IN-WATER RECOMPRESSION

The Forty-eighth Workshop of the Undersea and Hyperbaric Medical Society

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The Society, faculty, and attendees at the workshop would like to record their appreciation to Dr. Peter B. Bennett and the Divers Alert Network (DAN) for financial support of the highly important workshop.

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Tribute to Ed Beckman

Edward L. Beckman MD was a friend to many, a devoted family man, and admired worldwide for his dedication to scientific detail, and cheerful encouragement of his colleagues and students. He had an abiding interest in the physiology of physical stresses on the human body, beginning his scientific career at the University of Southern California Los Angeles in the Department of Aviation Medicine. He subsequently held appointments at The Naval Air Development Center in Johnsvtlle, Pennsylvania; The University of Pennsylvania Graduate School; School of Aviation Medicine at The Naval Air Station, Pensacola, Florida; Senior Medical Officer, USS INTREPID; Institute of Aviation Medicine, Farnborough, England; Naval Air Development Center, Johnsville, Pennsylvania; Naval Medical Research Institute, Bethesda, Maryland; University of Texas School of Public Health; University of Texas A&M College of Marine Sciences; and Baylor College of Medicine.

He published his first papers on acceleration physiology. This interest rapidly expanded to the effectiveness of anti G suits, aircraft ejection mechanics, launch and reentry of astronauts, weightlessness. fluid replacement during water immersion, homeostasis in the long distance underwater swimmer, wet and dry suites, suite warmers for cold exposure, and the stress of 100% oxygen exposure for long periods. In his later years he devoted his scientific talents to the problems and treatment of decompression sickness, osteonecrosis, and spinal cord injury. One cannot review these subjects without encountering seminal papers by E. L. Beckman.

Dr. Beckman's honors include The Liliencrantz Award of the Aerospace Medicine Association, US Navy Unit Commendation for work on SEALAB II and the US Navy Unit Commendations for work on Tektite I. He was a founding member of the Undersea Medical Society. It is most appropriate this Workshop on In Water Recompression Treatment be dedicated to Dr. Beckman not only because of his many contributions to undersea medicine but because he conceived of this workshop prior to his untimely death while serving as Medical Director at the University of Hawaii School of Medicine Hyperbaric Treatment Center.

Merrill P. Spencer MD

OPENING REMARKS

Merrill Spencer, M.D.

But here in Seattle, this symposium would not have occurred had it not been by the good offices and enthusiasm of Dr. Ed Kay.

I had the good fortune of having Ed take this up and work on it very hard, and also we'd like to thank all the speakers that have come from many miles to participate in what promises to be a very interesting discussion and presentations.

Ed, thank you very much for your organization.

(Applause)

Dr. Kay: Well, it's nice to see that we have such a good turn-out today. We had also scheduled a briefing on some of the legal aspects of in-water recompression, and the trial Rick Lesser is running didn't finish in time. So, he's not going to be here either, but we'll pick up on some of these issues as best we can later on.

Well, of course, the issue in-water recompression is very complex. As a treatment modality, it's really more of a conundrum. What Dr. Spencer and I've tried to do is to break down the problem into its component parts, into smaller, more manageable parts, like exposure, and even consider unusual things, like predation maybe, and certainly gas supply is important, how much oxygen do you need to recompress a diver, and what about oxygen toxicity?

Well, this conference -- talk about the important people in this conference. I'd be very remiss if I didn't recognize Peter Bennett and the excellent support from the Divers Alert Network, and whenever you see Peter Bennett, I want you to stop him in the hall and shake his hand and tell him thanks for giving us that $5,000 to get this conference on the road.

Well, you know, there's -- there's a movement afoot in medicine. It's called Evidence-Based and Evidence-Based Approach to Investigation, and this is not a new concept, but I think the emphasis on evidence-based medicine is. It is new.

We're going to be hearing a lot today from the experts, and I just wanted to point out for a minute that the experts are one the fifth tier of evidence-based grading, and as you go up in the quality of your information, you know, you go from randomized -- from non-randomized trials to randomized trials, and, of course, we don't have anything like that in terms of high-quality evidence to discuss, to clarify the issues in in-water recompression, but we're going to do the best we can to bring to the fore the evidence which is available, and hopefully a conference like this will initiate more enthusiasm.

Of course, our goals are to initiate that scientific scrutiny, to clarify these issues as best we can, recognizing the quality of the evidence that's available.

We want to identify the dangers of this procedure, and to, if possible, provide some standards for the diving community.

The guidelines and the proceedings will be published, and the proceedings will be available through the Undersea and Hyperbaric Medical Society for a nominal fee. I think it's $20, and, of course, today, we're here to recognize the work of Ed Beckman.

I'd like to start the conference by introducing Carl Edmonds. Carl, of course, is a noted author, and he is the Director -- former Director of the Diving Medical Center at Sidney. He's the former officer-in-charge of the Royal Australian Navy School of Underwater Medicine and past president of the South Pacific Underwater Medical Society. He is semi-retired, but we brought him out of retirement to talk to us today.

AUSTRALIAN UNDERWATER OXYGEN TREATMENT OF DCS

Carl Edmonds, Diving Medical Centre, Sydney

ABSTRACT

The problem of decompression sickness (DCS) in remote areas is described with particular reference to Australia and the Indo-Pacific islands. The various approaches of medevac, and underwater air, surface and underwater oxygen (UW O_2) are addressed and the techniques and equipment used in underwater oxygen therapy are documented.

The favourable experience with the original UW O_2 tables are compared with the less conservative, more hazardous, oxygen decompressions used by some abalone divers and the shorter but still successful exposures of the pearl divers. The latter imply that, with very prompt treatment, the 9 metre oxygen treatment may be reduced in duration.

The UW O_2 treatment table is an application, and a modification, of historical and current beliefs. It is not meant to replace recompression therapy in chambers. It is an emergency procedure, able to be applied with equipment usually found in remote localities and is designed to reduce the hazards associated with the conventional underwater air treatments.

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BACKGROUND

The three Australian diving groups that contributed to our experience in underwater treatment of DCS included -

- Pearl divers - in our Northern warmer waters.
- Abalone and other shell divers - in our Southern colder waters.
- Recreational divers - around our 20,000 km coastline and the surrounding tropical Indo-Pacific islands.

Pearl divers[1-5]

The Pearling Industry was established in 1866, in North Western Australia. Four years later it was commenced in the Torres Strait, in Northern Queensland. Then pearl grounds were discovered off Darwin, in our Northern Territory. Thus the whole of our North, with its warm waters and strong currents, was exploited for pearl and pearl shell.

During that time we lost almost 1,000 pearl divers, mainly from DCS.

The traditional pearl divers would sail many days out from the pearling port, dive most of the day, alternating their equipment with another diver, from dawn to dusk, from 7-20 fathoms (14-40 metres) deep, but sometimes deeper.

Some brilliant clinical descriptions of DCS were made by both the Naval Surgeon, Bassett-Smith[6], in 1892, and District Medical Officer in Broome, Dr Graham Blick[7], in 1909. Dr Blick looked after some 400 pearl divers, and described both clinical and autopsy findings.

In 1912 the Royal Navy brought some engine driven compressors to the area, increasing the depths accessible to diving, as well as the incidence of DCS. They also introduced some knowledge of decompression staging - including underwater regimes for treatment with compressed air.

Although the Royal Navy and other European groups did not survive the adverse environmental and diving conditions, the Japanese adapted the techniques and equipment, until 1975. Then, after more than 100 years of pearl diving in Australia, the last helmet dive was performed out of Broome. From then on the Australians, using surface supply breathing apparatus, have exploited these pearl fields .

Despite the high death rate, and even greater morbidity, the diving performed was well in excess of that believed possible by diving authorities. Thus they attracted a series of brilliant researchers and observers. These included Blick[7], Hugh Le Messurier and Brian Hills[8,9] to supplement the superb historians such as Sister Mary Bain[1], Hugh Edwards[2,3] and Ian Idriess[4,5].

In more recent years, Nishi[10], Wong[11] and Edmonds[12] have extended some of these observations and investigations.

Because of the prolonged delay in obtaining access to recompression facilities, and the fact it could take days to reach shore, the divers were obligated to recompress underwater, using compressed air. Also, the underwater air treatments had some degree of credibility, being described in both the Royal Navy and US Diving manuals, and diving texts such as Davis' *Diving and Submarine Salvage*[13].

The conditions for underwater air treatments are essentially based on empirical beliefs and the observation that exposure to greater pressure produced relief of symptoms, and a

gradual reduction in that pressure (ascent), permitted an asymptomatic return to the surface.

The technique was more applicable to this diving group because of the continuous supply of adequate compressed air (good quality compressors and men who knew how to use them), a relatively warm water environment and the ultimate in a full face mask - a metal helmet.

Some dramatic and impressive therapeutic successes were achieved, in cases that would appall most diving physicians. The severity of the cases was usually related to the extensive duration and pressure exposure, inducing severe DCS, sometimes aggravated by a "blow-up" or explosive decompression. Some cases were treated for between 36 hours and 3 days underwater, and often with a concomitant urinary catheterization required at sea, by sailors with no medical training.

Most of the severe cases presented with either unconsciousness or spinal DCS. The majority of the muscular-skeletal cases were treated only for a matter of a few hours. Treatment, fully or partially successful, often permitted the diver to continue his diving activities on subsequent days.

By 1981 the death rate was down to 1, the lowest on record. This was partly due to the more conservative diving practices (although the divers still dive well in excess of that permitted by the conventional tables), and the more efficient treatment - essentially underwater recompression.

Abalone divers[14]

Although abalone shells were collected both by Aboriginal and other Australians, the commercial abalone industry was only developed in 1962. It was mainly restricted to the southern half of Australia, where the colder waters encouraged the growth of this shellfish.

Many of the original divers were of a hardy nature, and were either untrained or extended their spear fishing and breath hold diving skills into this field. The lack of knowledge resulted in a great number of diving accidents, and information from *Australian Fisheries*[15], a government magazine, claimed that 60% of these divers suffered from partial deafness, 50% from lung damage, 12% from dysbaric osteonecrosis and 12% from "arthritis".

Again, because most of the abalone diving is carried out distant from major port facilities, and because there is usually only one or two people on board the vessel, DCS tended either to not be treated, or treated using underwater air by empirical or traditional schedules.

Gradually divers from the Royal Australian Navy, following their discharge from active duty, tended to move into the industry, increasing to some degree the knowledge of decompression staging.

The abalone divers shared, with their pearl diving brethren, a wildness of nature, disrespect for authority and a lack of faith in the ability of medical staff to assist them in the treatment of their illnesses.

Considering the relative paucity of recompression chambers in the 1960's (one chamber on each coast of Australia), distant from both pearl and abalone areas, their skepticism was probably justified.

Recreational divers[16]

During the mid 1960's Australia had one established recompression chamber (RCC), capable of applying conventional treatments in Sydney at the Royal Australian Navy (RAN). There was one other chamber, less reliable and requiring both courage and faith, at Fremantle in Western Australia. The RAN were committed to supplying the medical treatment for all divers; civilian, commercial and armed forces. The catchment area extended around Australia, for thousands of kilometres, encompassing many Indo-Pacific islands, on an *ad hoc* basis.

Recreational diving became wide-spread around Australia, especially on the Great Barrier Reef in Queensland, and the equally impressive reefs of Western Australia and the Indo-Pacific islands to our North. Unfortunately most of these diving areas were well away from major recompression facilities, and even aviation facilities. Much of the diving was performed on live-a-board boats or from islands. The latter were often nothing more than very small coral cays.

The RAN accepted responsibility for treatment of civilians in 1965, in lieu of any alternative, from most of the surrounding Indo-Pacific region. From 1968, to reduce the delay if the diver was significantly injured, we were as likely to take all the equipment (portable chambers, oxygen, appliances, etc.) to him, as we were to bring him to the chamber. It all depended on which was the quickest. We preferred RAAF Hercules aircraft, pressurised to 1 atmosphere, for transport. Only serious cases warranted medevac from such distances.

The conventional chamber treatment regimes were often inadequate to produce successful results in these cases. The initial treatments, involving recompression with air at a minimum depth of 30 metres and more frequently at 50 metres, were grossly inadequate.

The first case on which my assistance was sought was for the diving physician who had, that day, treated a diver in the recompression chamber. The fact that the diver/patient improved, while the physician got bent, did not inspire confidence in the reliability of the treatment table. Nor did a review of the success of the other cases treated conventionally.

DEVELOPMENT OF OXYGEN THERAPY

Surface oxygen[16]

In 1968 we started using surface oxygen whilst awaiting recompression, during the inevitable delays. Thus the diver would receive oxygen in transit to the chamber, or he would be placed on oxygen while we brought the chamber to him.

This decision was based on the writings of Paul Bert[17], the current beliefs on nitrogen washout and bubble resolution, and some of our own unpublished experiments with guinea pigs. Most clinicians who used this first aid regime, in both Australia[18] and France[19], seemed to be impressed with its success. It is now internationally accepted as a first aid measure[20].

Oxygen tables in recompression chambers

In 1965 Goodman and Workman[21] produced their oxygen tables, allowing us to start treatment of almost all DCS cases at 18 m. These really only became used for seriously ill divers, from about 1967. These oxygen treatments were also inadequate in many cases, possibly because of the delays and the development of complex pathophysiological effects of decompression only now being elucidated[22].

That was when we decided to experiment. If a patient got worse during treatment, then the treatment was modified for that type of case. We capitalised on the beneficial effects of both pressure and oxygen without preconception. We took the usually severely ill diver to the shallowest depth that produced a satisfactory (but not necessarily complete) clinical response i.e., one assessed as not to lead to permanent sequelae. We then decompressed with the maximum oxygen that would not produce convulsions. Each depth range had its own acceptable O_2 %, which was achieved by mixing air with 33% O_2, 40% O_2, 60% O_2 or 100% O_2. Dramatic treatment for a serious illness.

Those were called the Australian tables[16] and I would still revert to them for serious cases (not the indefinite cases with "soft" signs that now seem to predominate). We even avoided air breaks as we saw little value in perpetuating a nitrogen problem; also it seemed as if some patients deteriorated at or soon after the air break. We later used heliox instead of air breaks, to reduce the likelihood of respiratory oxygen toxicity.

The underwater oxygen (UW O_2) tables introduced soon after this, were no more than the shallow part of these "Australian tables"- from 9 m to the surface. This shallow segment of the tables had been frequently used alone in the RCC for:

- missed decompression
- the final segment of deeper treatments
- very recent cases (e.g. those who develop DCS near the chamber),
- minor cases of DCS,
- those in which we were not convinced of the diagnosis,
- those especially susceptible to oxygen toxicity, epileptics and
- delayed minor stable DCS cases e.g. musculo-skeletal DCS, days after the dive

(these respond equally well to surface oxygen, although it takes longer).

Independently, the French developed their Comex tables[23], which were intermediate between the formal but very limited US Navy tables and the more flexible and thus complex Australian ones. The Australian 9 m (UW O_2 and RCC) table differed little in effect from the COMEX 12 m (RCC) table

Underwater oxygen treatments

This was developed in the late 1960's at the RAN, and by 1970 was employed through many parts of the Indo-Pacific[16] - where chambers were not readily available. The origin of this treatment is not in dispute, as no one else was prepared to share the flack when the knowledge of it spread to the USA in 1973. It was also reported at an international conference in France, in 1978[24].

The UW O_2 regime is still employed by many of the divers in remote areas, such as in the Pacific islands, the abalone fields of southern Australia, and the pearl fields of the Australian north. But local variations in technique have developed.

In Hawaii it is preceded by a deep air dip. I have no experience of this modification, but there is good theoretical justification for it. I will elaborate on the others.

The UW O_2 treatment is now a part of many national diving manuals. It was included in the Royal Australian Navy manual as tables 81 and 82, but took 15 years and with some modifications, before it found its way into the US Navy Diving Manual[25].

PHYSIOLOGY

The value of substituting oxygen for air in the recompression chamber treatment of DCS, is now well established. The pioneering work of Behnke, Yarborough and Shaw[27,28] over 50 tears ago eventuated in the Goodman and Workman[21] oxygen tables 30 years ago.

The advantages of oxygen over air breathing include: Increasing nitrogen elimination gradients, avoiding extra nitrogen loads, increasing oxygenation to tissues, decreasing the treatment depths and exposure time, reducing vascular/haematological damage, with overall therapeutic efficiency. The same arguments are even more applicable when one considers UW O_2 - plus the added advantage of immediate reduction of the bubble size to almost half.

Certain other advantages of UW O_2 over underwater air are evident [26]. Attendant divers are not subjected to the risk of DCS or nitrogen narcosis, and the affected diver is not going to be made worse by premature termination of the treatment, if this is required. Hypothermia is much less likely to develop, because of the greater efficiency of the wet suits at these depths.

The underwater site chosen can often be in a shallow protected area (such as a bay or off a wharf), reducing the influence of adverse weather on the patient, diving attendants and boat safety. Communications between the diver and the attendants are not difficult, and the situation is not as stressful as the deeper, longer, underwater air treatments or even as worrying as in some third world recompression facilities.

TECHNIQUES AND EQUIPMENT[26]

Technique

Whenever oxygen is given , on the surface or underwater, the cylinder should be turned on and the flow commenced, before it is applied to patients or divers to breathe.

Oxygen is supplied at maximum depth of 9 metres (30 feet), from a surface supply. Ascent is commenced after 30 minutes in mild cases, or 60 minutes in severe cases if significant improvement has occurred (this time may be extended for another 30 minutes if there has been no improvement). Subsequent ascent is at the rate of 12 minutes per metre or 4 minutes/foot.

A diver attendant should always be present, but the ascent is controlled by the surface tenders. The duration of the 3 designated tables is 2 hours 6 minutes, 2 hours 36 minutes and 3 hours 6 minutes.

After surfacing the patient should be given periods of oxygen breathing, interspersed with air breathing, usually on a one hour on/one hour off basis to prevent significant recurrences. Omission of this procedure is the commonest mistakes made.

Acquire baseline and repeated expiratory spirometry measurements and chest X-ray when possible and practical (rarely).

Equipment

No equipment should be used with oxygen if it is contaminated, dirty or oil lubricated .

The ideal equipment required for this treatment includes the following: A large oxygen cylinder (e.g. 220 cubic feet/7000 litres, G size). This is usually available from local hospitals, although industrial oxygen can be used from engineering workshops. Breathing this oxygen at a depth between 9 metres (30 feet) and the surface, for this duration, is usually insufficient to produce either neurological or respiratory oxygen toxicity.

A 2-stage regulator, set at 550 kPa (80 psi) is fitted with a safety valve, and connects with 12 metres (40 feet) of supply line (HP hose). This allows for 9 metres depth; 2 metres from the surface of the water to the cylinder, and 1 metre around the diver.

A non-return valve is attached between the supply line and the full face mask (e.g. a Cressi-Sub). The latter is inexpensive and enables the system to be used with a semi-conscious or unwell patient. It reduces the risk of aspiration of sea water, allows the patient to speak to his attendants, and also permits vomiting without obstructing the respiratory gas supply. Many compromise by using a normal face mask.

The supply line is marked in distances of 1 metre from the surface to the diver, and is tucked under the weight belt, between the diver's legs, or is attached to a harness. The diver must be weighted to prevent him drifting upwards.

Some experienced divers use an oxygen re-breathing system. Recreational divers tend to prefer oxygen from a (well marked) designated scuba, a system with which they are familiar.

OBSERVATIONS AND RESULTS

The first 25 cases of UW O_2 treatment were chosen and monitored by me at the Royal Australian Navy School of Underwater Medicine. Usually these were in remote areas, and some would probably not have received medevac treatment, because of the logistics and/or expense.

The information soon became known to the diving population, especially those remote from recompression facilities, and the technique was applied by many different groups of people, and who were less selective in their choice of cases.

Different experiences and views have evolved from the three different groups of divers.

Recreational divers

There is no way of knowing the number of cases treated on the tropical islands of the Indo-Pacific. Some areas, with which I am more personally associated, have advised me of dozens of such cases in each of the following localities; Solomon Islands, Papua New Guinea, Rabaul / Kimbi, Torres Strait Islands.

The importance of post-treatment oxygen administration was evident and the value in reducing the need for medevac was obvious in most cases.

I am aware of other areas because of the cases referred back to me, or where I have been directly involved in the treatments (Christmas Island, Lord Howe Island, the Cook Islands, Naurau, Truk Lagoon and other areas of Micronesia, the Great Barrier Reef). I am aware of only one accident during these treatments, but the aetiology is problematical[30].

A diver who had been diving to 5 metres, snagged his air line, performed an emergency ascent, during which he may or may not have sustained pulmonary barotrauma, but during which he definitely did inhale a considerable amount of sea water into his lungs.

Because of the rapid ascent and the fear of the development of DCS, he was given oxygen through a full face mask at a depth of 8 metres. Within a few minutes he began to experience shaking of his limbs and appeared to be losing consciousness. He was surfaced, and treated for his salt water aspiration - which cleared over the next 24 hours. There were no sequelae but a provisional diagnosis of oxygen toxicity was made.

The actual events were clarified only after I attempted to follow up the case, and found that he was apparently cyanotic on the surface. I find it difficult to understand how one can become toxic to oxygen, having sustained a salt water aspiration that produces an appreciable reduction in arterial oxygen levels. It is not certain whether the problems were due to oxygen excess from the treatment, or hypoxia from salt water aspiration. Masseter spasm

and shivering are both common with hypoxia and the salt water aspiration syndrome. He did not have a typical epileptic fit.

Unfortunately the clinical data in this particular case is problematical, making differential diagnosis difficult. The heading of the article was misleading, to say the least.

Some of the cases subsequently treated, without any supervision have been much more seriously ill than we would have wished. Usually the results were surprisingly valuable in these cases, with UW O_2 used while waiting for medevac.

In more recent years the concept of treatment on oxygen has expanded even further, with the development of technical diving and the facilities available to some of these divers. I would personally have reservations regarding its unqualified use in such cases. The reasons for this are as follows:

1. These divers have already had an oxygen load, increasing the likelihood of oxygen toxicity.

2. The extended duration of the dive might well exceed the safe duration of that equipment if it is being used subsequently to supply the oxygen - either as regards oxygen depletion, or exhaustion of the carbon dioxide absorbent.

3. If the diver has already developed DCS following the use of mixed gas then the diving regime is unlikely to be a conservative one.

Abalone divers

In 1985, of the 200 or so registered professional abalone divers of Australia, 152 were submitted to a diving questionnaire, personal history taking, physical examination and various investigations[14].

These divers were exposed to excessive diving durations, and 58% of them routinely employed a dive profile which required some form of decompression, according to the US Navy Tables, but which was omitted.

Although they employed repetitive diving, and some multi-level diving, this was frequently not in the manner usually recommended. On the contrary, the dives tended to be deeper as the day progressed, with deteriorating sea conditions. Also the water temperature was often cold (4-10^0C).

At that time there was considerable ignorance in the field, as regards the UW O_2 techniques being employed by the RAN School of Underwater Medicine. Indeed, the few ex-RAN Divers that were working as abalone divers at the time were usually a source of mis-information, having only been exposed to the conventional oxygen treatment tables used in a recompression chamber. A popular belief evolved that oxygen could be safely used at 18 metres, as long as it was used for treatment.

Oxygen was rarely used for decompression per se, without DCS, at that time. It had a poor reputation and the majority of the divers neither employed oxygen as a treatment nor had it available on the boat. However,

8.6% had used oxygen for treatment on the surface,
7.9% also used it at a depth of 9 metres or less underwater,
5.3% also used it at a depth greater than 9 metres underwater.
No diver used it in excess of 18 metres.

Of the 625 cases of DCS that could be remembered by the 152 divers,

11% were treated in a recompression chamber. Over half were neurological DCS.
15% were treated on the surface, with O_2.
66% were treated underwater, on air and/or oxygen.
22% were not treated at all.

These figures are probably not precise, because of the inevitable vagaries of memory and denial.

The DCS incidence is especially misleading, as many of the divers would complain of joint and other symptoms post diving that they would not attribute to DCS. As a general rule, they would ignore minor symptoms, without considering them to be DCS, or they classified them as "niggles" - thus not requiring any treatment.

Problems with oxygen toxicity were documented. All of these cases were using oxygen at greater than 15 metres, a much greater depth than recommended.

An informal survey was undertaken in 1995 by letter (it is not easy to obtain replies from this occupational group) to ascertain the current status of UW O_2. After the 1985 Abalone Diver Survey, in which the UW O_2 regime was described, it would have been rewarding to report a safer oxygen use. Unfortunately this is not so. It has now superseded underwater air treatments, and is used frequently.

Most of the deeper divers (18m+) now routinely carry and use oxygen for treatment, and frequently for decompression. They employ a large variety of protocols. Some use the UW O_2 for treatment, as proscribed. Others return to the depth of the dive (as deep as 30 m). Others routinely decompress on oxygen from variable depths, to avoid DCS. Re-education appears warranted, as demonstrated by the problems reported.

There were a few cases of problems using oxygen in excess of the 9 metre depth, and often using it while continuing to harvest abalone, thereby employing their oxygen decompression time in a more lucrative manner. The cases were as follows:

1. Breathing oxygen at 12 metres caused his lips to "go funny" and he noted a tingling and numbness over the whole of his body;

2. Used oxygen mainly because of his navy training and his experience with this. The maximum depth and duration would be 1 hour at 15 metres. He would continue collecting abalone during that time and sometimes noted his right arm twitching and jerking, a loss of sight, appearance of star light objects underwater, twitching of the mouth and body. He claimed never to have lost consciousness underwater, however other abalone divers state that this is not so and that he had been rescued at least once by his boatman.

3. Lost consciousness after a few minutes (it must have been more than this as he had half filled his abalone bag) at about 18 metres.

4. After breathing oxygen for more than 10 minutes at 18 metres, his eyes went swimming and fuzzy and he started to twitch. These symptoms indicated to him that it was time to quit.

5. He dives to 30 metre regularly, and uses oxygen for both decompression and treatments from that depth. He frequently notices visual symptoms, such as "mini stick figures running around the edges of my vision". He will not alter this regime as he "feels better with it".

It can be seen by the above case reports that basic training in the use of oxygen underwater and its dangers, is required Since the 1985 Australian Abalone Diver Survey, most of the abalone divers have been using the underwater oxygen, but not always as proposed in reference[16]. The authorities have not accepted our offers to intervene with safety lectures to these divers.

We are unlikely to learn much of value from these divers, because of their radical use of underwater oxygen. We are more likely to encounter the hazards of its improper use.

Pearl divers

After 1989, when the deaths for pearl diving had been reduced to 0-1 per year, attention became focused on the next major problem, DCS. Information about the value of oxygen at 9 metres spread by word of mouth, from visiting lecturers.

It started to be used both for decompression and recompression treatment.

Wong[11], Nishi[10] and Edmonds[12] have described the diving schedules and the results, during the last decade. Of reference to this report is the divers experience with DCS and its underwater treatment[29].

During the 4 years, 1988-91, extending over 4 pearl diving seasons, a survey of 10% of subjects covered a total of 1,834 days dived by these open ocean shell divers. It comprised 11,776 dives, averaging 6.4 dives per day. The divers were exposed to depths between 10 and 54 metres.(Table 1).

Table 1 - Pearl Diver DCS Statistics

DEPTH m	DIVER DAYS	AVG DIVES per DAY (no.)	TUT avg	02 avg	DCS no.	DCS % diver days
45-54	140	4.4	152	96	19	13.6%
35-44	406	4.4	210	80	27	6.7%
25-34	322	4.7	285	73	7	2.2%
15-24	511	8.0	406	9	2	0.4%
10-14	455	8.3	444	-	?1	0.2%

DCS was the commonest medical disorder recorded (45%). The existence of a DCS diagnosis in the diving logs was verified by the recorded extra decompression time employed. This involved an administration or extension of O_2 at 9m for 30-45 min. The incidence of DCS from a diving day increases progressively from: 0.2% at 10-14m depths to 13.6% at 45-54m depths.

Of the 1,834 diver days worked (11,776 dives), there were 56 cases of DCS and 55 were treated successfully on the UW O_2 regime.

By extrapolation to the remainder of the Broome and Darwin fleets, we can presume a DCS case load of about 500 treated underwater on oxygen over those 4 seasons of diving. Only one required medevac.

Provisos must be noted.

1. All cases occurred at sea, and treatments were usually given within 30 minutes. Occasionally the diver would return to the depth of the dive to complete another "drift" before being treated with oxygen.
2. Except for the diver who required medevac, most divers continued diving on that or the next day without any more problems.
3. We have no idea of how this treatment influences the propensity to dysbaric osteonecrosis.

Like the abalone divers, their pearl divers have modified the treatment regime, but not in the same manner. Their consistent routine is to employ oxygen for 30 minutes at 9 meters, extendible if any symptoms persist, and then ascend at a relatively fast rate of 3 metres per minute.

As regards oxygen toxicity, the 1988-91 pearl divers survey[29] disclosed a great deal of oxygen exposure - for both decompression and recompression therapy. See Table 2. Based on this 10% sample, there was a total of 10,064 days diving with oxygen. It average 70 mins use per day (range = 10-150 minutes), spread over 1-5 dives with increasing durations - depending on the dive profiles.

There were no oxygen convulsions or toxicity's noted during this period. Nor have there been any since (personal communication, Dr Robert Wong, 1998).

Table 2 - Oxygen Decompression at 9 metres

DURATION O2	DIVER	MAX DEPTH	UW	DIVES
mins/day(avg)	days	metres	hours	no.
120 -150 (138)	147	43.3	3.24	4.95
90 -149 (95)	147	37.3	4.26	4.81
60 -89 (75)	420	34.2	4.65	4.83
30 -59 (37)	147	29.5	5.05	5.67
10 -29 (16)	203	22.2	6.48	8.0

This represented approximately 10% of the pearl divers 1988-91.

DISCUSSION

The physiological principles on which UW O_2 is based are well known and not contentious, although the indications for treatment may be.

It was originally hoped that the UW O_2 treatment would be sufficient for the management of minor cases of DCS and so avoid medevac requirements, and to prevent deterioration of the more severe cases while suitable transport was being arranged. When the regime was applied early, even in the serious cases, the transport was rarely required.

It is a common observation in recreational divers treatment that improvement continues throughout the ascent, at 12 minutes per metre. Presumably the resolution of the bubble is more rapid at this ascent rate than its expansion due to Boyle's Law.

The pearl divers, probably because of their speedy return to pressure and rapid treatment on oxygen, are able to reduce the duration on oxygen and cope with a faster ascent rate.

Critics of the underwater treatment technique often complain that its success is based on anecdotal cases. The vast numbers of divers employing it effectively make it more than anecdotal - and the numbers now exposed safely to shallow (9m or less) oxygen are extreme in both pearl divers and hyperbaric patients. The critics then often imply great danger citing one or two patients with symptoms that may be related to oxygen or may be related to the original dive i.e. genuine anecdotes!

Reports by Pyle and Youngblood[31] from the (predominantly) Hawaiian divers using in-water recompression highlighted both the disadvantages and value of this procedure. Of the 527 cases, 87.7 % got complete resolution, 9.7 % had mild residua and 2.7 % required additional recompression chamber therapy.

The UW O_2 recompression treatment is not applicable to all cases, especially when the patient is unable or unwilling to return to the underwater environment. It is presumably of less value in the cases where gross decompression staging has been omitted, or where a coagulopathy has developed. I would be reluctant to administer this regime where the patient has epileptic convulsions or is unconscious. Others are less conservative.

One of the common reservations in Australia[26] was that this underwater treatment regime is applicable to the semi-tropical and tropical areas (where it was first used), but not to the southern parts of the continent, where water temperatures may be as low as 4°C.

There are certain inconsistencies with this limitation. Firstly, if the diver developed DCS while diving in these waters, then he is most likely to already have effective thermal protection available to him. Also, the duration for the UW O_2 treatment is not excessive, and is at a depth in which his wet suit is far more effective than at his original diving depth. If he is wearing a dry suit the argument is even less applicable. The most effective argument is that it is used, and often very successfully, in the cold southern waters of Australia.

Some claim that the UW O_2 treatment is of more value when there are no transport facilities available. Initially this was also our own teaching, but with the logic that comes with hindsight, only a 3 hour gap is needed between the instituting of UW O_2 therapy and the arrival of transport, to enable us to use this system. It is probably more important to treat the serious cases early, even if full recovery is not achieved, than to allow the progression of pathology during those hours.

There is no doubt, especially in serious and delayed cases, that transport should be sought while the underwater treatment is being utilised.

There has been a concern that if this technique is available for treatment of DCS , other divers may misuse it to decompress on oxygen underwater, and perhaps run into subsequent problems. This has happened, but is more an argument in favour of educating divers, than depriving them of potentially valuable treatment . One could use this illogical argument to prohibit all safety equipment, including recompression chambers, and thereby hope to circumvent diving related problems.

It has been claimed that UW O_2 treatment is unlikely to be of any value for those patients suffering from pulmonary barotrauma. It may well be so in some cases. The treatment was not proposed for this. It is, however, possible that the treatment may be of value for mediastinal emphysema, and perhaps even a small pneumothorax.

When hyperbaric chambers are used in remote localities, often with inadequate equipment and insufficiently trained personnel, there is an appreciable danger from both fire and explosion. There is the added difficulty in dealing with inexperienced medical personnel not ensuring an adequate face seal for the mask. This problem is not encountered in underwater treatment. Medevac aggravates these difficulties and also introduces appreciable hazards of its own.

The customary supportive and pharmacological adjuncts to the treatment of recompression sickness are in no way superseded, and the superiority of experienced personnel with comprehensive hyperbaric facilities is not being challenged. The UW O_2 regime, as described, is considered as a first aid regime, not superior to portable recompression chambers, but sometimes surprisingly effective and rarely, if ever, detrimental.

Whether we approve of the concept or not, it will continue to be used for as long as it is needed. The various diving communities are widening the UW O_2 protocol, and this may reflect the different types of cases encountered and the speed of its application.

The relative value of current first aid regimes (the various UW O_2 procedures, including an additional deep air dip, and surface oxygen administration) needs to be clarified.

The most effective way that I can envisage us contributing to diving medical first aid for DCS in remote areas, is by demonstrating a safer but equally effective UW or surface treatment e.g., with a helium/oxygen or nitrox mixture that can be stored and used in emergencies, as oxygen is now.

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"TREATMENT OF DECOMPRESSION SICKNESS,

HAWAIIAN STYLE

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Edwin Hayashi
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A retrospective survey by personal interview of 44 of Hawaii's diving fishermen was carried out by the authors in 1981-82. Many of this group (22/44 = 50%) reported that they had used immediate in water recompression (IIWR) as a method of treatment for decompression sickness which it occurred while diving in the open ocean several hours away from the nearest decompression chamber.

These 22 divers who had used IIWR were subsequently re-interviewed in order to learn more about the technique used and the effectiveness of the procedure in ameliorating or curing decompression sickness (DCS). The data from this survey has been analyzed and the effectiveness of the IIWR treatment for decompression sickness has been evaluated.

The need for immediate recompression in the treatment of DCS has been emphasized for many years (U.S. Navy, 1963). However, when the U.S.N. recompression chambers at Pearl Harbor were made available for DCS treatment of Hawaii's diving fishermen (HDF) the emphasis for immediate recompression was skewed to immediate recompression in the recompression chamber. Hawaii's diving fishermen were thereafter admonished to come immediately to the recompression chamber at Pearl Harbor for treatment. However, the HDF netted and speared fish from small boats which they operated miles and hours away from the treatment chamber. They had learned that delay in treatment was detrimental to their recovery. They had learned, by trial and error, to treat DCS by immediate in water recompression (IIWR) using scuba. the effectiveness of this procedure was evaluated in this survey, and the parameters of IIWR treatments and their effectiveness were determined.

In order to understand the development of IIWR as a treatment for DCS, it is necessary to first understand the development of scuba diving in Hawaii.

The Cousteau-Gagnan self-contained underwater breathing apparatus was offered for sale in Honolulu in 1949. Skin divers who fished for monetary reward immediately recognized the commercial value of this device, which consisted of a gas bottle regulator and straps.

The waters around the Hawaiian Islands were clear and abounded with fish. Spearing or netting fish or collecting semi-precious coral by using scuba became a lucrative occupation. Upwards of 300 islanders used scuba diving either as a principal source of income or to augment other income. Of this group, more than 100 were still actively diving at the time of the survey of whom 44 were interviewed.

The ages of these divers varied from 61 to 31 years with a mean age of 42.5 years. The diving techniques developed by these divers are unique. They used small boats and carried many air tanks for diving and usually extra tanks for use in the case that one of the divers developed DCS. The survey revealed that the maximum number of dives made by any diver in one day was 12, with a mean value of 5.5 dives per day for all divers interviewed.
This explains why the most experienced diver had amassed a total of over 23,400 dives up to the time of the survey. The mean number of dives made to the date of the survey was 11,000 per diver. The deepest air dive reported was 350FSW on scuba. The mean maximum dive depth was 228 FSW. One group of black coral divers had worked a coral forest at over 300 FSW for over a month with 4-5 dives per week.

These HDF had learned by experience to make their deepest dive the first dive of the day, followed by less deep dives and then finishing the day by making a so-called "scrape" dive to catch lobsters, octopus or reef fish at dive depths of 60 ft. or less.

Since this is a retrospective survey of personal experiences, it is apparent that unless the divers maintained a log which chronicled all of the incidents of DCS which they had experienced over the preceding 15-20 years, then these data would not necessarily be reliable.. The divers did of course remember some specific incidents of DCS which they narrated. A few did keep logs, and these data were therefore available. It should also be remembered that the survey was of members of a small group of people, and they worked as teams. Therefore, each team member became a check against the others in augmenting, verifying or denying the memory of another. A further limitation in evaluation of IIWR results ensues from the lack of medicinal evaluation in most cases combined with the divers well known tendency for denial.

In addition, the records of treatment of DCS at the Pearl Harbor Treatment Centre were also available. Therefore by using these checks and balances we believe that the inferences derived from these data are essentially valid.

The divers interviewed reported the use of IIWR in the treatment of over 500 diving incidents of premonitory signs or frank decompression sickness. The treatment was successful except in 65 incidents. In 51 of these, divers reported significant improvement of bone pain but only to the point that they chose to "wait it out" or "bite the bullet" and used beer or aspirin as home remedies for 1-3 days until the ache subsided.

In addition there were 14 incidents of the total in which IIWR provided such inadequate recovery that further treatment was sought at the USN Recompression Treatment Chamber at Pearl Harbor. Of these, three (3) patients sought further relief from bone pain, ten (10) for spinal cord disease and one (1) for vestibular incordination. Of these, nine (9) still had significant residuals following discharge after treatment by USN procedures. The magnitude of these residuals varied from persisting complete paraplegia to continuing vestibular incoordination.

The signs and symptoms which were relieved varied from the mild "Bends Type" DCS (primarily paid and aches around the shoulders and arms) to the more serious CNS conditions that included loss of vision, vestibular dizziness, loss of sensation, paraplegia, quadriplegia and clouding of consciousness. However, it should be noted that this type of DCS treatment apparently does not protect divers against the chronic form of decompression sickness of the bone, i.e. dysbaric osteonecrosis, a disease which many HDF have developed (Wade et al., 1978).

The water depths that were used for IIWR ranged from the deepest estimated depth of 85 FSW to the shallowest estimated depth of 25 FSW, with an average treatment depth of 41.3 FSW. IIWR times showed a high of 200 minutes and a low of 20 minutes, with an average decompression time of 63.7 minutes. Recompression depths of 30 FSW or less or for durations of 30 minutes or less usually did not prove effective in treating DCS.

CASE HISTORIES.

One of the authors, (FF) has personally treated others several times and, likewise, has been treated himself by IIWR on two occasions. His personal treatments were for pain in the shoulder and arms. One one occasion after the onset of symptoms, he was rapidly taken to shallower water and two, one tank, "scrape" dives were made spearing fish in 55 to 45 FSW. Most of the pain disappeared immediately upon reaching depth, and relief continued while diving. He was very comfortable after the treatment dives.

In another incident (Figure 1), he initiated the IIWR of another diver who had made three dives ranging from 120 to 160 FSW. A few minutes after the third dive, the diver developed uncontrollable movements of both legs. The boat was already underway so FF piloted it toward shallower water. Within this few minutes the diver's lower body became paralyzed and he had no feeling from the nipple line down. He could not stand or move his lower extremities. A full tank of air was strapped to the victim who was still able to hold and breathe through the mouthpiece of the regulator. He was then lifted over the side of the boat and rolled into the water. FF was waiting in the water.

After checking the victim's breathing, he commenced pulling the disabled diver toward the bottom. No immediate benefit occurred at 40 FSW so FF towed the victim toward deeper water. In approximately 70 FSW, the victim started tugging and made noises and gave an "OK" hand signal. He further demonstrated that he had regained movement of his lower body.

The victim was instructed with hand signals to remain at the bottom holding onto or swimming around a large boulder. The boat was anchored directly above and a safety diver hung from a rope attached to the boat and watched from the surface while the victim recompressed. When the recompressing diver indicated low air pressure in his tank by engaging his reserve valve, the observing diver went to the bottom and exchanged tanks, thereby letting the victim have another full tank. The victim later ascended to 40 FSW and then to 20 FSW, where he stayed until the air supply was almost gone, and then surfaced. He felt a little tired that evening, but was observed to be walking normally and had had good return of strength in his legs and arms, as well as normal sensations throughout his body.

Another incident, which was reported by one of the divers interviewed, may explain why IIWR for the treatment of decompression sickness has been adopted by so many HDF. This incident was subsequently verified by other divers involved and by the County Coroner's Office. On this day of fishing, four divers were working in pairs at a site in about 165 to 180 FSW. Each pair alternated diving and made two dives each. Upon surfacing from the second dive, both divers of the second pair rapidly developed signs and symptoms of severe CNS decompression sickness. The driver of the boat and other diver decided to take both victims to the U.S. Navy recompression chamber, so they headed for the dock some 30 minutes away. However, one diver refused to go and elected to undergo IIWR. He took two full scuba tanks and told the boat driver to come back and pick him up after they got the other diver to the chamber. He was then rolled over the side of the boat.

The boat crew returned after two hours to pick him up, they found him swimming on the surface. He was asymptomatic and apparently cured of the disease. The other diver died of severe decompression sickness in the Med-Evac helicopter on the way to the recompression chamber.

SUMMARY.

It should be emphasized that this survey reports on a treatment for decompression sickness which has been empirically developed over many years of use by a specific population at risk. This population is small, and the procedure is directed toward the treatment of a disease process which results from use of diving techniques used by HDF. These HDF employ many repetitive scuba dives with relatively short surface intervals. This IIWR technique for treatment of DCS has found to be effective in treatment of DCS as it afflicts HDF. It is proposed neither for universal use nor as a complete treatment.

In recent years we have encouraged HDF to use oxygen in addition to air in carrying out IIWR. We recommend that they carry a tank of oxygen (of 120 cu ft or more capacity) in their boat for use in treating decompression sickness in water. They have been instructed in the use of the Australian emergency underwater oxygen treatment (Edmonds et al., 1976) and the Hawaiian emergency in-water, air-oxygen recompression treatment (Beckman, 1981: Figure 2). They have been encouraged to carry the necessary equipment (tank of oxygen and regulator with 30 ft tether) with them on their boat and to initiate treatment by either method immediately if any crew member develops signs or symptoms which could be related to decompression sickness. They have been further advised to seek medical consultation at the Hyperbaric Treatment Centre immediately after receiving this treatment. The results from use of the air/oxygen recompression treatment table have been excellent for those who have used it. Unfortunately, the problems of procuring oxygen for use on small boats still limits its usefulness for HDF.

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HDF REPETITIVE DIVES WHICH RESULTED IN CNS/DCS WITH SUCCESSFUL TREATMENT BY IMMEDIATE IN-WATER RECOMPRESSION

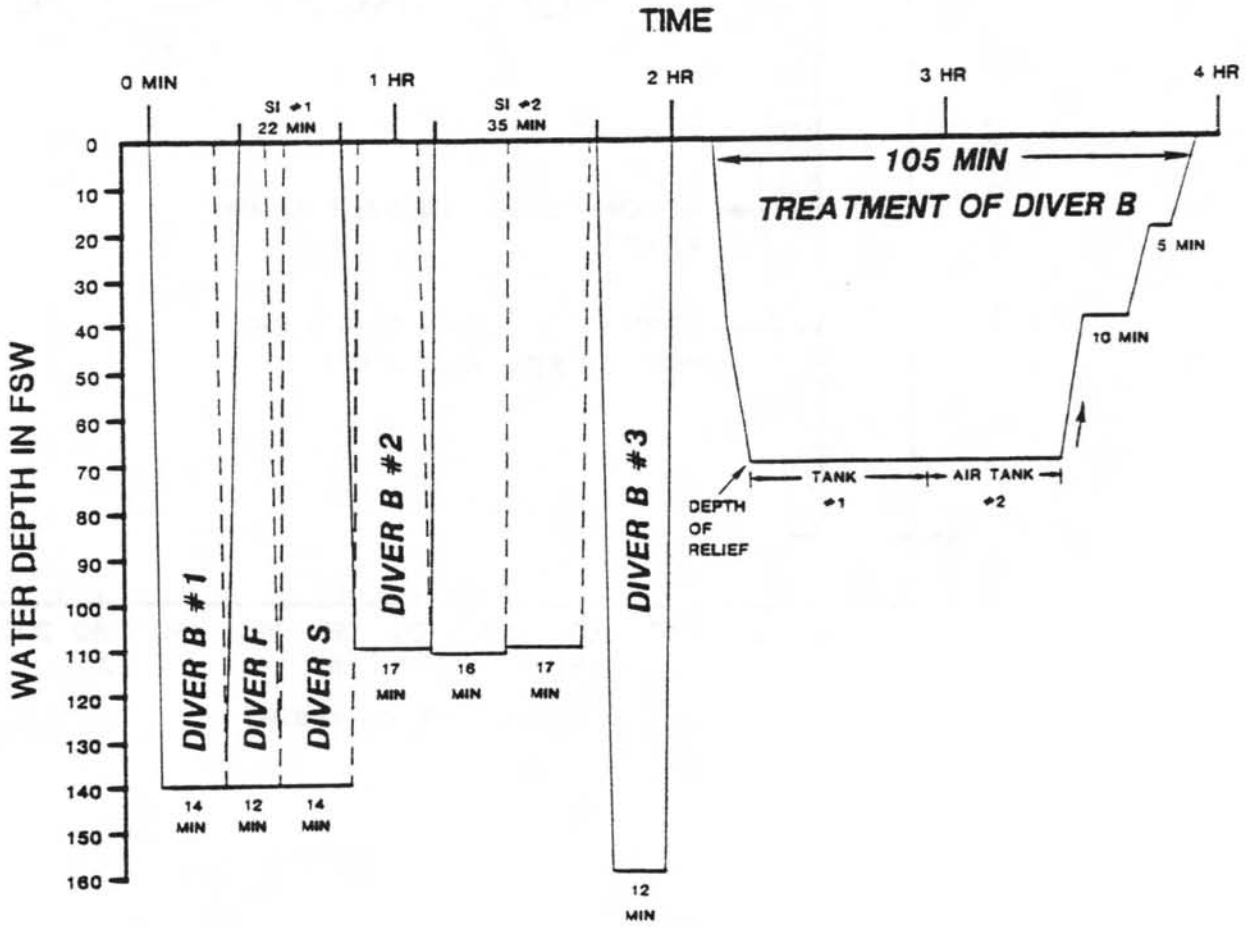
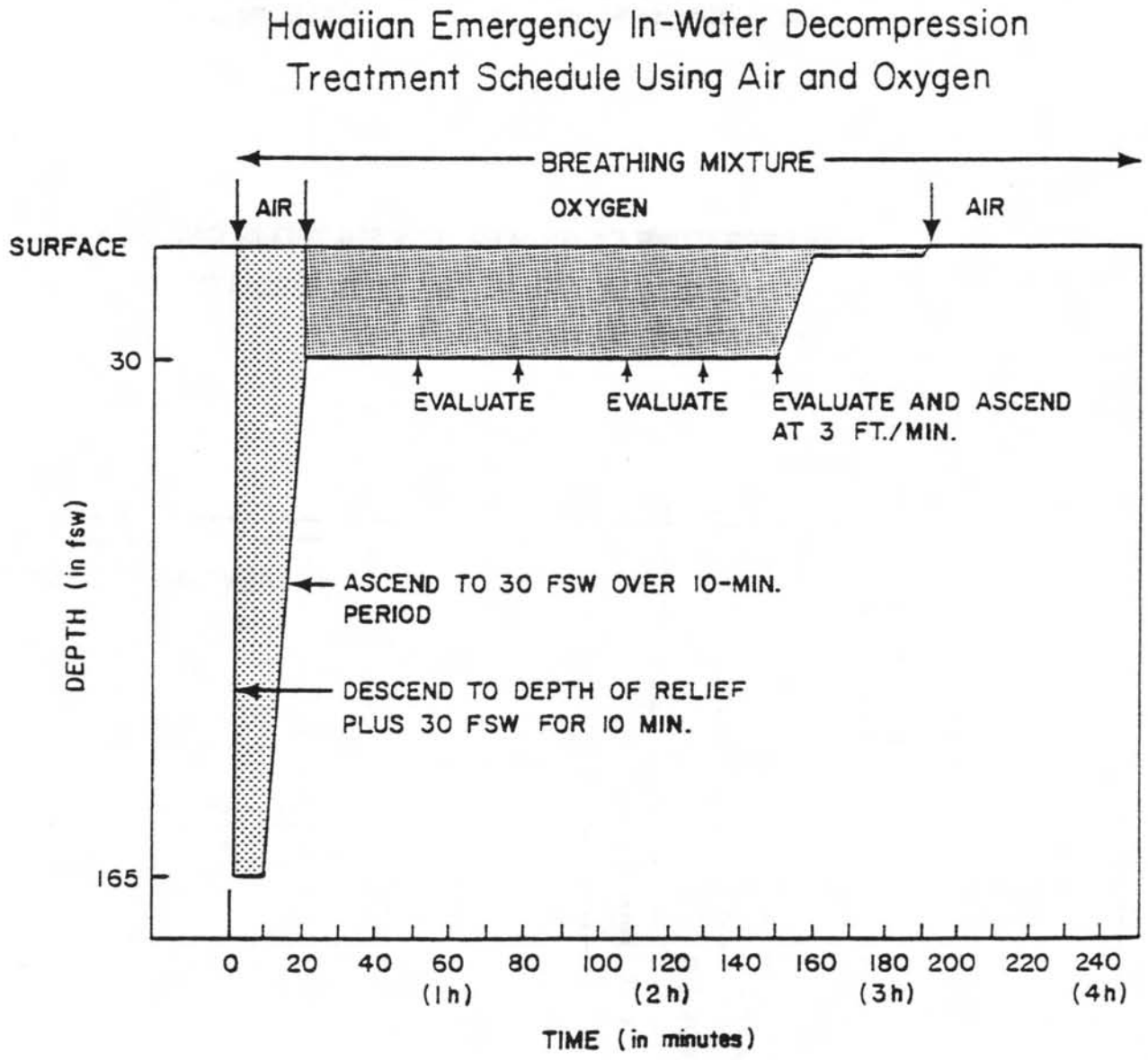


FIGURE 2. HAWAIIAN EMERGENCY IN-WATER RECOMPRESSION SCHEDULE FOR TREATMENT OF DCS



IMMEDIATE IN-WATER RECOMPRESSION

CONCLUSION:

IIWR USING COMPRESSED AIR SCUBA HAS BEEN FOUND TO BE EFFECTIVE AMONG H.D.F. IN THE TREATMENT OF ALL FORMS OF DCS IN ITS BEGINNING STAGES.

RECOMMENDATIONS:

1. IIWR USING THE HAWAIIAN AIR/OXYGEN TABLE IS RECOMMENDED FOR USE BY HDF IN THE TREATMENT OF DCS IN ITS PRODROMAL OR EARLIEST STAGES.

2. IIWR USING COMPRESSED AIR SCUBA IS ACCEPTABLE FOR EMERGENCY USE BY HDF IN TREATMENT OF DCS IN ITS PRODROMAL OR EARLIEST STAGES.

3. IIWR MAY BE USEFUL FOR EMERGENCY TREATMENT OF OTHER DIVERS WHO DEVELOP DCS WHEN A RECOMPRESSION CHAMBER IS NOT IMMEDIATELY AVAILABLE.

IN-WATER RECOMPRESSION: USN AND NOAA METHODS

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Abstract: *The use of in-water recompression is a useful option should an appropriate diving injury occur in an area where a recompression chamber is unavailable. The technique for safely managing in-water recompression is dependent on proper prior planning. This includes comprehensive training of the each member of the dive team and having the proper equipment operationally ready. Both the US Navy and NOAA have in-water recompression procedures that have been established for such an eventuality. This article describes those procedures.*

Background

The need for recompression for the primary treatment of decompression sickness has been well established. Decompression procedures to prevent the condition were the result of the work conducted during the early pioneering days of caisson work that witnessed incredible compressed gas exposures under extreme working conditions. Severe disease and even death was the regular cost of such work. Mortality and morbidity for professional diving has been nearly eliminated with implementation of decompression procedures to prevent decompression sickness and effective recompression therapy to treat it.

The establishment of recompression procedures for treating decompression sickness evolved with time. The importance of returning a stricken patient quickly to an elevated ambient pressure was recognized early as an important treatment goal. Various air tables were generated that required treatment pressurization that have included pressurization to 300 feet of seawater (FSW). Treatment failures however still occurred much too frequently despite air recompression to these various depths and times. The addition of oxygen as a therapeutic gas in the treatment of decompression sickness greatly enhanced the efficacy of recompression therapy. It did so at lower pressures and required less time for treatment. But despite the tremendous gains in treatment outcomes, there are still a minority of patients who are quickly recompressed and still suffer a poor outcome. This has even been demonstrated in controlled animal studies looking at severe exposures.

The use of in-water recompression therapy followed this same course. Divers experiencing problems were identified and lowered back into the water breathing compressed air only. Later procedures employed oxygen and are the preferred method when available. Various in-water procedures developed among the different diving organizations perhaps for various reasons, but all constrained by the same unforgiving realities of physiology and practical considerations of resources available. In-water

recompression should be considered an alternative for remote operations if deployment of a recompression chamber is not possible. It is a compromise due to operational constraints with risks that must be understood and accepted.

Any In-water procedure such as decompression, omitted decompression, and recompression require careful planning and supervision. In-water recompression (IWR) requires a significant commitment of time to complete the required decompression. The IWR tables demonstrate the expected requirement for longer periods of time than required during standard decompression. Likewise, the in-water omitted decompression procedures require in-water obligations less than the IWR recompression but more than the standard decompression procedures. This development of the various in-water procedures underscores the importance in preventing the rapid, uncontrolled evolution of the gas phase in body tissues by adequate decompression in an efficient controlled or staged ascent rather than the longer, protracted treatment method of recompression once gas formation has occurred. The ability to administer pressure early during standard in-water decompression, surface decompression, omitted decompression procedures, and even rapid recompression when symptomatic allows one to often effectively treat the mechanical aspects of bubble formation. These effective procedures can result in the elimination of the bubble in the tissues before they cause significant physiologic/humoral alterations that lead to a more refractory condition. Proficiency in these procedures is fundamental to expert dive planning. The old, overused adage of the ounce of prevention is worth a pound of cure can be rewritten with the words decompression and recompression substituted for prevention and cure respectively. Despite careful use of the tables during both the planning and operational stages of dives we conducted in the Persian Gulf 1980, we still included plans for air in-water recompression as outlined in the US Navy Dive Manual. The procedure required the following the USN air table 1A for in-water recompression. Contrast the time requirements for USN air table 1A with the lesser obligation for omitted decompression procedures:

-Repeat any stops deeper than 40 feet
-At 40 feet, remain for one-fourth of the 10 foot stop time
-At 30 feet, remain for one-third of the 10 foot stop time
-At 20 feet, remain for one-half of the 10 foot stop time
-At 10 feet, remain for 1 ½ times the scheduled 10 foot stop time

U.S. Navy In-water Recompression Procedures

The US Navy Dive Manual states "recompression in the water should be considered an option of last resort, to be used only when no recompression facility is on site and there is no prospect of reaching a recompression facility within 12 hours." It continues to recommend that it not be used for serious symptoms for fear of increased harm to the diver by subjecting him to the procedure.

As discussed earlier, one needs to look at the relationships of recompression to decompression. This point is particularly important when the diving supervisor is confronted with the asymptomatic diver who has omitted decompression. Interruption of

a diver's decompression can occur as a result of blow-up (unexpected ascent from greater than 20 FSW), equipment failure, out of air, or acute injury. In this case, the need is recognized that prompt return to depth is necessary with longer stop times than required by the offending dive to prevent the onset of decompression sickness. This can be conducted either in-water or in the recompression chamber. Figure 1 outlines the procedures for both in-water and chamber recompression for asymptomatic omitted decompression. Again, the table demonstrates the graded recompression response based on the severity of the decompression stress (depth and time of decompression stops) and the importance of the elapsed surface interval. This relationship is actually exploited in the surface decompression procedures that allow the diver to be pulled from the water without accomplishment of all his decompression and completing it in the recompression chamber. This gives the dive operation the flexibility to put a second team to work, avoid approaching storms or simply head back to port with decompression taking place onboard. It also eliminates the environmental factors that impact the diver during in water decompression. Addressing missed decompression is a sound, conservative practice and is performed for the protection of the diver to prevent decompression sickness. It is interesting to consider the impact of projecting this strict application of omitted decompression procedures to the sport diving community, which would result in an overwhelming surge in the use of recompression chambers worldwide if one simply waited at the marina and determined individual dive profiles.

The use of in-water recompression for decompression sickness has several methods depending on the actual circumstances. For symptoms that occur during the diver's decompression stops, an algorithm is available for an in-water option (figure 2). Initially, the diver descends 10 FSW and may continue deeper for an additional 10 FSW if needed. He then completes the remaining decompression stops for 1.5 times the required time. He will then complete recompression in the chamber upon completion of these stops.

Divers that are demonstrating signs of decompression sickness should be evacuated supine to the nearest available recompression chamber on 100% oxygen delivered by the highest partial pressure available given the delivery system (head tent, helmet>demand mask>nonrebreather mask>mask>nasal cannula) and with consideration to the volume of oxygen on hand. Hydrate the patient. Evaluation of the ABC's is continuously performed with additional attention to neurologic exam. Bladder catheterization may be required to avoid further injury. Use of an uncertified chamber may be considered to avoid use of in-water recompression procedures. A recommendation not to treat severe illness such as unconsciousness, paralysis, vertigo, respiratory distress, and shock is made.

If the decision to use in-water recompression is made, then the option of air versus oxygen needs to be made. In addition, the following equipment and precautions should be utilized:

1. Use a surface supplied UBA or a full-face mask to enhance communications and provide a non-aqueous breathing environment should the diver become unconscious or convulses. Maintaining a bite on a mouthpiece becomes difficult with time and/or in cold water.

Management of Asymptomatic Omitted Decompression.

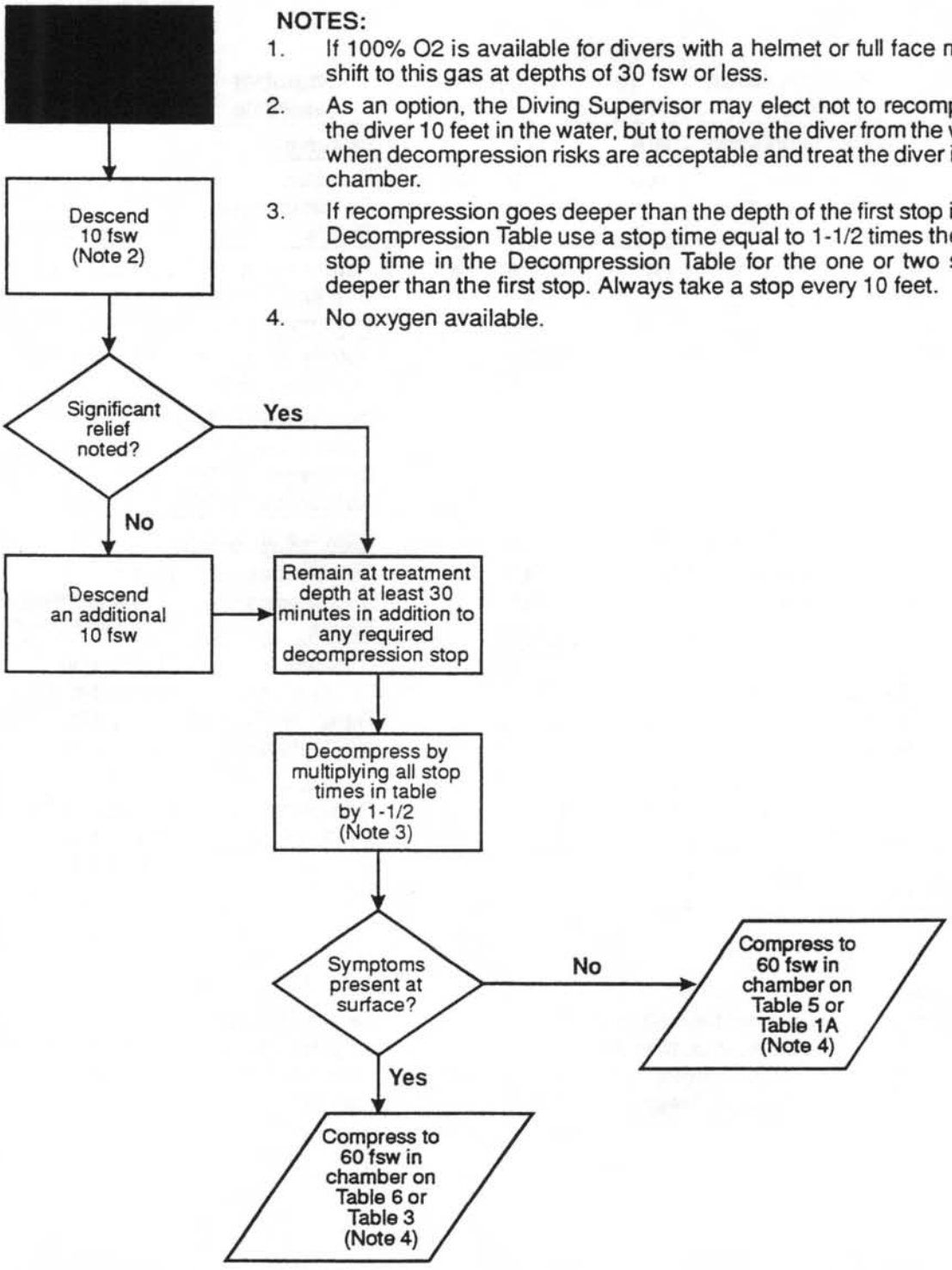
Depth at Which Ommission Began	Decompression Status	Eligible for Sur-D?	Surface Interval	Action	
				Chamber Available	No Chamber Available
20 fsw or shallower	No-Decompression	N/A	N/A	Observe on surface for 1 hr.	
	Decompression Stops Required	Yes	Less than 5 min.	Use Surface Decompression Tables	Perform Chamber stops in water (Note 1)
		No	Less than 1 min.	Return to depth of stop. Increase stop time 1 min. Resume decompression.	
		No	Greater than 1 min.	Return to depth of stop. Multiply 20 and 10-foot stop times by 1.5. Or: Treatment Table 5 (1A) for surface interval less than 5 min. Or: Treatment Table 6 (2A) for surface interval greater than 5 min.	
Deeper than 20 fsw	No-Decompression	N/A	N/A	Observe on surface for 1 hr.	
	Decompression Stops Required	Yes	Less than 5 min.	Use Surface Decompression Tables	Perform chamber stops in water (Note 1)
	Decompression Stops Required (Less than 30 min. missed)	No No	Less than 5 min. Greater than 5 min.	Treatment Table 5 (1A) (Note 2) Treatment Table 6 (2A) (Note 2)	Descend to depth of first stop. Follow the schedule to 30-fsw.
	Decompression Stops Required (Greater than 30 min.)	No	Any	Treatment Table 6 (2A) (Note 2)	Multiply 30, 20 and 10 fsw stops by 1.5.

Notes:

1. Sur-D Air only.
2. If a diver missed a stop deeper than 60 feet and oxygen is available, first compress to the depth of the first missed stop. Double this stop, then decompress to 60 feet using the appropriate decompression schedule doubling all stop times. Decompress from 60 feet on Treatment Table 5 or 6 as appropriate. If oxygen is unavailable, treat on a full Treatment Table 1A or 2A as appropriate.

figure 1

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



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

figure 2

2. Utilize a tender throughout the treatment
3. Stage or line marked every 5 feet with adequate weighting
4. Adequate air/oxygen supply, appendix A is an example
5. Thermal protection
6. Sufficient hose/umbilical length
7. Optimal location-sea state, storm, depth, water temp

The procedures for air utilizes USN treatment table 1A (figure 3). It requires 380 minutes in the water to a depth of 100 FSW initially. If the water is shallower than 100 FSW then remaining for thirty minutes and completing the table from that point using the maximum depth.

Hyperbaric oxygen is the preferred method for in-water recompression as it is for chamber treatment because of its efficacy. The use of a closed circuit oxygen rebreather is ideal for the challenge of delivering oxygen over two to three hours underwater. It is important that the diver has had prior training in its use since utilization of this UBA for recompression therapy does not eliminate the inherent dangers that it poses. The procedures (figure 4) are as follows:

1. After purging the UBA, the diver descends to 30 feet and remains there for one hour for type I and 1 ½ hours for type II decompression sickness.
2. The diver ascends to the surface with one-hour stops at both 20 and 10 FSW.
3. He breathes 100% oxygen for three hours on the surface.

NOAA In-water Procedures

NOAA utilizes either the US Navy or Australian in-water recompression procedures. The decision algorithm is outlined in figure 5.

Both tables bear similarities particularly in regard to treatment depth and time. The major differences lie in the slower Australian continuous ascent rate of 4 min/ft that does not have the same discreet stops. In addition, the Australian table does not have the caveats warning against its use in severe cases or for evacuations requiring 3 hours waiting.

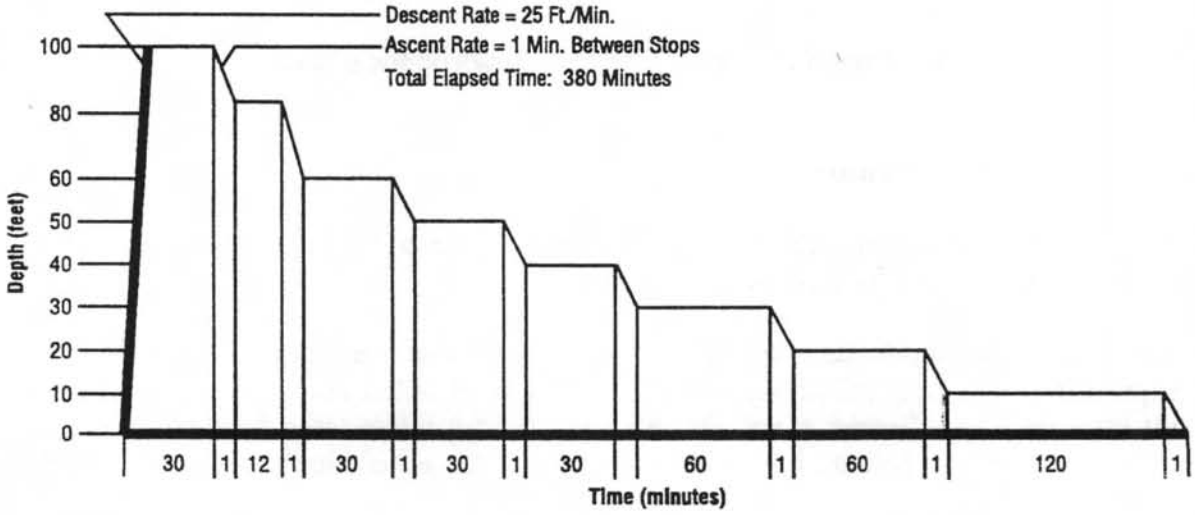
Future Prospects

The US Navy is currently testing man carried portable chambers. It has currently limited the field to one chamber that has a 60 FSW limit similar to monoplace chamber pressure restriction. There are other portable chambers that operate at maximum depths that range from 30 to165FSW. Their utility is that they provide rapid recompression since they are taken on station and therefore shares the same major advantage in-water recompression does but without many of the risks involved in returning to the water.

Air Treatment Table 1A

1. Descent rate - 25 ft/min.
2. Ascent rate - 1 minute between stops.
3. Time at 100 feet - includes time from the surface.
4. If the piping configuration of the chamber does not allow it to return to atmospheric pressure from the 10-foot stop in the one minute specified, disregard the additional time required.

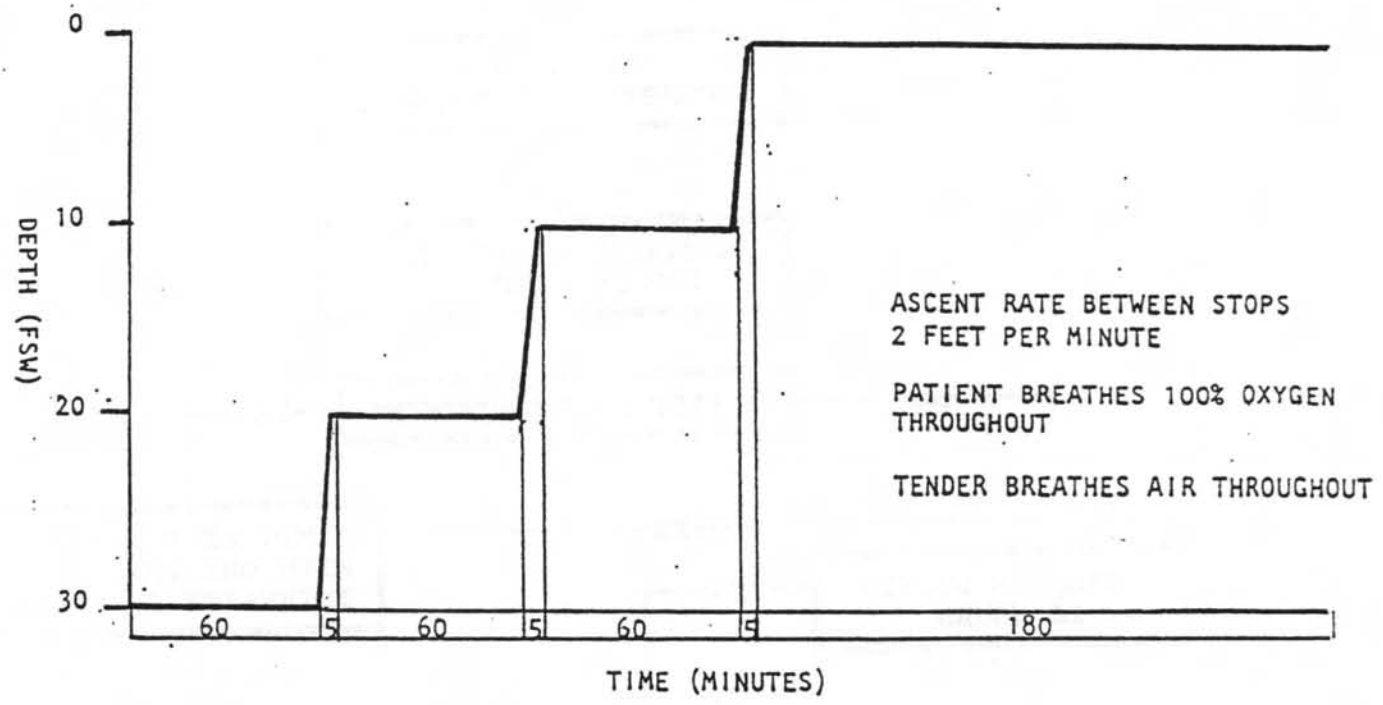
TABLE 1A DEPTH/TIME PROFILE



Air Treatment Table 1A.

figure 3

TYPE I SYMPTOMS



TYPE II SYMPTOMS

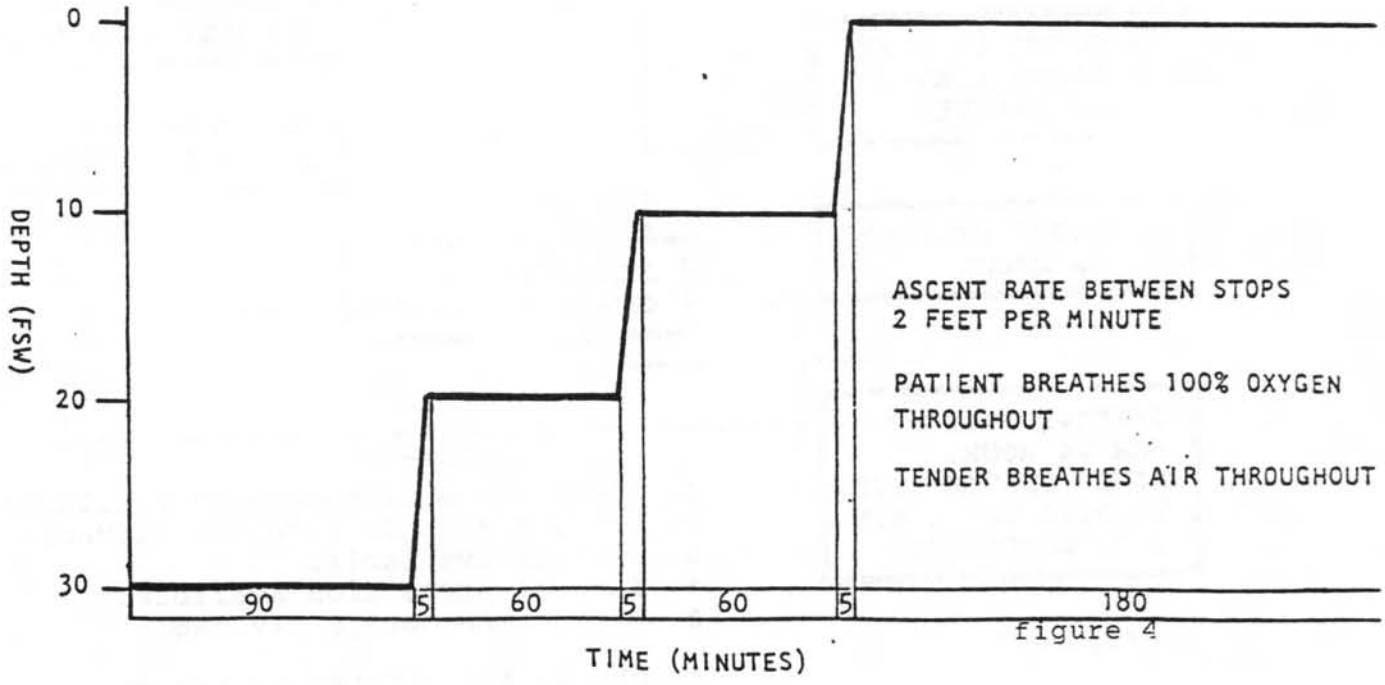
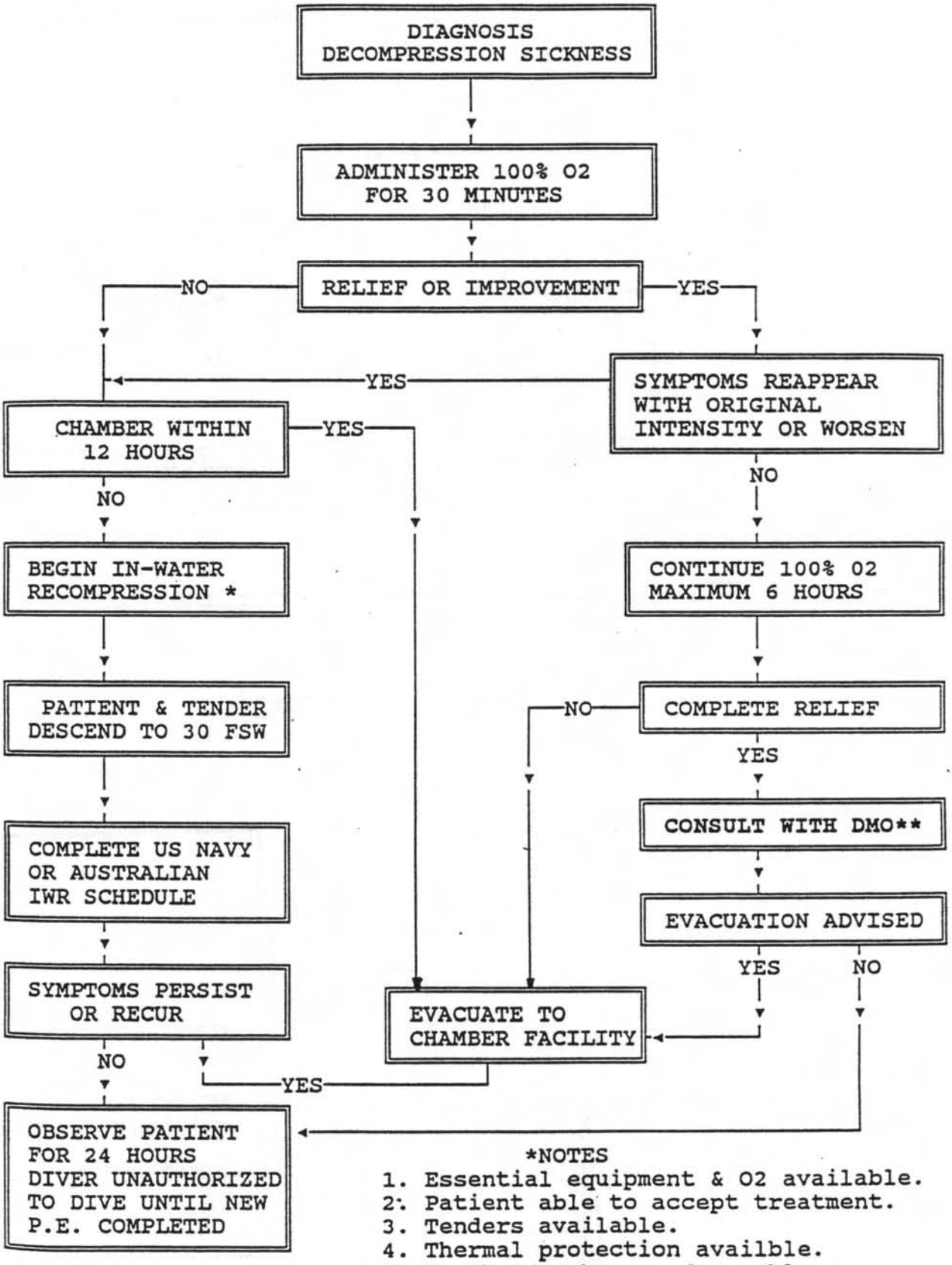


figure 4

IN-WATER RECOMPRESSION USING OXYGEN



*NOTES

1. Essential equipment & O2 available.
2. Patient able to accept treatment.
3. Tenders available.
4. Thermal protection availble.
5. Weather/anchorage favorable.

**** CAN BE ESTABLISHED AT ANY TIME.**

figure 5

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AIR AND OXYGEN REQUIREMENTS

A. Breathing Gas Requirements (Planning Only)

1. SCF Required = PAM X RMV

   a. SCF = Standard Cubic Feet
   b. P = Persons
   c. A = Atmospheres absolute
   d. M = Minutes
   e. RMV = Respiratory minute volume

2. RMV Rates (estimates)

a.	Full gear light work (photography)	0.5 SCF/min
b.	Full gear moderate work (swimming)	1.0 SCF/min
c.	Full gear heavy work (jetting)	1.5 SCF/min
d.	Patient at rest breathing 100% O2	0.5 SCF/min

3. Examples:

   a. A diver swimming at 66 feet for 25 minutes

      Air required = PAM X RMV
      = 1 x 3 x 25 x 1
      = 75 SCF

   b. Patient at rest at 30 feet for 60 minutes
      Oxygen required = PAM X RMV
      = 1 x 1.9 x 60 x 0.5
      = 57 SCF

   c. Patient at rest at 30 feet for 90 min
      Oxygen required (SCF) = PAM x RMV
      = 1 x 1.9 x 90 x .05
      = 85.5 SCF

   d. Patient at rest at surface for 180 minutes
      Oxygen required (SCF) = PAM x RMV
      = 1 x 1 x 180 x 0.5
      = 90 SCF

Appendix A

AIR AND OXYGEN SUPPLY

A. Cylinder Contents (standard cubic feet)

1. Fully charged cylinders (SCF/100 psi)

a. $\text{SCF/100 PSI} = \frac{V2}{P1 \div 100}$

V2 = Equivalent surface volume of fully charged cylinder (SCF)

P1 = Fully charged cylinder pressure (PSI)

2. Partially charged cylinders (actual contents - SCF)

a. $\text{Actual Contents (SCF)} = \frac{V2}{P1 \div 100} \times \frac{P2}{100}$

P2 = Actual cylinder pressure (PSI)

3. Examples

a. Steel SCUBA cylinders (71.2 SCF)

(1) Fully charged (2475 PSI)

$$\text{SCF/100 PSI} = \frac{V2}{P1 \div 100}$$

$$= \frac{71.2}{24.75}$$

$$\text{SCF/100 PSI} = 2.88$$

(2) Partially charged (1800 PSI)

$$\text{Actual contents (SCF)} = \frac{V2}{P1 \div 100} \times \frac{P2}{100}$$

$$= \frac{71.2}{24.75} \times \frac{1800}{100}$$

$$= 2.88 \times 18$$

$$\text{Actual Contents} = 51.8 \text{ SCF}$$

b. Aluminum SCUBA cylinders (80 SCF)

(1) Fully charged (3000 PSI)

$$\text{SCF/100 PSI} = \frac{V2}{P1 \div 100}$$

$$= \frac{80}{30}$$

$$\text{SCF/100 PSI} = 2.6$$

(2) Partially charged (2000 PSI)

$$\text{Actual contents (SCF)} = \frac{V2}{P1 \div 100} \times \frac{P2}{100}$$

$$= \frac{80}{30} \times \frac{2000}{100}$$

$$= 2.6 \times 20$$

$$\text{Actual Contents} = 52 \text{ SCF}$$

c. Oxygen storage cylinders (220 SCF)

(1) Fully charged (2015 PSI)

$$\text{SCF/100 PSI} = \frac{V2}{P1 \div 100}$$

$$= \frac{220}{20.15}$$

$$\text{SCF/100 PSI} = 10.9$$

(2) Partially charged (1600 PSI)

$$\text{Actual contents (SCF)} = \frac{V2}{P1 \div 100} \times \frac{P2}{100}$$

$$= \frac{220}{20.15} \times \frac{1600}{100}$$

$$= 10.9 \times 16$$

$$\text{Actual Contents} = 174.4 \text{ SCF}$$

A-3

EFFICACY OF IMMEDIATE IN WATER RECOMPRESSION IN THE TREATMENT OF CENTRAL NERVOUS SYSTEM DECOMPRESSION SICKNESS*

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SUMMARY

The primary goal of the experimental *in vivo* studies was to establish the efficacy and utility of immediate in water recompression (IIWR) of central nervous system decompression sickness. The experiments were completed during the 10 years of a contract granted by the Hawaii Department of Land and Natural Resources. We established the basic pathophysiology of experimental decompression sickness in dogs and shoats[1-9]. Then we established the efficacy and utility of IIWR in the treatment of experimental central nervous system decompression sickness[10-12]. Based on these experimental studies and the experiences of Hawaii's diving fishers, we believe that safe IIWR protocols can now be used for the treatment of divers suffering from decompression sickness (DCS).

INTRODUCTION

The U.S. Navy Diving Manual provides guidelines used not only Hawaiian divers but divers throughout the United States[13]. Hawaii's fishers dive and operate from boats twelve or more travel-hours away from recompression treatment facilities. The U.S. Navy Diving Manual contains seemingly contradictory instructions for use by divers like Hawaii's fishers. First the manual recommends that injured divers get immediate recompressive treatment. Then, the manual recommends that injured divers go to the nearest recompressive treatment facility for care. Hawaii's fishers and other divers afflicted with decompression sickness often have long and unavoidable delays before receiving recompressive treatment. Such delays often lead to significant irreversible injury or occasionally death of the injured divers. The use of IIWR treatment became the only practical way for some of Hawaii's fishers to treat DCS. Such treatments were done on an ad hoc basis and were frequently successful. We also believe that IIWR would reduce the severity of DCS.

In the animal experiments we compared the efficacy of immediate treatment of experimental decompression sickness with delayed treatment. We judged the efficacy of treatment by return of function. We also correlated recovery with the pathologic abnormalities found in the damaged tissues. Based on these studies, safe scientifically based IIWR protocols for DCS can be developed and tested.

APPROACH TO THE PROBLEM

The Hyperbaric Treatment Center in Honolulu has treated over 600 diving accident victims over the past 10 years. Such a large database remains insufficient to analyze the vagaries of decompression illness, particularly with respect to immediate in water recompressive treatment. Controlled studies using a suitable

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[1]*Dedicated in the memory of the late Edward L. Beckman MD

animal model is the only way to evaluate various treatment strategies. A suitable animal model must consistently develop decompression illness comparable to the disease as seen in humans. The model must be size scaled so that the metabolic rates and the response to intravascular bubbles will be similar to that observed in the human.

In animal experimentation physiologists want to measure all pertinent cardiovascular, pulmonary and neurophysiological variables. Such measurements are invasive and require anesthesia. To get these measurements, you need to intubate the animal, place intravascular cannulas, and insert recording electrodes into the central nervous system. Animals respond to these procedures. The responses may initiate coagulation, produce dependent vascular stasis and depress pulmonary gas exchange. Such responses also occur in DCS. To avoid these confounding problems, we used awake, unanesthetized and unrestrained dogs and pigs.

When we began the experimental studies we used a canine model that we had developed and used successfully for seven years. However, a change in the institutional animal use policy required that we no longer use dogs. Pigs were acceptable alternatives. Young shoats weighing between 50 and 60 kg also develop DCS of the central nervous system that closely emulates the human disease. The pathophysiologic changes are comparable to the canine model we had used previously.

Etiology: Clinically, DCS is a protean disease that affects all tissues of the body, but with different degrees of severity. The current dogma is that decompression illness results from the formation and growth of free nitrogen-laden gas bubbles. Early investigators recognized bubbles in blood of afflicted animals and humans. They thought that the bubbles embolized to produce the disease.

Yount et al. studied the physics of bubble formation and bubble growth in biological tissues[14]. Surfactant molecules coat and stabilize microbubbles in all tissues. Surfactant stabilized micronuclei (SSMN) is the term used to describe these structures. The SSMN are normally gas permeable and equilibrate their internal gas tensions with those of the tissues surrounding them. These micronuclei occur ubiquitously throughout the animal kingdom. A diver develops decompression sickness when he rapidly ascends to the surface after breathing air at depth. For example, when a diver descends to 150 feet sea water (FSW) and stays for twenty-five minutes, he will develop DCS with rapid ascent. At 150 FSW the body tissues rapidly build up nitrogen partial pressures equal to that of the compressed air that the diver breathes. The ubiquitous SSMN are permeable to gas, and they build up a tension of nitrogen gas equal to that of the tissues around them. When the diver rapidly ascends to the surface, he reduces the ambient pressure around his body. The nitrogen pressure in the SSMN becomes greater than the hydrostatic pressure on the SSMN and the bodily tissues. When this over pressure reaches 20 feet of sea water absolute (FSWA) or more than the ambient pressure, the SSMN expand to form unstable macrobubbles. Overpressure within the microbubble is inversely proportional to the size of the microbubble. An overpressure of 20 FSWA will rupture the shell of a 2.0 micron diameter microbubble. The 2 micron diameter micronucleus is probably the largest size of SSMN in the human body. Smaller microbubbles would require a greater overpressure to expand[15]. The reticuloendothelial system, particularly the spleen, will likely remove the larger SSMN.

Microbubbles measuring up to 2.0 microns in diameter grow to form the macrobubbles. Free macrobubbles (0.5 - 1.0 mm in diameter) form in the bodies of divers, aviators and caisson workers and cause DCS. Yount's idea explains

why macrobubbles may occur simultaneously in any tissue and particularly the lipid-rich tissues. Such tissues would be a rich source of surfactant and include the brain, spinal cord, blood, endolymph of the ear and the heads of the long bones of divers. If this thesis is correct, we can no longer postulate that all bubbles form in blood, then embolize through out the body.

Pathophysiology. Once bubbles have formed, we need to understand how they cause disease. Apparently millions of SSMN may exist in an animal without causing difficulty[15]. SSMN vary from less than 0.1 to 2.0 microns in diameter. When macrobubbles form from large SSMN, their diameters may grow from microns to millimeters, a thousandfold increase in size. When expressed as a change in volume, there may be a billion-fold increase in volume. This volume change would simultaneously increase the pressure in the bubble and in the tissue surrounding it.

The pathological effects of bubbles formed in decompression sickness are primarily due to autochthonous bubble formation in the tissues[3]. As autochthonous bubbles grow in a tissue, they exert pressure on the surrounding tissue and compress adjacent capillaries and stop blood flow. Such altered blood flow will cause local ischemia and necrosis, if not reversed quickly. Similarly, a bubble generated from an SSMN in the blood can flow with the blood until blocked by a vessel smaller then the bubble. Thus, the capillary loops of the pulmonary alveoli will likely trap venous bubbles. Arterial bubbles may embolize particularly to small vessels of the brain, spinal cord, kidneys, heart, lungs or bone.

Autochthonous bubbles caused by a too rapid decompression from a dive will form in all tissues, but certain organs, so-called target organs, are more severely afflicted. In large animals including man these organs include blood, lungs, bone and central and peripheral nervous systems.

1. **Blood. Bubbles are found in blood just as in other tissues. Blood represents about 7% of the body weight in shoats and man. So 7% of the bubbles generated in the body by rapid decompression would form in the blood. Up to 10,000 bubbles per minute (0.5-1.0 million total bubbles) may pass in the venous blood of the pulmonary artery of 30-40 kg dogs with DCS[16]. Arterial bubbles may embolize to the brain, lungs, kidneys and heart.**

2. **Lungs. Gas bubbles obstruct the pulmonary capillaries and cause difficulty in breathing and produce the clinical syndrome known as "chokes." Divers and caisson workers used this descriptive term to describe how they felt when they tried to smoke cigarettes. DCS may be mild or cause death. Generally DCS causes an increase in the respiratory rate because of a reduced gas exchange capacity of the lungs. The animal has difficulty breathing and takes deep labored breaths. The bubbles also increase the hydrostatic pressure of the pulmonary capillaries that may produce pulmonary edema.**

3. **The third target organ of DCS is bone. Divers and caisson workers may develop aseptic necrosis the heads of their long bones. Such bony changes were first identified in the early 1900's after X-ray evaluations of bones became available[17,18]. By using bone-seeking radioisotopes, Beckman et al. showed an acute change in the heads of the bones of divers[19]. The divers developed prolonged pain in the joint(s) after diving that showed the acute change on the bone scans. His studies showed that acute bone**

changes occurred in DCS within three days after the onset of the disease. Both dogs and pigs with experimental DCS usually have demonstrable bubbles in the synovial fluid and fatty marrow of the femoral heads.

4. The fourth target organ is the nervous system. Cerebrum, brain stem, cerebellum, spinal cord, nerve roots and peripheral nerves may be involved. In the canine experiments, autochthonous bubbles were found in all parts of the nervous system in DCS[4]. In the study of the central nervous system, clinical observations are important, e.g., when global ischemia of the brain is produced suddenly[20-21]. Unconsciousness occurred in the human within less than six seconds. However, the cells of the CNS do not "die" until several minutes have lapsed. Palmer et al. showed that divers treated for DCS may have apparent clinical recovery; yet, have injuries of the spinal cord as judged by post mortem histopathologic studies[22]. Therefore using both clinical and histopathologic techniques, evaluation of the effects of DCS upon the nervous system can be more accurately assessed. Such studies in both the dogs and pigs showed that both clinical and pathologic changes of the spinal cord relate to DCS. Lesions related to decompression illness were demonstrable within minutes and up to ten days after injury.

TREATMENT OF THE DISEASE

The landmark work of Professor Paul Bert in 1876-78 first described and delineated decompression sickness[23]. As a physiologist, he studied the mechanism by which decompression produced disease, and as a physician he studied methods for treatment of the disease. He investigated the use of oxygen breathing for treatment and the use of recompression. His insightful observations are highly relevant today and are worthy of quoting again.

"The data which have just been reported, and the results of which had already been listed in Table XVIII, show that one of our anticipations was completely realized. Under the effect of inhalation of pure oxygen, the gases contained in the veins and right heart diminished, then disappeared; the heart gurgles either did not appear or stopped when the respiration of oxygen began early. The danger of an immediate death, through stoppage of the pulmonary circulation, was therefore averted. But yet we could not save our animals; the paralysis persisted, and in spite of a real immediate improvement, ended in carrying off our experimental subjects. That is because the inhalation of oxygen could not bring back into the blood stream and dispose of the bubbles of gas which had stopped here and there in the capillaries of the central nervous system. And it could not, for an even better reason, cause the absorption of the bubbles which, as we have seen, escape into the interior of the tissues. Upon them, only recompression can have a beneficial effect. But, on the other hand, recompression cannot cause a considerable collection of gases in the right heart to be redissolved. We are, therefore, led to recommend the successive use of the respiration of oxygen, to eliminate the nitrogen stored up in the right heart, and recompression to dissolve the bubbles which have stopped in the capillaries or are scattered through the tissues. Even so, we cannot be sure of a cure, because the bubbles of gas, when they pass to a free state in the interior of delicate tissues, like those of the spinal cord, may have caused disturbances or lacerations there, the fatal effects of which cannot be averted by the disappearance of the bubbles. It is, then, upon preventive measures, that is, slow decompression, that industry must depend, and that is a point to which we shall return in our third part."

Engineers used these observations to build pressure caissons for bridge and tunnel construction. In 1909 Keayes reported on 3,692 cases of decompression

sickness that he treated during the construction of the tunnel under the East River in New York City[24]. Keayes used recompression as a primary treatment, which he found successful in 90% of his cases. Nevertheless, some men were permanently paralyzed and a few died.

The next important observation concerning IIWR treatment occurred during the rescue salvage of the sunken U.S. submarine, S-51 in the winter of 1925[25]. Very rough seas and cold water prevented safe decompression of the divers in the water. Divers were brought to the surface as rapidly as possible. They were immediately put in a compression chamber on the salvage ship and slowly recompressed. This method of decompression proved effective as the divers did not develop DCS. This observation proved that there is a short interval after surfacing before bubbles form and DCS develops.

In 1935 empirical surface decompression procedures were first tested under controlled laboratory conditions and subsequently in open water sea trials, as reported by Hawkins and Shilling[26]. These investigators tested divers to 167 FSW and increased dive-times using surface decompression until serious decompression sickness occurred. They used the 5-minute "depth to depth" rule for surface decompression. Subsequently, this procedure was incorporated into the operational diving procedures promulgated in the U.S. Navy Bureau of Ships "Diving Manual" in 1943[27].

In 1944 Gouze[28] reported on the question of what is a safe interval between the last stop in the water and recompression in the chamber. The depth of dives varied from 66-108 feet of sea water. The interval of ascent from 40 FSW until recompression in the chamber to 40 FSW varied from 3.5 to 14 minutes. Development of DCS after the recompression-decompression treatment in the chamber was used as a point for stopping the trials. Apparently there was a time limit after which this recompression-decompression would not prevent the occurrence of the DCS after any given depth of dive.

Nevertheless, these investigations lend credence to the empirically developed IIWR procedures evolved by Hawaii's diving fishers. When these divers experienced DCS of any type, they immediately returned to the water for recompression. Slow decompression usually gave relief from the immediate problem and generally prevented a recurrence of the disease. Figure 1 shows the effectiveness of IIWR for Hawaii's diving fishers[29].

Hawaii's diving fishers taught themselves how to use SCUBA gear after World War II. By trial and error, they learned how reduce the risk of DCS and to use IIWR treatment successfully. In the mid 1970s and 80s, these pioneering divers were questioned on how they prevented DCS and used IIWR treatment. At the time of the interviews, the older men had made an average of 11,475 dives each with a range of 5,200 to 23,000 dives. These divers fished using spears, traps, and nets. They used from two to eight scuba tanks (72 cu ft capacity) a day. The deepest air dive reported was 350 FSW, and the black coral divers worked at depths over 300 FSW. These divers averaged 23 years of diving experience (range 10 to 32 years).

These divers reported 527 incidents of DCS treated by IIWR treatment, an average of 22 per diver. The remarkable finding was that IIWR treatment was successful in 462 incidents (87 percent). See Figure 1. In another 51 incidents (10 percent) the divers improved but still suffered, usually from a mild pain or ache that could last several days. These divers used home remedies such as beer and aspirin or took hot or cold showers to relieve their symptoms. Occasionally,

tenderness of the affected limb or fatigue still existed, but relief from pain was satisfactory. In water treatment provided incomplete recovery and was unsatisfactory in 14 incidents (3 percent). These divers sought treatment at the Navy recompression chamber at Pearl Harbor.

In water recompression depths that proved successful averaged 41.3 FSW and ranged from 25 to 85 FSW. In water recompression times averaged about 60 minutes (range of 20-200 minutes). The signs and symptoms varied from the mild arm and shoulder pain to serious CNS abnormalities such as paralysis, loss of vision, movement, or sensation. However, IIWR treatment does not prevent the development of dysbaric osteonecrosis, a disease that many of Hawaii's diving fishers have developed[18].

How long does it take to form bubbles in the pulmonary artery before recompression will not reverse bubble formation? Intuitively, this seems to depend upon how severe the disease is. Clinical experience at the Hyperbaric Treatment Center reveals a few cases in which the disease has apparently been reversed spontaneously without treatment. Other cases of DCS were so overwhelming that death occurred before recompressive treatment. Neither the Hawaiian data on diving fishers nor the Navy data on surface decompression answers this question.

Our animal model of DCS produces a disease so severe that bubbles occur in the pulmonary artery within five minutes after the animal reaches sea level pressure. The disease is sufficiently severe to cause signs of disease (e.g., loss of function of limbs and/or respiratory disturbance). Disease develops in nearly all experimental animals. The model is useful for both clinical and pathologic studies. We used dogs weighing 20-30 Kg and shoats weighing 50-60 Kg.

Study of the efficacy of treatment implies that there is a standard treatment plan. There is no such protocol. Historically the depth of recompression and the duration of the decompression for humans varied tremendously. Even now treatment recommendations differ for each Navy of France, England, Canada, Germany and the United States. Therefore we developed a table for treatment of the experimental animals (dogs and shoats). We used air for recompression to 100 FSW for l0 minutes. Then we gradually reduced decompression over 190 minutes until we reached surface (sea level) pressures. We used recompression pressures to eliminate bubbles by Doppler detection after the animal had been subjected to a bubble-producing dive.

Morita reported the effect of various recompression pressures upon the length of time that bubbles appeared in the pulmonary artery of dogs subjected to a DCS producing chamber dive[30]. He observed that with a recompression pressure of 60 FSW, the bubbles were not detectable by a Doppler bubble detector over the pulmonary artery after 15 minutes. Overpressures of 80 FSW to 100 FSW were necessary to cause a decrease in the number of bubbles detected in the right pulmonary artery after some dives. These data imply that the recompression treatment pressure using air should meet two specific objectives. First the recompression must be at least 100 FSW. Second the recompression should start before the bubbles reach 250 microns or more in diameter. In any event recompression should start when possible immediately after the onset of the disease.

ELAPSED TIME BEFORE TREATMENT OF DCS

In the treatment of DCS time is the most important parameter governing the success or recompressive treatment. At a fixed recompression treatment facility,

treatment is initiated when possible after the patient arrives. However, the disease generally has been manifest for six or more hours.

In experiments, we initiated treatment immediately, as implied by IIWR treatment. We must address two questions:

1. What do we mean by immediate?

2. What are the time constraints that limit the effectiveness of IIWR?

The acceptance and use of surface decompression procedures by the U.S. Navy over the past 50 years provide a guide for the minimum "safe interval." These tables were designed to treat DCS that occurs during surfacing from a dive. The expected result of treatment is that the diver becomes asymptomatic. A gradient overpressure between the gas tension in the SSMN and the ambient pressure outside the skin of the SSMN of 20 FSW is sufficient to grow SSMN into a macrobubble.

For example, the U.S. Navy surface decompression table uses oxygen for a dive to 150 FSW for 30 minutes[13]. In Table P 7-35 the diver returns from 150 FSW to the surface in six minutes. He has 3.0 minutes on the surface to get his helmet off and enter the chamber. Then he has 1.0 minute to recompress to 40 FSW and start breathing oxygen. Bubbles would grow during the six minute ascent to the surface. On arrival at the surface, bubbles would be detectable in the pulmonary artery. One of us (ELB) has examined divers during this surface interval. Bubbles are detectable by Doppler detection over the pulmonary artery. One diver complained of pain in his left shoulder. Both the pain and the bubbles disappeared after recompression and oxygen breathing at 40 FSW. Based on Morita's data, Doppler detectable bubbles may not disappear for 15-30 minutes with 40 FSW overpressure of air[30]. This response time might be shorter with oxygen breathing. His data says that the bubbles would persist up to 18 minutes (3.0 minutes during the surface interval and up to 15 minutes during and after recompression). Growth of bubbles and dissolution curves would suggest a shorter time of around seven minutes before the bubble would no longer damage the tissue.

Kunkle and Beckman extended Yount's studies of bubbles[31-32]. They showed the rates of bubble growth and bubble dissolution at different overpressures. Bubble growth in gelatin approximates the data implied from the evaluation of divers on a U.S. Navy surface decompression schedule on oxygen.

Hills and Butler showed in vivo studies on bubble size that are comparable to the gelatin model[33]. They sized the bubbles that appeared in the right heart of dogs subjected to a decompression sickness producing dive. They reported that bubbles 24-32 microns in diameter appeared in five minutes and that the size of the bubbles detected increased with time after decompression. The largest bubble detected was 700 microns in diameter 40 minutes after decompression.

None of the above referenced studies address what is the maximum interval from the onset of DCS to recompressive treatment to prevent residual injury. This concern is the critical issue in the use of IIWR by Hawaii's diving fishers and other divers.

Hardman and Beckman described the pathogenesis of central nervous system decompression sickness as observed by them in dogs exposed to decompression sickness producing dives[4-5]. Their observations of pathology and times of

appearance are critical to this investigation. They reported as follows: The pathogenesis of central nervous system decompression sickness (CNS DCS) was studied in 50 dogs. The dogs received a pressure profile producing acute CNS DCS. Brain, spinal cord and visceral tissues were fixed in neutral buffered formalin and tissue sections were prepared for examination by light microscopy. Round to oval Space Occupying Lesions (SOLS), so-called autochthonous bubbles, and/or petechial hemorrhages were found principally in the spinal cord white matter and adipose tissue of the trunk and viscera of the animals. Autochthonous bubbles formed in the spinal cord white matter. Hemorrhages were often found eccentric to or filled the SOLS. SOLS remained up to six hours. By 8-12 hours, tiny (~ 1 mm) oval to round foci of necrosis appeared in the spinal white matter in a distribution pattern comparable to SOLS and hemorrhages. In animals surviving 12-24 hours infiltrates of neutrophils appeared in and around the necroses. After 24 hours macrophages appeared and reactive axonal swelling became evident. With resolution of the necroses, glial scars and degeneration attributed to Wallerian degeneration remained. In the dogs autochthonous bubbles formed in the spinal cord white matter, spinal nerves, and adipose tissue. Growing bubbles exerted pressure sufficient to obstruct capillary blood flow. Tissue hypoxia caused necrosis and hemorrhage. These data show that necrosis of the CNS tissue occurred between 8-12 hours. No amount of recompression treatment restored the function of tissue after 12 hours. This time table of pathological events was comparable in dogs and shoats.

To evaluate the efficacy of IIWR, we need to define what "immediate" means. What is the maximum time during which reversal of DCS occurs? This time table seems to vary for each of the target organs. We not only need to establish the maximum limit of a safe surface interval before recompression but also the ranges of times beyond which irreversible damage will probably occur. Although determination of such a maximum safe time limit is highly desirable, it is probably not achievable. The variability between individuals within one species and the interspecies variability (e.g., pigs to man) precludes a precise definition of a "safe" time. Therefore we will assign a time that will provide a high probability of safety (e.g., applicable in 95% of cases). The time that it takes to recompress the diver to treatment depth of 100 FSW, in the water (e.g., three minutes) plus the time during which the pressure must act to dissolve the bubble down to a size where it is not producing a pathological effect, (e.g., at 100 FSW with a less than 250 micron bubble) is about three minutes. There is an unavoidable time loss of about six minutes that will be added into a safe surface interval to arrive at a reasonable time for reaction to an emergency.

Although DCS is a protean disease afflicting all tissues of the body, it principally injures four organ systems-blood, lungs, bone and central nervous system. Therefore, safe surface intervals need to be defined for each of these tissues.

After an unsafe dive, bubbles are first detected in blood of the pulmonary artery by doppler bubble detection equipment. You may detect bubbles over the right pulmonary artery after a dive though no other signs or symptoms of the disease are manifest. Bubbles detected in blood may be very many. Dogs developed signs of decompression sickness when between 500,000 and 1,000,000 bubbles were present over an observation period of four hours. The maximum rate that bubbles were detected over the pulmonary artery was over 6,000 bubbles/minute.

Target one - Blood: The bubbles in blood embolize to the first organ that acts as a filter for venous bubbles, (e.g., the lungs). There is also a direct interaction between bubbles and the plasma to trigger the activation of complement. The

complement system is particularly involved in inflammation and coagulation. Such involvement is demonstrable in vitro[34]. Intuitively, one would expect that 500,000 venous bubbles would produce a catastrophic clotting reaction. However, disseminated intravascular coagulation (DIC) is rare in DCS and then only in severe or terminal cases.

Target two - Lungs: The lungs receive all of the pulmonary artery bubbles. The safe surface interval (SSI) for lungs depends upon two pathological processes: First, the occurrence of a bubble in lung parenchyma that enlarges and produces a "burst" lung and then forms arterial emboli. Second, embolization of the pulmonary arterial bed causes progressive obstruction of flow and gas exchange. As the number of emboli increases, the pulmonary artery pressure rises. Pulmonary edema follows and produces the subjective symptom of "chokes." The progress of pulmonary abnormalities is dependent upon the number of bubbles and the rate that the lungs can dissolve the bubbles. "Chokes" can occur in a few minutes or be progressive over an hour and then slowly subside. The safe surface interval is not predictable. Compression treatment would be required immediately for severe and life threatening bubble formation.

Target three - Bone: The "safe surface interval" for bone would be approximately the anoxic survival time for bone or six hours[35]. Recompression treatment given in less than six hours after the onset of DCS prevents the formation of bone "hot spots" (i.e., areas of bone necrosis) as shown by scintigraphy[19].

Target four - Central Nervous System (CNS): The anoxic survival time for the CNS is much more restrictive[20,21,36]. Irreversible brain damage begins after about seven minutes of anoxia for the brain and about 15 minutes for the spinal cord[37-39]. After cerebral circulatory arrest, a patient loses consciousness in less than 20 seconds. The U.S. Navy surface decompression table for oxygen is set very close to the time required for injury of the brain. The short safe surface interval for the brain will always control the treatment of CNS DCS.

We have estimated the safe surface intervals allowable before recompression will prevent irreversible injury. First we used the clinical examination as a guide and finally the histological examination of the tissues to establish whether injury occurred. The estimated safe surface intervals before recompression for the body's target organs in decompression sickness are:

Brain:	less than seven minutes
Spinal Cord:	less than fifteen minutes
Lungs:	ten to twenty minutes
Bone:	less than six hours
Blood:	no time limit

The course of DCS is dependent upon the severity of the disease. The disease is primarily a function of the number and size of free bubbles formed in the body and the rate at which they form. Bubble number and size are dependent on the gas overload and the preexisting SSMN.

To determine the presence of DCS we used both clinical and postmortem examinations. The clinical examination included observations immediately after a dive and continued through the treatment or euthanasia. If the animal was treated by recompression, the clinical examination was continued during and after treatment for whatever period was defined in the protocol (e.g., 30 minutes to 10 days). We followed the clinical course of awake drug-free dogs and pigs. We could detect intravascular bubbles by Doppler detectors in the dogs, but the pigs

would not tolerate the use of these instruments. Postmortem gross and histologic examinations were done by an experienced neuropathologist (JMH). Tissues from untreated and treated animals were compared. The surface interval before treatment varied from zero minutes to more than an hour to determine the efficacy of treatment. The post treatment survival time before euthanasia was varied to permit the assessment of the inflammatory response, glial scarring and Wallerian degeneration of the CNS[4]. Comparable assessment of bone injury was not done.

We did a controlled experiment using 27 shoats to determine the efficacy of IIWR treatment[11]. Three groups of nine shoats each were used to determine the efficacy of IIWR treatment of experimental DCS. We put all pigs in a dry chamber using a profile that has reliably produced clinical decompression sickness. Nine of the animals received no treatment. Nine animals were observed on the surface for 10 minutes then recompressed. The remaining nine animals were observed for 30 minutes before being recompressed. Treatment for the 18 animals was recompression to 100 FSW on air and then decompressed to sea level over 200 minutes. Following treatment the animals underwent complete post mortem examination about one hour after euthanasia. Evaluation of sections of the brain and spinal cord revealed hemorrhages and gas bubbles in all three groups of animals. The occurrence of hemorrhages observed in any section of the CNS tissue was taken as evidence of nerve injury. The hemorrhages were only found in the spinal white matter. The control group (untreated) of nine animals had spinal hemorrhages in seven animals (78%). The group treated after a 10 minute surface interval had three animals with spinal hemorrhages. The group treated after 30 minutes surface interval had two animals (22%) with spinal hemorrhages. See Figure 2 to show the efficacy of IIWR treatment. Such a response is comparable to the responses observed in Hawaiian divers (Figure 1) Since all treated animals improved clinically, we concluded that the immediate recompression on air was effective in improving CNS DCS. Even so, hemorrhages appeared in animals decompressed within 10 minutes after reaching the surface. This finding indicates that irreversible injury likely occurs very early in the course of CNS DCS.

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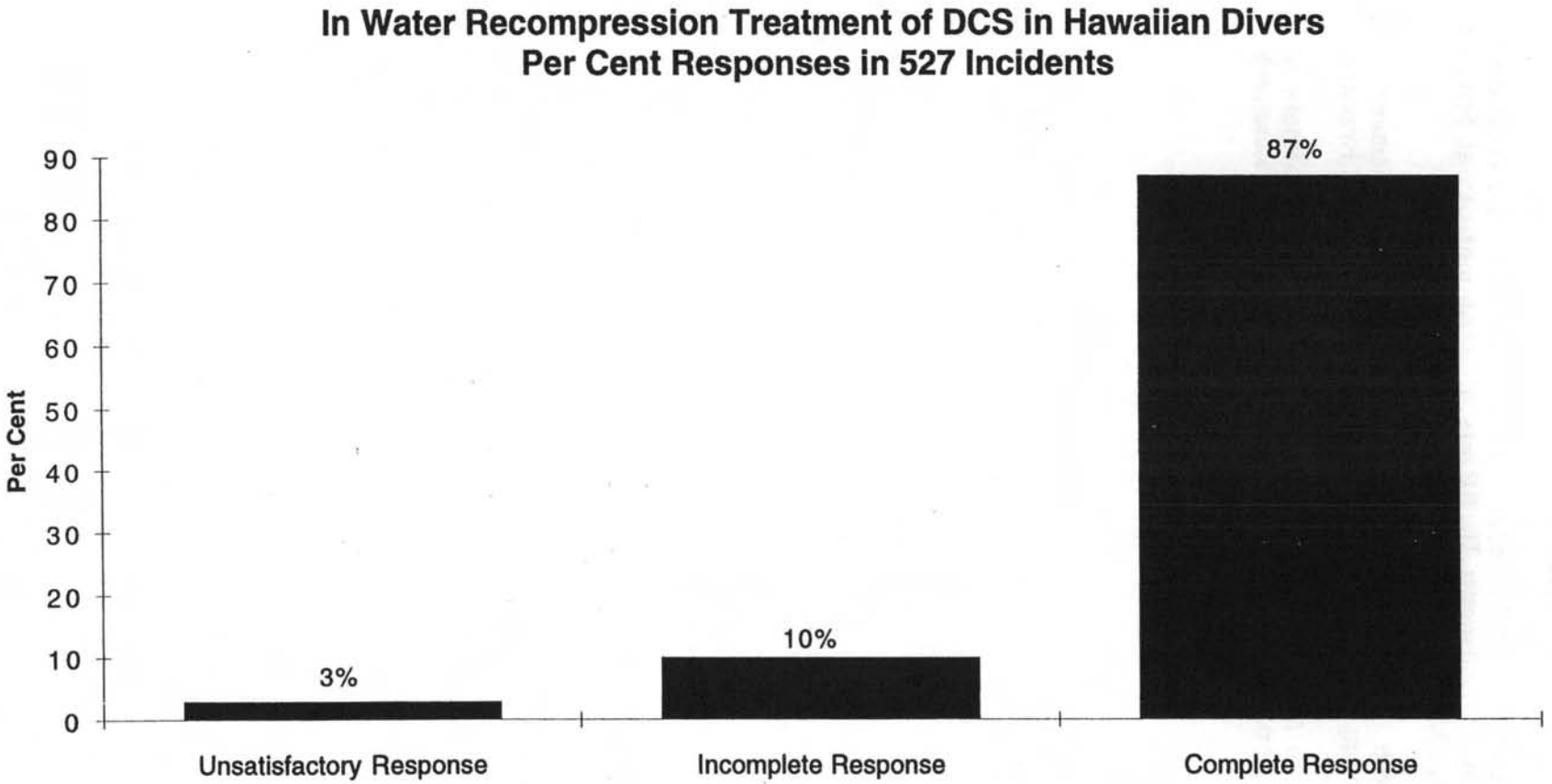

Figure 1.In Water Recompression Treatment of DCS in Hawaiian Divers Per Cent Responses in 527 Incidents

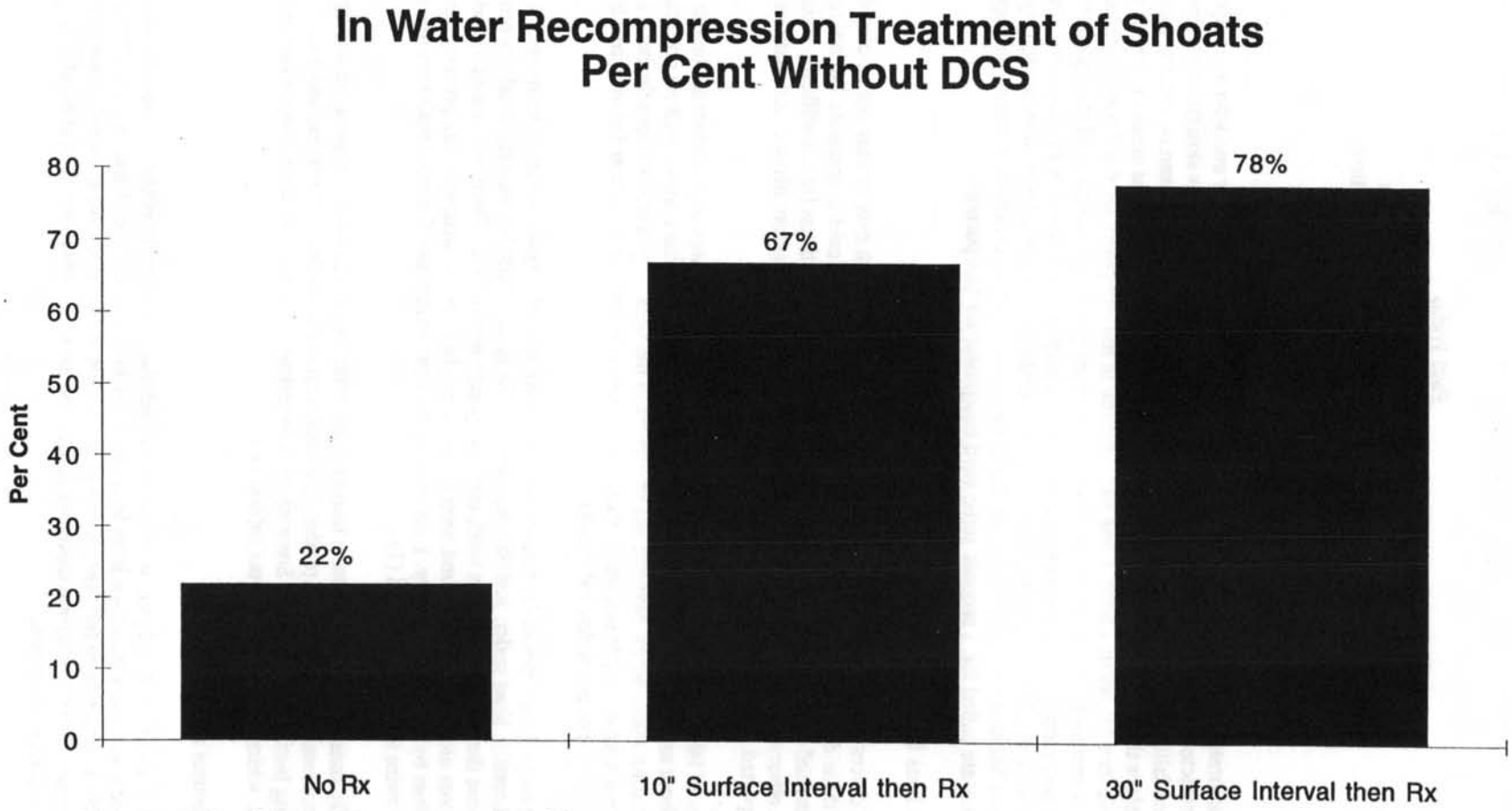

Figure 2.In Water Recompression Treatment of Shoats Per Cent Without DCS With And Without Treatment

BODY HEAT LOSS UNDER WATER AND THERMAL PROTECTION

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Abstract

Laboratory studies of subjects completely submerged in cold water and who are purposely poorly protected thermally produce body heat losses of 800 – 1300 kJ, low skin temperatures, shivering and falling rectal temperature, with afterdrop during rewarming. Exercise during acute cooling delays the fall in rectal temperature and suppresses shivering. These acute exposures to cold are tolerated for about 1 hour. Long slow cooling in the laboratory for 6 – 8 hours simulates dives that last several hours, or dives repeated several times per day. This can produce undetected hypothermia, which leads to fatigue, errors in performance, memory loss and unwillingness to dive again. In water recompression may be needed by a diver who is either acutely hypothermic or who has undetected hypothermia. Good thermal protection on return to water and supplemental heat are called for. I propose some field equipment for this purpose.

Body Heat Loss Under Water

In real life divers usually wear appropriate thermal protection, which may or may not be completely adequate, especially if the dive is prolonged, or repeated several times. Being cold is common, but not much is made of it. Mild, undetected or insidious hypothermia has been reported many times by scientifically trained observers. But since field observations are pretty thin on data, let us start with laboratory dives or simulations in which complete data are reported.

Three types of laboratory exposure to cold in water are: acute exposure nude except for bathing trunks, lasting typically about an hour; cold water exposure while the subject exercises, again with no protection; and mild exposure lasting many hours. Subjects are immersed to the neck, and heat loss from the head is thereby small. Although much loved by physiologists, these three types of exposure are rather far from real life diving and the data are chosen to enlarge physiological insight.

Modifying these three types of laboratory exposure have brought results closer to being useful. First, subjects are submerged totally, head under, just like real divers. This is important because the head is a prime site for heat loss. Second, some thermal protection is used, often purposely insufficient. Third, data taken include some measure of body heat loss along with surface and deep temperature data. In my laboratory the primary measurement has been body heat loss by direct calorimetry. I ask subjects to wear inadequate thermal protection that at least keeps them dry and to swim totally submerged (1).

I will briefly summarize what we have learned from acute exposures totally submerged and lightly protected, similar exposures with exercise to produce metabolic rates like those of divers and underwater swimmers and long slow cooling lasting 6 or 8 hours. Some of these experiments in my case were simulations using an insulated water cooled suit, which was also the direct calorimeter.

Acute exposures submerged

In water at 5, 10 or 15 °C subjects wearing cotton underwear and a thin rubber dry suit swam against a load until they reached a voluntary tolerance limit for cold. Body heat loss was rapid and reached 800 to 1300 kJ (about 200 to 300 kcal). Skin temperature fell quickly to just above that of the water, while rectal temperature fell slowly during the submersion. There was strong shivering despite the exercise. There was a marked afterdrop of rectal temperature during rewarming.

Acute cooling in the water cooled suit with exercise

The most interesting observation here was the delay in internal temperature drop; in fact rectal temperature often increased during the exposure despite low skin temperatures and a measured rate of body heat loss similar to that of the acute exposures submerged. Shivering was minor or absent.

Long slow cooling

In my laboratory this procedure was carefully controlled with a water cooled suit, as I am about to describe, but in other laboratories water immersion has been used (2). Our procedure was to cool the subject in the water cooled suit while measuring his metabolic rate continuously. Heat loss was controlled to be just greater than heat production so that there was no shivering. After 8 hours of this, body heat loss had accumulated to some 1300 kJ (300 kcal) or more, with only about 1 °C reduction in rectal temperature and a small (4 – 5 °C) but significant drop in skin temperature. There is good evidence that there is an early and full cutaneous vasoconstriction during this sort of cooling. During rewarming there is no afterdrop of rectal temperature.

In some studies of slow cooling, intense vasoconstriction has been observed at skin temperatures of 32 to 33 °C, which is surprisingly high. But also, and more important, potentially serious performance decrements occur (3, 4, 5)

Some Useful Observations

Rewarming from these several types of cold water exposure is interesting. In the acute exposures there is strong shivering and this stops quickly as rewarming begins. Shivering is quenched as heat is applied to the body, usually in the first 5 or 10 minutes, and long before the internal temperature has moved up. In fact shivering stops during the period of rectal temperature afterdrop. The skin temperature is rising quickly, which feels delicious, but the person is a long way from being warm. Rewarming is complete when the lost body heat has been restored, determined calorimetrically in the laboratory. At this point body temperatures are not back to where they started. Skin temperature is high and rectal temperature still below its initial level. Thus body temperature data are not helpful in judging when rewarming is complete. Outside the laboratory, to determine if rewarming is complete one should look for the physiological signs that the person is warmer than comfort. This includes flushed pink skin, rapid heart rate (higher than resting rate) and the onset of sweating on the forehead.

Rewarming from long slow cooling is especially interesting. There is no shivering and the rectal temperature is only mildly depressed, so a small amount of heat restores skin temperature and comfort. Nevertheless the person feels vaguely uncomfortable, feels deeply cold ("cold in my bones"), and often does not feel normal until he has had a night of sleep.

Estimating how much heat has been lost from the body is possible if both skin temperature and rectal temperature are measured after the exposure. That is, a calorimeter is not necessary if certain procedures are followed. First, skin temperature must be measured immediately after the exposure, since it increases rapidly after leaving the water. Conversely, rectal temperature should be measured until the afterdrop is complete, which normally takes 15 to 30 minutes, then the lowest value recorded; otherwise the calculation of heat loss will be incorrect. One uses weighting factors for estimating change in mean body temperatures, and then body heat loss. The correct weighting factors are 0.75 times change in rectal temperature and 0.25 for change in skin temperature (1). But this method only applies to acute cooling; long slow cooling does not lend itself to calculating heat loss from body temperature change.

Prolonged and Repeated Work in Cold Water

This was the topic of an Undersea Medical Society workshop (6). It may be pertinent in this discussion of In Water Recompression (IWR). It concerns an undetected mild hypothermia, which is a problem for surface divers, sport divers, scientific diving and others who dive for many hours in a day, or dive more than once in a day. One reason that mild hypothermia is undetected is that a man hates to admit he is cold; it is not manly. They have been wearing their wetsuits haven't they? They are not shivering, they are not blue. But they are in trouble in subtle ways. There is unusual fatigue. Errors creep into well-known tasks. A diver may show overtly or subtly that he is unwilling to dive

again. Excuses are made. How do we know this is the result of body cooling? Because well-designed dry suits with proper insulating garments relieve the problem.

The real world situation is similar to the laboratory studies of long slow cooling. The usual evidence of hypothermia, even mild hypothermia detected from low body temperatures, is lacking. But a lot of heat may have been lost and it is extremely hard to know how to rewarm and for how long.

Hypothermia and In Water Decompression

What has all this to do with IWR? I have read repeatedly that being cold makes decompression illness more likely and worse. By extension being cold is not a good starting point for recompression. A diver with a decompression problem should be rewarmed thoroughly, and it is sometimes hard to know just how to do this. He should be kept warm when he returns to pressure in the water and waits out the procedure. I presume that cutaneous vasoconstriction is undesirable. I presume that cold tissue holds more dissolved gas. I presume that an abnormal distribution of body temperatures is a potential barrier to bringing out the dissolved gas that caused the bends. Therefore it is worth speculating about how, from the thermal standpoint, to handle a diver with decompression illness who must be recompressed in the water.

Thermal Protection

Returning under water for recompression requires at least passive thermal protection as good or better than that the diver is already wearing. In many cases there should also be supplemental heat, since cold contributes to decompression illness. IWR should begin without delay, so early re-entry means rewarming will be largely accomplished under water. After thinking about IWR, I now propose a thermal protection scheme that makes good physiological sense.

Assume that the IWR requires an initial depth of 100 fsw, followed by stays at 30 and 10 fsw; the total time under water is 4 hours. Assume a water temperature of 10 °C. Assume a remote location so that the available equipment is already stowed on the dive boat. That is, there is a kit on board that is available if needed. The thermal package should contain a high quality dry suit, easily donned and large enough that one size fits all, since there is no need for the diver to be mobile or do work. A large, easily donned insulating garment is included. And there is a closed loop tube suit with pump and chemical heat source. The diver to be treated removes whatever thermal gear he (she) has been wearing, slips into the tube suit, the thermal underwear and the dry suit. No connections have to be made for heating. A switch starts the pump from its own battery and another activates the heat source.

The tube suit should cover the torso, upper arms and thighs. A positive displacement pump should recirculate water between the suit and heat exchanger at a rate of 2 liters/min, needing a power of about 50 watts for 5 hours. The chemical heat source delivers a steady 300 watts of heat through the heat exchanger for 5 hours. The source should be easy to quench, either manually, or with an overtemperature switch that cuts off heat when water temperature exceeds 42 °C. Assuming that the heat source delivers heat at only one rate, there should be a way to reduce heat input to the suit by, for example, letting cold sea water into the heat exchanger. Controls should be easy to find and simple to operate.

The only part of this proposed kit that needs development is, to my knowledge, the chemical heat source. There was exploratory work done some years ago, and perhaps more done more recently. I would hope that today's technology could meet the requirement.

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DISCUSSION MORNING SESSION

Moderated by Bill Hamilton, Ph.D.

Dr. Kay: Will the morning speakers come up and take your places at the table. I'm also going to be asking Dr. Bill Hamilton to chair the panel discussion.

Bill is our most recent recipient of the Craig Hoffman Award and has chaired several workshops like this one in the past, especially the Validation of Decompression Tables and Effectiveness of Dive Computers and Repetitive Diving. He just published a new book called *Guide to Diving With Oxygen-Enriched Air*. That's a nitrox book. I'll turn the podium over to Dr. Hamilton.

Dr. Hamilton: Before we open it up to questions, because we've cheated you a little bit in that you didn't get to ask questions at the time of the speakers, I'd like to do a little survey.

With a show of hands, please, how many people came in here with a fairly firm attitude about in-water recompression? That's almost everybody here. We're going to see if we can change that. Dr. Edmonds predicted we probably wouldn't. Let's see if you have some residual questions for any of the speakers, and I think it might be a little more orderly if I asked if anybody wants to ask Carl Edmonds a question.

Dr. Moon: Carl, I have two questions for you. You're absolutely right, whatever we say here will have no bearing on what people choose to do on their own island, and that's quite appropriate, but the mission here is rather, can we see the evidence that will enable us to give advice on this issue?

You presented a large number of cases that seem to have been obtained by retrospective memory dredging from the divers, but can you tell us how many cases you are aware of were actually supervised by a doctor on the scene and could be verifiable in terms of clinical response?

Dr. Edmonds: No, I don't think I can because most of the cases are in contact by phone, not by a doctor on the scene. You don't normally have a doctor on the scene who knows much about this.

Almost all the cases I know of, and certainly my first 25, were all by telephone conversation with the patient some thousands of miles away.

If you're talking about the other point you made, however, about retrospective assessments and memories, in fact, of all those pearl divers had diving logs. That wasn't retrospective really. The logs were done on the day of the dive.

So, I wouldn't actually agree with you on that.

Dr. Moon: The other question is, I think I heard you say it, but could you recommend who exactly would be appropriate for in-water recompression?

I think I heard you say that this is only a first aid measure for mild bends.

Dr. Edmonds: Well, I said that's what we originally intended it for, especially if there was going to be a delay in the MEDIVAC facilities. That is no longer my belief at all.

On the contrary. I believe if you've got a three-hour gap between the patient getting symptoms and getting a MEDIVAC going, then you might as well use it in that time. My feeling is that in that in those 3 hours when the decompression pathology cascade is going to occur, that's when things are going to progress, and that's when bends is going to become established. You might as well use the underwater oxygen immediately.

I don't have very many of the limitations now that I started off with, I'm afraid. I would use it on most divers who have bends and who are cleared to go back into the water. I would probably use it on them.

Dr. Moon: With the proviso that they be conscious, I think you said. Would you agree with that?

Dr. Edmonds: I certainly have written that in the past. Nowadays, I think if I had an unconscious patient in front of me and I had the facilities available, which is to me the full face mask, I almost wonder if I wouldn't now change my mind and take them back down.

Dr. Labosky: I may have missed it, and I'm sorry if I did, but would you then propose that this replace procurement of MEDIVAC system and evacuation to an appropriate hyperbaric facility?

Dr. Edmonds: Oh, under no circumstance. This is an interim treatment.

Dr. Labosky: So if you complete your protocol, then you would still want that diver to go to a diving treatment center?

Dr. Edmonds: I would much prefer that, and even if you don't have time to complete your protocol, it almost doesn't matter that much. This is one of the underwater oxygen treatments you can terminate at any stage.

Oh, no. I'm not suggesting you don't arrange for MEDIVAC. You don't know what's going to happen. Of course you'd arrange the MEDIVAC, and hope to hell that you'd be able to cancel it halfway through.

Dr. Bennett: This is becoming more and more of a problem because our divers, American divers, are diving all over the Pacific and running into problems where there is a long, long flight to get recompression.

Now, the question is, are the people skilled in doing it? It may be that the pearl divers are. It may be that the

abalone divers are, but if a recreational diver suddenly runs into a problem, should he, first of all, be put back in the water then, even though there's nobody trained to do it. Secondly, if this is the case, would more hyper-lights or those kinds of facilities be helpful if they were established in different places where most of the diving is done?

Dr. Edmonds: Well, to answer the last question first, of course, if you put chambers everywhere, you don't need it. That's an obvious answer.

Dr. Bennett: It's not quite as simple. We're talking about a hypo-light monoplace chamber with very limited facilities compared to putting the diver in the water. So, there's a choice here. Which would you have?

Dr. Edmonds: You're very naughty, Peter. You know darn well that I don't like one-man chambers.

Dr. Bennett: Well, you may not like one-man chambers, but you also may not like putting the man back in the water. I know you do like putting the man back in the water, but maybe it's much better to have them on the surface where you can have access to them, and you don't have the problems with the hypothermia and all these other problems we've been hearing about.

Dr. Edmonds: Sure. You have quite a different set of problems, and, of course, what I would really like to have is the option of what to do. So, to answer your first question, which to me is probably the more relevant one, what about the training?

Well, now, we devised this system. We tried to keep it simple. That's why we have the surface supply. That's why you can't go down deeper than 9 meters on our system. We've tried to keep this as simple as we possibly can with equipment you really do have on islands. You do have oxygen-welding equipment on islands. So, we've tried to stick with simple equipment and obey simple rules.

Now, every time someone adds an extra element of complication, you're going to make it more risky for the divers. Most dive shops have no problem at all in setting up this system.

So, if you've got a dive shop on an island, you've got someone who knows how to use the system. You don't have to have every recreational diver in the world knowing how to do it. You've got to have the dive shops know how to do it.

So, to me, that's where the training is, and it's often just a matter of them knowing what's required, and in fact, I think the only bit of equipment that they won't be able to make up easily in most areas is the full face mask, and that's the $25 Chrissy sub.

Dr. Bennett: Well, training could be done, and certainly DAN could institute some of that training, but it still comes down to something that's coming up this afternoon, and I'm sorry that I don't think Rick Lesser's going to be here, but our real problem is going to be a medical-legal issue.

Should you treat the person in a one-man chamber or should you put him in the water? Which is the most dangerous? Because that's where the lawyers are going to have us, and that's what we face every day.

Dr. Edmonds: Sure. And if you've got the option, you've got a problem. But if you don't have the option, then, you know ...

Dr. Hamilton: Thank you very much for the expert advice.

Before the next question, let me do something I should have done at the beginning of the discussion. It might be described as the dilemma of in-water recompression. When a patient is bad enough to really need the treatment, that's when you're a little reluctant to put them back in the water or to put them in the one-man chamber.

NASA faces this dilemma as Dr. Chimiak alluded to in putting something like the hyper-light on board the space station because they are concerned about using or not using the chamber for a really bad case. we won't use it. I'd like to just lay that on the table. And it's something that we sort of have to live with, the dilemma of in-water recompression.

Dr. Hatore: This is for Dr. Webb. Over the past 30 years, I've probably treated around 50 scuba divers for near drowning along with water non-divers, but I've noticed that when they all come into the ER, their rectal temperature always runs between 90 and about 92–93 degrees, and I always wondered how come their body temperature drops so drastically so quickly.

Dr. Webb: Well, I'm supposing that they were diving in cold water to start with.

Dr. Hatore: Monterey runs about 50 degrees, but they're all wearing quarter-inch wet suits.

Dr. Webb: Sure. And they're still wet probably if they haven't taken off the suit, they're still wet.

Dr. Hatore: Yes. Most of the time, they arrive in the ER with their whole suit on.

Dr. Webb: So, there's evaporative heat loss continuing at a very low metabolic rate. So, the rectal temperature does not recover quickly and you've got continued heat loss, probably from the time that you started and got them to the hospital. They probably were still losing heat.

Dr. Barrett: This is a question about Nicaragua that was just presented to the board. The situation there, as Commander Chimiak alluded to, is that there's not very much oxygen available for the treatment of severe decompression illness.

A lot of the boat captains are now practicing in-water decompression on air, and probably the minimum time to transportation to the chamber is about 4 hours. More likely it will probably take them about 8 hours to get someone to the chamber with limited oxygen.

Could you just comment on what you think of in-water recompression on air in that situation?

Mr. Farm: I guess I'm biased in that area, but I believe it has its place, in-water recompression and air. I think of the experiences that were cited, the 500 and something cases that we surveyed. Those were all done on air at that time because oxygen was not a tool that was available to the diving community, and even today, quite frankly, most of the boats use air. There are only a few that I can think of, and one example I showed where that particular boat carried a big bottle and that would really use oxygen.

We would like them to carry oxygen, but you cannot easily get it for the diving community, unless you get a prescription from the physician/ You wouldn't want them to be using that bottle of oxygen with unlimited use and to go down deeper or something like that. That's why we use the tethered line.

The other thing is that divers as a rule are not too eager to change their methods; it's going to take some evolution of time where you're going to get them to appreciate those things. I would try under certain circumstances. For the more case, I would be inclined to try this because you need to help them, and the last example I gave, the diver was actually out on his feet and a little bit out of his mind when they took him down. That's why they had to hold on to him and get him down next to the anchor rope.

When he came to, he thought his good buddies were doing him in under the water. That's when you have to do it. You have to check his breathing and everything else, and I know Peter's concerned about safety and liability, but with small crews like we have, there's a common understanding with my crew what we'll do.

So, permission is granted. That doesn't stop you from being sued, but you already said that this is what you want to do.

Dr. Edmonds: A little plug here. You know, if you really got divers 4 hours away from a reasonable first aid facility. I just can't believe that you shouldn't have oxygen on the boat, not for decompression sickness treatment, but for treatment of your drowning cases which are far more likely to happen.

So, I think it's wrong to have divers remote from first aid facilities without having oxygen available.

Dr. Hamilton: Anybody else on this question before we take the next one?

Mr. Farm: I'd just like to comment that we've encouraged dive charter boats in Hawaii to keep oxygen for surface use, not necessarily for in-water recompression, and there are a number of charter boats, especially on the other islands, on Maui and other places, that do keep that as a first aid for their operations out there. We haven't gone to them specifically to try to encourage any immediate in-water type things. These are the commercial guys, the guys that do it. Surface oxygen, yes, we certainly encourage that. In fact, that's how the DAN kits get sold in Hawaii for surface use.

Dr. Hamilton: Remember the question that was related to a community that does high-risk diving that doesn't have access to oxygen. Isn't that what you meant, Diana?

Dr. Stevenson: When you have slow cooling and there are no immediate signs, is that significant enough to warrant modification of your in-water recompression table.

Dr. Webb: I really don't know why you would modify the in-water recompression. I'd like to improve the chances of success by restoring body heat and protecting from further heat loss. I think that would be the way I'd put it.

Dr. Stevenson: My other question was, in your experience, how effective are heat-treat units for the administration of surface oxygen in core rewarming, viz. heated and humidified oxygen administration units.

Dr. Webb: Oh. Rewarming by respiratory inhalation on the surface? I'm not terribly experienced, but my calculations show me that even with highly humidified gas, air, oxygen, whatever at sea level, you don't gain a whole lot in terms of energy units. You make the person feel much better. It's a very reassuring thing to have, but in terms of actual gain of heat, it's rather small.

Dr. Hamilton: And it's difficult to humidify the gas, and if you don't humidify it, you're not going to gain anything.

Mr. Dunford: First, a comment, Carl Edmonds, about your industrial oxygen. I think that's an important thing to emphasize, is that industrial oxygen is perfectly good stuff to use.

The only thing I would add to that is especially in Third World countries, you need to know the pedigree of the bottle. In other words, what's been in it before they put oxygen into it, and it's on your hands. Sometimes these bottles will be filled with other gases, and it's good to try to find out what those other gases might be.

And the other question I have is for Dr. Webb. Tomorrow, we're going to have a workshop on indigenous diving, and a lot of these divers are diving in waters that are 80 degrees Fahrenheit, and they'll be in the water for 4, 5, 6, and sometimes 7 hours, I would like to know if you think that a native Honduran who is probably five-five and weighs a 150 pounds or less, who's been in the water for 6 hours diving, would he have a problem with hypothermia in an 80-degree water temperature.

Dr. Webb: So, would a man diving in 80-degree Fahrenheit water with no clothes, with no thermo-protection, I presume?

Mr. Dunford: Correct.

Dr. Webb: For as long as 6 or 8 hours?

Mr. Dunford: That's correct.

Dr. Webb: Well, he might be on the edge of this unsuspected hypothermia. It's quite possible.

Dr. Pyle: A question to Dr. Webb. One of the issues that doesn't often get talked about with in-water recompression is the need and/or value for hydration during the process.

My question to you is, to what extent can the thermal problems be mitigated by hydration with warm water in the course of the treatment?

Dr Webb: Are you suggesting warm water to drink?

Dr. Pyle: Correct.

Dr. Webb: Well, you've got so many calories per liter depending on the delta T, the difference between the water temperature and the internal temperature. You're talking 4, 5, 6 liters to do much good.

Dr. Sanchez: I have a tough time trying to convince myself that recompression in water is fine for third-world countries. The problem in Mexico is, we have by law all the diver providers have to have oxygen for surface oxygen from the dive site to the treatment area.

The questions are when to delay this transportation to the treatment area on surface oxygen and try recompression in the water, regardless of whether it's a commercial diver or a sports diver.

I get very tough times trying to see where it fits within our scheme of treatment, either surface oxygen, and then transport them as quickly as we can to the next chamber area.

So, what would you say or how would you justify to do the recompression in oxygen in the water and delay this transfer when you have surface oxygen?

Dr. Edmonds: Under those circumstances, I fully agree with your confusion and doubt, and it's even worse in that in some cases that I know of, we've tried surface oxygen first, and it really hasn't worked, and then we've had to put them under water, and that has worked quite well.

In other cases, as you say, putting them under water's going to delay that transfer. Now, I think under the conditions you've mentioned, I fully agree with your confusion, and I'd be equally confused, and I think I'd just toss a coin. I don't have an answer to that.

Dr. Hamilton: Before you go away, Cuau, you say that Mexico requires by law that you have surface oxygen for someone on their way to the treatment.

Now, aren't there divers in Mexico doing the same kind of lobster fishing that is done in Nicaragua and Honduras? Do those boats have oxygen for that purpose?

Dr. Sanchez: No. That's a different story. This is for sports divers. It's required to have as a provider oxygen on the boat from the dive site to the treatment area, and I reinforced this because before we were getting all these little bottles for 20 minutes of oxygen, and they were 8 hours out in diving sites. So, it was not of good use, and we changed the law, the phrasing of the law, to have enough oxygen from the diving site to the treatment area because there are diving sites that are 8 to 10 hours away by a dive boat in Mexico.

But the indigenous don't have oxygen in that case. That, I understand, but in some of the other ones, they do, yes.

Dr. Hamilton: And in fact, in Cozumel, the rescue scheme is awesome. They don't have helicopters, but they have fast boats that are sort of patrolling, waiting for somebody to call for them. They can get somebody picked up and back to shore very quickly, but that kind of situation isn't really a dilemma. That's prompt treatment. It's not a 12-hour delay. An 8-hour delay begins to cause the question.

Dr. Sanchez: I made that question because we're doing the national plan of rescuing divers regardless of where they're coming, and we have the possibility of using the one-man hyper-light chamber, and the transportable chamber, the Italian version, with 48-inch flexible or collapsible chamber, and the surface oxygen, and the in-water recompression.

So, once you try to set the standard for a country, it's very difficult to understand when you're going to use what, and it doesn't really help to toss a coin because you have to have the answer for the country.

Dr. Edmonds: Could I respond a little bit to that? Firstly, I'm surprised that you have so many chambers around Mexico that you can get to them in that time by boat, but I would beg you, if you're making some sort of standard plan for a country, for God's sake, don't stop your options. Leave them all open.

In other words, let them use the underwater oxygen if they think that's sensible. Let them use the surface oxygen. Believe me, I'm an advocate of surface oxygen. I introduced the thing.

So I'm not saying one's better than the other. I'm saying keep as many options open as you possibly can. Don't block off any of them. I'm surprised, however, that you'd want to get so many portable chambers around the place that you would use them. I tend to agree with Peter Bennett, whatever he said about those chambers.

Dr. Sanchez: You see, it's another problem of geography. Mexico has two Sierras on the side of the coastline. So, anything you want to do in transport to a reasonable treatment area, you have to go up more than 4,000 meters anyway. It's the same thing in Chile. The problem is geography. You're in the dive site, and within one hour, you're in 5,000 meters before you get to the treatment area. That's why you need those chambers.

Dr. Edmonds: Put them under water.

Dr. Shupak: I have a question of Dr. Webb. We know that actually the mild hypothermia like 34 degree centigrade temperature will probably not have an effect on the diver. On the other side, under these conditions, the nitrogen solubility in the blood will increase, thus facilitating the evacuation of nitrogen. So, that might be a benefit. Can I have your answer about this?

Dr. Webb: I've heard this discussed. Ed Beckman for one used to talk about the greater solubility of gas in cold tissue, and 34 degree centigrade temperature, you probably have some much colder tissues near the skin.

If you're trying to get the gas out of solution, then warm them up; if you're trying to hold it until you can do a definitive treatment, maybe you're right. You should keep them cold. That's a hard question.

Dr. Shupak: May I ask another question?

Dr. Hamilton: Let me respond again to this one and point out that at least in my opinion, the effect of temperature on solubility is a relatively small effect for the small change in absolute temperature that we have, but you have an enormous change in circulation as a result of the temperatures.

So, you're certainly dealing with another factor that's at least as important, probably a great deal more important, than the solubility factor. Would you agree with that, Paul?

Dr. Webb: Well, yes. If you're worried about decompression illness, it's not what's happening in the blood, but rather what's happening in the tissue itself, and do you want perfusion or not? If you want the perfusion, then you'd try to warm them up.

Dr. Hamilton: Your second question, Dr. Shupak?

Dr. Shupak: The second question is to Dr. Edmonds.

At the end of your lecture or presentation, you mentioned something about thinking about giving surface mixture -- I mean giving a mixture for in-water recompression of 40% oxygen and helium. Why that?

Dr. Edmonds: I apologize for mentioning that. I sometimes get carried away with lecturing and say things that I shouldn't. Some of us believe that helium is of some value in getting rid of nitrogen, and some work that Ralph Brown and I did many years ago, looking at a gas pocket, namely the middle ear, we worked out that the helium influx into the middle ear being faster than nitrogen outflow could be overcome by having a higher oxygen percentage, so that if you actually were to have 40% oxygen, you actually wouldn't increase the size of the bubble, but you would certainly get rid of your nitrogen much quicker.

So, that was just a way of getting rid of nitrogen. That was just a suggestion, you know. All I'm saying is we should be looking at other options, and that's one of the options that I personally liked. I'm not at all suggesting you use it, although I would.

Dr. Pyle: Just a comment. I know we've had some questions about triage. The afternoon panel will be charged with that as a primary dilemma, and we hope to address it in much greater detail this afternoon.

Dr. Hubner: This is to the whole panel. I've heard back and forth on oxygen, air, in-water recompression or decompression. We produce membrane systems that produce up to 40%, and I've been approached by different divers in places like the Malaysian area where they say they can't get oxygen. My question is, what would you see as the advantage of a nitrox mix of up to 40% versus air in the recompression?

Dr. Webb: The question is, are you better off if you've got 40% oxygen but you don't have 100% oxygen, to use that for your recompression?

Dr. Hubner: Correct.

Dr. Webb: It's better than air.

Dr. Hubner: Obviously, but I mean how much better?

Dr. Webb: Good answer. That's your answer. I mean it's better, but who can say how much in this situation? Too many factors.

Dr. Hubner: So, no studies have been done, and there's no evidence ...

Dr. Webb: Lots of studies have been done on the use of this mix as a diving gas, and it does work as a diving gas, but whether it will make the difference here when you're in a treatment situation, who can say? Certainly it would be better than air. Everybody agrees with that.

The question is, is it worth setting up the system to do that? Crank your system up and get out 90% oxygen, and then we've got something.

Dr. Hubner: Well, actually, now that you mention that, I have for these islands for on-surface made it so they can hook up a scuba bottle with nitrox in it already and do a second pass and breathe off that and have 80% oxygen. So, then my question would be, how's 80% oxygen on the surface compared to a 100%?

Dr. Webb: I would hazard that 80% oxygen can be used in the same place you would use a 100% oxygen, not quite as good, but it would work, and it has some advantages. You'd have a lower toxicity.

Unidentified: I suppose you're looking for actual studies which aren't present, but many of the treatment tables advocate a mix, a nitrox mix, particularly when you're going deeper in the treatment table. If that may help you, if you're trying to convince someone that oxygen is good, I guess you can show them the treatment tables now that use nitrox as an option in the treatment protocol.

Unidentified: Well, it's not a matter of trying to convince someone as far as the nitrox. It's a matter of they say they don't have oxygen, and I was wondering from the

panel as far as if there had been any studies shown on the differences of the nitrox versus the air versus pure O_2.

Unidentified: No, on recompression.

Dr. Labosky: Dr. Hardman, I was very interested in your discussion, and I wonder, could you define for us again what are the conditions within which we will see gray matter bubbles?

Dr. Hardman: I think every tissue can have bubbles in it. The problem is there will be a lot more gas off-loading in lipid-rich tissues where that nitrogen will be concentrated at a higher gradient. So, we don't see it very much.

The other reason is that gray matter in general everywhere is better supplied, blood-supply wise, than other areas. So, the circulation is increased in those areas as well.

I think the other thing that you need to think about in terms of the injury pattern is that we showed that there is some injury as soon as 10 minutes, but one of the big issues that's not been settled, and that's true not only for these lesions but for other types of major injuries to the nervous system, is once you have a certain amount of tissue damage, there's this cascading effect that's talked about. I would guess that in these severe spinal injuries, that would be an issue as well.

Otherwise, I don't see why you should have a response as you obviously do in patients at 6 or 8 or 10 or 12 hours after injury. The intervening tissue is vulnerable to this cascade and it is aborted because of the oxygen, and you probably haven't eliminated all the injury. You've just minimized the extent, it's a cascading effect.

Studies are needed in this area to see what that means because obviously with the evolution of treatment for cytosine blockages of one sort or another, this is an area that might help, and, eventually that would have direct benefit in trying to prevent or at least minimize the extent of the lesion.

Those are areas that our studies don't address directly at all.

Dr. Long: When I was in Cozumel diving recreationally, there's a fee that you pay to the dive master or the dive person that you go out with. That fee goes towards the stocking and preparation and manning of the chambers that are available.

Do you think that if the fees that are charged for licensing of commercial fisheries, such as urchin divers and sea cucumber divers in Maine and Alaska, are used to also cover chambers it might then be an option that would make the need for in-water recompression less likely?

Mr. Farm: I'm open for any way legally to raise money to improve the safety of the diving community; we have suggested other things, such as little surcharges to every bottle that's filled in the islands that would go to elements like this, but this gets to be a political football as to enforcement and everything else.

Dr. Lang: I'm really constructing a decision-making tree for our remote diving sites, and I think some of the answers we already have, and that is, is there a case of decompression sickness? Is it severe enough to not be able to wait and activate the emergency plan? Is there oxygen on site which we require at all our sites for surface administration? Sometimes it's difficult to ship to remote sites because of the International Air Traffic Transport Association requirements.

I'm also looking at in-water recompression. So far we've trained all the folks in oxygen administration on the surface. What I don't have a handle on yet is, and I'd like maybe Jim Chimiak to respond, whether adding that pressure head for in-water oxygen recompression is going to outweigh the risks and complexities of doing it.

CDR Chimiak: Again, that was in the air using in-water oxygen, I think, would be a useful thing as part of your protocol in this remote operation that you're contemplating. I would certainly recommend the use of it in that regard.

Dr. Lang: Thanks.

Dr. Edmonds: Could I add something there? You've made a statement in which you infer that you can't carry oxygen in aircraft.

If you've got a scientific operation going on, then there are all sorts of ways of using in-water or surface oxygen without carrying an oxygen bottle. One of the obvious ways is a superoxide, and you're carrying powder. You're not carrying a pressurized bottle.

So, you can actually take oxygen to the sites. I find this business of saying you can't use it or you can't get it is usually just the people who haven't thought of their alternative ways of getting it.

Dr. Chimiak: Have you heard of ValueJet?

Dr. Edmonds: They work, though.

Dr. Brubakk: I have two questions. One is about the bubbles in the spinal cord. There are no doubts that they are mostly found in the white substance, and it's always said that that is related to the fat content of the mylins. Is that fully correct?

Because according to our survey, when we started to look at what has actually been measured, it seems that all the measurements that exist give the solubility of the spinal cord very, very close to water. So, it may be that the reason is not directly a solubility question, but it has, for instance, something to do with circulatory matters. I don't know the answer to this, but perhaps you can take that first. I have a second question.

Dr. Hardman: Well, I agree with you in one respect. At least in the literature I've looked at; I haven't done any of those studies. So, I'm translating what I see. There are

differences in the chemistry of peripheral and central mylin, but they're pretty subtle. The lipid content in a sense is higher than other parts of the gray matter, but whether the solubility issues raised are necessarily different, I have no information on that.

There does need to be some basic rechecking of some of these things and to make sure that's true. I do agree, though, that vascularity probably is one of the key factors, too. I don't think you can dismiss it because when we did kill animals with a gas load on with no bubbles and then bring them back to the surface after death, the distribution is very similar to the brain—in fact, the highest concentration was seen in the dogs' brain stem.

That may or may not be statistically different, but the numbers were somewhat higher in that group. So, I agree, and I think that there are some basic issues like this that need to be looked at.

Remember there were some studies here, too, from Australia that showed that the surfactant properties of the spinal white matter are quite different from other places, and that could be a key factor.

So, clinically they may be different and important. Generally, I think we all tend to respond to these things in terms of what we know, not what we don't know. So we know that there is a high lipid content, and we assume that's true, but I think you're right. It needs to be explored further.

Dr. Brubakk: I think there is another issue about the recompression treatment that has not been raised here. That is the fact that we have studies documenting this, that there are a lot of divers who never report incidences of decompression sickness. We know, for instance, from our survey in Norway that the experienced commercial divers, about 70% of them, have had partly serious symptoms that they never told anybody about. My question is, one of the things that could be the reason for that is that usually, as it is in my country, and in many countries, perhaps reporting that they have decompression sickness with very serious consequences. The choppers are swirling around. Everybody's screaming, costs a lot of money. The trip is canceled and so on.

So, I think by having simple treatment methods on site would probably add to the number of people treated. So, we could believe that treatment is a good thing, then perhaps exploring this from this point of view may be of benefit.

Dr. Hamilton: Anybody up here have a response to that? Dr. Bennett?

Dr. Bennett: Since I didn't get on very well with Carl, I'm going to try with Jim. Jim, am I right if I say that from what I heard you say, from the U.S. Navy point of view, NOAA also, there was no evidence of in-water recompression being used. The Navy, from what I'm hearing you say, would use a monoplace hyper-light chamber instead?

CDR Chimiak: That's correct. We feel that the resources—and if you calculate doing the in-water recompression and looking at the idea of using rebreather thermal kit, active heating, ideas of a stage—you start running up costs in that direction as well as training on dry suit, rebreather training and that sort of thing.

The idea of going to these portable chambers is certainly an option, and they've got the costs, particularly at the 30-foot range, down to the $10–12,000 range.

So, you eliminate that idea of immediate recompression. The thing you do lose is the speed. The evidence is still out as far as the speed at which we need to recompress. Despite the excellent study at 10 minutes, we still had hemorrhage into the spinal cord after 10 minutes. That's very close to surface decompression.

As you know, that's where we bring the diver out of the water and do the rest of his decompression in the chamber. There's an interval at which you can get away with that without injury, and perhaps what we need to look at is during these cases that the sentinel, viz, abdominal pain, tingling, etc., with those warning symptoms we need to recompress quickly. Being ready in that situation is very important. Lastly, from the Diver's Alert Network, working with civilian divers, it's very difficult just to teach them how to do scuba.

I think many of them are poorly trained. Teaching them rebreather and teaching them some of the techniques that we're talking about here may be very difficult. It never happens to them. Therefore, they don't train with it.

The idea of chipping in in a local area and having an emergency response was tried in Pensacola. It was an utter failure because it never happens to you until it happens. As a result, there's no interest in this sort of training that you're going to need to do this effectively and do it safely.

So, I think it may be confined to the semi-professional; the professional, sea-harvesters and those that have a vested interest in their safety and well-being from a professional point of view.

As far as the Navy's concerned, we like the idea of having these chambers. We like the idea of being able to move that patient as soon as he's injured and start the evacuation process at that point. So, it has a lot of nice advantages. I go along with the portable.

We won't abandon that technique. It will remain in the dive manual as far as we're concerned. It's an option and certainly should remain so.

Dr. Labosky (?): There are political, legal, and training risks for organizations like the Divers Alert Network and the Navy and perhaps NOAA. I think we'll get into these this afternoon because they have a great bearing on whether DAN should recommend a procedure

like this.

But aside from that, for groups who aren't facing issues, legal issues, political issues, if you have the choice of a 3- or 4-hour boat ride back to a chamber as against in-water recompression, would the panel like to comment on the viability of that choice?

Dr. Hamilton: You've already commented, but go ahead, Carl, answer the question.

Dr. Edmonds: Of course, that's the question that's not answerable because the cases will differ. Some will do better in underwater, some will survive on surface oxygen.

But, honestly, you're talking about a rarity. Most of these places are not 3 hours away. The chamber is not 3 hours away by boat ride. That is a great rarity. The chamber is usually 12, 24 hours away. I think that is the break point. It's the 3 hour.

Dr. Labosky: So, if you're 3 hours or closer, go for it, I suppose.

Dr. Edmonds: Possibly, but you're going to make some mistakes in that, I can guarantee it. But there is no answer to my mind.

Unidentified: There's a kind of a guideline, and if you can get there in 3 hours, just go Start on oxygen, go, and if you have 12 hours to get to the chamber, probably go in the water.

Dr. Labosky: From the drift, I presume then that we may begin to see these monoplace chambers popping up, and the question I have is, will the transfer chambers be considered to be adequate to run our Table 6 by those who do them, who use them, and will this then be viewed as replacing transfer to an appropriate hyperbaric center?

Unidentified: We need another workshop on small chambers. But that's a very good question, and the same thing could apply to the in-water recompression. It does not. Carl made the case, and I think most of us agree, it does not replace treatment. It's something you do until you get a proper treatment.

Dr. Hanson: Dr. Hardman, in your studies, you didn't look at any of the animals between the surface immediate, surface arrival and 10 minutes, did you? Did you look at any animals earlier than the 10 minutes post-surface?

Dr. Hardman: Well, we practically couldn't get them fixed so we could. By the time we got them out where we could get to them—pigs are not easy to deal with. They're feisty sometimes, and, so our system, I'd say, on the average, it would take 10 minutes. That was part of the reason we used 10 minutes. That was as fast as we could get them where we could actually do what we needed to do.

Dr. Hanson (?): Do you have any plans for trying to get some other animal and look at that 10-minute interval?

Dr. Hardman: No, I don't have any problem with it. I think we were hoping we wouldn't find any injury at 10 minutes, and it would be a non-issue, but it is an issue.

I think in a scientific sense, it probably would be nice to know how much earlier it is. We do know, though, from other studies that the brain is not irreversible if it's under 5 minutes, probably it wouldn't be irreversibly damaged, unless there's actual severance of the axons.

From the ischemic problems that we know, that's probably not what happens. The severance occurs with a little bit of time. So, you probably could enhance that by 5 minutes and maybe prove what you're saying. I don't know.

Dr. Sutherland: Two-part. One a clarification. We're evaluating some chambers right now. It's kind of political.

(Question off-mike)

Unidentified: And for evacuation, what Dave means is during that evacuation, you'll actually run a treatment table 6 in the hyper-light chamber. There is a possibility, of course, as Dr. Labosky said, by the time you arrive at the chamber, you may have completed treatment table 6. At that point, you'd evaluate the patient accordingly.

(Question off-mike)

Unidentified: Yes. Stretchers.

(Question off-mike)

Unidentified: An anonymous person asked—I won't say who it was now—but asked me to ask the question: Is in-water recompression considered an experimental treatment?

Unidentified: I don't think anybody here would say that it is.

CDR Chimiak: Essentially, it is in the dive manual as in-water recompression, and it's been approved. Until it leaves NEDU, it's considered experimental from the Navy point of view, and maybe Dave might be able to comment on that.

Dr. Sutherland; Well, I'll answer it. It's in the dive manual right now. It's in there. We can use it. So, we would not call it experimental. We would just say it's an option that we would use only if we didn't have other options available, although the classic medical officer response is, like anything else, it depends on the situation. And that's why the dive manual gives us the ability to recommend changes for any of that stuff, including the tables, and that's a classic response that we'll give to everybody, that it depends on the situation. So, it lets us cover pretty much everything.

Dr. Hamilton: Dr. Labosky, are you making a comment relative to this last question?

Dr. Labosky: No.

Dr. Hamilton: Can you wait till after lunch? Because Dr. Kay wants to call us to lunch.

Dr. Kay: I'd like to formally adjourn this morning's session. Before I do so, I want to let everybody know that one of our sponsors, Best Publishing, is going to be

packing up after we're finished this morning. So, if anybody has any last-minute purchases they want to make, better stop by that booth before they leave, and we'll meet upstairs in the Metropolitan Room.

AFTERNOON SESSION

Moderated by Caroline Fife, M.D.

Excellent memorial to Ed at lunch time, and I'm going to introduce Ed Thalmann, who is a retired Navy captain. He is the Assistant Medical Director of the Divers Alert Network, the former senior medical officer of NEDU, and the former head of the Diving and Environmental Physiology lab at NAMRI. He's going to talk about oxygen toxicity. I like to refer to Ed as the Pollyanna of the diving world.

Oxygen Toxicity During In-Water Recompression

Edward D. Thalmann, Captain, Medical Corps, US Navy (retired)
Department of Family and Community Medicine, Department of Anesthesia, Divers Alert Network; Duke University Medical Center, Durham NC.

In-water recompression is approached with ambivalence by both the U.S. Navy and the Royal Navy. The Royal Navy has no in-water procedure and the U.S. Navy allows in-water recompression to be used only when evacuation to a recompression facility will not be delayed. The reason for this ambivalence rests with oxygen toxicity. The minimum treatment depth used for the vast majority of chamber recompressions is 60 feet of seawater (fsw) equivalent to 2.8 ata. Experience has shown that in the dry, resting chamber environment oxygen toxicity is unlikely at 2.8 ata, but occasionally occurs. For in-water recompression, a maximum treatment depth of 30 fsw has been proposed to reduce the likelihood of oxygen toxicity. However, it should be remembered, that in developing their 100% oxygen recompression tables, the US Navy found that even in those individuals who obtained complete relief at 30 fsw, there was a higher recurrence rate of symptoms than in those individuals who where recompressed to 60 fsw (1). This talk is not about efficacy, but I did want to make the point that in solving one problem, oxygen toxicity, we may be creating another, decrease in treatment efficacy.

In-water recompression is a practical subject and I will, therefore, keep this talk practical in nature. There are many standard references on diving medicine and physiology that one can consult to get details on the biochemical mechanisms of oxygen toxicity, the best starting place for the uninitiated being Bennett and Elliott (2). All I will say here is that oxygen toxicity is thought to be caused by oxygen free radicals overwhelming the natural defense systems in the cell thereby causing tissue damage. Two organ systems, the lung and the central nervous system (CNS), are the ones of concern in diving since that is where the brunt of the effect is concentrated.

Lung oxygen toxicity begins with some substernal burning during inspiration and eventually there is a reduction in vital capacity and mid-expiratory flow rates which revert to normal when the exposure is stopped. If exposures continues after the onset of the inspiratory discomfort, after many hours, there will be cellular proliferation and changes ultimately resulting in fibrosis and impairment of oxygen exchange. In practice, lung oxygen toxicity symptoms are initially noted only after many hours of oxygen exposure and the worst outcome in conscious divers is usually only a reduction in lung vital capacity or mid-expiratory flow rates. The reason is that the inspiratory pain eventually becomes severe enough that the diver will be unwilling to continue O2 breathing, and this usually occurs well before the permanent proliferative and fibrotic changes set in. Even with extended treatments at 2.8 ata using the protocol for a USN Treatment Table 6 with two extensions at 60 and 30 fsw, lung oxygen toxicity is limited to some mild, reversible, respiratory symptoms, will not limit treatment or put anybody's life in danger. The bottom line is, lung oxygen toxicity will not be of great concern for in-water recompression.

Oxygen toxicity involving the CNS is another matter. CNS oxygen toxicity is manifest by a spectrum of symptoms including tunnel vision, tinnitus or roaring in the ears, disorientation, aphasia, nystagmus or incoordination. These symptoms, while annoying or uncomfortable, do not put the diver at risk of injury. However, CNS oxygen toxicity may also result in a grand mal seizure. While the seizure, in and of itself, is not harmful, injury may occur from hitting something

Table 1 U.S. Navy 100% O2 Depth/Time Limits	
Depth (fsw)	Maximum Exposure Time (min)
20	240
25	240
30	80
35	25
40	15
50	10

while thrashing about or drowning may occur if a submerged diver loses his mouthpiece. So, the goal in dealing with CNS oxygen toxicity is to avoid seizures.

Oxygen toxicity is a PO2-time phenomenon, that is at high PO2 levels symptoms may occur in a short time, while at lower pressures, longer exposures are required. Table 1 shows the latest USN recommended oxygen depth time exposure limits based on studies done at the Navy Experimental Diving Unit (NEDU). Divers were submerged, breathed from a Draeger LAR V 100% oxygen rebreather, and did mild intermittent exercise during the entire exposure (3,4). The point to note here is that at the shallower depths, the permissible exposure times are much longer than at the deeper depths. The 240 min times reflect the maximum exposure times in the study. Even though an exposure time of 240 min was recommended at 20 fsw, a single O2 convulsion did occur after 75 min at that depth (3). There were 63 exposures with no symptoms, a factor heavily weighing towards recommending this time for operational use. One important observation from this study was the fact that when oxygen convulsions did occur, they did so with little or no warning. It appeared that if another symptom was perceived and recognized, enough time had passed such that a convulsion was unlikely to occur. In other cases, the convulsion occurred so rapidly after the first perception of a symptom (if one occurred at all) that the diver could take no action to avoid the convulsion. This means that there is no hierarchy of symptoms from mild to definite to convulsions. They occur randomly, in no particular order, and the convulsion may be the one and only symptom.

So the question is how can one avoid an oxygen convulsion? One way would be to reduce the PO2 below some threshold where symptoms would not occur. Harabin (5) analyzed the NEDU data cited above and developed a mathematical model for predicting the probability of an O2 symptom based on the PO2/time profile for a single depth exposure. In the NEDU study (3), the authors divided symptoms occurring during a dive into Probable, Definite, and Convulsions. Probable symptoms (light headedness, apprehension, dysphoria, lethargy, transient nausea) were those which could be due to oxygen toxicity but were equivocal enough that they could also have been attributed to other causes. Definite symptoms (muscle twitching, tinnitus, tunnel vision, disorientation, aphasia, nystagmus, incoordination) and Convulsions were symptoms which were definitely thought to be due to oxygen toxicity. If all symptoms which caused a diver to terminate an exposure (divers were instructed to stop their dive if they thought they had any symptom of O2 toxicity) were considered, then Harabin's model predicted a threshold of 1.3 ata (9 fsw). If only Definite symptoms and Convulsions were considered then the threshold was 1.7 ata (23 fsw). The problem here is that restricting depth for treatment of DCS to 23 fsw will probably have a negative impact on treatment efficacy.

One further note about Harabin's work. Although a mathematical model for CNS O2 toxicity was developed it applies only to single depth exposures where the PO2 at depth is constant. Multilevel exposures have been investigated (4,7) but no model has yet been developed which reasonably describes the outcomes in theses studies. So, for multi-depth 100% O2

exposures there is at present no reliable way to estimate the probability of an O2 convulsion occurring. Finally, the discussion above applies only to 100% oxygen breathing. In mixed gas diving at equivalent PO2 levels, nitrogen increases gas density causing CO2 retention which may result in an increased susceptibility to O2 toxicity. See Donald (6) for a discussion of the controversy surrounding this.

All of the depth/time limits discussed above are based on submerged exercising divers. Donald (6) showed that both exercise and immersion will increase susceptibility to O2 toxicity. That is why two hour exposures a 2 ata (a common clinical Hyperbaric Oxygen Therapy regimen) in a dry chamber with resting patients has a vanishingly small (but not zero!) probability of producing a symptom. So, one might consider that a 2 hour exposure at 30 fsw might be very safe. The problem is that with in-water recompression, the diver is immersed and Donald showed that immersion increases the probability of oxygen toxicity. He examined limits for resting, submerged divers and after 2 hours at 25 fsw had only one case of nausea out of 29 divers, but at 30 fsw there were two convulsions at 43 and 48 min.

Inserting short periods of air breathing to interrupt continuous oxygen exposures is a technique commonly employed to reduce the probability of oxygen toxicity, however, the degree of impact on the incidence of oxygen convulsions in humans is presently unknown.

`The bottom line here is that avoiding CNS oxygen toxicity by reducing the PO2 level will result in diminishing the therapeutic effect for treatment of DCS, while using 100% O2 at the minimally effective therapeutic depth of 30 fsw will put a diver in a situation where oxygen convulsions have been observed. So one cannot do therapeutic in-water recompression and be confident an oxygen convulsions will not occur. Those executing in-water recompression treatments must be trained to handle an in-water convulsion, and the treatment carried out in such a way to minimize diver injury should one occur.

The method prescribed by the US Navy for executing in-water recompressions (8) is to use a Draeger LAR V 100% O2 rebreather with a full face mask and adequate thermal protection. It also stipulates that in-water recompression is to be done only by individuals trained and qualified to use the LAR V. Part of that training includes how to handle an underwater convulsion. This includes the following steps:

a) **Assume a position behind the convulsing diver. Release the victim's weight belt unless he is wearing a dry suit, in which case the weight belt should be left in place to prevent the diver from assuming a face down position on the surface.**
b) **Leave the victim's mouthpiece in his mouth. If it is not in his mouth, do not attempt to replace it; however, if time permits, endure that the mouthpiece is switched to the surface position.** (*This step is not necessary if the diver is wearing a full face mask. Author)*
c) **Grasp the victim around his chest above the underwater breathing apparatus (UBA) or between the UBA and his body. If difficulty is encountered in gaining control of the victim in this manner, the rescuer should use the best method possible to gain control. The UBA waist or neck strap may be grasped if necessary.**
d) **Make a controlled ascent to the surface, maintaining a slight positive pressure on the diver's chest to assist exhalation.** (*If the diver is wearing a full face mask remain at depth until the convulsion subsides if at all possible. Author)*

e) **If additional buoyancy is required, activate the victim's life jacket. The rescuer should not release his own weight belt or inflate his own life jacket.**
f) **Upon reaching the surface, inflate the victim's life jacket if not previously done.**
g) **Remove the victim's mouthpiece and switch the valve to SURFACE to prevent the possibility of the rig flooding and weighing down the victim.** (*This is not necessary if the diver is wearing a full face mask. Also, if the diver is using open circuit SCUBA you will not be able to remove the mouthpiece if the diver is convulsing, and this should never be done forcefully. Once the convulsion stops, if the mouthpiece is secure and the diver is breathing then leave it in place. If not breathing, remove the mouthpiece and begin rescue breathing. Author.)*
h) **Signal for emergency pickup.**
i) **Once the convulsion has subsided, open the victim's airway by tilting his head back slightly.**
j) **Ensure the victim is breathing. Mouth to mouth breathing may be initiated if necessary.**
k) **If an upward excursion occurred during the actual convulsion, transport to the nearest chamber and have the victim evaluated by an individual trained to recognize and treat diving-related illness.**

The bold text is taken directly from the US Navy diving Manual (8) while the italics are mine.

The above procedure is directed at swimmers using a 100% O2 rebreather. Step k is problematic for in-water recompression because it is presumably being done because no recompression facility is close at hand. What this means is that to safely execute in-water recompression one must have total control over diver depth. The best way to do this is with a dive stage. If only a descent line is available then there must be some way of ensuring that the diver will not inadvertently sink or make an uncontrolled ascent to the surface if a convulsion occurs. As you can see, procedures and equipment must be worked out well in advance and individuals fully trained in their use. If an in-water convulsion occurs, then the reaction should be instinctual, and procedures executed meticulously.

The U.S. Navy procedures may not be directly applicable to procedures suitable for recreational or technical divers but they do provide a framework on which to base such procedures. Certainly any procedures developed should be at least as detailed and comprehensive as the Navy's.

Besides having absolute control over diver depth, proper thermal protection must be available to ensure that the diver does not get cold, which could increase the likelihood of oxygen toxicity. A full face mask is almost mandatory for in-water treatments, it is the best way to prevent drowning should a convulsion occur. Divers severely affected by DCS may not be able to keep a mouthpiece in place.

Given that there is no guarantee that CNS oxygen toxicity will not occur, one must consider what the benefit of in-water recompression is given the risk. There is not yet a sufficient body of evidence documenting what benefit is derived from a 30 fsw recompression. While

uncomfortable, joint pain is not life threatening, and probably not harmful unless there have been many untreated episodes. So one could argue that the proper treatment here is analgesics and eventual evacuation . If spinal cord or CNS symptoms occur then one wonders what the effect of a partial treatment will have on eventual outcome. In cases of life threatening DCS, it may not even be possible, or it may be extremely hazardous, to execute in-water recompression. If the divers disease is due to a rapid uncontrolled ascent then the possibility of arterial gas embolism must be considered, and then the problem of treating at depths below 30 fsw arises.

On thing is certain, **in-water recompression should never be attempted except as a treatment of last resort when it is certain that delaying recompression might put the diver in danger of significant morbidity, and when the procedure can be done safely by trained individuals and will not delay evacuation to a recompression facility.**

Having said that, if one plans on using in-water recompression then a rigorous training program must be put in place and those expected to perform the procedure fully trained. There must be training in the proper handling and maintenance of 100% breathing gear. The possibility of an oxygen convulsion must always be considered and procedures for handling this eventuality worked out and fully rehearsed in advance. Training must be hands on and include mock convulsions under as realistic conditions as possible.(This approach was used for all NEDU studies and there was no injury from any of the convulsions that occurred). Equipment must be available to absolutely control diver depth, even if he is convulsing. In addition a full face mask should be used, along with proper thermal protection.

One last note. The opinions expressed above are my own and should not be interpreted to reflect the policy or recommendations of the Divers Alert Network.

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Note: Both NEDU and NMRI reports are available through: National Technical Information Service, 5385 Port royal Road, Springfield VA 22161.

Mr. Dunford: Dr. Thalmann, you've basically started to lay out the concept of a lower threshold for recompressing someone in the water. I think you're suggesting that a Type 1 hit would not be an appropriate level of severity to put somebody in this condition in the water.

Now we have to go into Type 2. Where do we draw the line on Type 2? If we do draw the line on Type 2, how do we train somebody who is a recreational diver or even a professional diver to try and select the proper level of potential morbidity down the road, he has to make a decision on whether you should put this guy in the water or not? Any ideas?

Dr. Thalmann: But that's not what my talk was about. I think Richard Moon will cover some of that. This is a problem. Certainly in the military, if you're looking at the special forces, they go on their operations, some of them, knowing that they're not going to be able to evacuate. So, they have no option but to treat everything.

What can you get away without treating? Well, it all depends. Certainly if it's only pain, I don't think there's any evidence that a pain-only bend in the shoulder is any worse than a shoulder pain from playing tennis. So, I don't think there's going to be any long-term morbidity from that.

Once you get into very sick divers, you run right into what Carl said. You're between a rock and a hard place. You would like to treat them right away because of the rapidly evolving symptoms, but the treatment may put them in more danger. This is where training comes in.

In theory, if you could put the diver in a helmet, in a hot water suit, on a stage, and you had people that really knew what they were doing, you ought to be able to execute in-water recompression just as well as you could do it in a chamber. That's in theory.

But this requires, I think, a degree of training which may be unattainable in the sport diving community.

Dr. Edmonds: I've got a lot of comments I'm not going to make, but I've got a question. Once you get rid of all the indefinite cases, the confusing cases that have been on rebreathing sets because they really are very confusing, how many convulsions have we actually seen on open circuit oxygen at 9 meters or less?

Dr. Thalmann: I've heard of one. The man died. Don't get me wrong. I'm not saying that convulsions are so rampant that we're going to kill divers. My point is that if you're going to deliver oxygen in the water, you have to be prepared to treat a convulsion if it occurs. We may never use that training, but you need that training, and you need a plan.

Dr. Edmonds: No. What I'm trying to say is how many cases actually have happened, you know, on oxygen, 9 meters or less, open circuit?

Dr. Thalmann: On open circuit?

Dr. Edmonds: Yes. Once you start to rebreathe, you've got a carbon dioxide problem. You've got all sorts of problems that come into it. That's what's wrong with Donald's work after all. We didn't know what we were talking about in many of the cases. So, we really need to look at open circuit oxygen.

Dr. Thalmann: Well, are you suggesting that we don't have to worry about it?

Dr. Edmonds: Oh, no. I'm suggesting I'd love to have some data on one or two cases. I'm searching for them desperately.

Dr. Thalmann: Is your suggestion then we don't need to worry about oxygen?

Dr. Edmonds: No, not at all.

Dr. Thalmann: And therefore we don't need to train for it?

Dr. Edmonds: Well, if you're starting to use things like rebreathing sets underwater, yes, you've got to be trained.

Dr. Thalmann: I can assure you that none of the convulsions that we've experienced had anything to do with CO_2.

Dr. Edmonds: That's what Donald said.

Dr. Thalmann: No. We know it because we were monitoring it breath-by-breath.

Dr. Edmonds: Right.

Dr. Thalmann: So we're doing a scientific study fairly meticulously, and we knew how much CO_2 there was.

Dr. Edmonds: Okay.

Dr. Thalmann: You're not here looking at a study which was over-shadowed, as some of Donald's work was, by the possibility of increased CO_2, but as I said, we had one convulsion very shallow, and I have heard anecdotally of an O_2 convulsion occurring at the 20-foot stop during 100% oxygen breathing for decompression.

So, the probability is not zero. It may be low.

Dr. Edmonds: But even that case, I think, was from the deep dive region. Now I've got quite a few deep divers that have convulsed at 20 and 10 feet on air. So, you can't extrapolate from that.

Dr. Thalmann: Well I'm really just saying that you need to be prepared to treat a convulsion if it occurs. I don't think I've said anything about the incidence because I don't know what the incidence is, but I do know it's not zero.

Dr. Fife: That's all the time. I'm going to introduce Richard Pyle. He's currently working at the Collection of the Bishop Museum in Honolulu, Hawaii. He's working on his Ph.D. in zoology, but the reason we've got him here is that he has extensive experience with the crystalline rebreather. He's published many articles on in-water

recompression, and he's going to speak to us about the practical aspects of things, such as gas consumption and other technical challenges, and part of the technical challenge. So, for the moment, I just want to commend Ed Kay and Merrill Spencer for what they did to get this program together. It's always far more work than it looks.

KEEPING UP WITH THE TIMES: APPLICATIONS OF TECHNICAL DIVING PRACTICES FOR IN-WATER RECOMPRESSION

Richard L. Pyle
Ichthyology, Bishop Museum

Abstract

With the proliferation of so-called "technical" diving practices among recreational divers, has come an increased potential for Decompression Illness (DCI), and consequent increased interest in the topic of in-water recompression (IWR). Many of the reasons often cited for not conducting IWR (inability to deliver oxygen to a diver underwater, risk of oxygen-induced convulsions, complexities of staged in-water decompression procedures, insufficient logistical support, thermal concerns, etc.) are either negated, or are of less concern to trained technical divers, who must deal with such issues on a routine basis. This combination of increased potential need for, and increased ability to manage IWR by technical divers, make them ideal candidates for performing IWR under appropriate circumstances. Existing published methods of IWR might be improved upon in light of common technical diving practices, and a new method of conducting IWR specifically targeted at the technical diving community is proposed. Although the questions of whether IWR is a valid response to DCI, and if so, what specific methods are optimal represent the bulk of discussion surrounding the topic of IWR, more discussion (and perhaps standardization) is required for the most complex aspect of the IWR process; that is, how to decide whether a particular situation warrants the use of IWR.

Introduction

Perhaps the only aspect concerning the topic of in-water recompression (IWR; defined herein as any attempt to treat or relieve suspected symptoms of decompression illness [DCI] by returning an afflicted diver to the water – as distinguished from cases of "interrupted" or "omitted" decompression, where a diver returns to the water in order to complete omitted decompression prior to the onset of symptoms) that has escaped controversy, is the fact that the topic itself is highly controversial. This magnitude of dispute is not so surprising, considering that IWR involves a practice supported neither by conventional decompression theory nor clinical research data, which includes the placement of a person stricken with a very poorly-understood and potentially debilitating malady into a relatively hostile and uncontrolled environment. Only a few articles within academic publications include elaborated discussion of IWR, and most of those have originated in either Australia or Hawaii (1-6). A handful of other published articles (e.g., 7-10) include some brief discussion of IWR; but most references to IWR in primary and reference literature pertaining to general treatment of DCI has been limited to at best a paragraph or two (e.g., 11-13).

Over the past decade, an increasing number of non-commercial civilian divers have conducted dives involving alternative breathing mixtures (e.g., enriched air nitrox, pure oxygen, heliox, trimix), on profiles involving substantial decompression obligations. Collectively referred to as "technical"

diving, this expanding aspect of recreational diving has sparked the creation of several training agencies, annual international meetings, and the publication of several books, dedicated periodicals, and numerous popular and semi-popular articles. The divers engaged in these technical diving practices often find themselves at greater risk of incurring DCI due to the relatively extreme nature of their dive profiles and largely experimental decompression procedures. Therefore, many of these divers have gained an increased awareness of the need to be prepared to deal with the sudden onset of DCI (14). Consequently, this has led to an expanded interest in the topic of IWR among technical divers, resulting in presentations on the topic at annual meetings and a series of articles discussing IWR in the popular literature (15-18).

As pointed out by Pyle and Youngblood (19, 20), at the root of the controversy surrounding the practice of IWR is a basic conflict between theory and practice. The list of theoretical reasons why IWR has historically been discouraged is long, and includes the risks of additional nitrogen loading (when air or enriched air nitrox is breathed), risk of oxygen-induced convulsions (when pure oxygen is breathed), risk of drowning, insufficient supervision, risk to tending divers, thermal considerations, adverse environmental conditions (e.g., strong currents, rough seas), potentially adverse marine life, and reduced capacity for the afflicted diver and treatment supervisor to assess the nature of symptom progression during treatment. There are two theoretical considerations supporting IWR. First, there is the obvious advantage on the effect of *immediate* recompression on bubble growth; and second there is the advantage of increased inspired oxygen partial pressure (when pure oxygen is breathed), which could in some cases help counteract the effects of tissue hypoxia that may result from DCI-induced vascular obstruction. In stark contrast to the apparently overwhelming theoretical disadvantages of IWR, however, is the equally overwhelming apparent success rate among actual attempts at IWR (1, 4, 19, 20, this article).

The objectives of this article are threefold: first, to review the emerging practices of technical diving in the context of IWR; second, to examine existing published methods of IWR and propose a new method targeted at technical divers that represents a conglomeration of existing IWR methods with technical diving practices; and third, to discuss possible future directions that progress on refining IWR procedures might take.

Technical Diving Practices - Overview

In addition to being generally more at risk of experiencing DCI (due to more extreme dive profiles), technical divers are also perhaps among the most qualified and best suited for attempting IWR. This is a result primarily of generally increased awareness of diving physics and physiology (particularly DCI manifestations and oxygen toxicity issues), and perhaps more importantly, familiarization with factors related to IWR including breathing oxygen-rich mixtures underwater, staged decompression management, extended dive durations, and extensive thermal protection. Moreover, technical divers often conduct their operations with dedicated support personnel trained and prepared for dealing with unexpected emergencies.

Oxygen decompression

A common component of many technical diving operations is the practice of breathing pure oxygen for decompression at 20 ft (6 m) depth (21). This is among the most significant components of technical diving with respect to IWR, because all published IWR procedures involve breathing pure oxygen underwater. Because many technical divers routinely breathe pure oxygen for their normal decompression, they are not only trained for doing so (for the most part), but are also already equipped with appropriately cleaned and serviced cylinders and regulators (or even surface-supplied oxygen in some cases) for administering oxygen underwater. Moreover, there is a tendency for many technical dive operations to stock quantities of oxygen on-site that are considerably in excess of what is required for the planned dive, thereby reducing or eliminating the oft-cited criticism of IWR that sufficient quantities of oxygen are seldom available. One important disadvantage of routine in-water oxygen decompression by technical divers in the context of IWR is that, following long decompression dives involving in-water oxygen decompression, divers have been exposed to a much larger cumulative "dose" of oxygen, thereby possibly enhancing susceptibility to oxygen-induced convulsion should IWR be subsequently attempted.

Enriched air nitrox

The most ubiquitous of technical diving practices is the use of enriched-air nitrox (EAN). Although the most widespread use of this breathing mixture is for relatively shallow, non-decompression diving, various concoctions of EAN are almost universally an integral component of more extreme decompression diving, in the form of a decompression breathing mixture. Oftentimes a mixture containing 80% oxygen / 20% nitrogen (EAN-80) is used instead of pure oxygen for the final stages of decompression. Such a mixture might be worth considering as a breathing gas for IWR; the primary advantages being reduced potential for oxygen-induced convulsion and/or increased recompression depth , and the primary disadvantage being the presence of nitrogen causing a reduced off-gassing gradient across alveolar membranes (but still less nitrogen than breathing air at the surface). Other EAN mixtures available on-site in quantities during technical diving operations (e.g., EAN-50, EAN-40, EAN-36, etc.) might be useful as breathing gases during depth "spikes" associated with some methods of IWR. In any case, EAN can be considered a superior alternative to air as a breathing mixture for divers suffering DCI symptoms when no pure oxygen is available, regardless of whether IWR is attempted.

Helium

When conducting relatively deep (>165 ft / 50 m) diving operations, technical divers often employ helium among their breathing gas mixtures; either in the form of heliox (rare) or trimix (helium-nitrogen-oxygen mixtures; more common). This may have direct or indirect consequence on performing IWR in several different ways. For example, DCI symptom manifestation may be different following helium dives compared with air or other nitrogen-based dives (e.g., with regard to propensity towards neurological versus pain-only symptoms, or characterization of symptom onset), which may affect the relative importance of the immediacy of recompression. Also, there may be possible effects on the cost/benefit considerations of including a deep "spike" during IWR following a deep dive involving helium (i.e., the disadvantages of a "spike" while breathing EAN or air may be reduced if the primary constituent of excess dissolved gas in the diver's body is helium).

Furthermore, there is some indication that treatment using helium-based breathing mixtures has advantages over nitrogen-based breathing mixtures (22), although the role of this with respect to IWR is unclear.

Rebreathers

Although still relatively uncommon within the civilian diving community, closed circuit rebreathers are gaining broader popularity, especially among technical divers (23-27). As these devices, which offer greatly increased gas efficiency, become more and more prevalent, their role in the practice of IWR may expand. The primary disadvantage shared by all rebreathers is increased complexity and expanded range of failure modes (e.g., flooding the CO_2 absorbent canister), and hence greater need for specific training.

Oxygen rebreathers. The most basic kind of closed-circuit rebreather is the oxygen rebreather. Widely used in military operations around the world, these devices deliver pure oxygen to a diver via a closed-circuit breathing loop that includes a canister containing chemical CO_2 absorbent material. Oxygen utilization is based on diver metabolism, and very little gas is wasted. Hence, a very small supply of oxygen can sustain a diver for long periods of time. In some ways, pure oxygen rebreathers represent an ideal tool for use in the Australian method of IWR, because they greatly extend the duration a diver can remain underwater with a limited supply of oxygen. However, like all rebreathers, they can be dangerous in the hands of untrained users, and thus would only be appropriate as an IWR tool for divers already trained in their use. Furthermore, because of the restrictive depth limits of breathing pure oxygen underwater, oxygen rebreathers remain a useful tool primarily for military operations, and are uncommon among recreational technical divers.

Semi-closed rebreathers. Semi-closed rebreathers are so-named because not all of the breathing gas is recycled. An oxygen-rich supply gas is added to the breathing loop at a constant or variable rate (depending on the specific type of unit), and excess gas is vented from the loop. Far and away the most common of rebreather types among recreational divers, semi-closed rebreathers may be of use in the practice of IWR for their ability to greatly extent the functional use of a given supply of oxygen (although not as much as with pure oxygen rebreathers). Although generally designed to utilize an EAN supply gas mixture, substitution of pure oxygen as the supply gas would allow extended durations with a limited quantity of oxygen. However, although the use of oxygen as a supply gas would eliminate the concern of hypoxia typically inherent to semi-closed rebreathers, other complexities common to all kinds of rebreathers (e.g., risk of flooding CO_2 absorbent, specialized training) still apply.

Fully-closed mixed-gas rebreathers. The most sophisticated (but somewhat less common) kind of rebreathers within the technical diving community are fully-closed mixed-gas rebreathers. Offering the 'best of both worlds' (i.e., maximal oxygen utilization efficiency of pure oxygen rebreathers combined with extended depth capabilities of semi-closed rebreathers), these kinds of rebreathers possibly represent the ideal tool for conducting the Hawaiian method of IWR (or any other method involving a depth 'spike'). In generally, fully-closed mixed-gas rebreathers incorporate electronic control systems, which maintain a constant partial pressure of oxygen within the breathing loop. This means that the units can be set to provide 100% oxygen in shallow water, and add only enough 'diluent' gas (e.g., nitrogen or helium) to maintained the desired oxygen partial pressure. Hence, the

non-oxygen component of the breathing mixture is held to an absolute minimum at all depths. Alas, as with the other kinds of rebreathers, a great deal of specialized training is required for proper use of these devices; so-much so that they would be useful as a tool for IWR only to those individuals already properly trained in their operation. Nevertheless, the sorts of civilian divers who become trained for and use fully-closed mixed-gas rebreathers often have done so in order to dive to relatively great depths, or dive in very remote locations (where gas supplies are limiting), and thus may find themselves in a situation to conduct IWR.

Other technical equipment

All published methods of IWR prescribe the use of full-face masks (FFMs) in order to safeguard against the consequences of suffering from oxygen-induced convulsions underwater. To divers unfamiliar in the use of full face masks (of which there are many designs), FFMs may represent an additional hazard or source of stress in an already stressful situation (i.e., a situation in which the need for IWR is warranted). However, many members of the technical diving community have embraced the use of FFM's for diving, often to allow use of electronic through-water communication systems (another kind of technical dive equipment that may be of great value in an IWR situation); and hence are more prepared to use this kind of equipment. Yet another aspect of technical diving equipment of relevance to IWR is that of thermal protection. The risk of hypothermia in a diver engaged in IWR is often cited as a reason why IWR should not be attempted. Technical divers, however, are generally prepared for long-duration dives, including extended decompression times. Consequently, these divers tend to be familiar wit proper thermal protection equipment and practices (including drysuits and associated thermal underwear). However, even the best of thermal protection cannot necessarily be relied upon to keep the diver adequately warm in extremely cold situations (6).

Logistical support

Over and above the value of typical technical diving equipment and practices in the context of IWR, technical dives tend to be conducted with far more controlled and disciplined logistical support than most average recreational dives. Moreover, support personnel are often specifically trained and prepared for dealing with unexpected emergency situations, and therefore would likely be capable of managing an IWR effort.

Technical IWR Methodology

Existing methods

At least three formal methods of IWR have been published: the so-called "Australian Method", which is used by abalone divers in Australia (1); the U.S. Navy method (29); and the so-called "Hawaiian Method" (4), which is used by diving fishermen in Hawaii. The Australian method involves continuous breathing of pure oxygen at a depth of 30 ft (9 m) for a period of time ranging from 30 to 90 minutes, depending on severity of symptoms. Ascent is conducted while continuing to breathe oxygen at a slow and steady 1 ft/4 min (1 m/12 min). Upon surfacing, oxygen is breathed for 1-hour periods interspersed with 1-hour periods of breathing air for the following 12 hours. The

U.S. Navy method is similar, but the ascent is conducted as two 60-minute staged stops at 20 ft (6 m) and 10 ft (3 m), followed by continuous oxygen breathing for 3 hours after surfacing. The Hawaiian method is similar to the Australian method, but differs primarily in prescribing a depth "spike", descending while breathing air to a depth 30 ft (9 m) deeper than the depth at which symptoms resolve for 10 minutes, then returning to 30 ft (9 m) to commence breathing oxygen for an extended period of time.

Of the three methods, the Australian is most often cited, followed by the Hawaiian. The U.S. Navy method is seldom referenced for civilian use. Most authors who discuss IWR recommend the Australian method instead of the Hawaiian method, usually citing the risk of additional nitrogen loading during the air spike of the Hawaiian method as being too great to warrant the perceived benefit of increased ambient pressure exposure. Indeed, even among authors who discuss IWR, the vast majority condemn the practice of using air as a breathing mixture. The source of this condemnation appears to stem from the commonly-held believe among hyperbaric specialists that breathing air during IWR attempts tends to worsen symptoms more often than it improves them (11, 30). However, the empirical foundation of this widespread believe has been called into question (31). Published survey data of diving fishermen in Hawaii (4) indicate an apparently very high rate of success (in terms of symptom elimination or improvement) when using air as a breathing mixture for IWR (Figure 1).

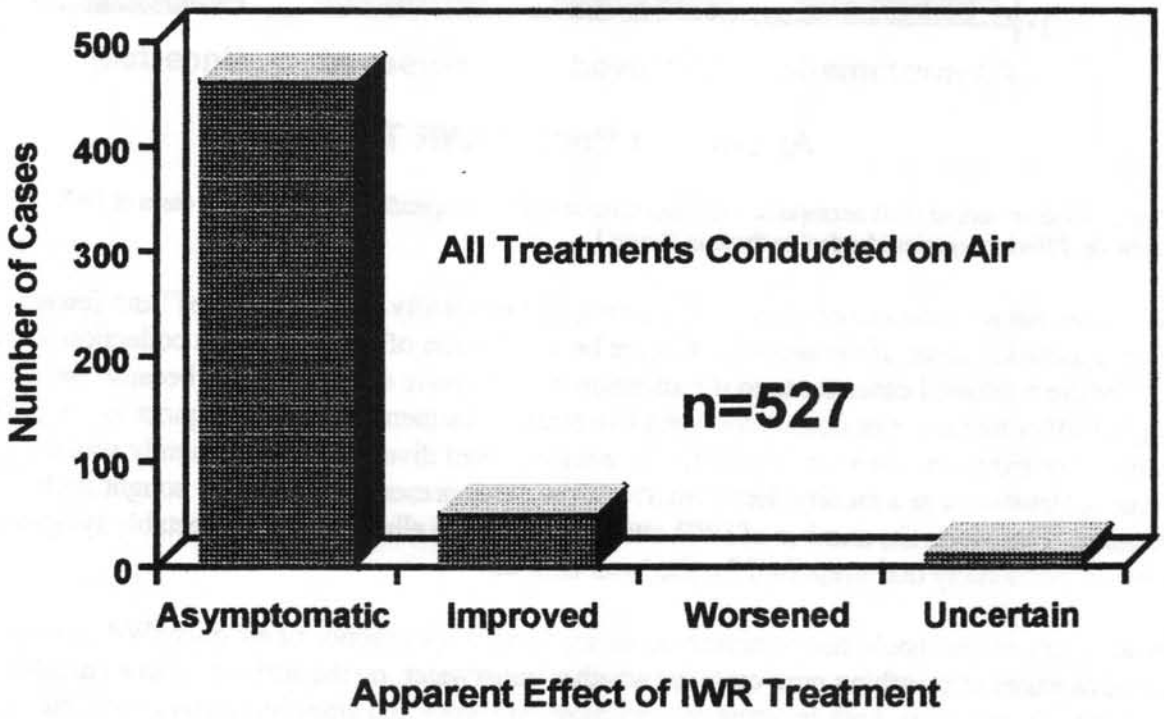

Figure 1. Success rates of IWR attempts among diving fishermen in Hawaii (4). "Asymptomatic" indicates diver felt no apparent residual DCI symptoms following IWR attempt; "Improved" indicates clear reduction of symptom severity to the point where subsequent treatment in a chamber was not sought; "Worsened" indicates exacerbation of DCI symptoms; and "Uncertain" indicates ambiguous outcome.

Because these survey data were obtained retroactively, and relied entirely on the recollection of the divers involved, these findings have been called into question. However, a similar survey of IWR cases for which detailed accounts have been published and cases with specific records of events (almost all of which involve air as the only breathing gas) reveals a similar trend (Figure 2).

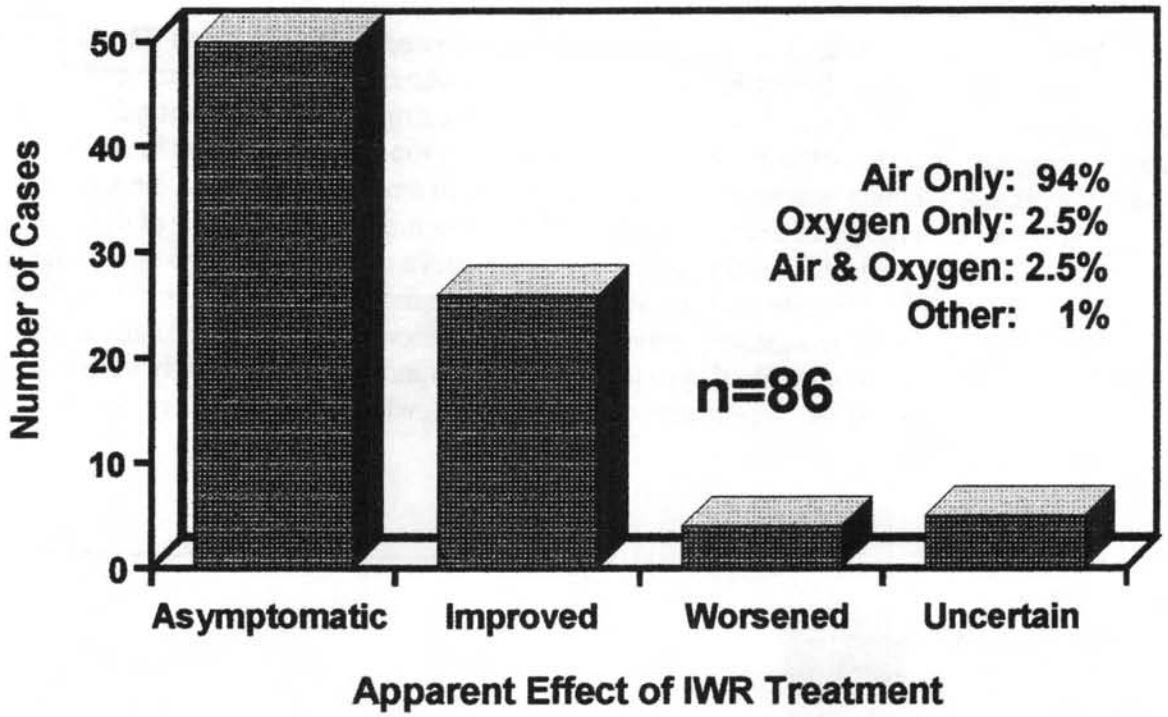

Figure 2. Success rates of IWR attempts among published or otherwise specifically documented cases of IWR, worldwide. Effect categories identical to those in Figure 1.

The somewhat less pronounced results indicating proportionally more "improved" and fewer "Asymptomatic" cases of the second set might be a reflection of bias in the data collection source. Most of the published cases came to the attention of hyperbaric specialists only because the diver sought further treatment or consultation at a hyperbaric treatment facility subsequent to the IWR attempt. For example, whereas 16% of these cases involved divers who subsequently sought additional treatment at a facility, less than 3% of the cases presented in Figure 1 sought such treatment. Therefore, the number of IWR attempts involving elimination of detectable symptoms is likely proportionally underreported for the latter data set.

These observations should not be construed as support for the practice of air-only IWR attempts. The advantages of breathing pure oxygen (whether underwater, on the surface, or in a chamber) are clear and unambiguous, both in terms of physiological theory and empirical observation. However, at the very least the data presented in the preceding figures challenge the notion that "If a victim has mild signs and symptoms of decompression sickness, the usual result [of an IWR attempt] is a much more seriously injured diver. If the initial symptoms are serious, the result is usually disastrous." (11).

So, if not founded in empirical experience, what is the source of general objection to the air "spike" of the Hawaiian IWR method? The most often-cited risk is that of additional nitrogen absorption. This seemingly indisputable contention is rooted in the conventional wisdom of decompression theory that DCI and its manifestations can (for the most part) be accurately modeled with hypothetical compartments representing levels of dissolved gas tensions throughout the body. As any hyperbaric specialist will admit, however, such models do not account for the entire DCI story. Modern approaches to DCI management acknowledge the roles played by other factors, primarily among them the physics governing gas-phase bubbles within aqueous solutions, and the biochemical (particularly immunological) responses of the body to the presence of disruptive intravascular. Thus, with decompression theory still relatively in need of further elaboration at fundamental levels, rejection of the "spike" on purely theoretical grounds seems unwarranted, and consideration of this practice is perhaps suggested by empirical experience.

Another aspect of published IWR methodology in need of scrutiny is the extent to which treated divers are exposed to elevated partial pressures of oxygen. Breathing pure oxygen at a depth of 9 m results in an inspired oxygen partial pressure of nearly 2 atm/bar. While this level is routine for dry hyperbaric chambers, it is somewhat excessive within the context of "safe" limits adopted by technical divers (1.6 atm/bar maximum; 1.4 atm/bar operational). To mitigate the effects of an oxygen-induced convulsion underwater, all published methods of IWR mandate the use of a full face mask by the afflicted diver. While it is certainly true that FFMs drastically reduce the probability of drowning during a convulsion, it is also true that their availability on-site (even during technical diving operations) is generally lacking, and the effect on untrained users may be amplification of stress levels. The fact of the matter is, IWR will be (and indeed already has been) conducted using oxygen underwater at the stated depth of 30 ft (9 m), without the benefit of FFM equipment.

Proposed method of IWR for technical divers

In response to these considerations, as well as personal observations of actual IWR efforts, I have developed my own method of IWR for use during technical diving operations in geographically remote localities. The specific methodology is summarized in the Appendix to this article.

This method differs from other published IWR methods in several respects. First of all, it includes a 10-minute period breathing 100% oxygen at the surface prior to re-entry into the water. This period allows for assessment of conditions as to whether IWR is appropriate, and provides a brief test to indicate whether surface oxygen alone will be sufficient to resolve symptoms. If IWR is to be performed, the diver descends to a depth of 25 ft (7.5 m) breathing 100% oxygen. This is shallower than the 30 ft (9 m) depth recommended by other IWR methods, with the intent of reducing the maximum inspired oxygen partial pressure from 1.9 atm/bar to just over 1.7 atm/bar. The advantage of this is reduced probability of oxygen-induced convulsion (especially important when a full face mask is not available), and the disadvantage is a reduction in ambient pressure. Because in many cases symptoms are relieved at depths of only 10 ft (3 m), the 25-ft (7.5-m) oxygen depth seems a more reasonable compromise between costs and benefits of recompression versus oxygen toxicity.

Like the Hawaiian Method of IWR, this method includes an optional deep spike while breathing air or (preferably) EAN. However, unlike the Hawaiian method, the spike is not conducted until after

10 minutes of breathing oxygen at 25 ft (7.5 m); and then, only if symptoms have not been substantially reduced. The purpose of delaying the spike is to allow time to assess the need for it. If 10 minutes of breathing oxygen at 25 ft (7.5 m) is sufficient to resolve symptoms, then the potential risks of a deeper spike might best be avoided altogether. On the other hand, if symptoms persist after 10 minutes at 25 ft (7.5 m), then the need for additional compression seems indicated and a spike is performed. The Hawaiian Method prescribes descending to a depth 30 ft (9 m) greater than the depth at which symptoms resolve. One potential problem with this approach is that symptom resolution may not be instantaneous, and therefore excessive depth may be achieved before making the decision to cease descent. The proposed new method breaks the spike up into 25-ft (7.5 m) increments, with two-minute assessment periods at each increment. If symptoms resolve after 2 minutes at a given spike depth increment, depth is no longer increased, and the remaining 8 minutes of the 10-minute spike duration are conducted at the current depth. If symptoms persist even after 2 minutes at a depth of 125 ft (38 m), spike depth is no longer increased, and the remaining 8 minutes of the spike duration are performed at 125 ft (38 m). After a total of 10 minutes at the maximum spike depth, the diver returns to 25 ft (7.5 m) following a slow ascent rate, and returns to breathing 100% oxygen. The primary reason for the divergence in specific spike methodology from what is described in the Hawaiian method is to reduce the probability of excessive spike depth. Also, it should be emphasized that a spike should not be performed if insufficient quantities of oxygen are not available to follow the spike up with an extended period breathing oxygen at 25 ft (7.5 m).

Regardless of whether a spike is performed, the period of breathing 100% oxygen underwater further differs from previous methods with the addition of 5-minute air or EAN breaks every 20 minutes. The technical diving community has borrowed this practice from hyperbaric treatment facilities in an effort to reduce probability of oxygen-induced convulsions. Presumably, the breaks were not included in previous IWR methods due to fear of additional nitrogen loading. However, given the apparent wide-spread success of air-only IWR, along with the fact that the concern for additional nitrogen loading does not seem to offset the value of the non-oxygen breaks during treatments in a chamber, the air breaks seem justified for IWR; and may be even more justified in view of the greatly increased dangers of suffering an oxygen-induced convulsion underwater.

At best, the new method of IWR described herein is a gross over-simplification of an optimal approach to treating DCI victims underwater. The fundamental problem with any standardized method of IWR is the difficulty of accounting for the wide variety of variables that can impact the decision to perform IWR. Even if all the factors could be taken into account, in many cases it is far from clear how those factors should affect IWR methodology. For example, are serious neurological symptoms more indicative of a need to perform IWR (to thwart permanent neurological damage before hypoxia leads to cell death); or are they more indicative of a need to *not* perform IWR (due to excessive risks of drowning, etc.)? This is only one of many factors that probably should affect the decision to perform IWR, but the way in which they should affect the decision is not clearly understood. Finally, the proposed new method is *not* intended as a replacement for any existing IWR method, but rather as an alternative to be considered by trained technical divers in appropriate circumstances.

Future Directions

The most important step in resolving the IWR controversy (i.e., the need to discuss the related issues from an open and objective perspective) has already been taken in the form of this workshop. The task at hand can be roughly summarized in the chart presented in Figure 3. The first question to decide is whether IWR should be attempted in any circumstances whatsoever. If not, then we can all go home – the controversy has been resolved. Following the assumption that the answer is not so simple, the next question involves whether IWR should be performed in all circumstances where DCI symptoms are presented. If so, there only remains the question of specific methodology. Again, assuming the answer is not so simple, the topic in need of most attention is the elucidation of circumstances in which IWR should, or should not be performed.

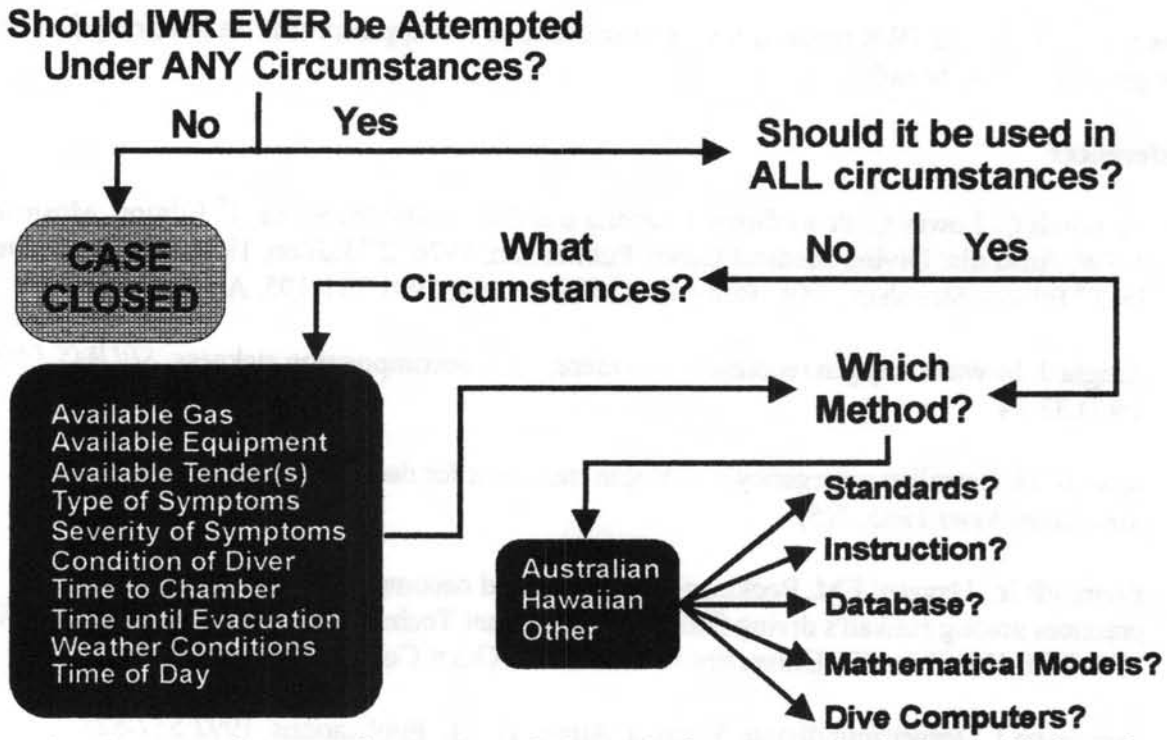

Figure 3. Flow chart representing questions of IWR that need to be resolved.

The IWR decision process will always be a complex one. The first step is to decide what the relevant factors are. Some of the more obvious ones are listed in Figure 3, but there are undoubtedly many others. The next step is to determine what role each factor should play in the decision making process. Unfortunately, neither theoretical nor empirical approaches to resolving these factors will provide the single best answer.

Once a clearer understanding of the associated factors and their roles in IWR has been gained, the next step is a resolution on IWR methodology. Is there one optimal method for all circumstances, or should several specific methods be defined, with each applied in specific circumstances? Or, should one dynamic method be devised, which changes according to the status of various specific factors on a case-by-case basis? To make progress towards some answers, empirical and theoretical approaches must be taken in the context of effective emergency management techniques.

Finally, what are the ultimate future directions to take with regard to IWR? Published standards would represent a very important step in the right direction, but would not end the issue entirely. Given the complex nature of IWR procedures and victim condition evaluation, perhaps a training course in the practice and administration of IWR could be developed and certifications offered by appropriate diving agencies. Perhaps the most important step (and one that should be undertaken sooner rather than later) is the establishment of a centralized database documenting IWR cases. In the very long-term future, if patterns emerge with enough consistency, mathematical models of IWR could be developed and possibly even incorporated into dive computers.

The road to resolving IWR issues is a very long one, and it is apparent that we have only just begun our progress along its path.

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Appendix – Pyle IWR Method.

Required Equipment

1. An adequate supply of oxygen that can be delivered to a diver underwater, either in the form of an appropriately serviced scuba cylinder, surface-supplied apparatus, or rebreather device (the latter for appropriately trained divers *only*!)
2. An adequate supply of air, EAN, or other diluted oxygen mixture that can be delivered to a diver underwater, either in the form of an appropriately serviced scuba cylinder, surface-supplied apparatus, or rebreather device (the latter for appropriately trained divers *only*!)
3. Weighted descent or decompression line marked at 10-ft (3-m) intervals, extending to a depth of 130 ft (40 m) or the maximum available depth, whichever is shallower.
4. Some means of communicating basic information between the diver and the surface support.

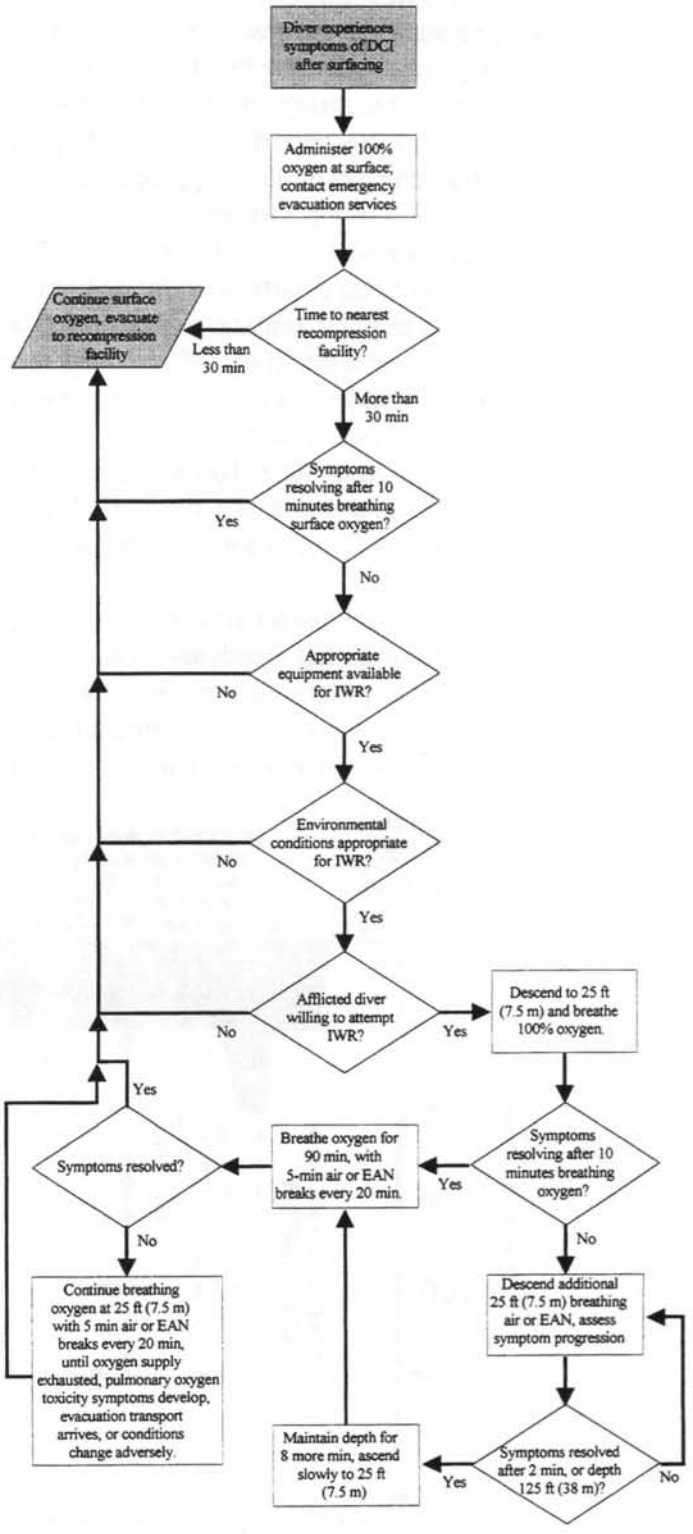
Recommended Equipment

1. A full face mask or diving helmet to be worn by the afflicted diver.
2. Means to physically attach afflicted diver to decompression line.

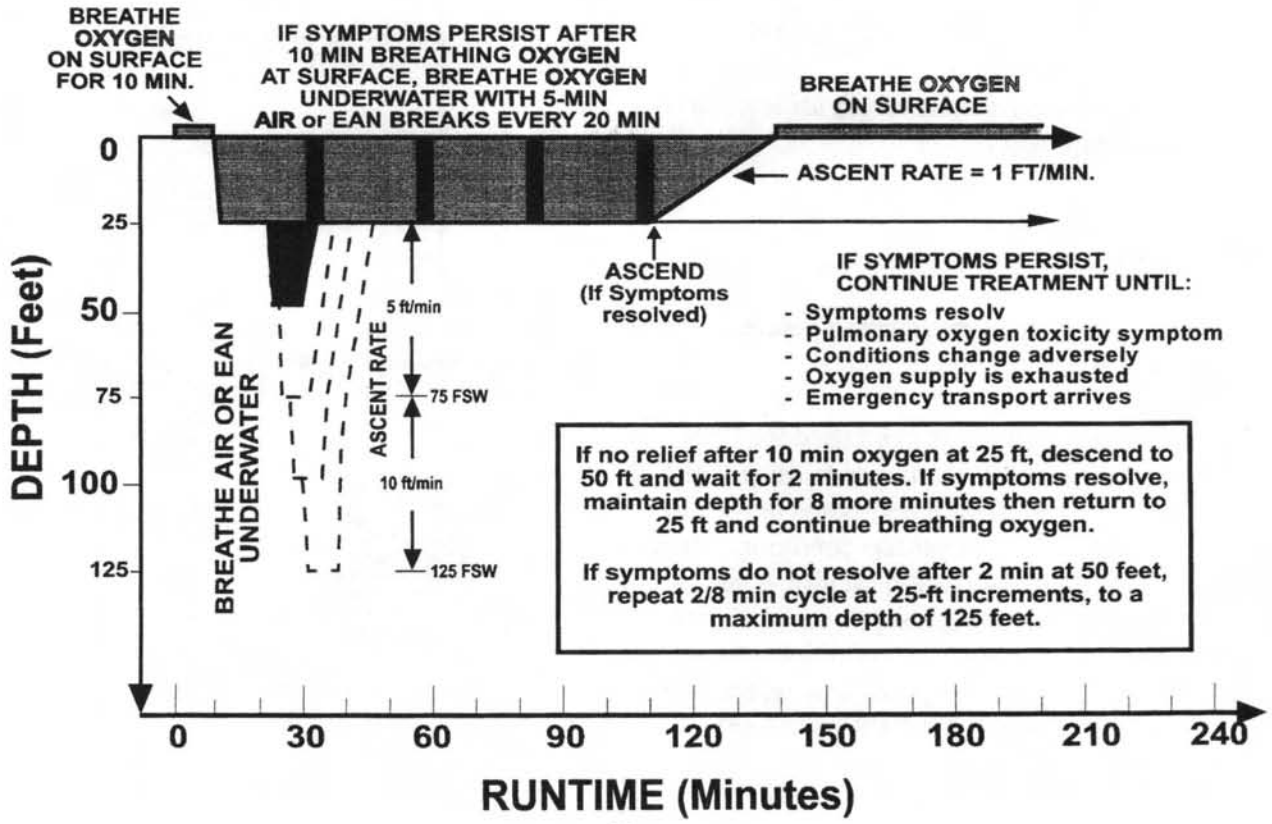
Method

Immediately upon recognizing potential symptoms of DCI:

1. Prepare equipment to be used for IWR.
2. Administer 100% oxygen to diver while at surface for 10 minutes, assess the progression of symptoms, and evaluate conditions (time to nearest recompression facility, diver disposition, oxygen supply, availability of tender diver, weather conditions, time of day, etc.), contact emergency evacuation services, and decide whether IWR is warranted.



3. If IWR is warranted and symptoms are not resolving within 10 minutes of commencement of surface oxygen, place afflicted diver at a depth of 25 ft (7.5 m) on weighted decompression line, breathing 100% oxygen for 10 minutes, under close observation of a tender diver who can maintain communication with surface support.
4. If symptoms are resolving after 10 minutes of breathing 100% oxygen at 25 ft (7.5 m), maintain depth and continue breathing oxygen for a period of 90 minutes, interspersed with 5-minute periods breathing air or EAN every 20 minutes.
5. If symptoms persist or continue to progress after the initial 10 minutes at 25 ft (7.5 m), change breathing gas to air or appropriate EAN, descend to a depth of 50 ft (15 m) and assess symptom progression for 2 minutes. If symptoms are resolving, maintain depth for 8 additional minutes, then ascend at a rate of 5 ft/min (1.5 m/min) to 25 ft (7.5 m) and perform step 4.
6. If symptoms persist or continue to progress after 2 minutes at 50 feet, descend to 75 feet and repeat step 5. Continue to repeat step 5 at 25-ft (7.5-m) depth increments until symptoms resolve, or a depth of 125 ft (38 m) is reached. After 10 minutes at maximum "spike" depth return to a depth of 25 ft (7.5 m) at a rate of 10 ft/min (3 m/min) below 75 ft (22.5 m), and 5 ft/min (1.5 m/min) above 75 ft (22.5 m), and perform step 4.
7. After 90 minutes of 100% oxygen with air or EAN breaks, if symptoms have resolved, ascend to surface at a rate of 1 ft/min (0.3 m/min) and continue breathing oxygen at surface until emergency evacuation transport arrives, diver suffers pulmonary oxygen toxicity symptoms, or 3 hours.
8. If symptoms persist or continue to progress after 90 minutes of 100% oxygen with air or EAN breaks, maintain depth and continue 20-min oxygen / 5 min air or EAN cycle until oxygen supply is exhausted, emergency evacuation transport arrives, diver suffers pulmonary oxygen toxicity symptoms, environmental or diver conditions change adversely, or symptoms resolve, then ascend at a rate of 1 ft/min (0.3 m/min).



DISCUSSION

Dr. Ladsen:. I want to compliment you on a very well-thought-out presentation. You did a very good job of organizing all the information that we all intuitively know but to see it put out in flow charts and everything is a very good thing.

On your recompression profile, I have one comment. I assume that you're recommending that a tender go down or a buddy go down with the diver on his profile?

Dr. Pyle: Absolutely.

Dr. Ladsen: In a situation where they've been doing repetitive dives, which I assume it would be or at least one long dive with a very short surface interval, you get down to a 125 feet for 10 minutes. You're blowing the table for the tender, and you're obligating him to stay in the water and not be able to go back up and call for more help. That's a caveat that I think needs to be thought out before any recommendations are made.

Dr. Pyle: Right. I agree with that, and I'll explain. My real method is much more complicated than I'm able to put on these visual aids. Generally in my and most technical diving operations, you have trained standby divers on the boat who haven't had historical exposure.... they haven't had a dive profile that day already. They're clean essentially.

Also, in these situations I would generally have given that diver access to enriched air nitrox which would give them a little more time to be as a tender diver following the patient down at 125 feet. That's another reason I drew the line at a 125 feet for decompression issues associated with the tender, and I made it sound over-simplified. The only thing I take into consideration when going deeper is whether symptoms resolve or not. Well, sometimes I'll stop the spike regardless. I don't do this every day. I don't get bent every day.

This is mostly my conceptualization of how I would deal with this situation, and I imagine that if there were a concern that a tender diver would be exposed to undue risk of decompression, I would stop the spike at shallower depth or it would be a more complicated procedure, but I completely agree with your point.

Unidentified: One other comment. If you're going to go to this much trouble to have standby divers and all this extra equipment, obviously you've made some pretty heavy equipment investments with the systems and stuff like that means you're anticipating a possible need for in-water recompression. Why don't you just go the extra mile and spend a few extra thousand dollars on a portable recompression chamber and not have to worry about it?

Dr. Pyle: Two answers to that question. The first is that this sort of logistical support is not uncommon among routine, technical-type divers, and so the question then becomes, should these routine technical-type divers routinely invest the amount of money to have a portable chamber on site? That leads me to the second answer to the question, which is, I probably shouldn't say this publicly, but if I were given a choice between one of these portable stretchers and getting back in the water, there would be no decision-making process at all. I would be back in the water.

Things that concern me about portable chambers are explosive decompression. I know maybe that's a bogey-man, maybe that's not really an issue, but for me, I'm more comfortable in the water. I feel that even if you can set up the chamber in 10 minutes, the water's only 2 minutes away. Maybe you could get in the water while they're setting up the chamber.

There are contingency plans. I have seriously looked into these portable chambers. I would recommend it to most serious technical diving operations. A lot of serious technical diver operations are looking into it, but for me personally, I'm actually very restrained in this talk, in the form of endorsing in-water recompression because, for me, it would be the knee-jerk response to almost any situation because it's immediate and because I wouldn't be here talking to you if it wasn't for that practice.

So, that's where my methodology came from, but you're absolutely right. I think that avenue needs to be explored more.

Dr. Edmonds: I agree with everything you said, and I was most impressed with the presentation. It really has filled in a big void in my understanding of this whole thing.

The two questions that I had in fact have just been asked. You've answered them very well, and the only argument I would have with you is please never put it on a computer.

Dr. Pyle: I just wanted to be the first one to suggest it, that's all.

Dr. Vann: Nice presentation, Richard. I might suggest that you want to add a slide that summarizes your experience. Could you give us just a couple words on that now?

Dr. Pyle: The article that I wrote along with David Youngblood, that was published in about four different places now, and most recently in the *SPUMS Journal*, and in fact, the *SPUMS* version of that article is the most unabridged. It's the most complete of all of those versions that were published and includes more than half of the cases that I've personally been involved with as cases histories, and it's described in fairly good detail.

If you want me to spend a few minutes just going over a few example cases of mine, one that I was talking with the folks I was having lunch with was when I got seriously

bent. When I was 19 and immortal. I did some stupid dives. I got a serious case of decompression sickness. I won't go into the details of how I got in that state, but basically I was having rapidly progressing quadriplegia. I was losing my legs and my arms. I was the only one on the boat. My dive buddy was still underwater. I was on the boat for about 10 minutes before I could hear my buddy's bubbles. I was lying down on the boat by this time, hear my buddy's bubbles breaking the surface near the boat.

I hobbled over to the side of the boat as best as my legs and arms would allow me to do, tapped him on the shoulder, buddy breathed with him for all of 7 minutes at 10 feet on air only and emerged, I could not detect any symptoms at all. My arms were completely recovered. My legs were completely recovered.

These symptoms were highly progressive up until the point that I went back in the water. They were arrested the moment I went into the water, and they remained undetectable by me anyway for about 20 minutes after exiting the water. That was long enough for me to drive the boat back to a dive shop, get some more tanks, get in the water, alert them, have them get the recompression chamber operable.

I spent the next 4 hours breathing air at about 20 feet. By that time, before I'd entered the water for that second attempted in-water recompression, my symptoms had progressed during that 20-minute interval that I was driving back to the dive shop to the point where my legs weren't quite right. My arms were okay, but my legs were not coordinated, and I was falling down on the deck of the boat.

Four hours of breathing air at 20 feet later, the symptoms were essentially identical when I left the water. They weren't any better. They weren't any worse. However, they were rapidly progressing up to the point. So, I think again I ceased the progression of the symptoms long enough for them to get a chamber ready, and then the story gets long after that, but from there on out, it was decompression chambers. That was my hard-core first experience with it.

Another case which I thought was interesting was where my dive partner—I was actually the bad boy that day. This is again back in the bad old days. He was doing his second dive to 200 feet that day. During the decompression from that dive, half of his body went limp at the 10-foot stop. He descended back to 70 feet, again breathing air.

I aborted my decompression about 20 minutes early, maybe not quite that much, but 15 minutes early, to give him my air supply, to go up to the boat and get another air supply for him. I turned out to be fine. He turned out to be fine after about 2½ hours. He was asymptomatic as far as he could tell that night and the next morning, but he went to the chamber the next day anyway just to verify and have himself checked out. That was another noteworthy case.

Another noteworthy case involved me. I'll try to keep these brief. It's also recorded in my paper with Youngblood, but I had a problem where I got detained at 200 feet unexpectedly, used up most of my air that I had planned on using for decompression and I had to omit 70-foot decompression ceiling to get back to the boat or face drowning. I explained to my diving partner, who was on the boat, that I needed another tank of air very quickly.

This would have been a case of interrupted decompression, except that while I was waiting for him to get a tank ready, I started getting very lightheaded. My legs started getting numb. My arms started getting numb. He passed me the tank. I put it under my arm. I held the regulator in my mouth and just allowed myself to sink.

When I reached the depth of about 50 feet, my head cleared up. My arms regained their strength. I followed the advice of my decompression computer, which at that time was flashing and buzzing and doing all kinds of nasty things. I probably shouldn't be telling this. This is again in the bad old days.

But I followed the advice of my decompression computer, stayed about 10 feet below it, up until I had about 20 minutes at 10 feet left according to the computer. By that time, I knew my partner would be very, very angry with me because he was meeting his girlfriend later that day.

So, I elected to abort the remainder of what my computer recommended as decompression time. So, basically I broke decompression anyway, in addition to the fact that I already had decompression illness symptoms and had absolutely no occurrence of symptoms thereafter, even though I truncated what my computer said I should be using for decompression.

Unidentified: What were the total numbers of cases you've been involved in?

Dr. Pyle: I think there were four, and then I think there were about an additional six, depending on whether the guy was really experiencing symptoms and whether it was really omitted decompression.

Most of them involved omitted decompression. In other words, where we were anticipating bad things to happen, and we're getting ready before the symptoms ever came on, by that time the person who was at risk was getting pretty anxious about this and imagining things or might not be imagining things. In some cases, it's pretty clear they're experiencing symptoms.

Depending on how rigorously you define in-water recompression, about 10 cases. Does that answer what you were after?

Dr. Sanchez: What happened if you're in your 125 spike? Your symptoms clear if you stayed your extra 8 minutes, you start ascending, with recurrence of symptoms at 100 feet and then you go down again?

Dr. Pyle: I would never go down again. I might pause for a brief time. That's one of those fuzzy logic things that I probably have to make up in the current situation.

How much oxygen do I have waiting for me to complete even longer decompression? How much thermal protection I have? Am I going to be able to endure an exceptionally long exposure at 25 feet to make up for the additional spike depth?

Chances are that in the majority of cases, I would go straight back to the 25-foot stop, as per the method regardless of the symptom progression. Go back to pure oxygen and just hope in the ensuing hour that the symptoms peak and regress back down again.

If they continue to get progressive, that would be a really nasty situation to be in because going to the surface means you're probably not going to get any better. Staying underwater means you're probably going to continue to get worse. I hope I'm never in that situation.

Dr. Sanchez: The reason I asked is because that's a nasty story of table 6A on air. Once you started coming out from 165 and then you have recurrence of symptoms.

Dr. Pyle: What do you do?

Dr. Sanchez: You start worrying a lot. You have very few options. My problem with your schedule. I'd rather see a shorter oxygen breathing period than going on the spike to depth because then you're getting yourself into a position in which you have very little options and very limited air supply.

Dr. Pyle: What is the experience in a chamber on table 6A treatments with the frequency of that situation arising? Is it common? Is it rare?

Dr. Sanchez: Well, let me tell you, if you're going to have problems, you're going to have problems in that coming from 165 to 60. You don't want to have those problems there, and if you're in the water, you really don't want to have those problems.

Dr. Pyle: Right. In every case that I've been involved with, except one, I never would have gotten to the spike phase because symptoms resolved within reaching, like I say, 10 feet of getting in the water, and that's breathing air.

There's a great case that happened in Australia recently of a diver who did 22 minutes at 220 feet or some pretty extreme exposure. She had a buoyancy compensator failure and blew to the surface quickly.

I think her first required decompression stuff, I have it written down at home, was something like 70 or 80 feet. I mean some serious decompression. Rather than go back down and continue the expected decompression obligation, the person elected instead to perform a modified Australian in-water recompression and returned to only a depth of 20 feet.

Now, bear in mind, this is a person who just skyrocketed to the surface with a 70-foot ceiling, returned to 20 feet breathing pure oxygen, and had mild neurological symptoms upon finally reaching the chamber X hours later. You don't really necessarily get bent. Just returning to 20 feet when you're supposed to be at 70 feet, at least in that case and a couple other cases I'm familiar with, seems to solve the problem.

So, the spike issue, I could be persuaded to eliminate the spike completely. And bear in mind, in my situation, I've got a fully closed rebreather which means that my oxygen partial pressure is maintained at whatever I darn well want it to be, and this means my nitrogen partial pressure is kept to a minimum.

So, I'm basically never breathing air. So, as I'm doing that ascent, my oxygen is getting richer and richer as I'm coming up. So with the equipment I have available, I think I might be in a slightly more advantageous situation to deal with these spikes but maybe not. I'm here to learn.

Dr. Arnold: My question would be you recommended the use of full face mask, but also you entertained using air breaks. How do you technically do that without a manifold?

Dr. Pyle: Well, for me, it's easy. My rebreather has a manifold on it. When I plug my rebreather into a full face mask, I can be breathing off the rebreather and switch a valve and go to open circuit and breathe air and switch back again. I don't have to take anything off my mask.

But that is a very good point. How do you take an air break? My solution to that before having the rebreather is a Hervy Morgan full face mask hood that had a manifold built into it. I carry a pony bottle of air strapped to my side and a surface supply of oxygen with a non-return valve. That was just a matter of flipping a valve to get my air break, but that's included in my more global complex issue of having the right equipment on hand to do it.

In Water Recompression: A Viewpoint

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In water recompression (IWR) of divers with decompression illness offers the potential of providing treatment when a chamber is not available. Furthermore, if bubble volume can be reduced quickly after on the onset of symptoms, it is possible that their associated damaging secondary effects can be attenuated.

During IWR, adequate thermal protection is essential. Head out immersion increases the rate at which inert gas is eliminated, and the rate accelerates as the the water temperature is increased (1). The greater tissue blood flow and tissue inert gas washout rate that accompanies immersion (2) might conceivably offer some advantage over recompression to an equivalent pressure in a dry chamber, at least in that one respect.

Indeed, in-water recompression while breathing oxygen has been used in remote areas of Australia. Dr. Carl Edmonds has provided evidence obtained from Australian pearl and abalone divers that, when used promptly under carefully controlled conditions (maximum depth of 30 fsw (9 meters), oxygen administered via tethered line and full face mask, thermal protection suit, buddy present, and ascent at 12 min/meter), in-water compression can be both safe and effective (3), with no reports of oxygen convulsions. Similar positive experience has been reported in Hawaiian fishermen using IWR breathing air (4). However, the efficacy of treatment is based entirely upon subjective relief as reported by the diver, retrospectively in the Hawaiian fishermen (4). It was dissatisfaction with air recompression tables that impelled the US Navy in the 1960's to adopt oxygen tables. It is therefore unlikely that IWR breathing air is particularly efficacious.

There are no reported cases of in-water recompression in which the response to treatment has been corroborated by a physician. Therefore it is difficult to be certain of the effectiveness of the technique breathing either air or O_2.

Moreover, breathing oxygen underwater has risks. Donald (5) has provided evidence that the risk of a hyperoxic convulsion is greater when immersed. Communication with the diver is less than ideal, and it is difficult to assess the clinical status of a diver underwater. The notion that immersion accelerates inert gas washout in bubble-damaged tissues has not been confirmed in either animal or human studies. Diuresis associated with a period of prolonged immersion may compound pre-existing dehydration, resulting in significant hemodynamic effects after leaving the water. The risk of hypothermia in cold water may be ameliorated by adequate thermal insulation, however without appropriate thermal protection there may be a substantial risk of hypothermia. Although water aspiration may be prevented by wearing a full face mask, aspiration of vomitus

may still occur. A full face mask will also not prevent airway obstruction during or after a convulsion, or the pulmonary barotrauma that may occur if the diver ascends in the water column at such a time.

While experienced divers may have used the technique successfully and safely, whether recreational divers could do so remains an open question. At this workshop Frank Farm presented information that Hawaiian divers typically make thousands of dives per year, and have often experienced bends themselves or have assisted fellow divers with treatment. On the other hand the median number of dives reported by recreational scuba divers in the DAN database of dive accidents is only around 100 (6).

Furthermore, the effectiveness of in-water recompression in comparison with standard chamber techniques has not been assessed. There is evidence, however, that for severe DCI, short shallow recompression tables are less effective than standard USN tables (7). Unless the hyperbaric oxygen exposure during IWR is fundamentally different in some way from dry chamber dives it is likely that the necessarily abbreviated oxygen exposure during an IWR treatment will not be as effective as a USN Table 6 treatment.

In the context of recreational diving IWR is only one of several possible first aid measures and thus the relevant question should not be focussed solely on IWR but on the more general issue of which one(s) to employ. Surface oxygen induces an increase in the gradient for diffusion of inert gas from bubble into tissue, and can be implemented more rapidly than IWR. Fluid resuscitation, placing the diver in the supine position warming him can all be similarly instituted rapidly. The effectiveness of these measures, all intrinsically safer than IWR, and not likely to delay movement of a boat, in comparison with IWR, is unknown. Adjunctive pharmacological compounds are likely to augment further the effectiveness of surface techniques.

The decision to use in-water recompression must therefore be based upon a balance of risks, and should be used only if the assessment of the responsible medical provider knowledgeable in diving medicine, preferably on-site, is that the potential risks are outweighed by its perceived benefits. It should only be implemented by individuals trained in its use and only when the appropriate equipment is available. In-water recompression should not be used in areas where adequate hyperbaric chambers are available or within a reasonable transport time to a chamber. In-water recompression should not delay transport to a recompression facility.

Several unanswered questions remain:

What is the best first aid measure for DCI?

Under what circumstances and by whom should any available measures be instituted, after what sort of training?

Is IWR more efficacious than the combination of surface oxygen and fluid resuscitation?

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INTRODUCTION TO THE FINAL PANEL DISCUSSION

Robert K. Overlock, M.D.

There are serious questions and immediate concerns that a person would be faced with when trying to come to a decision on immediate in-water recompression. First is the issue that you may or may not be given the opportunity to make the decision if the diver never tells you there is a problem. Paul Linaweaver, many years ago said, "The first three symptoms of decompression illness are denial." We had a case treated in Honolulu wherein the captain of the crew was well experienced in the use of in-water recompression and had in fact used it many times, but the diver in the chamber said, "Well, no, I really didn't tell him that my shoulder hurt, and that my arm was getting numb." By the time we received the patient, he had both weakness and numbness and was six hours away from having rapid treatment. So denial is the first very real problem.

There may also be a problem on the part of the person that might be willing to do the in-water treatment. If that person says, "Hey, no-no, you're not bent, we don't have to do all that, we don't have to stop what we're doing to treat you", that kind of thing will stand in the way. The same thing is true for instructors, dive masters, and. other people responsible for the diver training, because very often the diagnosis is not made or is delayed because the dive master says, "No, we didn't do anything wrong. Therefore, it can't be." Those of us with experience treating divers certainly know that it can be.

The next immediate concern is the availability and the nature of the resources. If you don't have the right stuff, I think that you may in fact shift the risk benefit ratio in the wrong direction, and that's always going to be a concern. The availability of resources needs to be looked at very carefully if you're going to be making triage decisions about where to go with this diver. If you have readily available recompression facilities that are appropriate for treatment, then you need to be moving in that direction. I think it has been clearly established, that treatment in a recompression chamber by people *who are trained and competent* probably constitutes the best scenario. On the other hand, if that treatment can't be carried out for six or seven hours because of the location of the dive or for any of the other reasons already mentioned today, then transport may not be the best decision for that diver.

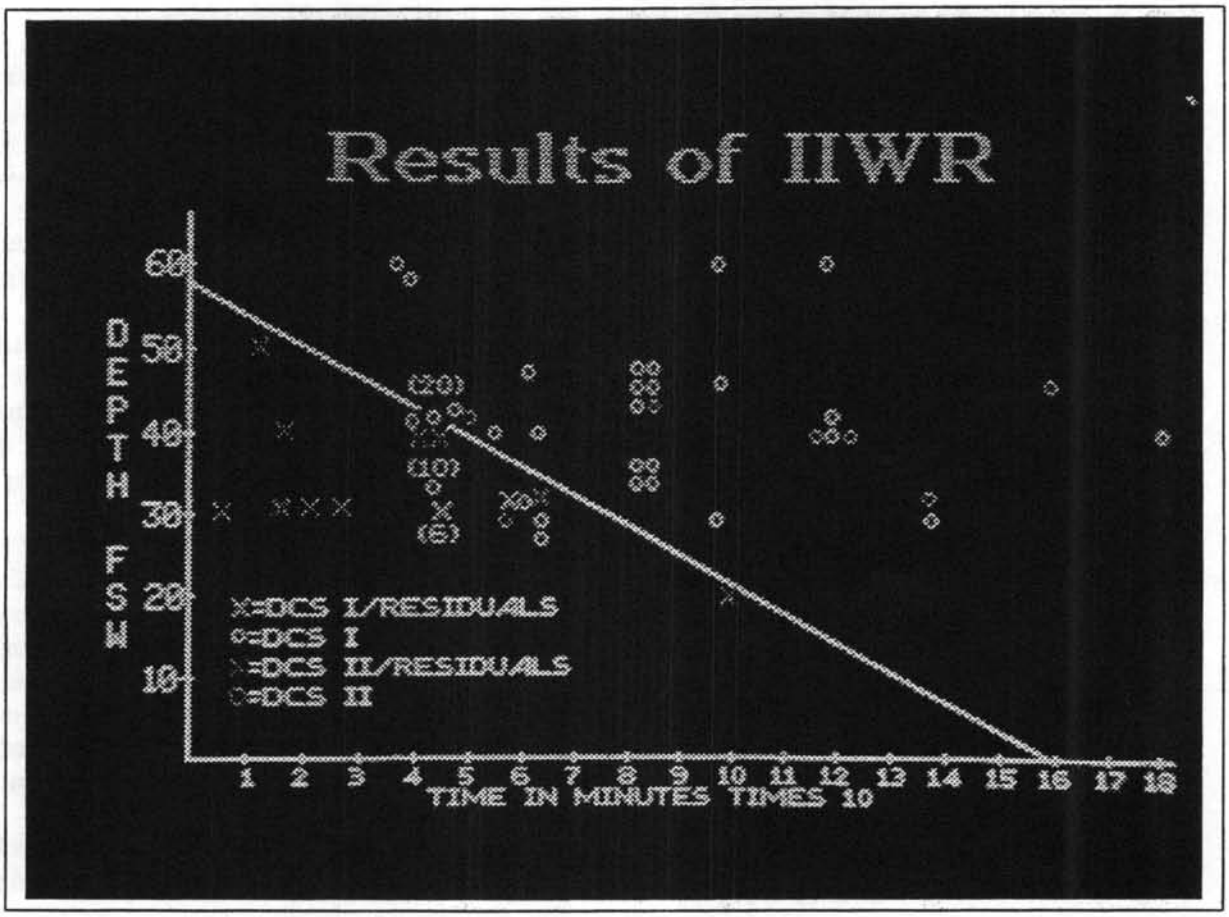
The availability of a portable recompression vehicle has been discussed. New materials and technology have provided us with several potentially viable alternatives to transportation or in-water treatment. If the diving operation can provide this equipment then treatment is immediately available at the dive site, and that represents the best of all worlds.

Transportation itself has very significant risks. Flying patients from a remote marginal airport on a dark and stormy or rainy night to treat a sore elbow doesn't make sense, because the risk of an accident in that kind of evacuation is relatively high.

Whoever is responsible for triage needs to be able to assess all of these resources, including adequate oxygen, and/or air or whatever other gas might be considered for immediate in-water recompression. And you'll hear me emphasize in every way I can the word "immediate". We're not talking about treating somebody hours later. We're not talking about even 20 minutes later. We are talking about within the first five to seven minutes after the onset of symptoms. If you delay much more than that the results are less predictable. Richard brought this out in his talk, by the way, *which* I should compliment. Doctors Beckman, Hardman, and Smith found a degree of irreversible damage after more than 10 minutes. We must consider the water, weather, personnel and their expertise, and the candidate. Is immediate in-water recompression appropriate in the situation at hand? All of these points have been mentioned before today, but I think these are the major issues that we're trying to deal with.

The reasons to consider in-water recompression:

1. Rapid initial bubble growth. We know from the physics of our situation that bubbles grow extremely rapidly in their first few minutes of life. If you reverse that process, by applying pressure while they're still in the 40 or 50 micron phase, then indeed you are likely to abort the process completely. Many of Rich's remarks, and those of others and certainly much of the experience that we've had with other divers would tend to verify that. I have one slide that I was going to show. Mr. Farm showed this slide earlier, and I'd like to point out a couple of things about it.



The vertical axis here is the depth of in-water treatment on air. The time line is actually time in minutes times 10. So, the line that's drawn here from roughly 60 feet to approximately a 160 minutes actually separates the majority of the failures represented by the X's from the successes represented by the circles. So the depths that we're talking about here are 45 to 50 feet for 45 to 50 minutes, where you begin to hit the majority of successful times. The parenthesis 20 here means that there are 20 cases at that point. And it seems fairly clear that if you get out here to 50 feet for 60 minutes, you're into the good zone by a long shot. But again, this needs to be carried out within the first 5 to 7 minutes, and that's all we need that slide for, unless there are questions about it.

What we want to avoid or abort is the rapid onset of paralysis and/or shock. We want to avoid further injury from bubble growth, and to do that safely we want to make sure that if we are to consider in-water recompression, that all of the resources are in fact available. The alternatives should clearly present a higher risk for poor results, if in-water treatment is to be considered. In other words, if you have a better system, a safer system, a system for which the risk:benefit ratio favors the patient more than in-water re-

compression, then that's the way to go. We're all responsible. We've all mentioned these very issues and I just want to say it again for the emphasis.

2. The reasons to avoid the use of in-water recompression would include the absence of the resources required for its success, adequate oxygen and air for immediate in-water recompression. A second reason might be immediacy. How long does it take for divers who has come up from a dive, to undress, taken a shower, remove their bathing trunks, go into the lounge, open the first beer, and then decide that they've got a symptom(s) that would make you want to consider in-water recompression? By the time you get him all suited up again and back in and ready to dive, can it still be immediate (within those 5 to 7 minutes)? Is that feasible? Can it be done?

These are the things that need to be considered because we are speaking here about immediate recompression. The water, the weather, the personnel, expertise. The diver or the care provider may be resistant to its use. If the diver doesn't want to get in the water, it is clear you don't want to put him in the water. I think that's pretty well been discussed already. The same thing holds true for the person carrying out the in-water or supervising the in-water recompression. If that person doesn't think this is the right thing to do then you don't want to be doing it. The diver condition itself may alter the risk:benefit ratio. If they're unable to dress, unable to hold themselves on line, unable to maintain depth, all of the possibilities that might ensue if these variables can't be controlled for, you may be putting the person at a higher risk.

The bottom line is that the window of opportunity is relatively narrow. You need to make the decision in a very short time, and you really don't have time to go out and gather up gear to do it. If you haven't already prepared ahead of time, this probably is ***not*** going to be a viable option. All the resources have to be available and ready for use. All of the people involved have to have diving expertise. Stakes are extremely high. You're juggling diver tissue injury and/or its salvage versus the risk of further injury. Since stakes are extremely high, that is why I think all of us are so very much concerned about how this kind of a workshop comes out. We don't want to be putting people more at risk.

If you use in-water recompression, you're going to be open to criticism. I think that's fairly clear, and even though it has been demonstrated to be successful, you still will be fighting a long tradition of condemnation of this procedure and for good reason in many cases. On the other hand, I overheard a remark that suggested that if it were clear to a person that this was the right option for that person at a given time. and it was also established clearly that the person responsible for the decision-making knew of this procedure and its efficacy under those given circumstances, they might indeed be liable for not doing it. So, there we are. It has significant potential for legal action, and I believe that as responsible physicians and care providers or diving supervisors and all the other positions we fill here, that is of major importance. The bottom line is it works, but it probably doesn't work for everybody under every circumstance nor does any other tool work for everyone in every circumstance.

It is appropriate that you try to do this as quickly as possible. Suppose you missed that window of 5 to 7 minutes. I think the time by which you miss the window determines how you alter the risk:benefit ratio. Ultimately this will always come to a clinical decision, and we all know of cases when late treatment had a marvelous result relatively quickly and we all know of the other kind as well. You need to integrate many data points to make this kind of decision and I would hesitate to say that you could come up with a universal policy that would fit all circumstances.

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Dr. Hamilton:. Suppose you missed the window.

Dr. Overlock: I think by the degree to which you miss the window, you begin to alter the risk:benefit ratio. Again, Bill, ultimately these will always require a clinical decision. We all know of cases that were treated late and had a marvelous result relatively quickly, and we all know of the other kind as well.

You need to integrate so many data points to make that kind of decision that I would hesitate to say could come up with a universal policy that would fit.

Mr. Dunford: I just want to amplify Bill's remark. I'm also a little disturbed by the 5–7 minute hard line that you've drawn here.

Dr. Hardman's data suggest that 10 minutes had some morbidity but not much more morbidity than 30 minutes for serious decompression sickness. So, maybe unless you have other data, maybe that 5–7 minutes is not such a hard line: If you publish 7 minutes as the ultimate outside limit, knowing diver mentality like I know diver mentality, 7.1 minutes, those people will not get in the water.

Unidentified: I'd also like to make a comment about the time limit because I think that may be a bit too restrictive. The reason I believe this is based on experimental evidence where we've actually tried to recompress animals. We waited until they had a maximum amount of gas bubbles and then recompressed them. That takes even in extreme severe dives in animals usually something like 20–30 minutes. We have been very impressed that even compressing them down to 10 meters on air, even if they were dead in the sense that they didn't breathe anymore and hardly had any heartbeat, they revived completely.

I'd just like to show you a slide. What you see here are several experiments. This time is the axis, and on this axis is the number of gas bubbles in the pulmonary artery where a hundred percent is set to the maximum amount of gas that we had after the dive. This line shows what happens if you don't do anything at all, if you simply let the animal bubble along. It takes a long. fore these bubbles are eliminated.

This curve is oxygen at the surface, and these three curves are either 10 meters on air, 18 meters on 100% oxygen or 30 meters on 50:50 heliox; the curves are very, very similar.

So, when it comes to elimination of gas from the central nervous system, it doesn't seem to matter very much if you do very shallow recompression or if you actually do a much deeper one.

But it's not the whole answer. If we could have the next slide? You see here, this is the initial dive, which is the standard dive, 40 minutes at 40 meters, 2 minutes decompression. The black ones are the gas bubbles in the pulmonary artery and the red ones are the gas bubbles in the jugular vein. This is standard treatment done in a way that we waited until all the bubbles were disappeared, and then we waited for another half hour and decompressed them slowly to the surface. One can see that the bubbles in the pulmonary artery have disappeared completely, but there are still some bubbles in the jugular vein for something like 10–15 minutes.

So, there may be more to this story, but in any case, at least as far as these experiments indicate, they indicate that even 10 minutes is very, very effective in increasing the elimination of gas bubbles, it also indicates that perhaps the 7-minute window is a bit too short.

Unidentified: I can't argue with that because the 7-minute window is a relatively arbitrary one based on the need for central nervous system tissue to have oxygen and nutrition. It's based on a worst case scenario, assuming that you provide neither of the above to the injured tissue. I didn't bring a slide because I didn't know I was talking about this to illustrate the rapid growth of bubbles early on. That's been well established by many, many people, and if you've got injured neural tissue, which is relatively fragile tissue, with bubbles growing., then how quickly do you want to get them back under pressure and back under control? As quickly as possible.

I think that's fairly clear, and the stories that we hear seem to be those divers who got in the water relatively quickly.

Dr. Vann: It strikes me that there's a critical question here that I don't think we can answer right now, but it bears upon this whole process and beyond this, too.

Is there a golden hour for DCI therapy? The answer is, we don't know. Now, you can make arguments on both sides, and this is going to require good solid clinical data. We can't do it with animal models. We can't do it with models of bubbles in gelatin or anything else. It's got to be done with human experience.

I think there was a recent paper at the last EUBS meeting that said if you don't catch them in an hour, then it doesn't make any difference how long you wait. But until we have this answered, it's going to be difficult to draw some solid conclusions about it.

Unidentified: I couldn't agree more, Richard, and the fact is it wasn't too many years ago that we were telling people that if you waited 24 hours, there was no sense being treated.

Dr. Overlock: The next issue is one that bears on, I think, the Divers Alert Network, and any organization that's going to make recommendations to its members or

recommendations that might be looked at as policy by some folks. Just to open up the discussion, it would strike me that it would make more sense to use DAN's limited resources to put in chambers that are close to the dive sites than to, say, train people to do in-water recompression with the potential liability for lawsuit because of somebody has a bad outcome, not necessarily related to the procedures, but just open that subject for discussion, see what people think.

Dr. Edmonds: Bob, I agree with your whole presentation, except for this implication that you've got this funny 5-minute period. I don't know about the sort of cases you see, but many of mine keep getting worse over a couple of hours and respond very well to treatment, even 2 or 3 hours later.

I think you're basing all your stuff on that one graph and I don't think that graph's good enough. To my knowledge, bubbles increase in size and volume and total volume for many hours; sometimes after weeks.

Dr. Overlock: They do keep increasing, but the quicker you get them under control, the better off they're going to be.

Dr. Edmonds: Of course, but why say 5 minutes? Why not say 30 minutes or 3 hours?

Dr. Overlock: Merely to emphasize the need for immediate treatment.

Dr. Edmonds: Oh, I agree with you thoroughly, but if you say 5 minutes, then as someone said, 10 minutes, they won't bother trying.

Dr. Overlock: But then perhaps it should be stated in a different way. Perhaps it really should be stated 5–7 minutes is optimal. Anything longer than that becomes less so.

Dr. Edmonds: Yes. But it may still work 6 hours later.

Dr. Overlock: There's no question in my mind about that, Carl. Really none. And I'd like to make that real clear.

One of the things that you try to do in a situation like this is to polarize the arguments so you'll bring out all aspects, and I think that it's served its purpose in that respect.

Dr. Moon: Just to unpolarize the issue a little bit, I think we need to say what kind of bends are we talking about. Do we seriously believe that somebody develops an elbow pain a few hours after a dive needs to be recompressed within a few minutes?

If you look at the recreational diving data, the median time from onset of symptoms to recompression therapy is about 24 hours, which is absolutely abysmal, and yet the number of individuals with severe permanent disability is very, very small.

So, I'm sure everybody would agree that we need to recompress divers as quickly as feasible, but whether it's 5 minutes, 5 hours or even 24 hours may not make that much difference.

Dr. Overlock: Indeed, you've caught my second bias, and that is simply that I am strongly biased by the bad cases, and those are the ones that I was addressing.

The guy with simple elbow pain or a sore pinkie or even, as DAN has been pointing out more and more lately, the little niggles that we're seeing so much more of now that we have a much larger diving population do not seem to be that emergent, and indeed there should be no risk to the patient in the treatment of those diseases.

So, whatever decision is made should be made on a no risk to the patient basis.

Dr. Moon: Well, that's exactly right. First, do no harm. I'm not sure we've totally come to grips with the potential harm that we can do to patients with in-water recompression or indeed in any other emergency first-aid maneuver.

Dr. Overlock: That's exactly right. Not only is in-water recompression fraught with potential for misstep, if you would, but so is transport, so is oxygen breathing. People have blown-up embolisms turning on oxygen systems. They all have some risk, and our job really is to make sure that everything we can do points to the best risk:benefit ratio for the patient.

It's so simplistic, but yet it is the right answer. We may not have the details. We may not have the data at this time to determine where the cut-off points are or how that matrix shifts with each of these options. But I think it would be wrong to deny the option completely, just as it would be wrong to say it's the only way to treat.

Michael ?who?: Bob, your response to Bill's question is that there are a lot of variables and it's going to come down to a clinical decision, unless it's only going to be diving doctors who can avail themselves of this immediate in-water recompression. In the decision-making process we need to get it to the level that the average diver can use.

I would submit that to flatten the learning curve and make these decisions, something that's already in place although not as extensive as we would like, is a 5-minute neuro. So somewhere in that decision-making tree, if the in-water recompression aspect gets plugged in somehow so you can go through and make those decisions, then that would help to decide once the parameters are outlined on how to go about deciding that.

Dr. Overlock: I think you're right, but very often the relatively disastrous cases that I tried to point toward have relatively serious deficits, as Richard described have relatively serious deficits, rapidly progressive. And it's very clear, I think, you don't need to be a physician to see somebody who's paralyzed from the nipples down. I mean it's that simple.

Dr. Hardman: I know in reviewing Dr. Beckman's recommendations about this, one of the things that he and I used to argue about a fair amount was related to this

issue, but the truth of the matter is, from what I'm hearing today, it sounds like we have shown by anecdotal and other data that the immediate compression can be done successfully. And it sounds as though we need some specific clinical trials to find out where these transition points could be, instead of letting it all happen in a haphazard kind of way.

Dr. Pyle: I just wanted to comment on a couple of things that have been talked about recently. In one sense, we have polarity in personalities here, but I don't think we have a polarity in what we're saying. The fundamental things we're saying are almost identical. We're saying that it does work in some cases and that it shouldn't be used in all cases and . It needs a lot more study because we really don't know what's going on. I completely agree it's anecdotal. It's convincing, but it's anecdotal.

As far as I'm concerned, you can just write that off as the impetus that piqued your interest in this topic enough to start paying attention more carefully. Divers Alert Network would be an optimal repository for information on this. Someone needs to collect information in a structured way so that we can see things a little more analytically than just off-the-cuff observations.

I had more points, but I've lost them for now. So, thanks.

Dr. Overlock: Carl, we'll get you next, but I'd like to say one thing. I think that we've made a significant step forward in the dimension of opening up discussion about it, and Richard's recommendation that DAN perhaps may be the repository of the data.

We'll never get anywhere if we don't learn what we're doing and how effective it is or is not. We'll never answer Richard's question about whether 7 minutes is a cut-off or 10 or 15 or 18 or 13 or 12. There's no way to answer that, and we certainly would be hard-pressed to set up a double-blind study to do this one.

So I certainly agree with the remark that an animal model isn't going to answer the question either. So, we really need to collect our human data, and we need to get it so that all the data gets collected.

One of the big problems is there's been such a horrendous bias against the concept of in-water recompression that nobody wants to admit they did it.

Dr. Vann: Well, we had this discussion last night, did we not. I certainly agree with you as being very involved in collecting that data at DAN, we're going through the agonies of revising our system, and letting it evolve. I'd like to ask all of you who operate recompression facilities, please work with us to submit this data and to help us improve the data quality. Help us improve the system so it will do more for you. It's going to take a while to do this, but we're in a 1-year pilot project right now for our revision from the DARF (Diving Accident Report Form) to the DIRF (Diving Injury Report Form). We will soon be coming out with an electronic or digital version of the DIRF, the EDIRF, which we will be sending around for everyone to evaluate and comment on, and hopefully to use. We know that if this is going to be used effectively, it's got to make your life easier. It's going to take us awhile to get there, but work with us and help us to do this.

Dr. Overlock: Very good. I'm going to ask you to wait just a moment while Carl responds.

Dr. Edmonds: This is not a response. This is a response to things that are being said here today, and it's sort of nit-picking. So, I'm going back into the arguments instead of the details.

I've heard some statements here that I think are sort of stupid and I'd like to just point out that I don't agree with them. Therefore you can decide whether they're right or not.

Firstly, I've heard statements that if you do in-water treatment, the communication is terribly difficult. That's nonsense. You lift the man out forward, and you speak as you would normally speak. It's as simple as that. Communication is dead easy and you've got a line to the top so you can communicate with the people on the surface.

Second thing, I've heard a couple of times now, once by chairman and one by a couple other speakers or twice by two other speakers, that if you have a minor problem, then you really want to put them in the water in a one-man chamber. If you've got a big problem, gee, you wouldn't want to put them in the one-man chamber or in the water. So, it's really a conundrum.

That's also nonsense because that's not how decompression sickness develops. It starts as a minor problem and gets worse progressively over the next 3 or 4 hours. So, what you want to do is stop the problem from developing into a major one.

It's not a matter of a minor versus a major problem. It's a matter of stopping the major problem developing.

I was very subdued when I heard the speaker say that, but I've thought about it now. The third thing I've heard is the tremendous training you've got to have to look after someone who's underwater convulsing. We train an awful lot of divers, and we've had an awful lot of underwater convulsions.

I'll tell you how much training there is done on it. You tell the guy's buddy, his attendant, if your fellow convulses underwater, just hold him where he is until the convulsion ends, then you bring him to the surface. That's the amount of training that our Navy clearance divers do, about 30 seconds of instruction.

There's been an inference, not actually said but inferred, that when you have this convulsion, it's going to have terrible things to do with your airway. The implication is you're going to be hypoxic. If you're going to quote Donald,

one of the things Donald said was you don't have to worry about that because it's an oxygen convulsion. They're not going to be hypoxic for a long while, not that I like quoting Donald.

The last thing that I'd like to point out is that we saw some presentations showing the inefficiency of short tables. I think that is unfair. Short oxygen tables do not give oxygen thereafter. You will get recurrences, but none of those tables that were mentioned, I believe, were given oxygen afterwards. So the recurrence rate is an irrelevancy to this discussion.

I think you're sometimes being misled by delightful selective parts of the literature. Well, how many friends have I got left?

Unidentified: In the study by Kindwall, what they actually showed was that the short tables were actually just as effective. The only thing they were not as effective for was AGE, and they are very low numbers. We don't know.

So the data that we have seem to indicate that normally the tables are better than the other. The point I'm going to make is about the clinical trials. A couple years back, we were in Palm Beach and discussed treatments. Here we are sitting seven years after the last meeting, and the only thing we can say is that, well, the only data we have are data for the U.S. Navy table 6. We don't have enough data to make any decisions, and believing that we're going to get data on clinical trials that will help us make decisions, I think, is very optimistic indeed.

I don't think there is any doubt that rapid recompression and oxygen will help you. It's not what we do not know very much about, but what is an effective treatment depth? What is the optimal depth and what is the optimal dose of oxygen. Those are questions I think we have to solve by doing careful experiments and simply seeing what is there and have objective outcomes. If it is humans or if it is animals, I don't really care. But I think we have to do some experiments that are geared towards that, even though we would probably be a bit reluctant to put people in the water—perhaps use Navy divers. They don't have a problem.

Dr. Overlock: Navy volunteers will do anything for time off, right? We already know that. Actually, I very much agree with you. There's little to really point to the optimal partial pressure of oxygen for the treatment of any of the diseases we treat. There's very little hard data to support a specific number.

If you go to 2.8, is that better than 2.6 or not quite as good? Or is it 3 times better than 2.2? Nobody seems to have hard data to support those positions and there are far more open-ended questions than there are answers.

Mr. Farm: I can appreciate where the subject of doing more trials and trying to determine how much time and how much depth would be best;, I kind of agree that maybe for the people in this room that are in the diving business that's very desirable.

The bottom line here is, I think most people will agree, some form of in-water recompression or immediate in-water recompression can be helpful, whether it be 7 minutes or 10 minutes or half an hour.

But I really think that this group, or whoever might have the right influence with this group, should identify some of these issues as the result of this workshop and perhaps get a few of them put together to see how we can overcome them.

Certain groups in Hawaii are going to continue with whatever they're doing. The problem we have with the recreational people is how do we educate them? How do we ensure the liability questions and everything else?

We know it works. How do we get this other part across? This is what we hope that the Society would be able to assist in.

CDR Chimiak: We are getting close to wrap-up time for the workshop. The question remains, is it air-in-water recompression and oxygen-in-water recompression?

I think we all agree that oxygen on the surface would be beneficial for a dive accident. Anyone can correct me at this point. The idea of adding additional pressure to that inhalation would also be deemed an appropriate first aid therapy, i.e., going to increased pressure of one atmosphere.

Would anyone disagree with that? The idea of adding oxygen under pressure?

I guess one of the questions then is, is air a problem with in-water recompression, and at what depth? I think we had some very good posters that both Frank Farm and Richard presented that seemed to show that in-water air would be beneficial. But there are some drawbacks to air and that is the increase in nitrogen load and whatnot.

So, can we perhaps agree and perhaps even set this up since we do have data recording at this point, set up a UHMS oxygen table, perhaps based on the Australian model? Richard's was a good one. He became more conservative with the 25-foot, but before we see any problems, let's not go back to 25 foot yet and cut ourselves short and stay at 30 feet. Report that to DAN and put the word out in technical diving literature. Report this information as accurately as we can, information that meets the standards that DAN demands for its database or else reject the information.

Over the next couple of years, we should have some information on the oxygen. The only problem is what air table should we use? It looks like you have a nice graph, but it doesn't tell the whole picture. It seems that there's an ascent rate we have to conform to. Form a UHMS table 2

that's on air in-water recompression. That again will report, because if we have five or six different tables coming in, again it just muddies the water and maybe doesn't give us a clear picture.

Mr. Farm: I'd like to point out that the reason that air was stressed in my presentation today is, when we presented the paper and did the research, we had recommended the Australian table as one of the first options that we presented to the Hawaiian divers because we liked the table. Going to 30 feet on oxygen was Richard's idea.

In fact, if we had advanced two more slides on my carousel, you would have seen it. The reason that I did not show it this time is because we tried to instruct the people out there to use this oxygen table. They either cannot get the oxygen very readily and have the set-up or they're too darn lazy to have all this equipment available. But they always have air, you see, and that's why we had to take perhaps the second best thing.

They've got air on the boat, and so they do it with air. We've told them to try to use the Edmonds method which was the first method presented in the International Congress in Australia. They don't really do it. The discussion of whether they can get oxygen freely. Not all of them are as sophisticated as Richard with rebreathers and do all those things.

That was the reason they didn't go to oxygen. We therefore used air because that's actually what they had been doing for the last few years.

CDR Chimiak: Mr. Farm, I agree. The use of oxygen is a good source, but I was saying that you had this option because that's the most readily breathing source out there and I think that's the one most people would use. In fact, in our area, for our diving fishermen, they use air in-water recompression also.

No. I was saying that since you have the most experience drawing up that particular table and instituting it as the one that perhaps we might all use. You know, of course, the Navy treatment table 1 alpha is totally unrealistic to complete under those circumstances. So you have the practical resources at your disposal to devise the actual table that we'll be testing.

Mr. Farm: Just for the record, even on my boat when we go out, we carry a 50:50 mix as the first tank we use for any in-water recompression. Just to build up a little more oxygen and maybe that happens only because I have access to it. You go buy a couple nice clean tanks; don't use them for anything else. Try to get this gas in it, and never use it until you really need it. Well, they're going to use it ahead of time because they'll feel it is safe, and then you've got a contaminated tank.

Dr. Sanchez: I would like to thank the workshop for opening a new possibility of treatment, especially in those areas where we don't have chambers.

My problem with this activity is, one, I wouldn't like to see an adequate treatment being delayed because an attempt to do in-water recompression with air. Two, we are losing the preventive part of the diving teaching. We should prevent accidents instead of just allowing divers to treat themselves with in-water recompression. I do understand it's very good when we don't have other resources Three, when I see the divers, the type of divers that do a lot of crazy things. It's going to be open to them and they are going to institute it as a treatment table for inadequate diving. We are going to see a lot of problems with that, especially in those areas in which they get half of the information with half of the equipment and half of a treatment. I see those problems especially in our countries where the dollars are difficult to get all the equipment and all the information.

Dr. Baker: One point I'd like to make is everything we discussed has been in the context of being out on a private boat.

On the Gulf Coast, we typically have boats that have 15–20 divers on them. So, if we are going to put this information out about in-water recompression, the boat captain's not going to sit out there for one person to go in and do that schedule. So, we'll have to define when it's appropriate, when it's not.

Dr. Overlock: Certainly that is one of the major issues.

Dr. Pyle: I couldn't get my brain together in time. I wanted to comment on this idea of never delaying treatment in a chamber.

If people who do this routinely get very favorable outcomes on a regular basis, transport to a chamber is delaying treatment. It can be flipped, you can just as easily say don't delay in-water recompression by wasting time trying to go to a chamber. I'm just pointing out that treatment is presumably administration of oxygen at increased ambient pressure. Nobody would argue that a recompression facility is a much, much better treatment. There's no dispute of that. But to say that you should never attempt in-water recompression because it's going to slow you down getting to the chamber for treatment.

For example, and this touches on another issue, if you're going to do it at all, you have to do the full treatment. Well, what about the issue of just waiting for the helicopter to arrive and breathing pure oxygen at 30 feet rather than on the boat? I mean maybe that's a bad idea, maybe it's a good idea. The helicopter's 3 hours away.

Unidentified: We'll all vote for that.

Dr. Pyle: So in that sense, you're not really delaying treatment, but that doesn't mean you don't do in-water recompression just because the treatment's on the way. I mean, to me it's a little bit more complicated than that.

Unidentified: I agree on that. What I disagree with is once you have repetitive diving accidents, there's something wrong there to me for the diver. You know, if you're a motorcyclist, and you have 10 motorcycle accidents in one month, there's something wrong in how you're driving.

Dr. Pyle: Right..

Unidentified: It's the same thing as how you're diving.

Dr. Pyle: I couldn't agree with you more.

Unidentified: That's the preventive part of diving. It means that you need to get better training or better schedules.

Dr. Pyle: That was the point that you made that I agreed with, which is that this should not be viewed as a crutch or an excuse to allow divers to allow themselves to do more dangerous dives, thinking I'll be able to treat myself, no big deal.

The first time I started giving presentations on this topic, my first big slide was—never has it been more true than the phrase a little knowledge is a dangerous thing—no knowledge is less dangerous, and lots of knowledge is less dangerous. If you know just enough to get yourself into trouble but not enough to get yourself out of trouble, then it can be much worse. That's what you really need to avoid. Which means you can't mention this topic, and that's why it's dangerous.

Right now, we're in the really dangerous period, and I'm partly responsible, and you're partly responsible. There's enough out there in the published literature that people said, you know, I heard something about you can get back in the water. They've heard about it, but there's not sufficient information to help them understand the dynamics.

So, right now, we're at this really critical threshold, where you either are going to back off and say never do it and stand by that and make sure everyone agrees with that Or you go over the hump and get the education flowing as fast as possible before too many people find themselves in the situation where they use it as an excuse to do more dangerous dives.

Unidentified: There are actually two issues involved there. One is the issue of whether people will in fact increase their risk-taking because they think they have an appropriate bail-out, and that's a very dangerous prospect. It's one of the things that really concerns me. The other issue is whether one uses the procedure in the appropriate circumstance to save tissue.

Dr. Edmonds: You won't get rid of decompression sickness by stopping people having recompression chambers.

Unidentified: No. What I say is the only thing that I agree on is, we see this type of diver, that once they have this additional treatment element, they're going to treat themselves because they are going to do worse diving because now they have this capability because they were not physicians and they are the only people that can treat you in Mexico.

For those remote areas for our commercial divers, it's an excellent possibility of saving lives. The problem is with this new group of technical divers. They are always going to the limit. They are going to use it to treat themselves.

Unidentified: I've always said that if you really want to avoid accidents among these emerging technical divers, you should have a workshop on how to limit testosterone levels in blood rather than ....

Dr. Kay: If you can think back a decade to a time before we were promoting oxygen, think back to how oxygen evolved as a surface gas for first aid, and some of these very same arguments that we're hearing right now were promoted as a reason not to use surface oxygen.

We heard that the divers would suck it up at any given moment to try to dive to crazier and deeper depths. We heard that you needed a week-long course to learn how to give it and we heard things that are just deja vu, how you say, all over again. I would propose that what we're hearing today is a little bit of the same apprehension that may not really be justified.

Unidentified: I think what you say is true, but we also find on some relatively rare occasions, people who have used the oxygen in the very same way. They use it as a vehicle to allow themselves to take increased risk. We actually do see cases of that. I'm not arguing against oxygen. Don't misunderstand me. You will probably have an element of abuse no matter what you do.

Dr, Delft: These same kinds of arguments were made against nitrox. Three years ago, nitrox was a black gas. Over 10 million cubic feet of nitrox were produced with our system. This isn't an advertisement. That's a lot of nitrox dived.

It isn't being abused from the records that exist and the histories that are being kept. It's not causing anyone to experience any additional problems in being treated as divers in terms of the recompression applications, which was one of those lists of potential disadvantages. I really think, Richard, that you can be more optimistic about seeing in-water recompression within your lifetime because I don't see it as being much different from nitrox.

Dr. Pyle: I do see it.

Dr. Moon: Making medical decisions in the face of uncertainty is nothing new to any of us. However, using surface oxygen as an emergency gas was perhaps in that category. But I think all of us have used oxygen in other settings before and realize that it had very few side effects.

What we have here, though, is really some data, again no implied criticism, that fails to measure up to the detail that we normally require.

When a new drug comes on the market, for example, I think there are very few physicians who would be willing to prescribe it without seeing its expected benefit under what circumstances and what its side effects are.

I would like to go back to what Jim Chimiak said, and that is, urge people to collect the data, to allow us to look at it, examine it, and see how it really works.

As a minimum, I think we need to know for each case the dive profile, the time of onset of symptoms, what those symptoms were, and if there can be any physical examination to record the depth of recompression and what gas was breathed, and to note what the outcome was.

I mean those are very simple pieces of data, and if we had those, I think we wouldn't be having this discussion at the moment. We'd either decide that it was a good idea or discard the issue.

I don't care if DAN collects the data or anybody else, but I think we just need a little bit more information than we currently have.

Dr. Hamilton: Are you willing to put blanks for that on your DAN form? In-water recompression is already there?

Dr. Moon: Yes. The opportunity to present it is there, but that's relatively new, too. I think that's the point that I've tried to make a little while ago is, now that we've opened the door, and we see that there's a need for the data, and that we need the data to be collected in an unbiased way, we have a hell of a lot better chance of doing it.

Dr. Edmonds: But with all due respect, do we not have most of the data?

Dr. Brubakk: First of all, surface decompression using oxygen has been used as a technique to prevent decompression sickness for many, many years in commercial and military diving. The results are no worse than any other technique you can use for diving. So we know that actually putting people under pressure and giving them oxygen will at least get rid of a lot of gas.

Now, to the clinical symptoms, Carl Edmonds has just presented a very large series from Australia where the pearl divers who use it seem to get away with it.

So, I'm not advocating that this is going to be the treatment way of the future, but this is an emerging technique. Couldn't we rather than insisting on more data on the effect, which I think any study will show that it is effective, if you manage to do it, rather discuss whether it's possible to do this safely That is the issue, to do it safely.
If we can do it safely, obviously we should do it. The question is, can it be safely done?

Unidentified: Well, I think, Alf, the devil is in the details. We don't know much about the clinical presentation of those cases in the pearl divers. Bob Wong was telling me in the break that he thought that perhaps a majority even were pain-only bends, and, so, I think there's very little argument about pain-only bends. It's unlikely to get worse if you treat it. There's very little risk.

What we don't know much about is what happens with the more serious cases, other than a few anecdotal reports that people have presented today.

Dr. Ladsen: One thing I'd like to mention. I think DAN's a fantastic organization, but one possible problem with them being responsible for collecting the data would be that because of their responsibility and their perception that they could be held liable for any recommendations that came out, they're probably going to be somewhat hesitant to make any recommendations.

So that although they may have a very good data-gathering capability, they may be somewhat more restrained than the technical diving community, for instance, in terms of analyzing that data and making recommendations. It puts them in a precarious position to ask them to do that.

So, it may be that DAN could collect the data but also make it available to other interested parties that may not have the same constraints to analyze and present that data.

Dr. Bennett: Understand how DAN works. We work by consensus. The data that we collect, all the data we collect, are available to anybody who wants it. That's the way it's done.

Now if we collect the data on this issue or any other, there is a need to come to some form of a statement of policy or position. We actually discussed one other area. We will call a meeting of the training agencies. We will get with them. We will have a kind of meeting like this, and we'll discuss it. We will come to a consensus of what we're going to do.

That means there's an industry support for what we do. DAN can't act in isolation. We're not a training agency. We can't say you will not dive. We're not the dive police either. So, it's an advisory-type mode that we take, and it's a consensus mode that we take in coming to decisions.

Unidentified: I think that's an important point that Dr. Bennett made. In fact the data are available to anyone, and the trick is to get it now.

Dr. Bopal: Richard, I think you misunderstood me. We see Type 1 DCS bends nowadays in the 1990s, but in the 1980s, they had all kinds of bends, neurological as well. They do about 25 or 30,000 dives a year, and in the 1980s, the incidence of decompression sickness was around 40–49%, and they used in-water oxygen recompression. There was not one single recorded case of oxygen toxicity.

Now, oxygen was first attempted in 1981. Previously, in the 1970s, all the cases were in-water air recompression. I just wanted to make that clear.

Dr. Moon: Thank you for correcting me. I didn't mean to misquote you. Not at all.

Dr. Sutherland: When I was doing abdominal pain

collection for submariners, all of our data seemed to show that corpsmen did a lot better than physicians at diagnosing appendicitis. When we went back and looked, it turned out that the corpsmen were only submitting those cases which looked good. So that's going to be one of the problems with any sort of data collection where you're depending on volunteerism.

Dr. Pyle: I agree. That is a problem in the bias of the collection of data. If your only source of information on this is recompression chamber facilities that are treating divers who in their interviews had said yes, I attempted in-water recompression, then you're getting a disproportionate slice of unsuccessful cases because all of the ones that didn't seek subsequent treatment. I agree with you. You need to find these people who do it on a day-to-day basis, give them some sort of form that they can understand and convince them that they won't be stigmatized for reporting no matter what the outcomes are.

Mr. Farm: I'd like to echo what Richard just said because if we do that, and although DAN was mentioned as perhaps a source of collecting the data, and if it's going to filter through the chamber system treatments, then all the ones that really did in-water and were successful will in all probability not be reported. You've got a real heavy bias on that type of situation.

The data are so skewed that they won't make much sense because they wouldn't be at the chamber unless they had a problem after the in-water. Somehow the more careful people take care of themselves by coming to the chamber anyhow after they're through. I guess this is what I meant by this type of data collection or what should be looked at by some of the people that are more knowledgeable in the field of how to do this and get the best results.

Dr. Boisen: Carl, I wonder if I could ask you a couple of specific questions for the record? First, when did you and the Australians first start using surface O_2?

Dr. Edmonds: 1968.

Dr. Boisen: Could that be put in the record, because it seems to me that some people have said it's only been around for about 10 years?

Dr. Edmonds: Probably in America, it has, but in both France and Australia at least, and probably some other countries it was certainly used a lot in the '70s.

Dr. Boisen: I think that's an important point to note. The second question is related to some of the original work that you did. When did you first start using in-water recompression? What date?

Dr. Edmonds: That would have been about 1969 to 1970. We were caught out in '73, and by that stage, we'd already got our first 25 cases, and we'd lost control. It was being used in other areas. But the first time it was actually reported was in 1973.

Dr. Boisen: And did you write this up in the first edition of your book?

Dr. Edmonds: Yes.

Dr. Boisen: I thought that should be in the record, because I think what we have now is a tried and tested system that, I think, I would agree with Alf, is what we really need to check the safety of it for insurance purposes. I don't think we should be behaving as if this is a new system.

Unidentified: You have two friends now.

Dr. Edmonds: Thank you.

Mr. Farm: Bob, I'd like to just make a comment here once more, and I know there was concern that if we discuss this topic or if it's presented prematurely to the public, there would be abuse. I go back to what I was saying. That is why we have to think it out and identify the issues so that when it is presented, it's 99% fool-proof as far as liability or anything else.

On the other side, I wanted to state in case some of you may have the wrong idea about how things are done in Hawaii. Please don't get the impression by what was shown here today that everybody in Hawaii is that kind of a diver or the recreational people were not promoting immediate in-water.

In fact, on the dive medicine updates that we go throughout the state and on the different islands where Bob Overlock, Donald, or the physicians go out, when the subject comes up, we take a step backwards and do not comment on it one way or the other.

What was presented here related to the hard-core commercial divers. So if they do ask occasionally when they come to the chamber to be treated, what do you think about in-water treatment, and you usually get a good political answer.

But for the divers that we know that go out 4 or 5 days a week and who have been in trouble and are diving the profiles, and these are the people we're referring to, not the recreational community and not the charter boats.

Dr. Overlock: One other comment that fits with that, Frank, is that these folks for the most part don't know what DAN is or who DAN is or what it means or anything about it. The same thing is true, one would assume, Carl, and correct me if I'm wrong, for your abalone divers in Australia.

They don't know very much about DAN, and they're not likely to fill out DAN forms.

Dr. Edmonds: DAN who?

Dr. Overlock: If we think about data collection, and I think the point has been made there may not be the need for data collection in terms of the efficacy of the treatment but rather for its safety profile. Again we need to be looking at the populations that are actually doing it.

Dr. Kay: Well, I wanted to address a question to Bill Hamilton and Richard Moon. It has to do with the data repository issue. I know that Peter's under a lot of pressure to keep things safe and to do not bite the hand that feeds him. The training agencies also have to be listened to. But the Undersea and Hyperbaric Medical Society has advisory panels.

In fact, we've made advisory statements on the treatments used in hyperbaric oxygen for non-diving issues of wound care, and I wonder whether it might be more appropriate to have a UHMS advisory panel make an in-water recompression treatment?

Dr. Overlock: Sort of an analog to the oxygen committee report kind of thing?

Dr. Kay: Correct.

Unidentified: Years and years ago, we found out that the workshop was a good mechanism for dealing with a sticky question. The Society can't take a stand on the question like this. They have done so with regard to what things can be treated with HBO, but what's that called? That's called a committee report.

In other words, the Society itself still can't get a consensus. There's no mechanism for doing that. So, they do it by means of workshops and this workshop has addressed this issue. We've pulled as much data together as we could get on the table in one day. So, in effect, basically we've taken the first step of what you're requesting.

A standing committee on the issue would be a fine idea.

Unidentified: I mean more as an on-going data repository, a place to centralize the data for further study.

Unidentified: Well, Ed, there are several mechanisms for a consensus statement or information to be disseminated via the UHMS, DAN, or of the training agencies. This is one mechanism today. There is nothing to stop the UHMS from studying the issue and making a statement as part of, for example, the hyperbaric oxygen committee report if a consensus comes out of this workshop.

I think the mechanics of it really don't matter very much. DAN already has a data collection infrastructure available. So, it would be an easy place to do it, but if somebody within the UHMS wants to do it, in parallel or separately, I think it's a moot point.

Dr. Bennett: I wanted to make two comments. The first is, remember that DAN speaks only for recreational divers. We are not speaking for pearl divers, we're not speaking for abalone divers or any other kind of diver. We speak for the recreational diving industry, very specific.

In terms of what we've been hearing about consensus and workshops, I would remind everyone that we did have a workshop on flying after diving and I was perhaps foolish enough to take the consensus of that workshop to the industry. I lost my head because we were voting that 24 hours was the exact time that we should take before flying. The industry came back very fast and said wait a minute, if you do 24 hours, that's about, I don't know, 7, 10, 11, 12 million dollars worth of money out of the industry's pockets. Where is the evidence for what you have said? If you have evidence, present it. Otherwise, change your tune.

Then we went back and reconsidered until we had evidence, which we're now collecting. The data luckily is coming up to have about the same kind of time. But I'm just saying, be cautious because if you go to the industry without substantial data, they will come back to you and ask you to prove it. What we have been talking about is the difficulty of collecting substantive data that is meaningful. That's the problem we have and that's why we're here. We're wrestling at the end of the day with the problems.

We all know the problems, but we haven't got a solution to it. So, we go back to the old one. Well, let's collect the data. Well, who was it said we've been here 10 years or whatever, and we had a chance to collect the data, but we haven't got it yet. Maybe in 10 years' time, we'll still be doing the same thing. I'm not sure how we're going to collect this data. Maybe DAN's data collection with the computers and having the field people out there collecting data will pick up some of these folks who are being treated in the water, but as to how many, we don't know. How many recreational divers are actually being treated in the water?

We know there are lots of pearl fishermen and abalone divers perhaps being treated, but how many recreational divers are doing this, other than maybe technical divers who are doing it quietly for themselves?

Unidentified: My question actually was: Is the scope of your data collection limited to the scope of the people you cater to, the recreational divers? Can you glean data from the non-recreational community to make recommendations for the recreational community?

Dr. Bennett: We get some information on commercial divers. We don't reject it. On the other hand, we don't go out and collect it because it costs money, a lot of money, to get this done. Our money comes from our members who are recreational divers. Another quick comment. The implications of any recommendations will have to be weighed very carefully because the implications will go far beyond what we initially intend Consider the perspective of the guy who is managing a dive boat that conducts diving operations in 120 feet of water 4 or 5 or 6 hours away from the nearest recompression chamber. Somebody comes up who is bent.

These recommendations may sway whether he recommends instituting in-water recompression for somebody that's a customer of his. Those kind of implications can be worrisome. So, that has to be seriously thought about.

Unidentified: There are many, many variables.

Dr. Pyle: May I have one last word to set the record straight, like Frank did?

Although I talk a lot about in-water recompression, bear in mind I haven't had any direct firsthand experience with it in over a decade. It's not like my friends and I are getting bent every other weekend. I'm not saying that just to cover my own rear-end. The reason I'm saying it is that the method I've presented here has been developed over these last 10 years during which time I have not had any first-hand experience with in-water recompression.

It is not necessarily an empirical method. It's my best amalgamation between what I know from my capabilities as a diver and what my understanding of reading the medical journals and books about the known physiologies.

Dr. Overlock: Thank you, Richard, and let me thank you again for a very excellent presentation today. I think you've added a good perspective to what we're doing.

Michael (WHO?): Then you've made yourself well known on the Internet regarding the initial episode and prevention is one of the things that we're here about today. I wonder if you have any comments regarding, I don't know how to put this really, Richard, but you must have been through a learning process, and the question is, how do you differ today? Is it just because you lived through the episode or was there something that you could have received in your initial training that might have helped you to avoid your original episode? Could you help us with that?

Dr. Pyle: The way I could have avoided my original episode would have been a federal law preventing diving before the age of 30 or at least a maturity test. I was smart back then. I was probably smarter back then than I am now. In some sense literally because I think I did actually sustain some brain damage from that particular incident, but I was no dummy. I was just cocky and arrogant. Several incidences have toned down my attitude about how important it is to make sure you're alive at the end of the dive.

One of them was that incident per se. That was probably one of the biggest slaps in the face, although I wasn't exactly an angel after that incident. Of course, getting married and having a daughter and all those sorts of things tend to make one more mature and one more retrospective about making sure safety is a growing issue.

I am not where I want to be. As I was telling Dr. Bennett, my goal is to make the most dangerous part of any diving expedition the drive to the airport. If I can get to that level, that's the level I'm looking at, and then I'll buy an armored car or something to try to reduce that probability. But I'm not there yet, and I recognize that. It's taken me years to get the level of maturity just to recognize that I'm not where I want to be necessarily.

I'm not omniscient. I don't know all the answers. It takes a lot of maturity to admit that to yourself. So, coming to these workshops are valuable to me because I have these conversations with people. I listen to what people have to say. These sorts of things mold me in different directions, in different ways, and I shape my outcome.

I don't know if that addresses really the question you were after or is your question: Help us with why did I get bent in the first place?

Unidentified: Well, of course, that brings up the other question of not only why you got bent but why you survived. So I don't want to make you live through the episode again, but reading it and listening to you today, one has to come to the conclusion that you were born with something that helped you to sway movement in your favor, if I make myself clear.

Dr. Pyle: I'm extraordinarily lucky, yes, but in many more ways than diving. I try never to rely on luck, but I don't know how to answer your question.

I recognize that my attitudes and my philosophies are dynamic. They're not static. I try to have a clear idea of where I want to be and I try to have an honest idea of where they are. I try to think about ways I can go from Point A to Point B, and this is one of the forums that allow me to do that.

I'm clearly demented, if you read the Internet postings that I rant and rave about. But the ratio of positive feedback to negative feedback is so extraordinarily biased that it's sort of encouraging that I might be going in more or less the right direction. I don't say that to pat myself on the back, but that's just the reality.

I'm sure the minute Rick Lesser gives me a call and says, by the way, somebody tried your deep decompression stuff and got bent. Here are your papers, come see us in court. Then my attitudes may change yet again. I'm sure that will be another life-changing event. One of the points I wanted to make earlier about this whole liability thing, I'm very naive about the whole liability issue and in the context of in-water recompression. I hope for my own sake, my family's sake, and my dive companion's sake I remain naive about the liability issues. I don't want to let those issues cloud doing what I believe to be the safest alternative, just because a community standard which dictates the outcome of court cases hasn't caught up or, maybe community standard's right. But quite often a large body that represents a community standard has a lot of inertia that doesn't catch up with the dynamic things individuals can keep up with. I know I'm naive when it comes to liability issues, but I want to stay that way. I don't want that to confound my decision-making process.

I'm in a luxurious position of pretty much allowing that because I make my own decisions about myself. I seldom

am in a position where I make decisions for others. So, those others are seldom in a position to sue me for it. My wife's never going to sue anybody else. She already knows this. So I probably didn't answer your question, but that's the best to my understanding.

Unidentified: Let me just go back a minute to something that Richard Pyle said that I think was eminently sensible. I think there's much to be said for keeping things simple as much as possible, and irrespective of the efficacy of in-water recompression, an attempt to see what happens, a therapeutic trial of surface oxygen, I think is an eminently sensible thing to do. If after a reasonable period of time symptoms begin resolving with surface oxygen, I don't think we necessarily want to advocate immediately jumping into the water.

Dr. Edmonds: I'd also like to make two requests. If we do make any suggestions or recommendations at all, that the only way to sort of assist these cases is if you have a medical diving expert on site.

I think that's sort of illogical and impractical and really it's like saying you're not allowed to do CPR unless you're an anesthetist.

So, you're not going to have a diving expert on the site. So please don't put that into any recommendation. The second thing I would ask, and I've got no reason for asking this, except to beg you, if you're going to even suggest underwater oxygen, would you please say a maximum of 1.8 atmospheres, maximum? I don't know if anyone would disagree with that, but otherwise we're going to get people using oxygen much greater than 1.8 atmospheres.

Unidentified: Carl, could you comment on your feelings about the Hawaiian method, and do you feel—because obviously for a lot of the commercial divers, even getting them to buy oxygen—that whole set-up is going to be a little bit impractical. Would you recommend the Hawaiian method over, perhaps, no method?

Dr. Edmonds: I honestly have no experience in the Hawaiian method. So it would be stupid of me to say how good it is or how bad it is.

I can see some problems with it, but that doesn't mean it's not good. It seems sensible. It seems a good idea, but if it was me, I'd prefer the oxygen, but I really I have no experience with the Hawaiian method.

CDR Chimiak: That particular treatment profile was suggested in a NOAA-sponsored paper and was published as such. Its utilization has never been accurately measured.

The only person I know who has actually taken off on that and modified it somewhat is Richard. You saw his modification of it.

That was a published table that has never, as far as I know, had any serious follow-up on data collection on its efficacy.

Unidentified: May I nit-pick with Carl for a moment? He said don't go over 1.8 atmospheres, but 9 meters or 30 feet of sea water is 1.9 atmospheres.

Dr. Edmonds: You're right. I'm happy to switch it to 1.8. Make an offer.

Unidentified: But that points out something very intriguing, and that is that it approaches Richard's 25 feet. What happens out there where divers are diving? In my experience, it frequently precedes what is entered into textbooks and into, shall we say, peer-reviewed papers. So there's something that we're hearing here from the practicing diver. I think we should listen to it.

Dr. Kay: I want to thank our panelists for the excellent job they did, and I want to thank you all for coming today. This has been a most rewarding experience for me, and I look forward to pondering the proceedings because that's my next job, trying to put all this together in a meaningful way, trying to represent each individual in just the right light.

I don't think we're going to have any recommendations. As Dr. Moon pointed out, it's a little premature to have guidelines published, but I think we've come a long way to improving our understanding. We certainly have initiated scientific scrutiny of this subject.

I thank you all for coming.