central nervous system oxygen toxicity.

PULMONARY OXYGEN TOXICITY

Poisoning and subsequent pulmonary damage can occur following prolonged oxygen exposure. It can begin to occur if oxygen in a concentration greater than 60% is inhaled for greater than 24 hours. In the undersea environment, this can occur while breathing air at 2.8 ATA (60 fsw). Long exposures to higher levels are encountered during recompression treatments. The initial symptoms of pulmonary oxygen toxicity usually begin with burning upon inspiration, and progress to pleuritic chest pain, substernal burning, cough, and shortness of breath. If left unchecked, the symptoms can progress to Adult Respiratory Distress Syndrome. The early changes of pulmonary oxygen toxicity are reversible with the discontinuances of oxygen treatments.

The cumulative effects of pulmonary trauma secondary to oxygen exposure can be measured by the Unit Pulmonary Toxicity Dose. This is a table developed to anticipate the level of insult that pulmonary tissues will receive as a result of exposure to oxygen at various pressures and lengths. These numbers should be used as guidelines only. Each patient must be evaluated separately for any indications of pulmonary injury. During treatments for DCS, treatments should be planned to minimize the UPTD. For mild DCS, the UPTD should be 615 or less. For prolonged therapy or serious DCS, the exposure should be kept below 1425. A dose of 1425 will

produce an approximate 10% decrease in vital capacity. These calculations do not make allowances for recovery time spent off of oxygen between treatments. As a note, treatments with doses of 600 UPTD have been administered twice daily with no evidence of cumulative toxicity.

pO_2	Kp	pO_2	Kp	pO_2	Kp	pO_2	Kp
0.5	0.0	1.7	2.07	2.9	3.7	4.1	5.18
0.6	0.26	1.8	2.22	3.0	3.82	4.2	5.3
0.7	0.47	1.9	2.36	3.1	3.95	4.3	5.42
0.8	0.65	2.0	2.5	3.2	4.08	4.4	5.54
0.9	0.83	2.1	2.64	3.3	4.2	4.5	5.66
1.0	1.0	2.2	2.77	3.4	4.33	4.6	5.77
1.1	1.16	2.3	2.91	3.5	4.45	4.7	5.89
1.2	1.32	2.4	3.04	3.6	4.57	4.8	6.01
1.3	1.48	2.5	3.17	3.7	4.7	4.9	6.12
1.4	1.63	2.6	3.31	3.8	4.82	5.0	6.24
1.5	1.78	2.7	3.44	3.9	4.94		
1.6	1.93	2.8	3.57	4.0	5.06		

Figure 1 – Kp factors for calculating UPTD

How to Figure UPTD

1. Convert the partial pressure of oxygen breathed at each depth to PO_2 in ATA.
2. Select the corresponding Kp from the Kp factors for calculating UPTD table.
3. Multiply the time of exposure in minutes, at that PO_2, by the corresponding Kp to get the UPTD for that depth.
4. Add the totals for various depths for total UPTD exposure.

UNIT PULMONARY TOXICITY DOSES FOR COMMON TREATMENTS

Treatment	UPTD
90 minutes O_2 at 45 fsw	270
U.S. Navy Treatment Table 5	333
U.S. Navy Treatment Table 6	645
U.S. Navy Treatment Table 6	
With 1 extension at 60 fsw	718
With 1 extension at 30 fsw	787
With 1 extension at 60 fsw and 1 at 30 fsw	860
U.S. Navy Treatment Table 6A	690
With 1 extension at 60 fsw	763
With 1 extension at 30 fsw	833
With 1 extension at 60 fsw and 1 at 30 fsw	906

CARBON MONOXIDE INTOXICATION

Carbon monoxide poisoning, because of the molecular affinity of CO to hemoglobin and the change of that affinity under pressure, creates a unique problem for the diver. At 1 ATA, CO has an affinity for hemoglobin that is 210 times that of oxygen. This means that carbon monoxide will preferentially bind to hemoglobin, displacing oxygen and producing cellular hypoxia. Oxygen under pressure will help to displace carbon monoxide. This allows divers to absorb carbon monoxide at depth while maintaining adequate oxygenation due to the increased partial pressure of oxygen. But once the diver begins to surface, the partial pressure of oxygen decreases and hypoxia induced by the carbon monoxide becomes more pronounced, producing unconsciousness as the diver gets closer to the surface. There are three primary reasons that we treat carbon monoxide poisoning with hyperbaric oxygen. The first is that by increasing the partial pressure of oxygen, the CO level will fall just by the laws of mass action. The higher the PO_2, the more CO can be displaced from hemoglobin. The second is that it is possible to supply the entire metabolic demand of an individual with oxygen dissolved in the plasma at 60 fsw. In effect, you could remove the body's entire supply of red blood cells (with CO still bound)

and the patient would be able to profuse oxygen to his vital tissues. The third reason is that the total elimination half-life of CO is dramatically reduced with increased oxygen partial pressure.

ALTERATION IN CARBOXYHEMAGLOBIN ELIMINATION HALF-TIME UNDER VARIOUS O_2 ENVIRONMENTS

O_2 Environment	HbCO*
1 ATA Air	250
1 ATA O_2	60
2 ATA O_2	46
3 ATA O_2	23

HbCO elimination Half-Time (in minutes)

INERT GAS NARCOSIS

Nitrogen narcosis is the most common form of narcosis associated with diving. For years it had been called rapture of the deep. That can occur at any depth. Nitrogen narcosis is a phenomenon that is directly related to pressure (depth). Any inert gas can cause a narcosis or a state of altered mental condition, given the right circumstances. Nitrogen is the most readily available, one of the most predictable, and certainly one of the most enjoyable altered states associated with diving. It is an overwhelming intoxication that can begin anywhere from 100 fsw to about 170 fsw, and will increase with depth. It can be fatal, however, and should not be taken lightly. Treatment is to simply ascend and decrease the partial pressure of the inert gas.

CHAPTER 5

ALTERATIONS IN CARDIOPULMONARY PERFORMANCE AT DEPTH

INTRODUCTION

Complicating cardiovascular performance at depth is the fact that any alterations in the rate of ventilation can dramatically affect the saturated gasses in the body. At sea level, any alteration in respiratory rate or muscular workload can cause subtle changes in the body's pH, which is easily compensated for by just as subtle changes in respiratory rate or modification in physical workload. Things are not quite as simple in the under water environment. Both the medium that you are working in and the complicating factors produced by that medium make the subtle changes that work on land ineffective. The increased viscosity of water increases the amount of energy required (and subsequent increased cellular metabolism), and the zero gravity prevents the normal leverage we take for granted in an air environment. Depth, with a small change in CO_2 production, can produce a significant change in perceived CO_2 amount in the body. This is a result of pressure modifications on the partial pressure of those gases induced by depth.

ALVEOLAR VENTILATION AND HYPERCAPNIA

The diver's ability to allow his partial pressure of CO_2 and O_2 to vary from what is normally found on the ground at sea level is very common in diving and has multiple causes. Every time you breathe, oxygen is extracted from inhaled air and diffused into the blood, while carbon dioxide diffuses out of the blood and into the about-to-be-exhaled air. The ultimate goal of the body is to consume O_2 at the same rate that CO_2 is produced. Then, the amount of CO_2 exhaled will equal what is produced, while the amount of O_2 inhaled will equal the amount consumed. Another complicating factor is that part of the respiratory tract does not allow the exchange of gases. This area includes the oral pharynx, and the upper and lower respiratory tract, not including the terminal bronchiole.

During hard work (exercise), CO_2 production and O_2 requirements increase in a linear fashion (it's physics, either do the math yourself or take my word for it). During hard work, these values may increase up to ten times over resting demands. Subsequently, the respiratory exchange rate must increase the same amount. A complicating factor of diving is that divers are using a finite amount of gas for breathing. When it is gone, so are you. Divers are continually motivating themselves to do more work with less O_2 while tolerating a higher CO_2 level.

Pulmonary Ventilation

Respiration is divided into 4 major phases:

1. Pulmonary Ventilation – the exchange of air between the alveoli and atmosphere

2. Diffusion of O_2 and CO_2 between the alveoli and the blood
3. Transport of O_2 and CO_2 in blood and body fluids to and from the cells
4. Regulation of ventilation and respiration

Pulmonary Mechanics
Normal quiet breathing is accomplished by movement of the diaphragm alone. No accessory muscles are needed for normal quiet breathing. During normal quiet exhalation, the diaphragm simply relaxes, and the elastic recoil of the lungs, chest wall, and abdominal structures compresses the lungs.

Pleural Pressure
Pleural pressure is the pressure of the fluid within the (potential) pleural space. This pressure is slightly negative, and helps to keep the alveoli open.

Alveolar Pressure
This is the pressure inside the lung alveoli. With an open glottis and no air flow it is 0 (zero). Inspiration causes a negative pressure and expiration causes a positive number.

Respiration
The work of inspiration can be divided into three parts:

1. Compliance work or elastic work – the work required to expand the lungs against the lung and chest wall elastic forces
2. Tissue resistance work – the work required to overcome the viscosity of the lung and chest wall structures
3. Airway resistance work – the work required to overcome the resistance of air moving from the atmosphere to the gas exchange tissue

During normal breathing, only a small percentage of the total workload is used to overcome tissue resistance and airway resistance.

During heavy exercise, when air flow through the respiratory passages is at a high velocity, the greatest proportion of work is used to overcome airway resistance.

In pulmonary disease, all three types of work are increased. It is usually an acute increase in respiratory passageway compromise (Asthma, RAD [reactive airway disease]) that will cause the greatest distress and immediate threat to life.

PULMONARY VOLUMES AND CAPACITIES

Volumes

1. Tidal Volume. The volume of air inspired or expired with each normal breath.
2. Inspiratory Reserve Volume. The amount of extra volume of air that can be inspired over and above the normal tidal volume (should be a large number).
3. Expiratory Reserve Volume. The extra amount of air that can be expelled by forceful expiration after the end of a normal tidal expiration.
4. Residual Volume. The amount of air that is left in the lungs after the most forceful expiration.

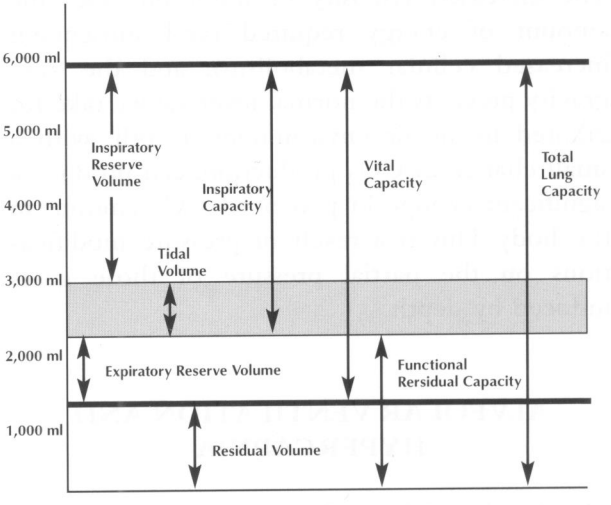


Figure 1 – Pulmonary Volumes and Capacities

Capacities

1. Inspiratory Capacity. The Tidal Volume plus the Inspiratory Reserve Volume. This is the amount of air a person can breath beginning at the normal expiratory level and distending the lungs to the maximum amount.
2. Functional Residual Capacity. The Expiratory Reserve Volume plus the Residual Volume. This is the amount of air that remains in the lungs at the end of normal expiration.
3. Vital Capacity. The Inspiratory Reserve Volume plus the Tidal Volume plus the Expiratory Reserve Volume. This is the maximum amount of air a person can expel from the lungs after filling their lungs to their maximum extent and then exhaling to the maximum extent.
4. Total Lung Capacity. The Vital Capacity plus the Residual Volume. This is the maximum volume to which the lungs can be expanded with the greatest possible Inspiratory effort.

Alveolar Ventilation. The rate at which air reaches the gas-exchanging tissues in the lung.

Dead Air Space. The space in the respiratory tract that fills the spaces were gas exchange does not occur. It is not the space in your body where farts are born.

Anatomic vs. Physiologic Dead Air Space. Anatomic dead air space is any air containing tissue that does not allow for the exchange for gas. Physiologic dead air space is alveolar air that must be added to Anatomic Dead Air due to non-functional or partial-functioning alveolus (emphysema). In a normal person, the anatomic and physiologic dead spaces are almost equal (about 150 ml). In a patient with COPD (chronic obstructive pulmonary disease), the anatomic dead air space can approach 2 liters. This is why patients with COPD tend to not make good scuba diver candidates.

NEURAL CONTROL OF ALVEOLAR VENTILATION

Normally, respiration is controlled by chemoreceptors, reflexes, and cortical input to the medulla. These interact with each other via positive feedback inhibition. Through these controls the body is able to finely control its levels of CO_2, O_2, and H^+ (hydrogen ions). The peripheral chemoreceptors (carotid and aortic bodies), and the central chemoreceptors (located in the floor of the fourth ventricle) serve as modifiers to stimulate the medullary respiratory center.

The peripheral chemoreceptors respond primarily to oxygen partial pressures of 60mm/Hg, even though they may still have some discharge at 100mm/Hg. This indicates that the peripheral chemoreceptors are more responsive to the partial pressure of low O_2, inducing increased ventilatory stimulus in a hypoxic state rather than a hypercapnic state. The central chemoreceptors, on the other hand, respond primarily to CO_2. These are the most sensitive receptors, being more responsible for maintaining the respiratory rate through CO_2 and H^+ levels than via peripheral O_2 levels. Subsequently, proper O_2 delivery can occur despite highly variable respiratory rates. Quite the opposite, the level of CO_2 is directly related to the respiratory rate. If you increase respiration, your partial pressure of CO_2 will drop significantly, causing an equally proportional change in the H^+ ion concentration and affecting the pH. Oxygen partial pressure does not have a similar effect. This explains why the body's respiratory rate is controlled by the body's partial pressure of CO_2, not O_2.

THE CHEMOSENSITIVE AREA

None of the 3 main respiratory centers of the medulla (Dorsal, Ventral, Pneumotaxic), are directly affected by CO_2 or H^+. Instead, a fourth area, the chemosensitive area, is highly sensitive to changes in blood concentration of either PCO_2 or H^+, but primarily H^+. This area in turn signals the respiratory areas of the brain. The primary problem with this system is that H^+ does not

cross the blood-brain barrier easily (under non-pathologic conditions, the amount is zero). Subsequently, these neurons are exquisitely more sensitive to levels of CO_2 in the blood than to blood H^+ levels. "Why is that so?" you might ask. It is because CO_2 can easily cross the blood brain barrier. When it does, it combines with water. This forms carbonic acid, which instantly disassociates into bicarbonate and H^+. For those of you who are chemists it goes like this:

$$CO_2 + H_2O_3 \Rightarrow H_2CO_3 \Rightarrow H^+ + HCO_3-$$

This stimulation only persists for a few days, and the primary stimulation begins to ebb after just a few hours, primarily because of renal excretion of bicarbonate into the blood that binds with H^+ in the CSF (cerebrospinal fluid).

CONTROL OF RESPIRATION THAT IS OXYGEN MEDIATED

The other control system for ventilation is the Peripheral Chemoreceptor System. The largest number of chemoreceptors is located in the carotid bodies, with the next largest collection in the aortic bodies. Carotid bodies are located bilaterally in the bifurcations of the common carotid arteries. Aortic bodies are located along the arch of the aorta. Each of these chemoreceptor bodies receives a special blood supply through a minute artery directly from an adjacent arterial trunk. The blood flow through these bodies is extreme – 20 times the weight of the bodies themselves, each minute. Subsequently, the removal of O_2 from this flowing blood is virtually zero. This means that the chemoreceptors are continually exposed to non-deoxygenated arterial blood at all times. So when arterial O_2 levels drop, the O_2 has no direct effect on the respiratory center. It will have an effect on the chemoreceptors that will in turn affect the respiratory center. The chemoreceptors are also affected by CO_2 and H^+. But since they have a much more dramatic effect on the respiratory center directly, this effect is negligible.

CHANGES IN RESPIRATORY CONTROL IN DIVING

Numerous studies have shown that the respiratory response to CO_2 can be depressed in some divers, beginning as shallow as 2 ATA (33 fsw). The depressive response is unpredictable between divers. Its mechanism seems unclear, but may be a partial response to accommodation (in frequent divers). The final effect of this poor response to CO_2 is that the diver will hypoventilate. This causes a gradual increase in serum CO_2 level, with subsequent decrease in pH. It can be further aggravated by the increased workload brought on by working in the under water environment. This further explains why divers must maintain a proper ventilatory rate, and not try to extend their bottom times by modification of respiratory rate.

PHYSIOLOGIC EFFECTS OF HYPERCAPNIA

Inhaled CO_2 and increased respiratory dead space should increase both tidal volume and respiratory rate and produce a sensation of air hunger. Increased mechanical workload such as increased air density and regulator resistance can increase the feeling of suffocation. Acute hypercapnia produces an uncompensated respiratory acidosis. This in turn results in a number of circulatory and central nervous system effects. These include increased cardiac output, an increase in pulmonary arterial pressure, and myocardial contractility. The rise in cardiac output is the result of increased contractility and venous constriction. The cerebral vasculature responds to CO_2 by vasodialating, giving the telltale "CO_2 headache." Ignoring this early sign can cause increased swelling, which can lead to further respiratory depression, and, ultimately, death. As the level of CO_2 rises, the peripheral vasculature will begin to dilate, inducing a state of hypotension that will further complicate the clinical picture.

The primary symptoms of hypercapnia are:

- Shortness of breath or the feeling of suffocation
- Tachycardia, which may be misinterpreted as a result of exercise by the diver
- Headache, which is generally mild to moderate initially, but becomes severe and throbbing after termination of the exposure
- Vertigo, ataxia, or light-headedness
- Burning eyes
- Progressive mental confusion, and, if left unchecked, unconsciousness

Secondary conditions that can result from hypercapnia:

- Increased risk of nitrogen narcosis
- Increased risk of central nervous system poisoning
- Increased risk of decompression sickness
- Reduced exercise tolerance

OXYGEN AND CARBON DIOXIDE TRANSPORT BY THE BLOOD

Oxygen is carried throughout the body by being bound to hemoglobin and dissolved in the plasma. The amount of oxygen saturated in the plasma will increase as the diver increases his depth. The percent saturation of hemoglobin to oxygen depends primarily on the partial pressure of oxygen, but will also be affected by temperature and carbon dioxide level (pH).

One phenomenon that assists the body in the elimination of CO_2 from the body is called the Haldane Effect. As the concentration of oxygen increases in the blood, it tends to displace carbon dioxide from the hemoglobin. This becomes very important in the elimination of carbon dioxide.

The Haldane Effect results from the fact that the combination of oxygen with hemoglobin in the lungs causes the hemoglobin to become a stronger acid. This displaces carbon dioxide from the blood and into the alveoli in two ways: 1) The more acidic hemoglobin has less tendency to form carbaminohemoglobin, displacing much of the carbon dioxide that is present in the carbamino form in the blood; and 2) The increased acidity of the hemoglobin also causes it to release an excess of hydrogen ions, and these in turn bind with bicarbonate ions to form carbonic acid. This can then disassociate into water and carbon dioxide, and the carbon dioxide is then released from the blood into the alveoli. In the tissue capillaries, the Haldane Effect causes increased pickup of carbon dioxide because of oxygen removal from the hemoglobin. In the lungs, it causes increased release of carbon dioxide because of oxygen pickup by the hemoglobin.

PULMONARY GAS TRANSFER IN THE HYPERBARIC ENVIRONMENT

The primary purpose of the lung is to allow for the exchange of O_2 and CO_2 with the blood for the metabolic needs of the body. This is achieved by bringing the environmental air into intimate contact with the body's blood flow, allowing for the exchange of gases via diffusion. Under normal circumstances, the air entering the alveoli will cross the alveolar membrane and mix instantaneously with the blood supply on the other side. This leads to a uniform alveolar blood/gas composition. In a resting individual, there is an approximate 0.7-second phase where the air is motionless in the alveoli, allowing for gas exchange. During exertion, this time can be reduced to 0.25 seconds or less. Because of the difference in binding potential of CO_2 and O_2 with hemoglobin, some deficits may begin to occur as the workload increases. One thing that can affect the diffusion of gases is trauma to the alveolar membrane. This can be caused by any of a number of insults, ranging from infection to the repeated trauma of breathing air under pressure. Any type of trauma or insult to the alveolar membrane will create thickening of the membrane (due to inflammation), which will in turn increase the amount of time and energy required for the gases to diffuse across.

Another problem stems from the fact that as divers breathe gases under increasing pressures, the density of these gases increases. As a result of this increased density, airway resistance increases, leading to an increase in work. In addition, as the density of air increases, the total number of molecules that must be exchanged across the membrane to maintain the metabolic needs of the body increases. This increase in the number of molecules still needs to be done in the same amount of time as gas exchange at normal atmospheric pressure. Yet another problem that must be addressed is that as the densities of gases increase, their ability to stay mixed decreases. This means that as the gases endure greater pressures, they tend to act as separate gases, diffusing across alveolar membranes at their own inherent rate. The more slowly diffusing gases are not given sufficient time to cross the membranes.

CARDIAC EFFECTS OF THE HYPERBARIC ENVIRONMENT

The bradycardic (slowing of heart rate) effects of hyperbaric air have been recognized since before the turn of the century (Heller, Mager, and Von Schrotter, 1897). They found that heart rate was uniformly depressed at all levels of exercise, so that the slope of heart rate change in relation to exercise remained constant. At a pressure of 4.5 ATA (115 fsw), the heart rate is depressed approximately 12 beats per minute. A similar decrease in rate can be seen by increasing the partial pressure of oxygen. But this is not as extreme as with pressure alone. In additional studies done to a maximum depth of 950 fsw, all of the changes were seen in the first 300 fsw. The extent of the bradycardia appears limited. Some EKG changes are also seen with exposure to pressure. The most common changes appear to be a prolonging of the Q-T interval, and the appearance of a nodal rhythm (which appears to be rate-dependent). Some of this may also be related to membrane conduction changes associated with pressures.

CHAPTER 6

HAZARDS OF THE UNDERSEA ENVIRONMENT

INTRODUCTION

There are numerous ways to get into trouble in the undersea environment. The really great thing about hyperbaric medicine is that people seem to be reinventing how they can hurt themselves at record paces. Anymore, people aren't satisfied to strap on a tank and go stare at fish in a nice tropical environment at 40 fsw. Now, everyone wants to dive with the sharks, dive through the ice, go cave exploring, go to extreme depths, breathe Nitrox, breathe Trimix or some other exotic gas. They want to dive open-circuit, closed-circuit, semi-open, semi-closed, "I thought I was supposed to leave it open, but found it was closed so I left it ajar" breathing apparatus. And then when they finally get their top-of-the-line, fully-turbocharged, radioactive, one-size-fits-all, no-side-effects, self-winding, guaranteed-on-time-delivery-with-no-reasonable-offer-refused dive gear (batteries not included), strap it on and descend through the ice, into the deep reaches of the cave, finally see the shark, and then they want to FEED THE THING. Rest assured. Our future in hyperbaric medicine is secure.

HUMAN PERFORMANCE LIMITATIONS UNDER WATER

Humans were designed to work best in an air environment at sea level (14.7 psi). Send them up to the mountains and their performance drops away. The same is true when you place a person under pressure. Even in an air environment, if humans breathe air at pressures greater than 14.7 psi, their performance drops off in a predictable fashion as the pressure increases.

Higher cognitive functioning seems to suffer at a greater rate than does the speed and performance of motor skills (but the latter suffer the same predictable decrease). Cognitive function begins to rapidly disappear after about 7 ATA (about 200 fsw), and dexterity tends to head out the door at about 10 ATA (300 fsw). This is breathing air (79% nitrogen, 21% oxygen), in an air environment (i.e., hyperbaric chamber, not under water). Human variability enters into the equation, with more experienced divers being able to tolerate depth much better than less experienced divers.

JUST ADD WATER

Working in a free-floating medium, such as water, produces problems as well as some noteworthy benefits. Some of the advantages include ease of body movement and increased lifting capability (with the use of air bags). Some of the disadvantages include loss of mechanical energy (there is about a 20% loss of mechanical energy in water) and diminished ability to apply leverage. Another limiting factor is man's ability to

work in a cold environment. It is a well-known fact that humans perform less well as their core temperature drops. Because of the conductive heat losses associated with water immersion, diving in water temperatures in the mid-80°Fs can still produce hypothermia if the diver remains immersed long enough. If you have an unresponsive diver that you think may be suffering from hypothermia, warm him up. If he gets better, you were probably right. If a diver suffers a cardiac arrest secondary to hypothermia, continue CPR until he has warmed up. The Golden Rule of Hypothermia: No one is pronounced dead until they are warm and dead.

Psychological factors can contribute to performance limitations. Frequently, communication is nonexistent, the equipment is uncomfortable, and the visibility is limited, allowing the diver's imagination to go into overdrive. This is frequently compounded by the isolation inherent to diving. Nighttime operations only intensify these problems.

BODY'S RESPONSE TO TEMPERATURE

Internal Temperature	Response
37°C (98.6°F)	Cold sensations, cutaneous vasoconstriction, ++ muscle tension, ++VO_2 (oxygen consumption)
36°C (96.8°F)	Sporadic shivering, suppressed by voluntary movements, with some uncontrolled bouts, additional rise in VO_2
35°C (95.0°F)	Mental confusion, decreasing will to struggle
34°C (93.2°F)	Amnesia, poor articulation, sensory and motor degradation
33°C (91.4°F)	Hallucinations, delusions, clouding of consciousness, 50% risk of death
32°C (89.6°F)	Atrial arrythmias
31°C (87.8°F)	Failure to recognize familiar persons
30°C (86.0°F)	No response to pain, muscle rigidity, pupillary reflex lost
29°C (84.2°F)	Loss of consciousness
28°C (82.4°F)	DTRs (deep tendon reflexes) lost, ventricular arrythmias
27°C (80.6°F)	V-Fib likely, flaccid skeletal muscles

UNDER WATER ORIENTATION (OR LACK THEREOF)

There are three sources that give us physical information about the environment that we are in. The first are called *teloceptors*, which give information about distance. These senses involve vision, smell, and hearing, and are concerned with the remote environment. *Exteroceptors* are the second source of sensors. These include the sensory organelles of the skin. They are sensitive to pressure, pain, warmth, and cold, and are responsible for the information we receive about our immediate environment. *Proprioceptors* are found in the muscles, tendons, joints, and in the labyrinth system. They are intimately interconnected and provide us with continual information as to the position of out body in relation to our environment.

Vertigo in the hyperbaric environment is termed alternobaric vertigo. It can be the result of failure to equalize pressure during descent or ascent, or the result of perforation of the tympanic membrane, allowing cold water to enter the middle ear. Merely descending to depth can induce vertigo through inert gas narcosis (usually at depths approaching 7 ATA). Other causes of vertigo include (but are not limited to): decompression sickness, oxygen toxicity, carbon dioxide poisoning, hypoglycemia, persistent sea sickness, residual alcohol intoxication, drug/pressure interaction, hyperventilation, hypoventilation, bad air mix, and the power of suggestion combined with a low-visibility environment. Episodes of vertigo have been reported in both experienced and novice divers. The danger lies not in the vertigo itself, but the action that the vertigo brings about. Disorientation may cause the diver to be unable to maintain depth, causing the diver to either descend or ascend. He may panic and have an uncontrolled ascent to

the surface, inviting risk of AGE or missed decompression. If the diver's symptoms are severe enough, vomiting into his mouthpiece can significantly increase his risk of airway obstruction, either by obstructing his regulator or by the aspiration of stomach contents. Directional orientation in the under water environment is limited at best, even in the most ideal situations. The primary method of maintaining geographical orientation is through vision. But because of field of view limitations induced by masks, even in great under water conditions (shallow and clear), a diver's innate navigational skills come up short of what they do on land. In a dark or limited visibility dive, any attempt to navigate without mechanical assistance would result in failure, even if it is a short course. For most divers, even with a compass and depth gauge, going from point A to point B under water is usually successful only when there is a third mechanical device to assist them – the rope connecting point A to point B.

THINGS THAT CAN GET YOU

Sharks

For those of us raised in the "Jaws" era, we know all too well that there are sharks out there just waiting to sample the dive boat smorgasbord. But of the over 300 species of sharks known to exist, fewer than 20 have been known to bite (eat) humans (we probably taste like chicken anyway). I won't spend any time on species identification, because if it's in the water with you and bites you, that's pretty much all you need to know. I find it unlikely that divers will find much comfort in being able to say "well, at least I knew the genus and species of what ate me."

The management for shark bites is the same as any injury that mimics getting attacked by a chain saw. Control for bleeding, treat for shock, use volume expanders (Intravenous Saline, Lactated Ringers, Plasmanate), and get to a definitive care facility. And bring all the parts with you, in case all of them are not still attached.

The jury is still out as to the best way to deal with sharks while you are still in the water. Some advocate staying on the bottom or as close to any structure as possible, but this is a time-limited option. You can try banging on your tank and hope it doesn't sound like a dinner bell, you can try blowing bubbles, or you can just make sure that you always dive with someone who swims slower than you, and get out of there as fast as you can.

Barracuda

This is another large fish with large teeth. Fortunately, attacks by these fish are very rare. They usually tend to be attracted by bright shiny objects (shiny regulators and such), or by freshly speared fish. Their large teeth can inflict nasty wounds, but they usually tend to be of the slash type. They can, however, produce significant hemorrhage. The management of these wounds is to prevent further hemorrhage and shock.

Stingrays

These bottom-dwelling sea creatures are very docile and non-aggressive. You have to go to them to get zapped. They have an exposed spineous process located at the base of their tail that they swipe at the individual who has managed to step on them. This spineous process contains a protein that once injected into the individual can produce heart block, respiratory depression, and blood pressure instability (hypotension). Treatment is supportive, and should include transport to a definitive care facility to manage seizures and cardiorespiratory depression. The onset of life threatening complications can occur as soon as 15-30 minutes.

Moray Eels

Morays tend to be timid bottom dwellers that tend to hide in rocky crevasses, and will do just about anything to avoid human contact. So if you get bitten by one, you probably deserve it.

Invertebrates

These injuries are the result of direct contact with organisms like jellyfish, coral, mollusks, fireworms, and urchins. The reaction to these injuries can range from local irritation to total

body failure, including respiratory arrest, cardiac arrest, hypotension, and paralysis. The treatment can range from local wound care to cardio-pulmonary resuscitation. The best treatment begins with avoidance.

NEAR DROWNING

Near Drowning definition: initial survival following immersion in liquid; the victim may die more than 24 hours later, i.e., from ARDS (acute respiratory distress syndrome). Near drowning victims must have suffocated first, and were subsequently resuscitated.

There are two scenarios that are associated with near drowning. The first is near drowning without aspiration. In this situation, laryngospasm prevents the entry of water into the thorax. Treatment of these patients evolves around the return of normal respirations. If normal respirations can be restored, these patients have an excellent chance of 100% recovery, providing that tissue hypoxia was not long enough to cause permanent damage.

The second is near drowning with aspiration. These patients have allowed the surrounding medium to pass through their vocal cords and into the alveolar spaces. This leads to an immediate hypoxic state with an associated metabolic acidosis. Other changes include fluid and electrolye changes, as well as direct tissue trauma. These immediate changes also affect other organ systems, including cardiovascular, renal, and central nervous system. A late consequence of aspiration near drowning is latent pulmonary infection. Unless the patient was diving in a sterile medium, and was able to selectively eliminate normal flora of the upper respiratory tract, they will develop a fulminating pneumonia. These patients need immediate hospitalization, with the application of antibiotics and corticosteriods as part of their care plan.

DIVING IN POLLUTED WATERS

I can't think of any reason that you would want to sport dive in polluted waters, but there is the occasion that may require rescue workers to do rescue or recovery work in waters that people would not normally dive in. The primary risk to these divers is exposure to coliform bacteria. Some of these organisms can produce exotoxins that can complicate patient care, making the patient more violently ill than just exposure to the pathogen itself. Numerous other organisms can be found, including Salmonella, Shigella, Pseudomonas, Vibrio, as well as parasites. Should any of your divers develop an illness consistent with the above diseases, continue to monitor the other divers for similar symptoms, and collect cultures from the ill divers to attempt to isolate the organism, and treat with appropriate antibiotics.

CHAPTER 7

DECOMPRESSION SICKNESS: CLINICAL MANIFESTATIONS AND PATHOGENESIS

INTRODUCTION

There are as many presentations of DCS as there are people diving. Flexibility in your diagnostic skills and a very high index of suspicion, when used in conjunction with the history and mechanism of injury, are still the mainstay of correct diagnosis. One of the biggest factors playing in the favor of the practitioner is that if you presume and treat for DCS, you won't be wrong. The most common thing you may confirm by overtreatment is that the patient probably did not have a decompression injury, and that further treatment is not indicated. If you fail to treat a true injury, however, the time lost will not be recoverable, and long-term sequelae may be the direct result of your failure to treat in an expeditious fashion.

TYPE I DECOMPRESSION SICKNESS (TYPE I DCS)

Type I DCS symptoms are those symptoms that only involve joint, skin, or lymph node pain and swelling. A good rule of thumb: if the pain is distal to the axilla, or distal to the groin, it can be safely treated as a Type I pain. If the pain is proximal to those points, it should be treated as a Type II DCS.

Musculoskeletal Pain Only

The most common symptom of DCS is joint pain. The pain can range from minimal to severe, from inconvenient to incapacitating. Even severe pain, as long as it is not accompanied by neurological deficits, is a Type I DCS.

Classic Presentation of Type I DCS (not to be confused with real life).

The pain begins gradually, and is initially difficult to localize. Over a period of time, it usually increases in intensity, and the patient may become much better at localizing the pain. The pain may or may not be changed with motion, but the patient is usually auto splinting.

The pain is consistently present at rest, unlike a traumatic (non DCS) injury. Occasionally, Type I DCS may be present when the diver reaches the surface, but it more classically presents with a delayed onset.

Cutaneous (Skin) Manifestations

The most common skin manifestation is itching. Itching alone generally does not require recompression, and will pass without sequelae.

Cutis Marmorata, a mottling of the skin, does require recompression. It usually starts with an intense itch and gradually progresses to a patchy red or blue rash that can mimic hives or meningococcus.

Lymphatic Symptoms

Lymphatic obstruction can occur, causing very well localized pain and swelling. Pressing will usually provide prompt relief of subjective symptoms, but the swelling may persist for some time. Reassurance, along with a single recompression, will usually suffice.

TYPE II DECOMPRESSION SICKNESS (TYPE II DCS)

Any symptom that does not go distal to either the axilla or the inguinal ligament can be presumed to be a Type II DCS. Even if it seems like it is just Type I of the skin of the trunk, you should give serious consideration to treating it as a Type II. You will never be faulted for treating a Type I as a Type II. You will be faulted for treating a Type II DCS as a Type I DCS.

Symptoms of Type II DCS may not be obvious to the diver at the onset. The diver may disregard the symptoms until they become more severe, with complaints of ataxia, hearing loss, or difficulty with urination. Often, a family member may bring the patient in because of a personality change. It is for this reason that you must also anticipate a worsening of symptoms prior to treatment.

Neurological Symptoms

These symptoms can strike at any level of the nervous system. It just depends where the bubble is. Symptoms can range from numbness and tingling, to muscle weakness and paralysis, to status epilepticus. Even *minor* neurological symptoms are DCS Type II.

Pulmonary Symptoms

If diffuse pulmonary intravascular bubbling occurs (chokes), the patient will develop symptoms consistent with pulmonary embolis and pulmonary edema. Patient will usually have chest pain, tachypnea, and probably a cough. He may develop fulminate pulmonary edema. Increasing pulmonary congestion will most likely lead to circulatory collapse and death (a bad thing).

Onset of Symptoms (Timeline)

If DCS is the result of shortened or omitted decompression, onset of symptoms is usually rapid, often by the time the diver reaches the surface. If DCS is the result of bad JuJu, symptoms can be delayed for several hours, or at least be ignored by the patient for several hours. Onset of symptoms directly correlates with severity of symptoms, survivability, and sequelae. It is imperative that all subjective symptoms that the patient may have are treated in an expeditious manner to minimize the risk of long term sequelae, secondary to treatment delay.

FACTORS AFFECTING THE LIKELIHOOD OF DECOMPRESSION SICKNESS

Dehydration

A common patient history is, "I had several drinks last night, was too hungover this morning to have anything except coffee (a diuretic) for breakfast, and the seas were rough so I didn't drink anything on the boat. I didn't really feel very well during the surface interval, so I wasn't able to drink much then either." Dehydration is probably the easiest predisposing factor to eliminate as a cause of decompression illness. The actual reason remains unclear, but is probably related to a decrease in tissue perfusion, in association with increased blood viscosity, which would limit gas elimination. A good rule of thumb: If you don't have to urinate on every dive, you probably aren't hydrated enough. This is based on immersion diuresis (the P Phenomenon). This is caused by compression of the extremities by the pressure of the water column that forces peripheral fluid into the central body. Volume detectors in the heart and kidney notice the perceived increase in body fluid, and in an effort to normalize volume again, cause the body to lose fluid by urination. If the divers don't rehydrate themselves shortly after reaching the surface, they can easily become clinically dehydrated, especially if they are going to be doing repetitive dives.

Exercise

Exercise, including exercise done before, during, and after diving, can contribute to DCS. By exercising before diving and exerting yourself while diving, you increase the amount of blood flowing to the muscles and soft tissues, and subsequently increase the amount of nitrogen that the diver will on-gas while at depth. Strenuous exercise after diving can increase the rate of nitrogen diffusion out of the tissues, causing bubble formation. Also, the lactic acid (a by-product of muscle activity) and CO_2 (which acts as an acid while dissolved in the blood) generated from exercising may contribute to bubble formation via subtle pH changes.

Heat and Cold

While it seems that these two environments should have opposite effects on nitrogen on-gassing, they are frequently similar. If you are diving in warm or hot water, you will on-gas more as a result of increased vasodialation and increased blood flow to tissue compartments. In a cold environment, you should theoretically on-gas at a much lower rate. Cold exposure causes cardiovascular changes that decrease the body's ability to both absorb and off-gas nitrogen. If you were to be uniformly cold through the dive, there should be no increase in risk of DCS. The problem stems from the fact that no one likes to dive cold. So the first thing divers do when they get into cold water is to try to warm up. The easiest way to do that is to swim a little harder, i.e., exercise, which increases the blood flow to the muscles, and subsequently rate of tissue on-gassing. Starting a dive warm, i.e., post exercise, and then getting cold can cause an increase in nitrogen load and then a decreased elimination, predisposing you to DCS.

Prior History of DCS

Divers who have a prior history of DCS are at increased risk of a repeat hit. This may be related to physiologic changes that occurred as a result of the previous hit, or because of the physiologic predisposition that individual may already possess that led to the first hit. My policy has been that if the patient had DCS as the result of a dive that exceeded the U.S. Navy Tables (and the dive is based on total bottom time at maximum depth as though it was a square profile, with time subtracted for the ascent and safety stop), then I would sign that person off to dive again, provided the diver paid special attention to predisposing factors and by diving a more conservative profile. If they were bent while diving within the tables, I will not sign them off to return to diving.

Obesity

Good thing for whales and seals, bad thing for scuba divers. Obesity has a protective effect on seals and whales. It should be noted that seals and whales hold their breath and don't use scuba. If your partner has been accused of having the body habitus of the aforementioned mammals, advise him or her to slim down. Adipose tissue can on-gas significantly more nitrogen on an extended dive, increasing the nitrogen reservoir that has to be off-gassed.

Age

As a rule, for older divers, the risk of DCS seems to be greater than for younger divers on any given profile. This may be related to decreased fitness, a reduction in tissue perfusion, vascular turbulence associated with atherosclerosis, or a combination.

Sex

There are several arguments for which sex is at higher risk for getting DCS. One line of thinking is that with a woman's genetic predisposition for a higher percentage of body fat, that females should be more likely to on-gas and subsequently develop DCS. It can also be argued that men are more likely to develop DCS because of their innate risk-taking tendencies, and likelihood to extend depth and bottom times.

Multiple Dives

Multiple dives can both increase and decrease the likelihood of DCS. While frequent diving, such as that done on live aboard dive boats, seems to contribute to DCS, Haldane discovered that newly exposed caisson divers were more likely to develop DCS than those that had extended exposures over a length of time.

While some sort of acclimation or accommodative effect appears to occur with multiple dives, it is possible that hydration and fatigue, in combination with complacency, may contribute to live aboard risks.

There are several factors affecting DCS. The important thing to remember is that even if you do everything right, you can still get bent. Divers must realize that if you get bent, it usually has no reflection on your abilities as a diver. It has everything to do with how your body eliminates nitrogen from your tissues after a dive. In the process of eliminating that nitrogen, sometimes bubbles get formed (more often than we had previously realized), and occasionally those bubbles get trapped in certain tissue spaces before they can be exhaled. Then you get bent. It's that simple. When you get bent, you should get treated – sooner rather than later. The greatest likelihood for a complete recovery comes from early treatment. The bad part about getting bent is that for every bubble that you know about, there may be more that you don't know about. So there is no such thing as just a little bent. If you think that you are bent, tell someone who will get you evaluated to determine if you are in fact suffering from DCS. The first thing you want to do is to start breathing oxygen. If that makes the symptoms go away, you're bent. You will need recompression therapy. It can't be assumed that breathing oxygen for a few minutes or a half hour will resolve your problems. You need to be evaluated by someone trained in hyperbaric medicine. And then if indicated, they will begin recompression therapy.

PATHOGENESIS OF DCS

Most of the trauma that occurs can be blamed on direct insult brought on by the physical effects of the bubble. Intravascular and subcutaneous bubbles not only cause direct trauma, but they also activate numerous vasoactive substances within the body. As the bubbles traumatize the blood vessel wall, they can allow for platelet activation and adhesion, further compromising blood flow. In cases of significant vascular trauma, consumption of platelets and clotting factors could, theoretically, lead to disseminated intravascular coagulation (DIC).

DYSBARIC OSTEONECROSIS

Dysbaric Osteonecrosis is localized areas of bone death, secondary to exposure to the hyperbaric environment of diving. As long as the necrosis is in the shaft of the bone (unless the lesion is large enough to cause a fracture), it will go undetected unless an x-ray is taken for some reason. If the necrosis occurs along an articular surface (a joint), then the patient will develop symptoms of arthritis, which can be serious if the joint is the hip or shoulder. When the hit occurs, there is no pain, so the patient is unaware of the damage as it occurs. Subsequently, it is difficult to anticipate and then treat for this disorder. Anyone, with even minor DCS symptoms, should be considered at risk for a bone hit, making treatment even of minor symptoms of paramount importance.

ULTRASONIC BUBBLE DETECTION

With the advent of ultrasonography, we can now detect the formation of bubbles as a result of diving. The problem is, it now seems that a significant number of dives result in bubble formation. So it is now clear that just the formation of bubbles does not imply that the diver will develop DCS. There is now speculation that some bubble formation may even be helpful. It is felt that bubbles may be able to transport a larger quantity of gas than could be transported in suspension. One concern that exists with using ultrasonic is the effect that the ultrasonic sound waves may have directly on the bubbles themselves. But whether the sound waves can change the morphology of the bubbles is still under investigation.

DIFFERENTIAL DIAGNOSIS OF DIVING INJURIES

On the Surface

While it would seem difficult to get DCS while you are still on the boat, it needs to be a part of your history so that you can rule out a non-diving insult that resulted in your patient's pain, i.e., falling on the boat and injuring a hip before the dive.

During Descent

Barotrauma is the most likely injury to occur during descent. Symptoms can range anywhere from pain to vertigo and incapacitation. Oxygen toxicity is a possibility but is unlikely, even if the patient is breathing 100% oxygen.

On the Bottom

Barotrauma is unlikely once the diver reaches the bottom, unless it is delayed as in the case of a round window rupture. Trauma may result from contact with objects on the bottom or marine life.

On Ascent

Alternobaric vertigo may present on ascent. Carbon monoxide poisoning will occur as the divers get closer to the surface. POIS and DCS can also become apparent during the ascent.

After Reaching the Surface

Symptoms that occur after arriving at the surface should be divided into two groups: those that occur within the first 10 minutes and those that occur after 10 minutes. Symptoms that occur in the first 10 minutes are generally more severe than those occurring later. Symptoms occurring in the first 10 minutes include AGE, POIS, hypoxia, and carbon monoxide poisoning. After 10 minutes, the symptoms are most likely to be DCS Type I or II.

THE PATIENT EXAM

The neurological exam is the foundation of evaluating a patient for decompression sickness. A normal neurological exam excludes the diagnosis of Type II DCS. This exam must be done thoroughly and competently to avoid misdiagnosis and poor patient outcome. It is of course presumed that life-threatening emergencies will be taken care of prior to the neurological exam.

CRANIAL NERVE EXAM

I Olfactory

Checks for ability to smell. Check for patency of nasal canal and then check for the patient's ability to detect common odors.

II Optic

Checks for ability to see. Check visual acuity. Check visual fields by facing the patient and moving an object equidistant between you and the patient through all of the eye fields. If you can see it and the patient can't, think problem.

III Oculomotor

Checks for motion of eye. Check the range of motion of the eye. At rest, the eye will look down and out with deficit. With motion, the eye can't look up, down, or in. The pupil is dilated, and the upper lid droops. Accommodation and consensual reflex is paralyzed. Incomplete lesions are possible.

IV Trochlear

Trochlear nerve innervates superior oblique muscle. Patient has difficulty turning the eye down and out.

V Trigeminal

Sensory to the face. Check all three branches. Motor to temporalis and masseter muscle.

VI Abducent

Innervates lateral rectus muscle. Patient can't move eye laterally. At rest, eye moves medially.

VII Facial

Innervates muscles of facial expression. Have patient raise eyebrows, squint, smile, puff out cheeks, show teeth. Taste to anterior 2/3 of tongue.

VIII Acoustic (Vestibulocochlear)
Hearing and balance. Check hearing. Weber and Rinne. Observe patients as they ambulate into office.

IX Glossopharyngeal
Sensory to back 1/3 of tongue. Check gag.

X Vagus
It's everywhere. Check for soft palate elevation during gag.

XI Spinal Accessory
Innervates SCM and trapezius. Have patient shrug shoulders, and turn head against resistance.

XII Hypoglossal
Motor to intrinsic muscles of the tongue. Have patients stick out their tongue, check for deviation.

NEUROLOGICAL ASSESSMENT

1. Mental Status
   Is the patient conscious, alert, and oriented to time and place? What is their mood?
2. Signs of Meningeal Irritation
   Nuchal Rigidity, Kernigs, and Brudzinski sign
3. Coordination/Balance
   Gait, heel-toe walk, Rhomberg, Stewart Holmes sign, rapid alternating movements, finger to own nose
4. Fine Motor Coordination
   Finger-nose-finger, heel-shin
5. Gross Motor
   Muscle atrophy, muscle tone (resistance to passive movement, cog wheel rigidity, spastisity, paralysis), strength (push, pull all major muscle groups [bicep/tricep, deltiod/lats, quads/hams, dorsi/plantarflex foot])
6. DTR
   Biceps, brachioradialis, triceps, patellar, achilles
7. Superficial Reflex. Babinski, upper, mid, lower abdominal, cremasteric, anal wink.
8. Sensory Testing
   Pain (sharp/dull), temperature, fine touch (cotton), proprioception, vibration, stereognosis, 2-point discrimination, graphesthesia

CHAPTER 8

PREVENTION OF DECOMPRESSION SICKNESS

HISTORICAL EVOLUTION OF DECOMPRESSION PROCEDURES AND CONCEPTS

The development of the deepsea diving suit in 1837 by Augustus Siebe and the pressurized caisson in 1841 by Triger opened up the compressed-gas environment to humans, allowing for the development of decompression sickness. The first cases of DCS came from Triger in 1841, when he reported about a caisson worker who developed severe limb pain after working at a depth of 65 fsw for 7 hours.

Some of the early hypotheses of DCS were:

- Exhaustion and cold caused reflex damage to the spinal cord.
- Compressed gas caused frictional electricity.
- Toxemia caused by excessive catabolism produced DCS.
- Expansion of the GI tract on ascent caused the problem.
- Blood forced internally by pressure caused congestion, stasis, and ischemic changes in internal organs, and that the rapid return of blood to the periphery during ascent led to anemia of the internal organs.

The first implication that gas bubbles might be responsible for DCS came in 1854, when Pol and Watelle discovered a consistent link between the amount of pressure, the duration of the exposure, the rapidity of decompression, and the onset of symptoms. They also noticed that some of the workers experienced relief of their symptoms with recompression. In 1878, Paul Bert demonstrated that the bubbles in tissue consisted primarily of nitrogen. He recommended recompression and decompression with oxygen, but did not stipulate any specific rates.

In 1900, Heller, Mager, and Von Schrotter began to develop actual decompression tables that would allow workers to spend extended periods of time at depth and come to the surface with stops at specific depths for precalculated times to allow for the elimination of saturated nitrogen. Their formula was based on the presumption that the body was of uniform architecture and density, with a nitrogen solubility close to that of blood. Their calculations revealed a tissue half-time of 9.7 minutes. They proposed that with a tissue half-time of this rate, decompression of 20 minutes per atm should prevent DCS. This is an adequate amount for dives of short duration, even to 5 or 6 ATA, but inaccurate for longer bottom times.

In 1908, Boycott, Damant, and Haldane noticed that the incidence of DCS in tunnel workers increased as exposure time approached 5 hours, but that the incidence remained constant when exposure times exceeded 5 hours. They had been doing simultaneous studies on goats, and

had found that they could reproduce similar results, except that a goat's exposure maximum was only about 3 hours. Since they figured that goats were 3/5 the size of humans, different tissues must have different half-times, and the human body should not be considered to be just one compartment, but a number of different compartments with different half-times. They speculated that the human body was composed of several compartments with half-times ranging from 5, 10, 20, 40, and 75 minutes.

The Royal Navy adopted the Haldane Tables in 1908. By this time, he had sufficient data to devise tables that went as deep as 200 fsw for as long as 30 minutes. But they were less than optimal for dives greater than about 10 minutes because of the linear ascent calculations being made. By the mid 1950s, it was determined that an additional tissue half-time compartment was needed. The tissue half-times were now decided at 5, 10, 20, 40, 80, and 120 minutes.

TISSUE COMPARTMENTS

From an anatomic perspective, there are three types of body tissue. These are muscle, connective (which includes blood), and nervous tissue. Everything in our body is made up of different percentages of these tissues. Because of the varying amounts of these substrates in different parts of the body, the body absorption and elimination of nitrogen will subsequently vary.

TISSUE HALF-TIMES

As you dive and breathe gas at pressure, you will increase the ambient partial pressure of nitrogen. It takes time to allow the amount of nitrogen to on-gas sufficiently to equilibrate and reach a steady state. Different compartments on-gas nitrogen at different rates; subsequently, different compartments will have different amounts of saturated nitrogen in them for the same amount of time at any given exposure. This difference in the rate of on-gassing is the half time. A tissue half-time is the amount time it take for the inert gas to move into and out of the body. A 5-minute half-time means that the compartment fills to 50% capacity with inert gas in 5 minutes, a 10-minute compartment fills to 50% capacity in 10 minutes, and so on. It does not fill the remaining 50% in one additional half-time. It will continue to saturate at a rate of 50% of the remaining tissue at a constant rate. After 6 half-times, the compartment is considered to be fully saturated.

The Haldane models figure off-gassing at the same half-time rate as on-gassing, even though there are several factors that can affect the rate of off-gassing. The most obvious factor is that as the level of the inert gas is decreased, the osmotic force driving the gas out continues to decrease. Another factor is that the half-time is based on blood flow to the area and the dissolvability of gas into that tissue. Since there are known hemodynamic changes that are the result of diving, the blood flow will change from that of the diving environment to that of the surface environment. In general, the body areas that have the best blood supplies are the faster tissues, such as lung and abdominal organs, and the slow tissues tend to be bone, fat, and, not surprisingly, fatty bone marrow. This may explain why these parts tend to be the ones where DCS is found.

Another factor that different tissue compartments bring into play is that not all of your compartments are decompressing at the same time. If you do a deep dive that does not saturate your longer compartments, as you ascend you will start to eliminate nitrogen from your shorter tissue compartments. However, your longer half-time compartments will continue to absorb nitrogen until you are at a sufficient partial pressure that allows them to also begin to off-gas. That is one reason that recreational divers can get bent while diving with a computer, when they are outside the tables. Some computers may give you credit for off-gassing (based on what compartments it is using) when you are still on-gassing.

REPETITIVE DIVE THEORY

A repetitive dive is defined as any dive conducted after 10 minutes of reaching the surface from a previous dive, and within 12 hours of the diver's previous dive. Any dive that occurs within 10 minutes of the previous dive is considered to be the same dive. On any dive that is made within 12 hours of the previous dive, the diver is credited with still having nitrogen remaining in his body. The repetitive nitrogen tables are based on the body's 120-minute tissues. In short, the residual nitrogen tables tell you to what equivalent depth the diver's 120-minute tissues have desaturated. These depths range from 26 fsw for an A group designator, to a depth of 50 fsw for a Z designator. Failure to add these additional nitrogen times to subsequent dives can lead to DCS.

SURFACE DECOMPRESSION THEORY

Surface decompression is a procedure that allows supersaturation of the body's tissues with nitrogen for a short period of time while still avoiding DCS. It allows a diver to omit a large portion of his in-water decompression, surface, and be placed in a dry surface chamber, and recompression in a chamber to finish out his decompression obligation out of the water. Surface decompression is generally desirable at all times, but is especially useful in foul weather, cold water, in the presence of hostile marine life, or when you need to leave an area in a hurry. The preferred decompression is done with oxygen at a depth of 40 fsw. A typical decompression will have the diver do abbreviated stops at 60 fsw, 50 fsw, 40 fsw, and 30 fsw (if indicated), ascending to the surface, and then descending to 40 fsw in a chamber while breathing oxygen. For purposes of comparison, we will look at 2 identical dives, 150 fsw for 40 minutes while breathing air. For standard in-water air decompression, the diver's first stop is at 30 fsw for 5 minutes, then ascend to 20 fsw for 19 minutes, and then finally ascend to 10 fsw for 33 minutes, giving you a total time at stops of 57 minutes. With surface decompression with oxygen (Sur D O_2), the diver spends 3 minutes at 40 fsw, and then 6 minutes at 30 fsw. He then ascends and is placed in a recompression chamber for an additional 27 minutes while breathing oxygen. His total "at stop" decompression time is only 36 minutes, and his in-water time is reduced by 48 minutes.

HIGHER ALTITUDE THAN SEA LEVEL DECOMPRESSION AND FLYING AFTER DIVING

Hyperbaric work that is conducted at altitudes above sea level require longer decompression times than do hyperbaric exposures done at sea level. Additionally, if you were to try to go to altitude following a dive, you would need to recompute your maximum depth to determine if you had done an adequate decompression dive. For example, if you were going to go to an elevation of 18,000 feet msl (mean sea level), you would have to recalculate your dive as though you had made your dive 33 fsw deeper than it actually had been to compensate for the decreased air pressure at your final stop. The following table demonstrates the differences of air pressure at various altitudes. Notice that to achieve the same pressure change that you would experience in ascending from 33 fsw to sea level requires that you ascend from sea level to 18,000 feet msl. It should also be noted that at 36,000 feet msl, there is insufficient pressure to drive oxygen across the alveolus. In order to breathe at that altitude, you must breathe gas that is under pressure.

To estimate the actual depth that you are diving at based on sea level equivalency, use the following equation:

Sea Level equivalent = 1/pressure (ATA [see chart next page]) x Depth of Dive.

If you wanted to dive to a depth of 125 feet while vacationing in the mountains at an altitude of 8500 feet msl, the fsw would be:

1/0.729 (ATA at 8,500 feet msl) x 125 (depth of dive) = 171 fsw.

As this equation demonstrates, altitude can significantly affect your dive profiles. Some considerations must be made if you plan to dive at sea level and then fly. Most modern jet aircraft are pressurized to about 8,500 feet. So once you are at altitude, that is the equivalent depth your body is decompressing.

Altitude (Feet)	Pressure (mm/Hg)	Pressure (ATA)
0	760	1.000
500	746	0.982
1,000	733	0.964
1,500	712	0.947
2,000	707	0.930
2,500	694	0.913
3,000	681	0.896
3,500	669	0.880
4,000	656	0.864
4,500	644	0.848
5,000	632	0.832
5,500	621	0.817
6,000	609	0.801
6,500	598	0.786
7,000	586	0.772
7,500	575	0.757
8,000	565	0.743
8,500	554	0.729
9,000	543	0.715
9,500	533	0.701
10,000	523	0.688
10,500	513	0.676
11,000	503	0.662
11,500	493	0.649
12,000	483	0.636
18,000	380	0.500

Figure 1 — Relationship Between Altitude and Pressure

Industrywide, the accepted no-fly time after diving (a no-decompression dive) is 12 hours after reaching the surface. A general recommendation is made not to go above 2,300 feet msl during the same time period. Previous experiments have clearly demonstrated the likelihood of DCS when combined with altitude after diving. In an experiment done by Duffner and Kiessling (1960), humans were pressed to 90 fsw for 30 minutes, received adequate decompression, and after a surface interval of 15 minutes, they were subjected to an altitude of 18,000 feet. 55% of the divers suffered DCS. Numerous other experiments have shown that there is an overwhelming likelihood of DCS in flying after diving in any dive profile that puts the diver into a repetitive dive group designation of E or greater.

BUBBLES, BUBBLES, BUBBLES

Bubbles form after more dives than a lot of divers are willing to admit. It is true that while you probably don't form bubbles on every dive, they are formed on a significant number of dives, including those that are well within the no-decompression limits. And while bubbles frequently form during dives, they only rarely create problems. Most bubbles form on the venous side of the circulation due to the lower pressure of the venous system, and are subsequently transported to the pulmonary circulation where they are simply exhaled. If you develop an excessive number of bubbles due to inadequate decompression, the pulmonary circulation will become overwhelmed with the bubbles, allowing some of them to return to the heart and subsequent systemic circulation. Once in the arterial circulation, the bubbles continue until the diameter of the arteriole or capillary becomes smaller than the diameter of the bubble. Once this occurs, blood flow distal to the bubble stops, causing hypoxia and eventual anoxia, creating the symptoms of DCS. The other physiological phenomenon that occurs as the bubble continues to injure the blood vessel is damage to the endothelial wall of the blood vessel, causing release of numerous vasoactive amines (histamine, interlukin, Hageman factor). This causes the clotting cascade to be activated, and as clotting begins to occur, even more blood vessels will become compromised, causing even more tissue hypoxia and anoxia. This in turn causes more inflammatory mediators to be released, which will increase local swelling and thereby contribute still more to the rapidly deteriorating situation.

Another way for bubbles to gain access to the systemic or arterial circulation is through a patent foramen ovale (PFO). This is a normally occurring hole in the atrial walls that may fail to close at birth in some individuals. While the pressure is normally higher in the left atrium, thus preventing blood (or bubbles) from moving from the right atrium to the left atrium, there are physiologic conditions that can increase right-sided pressure enough to overcome this pressure differential. The most common thing that divers do to overcome this pressure gradient is to Valsalva (holding your breath and making your abdominal muscles as hard as possible) to equalize their ears. This can raise the right atrial pressure to effectively open a patent foramen ovale and allow bubbles to become systemic, if that malformation is present. This is why this medical condition is incompatible with diving, even though it is found in anywhere from 10-30% of the adult population.

DIVING WITH A COMPUTER

Computers have become an everyday fact of diving. It seems that everyone is wearing them. Unfortunately, everyone seems to use their computer instead of paying attention to their dive profile or to the dive tables. Some divers are under the mistaken impression that the computers are able to measure the nitrogen levels in their body. Instead, they estimate the amount of nitrogen that would be absorbed and off-gassed, based on the tissue half-times that were devised by Haldane. The number of compartments that are measured by the computer worn by the diver vary from computer manufacturer to manufacturer, with some being more conservative and others being less conservative. The computers calculate how much nitrogen you started with in your body, the pressure around you at depth, the length of time that you spent at each depth, speed of gas uptake, and elimination in each compartment. It also calculates the maximum amount of nitrogen that each compartment is thought to tolerate upon ascent before desaturation (bubbling) occurs. It must be remembered that computers are based on models. As a rule, models are based on ideals. They tend to not take into account the wide variety of divers who are now diving, and the things that these divers tend to do between dives. Subsequently, divers seem to have a bad habit of developing DCS while they are allowing computers to be responsible for their nitrogen load, and not being responsible for themselves.

MEDICAL CONDITIONS THAT PRECLUDE DIVING

In a lot of respects, scuba diving is not much different than piloting an aircraft. In both instances, you are exposed to environmental pressures that are much different than are found on *terra firma*. Sensory inputs and subsequent perception of those stimuli can vary tremendously – more than would otherwise occur on the ground. And perhaps most importantly, if you have a medical problem, either in the cockpit or in the water, you are a long way from medical assistance. While some medical conditions can be a mere inconvenience on the ground, in an unforgiving environment, they can be fatal. Below are listed most medical conditions that have been agreed on by most hyperbaric-trained physicians as diseases that should raise eyebrows if you have (or know someone who has) one of these conditions and wants to dive. Hopefully, the explanations following the conditions will demonstrate the risks involved, and the reasons that we will not approve them for diving, and not try paint the medical profession as trying to keep people from doing a sport that we love.

ABSOLUTE DISQUALIFYING MEDICAL CONDITIONS FOR DIVING

Asthma (except childhood asthma that has not recurred since onset of adolescence)
The main problem in asthmatics is that with constriction of the breathing passages, it becomes more difficult to exhale than it is to inhale. If this were to happen at depth, it would be extremely difficult to get this diver to the

surface without inducing pulmonary overinflation. Because of the significant improvement in asthma management with modern medications, a study is presently underway by Lin Weaver M.D. et al. at LDS hospital, Salt Lake City, UT, to determine whether well-controlled asthmatics can safely dive.

Chronic Cystic, Obstructive or Restrictive Pulmonary Disease, or Chronic Bronchitis
Many of the reasons are the same as with asthmatics, with the added risk of pneumothorax.

Any History of Spontaneous Pneumothorax
If you had a spontaneous pneumothorax in the past, you are at risk to have one in the future. If you have a pneumothorax at depth, it will only expand as you ascend to the surface. Individuals who have had a traumatic pneumothorax can dive in the future.

Seizure Disorder or Frequent Syncope (fainting)
Either condition would be potentially nonsurvivable at depth.

Insulin-Dependent Diabetes
Because of the changes in metabolism and blood sugar requirements by cells at depth, there is an increased risk of critically low blood sugar, causing seizures, coma, and death.

Middle Ear Surgery with Placement of Prosthesis
These patients are at increased risk of inner ear trauma. While symptomatically, for the patient may be merely inconvenienced by this injury, the real dilemma comes in evaluating this patient after the fact. Specifically, are the new symptoms as the result of a problem with prior surgery, or the result of decompression sickness, requiring recompression therapy.

Sickle Cell Disease or Trait
Patients with sickle cell disease or trait are at risk of developing sickle crisis with any low oxygen insult. The risk occurs when divers ascend and begin to off-gas nitrogen. The presence of nitrogen bubbles in the venous circulation (and every diver forms them on every dive) can predispose these individuals to a sickle crisis. This will increase the likelihood of decompression sickness, which will in turn increase the severity of sickling.

Detached Retina
The area of detachment is a good place for nitrogen bubble formation, which can worsen the detachment, leading to blindness. It also presents a diagnostic dilemma regarding the location of the bubble. Is it really in the eye itself, or could this be a lesion in the brain causing similar symptoms?

Patent Foramen Ovale/Atrial Septal Defect, Ventricular Septal Defect
The infamous "hole in the heart." As many as 10% of the population has this problem. The risk is that as you ascend to the surface, bubbles can get from the right side of the heart, and instead of being exhaled via the lungs, they get forced through the hole into the left side of the heart and then are put into the atrial circulation, potentially lodging in distant body sites.

Recurrent Labrynthitis
If you have frequent (or infrequent) dizzy spells, and you have one after dive, we have no way of knowing if it is the result of DCS or your chronic disease.

Disk Disease with Neurological Deficit
If you develop numbness or tingling after a dive, there is no way to determine if the symptoms are the result of old disease or new bubbles.

Symptomatic Bradycardic Arrythmias (slow heart rate)
One phenomenon that occurs with diving is the decrease in heart rate with the increase in pressure. So as you go deeper, your heart rate gets slower. If you already have a condition that causes your heart to beat slowly, it can get much worse when diving.

Pregnancy
Animal studies have shown that bubble formation in the placenta is almost guaranteed, even

with moderate diving. At the very least, bubble formation can cause deformity or fetal death, along with a significant risk of arterial bubble formation in the mother.

RELATIVE DISQUALIFYING MEDICAL CONDITIONS FOR DIVING
(we are at least willing to talk about it)

Closed Head or Spinal Cord Injury with Sequelae
Again, the difficulty comes from trying to determine if any symptoms present after a dive are the result of bubble formation or from the previous injury. In addition, if there was bubble formation in an area that has no sensation or motor function as a result of a previous injury, it cannot be detected. This said, I realize that there are a number of spinal-cord-injured scuba divers enjoying the sport. I am certainly not trying to say that they should not dive, only that they must realize that there are increased risks associated with their diving that they must be aware of.

Chronic Perforation of Tympanic Membrane
The risk here is not from decompression injury. Since the TM is perforated, there is no risk of a pressure-related injury. The risk comes from infection from the water entering the middle ear. These divers can be very entertaining, since they have the ability to pinch off their nose, blow out through their nose (Valsalva), and get bubbles to vent out of the ear with the perforation.

Chronic Otitis Media
These individuals are at a much higher risk of not being able to equalize their ears because of the chronic infection. Not only are they at risk for pain on descent, if they do manage to get to depth, it may be difficult to get them to the surface without an overpressurization injury to the middle ear. And from a diagnostic standpoint, it is very difficult to tell the difference between this and a brain hit. Some of these people can be managed by decongestants and nasal steroids to allow diving.

Chronic Sinusitis
Similar to otitis media,with similar treatment.

Angina Pectoris
The major consideration with angina is that many dive areas are not very closely associated with hospitals that can treat an acute heart attack, should your angina progress while diving. Also, if you develop chest pain while on a commercial boat dive and abort your dive early, the boat may be unable to leave the dive site expeditiously because of the other divers in the water. This could lead to a significant delay in treatment.

Congestive Heart Failure
Fortunately, people with congestive heart failure (CHF) don't tend to be very athletic or dive. The medications used to control CHF have an unpredictable response in cardiac tissue at depth.

Claustrophobia
Most of these people discover early in the training period that diving is not for them.

Chronic Renal Disease
Because of the pressure exerted on the extremities while diving, this forces fluid in the extremities to the central body. This, in turn, puts increase fluid load on the kidneys (that's why you feel like you need to urinate every time you dive). If your kidneys can't handle this sudden extra load of fluid, it can end up getting shunted to the lungs, causing pulmonary edema.

Heavy Smoking
Heavy smokers are at increased risk of air trapping and spontaneous pneumothorax, which occurs at depth and would make it difficult at best to bring the diver back to the surface.

Migraine Headache
Again, it is a diagnostic dilemma. If a diver presents to the surface with a "migraine," it is difficult to determine if it is a recurring symptom or the first sign of decompression sickness.

Hernias
If a small gas bubble was trapped in a hernia, it would cause severe problems on ascent. The risk is extremely low, however, and many people do dive with hernias. Once fixed, there are no problems diving.

Recent Orthopedic Injury
Because of the alteration in blood flow around a healing fracture, the diver is at a much greater risk of developing DCS at the site of the fracture. This may lead to a condition called dysbaric osteonecrosis, which can lead to premature thinning of the bone and recurrent fractures that may not heal at a later time. Usually, divers can be released to return to diving six months after the fracture, providing healing was normal.

Recent Surgery
Because of alterations in blood flow to the repaired areas, it is generally recommended that you refrain from diving for six months after surgery to minimize the risk of DCS in that area.

Radiation Therapy
(absolute if it involves the lung)
Because of alterations in blood flow through irradiated tissue, this area is at increased risk of DCS. It may also contribute to systemic bubble development. Chest radiation increases the risk of spontaneous pneumothorax.

Mitral Valve Prolapse
(absolute if it involves congestive heart failure or arrythmia)
The prolapsing valve contributes to turbulence in blood flow, which in turn increases the risk of bubble formation. Any bubble formation from the mitral valve goes into the arterial circulation, with potentially devastating results.

Wolfe-Parkinson-White Arrythmia
A cardiac arrythmia. May cause a loss of consciousness if it occurs at depth.

Possibility of Being Pregnant
If you think you are pregnant, you shouldn't dive until you are sure that you aren't. I know of no studies that show a link between diving and getting pregnant.

Menstruation
Studies have shown that you are more likely to develop DCS if you dive while menstruating. Most likely related to fluid balance and blood viscosity, but may be hormonally related.

These are the generally accepted exclusions for diving. I will be the first to admit that I have gone on diving trips with people who have told me that they have some of the above conditions. Since the medical control of diving is nowhere near as stringent as it is to fly, many divers get away with pushing their luck, diving with these conditions. Certainly, as a hyperbaric physician, you have the power to sign off on any dive physical. But in today's litigenous society, you can be held responsible for any bad outcome that might occur as the result of a problem from pathology that is a published contraindication to diving.

CHAPTER 9

THE MEDICAL EVOLUTION OF BUBBLE RESOLUTION

INTRODUCTION

The goal of recompression therapy has always been to improve the outcome of those individuals stricken with decompression sickness. The problem was that no one initially knew why we got bent in the first place. Even once we figured out that nitrogen was the culprit, it took over 100 years to come up with treatment tables that were effective in both mild and severe cases, and minimized the risk of making the patient worse instead of better.

THE THREE PRIMARY OBJECTIVES OF RECOMPRESSION THERAPY

1. To reduce the size of the bubble, subsequently eliminating the mechanical trauma and vascular impediment, and promoting tissue reperfusion and reoxygenation.
2. To promote the resolution of the gas phase, so that symptoms will not appear during the subsequent ascent.
3. To avoid repeat bubble formation in the previously compromised tissue, so that recurring or reperfusion injury will not appear during subsequent decompression as the patient ascends to the surface.

HOW DEEP DO WE GO?

There are four basic hypotheses on how deep we should go with an injured diver's initial descent. Modern hyperbaric theory has evolved from these initial conjectures:

1. Compress to the depth of relief. For a straight air recompression, this technique has the advantage of minimizing the uptake of additional nitrogen during the treatment, making the subsequent decompression easier. The disadvantages come from the failure to maximally decrease the bubble diameter and resultant longer resolution time of distally hypoperfused tissues. In addition, if residual tissue supersaturation is greater than the depth of the initial recompression, the bubble morphology can continue to grow, causing subsequent reappearance of pathology at depth.
2. Compress to the depth of the previous dive. This procedure has the advantage that no tissue gas phase could continue to grow at the decompression depth, and that, on air recompression, the gas phase will be initially smaller and will resolve in a shorter time than in option 1 (assuming that relief is achieved before maximum depth).

3. Compress to the depth of relief, plus some arbitrary amount (i.e., 1 atm). This treatment plan takes advantage of some of the better points of 1 and 2 above.
4. Compress to some standard depth (165 fsw) which is likely to bring about resolution of symptoms in nearly all diving cases. This procedure has the capacity of providing a standard approach in delivering patient care. The trade-off with options 1 and 3 depends on the relationship between the depth of the preceding dive and the depth of relief to the standard depth.

EARLY RECOMPRESSION PROCEDURES

As early as 1854, cases of decompression sickness were noted to improve with recompression therapy. Yet, in spite of this observation, most cases of decompression sickness were treated with strict medical management which consisted of primarily morphine, atropine, and ergots. Even though studies were published showing the effectiveness of recompression therapy in 1863 (Foley), and that it was used successfully during the construction of the Brooklyn Bridge (which had a recompression chamber on site), it did not become an accepted therapy until 1896, during the construction of the Blackwall Tunnel in England. At this site, a moderate degree of success was achieved by recompressing the worker to the depth of relief or depth of the caisson, followed by a return to the surface over a 45-minute period. At about the same time, construction workers on the New York Hudson River Tunnel were experiencing the same degree of success with similar treatment protocols.

By 1909, during construction of the Pennsylvania Railroad tunnels under New York's East River, recompression therapy had taken on the standard practice of recompressing the worker to the depth of work the diver had emerged from. He was kept at that depth for a "brief" period of time, then decompressed to 34 fsw at a calculated rate. This is calculated as 2 times the max depth (converted into psig) which will equal the minutes of ascent to 34 fsw. Since 1 psi equals 2.25 fsw, a trip from 100 fsw to 34 fsw would take about 30 minutes. The ascent from 34 fsw to the surface was described as "a slow bleed to the surface" (hopefully, they are referring to the pressure bleed, not a patient bleed). There were a total of 3,692 cases treated in this manner at this site. Many of the patients required multiple treatments to resolve their symptoms. It was reported that 90% of pain cases were resolved and 100% of chokes cases were resolved. Of the 82 cases of spinal cord involvement, only 11 failed to resolve (13%). Of the 190 cases of vertigo, only 82 did not completely resolve, yet they all had some improvement in symptoms.

By 1917, the New York Public Service Commission embraced the policy that stricken workers would be recompressed to the depth at which they had been working. At long last, standards of care were finally being accepted.

In 1924, the U.S. Navy published its first standard therapy, "U.S. Navy Diving Manual, Bureau of Construction and Repair, 1924," which consisted of the following:

1. Compression of the diver to 45 psi (101 fsw).
2. Further compression to 60 psi (135 fsw) if 45 psi did not produce a significant improvement in the patient's symptoms.
3. Maintenance of 45 psi or 60 psi until the symptoms were relieved.
4. Decompression at the following rates:

Depth	Ascent Rate
60 psig-45 psig (135 fsw-101 fsw)	1 psi/min (2.25 fsw/min)
45 psig-30 psig, (101 fsw-67 fsw)	1 psi/3 min (2.25 fsw/3 min)
30 psig-15 psig (67 fsw-34 fsw)	1 psi/5 min (2.25 fsw/5 min)
15 psig-0 psig (34 fsw-surface)	1 psi/10 min (2.25 fsw/10 min)

Figure 1 – Decompression Rates

This produced a maximum decompression time of 285 minutes (4 hours, 25 minutes), providing the patient began at a depth of 135 fsw. If the patient had relief of symptoms at 101 fsw, it shortened the treatment by 15 minutes.

While these tables were a great step in the right direction, they did not prove to be entirely satisfactory. The need for retreatment was common, and while a good outcome was common in less severe cases, more serious cases often proved disappointing. As a result of the need for an improved treatment table, a modification of the above table was implemented in the mid-1930s and then was formally published in the 1943 U.S. Navy Diving Manual. This table (Figure 1) had the diver recompressed on air to the depth of relief (up to 300 fsw) and then remain there for 30 minutes. He was then decompressed to the surface based on the table.

IF NITROGEN IS THE CAUSE OF DCS, WHY DON'T WE USE A DIFFERENT GAS?

Several experts in the field (most notably, Philip B. James) have been trying for years to demonstrate the increased safety of helium as an adjunct to oxygen in both diving and recompression therapy. Dr. James has demonstrated in numerous scientific papers the decreased incidence of DCS in divers using helium rather than nitrogen. He has also brought to light how the occurrence of recompression injuries can be avoided when treating injuries in a chamber with a heliox mix rather than using straight oxygen or nitrogen/oxygen at various ratios and at different depths.

In a perfect world, we would probably all be using a heliox mix for both diving and treatments. Unfortunately, we don't. We are restrained by the limits of practicality and economics. As a result of these constraints, nitrogen remains the most readily accessible and the cheapest way to mix gas for diving and recompression. If there was a way to make helium more accessible to the diving public, and especially to the recompression chambers of the world, it would easily become the standard of care everywhere. If you are fortunate (?) enough to be bent in a part of the world that has the technology and the economics to provide its patients with heliox as its treatment modality, your likelihood of having a favorable outcome for your treatment of DCS will be maximized. It should also be clearly stated that the absence of helium should not prevent you from seeking and obtaining medical treatment in the event of DCS. Even more important than what gas you use in the treatment of your injuries is how rapidly you begin your treatment. The greatest chance for complete recovery is to begin treatment as soon as you realize there is a problem. By raising the bar and saying that every chamber must be able to deliver helium to treat dive injuries, we would only force chambers in many areas to shut down. This would only reduce the availability of recompression therapy to thousands of divers in need of potentially lifesaving therapy. With any luck, technology will be developed that will allow for the easy collection, storage, and delivery of helium. But until then, dive safe, dive smart, and dive conservatively.

DECOMPRESSION STOPS OF 1943 AIR RECOMPRESSION PROCEDURES TABLE 1

This table has been retained, and is still used as Treatment Table 8, which is used for HeO_2 (heliox) blowups. It is based on a tissue compartment time of 75 minutes.

At roughly the same time, Behnke and Shaw were doing experiments on dogs. These experiments began to show the efficacy of using O_2 and beginning the recompression at an even greater depth. By 1939, Behnke and Yarborough published a new alternative to the standard Treatment Table 1. This began at an initial depth of 165 fsw (75 psig), and after ascent to 60 fsw, the patient began to breath O_2. This modification became the first generally accepted treatment protocol that included the use of oxygen.

While again, this new table was a large improvement over the previous tables, as time progressed and diving became more popular outside the military community, it became apparent that more modifications needed to be made. As more divers began to use scuba equipment, two types of dive profiles began to be recognized in the evaluation of bent divers. Those types were described as standard and non-standard. The definition was quite simple. A non-standard dive was any dive not decompressed on a standard U.S. Navy decompression schedule (to include the ascent to the surface, in addition to any stops that should have been made), or performed by someone other than a graduate of the Navy Diving School. Non-standard dives were frequently done by civilian divers. More often than not, these involved grossly omitted decompression, and often involved long delays in seeking treatment. This is especially important when you consider the total number of dives being done by the military and the total number of injuries they incurred, in contrast with the number of injuries seen with non-standard dives, compared to the total number of non-standard dives being made.

Because of both the total number of cases being treated and the number of treatment failures

Treatment Depth (fsw)	Depth (fsw)														Total Time
	140	130	120	110	100	90	80	70	60	50	40	30	20	10	
100											14	42	52	68	175 min 2h 55m
150									22	30	35	30	52	68	249 min 4h 9m
200						7	22	24	26	30	35	42	52	68	306 min 5h 6m
250				13	18	19	22	24	26	30	35	42	52	68	351 min 5h 51m
300	4	14	16	16	18	19	22	24	26	30	35	42	52	68	387 min 6h 27m

Figure 2 – Decompression Stops of 1943 Air Recompression Procedures

AIR/OXYGEN RECOMPRESSION THERAPY

Depth (fsw)	Time (minutes)	Gas Mixture
165	30	Air
90	7	Air
80	22	Air
70	24	Air
60	26	Oxygen
50	30	Oxygen
40	34	Oxygen
40-Surface	15	Oxygen

Total Time to Surface – 188 minutes (3 Hours, 8 Minutes)
Total Air Time – 83 Minutes (1 Hour, 23 Minutes)
Total Oxygen Time – 105 Minutes (1 Hour, 45 Minutes)

Figure 3

DIVE INJURIES OF STANDARD DIVES COMPARED TO NON-STANDARD DIVES

Number of cases following

Years	Standard Dives	Non-Standard Dives	Percent of Cases Following Non-Standard Dives
1946	96	17	15%
1947-1955	303	40	12%
1956-1959	88	167	65%
1960-1961	124	52	31%
1962	40	27	40%
1963	25	48	66%
1964	28	32	53%
TOTALS	705	383	35%

Figure 4

(treatments that did not have a 100% resolution), Doodman and Workman (1965) began to re-investigate the use of recompression therapy that could utilize 100% oxygen for the entire duration of the treatment. It was believed that with 100% oxygen, you could promote tissue oxygenation, have faster bubble resolution, and have increased elimination of supersaturated inert gases. From those studies, it was determined that three criteria must be met for a treatment to be successful. Those criteria were:

1. A full treatment depth of 60 fsw.
2. At least 30 minutes of oxygen breathing at 60 fsw.
3. At least 90 minutes of total oxygen breathing time.

U.S. Navy Treatment Table 5 was developed by extending the oxygen time at 60 fsw by 10 minutes, and raising the total duration of oxygen breathing from 90 minutes to 120 minutes. U.S. Navy Treatment Table 6 merely doubled the total oxygen exposure of Treatment Table 5.

WHAT TABLE DO I USE?

Table 1A

- Symptoms using in-water recompression
- Asymptomatic omitted decompression
- Type I symptoms relieved at 66 fsw or shallower
- Exceeded Sur-D surface interval, with no symptoms

Table 2A

- Type I symptoms relieved deeper than 66 fsw
- Asymptomatic omitted decompression

Table 3

- Type II or arterial gas embolism symptoms relieved within 30 minutes at 165 fsw

Table 4

- Worsening Type II symptoms at 60 fsw
- Unresolved AGE symptoms after 30 minutes at 165 fsw
- Recurrence of symptoms 60 fsw or deeper

Table 5

- Type I symptoms relieved within 10 min of arrival at 60 fsw with normal pre-treatment neurological examination
- Asymptomatic missed decompression
- Treated and resolved in-water symptoms
- You can extend treatment times by 2 oxygen/air segments at both 60 fsw (25 minutes each) and 30 fsw (75 minutes each)
- If symptoms occur during ascent period, the diver should be recompressed to the depth they just ascended from and extend for one oxygen/air segment before attempting to ascend again

Table 6

- Type I symptoms not resolved within 10 minutes at 60 fsw or where neurological exam not done
- Recurrence of symptoms shallower than 60 fsw
- Type II or AGE symptoms responding to an initial 60 fsw recompression
- Asymptomatic missed decompression
- Symptomatic blowup from 60 fsw or less
- Treatment but unresolved in water symptomatic
- You can extend treatment times by 2 oxygen/air segments at both 60 fsw (25 minutes each) and 30 fsw (75 minutes each)
- If symptoms occur during ascent period, the diver should be recompressed to the depth they just ascended from and extend for one oxygen/air segment before attempting to ascend again

Table 6A

- AGE not responding at 60 fsw but resolving within 30 min at 165 fsw where initially 20 min or less are spent at 60 fsw
- Symptomatic blowup from deeper than 60 fsw

Table 7

- Type II or AGE symptomatic needing more time at 60 fsw
- AGE symptomatic relieved within 30 min at 165 fsw but where more than 20 min spent at 60 feet during initial recompression

Table 8

- Deep blowup from surface supplied heliox dives

Note 1: 21% oxygen in helium may be used instead of air if available
Note 2: Up to 50% oxygen in nitrogen mixes may be used to depth of 165 fsw

Table 9

- For residual treatment of AGE/DCS
- Can be used for initial treatment if determined by Diving Medical Physician
- For divers who may have recieved an injury on a heliox dive
- For divers whose symptoms have worsened during a treatment
- For divers whose symptoms worsened during treatment, after a heliox dive

It should be noted that Treatment Tables 1A, 2A, and 3 are air-only tables, and should not be used if oxygen is available. Table 4 should be used only with unresolved AGE symptoms after 30 minutes at 165 fsw.
The most commonly used treatment tables are the U.S. Navy Treatment Tables 5 and 6.

DRUG THERAPY CONSIDERATIONS FOR USE IN DECOMPRESSION SICKNESS

There are several drugs which need to be considered in the field of hyperbaric medicine. There are several that you will be administering to the patient while under your care, and even more that the patient will already have circulating through his body. Unfortunately, the ones in the latter category are ones that the patient frequently needs to maintain normal body homeostasis, and we will probably never understand the pharmacokinetics of its molecular properties under pressure. We tend to use those in the former group more frequently, whose results we can analyze, at least anecdotally.

Several points need to be considered when using medication in addition to hyperbaric therapy:

1. Drug therapy should never be used as a substitute for recompression therapy.
2. No drug or combination of drugs has ever been proven to be safe and effective for the treatment of DCS by controlled double-blinded studies in humans. Due to the sporadic nature of DCS, it is unlikely that we will ever be able to develop a protocol that would be useful in the evaluation of medications. Anecdotal reports don't count (they do, but I had to put in that disclaimer).
3. Usefulness of an drug or medication for the prophylaxis of DCS does not necessarily imply usefulness of the same drug or medication for the treatment of DCS.
4. Just because a drug will inhibit or reverse an action that is associated with DCS, i.e., platelet aggregation, does not mean that the drug will improve the clinical outcome of DCS (even though it frequently does).
5. Species difference may be important. What works in animals may not work in humans.
6. The potential hazards of a drug must be weighed against its potential benefits. Remember, above all else, DO NO HARM.

NOW, THE DRUGS

Oxygen

First and foremost. Probably the single most important drug (yes, it is a drug) that we use. Like any other drug that we use, it has toxic effects. If you give too much of it, your patient will die (a bad thing), or have permanent sequelae (still a bad thing). And like any other drug, it is susceptible to drug/drug interactions.

Intravenous Fluid

Virtually everyone that has DCS is dehydrated. If they say they are not dehydrated, then assume that they are lying. The P Phenomenon alone will cause divers to diurese, bringing them to the surface with a fluid deficit. Dehydration leads to hemoconcentration, which will also contribute to DCS by causing microvascular occlusion. Also, should you need to rapidly give medication, you need a rapid route. For all of these reasons, the patient should have an I.V. started. The choice of fluid is continually open to discussion, but as long as it's isotonic saline (0.9% NaCl) or Lactated Ringers (LR), either will suffice. The biggest difference is that LR has potassium, which can be used as an argument both for and against its use. The bottom line is that the choice of fluid is optional while the use of the I.V. is mandatory.

Steroids

Steroids are recommended in DCS to reduce brain and spinal cord swelling, as well as to reduce the inflammatory response seen in vascular and tissue injuries. The downside to steroids is that their use can potentiate oxygen toxicity.

Alcohol

While alcohol and post alcohol use seem to contribute to DCS, probably as a result of dehydration, there has been no indicated use of it as an adjunct for DCS by either the patient or the tender. And, as always, no drinking and driving.

Non-Narcotic Analgesics

Aspirin is a mainstay in DCS. It inhibits platelet aggregation, helping to minimize injury by occlusion to the microvasculature, and works locally to inhibit inflammatory mediators. Caution should be taken in the use of oral pain medications for any patient with DCS. One of the methods we have for the evaluation of resolution of symptoms is by how the pain is resolving. It is of paramount importance that we know that the patient is getting better as a result of recompression, not because we are masking the pain with pain medication.

Narcotic Analgesics

If you must give pain medication for the immediate treatment of pain, narcotics are the drug of choice, but they have significant side effects that must not be taken lightly. And they must be given intravenously. The most obvious side effect is that once the drugs are on board, you can't evaluate how the patient is responding to recompression. You now have to sort out if it is the depth of the treatment, or the effect of the drug that is making the patient feel better. Another undesired effect of narcotics is that they will decrease respirations. This will in turn cause a buildup in pCO_2. This slight elevation in pCO_2 will cause cerebral vasodilation, which will allow more dissolved O_2 in the blood to enter brain tissue. This in turn increases the risk of O_2 CNS toxicity. Additional problems commence if the drugs are given intramuscularly, since hyperbaric oxygen causes vasoconstriction. The vasoconstriction will impair the absorption of the medication. This might cause the provider to give additional medication. Then, once the patient reaches the surface and the vasoconstriction is relieved, the patient will get a large dose of the drug. The reason that narcotics (as well as any other medications) are given through I.V. is that you can give an amount that will give a predictable response, it is rapidly absorbed in a predictable fashion, and if you need to, narcotics can be reversed by the administration of a narcotic antagonist.

Diazepam (Valium®)

This is the drug of choice for seizure activity in the chamber. Its response is predictable and generally safe. The most serious side effect of Diazepam is it can depress respiration if given in a high enough dose. One of the advantages of Diazepam is that its effects can be reversed with a benzodiazepam antagonist. It can also be used as an anxioltic (anti-anxiety) for patients who may be feeling claustrophobic. For unknown reasons, the dosage of Diazepam needs to be increased in patients undergoing hyperbaric therapy. There does not seem to be an increased rebound sedation when the treatment is terminated.

Lorazepam (Ativan®)
Similar effects as Diazapam, but at a much lower dose and with a longer action. Can also produce an anterograde amnesia.

Vitamin E
Vitamin E appears to have a protective effect against pulmonary toxicity. The seizure threshold also appears to be raised. At present, the recommended dose is 400 units per 90 minutes of oxygen treatment per day.

Lidocaine
This may be the next drug to be used with enthusiasm in the field of hyperbaric medicine. Initial studies seem to indicate a significant improvement in the resolution of symptoms associated with DCS when lidocaine was used in conjunction with standard treatment. The standard treatment seems to be a 1mg/kg loading bolus, followed by a maintenance drip of 1-2 mg/min.

TAKING CARE OF YOUR TENDERS

In addition to taking care of divers that have been injured secondary to hyperbaric exposure, your tenders will also be placed in a similar environment with the same potential risks. Needless to say, the same precautions that divers are expected to adhere to, you need to have your tenders adhere to as well. There are also specific expectations that should be placed on tenders in addition to those expected of divers. The tender's expectations of you are higher. That includes documentation of tenders, both before and after their chamber exposure. Proper documentation cannot be stressed enough in today's highly litigenous society.

HYPERBARIC CHAMBER RULES

1. When you are on call, you are just that, on call. You need to be available for the entire time that you are on call. Don't be unavailable!
2. Don't plan on doing any diving when you are on call. It is hard to hear your phone under water, and it makes you sort of worthless as a tender.
3. If you are thinking about having any alcohol while you are on call, think again. If you are seen consuming alcohol while on call, or show up at the chamber demonstrating evidence of intoxication, you will be assumed to have a substance abuse problem. If you can't abstain from alcohol for 24 hours, let us know and we will help get you into treatment.
4. After acting as a tender, you must stay in the chamber area until released by the dive supervisor. After being a tender, you may not do any strenuous exercise for 12 hours (or until your "clean" time). Strenuous exercise is anything that causes you to break a sweat. This includes trail hiking, walking almost anywhere, or doing almost anything. You may not fly for 12 hours after reaching the surface from a treatment.
5. If you have an acute medical condition that you think should preclude you from being on call, inform your team leader. If you are too hung-over to be on call, see #3.
6. If you have a personal, religious, ethnic, moral, dietary, sexual, emotional, perverted, or visual (i.e., I can't see going in the chamber) problems, that's OK. No one will be forced to go in the chamber. Check with your team leader. We will find a way for you to participate. If you have a pathological condition that will keep you out of the chamber, please don't wait until we are about to put you in with a patient to bring it up.
7. Common sense prevails. If you see something that seems unsafe, stop whatever is going on until it is safe.

8. No talking during traveling. Any sounds heard will be assumed to be someone in trouble and we will stop traveling.

9. Tenders will always be observed during traveling. Tenders will show the OK sign when traveling. If an OK sign is not observed, it will be assumed that there is a problem and we will stop traveling.

10. Upon exiting the chamber, eye contact MUST be made with the Dive Supervisor prior to your post-dive exam. If you wander off after exiting the chamber, you will be presumed to have a Type II hit and you will be treated as such.

11. There is NO SMOKING in or around the chamber. If you are there for a treatment, YOU MAY NOT SMOKE. YOU MAY NOT STEP OUT AND SMOKE. Smoking and hyperbaric oxygen are incompatible.

12. There is a dress code. Adhere to it. We don't have a dress code because we are fashion buffoons (we do it in spite of that fact). Improper clothing can contribute to fires. Safety is not taken for granted.

THE RECOMPRESSION CHAMBER

There are two basic types of recompression chambers: monoplace and multiplace chambers. There are also two types of multiplace chamber: single compartment and multiple compartment chambers. The primary disadvantage of single compartment chambers is that once you begin to pressurize the chamber, nothing can go in or come out. Personnel and supplies that are with the patient at the start of treatment must complete treatment with the patient. And in the event that the needs of the patient change, the patient must be brought to the surface in order to accommodate them.

Monoplace chambers are being used more and more frequently. They are lightweight, portable, and relatively inexpensive. They accommodate one patient at a time, and are small enough that several can be placed in one room, allowing for multiple treatments to be conducted simultaneously. The primary disadvantage lies in that they are constructed to allow for limited pressures. Most have a maximum operating depth of 30-45 fsw.

Multiple-compartment chambers allow for the ability to move tenders and supplies in and out of the chamber while maintaining the same depth. The only requirement of the multi-compartment chambers is that all compartments be able to safely reach and maintain the maximum depth of treatment.

CHAMBER FIRES

Fires are of major concern to the hyperbaric medical environment. Fortunately, because of strict adherence to safety standards, the risk of fire in a modern chamber is very slight. In a review of the literature, virtually all fires involving hyperbaric chambers involve a source of ignition that was brought into the chamber by a patient.

Three requirements are needed in order for combustion to occur: a flammable fuel, a source of ignition, and an atmosphere capable of supporting combustion. The temperature necessary for ignition at 200 fsw is 100 degrees less than at the surface. The burning rate also increases as depth increases. The burning rate is twice the sea level at 100 fsw. The burning rate also increases as the concentration of oxygen increases. Increasing the concentration of oxygen from 21% to 25% increases the burn rate by 25%. To date, chamber fires have been limited to environments that were higher than 28% oxygen. This is one of the primary reasons that the patient must exhale into the mask while on an oxygen period, otherwise the exhaled gas will increase the O_2 concentration of the chamber environment to dangerous levels. Oxygen concentrations inside the chamber should never be allowed to go above 24%.

The most important part of fire avoidance is prevention. All flammable materials must be kept out of the chamber. The primary source of ignition is usually an electrical spark. Any electrical appliance that goes into the chamber must be sealed in such a way that it will not spark. Battery operated equipment must have a power source that does not give off hydrogen gas. When balloons are taken into the chamber for demonstration purposes, instructions must be given NOT to rub them on your head so that they stick to the ceiling. It is difficult for static electricity to be generated in an environment with a humidity of at least 60%. If you need to start an I.V., give a shot, or administer medications into I.V. tubing, you may NOT use an alcohol pad to cleanse the area. The alcohol makes a great source for ignition, and the pad makes a great wick (see the connection). Garments should be 100% cotton. While cotton is not fireproof, it does not promote static electricity. Swimwear, unless it is 100% cotton, is not to be worn in the chamber. It is imperative that the patient and the tenders adhere to a dress code.

CHAPTER 10

CONSIDERATIONS TO BE MADE BEFORE, DURING AND AFTER RECOMPRESSION THERAPY

EVALUATION OF DIVE INJURIES

Severity of injuries is determined by how much distress or pain the patient is in. Any obvious disorders of mental status, gait, limb movement, respiration, or circulation qualifies as emergent.

Category A-Emergent
Symptoms are severe, involve the inner ear, cardiorespiratory system or CNS, and/or are progressive or relapsing. These patients need to be pressed now. Do your physical exam on the way down to depth.

Category B-Urgent
The only symptom is pain. Pain is static or has progressed only minimally over the last few hours. This patient should have a thorough exam before going to depth.

Category C-Timely
Symptoms are not severe and may take a detailed exam to localize injury. Symptoms are static or progressing very slowly. These patients have time for a thorough physical exam to rule out the possibility of a non-diving pathology.

PATIENT RESPONSE

All recompressions initially go to 60 feet. Once at 60 feet, any decision is based on patient response.

Deterioration
Patient is in a life-threatening situation.

Progressive
Patient is getting worse.

Stable
Unchanged.

Improving
Patient reports that there is a clear improvement in symptoms. A subjective finding only.

Significant Improvement
Patient improvement is obvious to chamber personnel.

Resolved
Complete relief of symptoms is reported by the patient and confirmed by subsequent exam.

DELAYS IN ASCENT

For any number of reasons, your patients or tenders may have a delay that occurs on their ascent to the surface. Where the delay occurs, and for how long it occurs, will influence how you manage the remainder of the ascent.

If you have a delay in the treatment period when you are travelling from 60 fsw to 30 fsw at 1 fpm, then there is nothing to do. You simply

continue your ascent at 1 fpm. You do not attempt to make up the lost time that you encountered with your delay. Your ascent time will be extended by however long your delay was. The same for travels from 30 fsw to the surface at 1 fpm. If you are ascending from depth with a tender, or a patient that you have just pressure tested, and are ascending at a rate of 30 fpm, then a hold is calculated in differently. The depth that the hold occurs at also influences how the delay is managed.

If the hold occurs at a depth of 50 fsw or deeper, and is longer than 60 seconds, then that delay is added to the diver's total bottom time. His total bottom time must then be recomputed, and if he has violated no-decompression limits, then his ascent to the surface will have to be modified to include the proper decompression stops. For added safety, any delay should be rounded up to the next whole minute.

For delays shallower than 50 fsw, any delay will be added to the diver's first decompression stop (increasing the time of the first stop by the number of minutes of the delay). If it is a no-decompression dive, then the delay is credited as decompression time, provided the delay is not so long that it doesn't exceed the no-decompression limit or that depth.

At the other end of the spectrum is a rapid ascent. If you find that you have not been paying attention to your 30 fpm ascent rate and suddenly find yourself at a shallower depth than you should be, stop your ascent and let the clock catch up to you. Do not descend to reorient to the clock. Just wait, and when the clock catches up to you, continue your ascent to the surface at the correct rate. If your patient develops DCS as a result of your rapid ascent, you are then obligated to treat your patient as a missed decompression, descend to 60 fsw and treat based on the type of symptoms that they developed (Table 5 or Table 6).

TENDER BOTTOM TIME

Once you have decided to place a patient in the chamber, you now have to decide how you are going to use your tenders. If you have an endless supply of tenders, a good rule of thumb is to plan to leave your tenders in the chamber for 50-75% of their no-decompression limit for the depth of the treatment. So at 60 fsw, your tenders should have a total bottom time (left surface to left bottom) of 30-45 minutes. If you have a limited supply of tenders, you can use a single tender for the entire treatment, but for an added margin of safety, the tender should go on oxygen during the travel from 30 fsw to the surface.

VARIATIONS IN DEPTH AT TREATMENT LEVEL

All treatments should be kept to the prescribed depth plus or minus less than one fsw. If there is a variation in the depth, then the chamber should be returned to the correct depth as soon as possible. This means that if you are shallow, you should descend as rapidly as can be tolerated by the occupants. If the chamber has descended to a deeper depth, then the chamber should be brought back to the proper depth at an ascent rate not to exceed 30 fpm. If you are performing frequent tender swaps, then the bottom time must be recalculated on a deeper schedule to match the new maximum depth.

PROBLEMS ENCOUNTERED DURING DESCENT

The most common problem to be encountered during descent is going to be the inability to clear your ears. This can be countered in any of a number of ways. The easiest is to slow the rate of descent, allowing the chamber occupants adequate time to clear. It may require that you ascend a few feet before you re-continue the descent to allow the initial insult to resolve. If the patient is too critical to slow the travel then your options are more limited. The easiest thing

to do is to just continue the travel and hope that the tympanic membranes spontaneously rupture. The patient's pain will be relieved as soon as the rupture occurs, and equalization will no longer be a problem. Healing, as a rule, will occur spontaneously, but healing tends to resolve at a slower rate than if the tympanic membrane is perforated surgically. If you have the luxury of time then it is a simple procedure to do a myringotomy with a 21 gage, 3-1/2 inch spinal needle. Simple topical anesthesia will make the procedure more tolerable (for the patient). This can be done through the view head of any standard otoscope.

Other problems include claustrophobia and apprehension/agitation. Since all patients are put in the chamber with an I.V., it is no problem to give an anxiolitic. Valium™ works quite well, and has a relatively short half-life, clouding your physical exam for a shorter period of time than Ativan™ would. It needs to be remembered that for some reason, it takes a greater dose of these drugs to produce the same effect than it would normally require at the surface. Another advantage of the benzodiazapans is that the unwanted effects can be reversed with Flumazinil™, a benzo-antagonist.

INTRAVENOUS HYDRATION AND OTHER TUBES

Every acute patient in the chamber needs an I.V. of either Lactated Ringers or Normal Saline. No dextrose-containing solutions should be used during recompression therapy. The patients should be vigorously rehydrated until they can produce a dilute urine. Careful attention must be paid to the drip chamber if the patient is not on a pump, as the size of the bubble will change with depth (Boyle's Law). A catheter can be placed to monitor urine outflow, but the balloon must be inflated with water, not air, to avoid size changes associated with depth changes. The same goes for endotracheal tubes. The cuff must not be inflated with air. Fluid must be used.

RETURN TO DIVING AFTER DECOMPRESSION SICKNESS

Divers with Type I DCS symptoms, who have complete resolution of symptoms with Treatment Table 5, may return to diving after 48 hours when signed off by a qualified physician. Divers who had Type I DCS symptoms that were successfully treated on Treatment Table 6 can resume diving after seven days. Divers whose only Type II symptoms were patchy peripheral parasthesias, and whose symptoms were completely resolved by the beginning of the second oxygen treatment, may resume diving after 14 days. Any other Type II symptoms should wait at least four weeks. These are general recommendations. There are some divers who should probably never return to the water.

It is also recommended that divers with any type of DCS undergo cardiac ultrasonography prior to returning to diving to rule out a patent foramen ovale or other type of right to left shunt. An MRI, with and without contrast, can rule out any type of occult intercranial pathology caused by bubble formation. Baseline neuropsycological testing is also recommended to establish a baseline level of functioning in case there are any changes noted later in life. While this may not be of much help to the individual, it may prove to be valuable for divers in the future.

CHAMBER OPERATING TEMPERATURES

Because the body's metabolic rate is influenced by surrounding temperatures, specific guidelines must be adhered to for monitoring the temperature inside the chamber. The inside chamber temperature can be monitored by a number of different types of thermometers, providing that they are neither electric or mercury.

The temperature limitations are as follows:

Chamber Operating Temperatures

Internal Temperature	Maximum Treatment Time
104°F (40°C) or greater	No treatment allowed
94°F - 104°F (34.4°C - 40°C)	2 Hours (Treatment Table 5)
85°F - 94°F (29.4°C - 34.4°C)	6 Hours (Treatment Table 5, 6, 6A)
Under 85°F (29.4°C)	Unlimited

APPENDIX A

FLOW CHARTS TREATMENT TABLES

GENERAL

This appendix contains the diving accident treatment flow charts and a number of U.S. Navy Treatment Tables used to recompress divers who have experienced decompression sickness or arterial gas embolism as a result of their diving activities. The information in this appendix reflects treatment procedures recommended by the NOAA Diving Program and taught in the NOAA training program. All of the tables in this appendix have been widely used in the field and have been shown to be safe and effective.

Diving Accident Treatment Flow Charts

The flow charts shown are decision trees designed to aid dive supervisors, diving physicians, Diving Medical Technicians, chamber operators, and other health care professionals who must decide how best to treat stricken divers. Use of the decision tree requires only that the diver's condition be observed; a medical diagnosis is not required for treatment to begin.

Recompression Treatment Tables

The recompression treatment tables recommended by NOAA are shown on the following pages. Instructions for the use of these tables appear with each table and should be followed precisely.

RECOMPRESSION TREATMENTS WHEN CHAMBER IS AVAILABLE

Oxygen Treatment Tables are more effective and, therefore, preferable over Air Treatment Tables. Treatment Table 4 can be used with or without oxygen but should always be used with oxygen if it is available.

Symptoms During Decompression and Surface Decompression: If symptoms of decompression sickness occur in the water during decompression, follow Flow Chart 1. After completing recompression treatment, observe the diver for at least 6 hours. If any symptoms recur, treat as a recurrence of Type II symptoms. As an option, the on-site Diving Supervisor may elect not to recompress the diver 10 feet in the water, but to remove the diver from the water when decompression risks are unacceptable and treat him in the chamber. When this is done, the surface interval should be 5 minutes or less, with the diver always treated as having Type II symptoms.

Treatment of Symptoms During Sur-D Surface Interval: If surface decompression procedures are used, symptoms of decompression sickness may occur during the surface interval. Because neurological symptoms cannot be ruled out during this short period, the symptomatic diver is treated as having Type II symptoms, even if the only complaint is pain.

Treating for Exceeded Sur-D Surface Interval: If the prescribed surface interval is exceeded but the diver remains asymptomatic, the diver is treated with Treatment Table 5, or Treatment Table 1A if no oxygen is available. If the diver becomes symptomatic, the diver is treated as if Type II symptoms were present. Any symptoms occurring during the chamber stops are treated as recurrences in accordance with Flow Chart 3.

Recompression Treatments When Oxygen Is Not Available: If no oxygen is available, select the appropriate Air Treatment Table in accordance with Table 1A, Table 2A, Table 3, and Table 4.

Use Table 1A if pain is relieved at a depth less than 66 feet. If pain is relieved at a depth greater than 66 feet, use Table 2A. Table 3 is used for treatment of serious symptoms where oxygen cannot be used. Use Table 3 if symptoms are relieved within 30 minutes at 165 feet. If symptoms are not relieved in less than 30 minutes at 165 feet, use Table 4.

Descent/Ascent Rates for Air Treatment Tables: The Air Treatment Tables (1A, 2A, 3, and 4 using air) are used when no oxygen is available. They are not as effective as the Oxygen Treatment Tables. The descent rate is 20 feet per

minute; the ascent rate is not to exceed 1 foot per minute.

Recompression Treatments When Oxygen Is Available: Use Oxygen Treatment Tables 5, 6, 6A, 4, or 7, according to Flow Charts 2, 3, and 4. The descent rate is 20 feet per minute. Upon reaching treatment depth not to exceed 60 fsw, place the patient on oxygen. For depth deeper than 60 fsw, use treatment gas if available. Additional guidelines for each treatment table are given below.

Treatment Table 5: Treatment Table 5 may be used for the following:

- Type I (except for cutis-marmorata) symptoms when a complete neurological examination has revealed no abnormality
- Asymptomatic omitted decompression of shallow surfacing (20 fsw or less)
- Asymptomatic omitted decompression of rapid ascent (from deeper than 20 fsw) if the missed decompression is less than 30 minutes
- Asymptomatic divers who have exceeded surface interval limits following a Sur-D dive
- Treatment of resolved symptoms following in-water recompression
- Follow-up treatments for residual symptoms

Performance of Neurological Exam at 60 fsw: After arrival at 60 fsw a neurological exam shall be performed (see Appendix II) to ensure that no overt neurological symptoms (e.g., weakness, numbness, incoordination) are present. If any abnormalities are found, the stricken diver should be treated using Treatment Table 6.

Extending Oxygen Breathing Periods on Treatment Table 5: Treatment Table 5 may be extended by two oxygen breathing periods at 30 fsw. Air breaks are not required prior to an extension, between extensions, or prior to surfacing. In other words, the Diving Supervisor may have the diver breathe oxygen continuously for 60 minutes at 30 fsw and travel to the surface while breathing oxygen. If the Diving Supervisor elects to extend this treatment table, the tender does not require additional oxygen breathing than currently prescribed.

When Use of Treatment Table 6 is Mandatory: Treatment Table 6 is mandatory if:

- Type I pain is severe and immediate recompression must be instituted before a neurological examination can be performed, or
- A complete neurological examination cannot be performed, or
- Any neurological symptom is present.

These rules apply no matter how rapidly or completely the symptoms resolve once recompression begins.

Complete Relief after 10 Minutes: If complete relief of Type I symptoms is not obtained within 10 minutes at 60 feet, Table 6 is required.

Musculoskeletal Pain Due to Orthopedic Injury: Symptoms of musculoskeletal pain that have shown absolutely no change after the second oxygen breathing period at 60 feet may be due to orthopedic injury rather than decompression sickness. If, after reviewing the patient's history, the Diving Medical Doctor feels that the pain can be related to specific orthopedic trauma or injury, Treatment Table 5 may be completed. If no Diving Medical Doctor is on site, Treatment Table 6 shall be used.

Note
Once recompression to 60 feet is done, Treatment Table 5 shall be used even if it was decided symptoms were probably not decompression sickness. Direct ascent to the surface is done only in emergencies.

Treatment Table 6: Treatment Table 6 is used for the following:

- Type I symptoms where relief is not complete within 10 minutes at 60 feet or where a neurological exam is not complete

- Type II symptoms
- Cutis marmorata
- Arterial gas embolism
- Symptomatic uncontrolled ascent
- Asymptomatic divers with omitted decompression greater than 30 minutes
- Treatment of unresolved symptoms following in-water treatment
- Recurrence of symptoms shallower than 60 fsw

Treating Arterial Gas Embolism: Arterial gas embolism is treated by initial compression to 60 fsw. If symptoms are improved within the first oxygen breathing period, then treatment is continued using Treatment Table 6. Treatment Table 6 may be extended for two oxygen breathing periods at 60 fsw (20 minutes on oxygen, then 5 minutes on air, then 20 minutes on oxygen) and two oxygen breathing periods at 30 fsw (15 minutes on air, then 60 minutes on oxygen, then 15 minutes on air, then 60 minutes on oxygen). If there has been more than one extension, the tenders' oxygen breathing period is extended 60 minutes at 30 feet.

Treatment Table 6A: Arterial gas embolism or severe decompression symptoms are treated by initial compression to 60 fsw. If symptoms improve, complete Treatment Table 6. If symptoms are unchanged or worsen, assess the patient upon descent and compress to depth of relief (significant improvement), not to exceed 165 fsw. Once at the depth of relief, begin treatment gas (N202, HeO2) if available. Stay there for 30 minutes. A breathing period of 25 minutes on treatment gas, interrupted by 5 minutes of air, is recommended at depth to simplify time keeping. The patient may remain on treatment gas during ascent from treatment depth to 60 fsw since the P02 will continually decrease during ascent. Decompress to 60 fsw at a travel rate not to exceed 3 ft/min. Upon arrival at 60 fsw, complete Treatment Table 6. Consult with a Diving Medical Doctor at the earliest opportunity. The Diving Medical Doctor may recommend a Treatment Table 4. Treatment Table 6A may be extended for two oxygen breathing periods at 60 fsw and two oxygen breathing periods at 30 fsw. If deterioration is noted during ascent to 60 feet, treat as a recurrence of symptoms (Flow Chart 4).

Treatment Table 4: If a shift from Treatment Table 6A to Treatment Table 4 is contemplated, a Diving Medical Doctor shall be consulted before the shift is made. Treatment Table 4 is used when it is determined that the patient would receive additional benefit at depth of significant relief, not to exceed 165 fsw. The time at depth shall be between 30 to 120 minutes, based on the patient's response.

Recurrence of Symptoms: If deterioration is noted during ascent to 60 feet, treat as a recurrence of symptoms (Flow Chart 4).

Oxygen Breathing Periods: If oxygen is available, the patient should begin oxygen breathing periods immediately upon arrival at the 60-foot stop. Breathing periods of 25 minutes on oxygen, interrupted by 5 minutes of air, are recommended. This simplifies timekeeping. Immediately upon arrival at 60 feet, a minimum of four oxygen breathing periods (for a total time of 2 hours) should be administered. After that, oxygen breathing should be administered to suit the patient's individual needs and operational conditions. Both the patient and tender must breathe oxygen for at least 4 hours (eight 25-minute oxygen, 5-minute air periods), beginning no later than 2 hours before ascent from 30 feet is begun. These oxygen-breathing periods may be divided up as convenient, but at least 2 hours' worth of oxygen breathing periods should be completed at 30 feet.

Treatment Table 7: Treatment Table 7 is considered an heroic measure for treating non-responding severe gas embolism or life-threatening decompression sickness. Committing a patient to Treatment Table 7 involves isolating the patient and having to minister to his medical needs in the recompression chamber for 48 hours or longer. Experienced diving medical personnel must be available before committing to Treatment Table 7.

Considerations: A Diving Medical Doctor shall be consulted before shifting to a Treatment

Table 7 and careful consideration shall be given to life support capability. In addition, it must be realized that the recompression facility will be committed for 48 hours or more.

Indications: Treatment Table 7 is an extension at 60 feet of Treatment Tables 6, 6A, or 4 (or any other nonstandard treatment table). This means that considerable treatment has already been administered. Treatment Table 7 is not designed to treat all residual symptoms that do not improve at 60 feet and should never be used to treat residual pain. Treatment Table 7 should be used only when loss of life may result if the currently prescribed decompression from 60 feet is undertaken.

Time at Depth: When using Treatment Table 7, a minimum of 12 hours should be spent at 60 feet, including time spent at 60 feet from Treatment Table 4, 6, or 6A. Severe Type II decompression sickness and/or arterial gas embolism cases may continue to deteriorate significantly over the first several hours. This should not be cause for premature changes in depth. Do not begin decompression from 60 feet for at least 12 hours. At completion of the 12-hour stay, the decision must be made whether to decompress or spend additional time at 60 feet. If no improvement was noted during the first 12 hours, benefit from additional time at 60 feet is unlikely and decompression should be started. If the patient is improving but significant residual symptoms remain (e.g., limb paralysis, abnormal or absent respiration), additional time at 60 feet may be warranted. While the actual time that can be spent at 60 feet is unlimited, the actual additional amount of time beyond 12 hours that should be spent can only be determined by a Diving Medical Doctor (in consultation with on-site supervisory personnel), based on the patient's response to therapy and operational factors. When the patient has progressed to the point of consciousness, can breathe independently, and can move all extremities, decompression can be started and maintained as long as improvement continues. Solid evidence of continued benefit should be established for stays longer than 18 hours at 60 feet. Regardless of the duration at the recompression below 60 feet, at least 12 hours must be spent at 60 feet and then Table 7 followed to the surface. Additional recompression below 60 feet in these cases should not be undertaken unless adequate life support capability is available.

Decompression: When using Treatment Table 7, tenders breathe chamber atmosphere. Chamber oxygen should be kept above 19% and carbon dioxide below 1.5% surface equivalent (sev) (11.4 mmHg). Decompression on Treatment Table 7 is begun with an upward excursion at time zero from 60 to 58 feet. Subsequent 2-foot upward excursions are made at time intervals appropriate to the rate of decompression:

Depth	Rate	Time Interval
58-40 feet	3 ft/hr	40 min
40-20 feet	2 ft/hr	60 min
20-4 feet	1 ft/hr	120 min

Preventing Inadvertent Early Surfacing: Upon arrival at 4 feet, decompression should be stopped for 4 hours. At the end of 4 hours at 4 feet, decompress to the surface at 1 foot per minute. This procedure prevents inadvertent early surfacing.

Time Intervals: The travel time between subsequent stops is considered as part of the time interval for the next shallower stop. The time intervals shown above begin when ascent to the next shallower stop has begun.

Oxygen Breathing: On a Treatment Table 7, patients should begin oxygen breathing periods as soon as possible at 60 feet. Oxygen breathing periods of 25 minutes on 100% oxygen, followed by 5 minutes breathing chamber atmosphere, should be used. Normally, four oxygen breathing periods are alternated with 2 hours of continuous air breathing. In conscious patients, this cycle should be continued until a minimum of eight oxygen breathing periods have been administered (previous 100% oxygen breathing periods may be counted against these eight periods). Beyond that, oxygen breathing periods should be continued as recommended by the

Diving Medical Doctor, as long as improvement is noted and the oxygen is tolerated by the patient. If oxygen breathing causes significant pain on inspiration, it should be discontinued unless it is felt that significant benefit from oxygen breathing is being obtained. In unconscious patients, oxygen breathing should be stopped after a maximum of 24 oxygen breathing periods have been administered. The actual number and length of oxygen breathing periods should be adjusted by the Diving Medical Doctor to suit the individual patient's clinical condition and response to oxygen toxicity.

Sleeping, Resting, and Eating: At least two tenders should be available when using Treatment Table 7, and three may be necessary for severely ill patients. Not all tenders are required to be in the chamber, and they may be locked in and out as required following appropriate decompression tables. The patient may sleep anytime except when breathing oxygen deeper than 30 feet. While asleep, the patient's pulse, respiration, and blood pressure should be monitored and recorded at intervals appropriate to the patient's condition. Food may be taken at any time and fluid intake should be maintained.

Ancillary Care: Patients on Treatment Table 7 requiring intravenous and/or drug therapy should have these administered in accordance with a Diving Medical Doctor.

Abort Procedures: In some cases, a Treatment Table 7 may have to be terminated early. If extenuating circumstances dictate early decompression and less than 12 hours have elapsed since treatment was begun, decompression may be accomplished using the appropriate 60–foot Air Decompression Table as modified. The 60–foot Air Decompression Tables may be used even if time was spent between 60 and 165 feet (e.g., on Table 4 or 6A), as long as at least 3 hours have elapsed since the last excursion below 60 feet. If less than 3 hours have elapsed, or if any time was spent below 165 feet, use the Air Decompression Table appropriate to the maximum depth attained during treatment. All stops and times in the Air Decompression Table should be followed, but oxygen-breathing periods should be started for all chamber occupants as soon as a depth of 30 feet is reached. All chamber occupants should continue oxygen-breathing periods of 25 minutes on 100% oxygen, followed by 5 minutes on air, until the total time breathing oxygen is one-half or more of the total decompression time.

If more than 12 hours have elapsed since treatment was begun, the decompression schedule of Treatment Table 7 shall be used. In extreme emergencies, the abort recommendations may be used if more than 12 hours have elapsed since beginning treatment.

Treatment Table 8: Treatment Table 8 is an adaptation of a Royal Navy Treatment Table 65 mainly for treating deep uncontrolled ascents when more than 60 minutes of decompression have been missed. Compress symptomatic patient to depth of relief not to exceed 225 fsw. Initiate Treatment Table 8 from depth of relief. The Table 8 schedule from 60 feet is the same as Treatment Table 7.

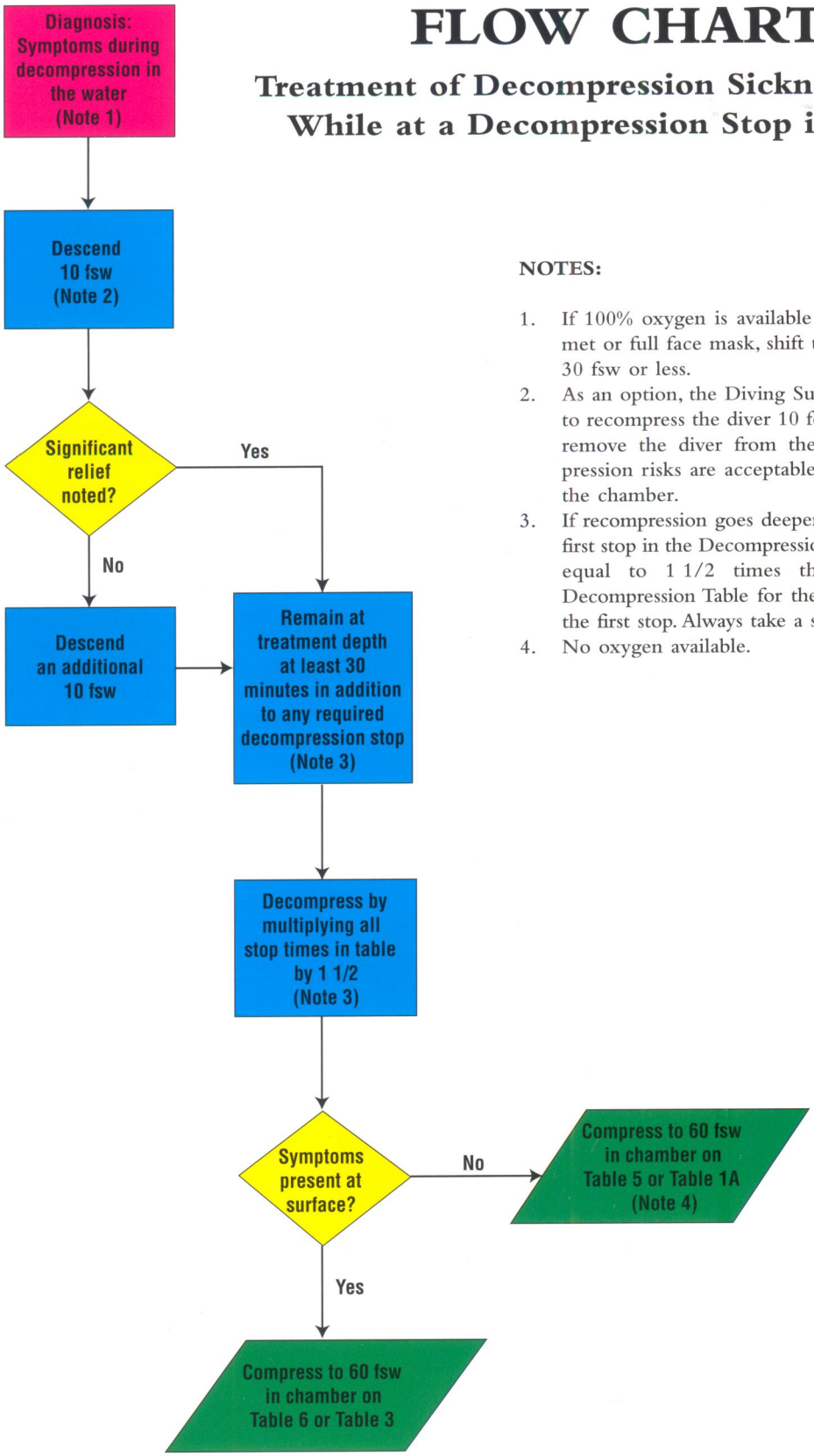
Treatment Table 9: Treatment Table 9 is a hyperbaric oxygen treatment table using 90 minutes of oxygen at 45 feet. This table is recommended by the Diving Medical Doctor cognizant of the patient's medical condition. Treatment Table 9 is used for the following:

- Residual symptoms from AGE/DCS

This table may also be recommended by the cognizant Diving Medical Doctor when initially treating a severely injured patient whose medical condition precludes long absences from definitive medical care.

FLOW CHART 1

Treatment of Decompression Sickness Occurring While at a Decompression Stop in the Water

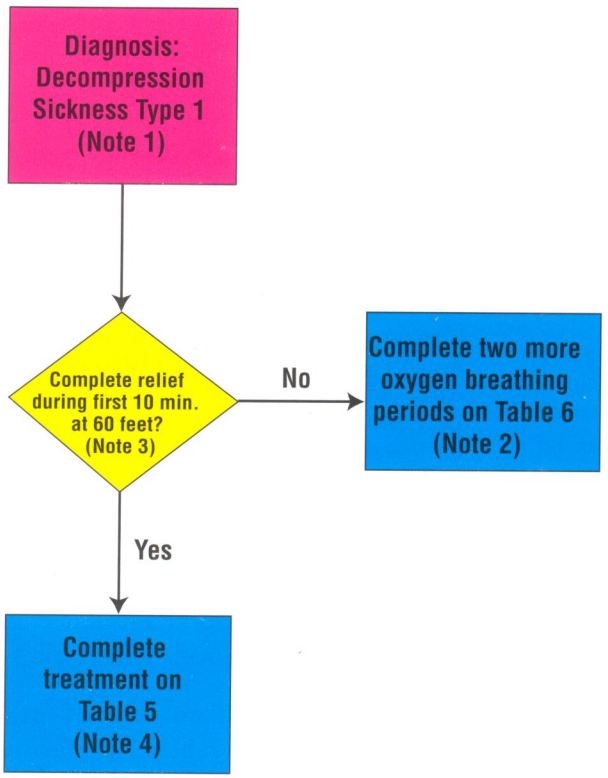


NOTES:

1. If 100% oxygen is available for divers with a helmet or full face mask, shift to this gas at depths of 30 fsw or less.
2. As an option, the Diving Supervisor 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.
3. If recompression goes deeper than the depth of the first stop in the Decompression Table use a stop time equal to 1 1/2 times the first stop in the Decompression Table for the one stop deeper than the first stop. Always take a stop every 10 feet.
4. No oxygen available.

FLOW CHART 2

Treatment of Type I Decompression Sickness Treatment from Diving or Altitude Exposures

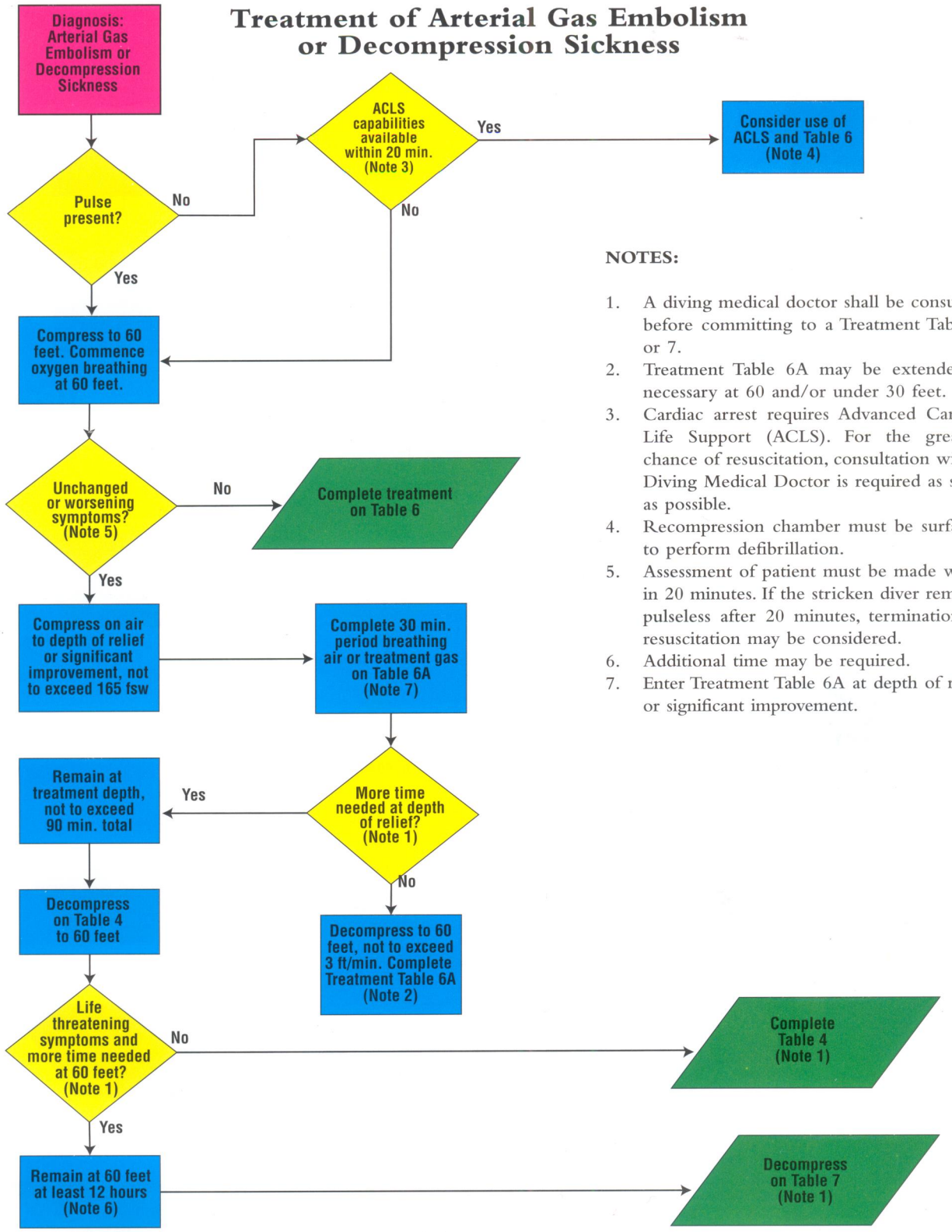


NOTES:

1. If a complete neurological exam was not completed before recompression, treat as a Type II symptom.
2. Treatment Table 6 may be extended up to four additional oxygen-breathing periods, two at 30 feet and/or two at 60 feet.
3. Diving Supervisor may elect to treat on Treatment Table 6.
4. Treatment Table 5 may be extended two oxygen-breathing periods at 30 fsw.

FLOW CHART 3

Treatment of Arterial Gas Embolism or Decompression Sickness

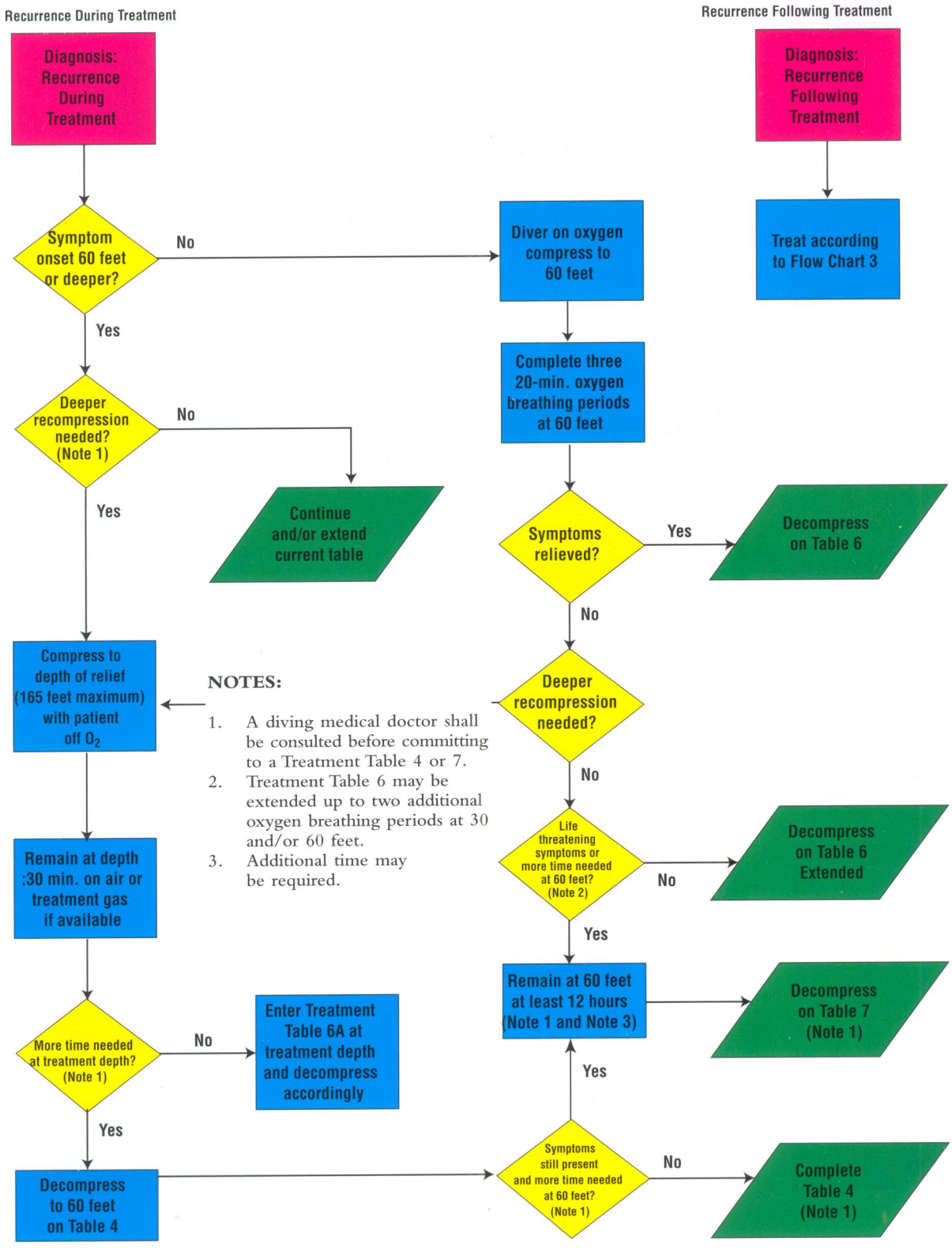


NOTES:

1. A diving medical doctor shall be consulted before committing to a Treatment Table 4 or 7.
2. Treatment Table 6A may be extended if necessary at 60 and/or under 30 feet.
3. Cardiac arrest requires Advanced Cardiac Life Support (ACLS). For the greatest chance of resuscitation, consultation with a Diving Medical Doctor is required as soon as possible.
4. Recompression chamber must be surfaced to perform defibrillation.
5. Assessment of patient must be made within 20 minutes. If the stricken diver remains pulseless after 20 minutes, termination of resuscitation may be considered.
6. Additional time may be required.
7. Enter Treatment Table 6A at depth of relief or significant improvement.

FLOW CHART 4

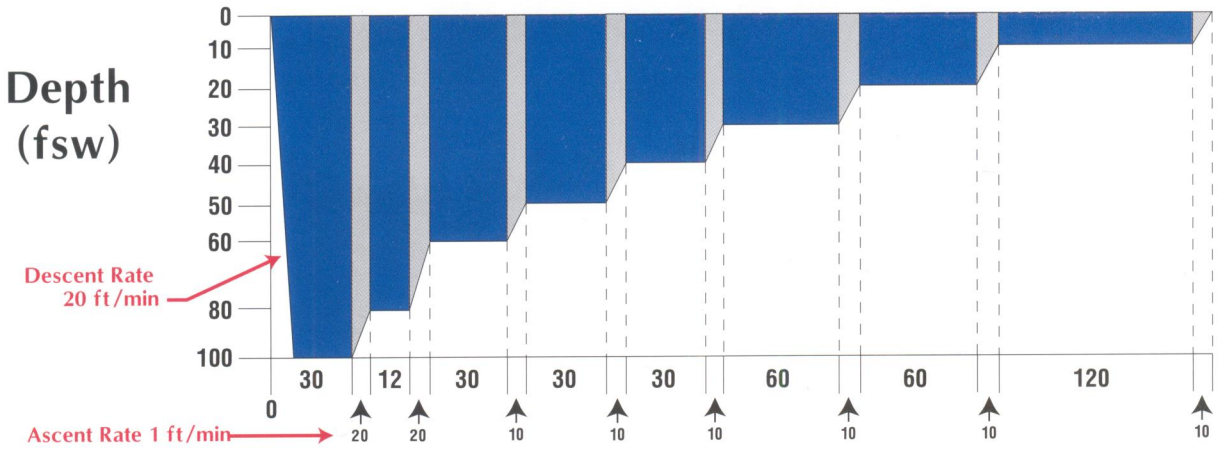
Treatment of Symptom Recurrence



AIR TREATMENT TABLE 1A

1. Descent rate — 20 ft / min.
2. Ascent rate — 1 ft / min.
3. Time at 100 feet includes time from the surface.

Depth / Time Profile



Time at Depth (minutes)

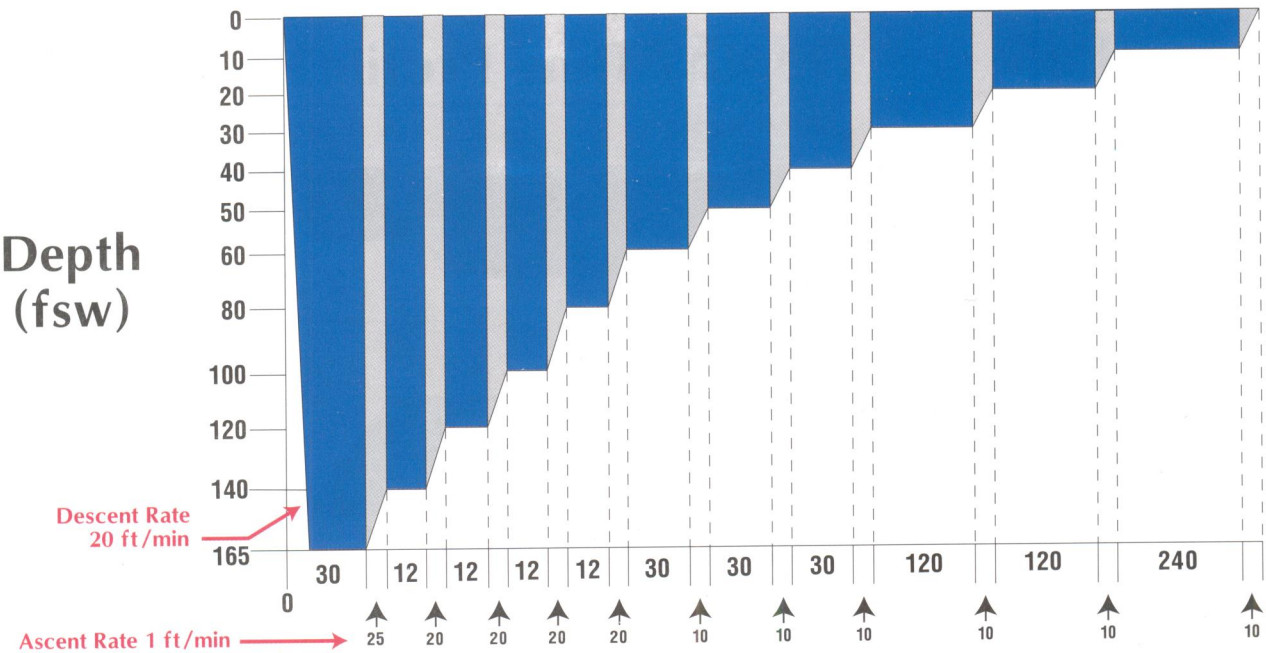
Total Elapsed Time: 472 Minutes (7 hours 52 minutes)

Depth (feet)	Stop Time (minutes)	Ascent Time (minutes)	Breathing Media	Total Elapsed Time (hrs:min.)
100	30		Air	0:30
80	12	20	Air	1:02
60	30	20	Air	1:52
50	30	10	Air	2:32
40	30	10	Air	3:12
30	60	10	Air	4:22
20	60	10	Air	5:32
10	120	10	Air	7:42
0		10	Air	7:52

AIR TREATMENT TABLE 2A

1. Descent rate — 20 ft / min.
2. Ascent rate — 1 ft / min.
3. Time at 165 feet includes time from the surface.

Depth / Time Profile



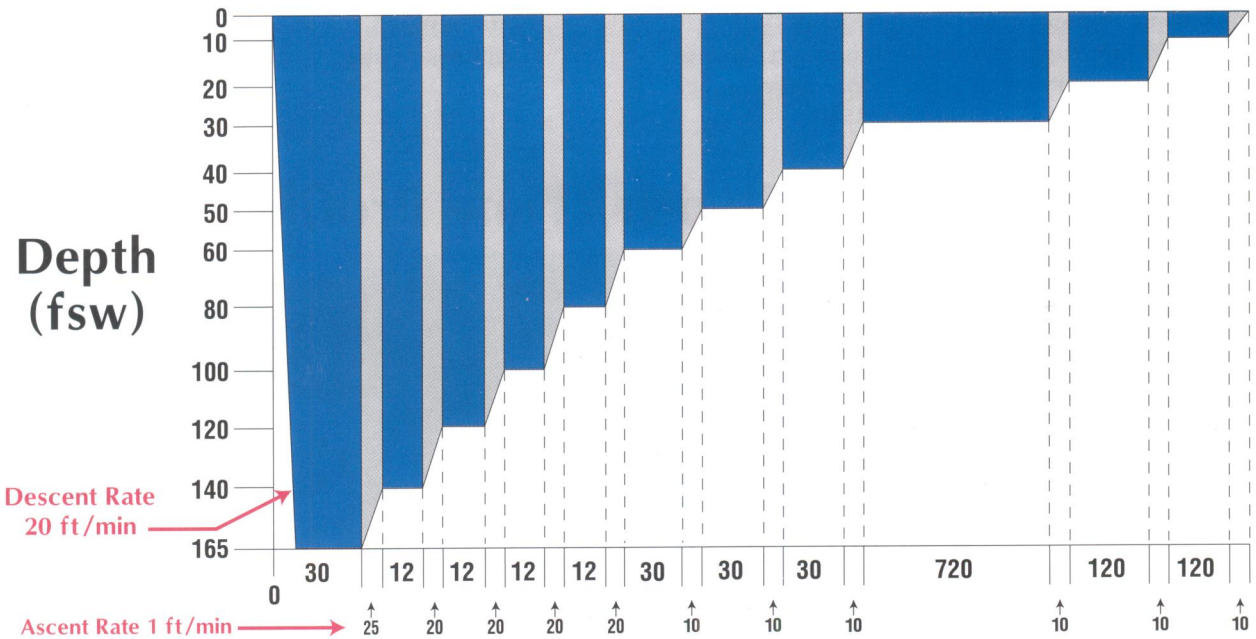
Total Elapsed Time: 813 Minutes (13 hours 33 minutes)

Depth (feet)	Stop Time (minutes)	Ascent Time (minutes)	Breathing Media	Total Elapsed Time (hrs:min.)
165	30		Air	0:30
140	12	25	Air	1:07
120	12	20	Air	1:39
100	12	20	Air	2:11
80	12	20	Air	2:43
60	30	20	Air	3:33
50	30	10	Air	4:13
40	30	10	Air	4:53
30	120	10	Air	7:03
20	120	10	Air	9:13
10	240	10	Air	13:23
0		10	Air	13:33

AIR TREATMENT TABLE 3

1. Descent rate — 20 ft/min.
2. Ascent rate — 1 ft/min.
3. Time at 100 feet includes time from the surface.

Depth / Time Profile



Time at Depth (minutes)

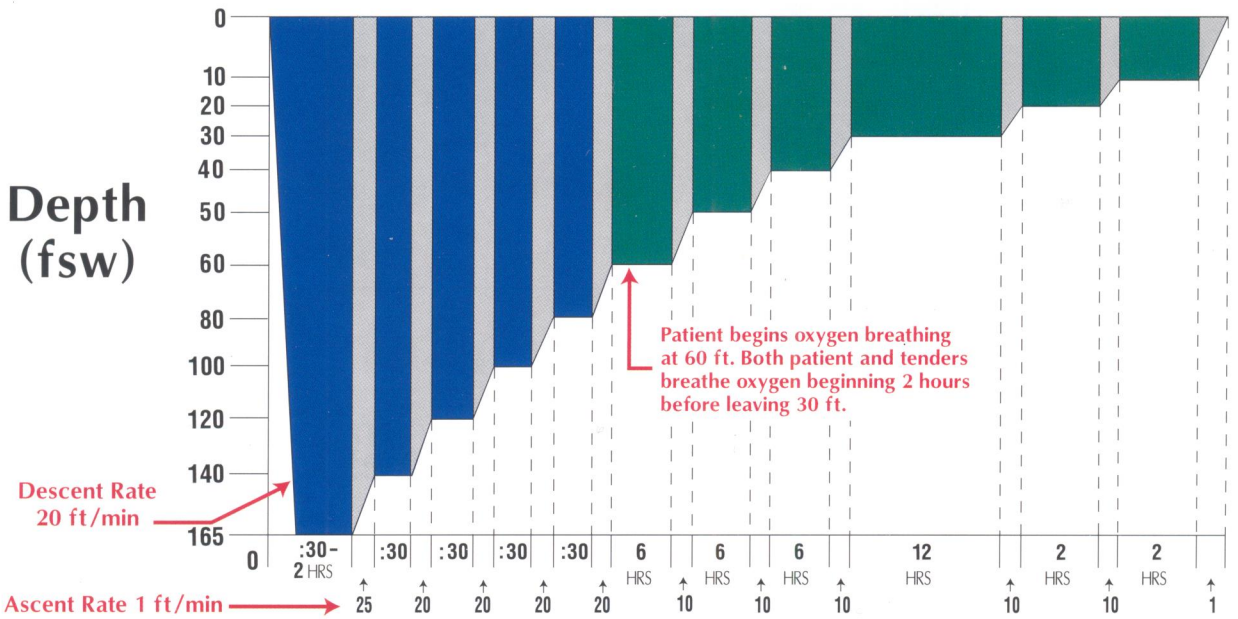
Total Elapsed Time: 1293 Minutes (21 hours 33 minutes)

Depth (feet)	Stop Time (minutes)	Ascent Time (minutes)	Breathing Media	Total Elapsed Time (hr:min.)
165	30		Air	0:30
140	12	25	Air	1:07
120	12	20	Air	1:39
100	12	20	Air	2:11
80	12	20	Air	2:43
60	30	20	Air	3:33
50	30	10	Air	4:13
40	30	10	Air	4:53
30	720	10	Air	17:03
20	120	10	Air	19:13
10	120	10	Air	21:23
0		10	Air	21:33

TREATMENT TABLE 4

1. Descent rate — 20 ft/min.
2. Ascent rate — 1 ft/min.
3. Time at 165 feet includes time from the surface.
4. 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 two hours before leaving 30 feet.
5. Ensure life support considerations can be met before committing to Table 4. Internal chamber temperature should be below 85° F.
6. If oxygen breathing is interrupted, no compensatory lengthening of the table is required.
7. If switching from Treatment Table 6A or 3 at 165 feet, stay a maximum of two hours at 165 feet before decompressing.
8. If the chamber is equipped with a high-O_2 treatment gas, it may be administered at 165 fsw, not to exceed 2.8 ata O_2. Treatment gas is administered for 25 minutes interrupted by 5 minutes of air.

Depth / Time Profile



Time at Depth (minutes)

Total Elapsed Time:
39 Hours 6 Minutes
(30 minutes at 165 fsw)
to 40 hours 36 minutes
(2 hours at 165 fsw)

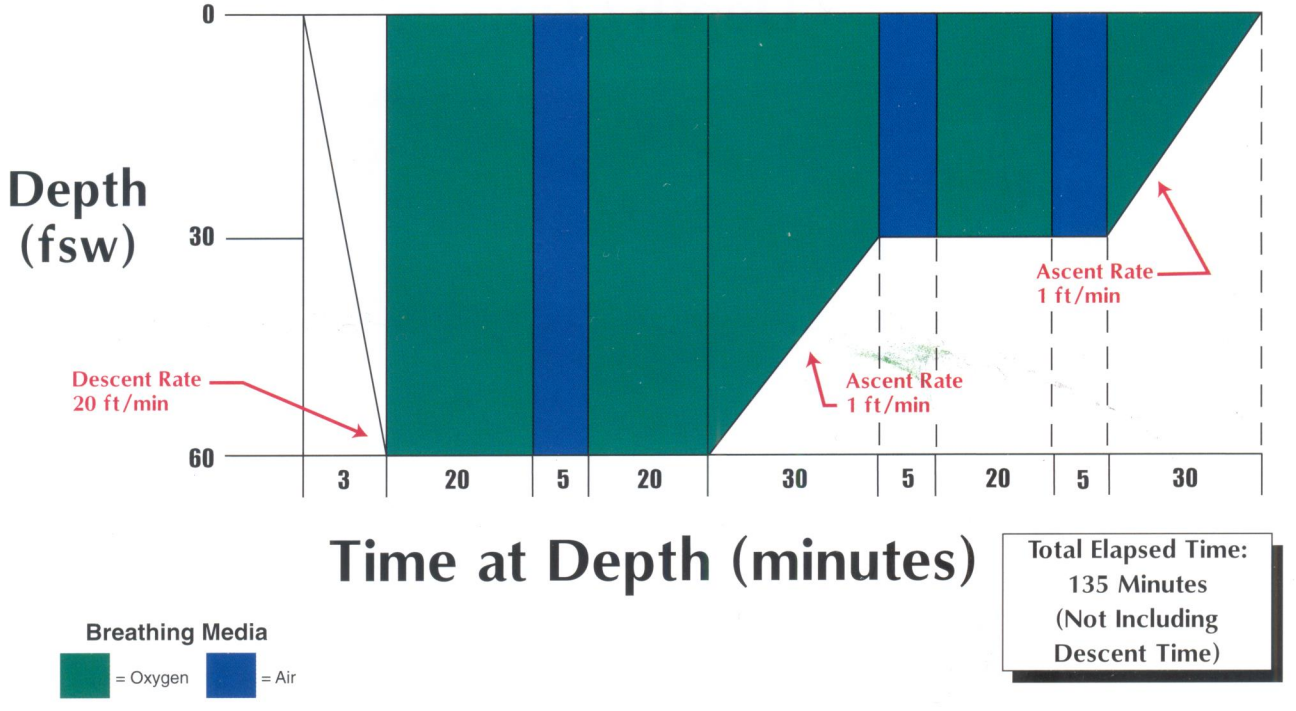
Breathing Media

= Oxygen = Air

TREATMENT TABLE 5

1. Descent rate — 20 ft/min.
2. Ascent rate — Not to exceed 1 ft/min. Do not compensate for slower ascent rates. Compensate for faster rates by halting the ascent.
3. Time on oxygen begins on arrival at 60 feet.
4. If oxygen breathing must be interrupted because of CNS Oxygen Toxicity, allow 15 minutes after the reaction has entirely subsided and resume schedule at point of interruption. *
5. Treatment Table may be extended two oxygen-breathing periods at the 30-foot stop. No air break required between oxygen-breathing periods or prior to ascent.
6. Tender breathes 100% O_2 during ascent from the 30-foot stop to the surface. If the tender had a previous hyperbaric exposure in the previous 12 hours, an additional 20 minutes of oxygen breathing is required prior to ascent.

Depth / Time Profile

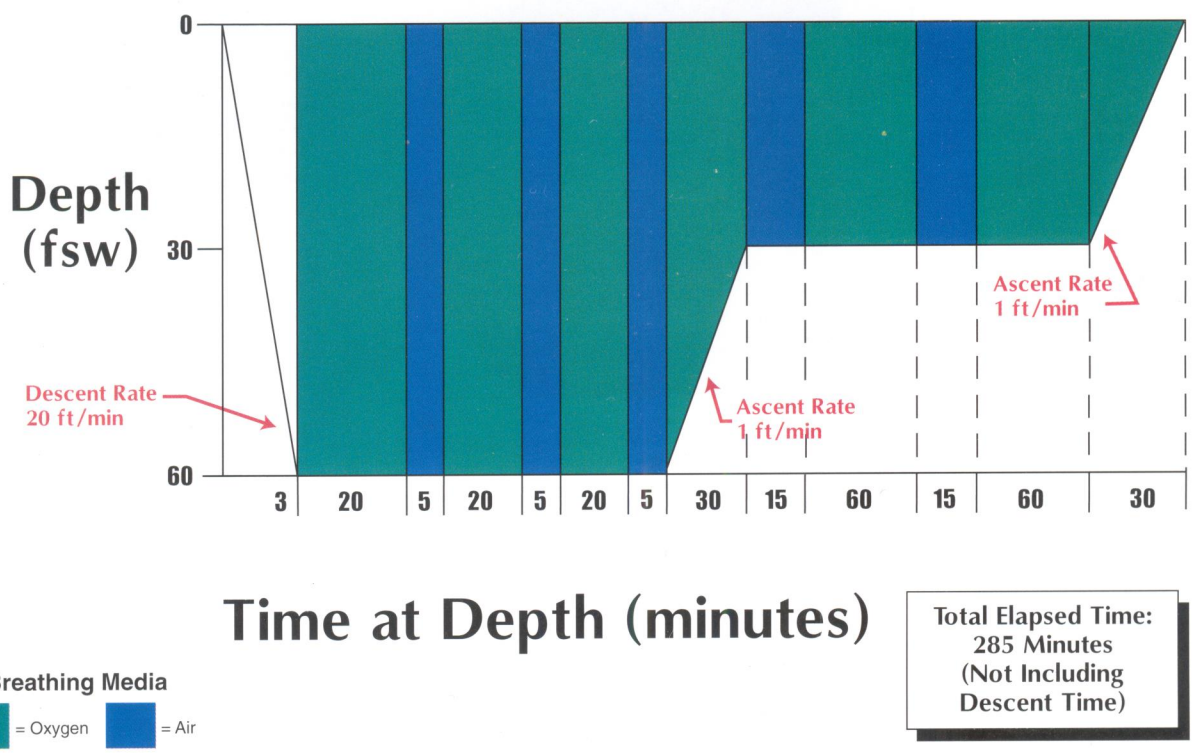


*** Procedures In the Event of Oxygen Toxicity.** At the first sign of CNS oxygen toxicity, the patient should be removed from oxygen and allowed to breathe chamber air. Oxygen breathing may be restarted 15 minutes after all symptoms have subsided. If symptoms of CNS oxygen toxicity develop again, interrupt oxygen breathing for another 15 minutes. If CNS oxygen toxicity develops a third time, contact a Diving Medical Doctor as soon as possible to modify oxygen breathing periods to meet requirements.

TREATMENT TABLE 6

1. Descent rate — 20 ft / min.
2. Ascent rate — Not to exceed 1 ft/min. Do not compensate for slower ascent rates. Compensate for faster rates by halting the ascent.
3. Time on oxygen begins on arrival at 60 feet.
4. If oxygen breathing must be interrupted because of CNS Oxygen Toxicity, allow 15 minutes after the reaction has entirely subsided and resume schedule at point of interruption. *
5. Table 6 can be lengthened up to two additional 25–minute periods at 60 feet (20 minutes on oxygen and five 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.
6. Tender breathes 100% O_2 during the last 30 minutes at 30 fsw and during ascent to the surface for an unmodified table or where there has been only a single extension at 30 or 60 feet. If there has been more than one extension, the O_2 breathing at 30 feet is increased to 60 minutes. If the tender has had a hyperbaric exposure within the past 12 hours, an additional 60–minute O_2 period is taken at 30 feet.

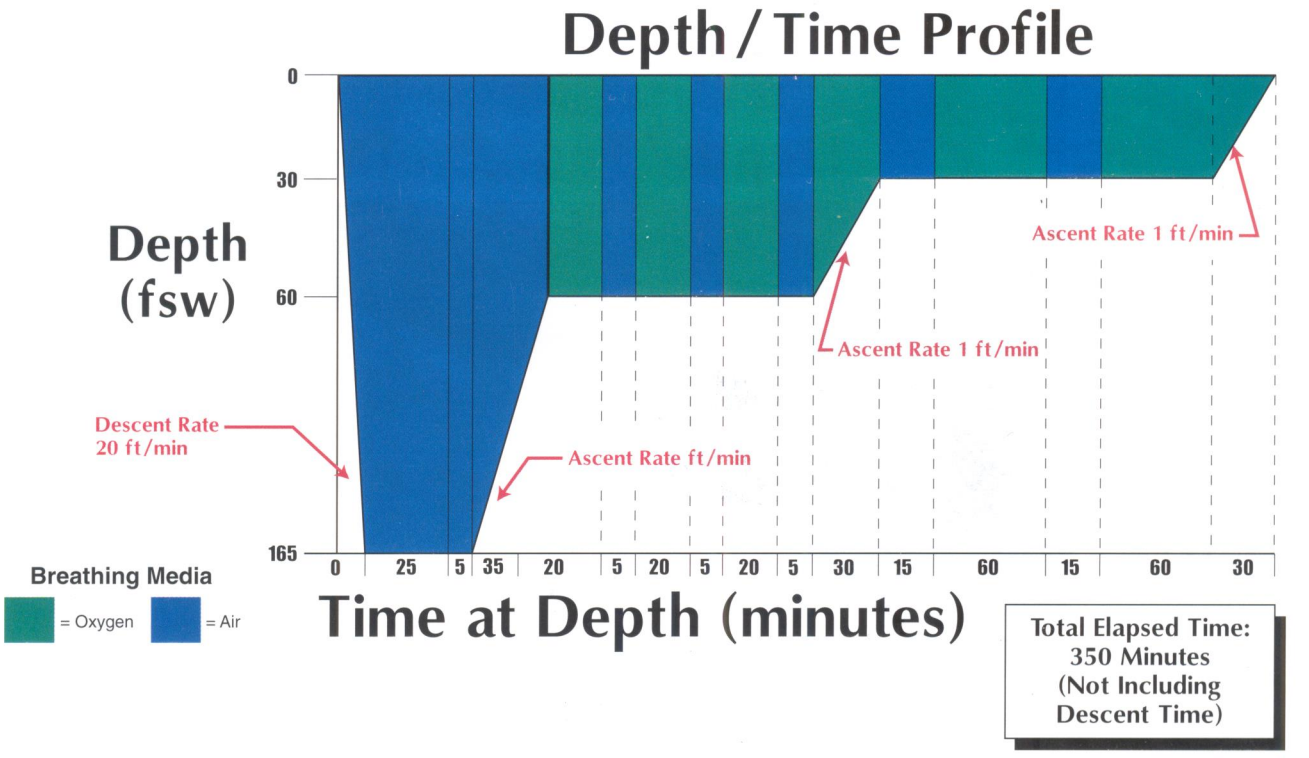
Depth / Time Profile



*** Procedures In the Event of Oxygen Toxicity.** At the first sign of CNS oxygen toxicity, the patient should be removed from oxygen and allowed to breathe chamber air. Oxygen breathing may be restarted 15 minutes after all symptoms have subsided. If symptoms of CNS oxygen toxicity develop again, interrupt oxygen breathing for another 15 minutes. If CNS oxygen toxicity develops a third time, contact a Diving Medical Doctor as soon as possible to modify oxygen breathing periods to meet requirements.

TREATMENT TABLE 6A

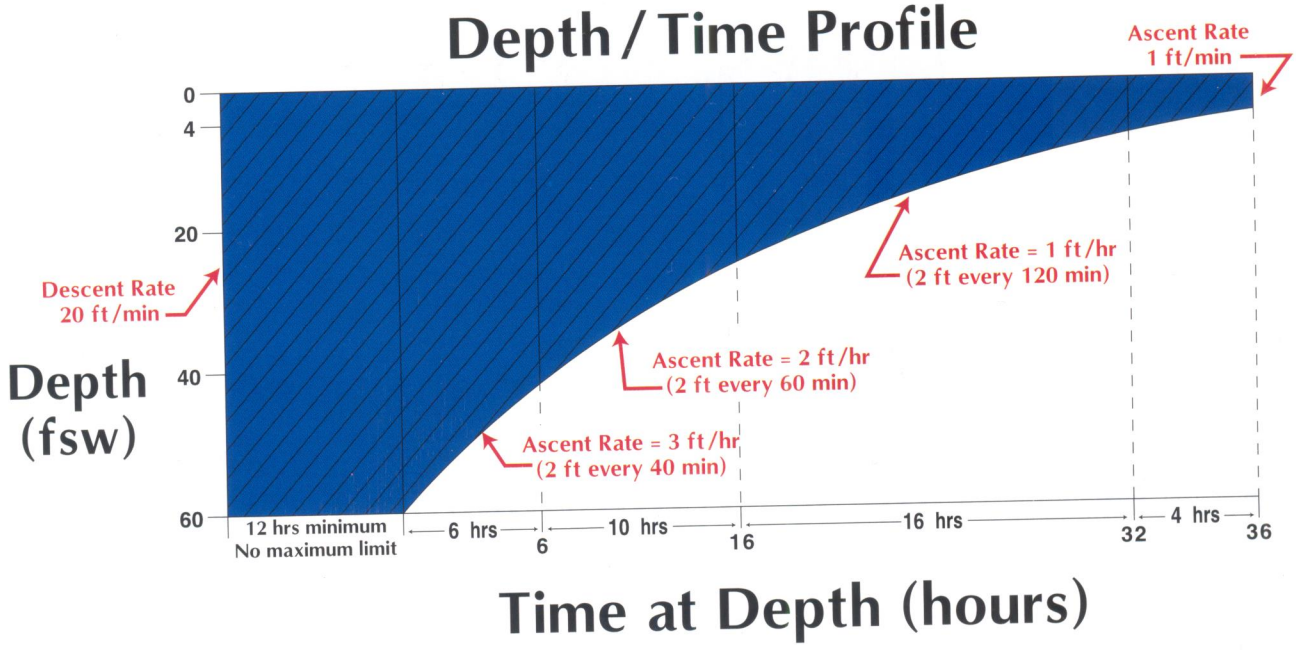
1. Descent rate — 20 ft/min.
2. Ascent rate — 165 fsw to 60 fsw, not to exceed 3 ft/min, 60 fsw and shallower, not to exceed 1 ft/min. Do not compensate for slower ascent rates. Compensate for faster rates by halting the ascent.
3. Time at treatment depth does not include compression time.
4. Table begins with initial compression to depth of 60 fsw. If initial treatment was at 60 feet, up to 20 minutes may be spent at 60 feet before compression to 165 fsw. Contact a Diving Medical Doctor.
5. If a chamber is equipped with a high-O_2 treatment gas, it may be administered at 165 fsw and shallower, not to exceed 2.8 ata O_2. Treatment gas is administered for 25 minutes interrupted by five minutes of air. Treatment gas is breathed during ascent from the treatment depth to 60 fsw.
6. Deeper than 60 feet, if treatment gas must be interrupted because of CNS oxygen toxicity, allow 15 minutes after the reaction has entirely subsided and resume schedule at point of interruption. *
7. Table 6A can be lengthened up to two additional 25-minute periods at 60 feet (20 minutes on oxygen and five 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.
8. Tender breathes 100% oxygen during the last 60 minutes at 30 fsw and during ascent to the surface for an unmodified table or where there has been only a single extension at 30 or 60 fsw. If there has been more than one extension, the O_2 breathing at 30 fsw is increased to 90 minutes. If the tender had a hyperbaric exposure within the past 12 hours, an additional 60 minute O_2 breathing period is taken at 30 fsw.
9. If significant improvement is not obtained within 30 minutes at 165 feet, consult with a Diving Medical Doctor before switching to Treatment Table 4.



*** Procedures In the Event of Oxygen Toxicity.** At the first sign of CNS oxygen toxicity, the patient should be removed from oxygen and allowed to breathe chamber air. Oxygen breathing may be restarted 15 minutes after all symptoms have subsided. If symptoms of CNS oxygen toxicity develop again, interrupt oxygen breathing for another 15 minutes. If CNS oxygen toxicity develops a third time, contact a Diving Medical Doctor as soon as possible to modify oxygen breathing periods to meet requirements.

TREATMENT TABLE 7

1. 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 20 ft/min to begin Table 7.
2. Maximum duration at 60 feet is unlimited. Remain at 60 feet a minimum of 12 hours unless overriding circumstances dictate earlier decompression.
3. Patient begins oxygen breathing periods at 60 feet. Tender should breathe only chamber atmosphere throughout. If oxygen breathing is interrupted, no lengthening of the table is required.
4. Minimum chamber O_2 concentration is 19%. Maximum CO_2 concentration is 1.5% SEV (11.4 mmHg). Maximum chamber internal temperature is 85^{O} F.
5. Decompression starts with a 2-foot upward excursion from 60 to 58 feet. Decompress with stops every two 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 four feet for four hours and then ascend to the surface at 1 ft/min.
6. Ensure chamber life-support requirements can be met before committing to Treatment Table 7.
7. A Diving Medical Doctor shall be consulted before committing to this treatment table.



TREATMENT TABLE 8

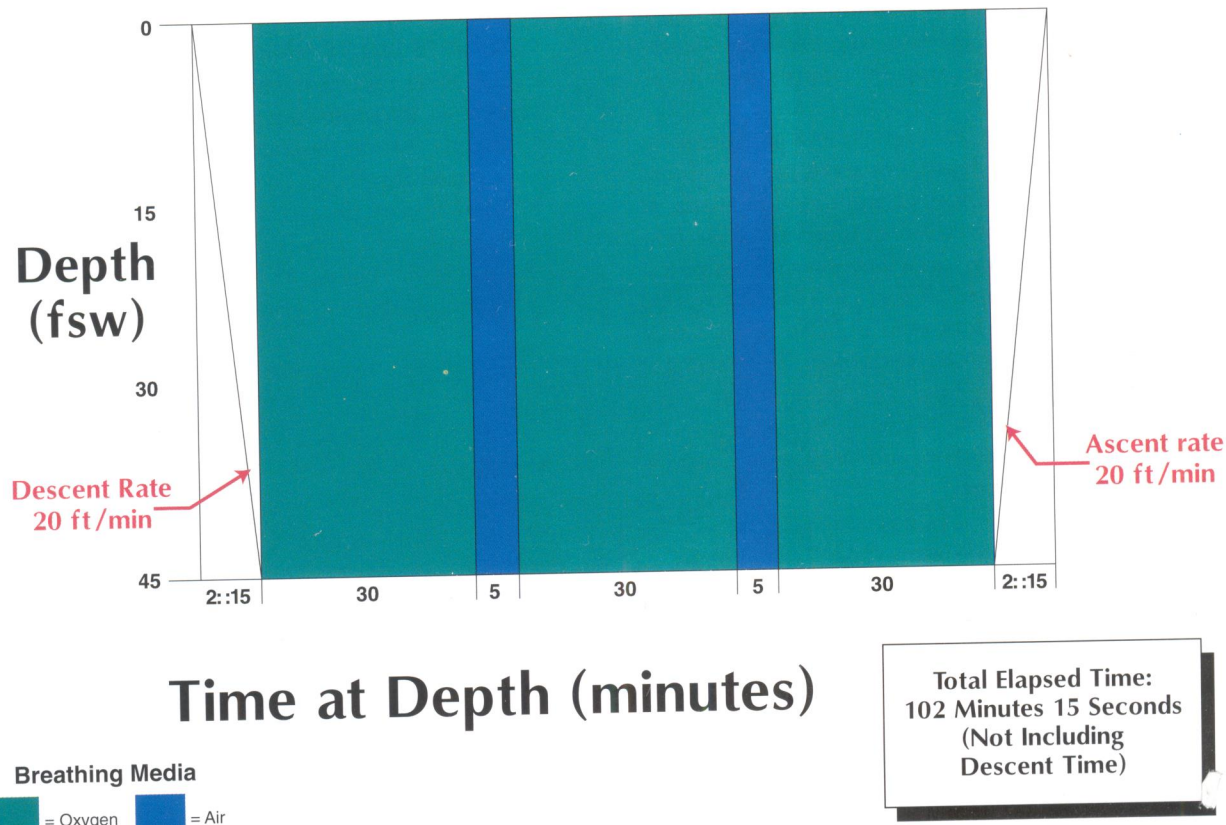
1. Enter the table at the depth which is exactly equal to or next greater than the deepest depth attained in the recompression. The descent rate is as fast as tolerable.
2. The maximum time that can be spent at the deepest depth is shown in the second column. The maximum time for 225 fsw is 30 minutes; for 165 fsw, 3 hours. For an asymptomatic diver, the maximum time at depth is 30 minutes for depths exceeding 165 fsw and 2 hours for depths equal to or shallower than 165 fsw.
3. Decompression is begun with a 2-fsw reduction in pressure if the depth is an even number. Decompression is begun with a 3-fsw reduction in pressure if the depth is an odd number. Subsequent stops are carried out every two fsw. Stop times are given in column three. The stop time begins when leaving the previous depth. Ascend to the next stop in approximately 30 seconds.
4. Stop times apply to all stops within the band up to the next quoted depth. For example, for ascent from 165 fsw, stops for 12 minutes are made at 162 fsw, and at every two-foot interval to 140 fsw. At 140 fsw, the stop time becomes 15 minutes. When traveling from 225 fsw, the 166 foot stop is 5 minutes; the 164–foot stop is 12 minutes. Once begun, decompression is continuous. For example, when decompressing from 225 feet, ascent is not halted at 165 fsw for 3 hours. However, ascent maybe halted at 60 fsw and shallower for any desired period of time.
5. While deeper than 165 fsw, a helium-oxygen mixture with 16–21% oxygen may be breathed by mask to reduce narcosis. At 165 fsw and shallower, a heliox mix with a PO_2 not to exceed 2.8 ata may be given to the diver as a treatment gas. At 60 fsw and shallower, pure oxygen may be given to the diver as a treatment gas. For all treatment gases (HeO_2, N_2O_2, and O_2), a schedule of 25 minutes on gas and 5 minutes on chamber air should be followed for a total of four cycles. Additional oxygen may be given at 60 fsw after a 2-hour interval of chamber air. See Treatment Table 7 for guidance.
6. A high-O_2 treatment mix can be used at treatment depth and during decompression. If high O_2 breathing is interrupted, no lengthening of the table is required.
7. To avoid loss of the chamber seal, ascent may be halted at four fsw and the total remaining stop time of 240 minutes taken at this depth. Ascend directly to the surface upon completion of the required time.
8. Total ascent time from 225 fsw is 56 hours, 29 minutes. For a 165-fsw recompression, total ascent time is 53 hours, 52 minutes, and for a 60-fsw recompression, 36 hours, 0 minutes.

Depth (fsw)	Max Time at Initial Treatment Depth (hours)	2-fsw Stop Times (minutes)
225	0.5	5
165	3	12
140	5	15
120	8	20
100	11	25
80	15	30
60	Unlimited	40
40	Unlimited	60
20	Unlimited	120

TREATMENT TABLE 9

1. Descent rate — 20 ft/min.
2. Ascent rate — 20 ft/min. Rate may be slowed to 1 ft/min depending upon the patient's medical condition.
3. Time at 45 feet begins on arrival at 45 feet.
4. If oxygen breathing must be interrupted because of CNS Oxygen Toxicity, oxygen breathing may be restarted 15 minutes after all symptoms have subsided. Resume schedule at point of interruption. *
5. Tender breathes 100% O_2 during last 15 minutes at 45 feet and during ascent to the surface regardless of ascent rate used.
6. If patient cannot tolerate oxygen at 45 feet, this table can be modified to allow a treatment depth of 30 feet. The oxygen breathing time can be extended to a maximum of three to four hours.

Depth / Time Profile



* **Procedures in the Event of Oxygen Toxicity.** At the first sign of CNS oxygen toxicity, the patient should be removed from oxygen and allowed to breathe chamber air. Oxygen breathing may be restarted 15 minutes after all symptoms have subsided. If symptoms of CNS oxygen toxicity develop again, interrupt oxygen breathing for another 15 minutes. If CNS oxygen toxicity develops a third time, contact a Diving Medical Doctor as soon as possible to modify oxygen breathing periods to meet requirements.

APPENDIX B

FORMULAS CALCULATIONS TABLES

1 pound per square inch (psi) =
- 2.31 feet of fresh water (ffw)
- 2.25 feet of sea water (fsw)
- 0.068 atm
- 2.036 inches mercury
- 5.17cm mercury

1 atmosphere =
- 14.7 psi
- 22.92 inches mercury
- 33.9 feet of fresh water
- 33 feet sea water
- 1.013 bar
- 760mm mercury

1 foot of sea water= 0.445psi

Atm=Depth/33
ATA=(Depth/33) +1

To convert feet to meters
- Feet/3.28=Meters

To convert meters to feet
- Meters x 3.28=Feet

To convert kilograms to pounds
- kg x 2.2=pounds

To convert pounds to kilogram
- pounds/2.2=kilograms

To convert Fahrenheit to Celsius
- °C=5/9 x (°F-32)

To convert Celsius to Fahrenheit
- °F=(1.8 x °C) + 32

Sea level equivalent=
- 1/pressure (ATA) x Depth of Dive

Altitude (Feet)	Pressure (mm/Hg)	Pressure (ATA)
0	760	1.000
500	746	0.982
1,000	733	0.964
1,500	712	0.947
2,000	707	0.930
2,500	694	0.913
3,000	681	0.896
3,500	669	0.880
4,000	656	0.864
4,500	644	0.848
5,000	632	0.832
5,500	621	0.817
6,000	609	0.801
6,500	598	0.786
7,000	586	0.772
7,500	575	0.757
8,000	565	0.743
8,500	554	0.729
9,000	543	0.715
9,500	533	0.701
10,000	523	0.688
10,500	513	0.676
11,000	503	0.662
11,500	493	0.649
12,000	483	0.636
18,000	380	0.500

Relationship Between Altitude and Pressure

pO_2	Kp	pO_2	Kp	pO_2	Kp	pO_2	Kp
0.5	0.0	1.7	2.07	2.9	3.7	4.1	5.18
0.6	0.26	1.8	2.22	3.0	3.82	4.2	5.3
0.7	0.47	1.9	2.36	3.1	3.95	4.3	5.42
0.8	0.65	2.0	2.5	3.2	4.08	4.4	5.54
0.9	0.83	2.1	2.64	3.3	4.2	4.5	5.66
1.0	1.0	2.2	2.77	3.4	4.33	4.6	5.77
1.1	1.16	2.3	2.91	3.5	4.45	4.7	5.89
1.2	1.32	2.4	3.04	3.6	4.57	4.8	6.01
1.3	1.48	2.5	3.17	3.7	4.7	4.9	6.12
1.4	1.63	2.6	3.31	3.8	4.82	5.0	6.24
1.5	1.78	2.7	3.44	3.9	4.94		
1.6	1.93	2.8	3.57	4.0	5.06		

Kp factors for calculating UPTD

MEDICAL CONDITIONS THAT PRECLUDE DIVING

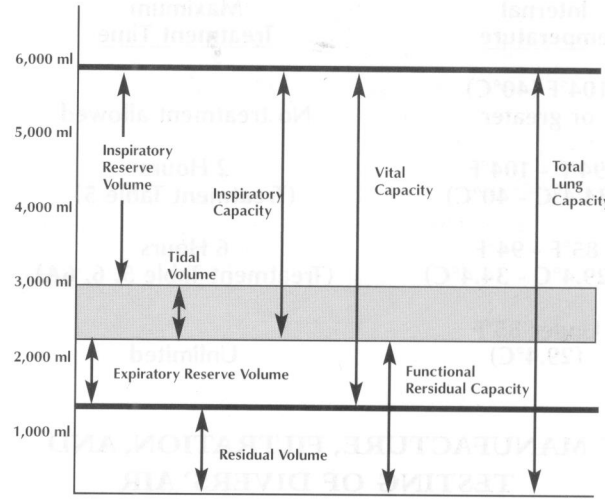
Absolute disqualifying conditions for diving

- Asthma – except childhood asthma that has not recurred since onset of adolescence
- Chronic cystic, obstructive or restrictive pulmonary disease, or chronic bronchitis
- Any history of spontaneous pneumothorax
- Seizure disorder or frequent syncope
- Insulin dependent diabetes
- Middle ear surgery with placement of prosthesis
- Sickle cell disease or trait
- Detached retina
- Patent foramen ovale/atrial septal defect
- **Ventricular septal defect**
- Recurrent labrynthitis
- Disk disease with neurological deficit
- Symptomatic bradycardic arrythmias
- Pregnancy

Relative Disqualifying Medical Conditions For Diving

- Closed head or spinal cord injury with sequelae
- Chronic perforation of tympanic membrane
- Chronic otitis media
- Chronic sinusitis
- Angina Pectoris
- Congestive heart failure
- Claustrophobia
- Chronic renal disease
- Heavy smoking
- Migraine headache
- Hernias
- Recent orthopedic injury
- Radiation therapy (absolute if it involves the lung)
- Mitral Valve Prolapse (absolute if it involves failure or arrythmia)
- Wolfe-Parkinson-White arrythmia
- Possibility of being pregnant

Pulmonary Volumes and Capacities



BODY'S RESPONSE TO TEMPERATURE

Internal Temperature	Response
37°C (98.6°F)	Cold sensations, cutaneous vaso-constriction, ++ muscle tension, ++VO_2(oxygen consumption)
36°C (96.8°F)	Sporadic shivering, suppressed by voluntary movements, with some uncontrolled bouts, additional rise in VO_2
35°C (95.0°F)	Mental confusion, decreasing will to struggle
34°C (93.2°F)	Amnesia, poor articulation, sensory and motor degradation
33°C (91.4°F)	Hallucinations, delusions, clouding of consciousness, 50% risk of death
32°C (89.6°F)	Atrial arrythmias
31°C (87.8°F)	Failure to recognize familiar persons
30°C (86.0°F)	No response to pain, muscle rigidity, pupillary reflex lost
29°C (84.2°F)	Loss of consciousness
28°C (82.4°F)	DTRs (deep tendon reflexes) lost, ventricular arrythmias
27°C (80.6°F)	V-Fib likely, flaccid skeletal muscles

Chamber Operating Temperatures

Internal Temperature	Maximum Treatment Time
104°F (40°C) or greater	No treatment allowed
94°F - 104°F (34.4°C - 40°C)	2 Hours (Treatment Table 5)
85°F - 94°F (29.4°C - 34.4°C)	6 Hours (Treatment Table 5, 6, 6A)
Under 85°F (29.4°C)	Unlimited

MANUFACTURE, FILTRATION, AND TESTING OF DIVERS' AIR

CONSTITUENT	FED SPEC
Oxygen (O_2) (percent by volume)	20-22%
Carbon Dioxide (CO_2) (by volume)	500ppm (max)
Carbon Monoxide (CO) (by volume)	10ppm (max)
Total Hydrodrocarbons (as CH_4) (by volume)	25ppm (max)
Oil, Mist, Particulates (weight/volume)(max)	.005mg/L
Odor (not pronounced or objectionable)	
Separated Water	none
Total Water (weight/volume) (max)	0.02mg/L
Nitrogen Dioxide (NO_2) (by volume)	2.5ppm (max)
Nitrous Oxide (N_2O) (by volume)	2.0ppm (max)
Sulfur Dioxide (SO_2) (by volume)	2.5ppm (max)
Halogenated Compounds (by volume)	
Refrigerants	2.0ppm (max)
Solvents	0.2ppm (max)
Acetylene (C_2H_2)	0.1ppm (max)
Ethylene (C_2H_4)	0.4ppm (max)