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THE TWENTIETH UNDERSEA MEDICAL SOCIETY WORKSHOP

TREATMENT OF SERIOUS DECOMPRESSION SICKNESS AND ARTERIAL GAS EMBOLISM

DUKE UNIVERSITY

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UNDERSEA MEDICAL SOCIETY, INC.
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Chairman

Jefferson C. Davis

The Twentieth Undersea Medical Society Workshop

Undersea Medical Society, Inc.
9650 Rockville Pike
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FOREWORD

Most cases of decompression sickness (DCS) and arterial gas embolism (AGE) respond to treatment on a standard table and remain clear. To define the goals of this workshop, the following points must be made.

The response of decompression sickness and arterial gas embolism to compression treatment ranges across a spectrum:

1. Prompt, complete, and permanent clearing upon compression.

2. Prompt improvement with gradual and permanent resolution during a standard treatment table.

3. Little improvement initially on compression but gradual resolution during a standard treatment table.

4. Prompt improvement or clearing with deterioration again during treatment, usually on attempted decompression but sometimes at a stable depth or after completion of the treatment table.

5. Improvement but persisting residual manifestations.

6. Fixed neurological deficit, or shock pattern that does not respond to compression, leading to permanent deficit or death during or after chamber treatment.

Where on this spectrum a particular case lies will depend on several factors:

1. Severity and target organ affected by the process, which probably depend on the nature of the inciting event. The greater the violation of decompression procedures, the greater the volume of gas liberated from solution in body tissues in DCS. In AGE, the greater and more prolonged the pulmonary overpressure, the larger the volume of gas introduced into the arterial circulation. The importance of the organ is demonstrated by the observation that limb bends alone, with no neurological or vasomotor component, will clear well on compression even after many hours' delay in reaching the chamber, while a massive AGE with a large volume of arterial gas occluding the cerebral arteries can result in irreversible brain damage within a few minutes. (Note: This possibility must never be allowed to interfere with a trial of recompression treatment--remarkable improvement has been seen after delays of hours or even days.)

2. Interval between accident and compression therapy. In addition to relative and variable tissue ischemia produced by bubble blockage of the arterial supply or venous drainage, secondary blood-bubble interface reactions trigger a chain of events leading to hemoconcentration, tissue edema, and intravascular coagulation.

3. Adequacy of immediate care en route to the chamber, such as oxygen inhalation, and cardiopulmonary resuscitation.

4. Adequacy of treatment profile used in the chamber and the quality of the subsequent intensive care.

In regard to these points, the first three responses described in the spectrum above 1) prompt, permanent clearing; 2) prompt improvement and gradual full clearing; and 3) gradual clearing during a treatment table, are satisfactory with present methods. Response 6, permanent damage, cannot be altered and unless better planning for and administration of immediate care en route to the chamber are effective in preventing patients from reaching this point, little improvement in outcome can be expected. Hence, our focus is on the two remaining responses: 4) prompt improvement or clearing with later deterioration; and 5) improvement but with residual manifestations, both of which are unsatisfactory, and which we hope to alter significantly.

Since decompression accidents in saturation diving usually occur under conditions in which on-scene and experienced treatment support is available, the workshop participants decided to focus on difficult cases of DCS and AGE that occur after relatively shallow compressed air diving. The emphasis of this report is therefore on sport scuba divers, scientific divers, and compressed air commercial divers. Further, the workshop focused on treatment in compressed air chambers that do not have extensive life-support capability. By involving operators of outlying chamber installations that have limited budgets and personnel, the participants looked for feasible methods of upgrading treatment in such facilities. Minimum treatment gas, medical supplies, and training and staffing requirements were therefore taken into consideration in the treatment options developed.

Finally, the group recognized that improvements in the prevention and treatment of DCS and AGE must derive from continued studies. Workshop participants included clinical and basic research scientists, who described the state of the art and provided general guidelines for future studies. The papers that follow focus on the factor that most often produces difficult cases -- delayed arrival at the compression chamber. Even in those cases delayed for many hours by misdiagnosis and transit time, the standard treatment tables in use around the world resulted in satisfactory outcomes in a surprisingly constant 80 to 85% of cases. Thus, the greater achievable gain may be in the area of immediate intensive fluid therapy, resuscitation, and oxygen administration en route to the chamber. Better care during transport can be expected to improve the responses of patients to established treatment tables upon arrival at the chamber. The modern understanding of the pathophysiology of DCS and AGE urgently dictates that we remember that these are seriously ill patients who demand proper intensive care by a trained physician. It is no longer enough to simply turn a valve and

watch a clock and hope for the best. Serious DCS and AGE cases are medical emergencies that must be given comprehensive medical care en route to and in a compression chamber.

If such care is given, we will be left with fewer delayed cases that require careful consideration of the treatment options developed by the workshop members and described in the conclusion section of this report.

BACKGROUND*

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The affliction (decompression sickness) identified by Pol and Watelle (1854) and later described by Bert (1878) opened a new research area for preventive and therapeutic medicine. The decompression procedures developed by Haldane in the early 1900's greatly reduced the incidence of decompression sickness to a manageable level, but Haldane's approach and its subsequent modifications have not eliminated the problem. Because a certain percentage of persons undergoing decompression are going to be stricken with decompression sickness, an adequate therapeutic regimen will always be necessary.

The beneficial effects of recompression were recognized early. During the building of the Brooklyn Bridge, Smith (1873) had a special iron treatment lock (9 ft by 3½ ft) built. Workers stricken with decompression sickness were recompressed in this lock to a pressure equal to that at which they had been working previously; when the pain was relieved, the pressure was gradually reduced over a period of several hours.

Overwhelming evidence that recompression was the treatment of choice in handling decompression sickness was provided by Keays in 1909. But recompression treatment procedures varied depending upon who was in charge. Neither the extent of recompression nor the subsequent decompression schedule was standardized in any way. Ryan (1912) suggested that recompression should be to a pressure equivalent to two-thirds of the original working pressure. In 1917 the New York Public Service Commission adopted the policy of recompressing stricken patients to the pressure at which they had been working originally (Levy 1917). Recompression treatment procedures made little significant progress until about 1920 because of the lack of experimental data and the fragmented, non-uniform clinical evidence. In 1924 the U.S. Navy published in its Diving Manual the first standard recompression treatment procedure.

The results of treatments using the Navy's air recompression

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*Reprinted from Recompression Treatment Tables Used Throughout the World by Government and Industry, by the same authors, published by the Naval Medical Research Institute (NMRI Report 78-16), 1978.

procedure were not completely successful. Over 50% of the individuals treated suffered a recurrence of symptoms. The air treatment afforded relief in mild cases of decompression sickness, but often failed in the more serious cases.

Several early investigators had suggested, based on theoretical grounds, that the use of oxygen might be beneficial in the treatment of decompression sickness. In 1937 Behnke and Shaw conducted empirical research on the subject, and in 1944 an oxygen treatment table was promulgated in a News Letter distributed by the Bureau of Medicine and Surgery, U.S. Department of the Navy. Reports from the field and experience at the Navy Experimental Diving Unit showed that neither the new oxygen treatment table nor the air treatment table included in a U.S. Navy Diving Manual available in 1942 produced the desired therapeutic results. Recurrence of symptoms still ran about 50% of those treated. To verify field reports and to formulate adequate and comprehensive tables for the treatment of decompression sickness and air embolism, investigators at the Naval Medical Research Institute (NMRI) and the Navy Experimental Diving Unit (NEDU) performed a series of tests (Van Der Aue, White, Hayter, Brinton, Kellar, and Behnke 1945). The details and experimental results of this study will be considered in depth because of their impact on treatment procedure used here and around the world.

In these experiments, 33 Navy enlisted men served as subjects who made hard-working dives to 130 feet for 1 hour using standard U.S. Navy decompression schedules. The subjects were recompressed on the treatment table under evaluation 30 to 60 minutes after surfacing from the dive.

To allow the reader access to the actual results obtained, we quote verbatim the results from O. E. Van Der Aue, W. A. White, Jr., R. Hayter, E. S. Brinton, R. J. Kellar, and A. R. Behnke's paper, Physiological factors underlying the prevention and treatment of decompression sickness (Project X-443, Rpt. No. 1, U.S. Naval Medical Research Institute, Bethesda, Md., 26 April 1945).

> The purpose of a work dive prior to the application of the treatment table was to saturate the body tissues with nitrogen to such degree that a second exposure unless followed by prolonged decompression would be certain to produce bends. For example, following a work dive, the application of the treatment decompression outlined in the table published in the BUMED News Letter gave rise to bends in six out of ten individuals and it was necessary to recompress three of the men in order to alleviate symptoms. When the treatment table, however, was modified to include an additional hour of decompression, no symptoms developed. This illustrates the critical nature of the time factor that separates safe

treatment from treatment that is inadequate.

The failure of bends to develop following the application of the second or "treatment" decompression was, therefore, the criterion used to determine adequacy of treatment. (p. 4)

Experimental Results (pp. 5-8)

Tests of the BUMED News Letter 165-foot air-oxygen treatment table.- This table provides for the following treatment for patients whose only symptom is pain:

Depth (feet of seawater)	165	140	120	100	80	60	50	40	To surface
Time at depth (minutes)	30	12	12	12	12	30*	30*	30*	5*

*Breathing oxygen.

Ten subjects were exposed to the pressures of the table one hour after the wet dive. Three subjects (Abe, Mey, Cun) developed joint pain requiring recompression for relief after completion of the treatment table. Three subjects (Pac, Sim, Bun) had mild pain lasting fifteen to twenty minutes but recompression was not necessary to relieve the pain....* This confirmed the field reports that the 165-foot treatment table was not entirely satisfactory.

Tests of modifications of the BUMED News Letter 165-foot treatment table.- In an attempt to rectify the apparent inadequacies of the table, an additional thirty minutes of oxygen breathing was added at 30 feet according to the following table:

Depth (feet of seawater)	165	140	120	100	80	60	50	40	30	To surface
Time at depth (minutes)	30	12	12	12	12	30*	30*	30*	30*	5*

*Breathing oxygen.

One of the two divers subjected to this modified table developed joint pain requiring recompression after surfacing. ....These findings indicated that the table as modified was not satisfactory.

*References to table numbers in the quoted report have been deleted to avoid confusion with table numbers pertinent to the present report.

The addition of sixty minutes of oxygen breathing at 30 feet was required to make the table effective:

Depth (feet of seawater)	165	140	120	100	80	60	50	40	30	To surface
Time at depth (minutes)	30	12	12	12	12	30*	30*	30*	60*	5*

*Breathing oxygen.

Following the work dive three men (Kos, Kra, and Kes) developed bends within a period of one hour after decompression. To these three and the remaining eight men performing the work dive, the above outlined table of recompression was applied. Bends did not develop or recur subsequently.....There were no symptoms indicative of oxygen poisoning.

Test of 165-foot air treatment tables.- The following modification of the air treatment table of the BUMED News letter was tested:

Depth (feet of seawater)	165	140	120	100	80	60	50	40	30	20	10
Time at depth (minutes)	30	12	12	12	12	30	30	30	240	120	120

Two subjects exposed to the pressure of this table one hour after the 130-foot dive and four subjects exposed thirty minutes after the dive complained of fatigue following the test...

The table was further modified as follows:

Depth (feet of seawater)	165	140	120	100	80	60	50	40	30	20	10
Time at depth (minutes)	30	12	12	12	12	30	30	30	120	120	240

Two subjects were exposed to the pressures of this table one hour after the 130-foot dive and four subjects thirty minutes after the dive. All the subjects remained completely asymptomatic....

Tests of a 100-foot air-oxygen treatment table.- The following table, developed by Yarbrough and Behnke and Behnke, was tested:

Depth (feet of seawater)	100	80	60	50	40	To surface
Time of depth (minutes)	30	12	30*	30*	30*	5*

*Breathing oxygen.

Tests were performed under two conditions: (a) one hour after exposure to the usual 130-foot dive and (b) thirty minutes after the 130-foot dive. None of twelve subjects exposed to the pressures of this treatment table thirty minutes after the wet dive developed symptoms of caisson disease....Twelve subjects remained asymptomatic after exposure to the pressures of this treatment table one hour after the 130-foot dive....This table was considered to be satisfactory.

Tests of a 100-foot air treatment table.- The following 100-foot air treatment table, a modification of the 150-foot air treatment table of the Diving Manual, was devised:

Depth (feet of seawater)	100	80	60	50	40	30	20	10
Time at depth (minutes)	30	12	30	30	30	60	60	120

The table was tested under two conditions: (a) thirty minutes after exposure to the usual 130-foot dive and (b) one hour after the 130-foot dive. None of eight subjects had symptoms of caisson disease following exposure to the pressures of the treatment table thirty minutes after the wet dive....All three subjects were asymptomatic after exposure to the treatment table one hour after the wet dive....This table was also considered to be satisfactory.

Tests of treatment tables providing for prolonged recompression.- The following table was tested without a preceding 130-foot dive:

Depth (feet of seawater)	165	140	120	100	80	60	50	40	30	20	10
Time at depth (minutes)	120	12	12	12	12	120 22 hr.	*120	120	120	120	120

*Breathing oxygen.

Six subjects were exposed to the pressures of this table. All the subjects were extremely fatigued on surfacing. Three subjects developed substernal soreness on deep inspiration at the 60-foot depth while breathing oxygen and one who did not breathe oxygen also suffered from substernal soreness after three hours at 60 feet. Two subjects had numbness of the fingers throughout the period of oxygen breathing and one had mild nausea during the last thirty minutes of oxygen breathing. Four subjects developed joint pain after surfacing, two of whom required recompression for relief of the symptom....It appeared that the table was faulty in the following respects: too rapid decompression from 165 to 60 feet, the danger of oxygen poisoning as a result of the two-hour period of oxygen breathing at 60 feet, prolonged breathing of dense air at 60 feet, and too rapid decompression from 60 feet to the surface.

The table was modified as follows:

Depth (feet of seawater)	165	140	120	100	80	60	50	40	30	20	10
Time at depth (minutes)	120	30	30	30	30	6 hr.	6 hr.	6 hr.	12 hr.	120	120

Six subjects were exposed to the pressures of this table without a previous wet dive. None developed joint pain, paresthesias, substernal soreness or nausea, but all were moderately fatigued after surfacing. Two subjects developed moderate frontal headache, one at the 20-foot depth and the other six hours after surfacing....

Tests of a decompression table for tenders.- The following table was tested:

Depth (feet of seawater)	165	140	120	100	80	60	50	40	30	To surface
Time at depth (minutes)	30	12	12	12	12	30	30	30	60*	5*

*Breathing oxygen.

Ten subjects were exposed to the pressures of this table without a preceding wet dive. None of the subjects developed symptoms of caisson disease....

The results of the Van Der Aue et al. (1945) study provided the treatment tables that were used by the U.S. Navy and many foreign countries until about 1965. It is interesting to note that these widely accepted treatment procedures are based upon a study involving only 33

subjects and that some of the individual treatment tables are based on as few as 6 subjects. Another interesting point is that in most cases the treated subjects did not actually have manifest symptoms of decompression sickness prior to the therapeutic recompression. In several instances they were symptom-free prior to recompression-experienced decompression sickness during the treatment. The treatment tables were extended until all subjects could tolerate both the initial pressure exposure and the therapeutic recompression without manifesting symptoms of decompression sickness.

This study and the resulting recompression treatment tables stood as the U.S. Navy's treatment procedure for the next 20 years. During this time these tables, or slight modifications of them, were adopted by several foreign navies and numerous foreign and domestic commercial companies. As evidence accumulated it became apparent that the success rate for the more severe cases of decompression sickness was considerably lower than desired. Rivera (1964) published a statistical evaluation of the treatment tables, which showed the following success rates following an initial recompression and after repeated treatments.

Treatment table	Success rate following first recompression (%)	Final success rate (%)
1	92	100
1A	86	98
2	91	99
2A	90	99
3(O_2)	93	96
3(Air)	80	90
4(Air)	55	61
4(O_2)	42	42
4(He-O_2)	33	42

Failure rates for the initial recompression and lack of success in treating severe cases of decompression sickness led Goodman and Workman (1965) to the development of the minimal-recompression oxygen treatment table. Often called simply "the oxygen treatment table," it has been widely used throughout the world. It is still the treatment of choice.

W O R K S H O P

C O N T R I B U T I O N S

TREATMENT OF DECOMPRESSION ACCIDENTS AMONG SPORT SCUBA DIVERS WITH DELAY BETWEEN ONSET AND COMPRESSION

Jefferson C. Davis, M.D.

Studies at the U.S. Air Force School of Aerospace Medicine between 1962 and 1965 demonstrated that the etiology and pathophysiology of altitude decompression sickness are essentially the same as those in stricken divers. With the assistance of Workman and Goodman, minimal pressure-oxygen treatment tables were found to be especially effective in treating altitude decompression sickness. The USAF installed eight compressed air chambers, man-rated to 225 fsw and fitted with a built-in breathing system (BIBS), at various locations around the world to supplement naval and civilian chambers for the treatment of aviators. An initial cadre of flight surgeons (all graduates of the 9-week basic course in Aerospace Medicine or the 3-year Residency in Aerospace Medicine), aerospace physiologists (with advanced degrees in physiology), and technicians attended 2-week courses in the recognition and treatment of diving casualties at the U.S.N. School of Diving and Salvage.

For all USAF flight surgeons attending the basic flight surgeon's course, heavy emphasis was placed on recognition, immediate care, and referral of decompression accidents. Those graduates of the 9-week basic flight surgeon's course who were destined for bases that had compression chambers remained at the School of Aerospace Medicine for a 2-week course on compression therapy. Telephone consultation with physicians at the School of Aerospace Medicine was required on each case treated in USAF chambers. In all difficult cases, especially diving cases, the medical team at the School of Aerospace Medicine also consulted its mentors in the U.S. Navy. Between 1965 and 1979, over 200 cases of altitude decompression sickness were successfully treated in USAF chambers according to USN Treatment Tables 5 and 6.

DISCUSSION OF CASES

During the same period (1965-1979), 67 sport scuba divers suffering decompression sickness or arterial gas embolism were also treated in USAF compression chambers. Civilian compression chambers were provided telephone assistance on request; among approximately 200 such requests, our records are complete enough to include 25 cases in this report. Though this series of 92 cases is small, it may be important for comparison. Except for cases where erroneous treatment tables were used before we were called, the rules of Tables 5, 5A (before deletion), 6, and 6A were followed in all cases.

This paper summarizes the treatment experience gained in treating 92 cases of decompression sickness (DCS) and arterial gas embolism (AGE)

among sport scuba divers. There were 67 cases of DCS, and 25 of AGE resulting from pulmonary overpressure accidents in scuba diving. The two distinguishing features of this series are the long delays between onset of clinical manifestations and start of compression therapy, and the variation in diving habits and training among these sport scuba divers.

Decompression Sickness Cases

Among the 35 cases of limb bends treated, 77% arrived at the chamber more than six hours after onset. This is twice the percentage (38%) of civilian divers delayed more than six hours reported in the Rivera study. The average delay for our cases was 18 hours.

TABLE 1

Treatment results: limb bends (35 cases)

Table Used	No. Cases	Clear After 1 Treatment	Recurred; 2 Treatments Required
5	7	7	0
6	28	26	2
Total	35	33	2

NOTE: Mean time, onset to start of recompression: 18 hours; range, 2 - 85 hours.

Even with this delay, responses to Tables 5 and 6 were good; repeated tables were required in only two cases. At present, we use Table 6 for any case delayed in reaching the chamber, regardless of how rapidly relief is achieved at 60 fsw. The only episode of oxygen poisoning in this group was one patient who convulsed 18 minutes into the first oxygen breathing period at 60 fsw. The only adjunctive therapy was intravenous fluid therapy, used in two cases.

Table 2 is a summary of the treatment and results in 32 cases of neurological decompression sickness.

Table 2

Treatment results: neurological decompression sickness
(32 cases: 29 spinal cord, 3 brain)

Table Used	No. Cases	Clear After 1 Treatment	Clear With Repeated Treatments	Permanent Residua
5 (in error)	3	1	1	1
6 (with extensions)	21	13	5	3
5A	1	0	1	0
6A	6	4	1	1
4	1	0	0	1
Total	32	18	8	6

NOTE: Mean time, onset to start of recompression: 16 hours; range, ½ - 48 hours. (Only 3 cases had delays of less than 4 hours.)

Three of these cases had brain manifestations and 29 had spinal cord involvement and, except for one case in which treatment began 30 minutes after onset, all were delayed -- only three of the 32 cases arrived in less than four hours. A 60-foot oxygen table was used initially in 24 cases, with ultimate resolution in 21 cases; however, several patients required repeated treatment. Treatment of eight cases began at 6 ATA and was followed by ultimate clearing in six cases. Four of the six cases treated on Table 6A (the gas embolism table) cleared permanently after only one treatment. Of the two recurrences after treatment on Table 6A, one was successfully treated on a repeated Table 6A and then on a series of daily modified Table 5's (three 20-minute oxygen periods at 60 fsw). Another patient remained paralyzed despite treatment on daily Table 5's. One patient treated on Table 5A demonstrated the pattern dictating that choice of table (symptoms cleared in less than 10 minutes at 165 fsw) but had a severe recurrence after treatment and was successfully treated on Table 6A and daily Table 5's for six days. We discontinued the use of Table 5A three years ago.

The spinal cord case treated initially on Table 4 was further complicated by attempted treatment with compressed air in the water -- one 72 ft^3 scuba tank had been given at 30 fsw and one at 100 fsw, before the diver was delivered paralyzed to a civilian chamber. This happened at a remote island chamber several years ago, and was the chamber's first spinal cord case; the team at the chamber had not yet learned of oxygen treatment tables. We were called when there had been no relief by 30 fsw on Table 4, and at that point the patient was returned to 60 fsw and treated on an extended Table 6, with no response. Absent anal sphincter tone, absent bulbocavernosus reflex, and an atonic urinary bladder with motor paralysis persisted, but cutaneous sensation

returned during the next 14 days of treatment with modified Table 5's. The patient was sent to a rehabilitation unit, where significant return occurred over the next six months including anal sphincter tone, bladder function, and the ability to walk (albeit with a spastic gait and using canes). This case is counted as a treatment failure.

Despite an average delay of 16 hours before compression therapy, 13 of 21 cases treated on Table 6 recovered fully with one treatment. In the remaining eight patients treated initially on Table 6, return of function was either incomplete or had deteriorated after the first treatment. Of these, five patients responded fully to repeated treatment with Tables 6, 6A, or daily Table 5's. Three patients failed to respond and remained paralyzed despite repeated treatments. Table 5 has never been recommended for anything other than limb bends that clear in less than 10 minutes at 60 fsw, but it was used erroneously in three of these cases. One of the three, a patient with brain manifestations, did remain clear but the other two required re-treatment and one had some residual sensory impairment even after repeated treatment.

Two of these neurological cases demonstrated some degree of pulmonary oxygen poisoning during the course of Table 6's and repeated Table 5's, but the poisoning was limited to subjective burning and irritation and cleared completely as soon as oxygen was discontinued. We used adjunctive dexamethasone and one of the detrans in almost all cases. Dexamethasone requires 12 hours for maximum effect in CNS edema, so we recommended that it be given i.v., en route to the chamber or as soon as possible thereafter.

Arterial Gas Embolism Cases

Table 3 summarizes 25 cases of arterial gas embolism.

Table 3

Treatment results: arterial gas embolism
(25 cases)

Table Used	No. Cases	Clear After 1 Treatment	Recurred: Cleared With Repeated Treatment	Died
5A	4	3	1	0
6A	17	11	2	4
4 to 60 fsw then 6	1	1	0	0
6 (in error)	3	2	1	0
Total	25	17	4	4

NOTE: Mean time, onset to start of recompression: 4 hours; range, 10 min to 12 hours. (In only 3 cases was the delay less than 2 hours.)

The long delay before arrival at the chamber is the most significant factor in this AGE series, and it resulted in the four fatalities, each of whom had suffered irreversible brain damage before reaching the chamber. In only three cases could treatment begin in less than two hours after the incident. The mean time from onset to chamber treatment was four hours. The 12-hour-delay case was one of the four fatalities. Three of our less serious cases were mistakenly treated at only 60 fsw. Two cleared fully with one Table 6, but one required a repeated Table 6 to control recurrent seizure activity. Of the 22 cases treated at 165 fsw, 18 ultimately cleared fully, although some required repeated treatments. Of the four patients who died, two died in the chamber and two died in the hospital after a Table 6A or a Table 4. Of the four fatalities, none showed any response at any depth, despite two hours at 165 fsw in one case.

Of the 17 cases treated on Table 6A, 11 (65%) responded with permanent clearing. Of these, only one began compression therapy within two hours of onset, and the mean delay for these 11 cases was three hours, with a range from five minutes (one case) to the longest, seven hours. Two cases had recurrent neurological manifestations after responding initially to Table 6A; these then cleared after repeated daily Table 5's. Three of the four fatal cases (all treated on Table 6A) arrived at the chamber six hours after the accident, and one was delayed 12 hours. Upon arrival all four were unresponsive to pain, three were intubated and required mechanical ventilation, and one patient was having continuous major seizure activity.

Of the 18 survivors, 12 cases (66%) cleared completely during the initial stay at 165 fsw on Tables 6A, 5A, or 4. We no longer use Table 5A, but four early cases were treated on this table. Two of the three cases that responded well showed significant improvement by the time of arrival at the chamber, where they were treated for their history and for very minimal subjective symptoms. The third patient regained consciousness at 165 fsw and stayed clear after treatment on Table 5A. The recurrent case arrived at the chamber hemiplegic eight hours after the accident. He responded fully at 165 fsw and stayed clear until he arrived at the surface on a Table 5A, where his hemiplegia returned. He responded fully to a repeated Table 6. One case responded at 165 fsw in a portable compressed-air chamber; in this case, Table 4 was followed to 60 fsw during transport of the portable chamber to a large chamber facility. At 60 fsw, oxygen breathing began and there was full recovery on Table 6, with treatment extensions at 60 fsw and 30 fsw.

Table 6 was erroneously used initially in three cases. Two of these cases had improved significantly upon arrival at the chamber and then cleared fully on Table 6. The patient with symptoms that recurred after treatment on Table 6 had been delayed for nine hours before reaching the chamber. During that time, he had required cardiopulmonary resuscitation for cardiac and respiratory arrest at the surface after emergency ascent from 60 fsw. This ascent followed a deep dive to 200 fsw. Water

recompression using scuba air had been attempted unsuccessfully. At the chamber nine hours later, the patient showed primarily spinal cord decompression sickness symptoms with marked weakness of the lower extremities. He improved on the initial Table 6 but had returned to his original status four hours after the treatment. After three daily Table 5's, his only residual symptom was spotty sensory loss over the left lower extremity. Central nervous system oxygen toxicity, manifested by nausea, occurred twice during the series but responded to air breaks and continuation of Table 6A. Intravenous dexamethasone and dextran 40 or 70 were used routinely.

Another previously unreported case not included in this series but pertinent to the discussions of this workshop concerns aircraft transport of these patients. The importance of maintaining sea-level cabin pressurization during the transport of decompression sickness and gas embolism patients is demonstrated by the following case; the only records available from this 1963 case are from telephone memos. A young male civilian sport scuba diver was at 100 fsw for about 30 minutes and surfaced directly after a dive near Puerto Rico. Five minutes after surfacing he complained of chest pain and dyspnea and by 10 minutes after surfacing he had weakness progressing to paralysis of the lower extremities. He was flown to Cuba in an unpressurized DC-3. The exact flight altitude is not recorded but was said to be about 5,000 feet above sea level. He arrived five hours after onset and died two and one-half hours into a treatment table (presumably Table 4). Details of his death are not known, but chokes and decompression sickness shock, exacerbated by altitude exposure, may be assumed.

SUMMARY

Of 92 treated cases of decompression sickness and gas embolism among sport scuba divers, there were five cases with permanent impairment and four fatalities. This amounts to an overall 10% failure rate for cases with long delays between onset and treatment, and this figure may not be reducible, but we must continue efforts to improve on it. Nagging questions remain:

1. Would more aggressive drug and fluid therapy as first aid on the way to the chamber have helped?
2. Would the use of enriched oxygen mixtures at 6 ATA or deeper have helped?
3. Would more aggressive intensive care in the chamber during treatment have helped?
4. At what point should nitrox saturation have been used and how should patients who need it be selected?

5. Some patients surely have permanent irreversible brain or spinal cord damage upon arrival at a chamber. At what point should the medical team give up and stop treatment?

6. What is the relative contribution, in patients who respond poorly, of persistent bubbles, edema, intravascular coagulation, and small vessel hemorrhage into tissue?

7. Are repeated hyperbaric oxygen treatments really of value?

TREATMENT OF AIR DECOMPRESSION ILLNESS IN THE ROYAL NAVY

Surgeon Commander David R. Leitch, Royal Navy

CURRENT STATE OF THINKING

The last three years have seen an evolution in the philosophy of treating dysbarism that began at the European Undersea Biomedical Society workshop on treatment in 1975. The RN has changed its policy under the influence of its underwater medicine specialists, but those doctors away from the center of diving activity have continued to be bound by the existing rules of the *Diving Manual*. This period should soon be ended by the publication of the revised treatment section of the *Manual*. The outline of treatments presented in this paper agrees with what will soon become standard RN practice.

The objectives of the *Manual's* revision of treatment procedures are:

1. To emphasize using oxygen at 18 m to such an extent that using air would be exceptional.
2. To provide easy-to-follow guidelines for users.
3. To incorporate the experience gained in more difficult cases over the last three years.
4. To adopt USN Table 6A as the preferred treatment of arterial gas embolism (AGE).
5. To try to avoid protracted and difficult air therapy.
6. To provide the user with written directions that may be used in difficult cases when communications are unsatisfactory.

THE TABLES TO BE USED

The only new tables to be included are USN Table 6A (RN 63) and the RN submarine escape training tank table now designated Table 73, that is, Table 54 (T 54) to 18 m, followed by a bleed-type decompression that is actually carried out in 1-m increments. The tables (Table 1) are presented in three series, 50 (air), 60 (O_2 primarily), and 70 (air bleed decompression).

The air tables of the 50 series are modified USN air tables. They differ from the USN tables in the following ways:

TABLE 1

ROYAL NAVY TREATMENT TABLES

Table	USN Equivalent	Comment
51	1A	Deleted from treatment options.
52	2A	a) Descent not included in elapsed time. b) Travel between stops is 5 min not 1 min (Applies to T 51 - T 55) c) 3 m stop is only 2 hr not 4 hr.
53	3	See T 52.
54	4	See T 52. Time on the bottom is a mandatory 2 hr. not 1/2-2 hr.
55	4	See T 54. No O_2 is used so that stops become: 9 m for 12 hr; 6 m for 4 hr; 3 m for 4 hr.
61	5	Same table - same rules.
62	6	Same table - same rules.
63	6A	Same table - same rules.
71	none	T 71 and T 72 are options to T 54 and T 55. They are bleed decompressions. T 71 allows 30 min at 70 m. O_2 may be administered where appropriate.
72	none	Allows 2 hr at 50 m and decompresses from there at the same rate at T 71.
73	none	Allows 2 hr at 50 m and is a bleed profile broken into 1-m steps from 18 m to surface. It is the preferred table for AGE and incomplete cures of serious DCS. The profile is essentially that of T 71.

NOTE: 1) Air treatments all have 5 min between stops. The time is not included in the stops but is included in the elapsed time; 2) O_2 tables of the 60 series have ascent rates of 3 m in 10 min from 18 m to 9 m to 0 m; 3) The bleed decompression in the 70 series is based on the absolute pressure ratio of 1.3:1 in 5 hr.

a. Descent time is not included in the running time.

b. Ascents between stops take 5 rather than 1 min.

c. There are a number of individual table differences.

(i) Table 51 has been removed from the available tables so that all air tables now start at 50 m.

(ii) Table 52 has only a 2-hr rather than 4-hr stop at 3 m.

(iii) Table 54 has a mandatory bottom time of 2 hours, not a bottom time range of ½ to 2 hours.

(iv) Table 55 is used when oxygen is not available; it is T 54, but has stops of 12 hours at 9 m, 4 hours at 6 m, and 4 hours at 3 m.

The tables of the 60 series are the same and are used in the same way as USN Tables 5, 6, and 6A. Table 63 (USN 6A) is used exclusively for AGE.

The tables of the 70 series now also include T 73, for use in difficult treatments of AGE. Any of the three tables may also be used in difficult cases to complete long air therapies when large stages are feared.

ADDITIONAL TREATMENT

a. Oxygen: The option of using additional oxygen is available in Table 62 and in any of the long air tables. Oxygen-rich mixtures may also be used as appropriate.

b. Steroids: Steroids are prescribed in most case of AGE in the form of dexamethasone, 12 mg i.v. immediately, followed by 8 mg in 6 hours, continued at 6-hour intervals for 48 hours. There is a move afoot to increase the steroid dose to 2 g hydrocortisone equivalent immediately, 1 g in 3 hours, and 1 g in 6 hours, followed by a tapering off over 48 hours. This dosage is as yet speculative. Steroids have also been used in cases of serious DCS.

c. Dextran: Dextran, up to two units in 48 hours, is used in difficult cases of serious DCS.

d. Follow-up oxygen: One hour daily of oxygen (PO_2 = 2 bar) until no improvement occurs as a result of the treatment.

GUIDELINES FOR TREATMENT

Cases are grouped into two categories, based on severity rather than type of illness (Fig. 1):

1. Life-threatening or definite AGE of less than five hours' duration.
2. Not life-threatening.

A rider or third group qualifies these two: if the delay between onset and compression exceeds five hours, oxygen at 18 m is to be used. For cases in categories 1 and 2 above, the guidelines shown in Fig. 1 are straightforward. Those cases that are the object of this workshop lie in the right-hand end box of the figure.

The instructions with the tables outline the actions required in cases involving relapses, blow-ups, or missed decompressions.

Experience with the Treatments

This paper presents results from 46 reasonably complete cases of air divers with DCS or AGE treated in the context of the RN guidelines during the last three years. Not all the cases were treated by the RN at first hand. Many of the problems had occurred by the time RN help was sought in the difficult cases.

The number of cases is too small to allow firm conclusions, but some expected trends are present and some insights available. The types of cases are shown in Table 2, and the onset times and delays in treatment are shown in Table 3. Table 4 gives an interpretation according to the new rules, of the validity of the treatments given and also notes other pertinent facts. Five patients made spontaneous recoveries to the point of absence of clinical signs; three were treated and two were not treated; of these, one later relapsed and required treatment. Six cases arrived at the surface unable to walk, and all were subsequently able to walk unaided.

Arterial gas embolism (Table 5). Of the 14 cases, the only two that had not recovered by the 25-minute point were one patient whose symptoms were 29 hours old, and one case that had been wrongly treated, ending up on an N_2-O_2 saturation treatment table.

The five patients treated on T 63 surfaced without problem, as did the four treated on T 73. It is possible that these cases could have been treated on T 63, but under the prevailing rules they were treated as described. Half of the cases received steroids. Four cases were incorrectly treated, but only one of these subsequently developed problems.

ROYAL NAVY
1979

FIGURE 1

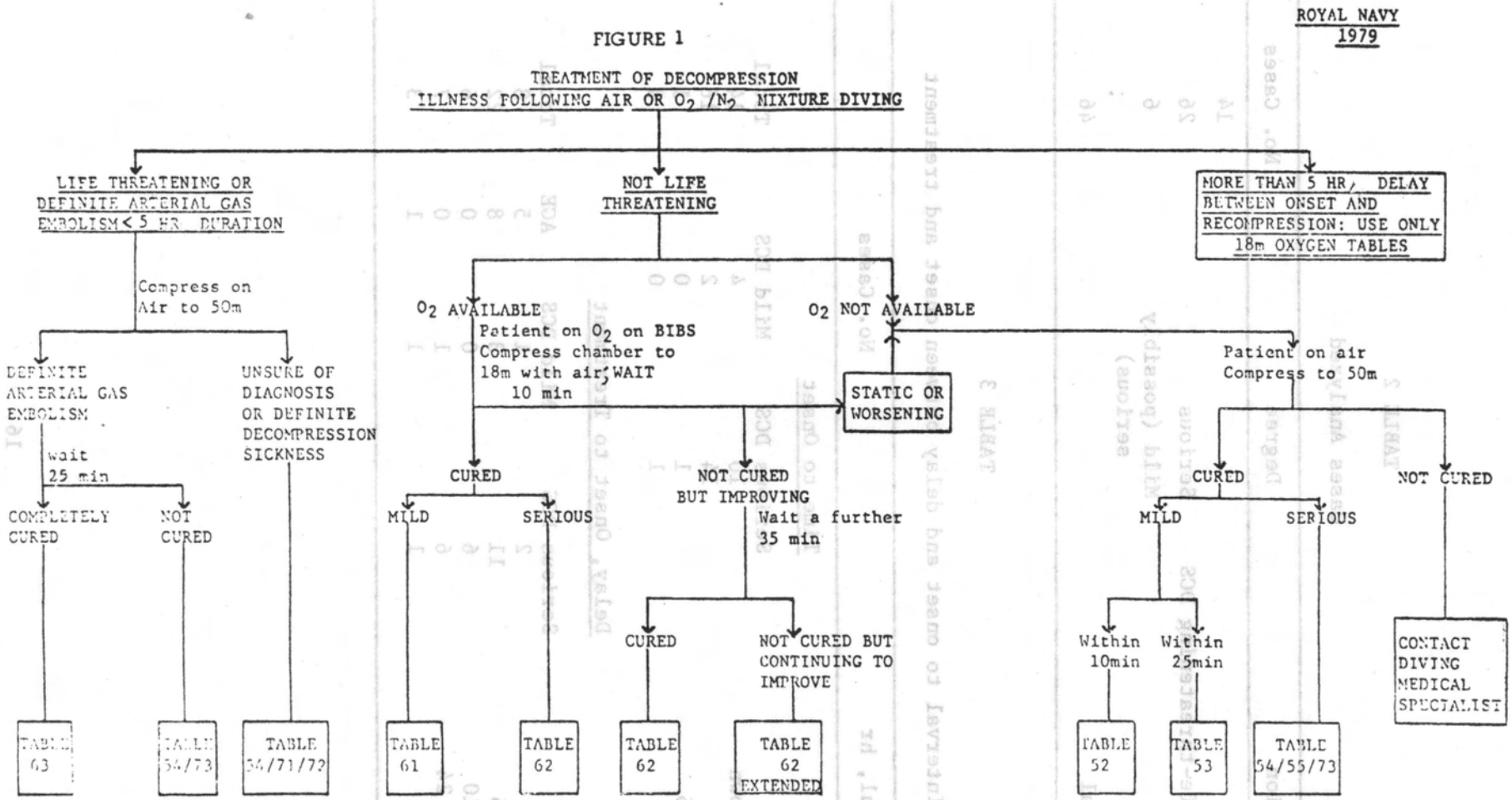

TABLE 2

Cases Analyzed

Condition	Degree	No. Cases
AGE		14
Non-life-threatening DCS	Serious	26
	Mild (possibly serious)	6
Total		46

TABLE 3

Interval to onset and delay between onset and treatment

Interval, hr	No. Cases			
	Time to Onset			
	Serious DCS	Mild DCS		Total
Not known	10	4		14
1	14	2		16
1 - 6	1	0		1
>6	1	0		1
	Delay, Onset to Treatment			
	Serious DCS	Mild DCS	AGE	Total
1	2	1	5	8
1 - 5	11	3	8	22
5 - 10	6	0	0	6
10 - 24	6	1	0	7
>24	1	1	1	3

TABLE 4

Interpretation within rules

Description of Treatment	No. Cases
Correctly treated throughout	37
Wrong treatment at some phase	9
Wrong treatment begun	6
Wrong treatment later followed	4
Not treated at all	1
Spontaneous initial recovery (no signs), AGE	3
(1 treated, 1 relapsed) DCS	2
Complications from wrong action	5
Surfaced with signs	3
Relapse during treatment	3

TABLE 5

Treatment of arterial gas embolism

Treatment	No. Cases	Cured by 25 min	Cured at Surface	Adjuvant Therapy	Delay >5 hr
Correct	10	9	9	7	1
T 63*	5	5	5	2	-
T 54**	1	0	0	1	1
T 73	4	4	4	4	-
Incorrect	4	3	3	1	-
No R_x***	1	-	-	-	-
T 53	1	1	1	-	-
T 62	1	1	1	-	-
T 61****	1	0	0	1	-

*Includes one patient left with minimal symptoms by start of treatment; **29-hour delay in treatment, extra O_2 at 9 m, surfaced with severe signs; ***had mild headache for some hours after recovery of sight; ****this case was reported in Miller et al., Lancet, 22 July, 169-171, 1978.

Oxygen and minimal pressure treatment of DCS (Table 6). Twenty-seven cases are presented, of which three were wrongly treated initially; of these, two were cured anyway. An additional case that spontaneously recovered required later treatment and another similar case was also treated.

Six of the 27 cases were essentially mild DCS and were successfully treated as such on T 61. These patients all had symptoms that might have suggested a more sinister problem, e.g., headache, bilateral costal margin pain, back pain, and subjective sensory changes.

The 21 serious cases responded as follows: cure by 10 min, 43% (n=9); cure by 45 min, 57% (n=12); cure at surfacing, 67% (n=14); required air treatment for severe relapse, 1 case; surfaced with mild symptoms, 4 cases; surfaced with signs, 2 cases; and surfaced on air with mild symptoms after an oxygen hit, 1 case.

Of 10 cases not cured by 45 minutes, only four received extra oxygen before surfacing; two that did not receive extra oxygen could not receive it, which left four cases. These compare at the surface as follows:

Extra O_2: cured 1; mild symptoms, 2; signs, 1.
Straight T 62: cured, 3; mild symptoms, 1.

Only in five difficult cases was dextran and/or dexamethasone used.

50-m Air treatment of DCS (Table 7). This table was only the correct primary treatment in four of the six cases; the other two consisted of a case wrongly begun at 30 m and another resulting from a patient who relapsed severely on T 62. It can only be said that none of the treatments was straightforward, but nevertheless, of the four initial treatments, three surfaced completely cured.

The effect of delays exceeding 5 hours on the correct O_2 therapy of serious DCS (Tables 8 and 9). As one would expect, delay results in a longer time to achieve a cure and a reduction in the number of cures. With delays under 5 hours, 80% were cured by arrival at the surface, but in cases in which delay exceeded 5 hours, this rate was only 50%. There were also more residual signs and symptoms in this latter group.

The use of repeated O_2 therapy for cases with residual signs. Six patients completed the initial therapy with residual signs; four of these received additional oxygen over a period of days, once a day at a PO_2 of 2 bar for one hour, followed by 30 minutes of decompression on oxygen. This treatment was continued only as long as clinical improvement was observed with each treatment. Both of the untreated

TABLE 6

Results of treatment of decompression sickness with O_2 as first option

Condition	Cases	Cure by 10 min	Cure by 45 min	Cure at Surfacing	Symptoms at Surface	Signs at Surface	Adjuvant Therapy	Relapse	Air Follow-up	Repeated O_2 Therapy
	27	14	18(4)	20(4)(-2)	24(4)	27(2)(+1)	5	4(+1)	1	2
Mild DCS T 61	6	5	6(1)	6(0)(0)	6 (0)	-	-	-	-	-
Serious DCS	21	9	12(3)	14(4)(-2)	18 (4)	21(2)(+1)	5	4(+1)	1	2
No R_x	1(-1)	0	0(0)	1(1)(0)	-	-	-	-	-	-
T 61	2(-1)	2	2(0)	1(0)(-1)	-	-	-	1	-	-
T 62	14(+2)	7	10(3)	12(3)(-1)	14 (2)	16(1)(+1)	3	2	1	1
T 62+O_2	4	0	0(0)	1(1)(0)	3 (2)	4(1)	2	-	-	1

Figures in brackets without signs indicate new cases in the column; figures in brackets with +/- signs indicate a shift from or to another treatment.

TABLE 7

Results of treatment of decompression sickness with air

	Cases	Cure by 25 min	Relapse	Extra O_2	Cure at Surfacing	Symptoms at Surface	Signs at Surface	Adjuvant R_x	Repeated O_2
First treatment	4	2	3	2	3	1	0	2	0
Second treatment	1	0	0	1	0	0	1	1	0
First at wrong depth	1 (went to complicated T 55/73 therapy)	0	1	0	0	0	1	1	1

TABLE 8

Effect of treatment delay on course of therapy (serious DCS only)

Treatment	No. Cases	Cured by 10 min	Cured by 45 min	Cured at Surfacing	Symptoms at Surface	Signs at Surface	Adjuvant R_x	Relapse
				Delay <5 hr				
O_2 first	10	5(5)	8(3)	8(0)	9(1)	10(1)	1	2
T 61	1	1(1)	1(0)	1(0)	1(0)	1(0)	-	-
T 62	8	4(4)	7(3)	7(0)	7(0)	8(1)	-	1 to air
T 62 + O_2	1	0(0)	0(0)	0(0)	1(1)	0(0)	1	-
Air only	3 (1 from O_2 relapse; 1 from wrong air treatment; 1 surfaced with symptoms after all additional treatment)							
				Delay >5 hr				
O_2 first	11	3(3)	3(0)	6(3)	9(3)	11(2)	3	2
T 61	1	1(1)	1(0)	0(-1)	-	-	-	1
T 62	7(+1)	2(2)	2(0)	5(3)	7(2)	8(1)	2	1
T 62 + O_2	3	0(0)	0(0)	1(1)	2(1)	3(1)	1	0
Air first, T 54/55	3 (2 cured by 25 min; 3(1) at surfacing; 1 received extra O_2, 1 received adjuvant R_x, and 2 had relapses)							

TABLE 9

Success in correctly conducted O_2 treatments related to delay between onset and treatment of serious DCS

Delay Before Treatment	No. Cases Treated	Accumulative Cure By 10 min	By 45 min	At Surfacing	Surfaced with Symptoms	Surfaced with Signs
<5 hr	9	44	78	78	11	11
>5 hr	10	20	20	50	30	20

Values are percentages.

cases walked unaided again, one within 2 weeks and one in 3 months. The relation between number of oxygen treatments and time to recovery is shown in the table below.

No. of Oxygen Treatments	Time to Unaided Walking
4	not known
9	5 weeks
3	6 weeks
4	8 months

No conclusion can be drawn from these figures; they show no apparent difference either in recovery time or degree of recovery between those treated and those untreated.

CONCLUSION

1. The sample of cases presented in this paper is heavily and deliberately biased toward problem cases because they were the focus of this workshop. All the real problems occurred outside the sphere and control of the Royal Navy.

2. Evidence from a small number of cases cannot be totally relied on, but some tentative conclusions may be drawn.

   a. Tables 63 and 73 appear to be equally satisfactory for the treatment of AGE. Three of the four cases treated on T 73 were the result of submarine escape training tank accidents.

   b. The oxygen tables, even properly used, appeared at first to be less than adequate to treat serious DCS. The cure rate at arrival at the surface was 67%, but adding those cases with only minimal symptoms but no signs brings this rate up to 90%. When this rate is compared with that of Rivera (1964) and Slark (1962) and the results for air treatments, this outcome is favorable. When compared with the 65% success rate for oxygen treatments (Bornmann 1967), the results are as good.

   c. No comment can be offered on the efficacy of extending the oxygen sessions of T 62, beyond saying that by the time of surfacing the results were similar to those obtained by using T 62 alone. Since the cases not cured by 45 minutes demonstrate reasons enough to use extended oxygen, it must be assumed that where extra oxygen was given the cases were doing less well and that therefore extra oxygen was efficaceous.

d. When air was used, even correctly, none of the treatments was straightforward, although all four patients given the correct air therapy as a first resort surfaced free of clinical signs. The remaining two patients reflected their difficult histories, i.e., one wrong treatment and one severe relapse from a T 62.

e. As the reports referred to show, delay in treatment is detrimental to outcome, but there is no evidence that limiting cases involving delays of more than 5 hours to oxygen therapies is wrong; the cure rates at surfacing were 80 and 50%, respectively, and the success rate for removing clinical signs was 90 and 80%, respectively.

f. Using steroids to treat AGE has almost become mandatory, but using steroids and dextran in serious DCS still remains a measure of last resort. No evidence is available about the relative efficacy of these treatments in this series.

g. Of the six patients who were unable to walk at the conclusion of their initial treatments, all eventually walked unaided. Four received additional oxygen therapy, as described earlier. There is no doubt that these therapies did hasten improvement, because improvement was seen during the therapy, but it may be that this improvement was only hastened by a matter of days. We are also aware of a case in which no improvement was produced by the administration of late oxygen. We have no evidence whether or not oxygen actually improves the result, since the criterion of unaided walking is a very crude measure of function.

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U.S.N. TREATMENT METHODS

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The U.S. Navy records about 70,000 dives annually, of which about half are working dives. Training, requalification, equipment testing, recreation, and experimental dives account for the other half of the dives. The number of major accidents is approximately 70 annually; this is an accident rate of less than one-tenth of one percent. The number of decompression sickness cases in the U.S. Navy diving community is 40 to 50 annually; half of these are from experimental dives. Gas embolization occurs about twice annually, and there is about one fatality a year. The emphasis is on prevention of diving accidents. This Navy experience must be considered when one examines the method of treatment for diving accidents in the U.S. Navy.

Tom Berghage reminds us that the majority of Navy dives are performed in water less than 60 feet deep. Open-circuit scuba dives make up one-half of the annual dives logged. The U.S. Navy allows only no-decompression diving with open-circuit scuba. In the Navy, the diving apparatus and the diving procedure utilized are governed by the work to be performed and the depth. For example, most Navy diving is for ship husbandry, recreation, or combat swims. Salvage dives using air or deep salvage work using helium-oxygen are less common. The Navy does no very deep helium-oxygen bounce diving and has no pressurized bell and deck decompression chamber system capable of bounce diving. The decompression tables are made specifically for the underwater breathing apparatus and the work to be performed. The dive station organization and the number of people on station are set by instruction. The level of training is appropriate for the diving apparatus and the work to be performed.

If the dive is to be deeper than 170 fsw, a recompression chamber is always on scene. Recompression chambers are of a fairly standard configuration, are always capable of compression to at least 200 fsw, and have air of certain capacity and quality. Oxygen must be available by mask. Each station has trained operators on call and hospital corpsman diving technicians are usually available at all localities. When diving deeper than 170 fsw, a medical officer must always be available and he is always a graduate of the 8-week course at the Naval School of Diving and Salvage. Diving procedures are quite detailed in Navy diving manuals and are generally closely followed by all Navy diving activities.

It should be remembered that the U.S. Navy *Diving Manual* is for the use of this particular organization with its special diving procedures. The sections on diving emergencies are written for U.S. Navy divers, diving in U.S. Navy circumstances.

The U.S. Navy does treat approximately 150 non-U.S. Navy divers annually, so many commands are not short of experience with very difficult cases.

TREATMENT OF DECOMPRESSION SICKNESS AND GAS EMBOLISM

Guidance for treatment of decompression sickness and gas embolism can be found in the Navy manuals. The guidelines are written as if everyone were using a U.S. Navy double-lock recompression chamber with oxygen by mask. Readers of the manuals are instructed to take action in each case, in order of priority as follows: first aid, preliminary examination, diagnosis, compression, and detailed examination.

Compression is not delayed for detailed examination if the diagnosis of serious decompression sickness or gas embolism is probable. If no differentiation can be made between the diagnosis of serious decompression sickness and gas embolism, the diver is treated for gas embolization.

Signs and symptoms of the three diving illnesses treated by recompression are decompression sickness, pain only; decompression sickness, serious symptoms; and gas embolism. Treatment is initiated according to the working diagnosis. Flow charts are given to aid in the choice of proper treatment procedures.

Treatment Tables 5, 6, and 6A are used when oxygen is available in the treatment chamber. Table 4 may be utilized in serious decompression sickness cases that have worsening symptoms during the first 20-minute oxygen breathing period at 60 ft and when there is a need for deeper compression. Tables 1A, 2A, 3, and 4 are less effective than Tables 5, 6, and 6A and are for use only when no oxygen is available in the chamber. The exception is the special use of Table 4 noted in the preceding paragraph.

The patient is observed for recurrence of symptoms for six hours after treatment.

After the completion of the treatment table and after a surface interval sufficient to allow complete medical evaluation, hyperbaric oxygenation Treatment Table 5 may be warranted in cases with residual damage to the central nervous system or the organs of special sense, if recommended by appropriate medical consultants.

Emergency consultation is available on 24-hour call at the Navy Experimental Diving Unit and at the Naval Medical Research Institute.

Divers treated for pain-only decompression sickness should not dive for at least seven days after treatment. Divers treated for serious decompression sickness or gas embolism should not dive for at least four weeks thereafter.

Choice Diagrams

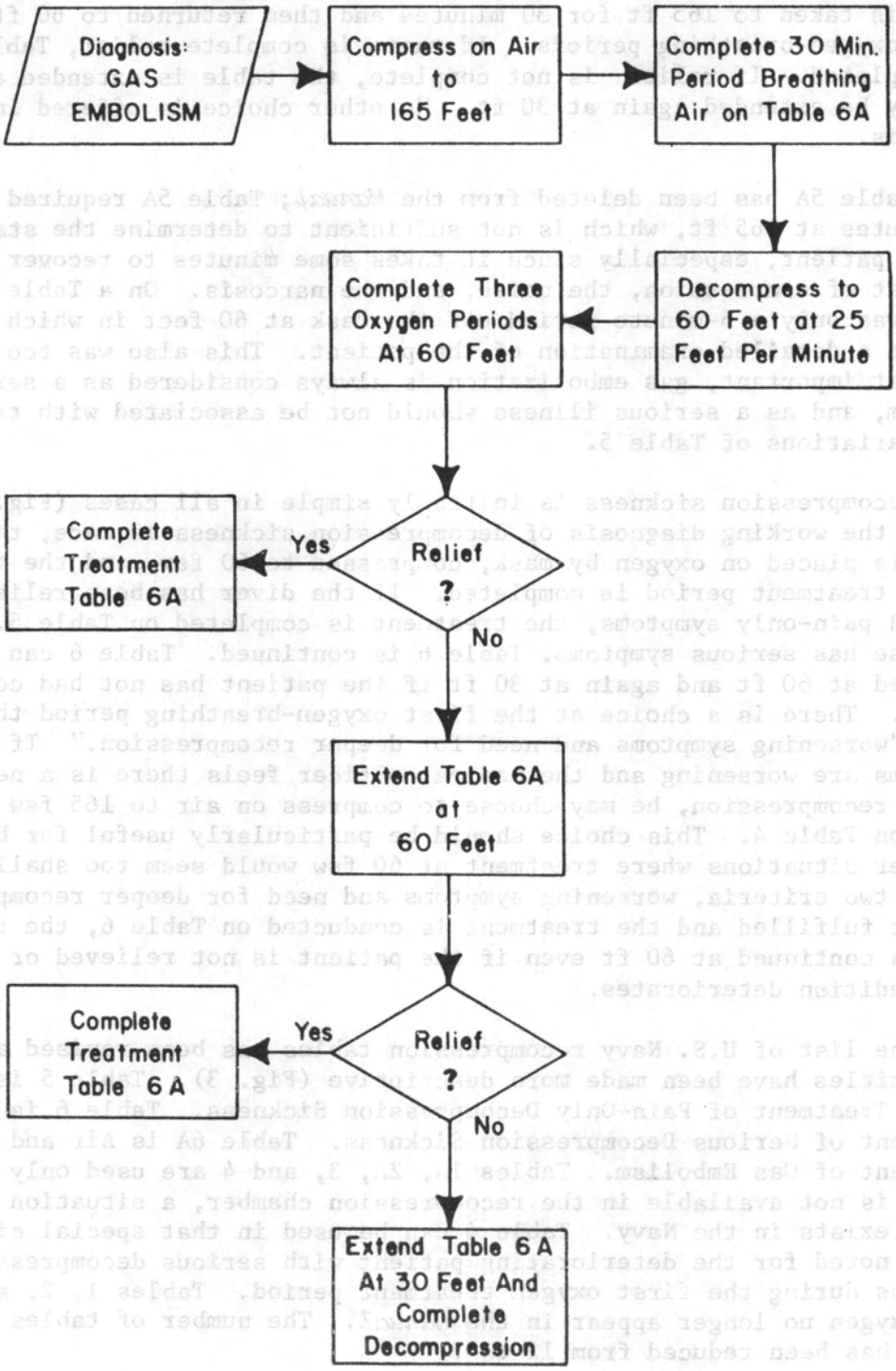
Gas embolism treatment is simply Table 6A (Fig. 1). The stricken diver is taken to 165 ft for 30 minutes and then returned to 60 ft for three oxygen-breathing periods. If there is complete relief, Table 6A is completed. If relief is not complete, the table is extended at 60 ft and may be extended again at 30 ft. No other choice is offered in the diagrams.

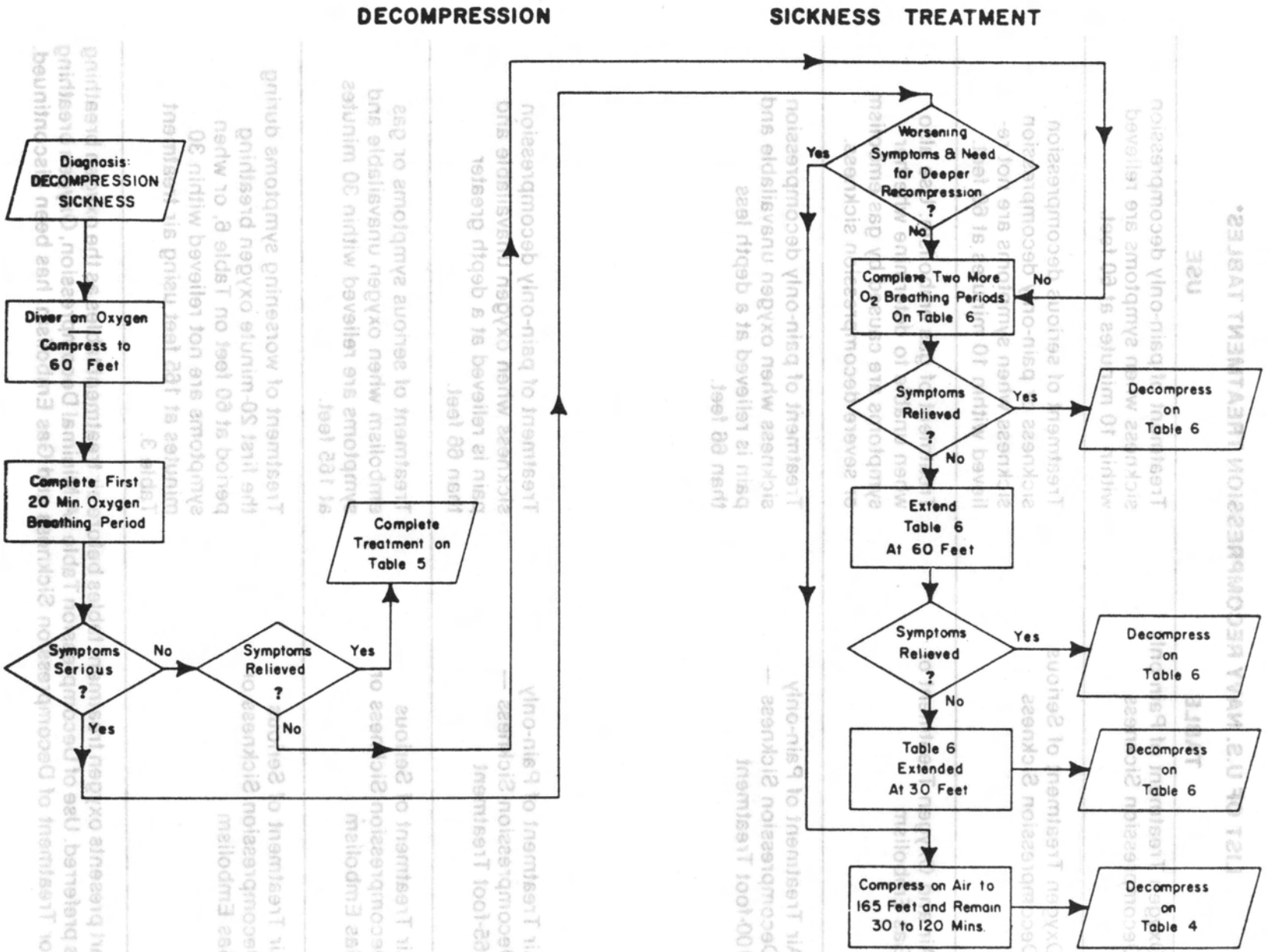
Table 5A has been deleted from the *Manual*; Table 5A required only 15 minutes at 165 ft, which is not sufficient to determine the status of the patient, especially since it takes some minutes to recover from the heat of compression, the noise, and the narcosis. On a Table 5A, there was only a 5-minute period off the mask at 60 feet in which to conduct a detailed examination of the patient. This also was too short. And most important, gas embolization is always considered as a serious symptom, and as a serious illness should not be associated with treatment variations of Table 5.

Decompression sickness is initially simple in all cases (Fig. 2). First, the working diagnosis of decompression sickness is made, the diver is placed on oxygen by mask, compressed to 60 fsw, and the first oxygen treatment period is completed. If the diver has been relieved and had pain-only symptoms, the treatment is completed on Table 5. If the case has serious symptoms, Table 6 is continued. Table 6 can be extended at 60 ft and again at 30 ft if the patient has not had complete relief. There is a choice at the first oxygen-breathing period that says, "worsening symptoms and need for deeper recompression." If the symptoms are worsening and the medical officer feels there is a need for deeper recompression, he may choose to compress on air to 165 fsw and treat on Table 4. This choice should be particularly useful for blow-up or other situations where treatment at 60 fsw would seem too shallow. If the two criteria, worsening symptoms and need for deeper recompression, are not fulfilled and the treatment is conducted on Table 6, the treatment is continued at 60 ft even if the patient is not relieved or if his condition deteriorates.

The list of U.S. Navy recompression tables has been revised and their titles have been made more descriptive (Fig. 3). Table 5 is Oxygen Treatment of Pain-Only Decompression Sickness. Table 6 is Oxygen Treatment of Serious Decompression Sickness. Table 6A is Air and Oxygen Treatment of Gas Embolism. Tables 1A, 2A, 3, and 4 are used only if oxygen is not available in the recompression chamber, a situation that seldom exists in the Navy. Table 4 can be used in that special circumstance noted for the deteriorating patient with serious decompression sickness during the first oxygen treatment period. Tables 1, 2, and 3 with oxygen no longer appear in the *Manual*. The number of tables included has been reduced from 12 to 7.

GAS EMBOLISM TREATMENT




DECOMPRESSION SICKNESS TREATMENT
Diagnosis: DECOMPRESSION SICKNESS
Diver on Oxygen — Compress to 60 Feet
Complete First 20 Min. Oxygen Breathing Period
Symptoms Serious ?
No
Yes
Symptoms Relieved ?
Yes
No
Complete Treatment on Table 5
Worsening Symptoms & Need for Deeper Recompression ?
Yes
No
Complete Two More O2 Breathing Periods On Table 6
No
Symptoms Relieved ?
Yes
Decompress on Table 6
No
Extend Table 6 At 60 Feet
Symptoms Relieved ?
Yes
Decompress on Table 6
No
Table 6 Extended At 30 Feet
Decompress on Table 6
Compress on Air to 165 Feet and Remain 30 to 120 Mins.
Decompress on Table 4

LIST OF U.S. NAVY RECOMPRESSION TREATMENT TABLES*

TABLE		USE
5 —	Oxygen Treatment of Pain-only Decompression Sickness	Treatment of pain-only decompression sickness when symptoms are relieved within 10 minutes at 60 feet.
6 —	Oxygen Treatment of Serious Decompression Sickness	Treatment of serious decompression sickness or pain-only decompression sickness when symptoms are not relieved within 10 minutes at 60 feet.
6A —	Air and Oxygen Treatment of Gas Embolism	Treatment of gas embolism. Use also when unable to determine whether symptoms are caused by gas embolism or severe decompression sickness.
1A —	Air Treatment of Pain-only Decompression Sickness — 100-foot Treatment	Treatment of pain-only decompression sickness when oxygen unavailable and pain is relieved at a depth less than 66 feet.
2A —	Air Treatment of Pain-only Decompression Sickness — 165-foot Treatment	Treatment of pain-only decompression sickness when oxygen unavailable and pain is relieved at a depth greater than 66 feet.
3 —	Air Treatment of Serious Decompression Sickness or Gas Embolism	Treatment of serious symptoms or gas embolism when oxygen unavailable and symptoms are relieved within 30 minutes at 165 feet.
4 —	Air Treatment of Serious Decompression Sickness or Gas Embolism	Treatment of worsening symptoms during the first 20-minute oxygen breathing period at 60 feet on Table 6, or when symptoms are not relieved within 30 minutes at 165 feet using air treatment Table 3.

* This chart presents oxygen treatment tables before air treatment tables as the oxygen breathing method is preferred. Use of Decompression Table 5A-Minimal Decompression, Oxygen breathing method for Treatment of Decompression Sickness and Gas Embolism, has been discontinued.

Recurrence of signs or symptoms during treatment is treated at 60 ft with oxygen using Table 6 or Table 6 extended (Fig. 4). There is no invitation to go to 165 fsw when the recurrence occurs at 60 fsw or shallower. If the symptom onset is deeper than 60 ft, as might be the case on Table 4, the diver is compressed on air to the depth of relief, but is not automatically taken to 165 ft.

Recurrence after treatment is treated with either Table 5 or 6, depending on the seriousness of the symptoms and the relief of the symptoms (Fig. 5).

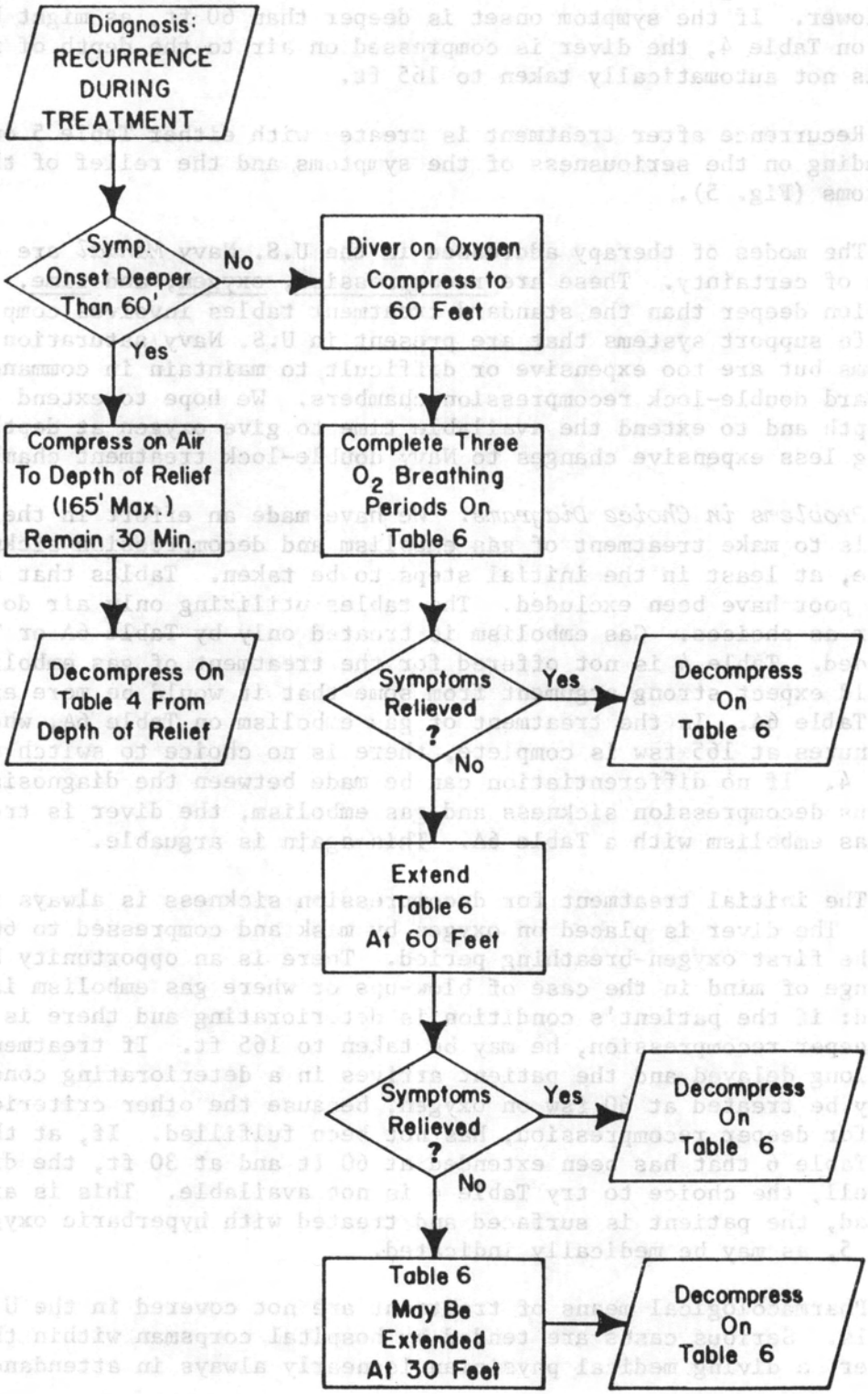
The modes of therapy addressed in the U.S. Navy *Manual* are only those of certainty. These are recompression, oxygen, and time. Recompression deeper than the standard treatment tables involves complexities of life support systems that are present in U.S. Navy saturation diving systems but are too expensive or difficult to maintain in commands having standard double-lock recompression chambers. We hope to extend the time at depth and to extend the available time to give oxygen at depth by making less expensive changes to Navy double-lock treatment chambers.

Problems in Choice Diagrams. We have made an effort in the Navy's manuals to make treatment of gas embolism and decompression sickness simple, at least in the initial steps to be taken. Tables that are known to be poor have been excluded. The tables utilizing only air do not appear as choices. Gas embolism is treated only by Table 6A or Table 6A extended. Table 4 is not offered for the treatment of gas embolism, but I would expect strong argument from some that it would be more efficacious than Table 6A. In the treatment of gas embolism on Table 6A, when the 30 minutes at 165 fsw is complete, there is no choice to switch to a Table 4. If no differentiation can be made between the diagnosis of serious decompression sickness and gas embolism, the diver is treated for gas embolism with a Table 6A. This again is arguable.

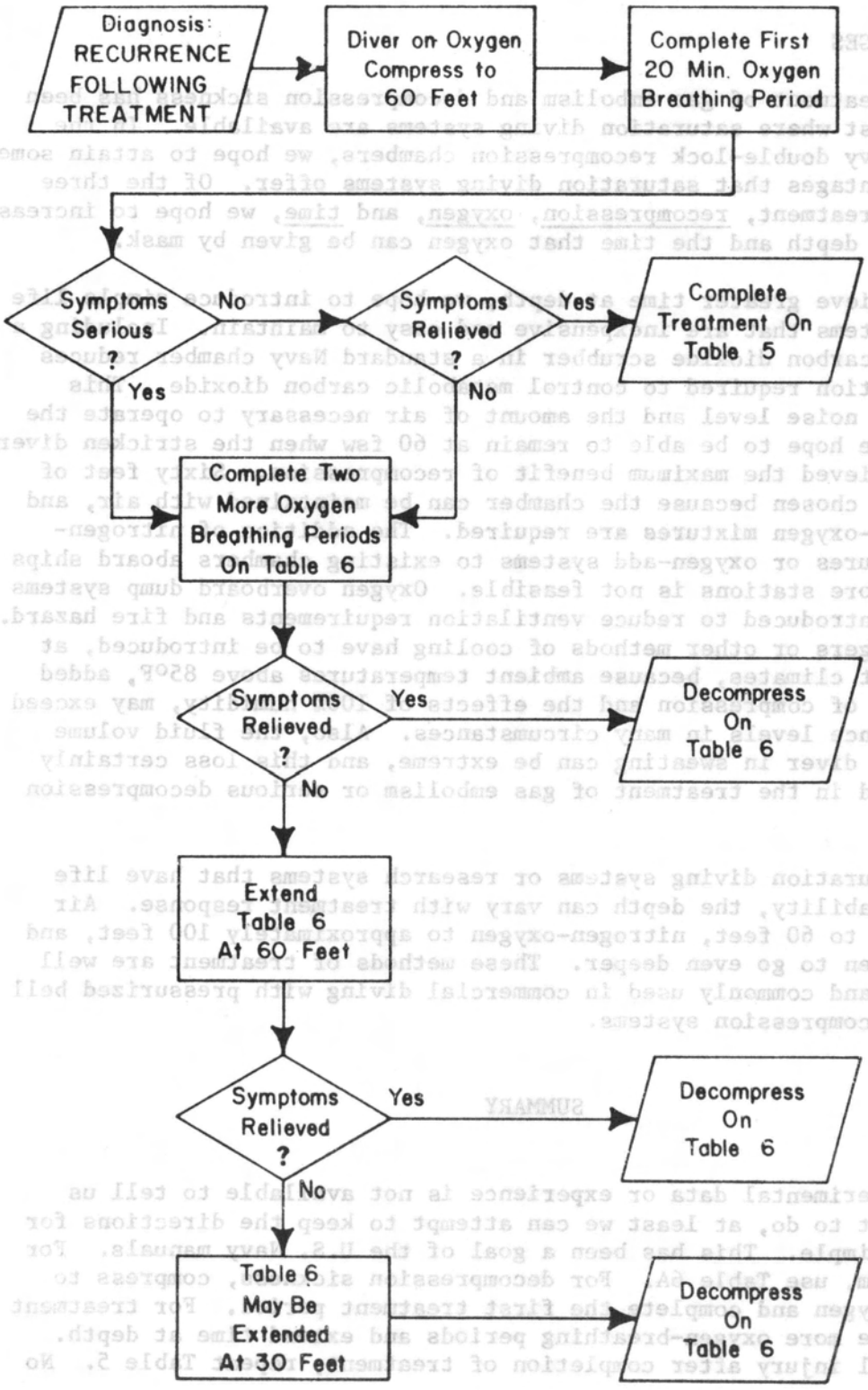
The initial treatment for decompression sickness is always the same. The diver is placed on oxygen by mask and compressed to 60 ft for the first oxygen-breathing period. There is an opportunity here for a change of mind in the case of blow-ups or where gas embolism is suspected: if the patient's condition is deteriorating and there is a need for deeper recompression, he may be taken to 165 ft. If treatment has been long delayed and the patient arrives in a deteriorating condition, he may be treated at 60 fsw on oxygen, because the other criterion, the need for deeper recompression, has not been fulfilled. If, at the end of a Table 6 that has been extended at 60 ft and at 30 ft, the diver is not well, the choice to try Table 4 is not available. This is arguable. Instead, the patient is surfaced and treated with hyperbaric oxygenation Table 5, as may be medically indicated.

Pharmacological means of treatment are not covered in the U.S. Navy manuals. Serious cases are tended by hospital corpsman within the chamber, a diving medical physician is nearly always in attendance, and

RECURRENCE DURING TREATMENT



RECURRENCE FOLLOWING TREATMENT



consultation with NMRI, NEDU, or any chosen diving medical officer is always available.

FUTURE CHANGES

The treatment of gas embolism and decompression sickness has been improved most where saturation diving systems are available. In the standard Navy double-lock recompression chambers, we hope to attain some of the advantages that saturation diving systems offer. Of the three facets of treatment, recompression, oxygen, and time, we hope to increase the time at depth and the time that oxygen can be given by mask.

To achieve greater time at depth, we hope to introduce simple life support systems that are inexpensive and easy to maintain. Including a fan-driven carbon dioxide scrubber in a standard Navy chamber reduces the ventilation required to control metabolic carbon dioxide. This reduces the noise level and the amount of air necessary to operate the chamber. We hope to be able to remain at 60 fsw when the stricken diver has not achieved the maximum benefit of recompression. Sixty feet of seawater is chosen because the chamber can be maintained with air, and no nitrogen-oxygen mixtures are required. The addition of nitrogen-oxygen mixtures or oxygen-add systems to existing chambers aboard ships or small shore stations is not feasible. Oxygen overboard dump systems are being introduced to reduce ventilation requirements and fire hazard. Heat exchangers or other methods of cooling have to be introduced, at least in hot climates, because ambient temperatures above 85°F, added to the heat of compression and the effects of 100% humidity, may exceed heat tolerance levels in many circumstances. Also, the fluid volume lost by the diver in sweating can be extreme, and this loss certainly does not aid in the treatment of gas embolism or serious decompression sickness.

In saturation diving systems or research systems that have life support capability, the depth can vary with treatment response. Air can be used to 60 feet, nitrogen-oxygen to approximately 100 feet, and helium-oxygen to go even deeper. These methods of treatment are well worked out and commonly used in commercial diving with pressurized bell and deck decompression systems.

SUMMARY

If experimental data or experience is not available to tell us exactly what to do, at least we can attempt to keep the directions for treatment simple. This has been a goal of the U.S. Navy manuals. For gas embolism, use Table 6A. For decompression sickness, compress to 60 ft on oxygen and complete the first treatment period. For treatment failure, use more oxygen-breathing periods and extend time at depth. For residual injury after completion of treatment, repeat Table 5. No

drugs are recommended, but difficult cases are managed by diving medical officers and additional consultation is always available. In systems with life support apparatus, the approach is the same as in saturation diving. The stricken diver is taken as deep as necessary and for as long as necessary.

A complaint has been voiced that the U.S. Navy tables become a standard from which others hesitate to deviate. The USN tables are the standards for the minimum depth and oxygen and time -- one should never do less -- but one can always do more.

There are several areas where the U.S. Navy could improve the treatment of decompression sickness and gas embolism and offer guidance to others. I hope the Navy will be able to improve its standard double-lock recompression chambers by adding carbon dioxide scrubbers, and heat exchangers in locations where thermal stress is a problem. This will give the capability to conduct saturation treatments at 60 fsw or shallower and offer a great deal more flexibility in treatment methods. Another action that will be taken is offering guidance for saturation treatment in the next change to the saturation diving section of Volume II of the U.S. Navy *Diving Manual*.

TREATMENT OF SERIOUS DECOMPRESSION SICKNESS

Xavier Fructus, M.D.

When the total number of divers is taken into account, serious decompression sickness (DCS) is more common among recreational divers than among those who dive professionally. First, time and depth are not as well controlled among sport divers, and second, there are often considerable delays in instituting recompression therapy because of the distance of the diving site from the hyperbaric chamber.

In contrast to the recreational diver, those who dive professionally benefit from certain safety precautions: 1) diving schedules and protocols are more rigidly respected (except in cases of equipment failure); and 2) in case of accident, therapeutic recompression is more rapidly instituted (French law requires a multiplace hyperbaric chamber in the vicinity of all underwater worksites).

In France, most recreational diving accidents occur on the Mediterranean coast. Over a seven-year period, Drs. Wolkiewiez and Plante-Longchamp of Nice and Dr. Prim of Var Province treated diving accidents that occurred along the coast between Toulon and the Italian border. They saw a total of 140 diving accidents, of which two-thirds were DCS and the balance pulmonary barotrauma with air embolism or near-drowning. Of the DCS cases, about one-half consisted of neurologic DCS. The delay involved in transporting the victim was often catastrophic and included time spent in transit from the dive site to the hospital in Nice. It is common knowledge that delayed treatment is less effective.

However, certain observations on the physiopathology have served as the basis for instituting medical treatment before recompression. The author has published an account of a case of neurologic DCS that occurred off the coast of Sardinia in 1970. With two companions, the author dove to 40 meters, observing the pertinent French decompression tables. A fellow diver, W., stayed on the bottom for less time than the author or the third diver; however, W. performed several Valsalva maneuvers during ascent. A few minutes after surfacing, he presented with lumbar back pain and a pins and needles sensation in both lower extremities. This progressed suddenly to weakness of both lower extremities, sufficient to prevent upright posture. Physical examination revealed paresthesia of both legs and an upgoing Babinski sign on the left side.

The divers were hours away from a recompression chamber, even if the group was successful in alerting a helicopter. The following interim treatment was instituted: 1) pure oxygen by mask; 2) one liter of fruit juice by mouth over one hour; and 3) one gm aspirin by mouth. During the first half hour, the signs and symptoms grew worse. During the

second half hour, however, the patient became asymptomatic, results of a physical examination returned to normal, and there was no recurrence. Obviously, this incident could represent a case of spontaneously resolved DCS, but the possibility that this interim treatment was effective was intriguing.

Hearing of this case, Wolkiewiez and Plante-Longchamp had the idea of applying similar treatment to recreational divers with DCS en route to recompression facilities. Mask oxygen was the first treatment to be adopted. Oral aspirin and large quantities of oral fluids (one liter of water over an hour) were soon added to the routine. Finally, drawing upon experience using plasma expanders and corticosteroids as ancillary treatment during recompression, Drs. Wolkiewiez and Plante-Longchamp devised a complete protocol (Table 1). It is meant to be administered as early as possible to victims of neurologic decompression sickness. It includes four elements.

TABLE 1

Medical treatment of CNS decompression sickness during transport to hyperbaric chamber

Step	Treatment	Dose
1	100% oxygen by mask	
2	corticoids i.v.	
	hydrocortisone hemisuccinate or	1000 mg
	dexamethasone or	30 mg
	medrocortisone	160 mg
3	aspirin, i.v. (not available in U.S.)	1000 mg
4	dextran 40, i.v. infusion	500 ml

Derived from Wolkiewiez and Plante-Longchamp.

Note that the doses of corticosteroid are quite high. Drs. Wolkiewiez and Plante-Longchamp believe it is at these high dosages that maximum anti-inflammatory action, anti-edema effect, and restoration of microcirculation are obtained. The dosages given are for a single administration.

The pharmacologic effect of the aspirin is based more on empiric than on experimental evidence. In any case, it is necessary to rule out a possible allergy to salicylates before administering the aspirin. (Ed. note - intravenous aspirin is not available for use in the U.S.)

As far as plasma expanders are concerned, we French prefer dextran of average molecular weight equal to 40,000 (Rheomacrodex®). We believe this to be superior to 70,000 molecular weight dextran for maintaining the victim's renal function. Although French professional divers must pass rigid medical examinations yearly, recreational divers may have impaired renal functions. Wolkiewiez and Plante-Longchamp believe that dextran is superior for its anti-sludge action. However, they also think that Ringer's lactate is more effective in restoring volume. They plan to combine perfusions of both fluids in the future.

This therapeutic protocol has proved to be so effective that a potential danger may arise. The results obtained may be transient, and may then be followed by a serious recurrence of DCS if recompression is not carried out. Complete treatment must include the preliminary medical therapy plus speedy transport to a hyperbaric chamber. Once the patient is at the facility, a doctor with experience in diving medicine may make the decision whether or not to compress the victim.

Finally, we shall examine the tabulated results obtained by our colleagues in Nice. First, the efficacy of the first aid treatment will be considered, and then the final outcome of recompression therapy in this series of 67 cases (see Tables 2 through 8). All cases involved neurologic DCS with delays of three to 24 hours in reaching the chamber (mean delay about 10 hours).

TABLE 2

Cases treated during transport

Type of CNS Decompression Sickness	Transport Without Treatment	First Aid During Transport		
		O_2	Aspirin + O_2	Complete protocol
Cerebral (23 cases)	8	2	1	12
Spinal (38 cases)	6	1	4	27
Mixed (6 cases)	0	0	0	6

TABLE 3

Results of first aid treatment in cerebral DCS

Condition Before Recompression	Transport Without Treatment	First Aid During Transport			Total
		O_2	Aspirin + O_2	Complete Protocol	
Unchanged	8	0	0	3	11
Improved	0	1	1	3	5
Asymptomatic	0	1	0	6	7
Total	8	2	1	12	23

TABLE 4

Results of first aid in spinal DCS

Condition Before Recompression	Transport Without Treatment	First Aid During Transport			Total
		O_2	Aspirin + O_2	Complete Protocol	
Unchanged	6	0	2	7	15
Improved	0	1	2	15	18
Asymptomatic	0	0	0	5	5
Total	6	1	4	27	38

TABLE 5

Results of first aid treatment in mixed DCS

Condition Before Recompression	Transport Without Treatment	First Aid During Transport			Total
		O_2	Aspirin + O_2	Complete Protocol	
Unchanged	0	0	0	3	3
Improved	0	0	0	3	3
Asymptomatic	0	0	0	0	0
Total	0	0	0	6	6

TABLE 6

Results of first aid during transport

Type of CNS Decompression Sickness	Transport Without Treatment		First Aid During Transport	
	Unchanged	Improved or asymptomatic	Unchanged	Improved or asymptomatic
Cerebral (23 cases)	8/8	0/8	3/15	12/15
Spinal (38 cases)	6/6	0/6	9/32	23/32
Mixed (6 cases)	0/0	0/0	3/6	3/6
Total (67 cases)	14/14	0/14	15/53	38/53
Percentage	100%	0%	28%	72%

TABLE 7

Results of CNS DCS treatment on various treatment tables

Table	Complete Relief (A)	Minor Sequelae (B)	A + B (percentage)	Major Sequelae
USN V and VI	14	16	30 (88%)	4
French Navy Gers A	7	5	12 (92%)	1
French Navy Gers B and Comex 30	3	3	6 (75%)	2

The Gers A and B tables may be found in Appendixes 4 and 5, and the Comex 30 and 30A tables in Appendixes 2 and 3.

TABLE 8

Results of CNS DCS treatment, all tables, by type of DCS

Type of CNS DCS	Complete Relief (A)	Minor Sequelae (B)	A + B	Major Sequelae
Cerebral (16 cases)	11	4	15	1 (death)
Spinal (33 cases)	13	14	27	6
Mixed (6 cases)	0	6	6	0
Percentage (55 cases)	44	44	88	12

ADJUNCTIVE TREATMENT METHODS

Eric P. Kindwall, M.D.

Recompression remains the sine qua non of the treatment of decompression sickness, but over the past few years adjunctive measures have assumed great importance, with emphasis on the period immediately following the onset of symptoms and before recompression.

FLUIDS

Dehydration has long been clinically associated with greater susceptibility to decompression sickness, and early hydration is receiving greater medical attention in the symptomatic case. Fructus has presented evidence (this workshop report) derived from Wolkiewiez and Plante-Longchamp that patients who were hydrated early after onset of symptoms had a decrease in symptoms compared to unhydrated patients, before actual recompression. Hallenbeck et al. (1976) showed that significant plasma loss occurs because of capillary leakage, and it appears that any means of increasing blood volume will produce an immediate but temporary improvement. Forcing oral fluids, at the rate of one liter an hour for the first two hours, in the conscious patient or, preferably, using intravenous infusion of Ringer's lactate, dextrose 5%, and saline or normal saline may be crucially important in the immediate treatment of decompression sickness. The use of plain dextrose (5%) and water is discouraged because the glucose will be metabolized away, leaving water, which may worsen neural tissue edema. Since the typical patient may be dehydrated or behind on fluids either because of an inadequate oral intake (in tropical environments) or may perhaps be suffering from post-alcoholic dehydration, it is important that enough fluid be given to ensure an adequate urine flow -- 1 to 2 ml per kg per hour is a suitable urinary output. A urinary catheter should be used if the patient is unable to urinate and becomes distended.

THE DEXTRANS

If fluid volume replacement with saline or Ringer's lactate proves ineffective, dextran is considered. Dextran 40 (low molar dextran, Rheomacrodex®) and Dextran 70, its heavier analog, have been used for some time as plasma expanders. Early studies seemed to show that dextran had an effect in addition to blood volume expansion that might be helpful to divers. Dextran 40 was thought to alter the electrical charge on the surface of red cells, thereby inhibiting Rouleaux formation. For this reason, it was my practice to prescribe at least one unit of low molar dextran for any diver suffering symptoms of decompression sickness if the symptoms had been present longer than two hours before recompression. At the European Undersea Biomedical Society workshop on the problems of

decompression sickness in the North Sea, Ah-see (1976) reported that Dextran 70 probably was superior in that respect. To achieve this effect, either Dextran 40 or 70 would have to be given in volumes on the order of 4 to 5 liters. The original Swedish studies upon which we had previously based our assumptions had involved mice with extremely high hematocrits, and the data did not apply to the condition of the usual diver. For this reason it appears that the use of the dextrans should be restricted to the treatment of decompression sickness shock, where plasma expansion is the overriding concern. Dextran 40 is rapidly excreted through the kidney but exerts a stronger osmotic pressure because of its smaller molecule size. Because of its rapid excretion, Dextran 70 should probably be given after the first unit of Dextran 40 if blood volume needs to be continually bolstered.

PLASMA, RECONSTITUTED PLASMA, PLASMANATE

Again, if saline or Ringer's lactate fails to maintain fluid volume, plasma can be used. Plasma has been useful in bends shock, as might be expected. Cockett (1964) showed that a group of experimental dogs given plasma survived severe decompression sickness, whereas the controls did not.

Of more recent interest is the plasma colloid osmotic pressure. This determination can be made in some hospitals, and if it falls below 19 to 20 mmHg, albumin would be administered until colloid osmotic pressure is restored. Pulmonary and generalized edema become evident at pressures of 15 to 16 mmHg, and there is no survival if colloid osmotic pressure drops below 11 mmHg. Fluid is not lost from the body in decompression sickness shock but simply extravasates into the tissues from where, at least theoretically, it may be retrieved and returned to the vascular compartment. Maintaining plasma colloid pressure to prevent extravasation would be better than simple volume replacement, especially in cases where pulmonary edema is a concomitant. In shock, colloid osmotic pressure does not bear a simple relationship to the amount of circulating albumin.

HEPARIN

Cockett (1972) showed that heparinized dogs subjected to a decompression insult survived decompression sickness, whereas control dogs did not. Philp (1964) has shown that obese rats were able to survive decompression when heparinized, while their unheparinized controls did not. Heparin's lipemia-clearing ability rather than its anti-coagulant effect was considered to be the mechanism of action. Reeves and Workman (1971) did not show any beneficial effects, however, in dogs heparinized after mild to moderate decompression sickness. This is expected since heparin has little effect on platelets but acts mainly on the intrinsic clotting system. Heparin does not remove existing thrombi but prevents further clotting. Most physicians would not use it initially, but in

seriously injured divers who will be at bed-rest for prolonged periods it could be used to prevent thrombo-embolism. The suggested dose is 2500-3000 units given intravenously every 8 hours if necessary. In any case, heparin is definitely contraindicated in vestibular decompression sickness (Farmer 1976). Waite (1967) documented cerebral hemorrhage after experimental air embolism in a dog, so heparin's use in burst lung would also appear to be contraindicated.

ASPIRIN

Theoretically, aspirin should have value because it appears to inhibit platelet aggregation. Hallenbeck et al. (1976) noted that 600 mg or two tablets taken orally will exert this effect maximally in 30 minutes. Again, once platelet aggregation is complete and vessel blockade has been established, its theoretical value is nil. For this reason, to have an effect, it would have to be given immediately after the earliest symptoms appear. There are no experimental data to demonstrate aspirin's prophylactic value when taken before decompression, but from the theoretical viewpoint, one is tempted to make the assumption.

STEROIDS

Steroids are given because they tend to stabilize vascular endothelium and have an anti-edema effect, which has been noted by neurosurgeons. For this reason the indication is usually serious decompression sickness affecting the brain and spinal cord. Early work seemed to show that steroids exacerbated oxygen toxicity, but clinically this has not been observed even with high steroid dosage. Pre-treatment with steroids has not increased the risk of convulsions on U.S. Navy Treatment Tables 5 and 6. The recommended dosage would appear to be one gram of a rapidly acting steroid such as hydrocortisone hemisuccinate given i.v. in a bolus, with 4 mg of dexamethasone 21-phosphate given concomitantly intramuscularly. The latter drug is continued in 8 mg dosage every six hours for two to three days. It then may be stopped abruptly without tapering or continued if clinically indicated or if a neurologic consultant so advises. It must be remembered, however, that even intravenous steroids generally have a lag period of some hours before having demonstrable effect. It is no longer felt that steroid peptic ulcer is a danger when therapy is limited to 72 hours.

GLYCEROL

The value of glycerol in the treatment of cerebral edema was demonstrated by Tourtellotte (1972), Matthew (1972) and Reinglass (1974). It is given orally, 0.8 ml per kg of body weight, in a 50% solution of water that may be flavored with lemonade to make it more palatable. Glycerol would appear to have many advantages for the diver when compared with other edema-reducing agents, as summarized by Saper and Yosselson (1975). It is superior to mannitol and urea in that it does not derange electrolyte balance, and it exerts its maximal effect in one hour, with

a duration of effect of six hours. This is faster than all the other agents. It does not cause rebound edema and -- a particular advantage to the diver who may be remote from the services of a paramedic or physician -- it can be given orally by a layman. The only major disadvantage of glycerol is that it is an unpalatable concoction that may induce nausea and vomiting when ingested by the patient. In such cases it could be given via nasogastric tube.

DIGITALIS

In decompression sickness shock, if heart rate is high despite adequate fluid supplementation, digitalis has been useful. In a case of bends shock which the author treated, it appeared to be the drug that saved the patient's life when everything else had failed. The patient may be quickly digitalized intravenously with deslanoside 0.8-1.6 mgm. Digitalization is then maintained with digoxin or one of the other digitalis preparations.

NARCOTIC ANALGESICS

The use of opiates is usually contraindicated unless there is some overriding necessity. Respiratory depression caused by morphine or one of its analogs produces a rise in the arterial PCO_2 which may in turn bring on early CNS oxygen toxicity during hyperbaric treatment. Additionally, potent analgesics mask symptoms and hinder evaluation of response to treatment.

ANTI-CONVULSANTS

Diazepam appears to be the drug of choice in the event of convulsions due to CNS damage. Barbiturates are not as safe because they produce more respiratory depression, again with the increased liability of CNS oxygen toxicity during treatment. Dilantin does not appear to protect against oxygen seizures, although diazepam and the barbiturates seem effective. Diazepam may be administered intravenously in 10 mg doses as required. Extreme caution must be observed in controlling convulsions in patients receiving hyperbaric oxygen because permanent neurologic damage may result if convulsions are suppressed and normal time-depth limits for oxygen breathing are exceeded.

SEDATIVES

If the patient is extremely agitated or restless and requires sedation during transport, or if agitation is so great that sedation is required to carry out treatment, diazepam is again the preferred drug. The intravenous route is best and the dosage is the same as that indicated for convulsion suppression.

At the present time there are no theoretical or clinical data to support or suggest the use of other adjunctive agents in the treatment of decompression sickness.

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THE IMMEDIATE CARE, TRANSPORT, AND CHAMBER INTENSIVE CARE OF DIVING CASUALTIES

Phillip B. James, M.D.

The immediate care given to a diving casualty clearly depends on 1) the condition of the diver; 2) the location of the accident; 3) the skill of the personnel available; and 4) the drugs and equipment on site. Dealing first with the clinical state of the diver, it is surely academic to be concerned with the distinction between cerebral decompression sickness and cerebral gas embolism. It is difficult to be sure that gas entrapment has not occurred in the lung on ascent. Decompression sickness and gas embolism are likely to occur together in a blow-up from depth in commercial diving when the time at depth has exceeded the no-stop decompression limits. Serious decompression sickness, which is commoner in air diving than in mixed gas diving, must not be regarded in any way as progression from the milder forms of decompression sickness, and it is important that divers should be taught this. The point is that while limb bends and spinal cord decompression sickness or chokes may result from the same decompression insult, their pathophysiology and clinical implications are quite different. It is also necessary to consider the possibility of the inhalation of water as a factor in the diver's condition when the ascent has been made in the water, as is the case in emergency ascent with scuba.

There are three categories in which the seriously ill diver can be placed: Apparently dead, unconscious but breathing spontaneously, and conscious but with neurological deficit. Clearly the worst case is the apparently dead diver who is apneic and has no apparent pulse.

The immediate priorities are FIRST AID:

1. To clear the airway.
2. To institute artificial ventilation.
3. To give external cardiac massage.

There has been some discussion in the U.K. about the unfortunate tendency for mouth-to-mouth, or nose, resuscitation to inflate the stomach rather more capably than the lungs, with inevitable vomiting, particularly if external cardiac massage is also used. In training it is imperative to teach that the head should be well extended, and it

is sensible to recommend light pressure on the junction of the cricothyroid cartilages with the side of the hand to compress the esophagus. Care must be taken to avoid over-pressurization of the lung on positive pressure ventilation in cases of lung barotrauma because of the danger of pushing more gas into the circulation. Cardiac massage should be instituted when cardiac activity has ceased. Cessation is less likely than in, say, myocardial infarction, which is the usual clinical situation in which cardiac arrest is seen. Enthusiastic closed chest massage frequently causes injuries to the thorax and even abdomen that are not survivable. A NOAA report on sport diving fatalities highlighted this problem in 1974 (Underwater Fatality Statistics), citing a particular example and suggesting that further research was needed. The problem is clearly more acute in the older diver with a less flexible chest wall. The emphasis must be on the careful training of all divers in this crucial area.

The unconscious diver breathing spontaneously is probably best stabilized by an oropharyngeal airway. If he is not deeply unconscious, the airway will be coughed out.

Other measures obviously depend on what is available, and may include: 1) oxygen, continuously; 2) **rapidly acting** steroids (hydrocortisone, 1 gm. i.v., or methyl prednisolone); and 3) intravenous fluids.

Dr. Charles Wells' work on dogs (1976, 1978) indicates that at least in the short term the fluid used does not matter too much. There may be a consensus not to use dextrose in water, but dextrose in saline, saline, and Ringer's lactate are all clearly suitable. In the U.K. we have additionally a recommendation to use Dextran 70, but this is open to debate.

4) Drugs suggested: a) aspirin; the injectable form is NOT available in most situations; b) Valium^{R} is of particular use in the control of convulsions; 5) Mannitol was used by Saumarez, Bolt and Gregory (1973) as an infusion of 500 ml over several hours in an attempt to reduce cord and cerebral edema.

The final category is the conscious patient with a neurological deficit, for whom there is also the possibility of using oral fluids, although clearly intravenous therapy is to be preferred. As in the management of all casualties, regular monitoring of the vital signs before, during, and after the transport of a diving casualty is essential.

The importance of transporting a diving casualty to a hyperbaric facility must be stressed because of relapse. Transport should be at 1 ATA or as close to it as possible. A number of aircraft are able to maintain cabin pressure at 1 ATA throughout flight (see Workshop

Conclusions). Helicopters should restrict altitude if possible to 500 ft. An alternative in the United Kingdom is to transport the hyperbaric facility to the casualty. The International Underwater Contractor (IUC) Company's titanium chambers could be used for the on-site treatment of a scuba casualty, with subsequent transfer under pressure.

In sport diving in cold water, the diving casualty may well be suffering from hypothermia in addition to decompression sickness. In the presence of less serious symptoms, that is, rashes with no other symptoms, joint aches that do not worsen with time, or subcutaneous emphysema, the patient may be slowly rewarmed. Serious symptoms, however, are associated with hypoxia, and hypothermia has been shown to have a strong protective influence by reducing the oxygen requirement, particularly of nervous tissue. It is therefore sensible to recommend that no attempt be made to rewarm a seriously ill diving casualty.

Intensive care in the chamber is, of course, additional to pressure and the use of high partial pressures of oxygen. The measures used clearly depend on the back-up outside the chamber, the availability of through-chamber monitoring and the condition of the diver. If the facilities allow, then clearly it is necessary to establish the full clinical parameters: blood samples for full blood analysis; electrolytes; blood gas analysis; and special investigations (X-ray, ENG, ECG may be done where appropriate).

In some instances evacuation may be to a sophisticated medical center with experienced personnel. However, there have been many instances where intensive care has been required in small chambers and the staff have had little experience with hyperbaric conditions. The following information may be useful:

1. Ampules must be opened and re-usable bottles vented when pressurized.

2. Blood sample bottles must be left uncapped when pressurized and depressurized.

3. Intravenous fluids should ideally be in plastic bags that are collapsible. If glass bottles are used, the air space must be vented during decompression, either by an extended vent or by inversion during a staged decompression.

4. Check lists should be used. In the stress of the moment, particularly at raised partial pressures of nitrogen, it is easy for important observations to be omitted.

5. A chart of pulse, blood pressure, respiration rate, and fluid intake and output should be started as soon as possible. Since this is more usually a nursing duty it is again easily forgotten in the acute stages.

6. An aneroid sphygmomanometer must be used. Any instrument containing mercury, including thermometers, must not be used in a chamber.

7. Foot protection should be worn to guard against injuries.

8. Warm clothing, ideally non-flammable, may be required, especially during decompression when the chamber temperature falls.

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NOTES ON THREE RECOMPRESSION TREATMENT RESEARCH PROJECTS

Cdr. T. E. Berghage

Three small-animal research projects dealing with recompression therapy have been undertaken at the Naval Medical Research Institute. Small rodents (rats) have been used in these studies, for economic reasons; using them allows a researcher to screen several potentially important variables rather rapidly and at relatively low cost. By using several hundred animals in each study, the statistical variability associated with the decompression sickness phenomenon can be dealt with and the underlying relationships illuminated. The first study to be reviewed, although it did not deal with the topic of this workshop (the treatment of severe decompression sickness), is important to lead into the second study.

STUDY 1. TREATMENT OF DECOMPRESSION SICKNESS OCCURRING UNDER PRESSURE

This study was undertaken to evaluate the efficacy of three major treatment variables: recompression magnitude, oxygen partial pressure, and time. The dependent measure for this study was the change in the ED_{50} pressure-reduction value. (The ED_{50} is the pressure reduction that will produce signs of decompression sickness in 50% of the animals exposed.)

As shown in the dive profile in Fig. 1, an abrupt decompression to 12 ATA after a 40-minute exposure at 30 ATA will produce decompression sickness in 50% of the research animals. A 20-minute observation period is needed at 12 ATA to detect 99% of the signs of decompression sickness (DCS). If a treatment regimen is introduced after only 2 minutes at 12 ATA, the ED_{50} pressure can be altered and the degree of alteration is modified by the magnitude of the recompression, the oxygen partial pressure, and the treatment time. The dive profile, with the treatment

Figure 1

regimen, is shown in Fig. 2. Figures 3, 4, and 5 display the changes in the ED_{50} values caused by manipulating the three experimental variables. It is apparent from the data in the figures that oxygen partial pressure and time are the most cogent treatment variables for the management of decompression sickness occurring under pressure. Recompression appears to have little if any positive effect on such cases. When one considers Boyle's Law, this is not an unexpected result. According to Boyle's Law, tremendous increases in pressure are required to reduce bubble volumes when the bubbles are formed under increased pressures. The deeper the depth at which DCS occurs, the less the value of recompression. At depths (pressures) as great as those used in this study, there is even an indication that recompression is disadvantageous. The amount of additional gas taken up by the body is greater than that eliminated through bubble resolution. For decompression sickness that occurs under increased pressure, it appears that the only therapeutic tools presently available to ameliorate the condition are elevated oxygen partial pressures, time, and adjunctive drugs and fluids.

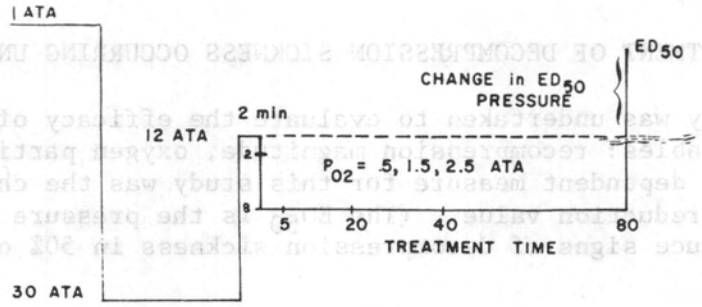


Figure 2

STUDY 2. THE THERAPEUTIC VALUE OF RECOMPRESSION AND ELEVATED OXYGEN PARTIAL PRESSURES

If Boyle's Law is the principle that governs the efficacy of recompression therapy, then, as shown in Fig. 6, there should be a "diminishing marginal return" in the use of recompression to treat decompression sickness. Barnard and Hanson (1973) have demonstrated with a mouse study (Figs. 7 and 8) that the principle of a diminishing marginal return does indeed exist in recompression therapy. We have since reproduced this same effect in rats (Fig. 9). This study, although not completed yet, extends the Barnard and Hanson (1973) results in that it assesses the combined effects of recompression and elevated oxygen partial pressures. The partial results so far obtained tend to indicate that the two therapeutic procedures interact with each other to produce a degree of synergism. The initial results are shown in Table 1; these should be viewed with a degree of skepticism until the entire study is completed.

TABLE 1

Incidence of decompression sickness after a 20-min treatment with various combinations of recompression and elevated oxygen partial pressure.

Recompression Magnitude (atm)	Oxygen Partial Pressure (ATA) .25	.50	1.00	2.00	4.00
1	25%	35%			
2		25%			
3			25%	20%	
5					
7			10%	20%	0%
9	27%			10%	

0 | .20 | 80%

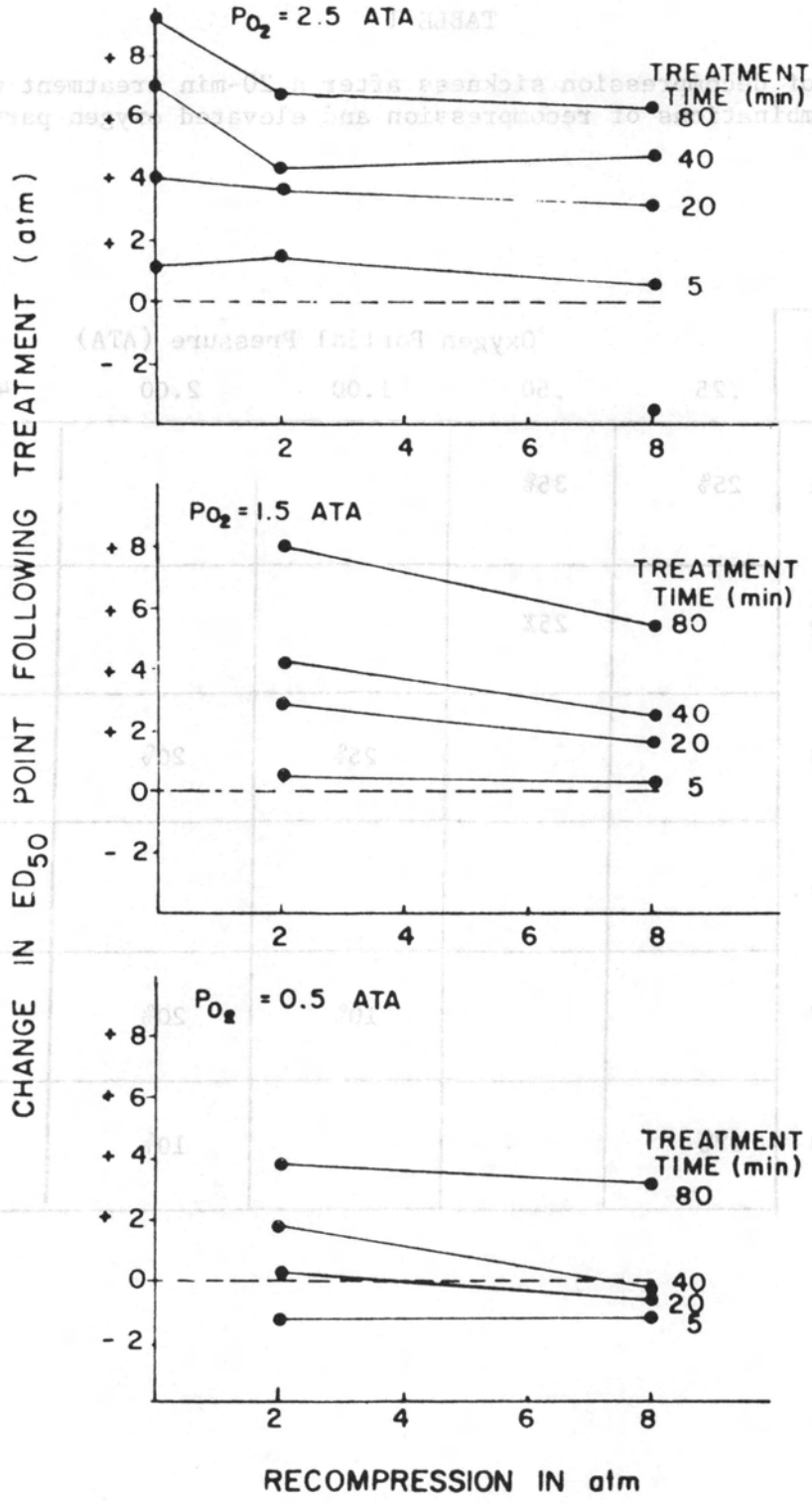

P_{O_2} = 2.5 ATA
TREATMENT TIME (min)
80
40
20
5
P_{O_2} = 1.5 ATA
TREATMENT TIME (min)
80
40
20
5
P_{O_2} = 0.5 ATA
TREATMENT TIME (min)
80
40
20
5
CHANGE IN ED_{50} POINT FOLLOWING TREATMENT (atm)
+8
+6
+4
+2
0
-2
2
4
6
8
RECOMPRESSION IN atm

Figure 3

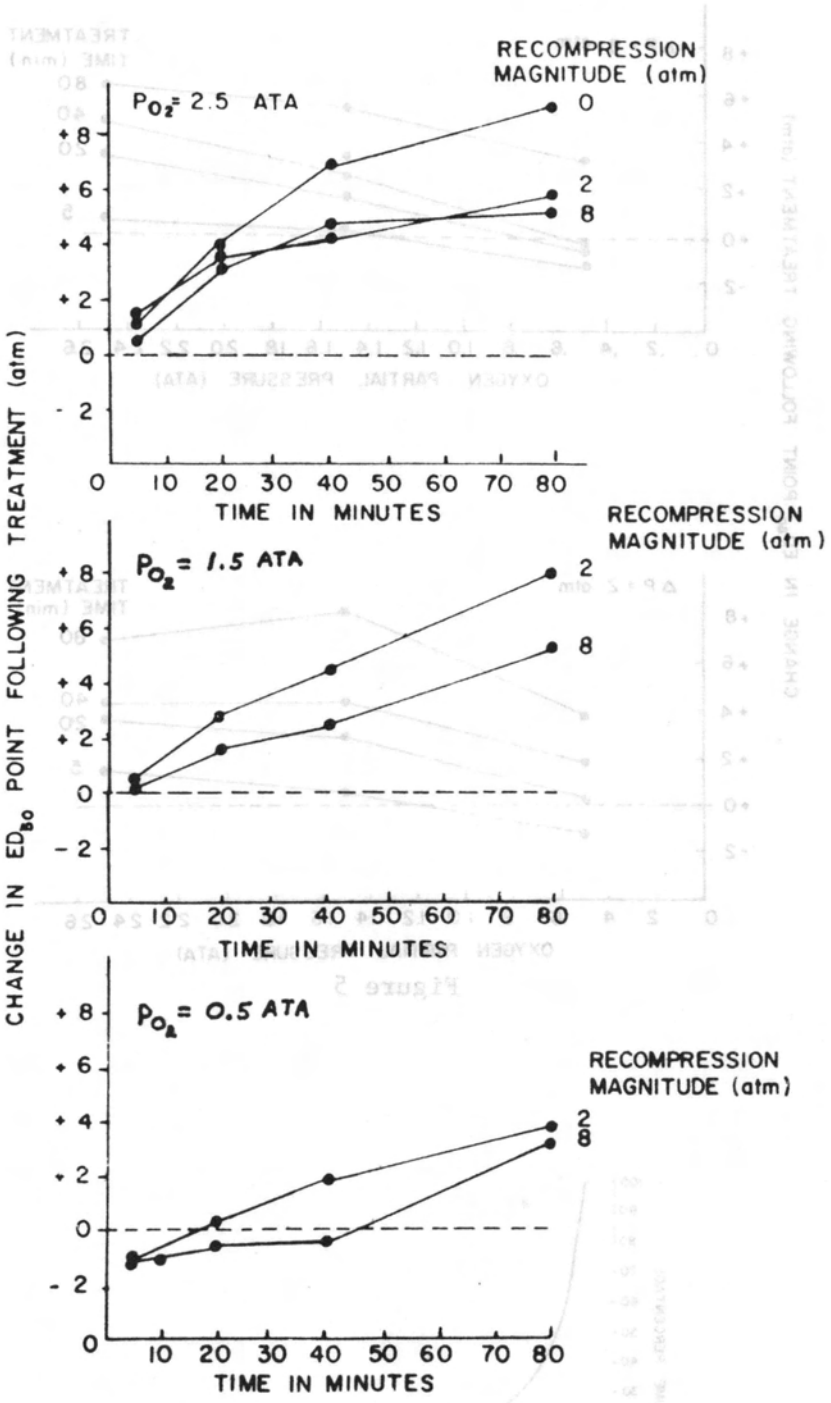

RECOMPRESSION MAGNITUDE (atm)
P_{O_2}= 2.5 ATA
0
2
8
+8
+6
+4
+2
0
-2
0 10 20 30 40 50 60 70 80
TIME IN MINUTES
RECOMPRESSION MAGNITUDE (atm)
P_{O_2}= 1.5 ATA
2
8
TIME IN MINUTES
P_{O_2}= 0.5 ATA
RECOMPRESSION MAGNITUDE (atm)
2
8
TIME IN MINUTES
CHANGE IN ED_{50} POINT FOLLOWING TREATMENT (atm)

Figure 4

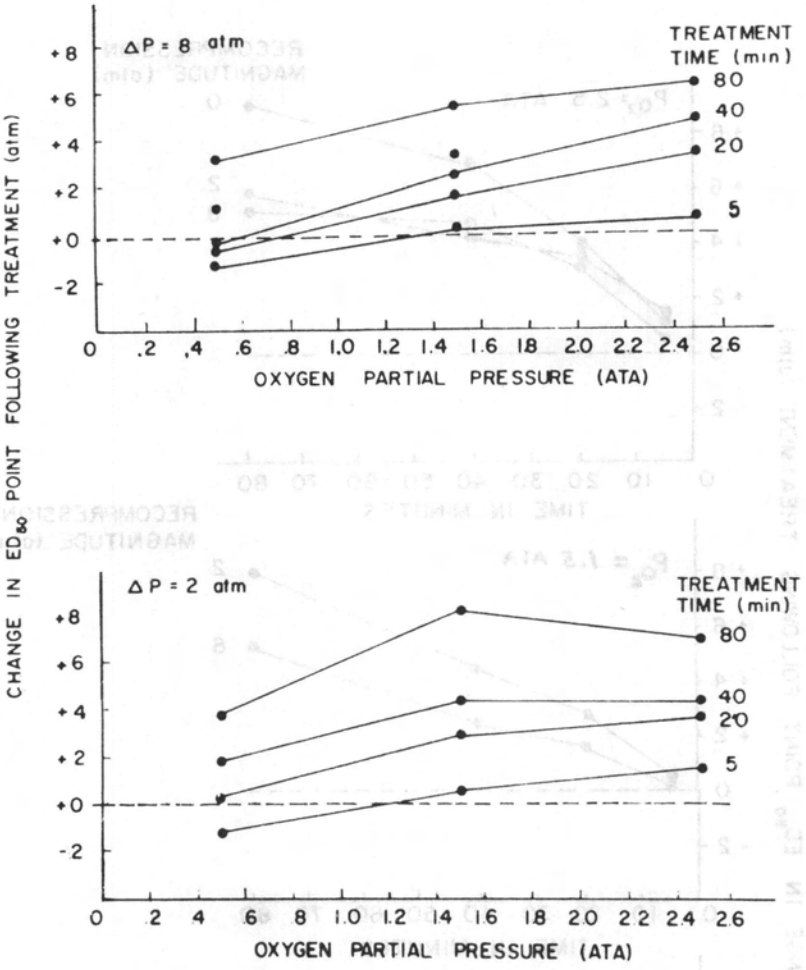


Figure 5

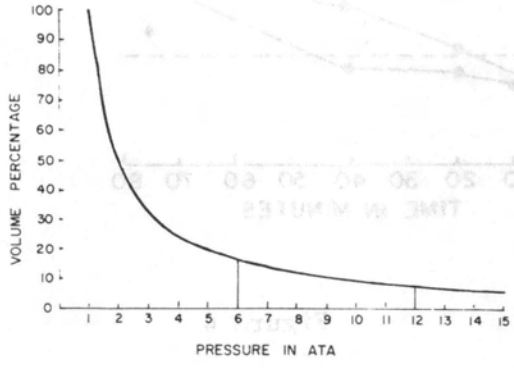


Figure 6

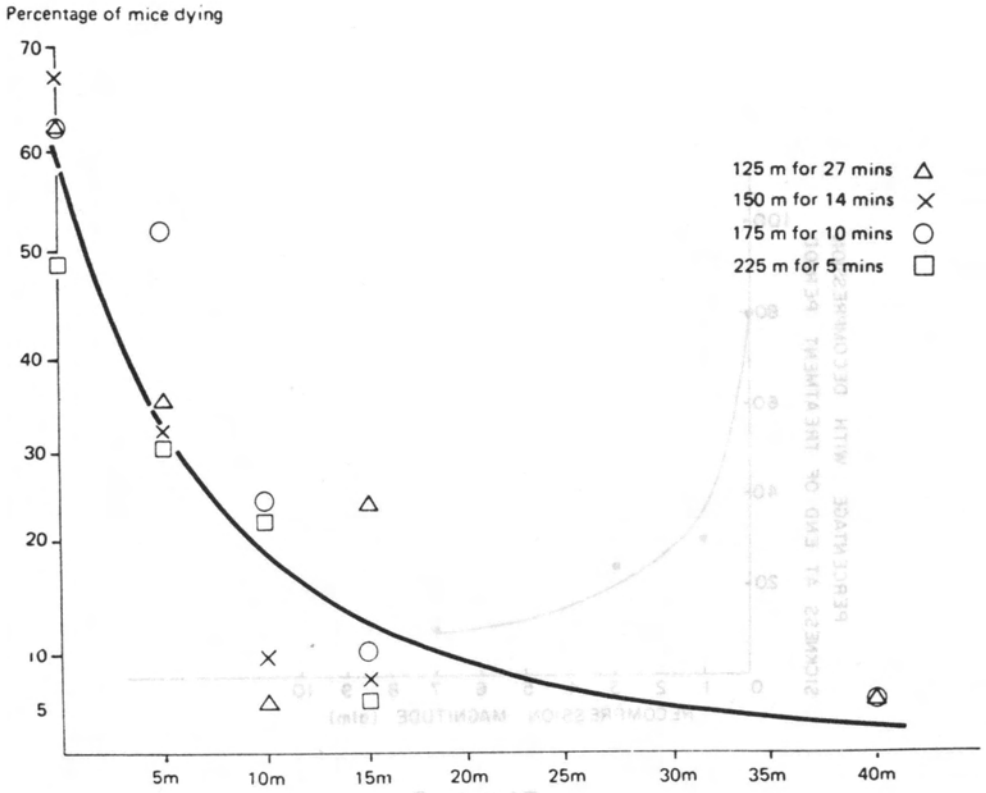


Figure 7

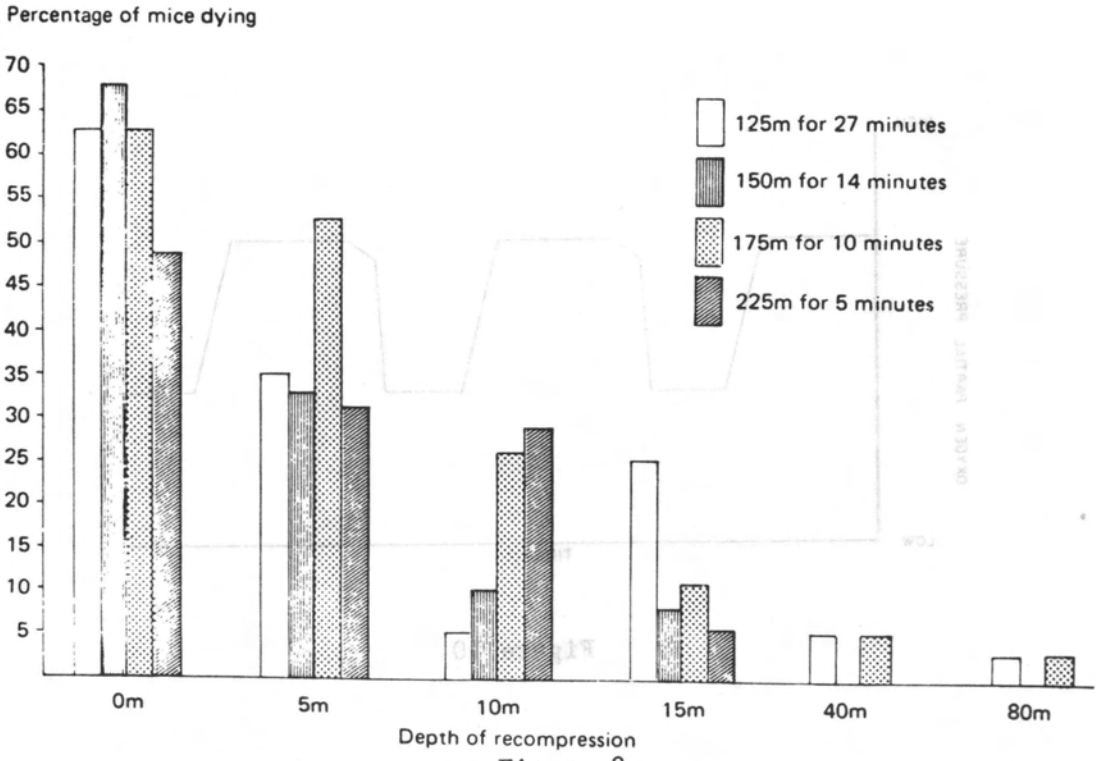


Figure 8

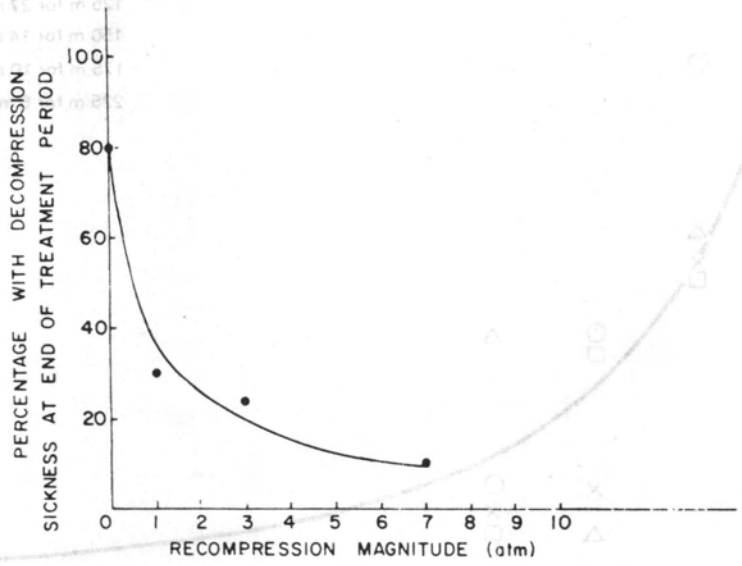


Figure 9

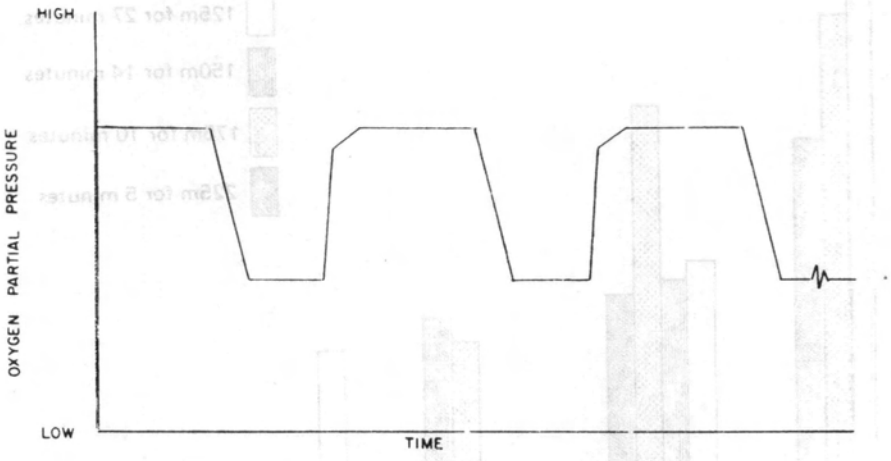


Figure 10

STUDY 3. INTERMITTENCE PROCEDURES FOR POSTPONING OXYGEN TOXICITY

Present treatment procedures call for a sequence of high oxygen partial pressures interrupted by periods of low oxygen (Fig. 10). The most common intermittence relationship used is 20 minutes on high oxygen and 5 minutes on low oxygen. The idea of intermittent high and low oxygen has been incorporated into the treatment of decompression sickness in an effort to postpone the onset of oxygen toxicity. Although there have been two or three experimental studies designed to determine the optimum time for the high and low oxygen periods, the mathematical relationship between the two exposure periods has never been illuminated. We undertook Study 3 in an effort to define this relationship.

Our independent variables (experimental variables) were time on high oxygen and time on low oxygen; our dependent measure was the total time on high oxygen. After 33 dives we have not yet adequately defined the relationship between the two oxygen intervals, but we have discovered that a weighted average of the two intervals provides a good estimate of toxic limits of the intermittent exposures (Fig. 11). If one weighs the individual oxygen partial pressures by their respective exposure times and divides by the sum of the times, one gets the average oxygen partial pressure: $\frac{PO_2 = t_1 (PO_2) + t_2 (PO_2)}{t_1}$. A continuous oxygen exposure has the same toxic effect as a fluctuating oxygen exposure with an equivalent average partial pressure. This is demonstrated in the experimental results plotted in Fig. 12. The solid black dots are the average oxygen partial pressures calculated from the fluctuating oxygen exposures. The "X's" are the ED_{50} toxic values for continuous oxygen exposures. It is apparent that there is very little difference, if any, between the continuous oxygen exposures and the weighted average of the fluctuating oxygen exposures. If we are able to confirm this principle using large animals and man, it will then be possible to equate the toxicity of various intermittent oxygen profiles. Fig. 13 presents the theoretical relationships we might expect for man if the weighted average concept proves to be viable. These relationships are based upon a low oxygen partial pressure of 0.2 ATA.

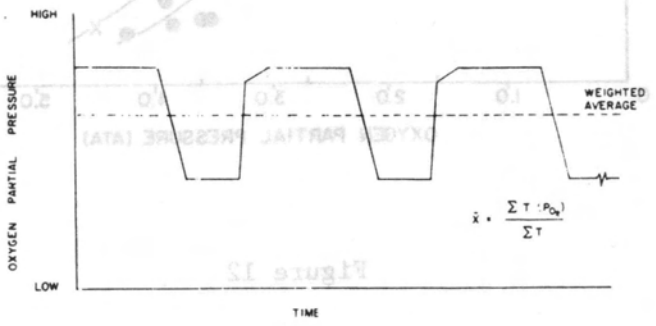


Figure 11

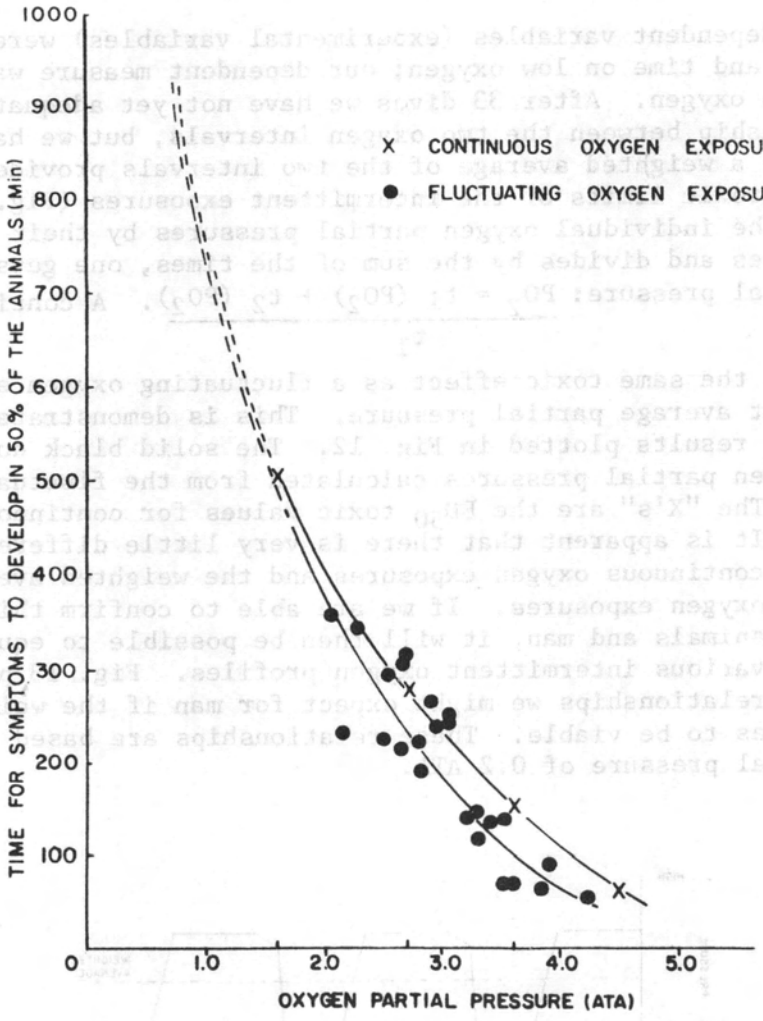

1000
900
800
700
600
500
400
300
200
100
0
TIME FOR SYMPTOMS TO DEVELOP IN 50% OF THE ANIMALS (Min)
X CONTINUOUS OXYGEN EXPOSURE
● FLUCTUATING OXYGEN EXPOSURE
1.0
2.0
3.0
4.0
5.0
OXYGEN PARTIAL PRESSURE (ATA)

Figure 12

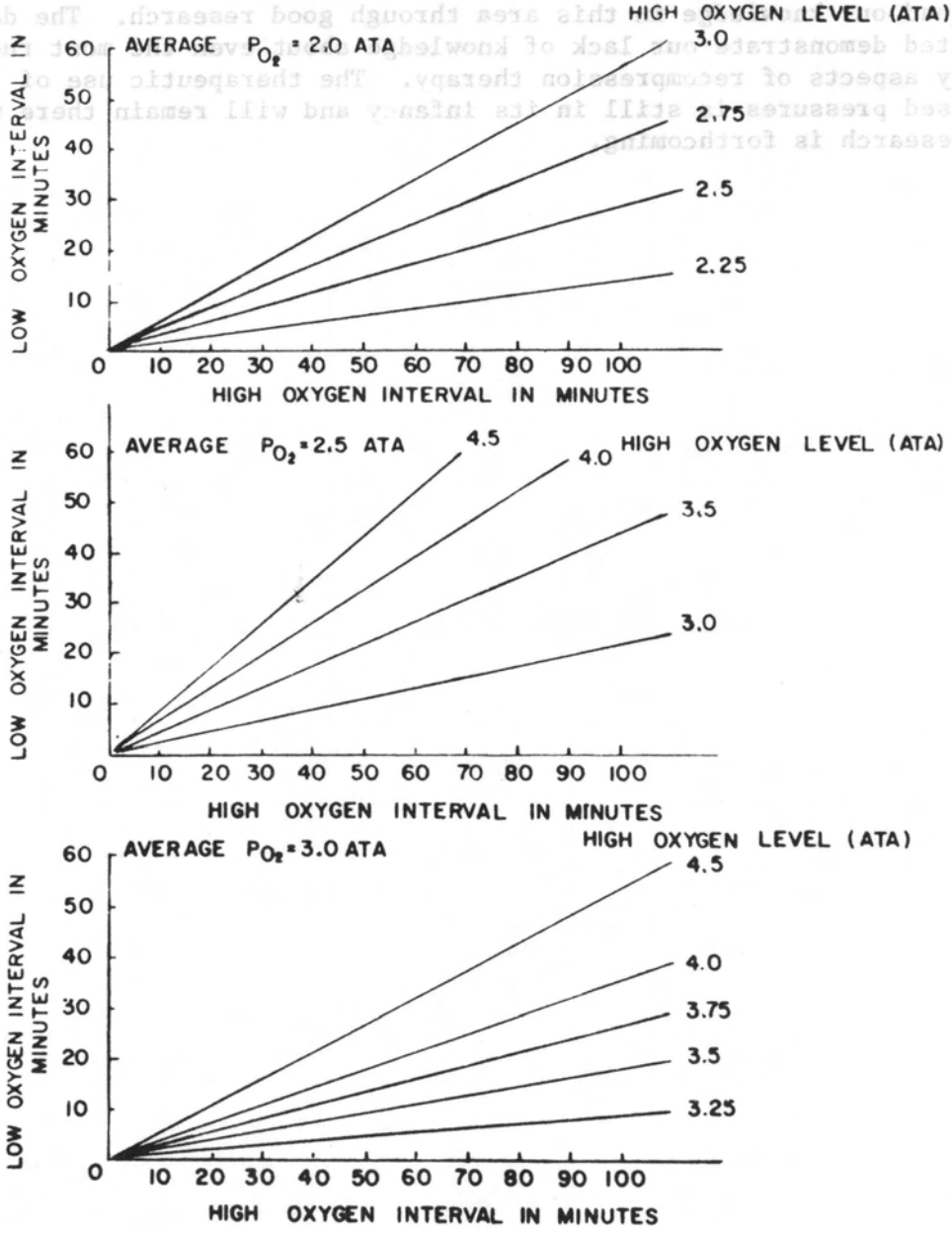
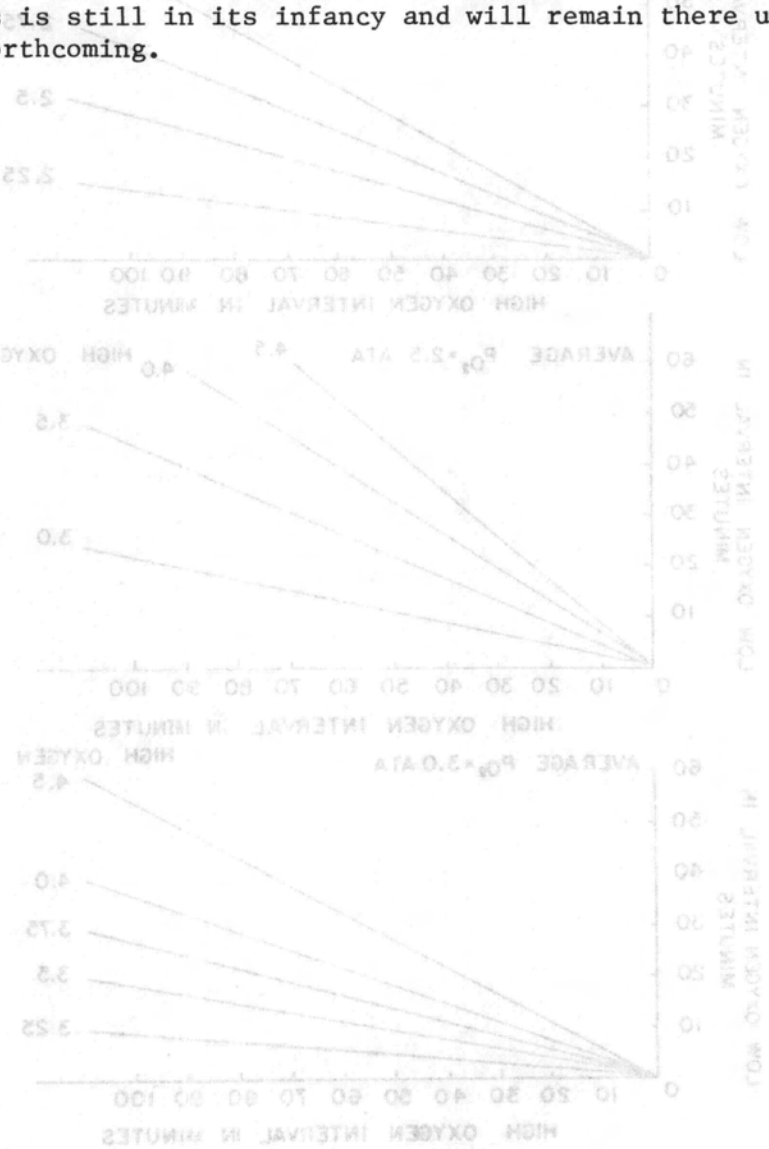

AVERAGE P_{O_2} = 2.0 ATA
HIGH OXYGEN LEVEL (ATA)
3.0
2.75
2.5
2.25
LOW OXYGEN INTERVAL IN MINUTES
HIGH OXYGEN INTERVAL IN MINUTES
AVERAGE P_{O_2} = 2.5 ATA
HIGH OXYGEN LEVEL (ATA)
4.5
4.0
3.5
3.0
LOW OXYGEN INTERVAL IN MINUTES
HIGH OXYGEN INTERVAL IN MINUTES
AVERAGE P_{O_2} = 3.0 ATA
HIGH OXYGEN LEVEL (ATA)
4.5
4.0
3.75
3.5
3.25
LOW OXYGEN INTERVAL IN MINUTES
HIGH OXYGEN INTERVAL IN MINUTES

Figure 13

A brief description of the results of three studies has been presented. A full description of the experimental procedures has been published in Undersea Biomedical Research. I am presenting this preliminary report of these data in an effort to stimulate ideas concerning the treatment of severe decompression sickness. There is a real need to expand our knowledge in this area through good research. The data presented demonstrate our lack of knowledge about even the most rudimentary aspects of recompression therapy. The therapeutic use of increased pressures is still in its infancy and will remain there until good research is forthcoming.



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CAYMAN ISLANDS DIVERS' COMPRESSION CHAMBER

David Myers

The Cayman Islands divers' compression chamber is fairly typical of many diver treatment chambers in remote locations. In some respects, ours is more sophisticated than many. Our facilities are reasonably good, but our limitations are many.

The Cayman Islands are situated south-southwest of Miami, Florida, and west-northwest of Jamaica. The largest of the islands, Grand Cayman, is a mere seven miles by 22 miles at most, and yet it has hundreds of miles of magnificent living reefs around it that range from 10 to hundreds of feet deep. And many of these are only a five-minute swim from shore! We are a "high density" sport diving area; there are over a quarter million sport scuba dives annually, over 95% of which are performed by tourists.

Our chamber building is a 16 x 20 foot concrete block and air-conditioned structure. An 8 x 10 foot mechanical room adjoins the rear of the building. The building's doors are 8 feet wide, enabling an ambulance to back within 6 feet of the chamber hatch, which is wide enough to allow a stretcher to pass. There are two air storage cylinders containing 3500 cubic feet of air at a maximum of 1200 psig. These are filled by a 2.8-cfm compressor. This compressor is one of our weakest links; we hope in the near future to replace it with a much larger unit.

The chamber itself, manufactured by Perry Oceanographics, is 12 feet long by 5 feet in diameter. The inner lock is 8 feet long and the outer lock is 4 feet. Unfortunately, the chamber is not fitted with a medical lock. Each lock has a viewing port, a low-voltage external light, and a sound-powered phone. The inner lock is plumbed for up to four overboard dump oxygen masks, though at present we only own one. Four standard oxygen masks are fitted two to each lock. There are three external pressure gauges with valving to allow the center gauge to be used to cross-check either lock. Additionally there are valves for oxygen supply and exhaust and for a large and accurate battery-operated clock. We do not yet own an oxygen analyzer. All exhaust air and oxygen are plumbed to dump outside the building.

The inner lock has two bunks for patients and two folding stools for attendants. Bedding includes mattresses, pillows, sheets, and blankets, all of fire-resistant material. A medical kit, sand and water buckets, oxygen masks, and a hand-held hose and nozzle water system for fire fighting complete the inner lock fittings.

The low-pressure compressor, used for chamber ventilation, is a 25-cfm 5-hp unit supplying air at 100 psig. The compressed air first enters a 60-gallon receiver tank and then passes through an aftercooler that reduces the air's temperature to ambient and removes entrained moisture through an automatically discharging filter. The air then passes through a refrigerating aftercooler that further reduces its temperature and humidity. Entrained moisture is again collected by an automatically discharging filter. Finally the air passes through a multisorb and charcoal filter to remove impurities, and a small particulate matter filter.

A minimum of four large oxygen cylinders is kept on hand at all times; two are always connected to the chamber via an automatic change-over valve, and the other two are standbys requiring not more than three minutes to put on line.

With the exception of one diving medical doctor, the chamber is staffed entirely by volunteers. Most are experienced divers and a few are nurses. There are at present 22 chamber team members on the island. Operators and attendants attend a lengthy course and must pass rigorous theoretical and practical exams. Thereafter they work as apprentices until they have proven their competence in diagnosis, treatment protocol, medical attending, and chamber operations. Inside chamber attendants need not qualify as operators, but operators must qualify as attendants. At present we have only four senior operators; these are people who have attended at least one Undersea Medical Society sponsored course in diving medicine and have worked on at least 20 treatments, both as operators and attendants. Because all team members hold other full-time jobs, extended and multiple treatments sorely tax our limited personnel resources. We are therefore most anxious to learn of more effective ways of treating serious and life-threatening cases.

During the past six years, we have treated 37 patients, for a total of 58 treatments. All but four were, and remained, asymptomatic after treatments were completed, and all except one were treated on oxygen treatment tables.

Of the four patients with residual symptoms, one had a massive air embolism that was completely clear within one week. The other three patients had spinal cord hits. The first had severe deficiencies of the lower extremities but was not paraplegic; he was able to walk unaided at the conclusion of the treatments. At six-month follow-up, this patient showed only some numbness in the soles of both feet. The second patient was delayed for 29 hours in reaching the chamber and was totally paraplegic. After treatment and two weeks of physiotherapy, he could walk with crutches; at the 15-month follow-up, he showed an apparently permanent limp in the right leg. The third case was 49 hours delayed and totally paraplegic. Two days after termination of treatment with i.v. fluids, daily hyperbaric oxygen, and physiotherapy, he walked over a mile unaided. Physiotherapy continued and at the six-week follow-up

examination, he had only a slight deficiency in the right leg.

Altogether we have treated eight limb-bends cases, 17 neurological decompression sickness cases (one of which was a labyrinthine case) and 11 air embolisms. One of these latter cases was completely phoney and was perpetuated by a man who simply wanted a ride in the chamber. This means that of our legitimate patients, 22.2% had limb bends, 47.2% had neurological DCS, and 30.6% had air embolisms.

One final comparison. Previously in this workshop, Captain Spaur reported that the USN annually conducts about 70,000 dives and has about 70 diving accidents of all types. Even if all of these accidents were decompression sickness or gas embolism, the percentage would work out to an admirable 0.1%. Last year's dives in the Cayman Islands totalled around 250,000, and with eight chamber cases, that works out to 0.0032%! I think this speaks rather well of the local dive masters and of sport scuba divers' training.

WORKSHOP CONCLUSIONS

J. C. Davis, M.D., Chairman

The difficult nature of the delayed cases of decompression sickness (DCS) and arterial gas embolism (AGE) considered by participants of this workshop makes it possible that the current estimate of a treatment failure rate of 15 to 20% may be irreducible. At some point, some of the patients from this failure group suffered irreversible neurologic tissue damage or irreversible shock, and no treatment regimen could then have been successful. Regarding chamber treatment, it is also important to remember that standard, established treatment tables today have a success rate of 80 to 85% using pressure, oxygen, time, and adjunctive drug and fluid therapy on established schedules, even in cases that have long delays before reaching the chamber. This may be the best possible outcome, but this workshop group looked for areas that might be improved upon.

There were certain areas of agreement among workshop participants. The overriding conclusion may appear obvious, but experience with treatment failures suggests that it should be emphasized: serious decompression sickness and arterial gas embolism deserve to be managed with comprehensive, current medical care administered under the direction of a trained physician. Many of the advances in modern intravenous fluid therapy, advanced life support, diagnostic laboratory studies, acid-base balance, and drug therapy have not yet generally been incorporated into the treatment of these serious multi-organ disorders. It is true that most cases of decompression sickness treated immediately respond to pressure and oxygen on standard treatment schedules, but delayed cases, especially those with neurologic or vascular involvement, deserve consultation with a physician experienced in diving medicine and intensive care. Because delayed arrival at a recompression chamber accounts for the most serious cases, the majority of such cases are sport scuba divers. Certainly in DCS and AGE, compression therapy is the central and mandatory treatment, proved highly effective by long years of experience. With the exception of preliminary studies such as those by Wells (Underwater Physiology Symposium Proceedings, 1976, 1978) on the value of intravenous fluid therapy of DCS, data supporting fluid and drug therapy generally do not exist. On the other hand, deductions from pathophysiological studies and clinical experience point toward more vigorous and intensive medical management throughout the treatment, from accident scene, during transport, and in the chamber. Based on the papers presented, workshop discussions, and the recommendations of task groups comprised of workshop attendees, the following recommendations are offered.

Immediate care of decompression sickness and arterial gas embolism when a compression chamber is not on-scene. Which treatment procedures are selected from the following list will depend on the level of medical expertise and supplies available. With the exception of the time needed to perform immediate life-saving procedures, nothing should be allowed to delay the fastest possible transportation to the nearest compression chamber. En route, the following procedures should be observed.

1. Cardiopulmonary resuscitation, if required.

2. Patient supine or head low, in the left side position for suspected arterial gas embolism until at treatment pressure in a compression chamber. It is important to ensure an open airway and to prevent aspiration of vomitus.

3. One hundred percent oxygen should be administered by mask or endotracheal tube. When a mask is used, it is only possible to deliver nearly 100% oxygen with a tight double-seal mask (aviation or anesthesia mask). Continuous 100% oxygen administration and maintenance of intravascular volume are considered the most important features of immediate treatment, and should be continued until reaching the chamber.

4. In conscious patients, give oral non-alcoholic liquids such as fruit juices or balanced electrolyte beverages (one liter in the first hour). Urine output should be kept at 1 or 2 ml/kg/hr the first hour).

5. In unconscious patients and in patients with any manifestation more serious than limb bends, intravenous fluid replacement is preferred. Ringer's lactate, normal saline, or 5% dextrose in saline should be given to maintain urine output at 1-2 ml/kg/hr.

6. With spinal cord involvement, an in-dwelling urinary catheter should be considered.

7. For any neurologic involvement, give hydrocortisone hemisuccinate, 1.0 gm i.v., or dexamethasone, 20-30 mgm i.v.

8. For intractable vomiting or unmanageable agitation, intravenous Valium® can be infused through a flowing i.v. Direct intravenous injection will produce phlebitis. Remember that Valium will mask symptoms and signs of labyrinthine involvement.

9. For its anti-platelet activity, 0.5 - 1.0 gm of oral aspirin has been suggested, but opinion is divided regarding its efficacy. Except for the risk of reaction in hypersensitive

individuals and the risk of gastric irritation, the safety of this drug makes this recommendation reasonable.

10. Early contact with the compression chamber to which the patient is to be transported. This is mandatory to be sure the chamber is available and a crew is assembled and awaiting the patient's arrival.

11. Mode of transportation. A major determinant of successful outcome is shortening the time from onset to compression chamber treatment. If the distance is too great for surface transportation, air evacuation must be used. It is critically important that the patient not be exposed to decreased barometric pressure at altitude. Several aircraft are capable of maintaining sea level cabin pressure up to flight altitudes as great as 18,000 feet or higher. Such aircraft are: the military C-9 aeromedical evacuation jet aircraft, the C-130 Hercules, Boeing 707, 727, 737, and 747, DC-8 and DC-10, BAC-111, Cessna Citation, and Learjet (preferred for its speed and pressurization system). If the patient is moved by helicopter, the pilots must be instructed to keep the flight altitude as low as safely possible but not greater than 500-800 feet above ground level. One hundred percent oxygen and fluid therapy must be maintained in-flight.

12. The workshop addressed the question of treatment of DCS and AGE in monoplace hyperbaric oxygen chambers of the type currently in use about the world to treat certain non-diving disorders. The consensus workshop statement is that the use of such chambers is severely limited for treatment of DCS and AGE. Reasons can be summarized:

    (a) Treatment time and depth are limited in such chambers by oxygen toxicity, patient isolation, and structural constraints. The decision to keep the difficult case under pressure for hours or days cannot therefore be made.

    (b) The current thrust of DCS and AGE treatment is to perform diagnostic procedures and institute intensive care while the patient is under pressure, to prepare him/her for eventual decompression.

    (c) Effective initial and en route adjuvant treatment, such as that delineated in Dr. Fructus' workshop paper, can be carried out during transportation to a multiplace chamber.

    Some members of the workshop suggest that, in the rare instance where a life-threatening emergency occurs in the immediate vicinity of a monoplace oxygen chamber but far from a multiperson chamber, the attending physician might

choose to treat in the monoplace chamber. However, the majority of workshop participants consider the monoplace oxygen chamber inadequate for definitive treatment of DCS and AGE. (This statement is not meant to detract from the valuable work in hyperbaric oxygen therapy being conducted in hospitals using monoplace hyperbaric oxygen chambers for certain non-diving disorders.)

Portable chambers for transporting patients from remote diving sites to adequate compression chambers were not discussed; these chambers are generally used only by the Navy and commercial divers.

13. The workshop members also considered the underwater oxygen treatment of decompression sickness procedures being used in regions of the Indo-Pacific. In remote conditions, with expert and experienced personnel, and when procedures have been fully planned and the proper equipment is at hand, workshop members recognize that the technique has value. The warm waters in that part of the world also reduce the problem of hypothermia that would be encountered in the Caribbean or other sub-tropical waters. There are recognized and significant risks involved in this technique, however, and the workshop could not recommend its widespread use. Certainly, many tragic experiences resulting from attempts to administer compressed air in-water recompression therapy dictate against the use of this technique.

COMPRESSION CHAMBER TREATMENT

This workshop did not consider treatment of decompression sickness occurring in saturation or deep mixed-gas diving, or the treatment of AGE and DCS in sophisticated chambers with great depth and life-support capability. The equipment and staff available in these situations make it possible to use the depth, gas mixtures, and therapeutic saturation decompression needed to achieve the best results possible.

The workshop also did not focus on cases occurring in naval and commercial diving because standard diving practices, immediate treatment in on-scene compression chambers, and treatment using established treatment tables provide excellent results.

The workshop group concentrated on sport, scientific, and "semi-professional" divers, all of whom often dive in areas remote both in distance and time from compression chambers. Cases drawn from these groups of patients were presented in the papers by Davis, Leitch and Fructus. Further, workshop consideration was limited to those situations in which treatment was conducted in a compressed air chamber with the treatment gas supplied by a built-in breathing system (BIBS).

In the United States, most treatment is conducted according to U.S. Navy Treatment Tables 5, 6, or 6A, as described in Spaur's paper. Even in cases delayed by several hours in reaching the chamber, these schedules have an 80 to 85% success rate. Thus, the workshop participants do not recommend changes in the initial treatment flowcharts; instead, the group directed attention toward possible options for those situations in which the patient fails to respond to established procedures. Besides those charts presented in this workshop and in the paper by Miller (Appendix 1), there are many effective flowcharts in use about the world. The participants attempted to identify problems that might arise on following any flowchart and to offer options. Specific problems considered were:

a. The patient at 165 fsw who either has not responded to treatment after 30 minutes on Table 6A or who deteriorates on an attempt to decompress to 60 fsw. Possible treatment options for this situation include the following:

(1) Remain at 165 fsw for up to two hours and, if the patient responds well to fluid resuscitation and steroids, decompress to 60 fsw according to USN Table 4, enter Table 6, and use with all extensions. An air-breathing tender must be locked in at 60 fsw because the original tender must be treated with oxygen on an extended Table 6 with the patient.

(2) If the patient has not responded satisfactorily by two hours at 165 fsw, the nitrogen-oxygen saturation method of Miller, Fagraeus, Bennett, Elliott, Shields, and Grimstad (App. 1) should be considered. The equipment and gas supplies needed to adapt a compressed air chamber and the treatment schedules used are set forth in Appendix 1, a reprint of these authors' paper in *Lancet*.

(3) The use of a 50% oxygen-50% nitrogen breathing mixture at 165 fsw during Table 6A or intermittently up to two hours. This mixture is equivalent to breathing 100% oxygen at 3 ATA.

b. Patient at 60 fsw on Table 6 or 6A who fails to respond satisfactorily or who deteriorates on attempted decompression despite maximum possible oxygen extensions at 60 fsw. Possible treatment options include the following:

(1) Remain at 60 fsw for an indefinite period breathing chamber air with oxygen breathing periods as needed to avoid oxygen poisoning until maximum benefit has apparently been achieved, and then decompress on an air saturation decompression table. A CO_2 scrubber reduces the ventilation

requirement in a compressed air chamber, and an oxygen analyzer is needed to ensure a safe oxygen level in the chamber.

(2) Another option was considered for the patient in a compressed air chamber who fails to respond at 60 fsw on Table 6. The patient is compressed to 100 fsw breathing chamber air and, preferably, given 50-50 nitrogen-oxygen by BIBS for 45 minutes according to the COMEX Therapeutic Table CX 30 (Appendix 2). If 50-50 nitrogen-oxygen is not available, the patient breathes chamber air according to COMEX Therapeutic Table CX 30A (Appendix 3) for 60 minutes at 100 fsw. If significant relief is achieved, the patient can be decompressed according to these COMEX tables to 60 fsw, where USN Table 6 is again followed. Again, the tender should breathe oxygen during decompression according to Table 6. The only special requirement is a supply of 50-50 nitrogen-oxygen.

(3) If the patient does not achieve significant relief or is slowly improving at 100 fsw, the option remains to stay at 100 fsw, use pure nitrogen to drop the chamber atmosphere PO_2 to 0.5 bar (according to the Appendix 1 table), and continue fluid resuscitation and steroids for as long as needed, and then to decompress according to the schedule on the last page of Appendix 1.

c. Patient who responds satisfactorily or partially to initial treatment on Tables 6 or 6A, then has a recurrence within a few hours after surfacing or has a residual deficit.

(1) Table 6 can be repeated for recurrence after surfacing from the first treatment dive. Residual manifestations have been treated once or twice daily with Table 5 or 6 and continuation of steroid therapy and physiotherapy. This daily hyperbaric oxygen therapy is continued as long as definite improvement can be seen at depth breathing oxygen with each treatment. Proof is lacking that these repeated hyperbaric oxygen treatments improve the long-term outcome, but experience seems to be accumulating that this regimen hastens recovery and leads in some cases to a better outcome.

d. Patient with persistent serious manifestations at depth but with pulmonary oxygen poisoning due to preceding circumstances.

Use nitrogen-oxygen saturation therapy (see Appendix 1).

Note: The availability of telephone or radio communication from any chamber in the world makes it possible to obtain assistance in difficult cases. Even experienced diving medicine physicians frequently call for other opinions.

These treatment options are not intended to replace any standard treatment method.

Compressed air chamber facilities that treat DCS and AGE may wish to prepare in advance. Routine USN tables require at least a compressed air chamber with a capability of 165 fsw and 100% oxygen BIBS. An overboard dump and a chamber environment oxygen analyzer are recommended. For a treatment facility to be able to choose any of the options suggested in this workshop, a source of pure nitrogen, 50-50 nitrogen-oxygen, and a CO_2 scrubber are also needed. Workshop participants had strong concerns about keeping a source of pure nitrogen at the chamber site because of the possibility that pure nitrogen might accidentally be plumbed to the BIBS. If a facility wishes to maintain nitrogen availability, this gas should be stored in a place other than the chamber area. It was recommended that any gas in the chamber area be respirable to avoid any risk of an error.

SUMMARY

This workshop dealt with the treatment of decompression sickness and arterial gas embolism cases with delayed arrival at compression chambers and those cases that fail to respond satisfactorily to standard treatment tables. Most cases respond well to established treatment schedules. The treatment aspects most likely to be amenable to improvement occur at present in the interval required to transport victims to adequate compression chambers and the intensive medical care administered initially and en route. Oxygen inhalation, steroids to reduce CNS edema, and fluid therapy to maintain intravascular volume and perfusion are stressed.

Though pressure, time, and oxygen administered according to established treatment schedules are usually effective when used early in the treatment of DCS and AGE, delayed cases must be viewed as serious multiorgan disorders. Compression is the cornerstone of therapy, but vigorous intensive care by a physician -- including ongoing diagnostic studies, fluid resuscitation, maintenance of acid-base balance, the administration of steroids, and other intensive care required during compression therapy -- is also of the utmost importance.

Finally, the diving world must be informed that prevention is still the key. Decompression sickness and arterial gas embolism are serious disorders that sometimes cannot be corrected no matter what the treatment. There is a tendency to believe that any case arriving at a chamber alive can be cured. This erroneous impression fails to recognize that irreversible changes in nervous or cardiovascular systems cannot be undone. Despite any known treatment, in some cases there will be treatment failures or incomplete recovery.

APPENDIXES

APPENDIX 1

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NITROGEN-OXYGEN SATURATION THERAPY IN SERIOUS CASES OF COMPRESSED-AIR DECOMPRESSION SICKNESS

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Summary Decompression sickness and arterial air embolism which follow exposure to raised environmental pressures of compressed air are usually adequately treated by accepted recompression procedures of relatively short durations. With serious cases, however, conventional treatment may not allow sufficient time at depth for the complete resolution of manifestations because of the need to avoid pulmonary oxygen toxicity which is associated with a prolonged period of breathing compressed air. Treatment by nitrogen-oxygen saturation at a pressure equivalent of 30 m (100 ft) sea water is proposed. Based upon the success of three refractory cases treated by this procedure, recommendations are made for the conversion of standard compressed-air chambers into an emergency saturation mode for therapy.

Introduction

THE treatment of acute decompression sickness following compressed-air diving is in the first instance by immediate recompression by the raising of environmental pressure to 18 m (60 ft) while breathing oxygen.[1] This usually provides immediate and complete relief of symptoms, although some refractory cases may require either a fairly prolonged duration at this pressure or further recompression to 50 m (165 ft). The treatment of arterial air embolism arising from pulmonary barotrauma is also by immediate recompression, but directly to 50 m (165 ft) on compressed air. If treated rapidly the majority of such cases respond promptly and the subsequent decompression is by accepted treatment tables.[2,3]

Problems can arise with both decompression sickness and air embolism when the response to recompression is not complete. Recognised therapeutic tables do not allow more than 2 h at 50 m (165 ft) because of the risk of inducing pulmonary oxygen toxicity when breathing compressed-air (Po_2=1·26 bar), and because of the lack of adequately proven decompression procedures.

There are several possible options. The first is to adhere to accepted treatment tables at the risk of a worsening in the patient's clinical state during decompresion, or possibly of death.[4] After surfacing with residual symptoms the patient may then be given repeated shallow hyperbaric oxygen treatments for some days.

Alternatively, if the duration at 50 m (165 ft) is extended beyond 2 h, perhaps up to 4 h, the hazard of pulmonary oxygen toxicity is increased and the risks associated with decompressing the incompletely cured diver persist.

With still further recompression on air, either to the depth of relief or to 70 m (230 ft), the risk of pulmonary oxygen toxicity is further increased both at depth and during the subsequent decompression on air. Thus the duration at maximum depth is restricted to 30 min (R.N. table 71).

If the breathing medium is changed from air to an appropriate helium-oxygen mixture in order to go deeper, it is possible to recompress to the depth of relief. This has occasionally been associated with complications due probably to counter-diffusion between helium and nitrogen.[5] The initial worsening of symptoms seen with a change from helium-oxygen to compressed air during the treatment of a helium-oxygen diver, has subsequently required extreme recompression followed by prolonged treatment and decompression.[6]

Another possible method of avoiding pulmonary oxygen toxicity is to reduce the partial pressure of oxygen by using a nitrogen-oxygen mixture. This gives unlimited time for significant symptom relief at a convenient depth. Such an approach is possible with techniques commonly used in helium-oxygen diving and is the subject of this paper.

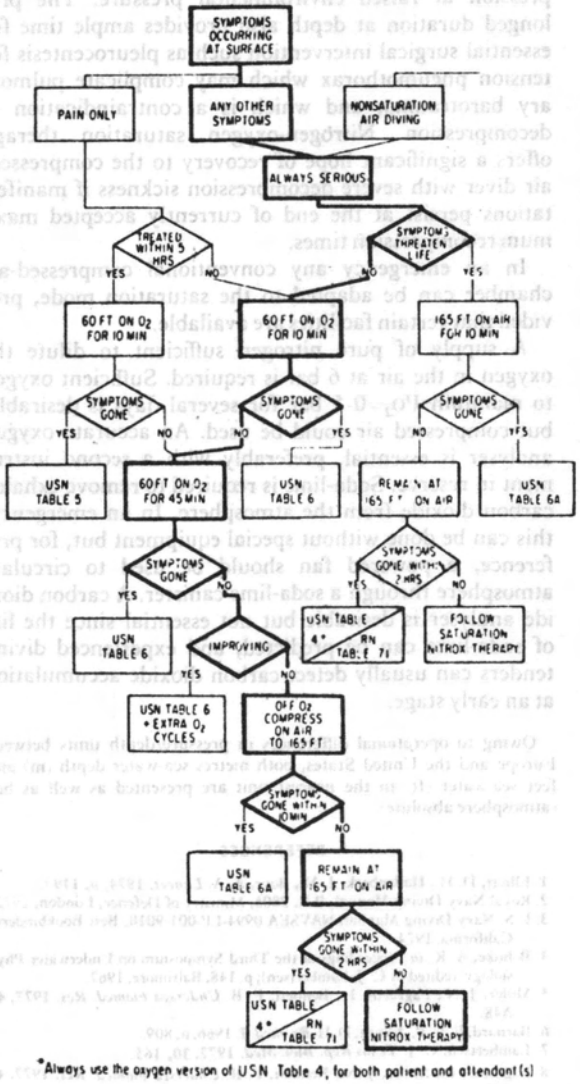
Case-reports

Case 1

In January, 1977, a 23-year-old commercial diver carried

out a 36 m (115 ft) dive in Scotland breathing compressed-air from a diving bell for 27 min. His decompression according to accepted diving tables included a transfer under pressure to a deck chamber at 6 m (20 ft). Because of a faulty hatch seal he was accidentally decompressed twice to atmospheric pressure after which chest pain and a marked weakness of his right leg developed. During subsequent oxygen treatment at 18 m (60 ft) his symptoms, which had initially regressed, recurred and worsened, necessitating further recompression with air to 50 m (165 ft) according to the U.S.N. table 6A (30 min at 50 m) but with no relief of his symptoms. After his return to 18 m (60 ft) he became paraplegic below T_4.

The patient was recompressed to 50 m (165 ft) breathing air for 2 h during which time adjuvant therapy was started, including dexamethasone 12 mg intravenously followed by 6 mg intravenously every 6 h, 500 ml dextran-70 4-hourly, and urinary bladder catheterisation. Decompression on R.N. table 71 was made to 30 m (100 ft) on compressed air when examination by a consultant diving physician some 22 h after the incident revealed a complete paralysis of the right lower limb, partial paralysis of the left, paralysis of the rectus abdominis, and paræsthesia below T_{10}. There was also very slight nystagmus to the right and weakness of the small muscles of both hands.



*Always use the oxygen version of USN Table 4, for both patient and attendant(s)

Fig. 1—Segment of binary decision tree in compressed-air decompression sickness.

Decision pathway outlined for case 2.

However, shallow, rapid respiration and severe burning retrosternal pain indicated that pulmonary oxygen toxicity had reached significant proportions. The patient's attendant, too, had similar symptoms of pulmonary oxygen toxicity. Reduction of chamber Po_2 from 0·75 to 0·35 bar was followed by rapid improvement in the pulmonary condition of both patient and attendant.

Decompression began on a nitrogen-oxygen saturation table[7] and a slow improvement in his neurological state continued throughout the decompression. On arrival at the surface, about 75 h after the incident, strength was returning to the left lower limb and movement of all muscle groups except tibialis anterior was present on the right. Sensory symptoms had improved considerably. He was then treated with daily hyperbaric oxygen for 4 days (one R.N. table 61, and three 2-h periods at 2·0 bar) with further improvement. Subsequently, he regained normal bladder function and reasonable strength in his left lower limb, but still had some weakness on the right. He could walk well with crutches, and over the succeeding year has shown progressive but slow improvement.

Case 2

A 40-year-old diving instructor with a past medical history of partial lung resection in childhood and some weeks in hospital in 1976 for low back pain performed a single air dive in Florida to 80 ft (24 m) for 22 min in August 1977. Within several minutes of surfacing he had tingling and significant weakness in both legs. He was recompressed on oxygen to 60 ft (18 m) for an extended U.S.N. table 6 with apparently good results. Within minutes of surfacing from this therapy his symptoms recurred. They became worse and were complicated by paralysis of the bladder. He was then flown at a low altitude 500 miles (800 km) to Duke University Medical Center, where he arrived 18 h after his dive. Upon his arrival, clinical examination revealed signs of a lesion of the spinal cord from T_{10} to S_5.

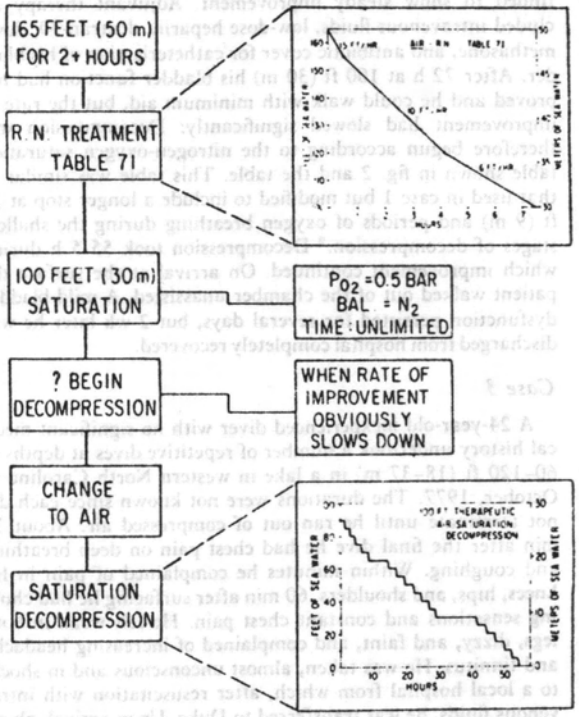


Fig. 2—Procedures used in treatment of cases 2 and 3.

The management of case 1 was more complex and the gas mixtures used during saturation decompression contained a constant Po_2=0·35 bar to alleviate pulmonary oxygen toxicity.

DECOMPRESSION TABLE USED FOR CASE 2

Depth		Time at stop	Breathing mixture
Feet	Metres	(h : min)	
100	30·0	30 min to 90 ft	Air
90	27·0	00:50	,,
85	25·5	01:20	,,
80	24·0	01:30	,,
75	22·5	01:40	,,
70	21·0	01:50	,,
65	19·5	02:00	,,
60	18·0	06:00	,,
55	16·5	02:20	,,
50	15·0	02:40	,,
45	13·5	02:40	,,
40	12·0	00:10	Oxygen
40	12·0	02:30	Air
35	10·5	02:30	,,
30	9·0	12:00	,,
25	7·5	02:00	Oxygen/air*
20	6·0	02:20	Air
15	4·5	02:40	Oxygen/air*
10	3·0	02:30	Air
5	1·5	02:40	,,

Total decompression time—55 h 30 min.
Decompression time between stops=10 min.
*Oxygen delivered in 4 recurrent cycles—25 min O_2/5 min air.

The sequence of treatment followed a binary decision tree (fig. 1) developed to engender a more rational approach to the treatment of decompression sickness.[8] At 60 ft (18 m) on 100% oxygen there was no improvement after 10 min and still none after 45 min. He was then further recompressed to 165 ft (50 m) on air. After 30 min at this depth some improvement was noted but after 4 h at 165 ft (50 m) there was insufficient improvement to warrant conventional decompression. Accordingly, the patient was slowly decompressed using R.N. table 71 to 100 ft (30 m) without any deterioration. He was then kept at 100 ft (30 m) with an inspired Po_2=0·5 bar, and he continued to show steady improvement. Adjuvant therapy included intravenous fluids, low-dose heparin, dextran-70, dexamethasone, and antibiotic cover for catheterisation of his bladder. After 72 h at 100 ft (30 m) his bladder function had improved and he could walk with minimum aid, but the rate of improvement had slowed significantly. Decompression was therefore begun according to the nitrogen-oxygen saturation table shown in fig. 2 and the table. This table was similar to that used in case 1 but modified to include a longer stop at 30 ft (9 m) and periods of oxygen breathing during the shallow stages of decompression.[9] Decompression took 55·5 h during which improvement continued. On arrival at the surface the patient walked out of the chamber unassisted. A mild bladder dysfunction persisted for several days, but 2 wk later he was discharged from hospital completely recovered.

Case 3

A 24-year-old inexperienced diver with no significant medical history undertook a number of repetitive dives at depths of 60–120 ft (18–37 m) in a lake in western North Carolina in October, 1977. The durations were not known since each did not terminate until he ran out of compressed air. About 30 min after the final dive he had chest pain on deep breathing and coughing. Within minutes he complained of pain in his knees, hips, and shoulders. 60 min after surfacing he had choking sensations and constant chest pain. He felt weak in both legs, dizzy, and faint, and complained of increasing headache and tinnitus. He was taken, almost unconscious and in shock, to a local hospital from which, after resuscitation with intravenous fluids, he was transferred to Duke. Upon arrival, about 8 h after the final dive, he had severe generalised decompression sickness including numbness of both legs, marked weakness of the left, and complete paralysis of the right hip and leg.

Initial treatment again followed the binary decision tree but, because of the severity of symptoms, the patient was taken directly to 165 ft (50 m), where over the next 2 h his general condition improved considerably. Since the patient had severe residual localised signs nitrogen-oxygen treatment was started. Accordingly, he was slowly decompressed on R.N. table 71 breathing compressed-air to 100 ft (30 m) without any deterioration. Once at 100 ft the inspired Po_2 was reduced to 0·5 bar. The patient improved, but more slowly than did case 2. A similar adjuvant therapeutic regimen was given. Decompression, again lasting 55·5 h, was unventful. The patient surfaced still with a marked paralysis of his right leg and some weakness of his left, but with resolution of all other symptoms. 2 months later, recovery was complete.

Discussion

The results obtained in each of the three patients treated with the nitrogen-oxygen regimen compare favourably with past experience in which permanent residual injury or death has resulted. The use of nitrogen-oxygen mixtures with an inspired Po_2 of 0·5 bar or less allows a potentially indefinite duration of recompression at raised environmental pressure. The prolonged duration at depth also provides ample time for essential surgical intervention such as pleurocentesis for tension pneumothorax which may complicate pulmonary barotrauma, and which is a contraindication to decompression. Nitrogen-oxygen saturation therapy offers a significant hope of recovery to the compressed-air diver with severe decompression sickness if manifestations persist at the end of currently accepted maximum recompression times.

In an emergency any conventional compressed-air chamber can be adapted to the saturation mode, provided that certain facilities are available.

A supply of pure nitrogen sufficient to dilute the oxygen in the air at 6 bar is required. Sufficient oxygen to maintain Po_2=0·5 bar for several days is desirable, but compressed air could be used. An accurate oxygen analyser is essential, preferably with a second instrument in reserve. Soda-lime is required to remove exhaled carbon dioxide from the atmosphere. In an emergency, this can be done without special equipment but, for preference, a powered fan should be used to circulate atmosphere through a soda-lime canister. A carbon dioxide analyser is desirable but not essential since the life of soda-lime can be predicted, and experienced diving tenders can usually detect carbon dioxide accumulation at an early stage.

Owing to operational differences in pressure/depth units between Europe and the United States, both metres sea-water depth (m) and feet sea water (ft) to the nearest unit are presented as well as bar (atmosphere absolute).

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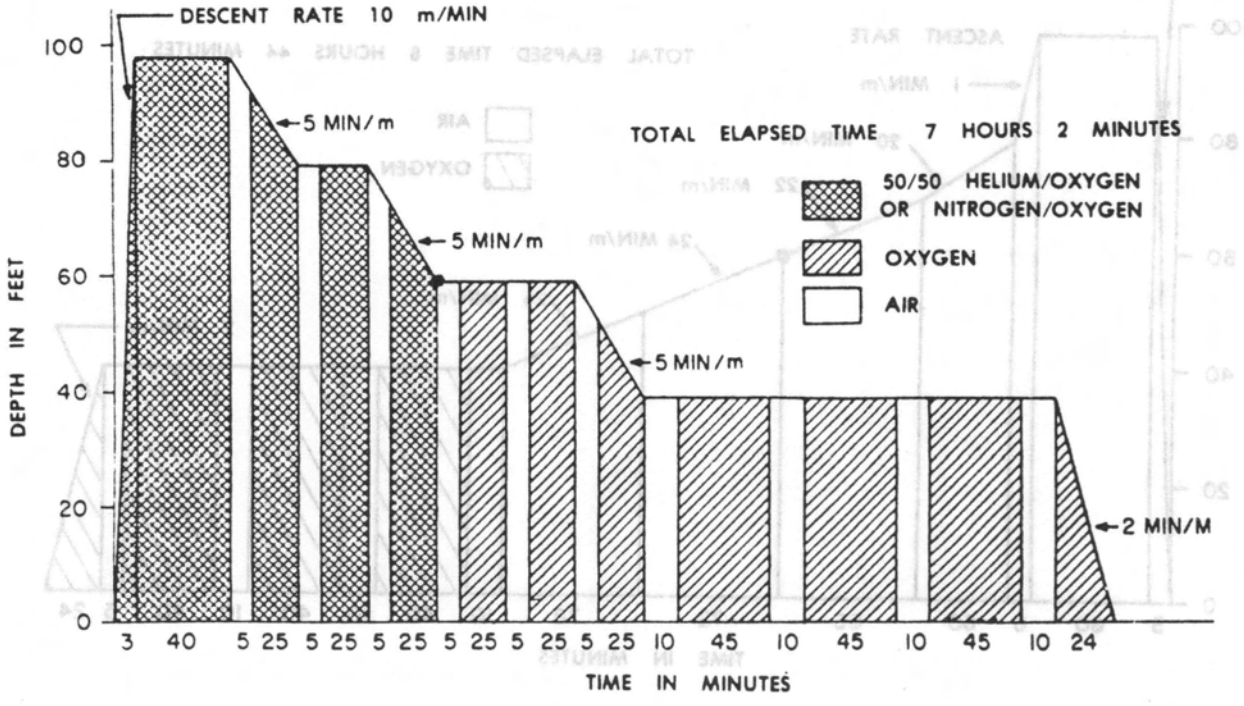
APPENDIX 2

COMEX THERAPEUTIC TABLE CX 30*

1. Use--treatment of vestibular and general neurological decompression sickness occurring after either a normal or shortened decompression.
2. Descent rate--as quickly as possible, in 2 or 3 minutes.
3. Ascent rate--between 30 and 24 meters--5 min/m
   24 and 18 meters--5 min/m
   18 and 12 meters--5 min/m
   12 and 0 meters-- 2 min/m
4. Time at 30 meters does not include the compression time.

*COMEX (1976).

Depth (ft)	Depth (meters)	Time (min)	Breathing media	Total elapsed time (hours)	(min)
98	30	40	50/50		43
98-79	30-24	30 (5 / 25)	Air / 50/50	1	13
79	24	5	Air	1	18
79	24	25	50/50	1	43
79-59	24-18	30 (5 / 25)	Air / 50/50	2	13
59	18	5	Air	2	18
59	18	25	Oxygen	2	43
59	18	5	Air	2	48
59	18	25	Oxygen	3	13
59-39	18-12	30 (5 / 25)	Air / Oxygen	3	43
39	12	10	Air	3	53
39	12	45	Oxygen	4	38
39	12	10	Air	4	48
39	12	45	Oxygen	5	33
39	12	10	Air	5	43
39	12	45	Oxygen	6	28
39	12	10	Air	6	38
39-0	12-0	24	Oxygen	7	2



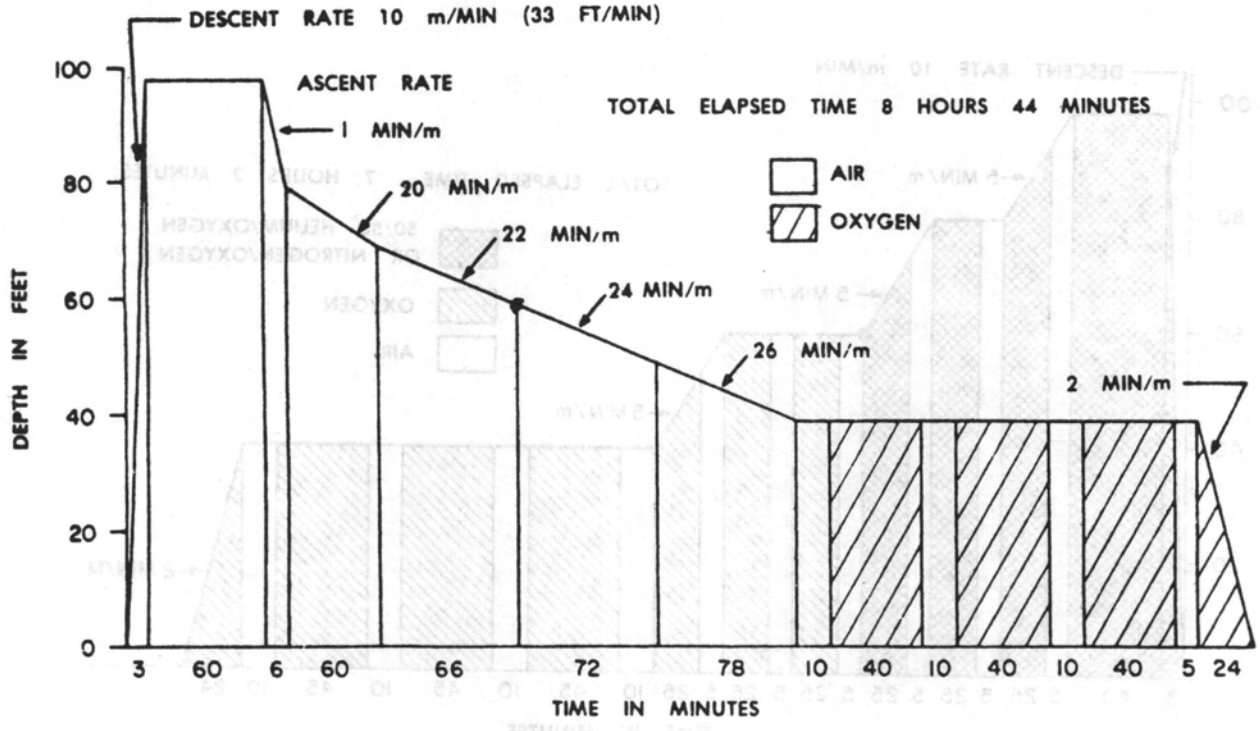
APPENDIX 3

COMEX THERAPEUTIC TABLE CX 30 A*

1. Use--treatment of musculoskeletal decompression sickness when signs of oxygen poisoning are present.
2. Descent rate--as quickly or possible using air, 2 to 3 minutes.
3. Ascent rate--continuous ascent at the rates shown below.
4. Time at 30 meters (98 ft) does not include the compression time.

*COMEX (1976).

Depth (ft)	Depth (meters)	Time (hours)	Time (min)	Rate min/meter	Breathing media	Total elapsed time (hours)	Total elapsed time (min)
98	30	1		-	Air	1	3
98-79	30-24		6	1	Air	1	9
79-69	24-21	1		20	Air	2	9
69-59	21-18	1	6	22	Air	3	15
59-49	18-15	1	12	24	Air	4	27
49-39	15-12	1	18	26	Air	5	45
39	12		10	-	Air	5	55
39	12		40	-	Oxygen	6	35
39	12		10	-	Air	6	45
39	12		40	-	Oxygen	7	25
39	12		10	-	Air	7	35
39	12		40	-	Oxygen	8	15
39	12		5	-	Air	8	20
39-0	12-0		24	2	Oxygen	8	44



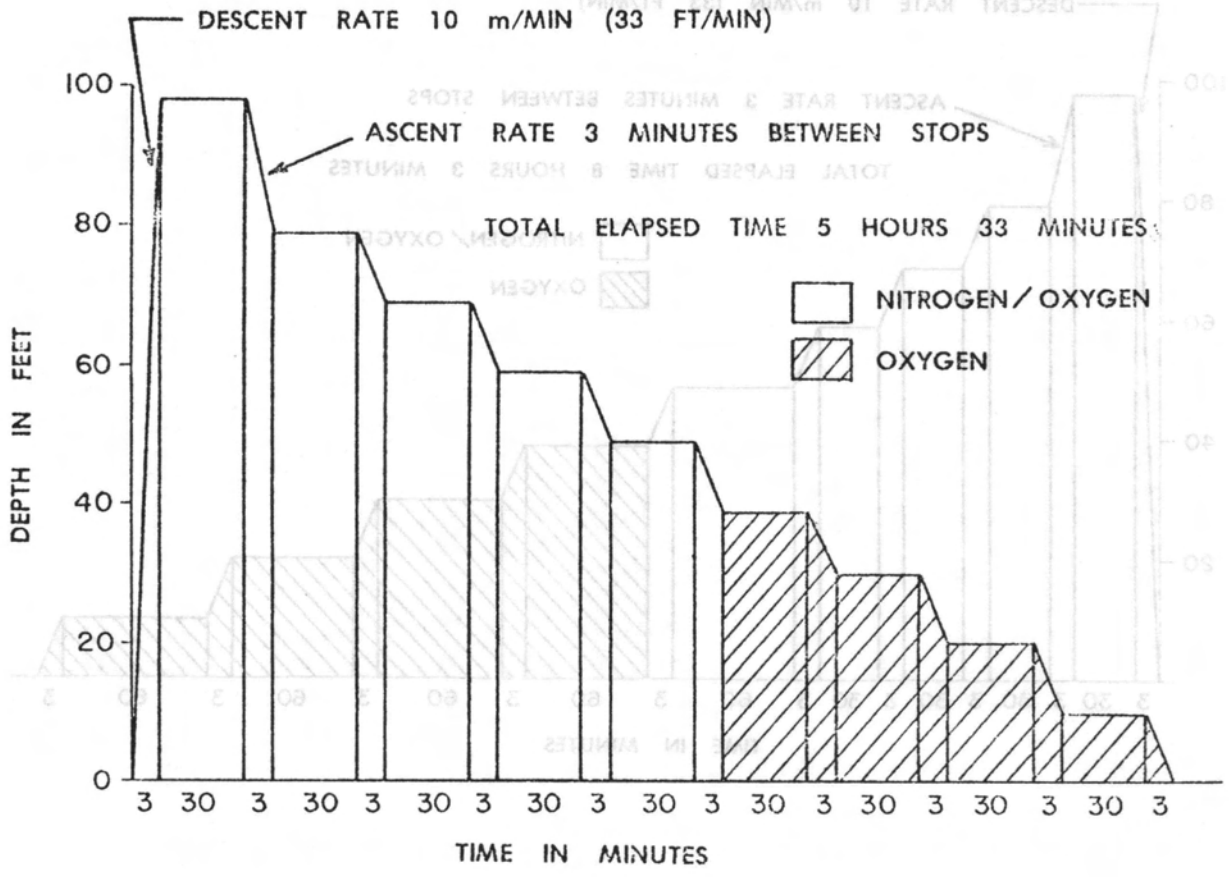
APPENDIX 4

FRENCH NAVY RECOMPRESSION TREATMENT TABLE A
(GERS 1968)*

1. Use--treatment of mild decompression sickness occurring during dives to less than 40 meters (131 ft).
2. Descent rate--10 m/min (33 ft/min).
3. Ascent rate--2 min/m (1.64 ft/min) between stops.
4. Time at 30 meters (98 ft) does not include the compression time.

*GERS (1968).

Depth (ft)	Depth (meters)	Time (min)	Breathing media	Total elapsed time (hours)	Total elapsed time (min)
98	30	30	40% O_2		33
79	24	30	40-60% O_2	1	15
69	21	30	60% O_2	1	51
59	18	30	60% O_2	2	27
49	15	30	60% O_2	3	3
39	12	30	100% O_2	3	39
30	9	30	100% O_2	4	15
20	6	30	100% O_2	4	51
10	3	30	100% O_2	5	27
10-0	3-0	6	100% O_2	5	33



APPENDIX 5

FRENCH NAVY RECOMPRESSION TREATMENT TABLE B
(GERS 1968)*

1. Use--treatment of mild decompression sickness occurring during dives to greater than 40 meters (131 ft) or for moderately severe decompression sickness occurring on dives to less than 40 meters.
2. Descent rate--10 m/min (33 ft/min).
3. Ascent rate--2 min/m (1.64 ft/min) between stops.
4. Time at 30 meters (98 ft) does not include the compression time.

*GERS (1968).

Depth (ft)	Depth (meters)	Time (min)	Breathing media	Total elapsed time (hours)	(min)
98	30	30	40% O_2		33
79	24	30	40-60% O_2	1	15
69	21	30	60% O_2	1	51
59	18	30	60% O_2	2	27
49	15	60	60% O_2	3	33
39	12	60	100% O_2	4	39
30	9	60	100% O_2	5	45
20	6	60	100% O_2	6	51
10	3	60	100% O_2	7	57
10-0	3-0	6	100% O_2	8	3