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RECOMPRESSION THERAPY FOR DECOMPRESSION ILLNESS

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Key Words

Decompression illness, oxygen, treatment.

Abstract

Recompression therapy for decompression illness was developed empirically based on observations by compressed air workers. The rationale that was developed fit the evidence that the disease was caused by bubbles, and it has been presumed that the major mechanism of action is related to physical reduction of bubble size. Oxygen was later added to increase the gradient for diffusion of nitrogen from bubbles, and to relieve tissue hypoxia. Definitive treatment of decompression illness (DCI) includes the administration of oxygen under pressure. Current recommendations include initial recompression to 2.8 bar, using USN, RN or closely related commercial procedures. A review of experimental data and experience with recompression tables is discussed. Expedient application of recompression using oxygen along with standard resuscitative measures is usually successful in treating decompression illness. Recent evidence suggests that pharmacological effects of hyperbaric oxygen, in addition to the physical effects on bubble size, gas diffusion and oxygenation, may be important in resolving the disease.

Introduction

Recompression therapy dates back to the 19th century. The bridge across the Mississippi River at St. Louis, completed in 1874, at was an engineering milestone in the United States, because the bottom of the Mississippi is covered in mud and it was impossible until that time to construct piers using traditional bridge building techniques.

In order to excavate down to bedrock the engineers used what resembles an upside-down cup (caisson), into which was pumped compressed air to maintain the internal pressure equal to that of the hydrostatic pressure outside. As the caisson rested on the bottom, the air pressure prevented the ingress of water and mud, allowing workers inside to facilitate pumping of the mud to the surface. The caisson gradually sank by its own weight, aided by the mass of the bridge pier being constructed atop the caisson. Once on bedrock, the caisson was filled with concrete, locking the bridge pier permanently into place. This was the first major use of caisson construction work in the United States.

At the end of a shift the men decompressed in an independently pressurised lock. As the depth (and hence the ambient pressure in the caisson) increased, the men were subjected to progressively increasing decompression stress. Many of the men developed neurological decompression sickness (DCS) and 14 of them died. It is perhaps of note that as a result the engineer, James Eads, hired a local doctor, Dr Alphonse Jaminet, who then became the first occupational physician in the United States concerned with the welfare of men working under pressure, to take care of the men. This man, although not knowing the pathophysiology of decompression sickness, elucidated several procedures and principles for the prevention of this illness that are still believed correct to this day.¹

One of Dr Jaminet's contributions is an account of an episode of spinal cord bends that he experienced after leaving the caisson following a visit to the work site. With no definitive treatment available, other than tincture of time, he went home, drank some wine and gradually got better. Unfortunately this was not the fate of Washington Roebling, the engineer of the Brooklyn Bridge, built a few years afterward using the same technology, who became permanently disabled by spinal cord DCS after helping to fight a fire in the caisson.

It was noted that when men with DCS re-entered the caisson on their next shift they felt better and their symptoms often resolved. This was the beginning of recompression treatment, which has continued to be developed to this day. The use of recompression therapy was not routine, however, until the East River tunnels in New York City were constructed between 1906 and 1909, and described by Dr. Frederick Keays.² Keays observed that 89% of bends cases treated with one or two recompressions experienced complete relief, compared with only 75% of cases treated “by medical means”. He also observed the relationship between delay to treatment and residual symptoms. Eight percent of cases treated within less than two hours were left with residual symptoms, vs. 25% of cases treated 12 or more hours after symptom onset. The history of the development of recompression tables has been lucidly described by Chris Acott.³

Recompression treatment

It is now understood that bubbles are the initiating cause of decompression illness, and therefore it is logical to believe that increasing the ambient pressure, and so reducing the bubble size, will be therapeutic. Boyle’s Law predicts that bubble volume is inversely proportional to the ambient pressure. If the bubble is spherical, the reduction in volume will be accompanied by a less impressive reduction in diameter. At 2.8 bar (18 m), a commonly used pressure for the treatment of decompression illness, the immediate reduction in bubble diameter is only about 30%.

Another effect of recompression with oxygen is the relief of tissue hypoxia and oedema. Recently in animal models of arterial gas embolism,⁴ ischaemic myocutaneous flap⁵ and carbon monoxide poisoning⁶⁻⁸ there has been evidence that leucocyte adherence to damaged endothelium plays a pathophysiological role. Furthermore, in humans hyperbaric oxygen administration reduces leucocyte adherence by inhibiting beta-2 integrin function.⁹ This is likely to be another beneficial effect of recompression treatment for DCI.

Over the years there has been considerable discussion about the optimum depth and PO₂. Recompression chamber operations are usually limited to 50 m (6 bar); inspired PO₂ is usually within the range 2.5-3 bar, above which there is an unacceptable risk of toxicity. A wide range of treatment depths and inspired PO₂ values has been published (Table 1).

One of the first studies to address the issue of treatment depth was performed by Waite, who injected air into the carotid arteries of dogs and, using a cranial window to observe bubbles directly, studied the resolution of these bubbles as a function of ambient pressure.¹⁰ He found that at a chamber pressure of 4 bar (30 m) all of the bubbles originally visible in the cranial window had disappeared.

TABLE 1
AMBIENT PRESSURE AND PO₂ OF RECOMPRESSION TABLES

Table	Maximum pressure (bar)	PO ₂ at maximum pressure (bar)
Comex Table 12 ^{27,45}	2.2	2.2
USN Tables 5, 6 ¹⁹	2.8	2.8
Comex Table 30 ²⁷	4	2
USN Table 6A ¹⁹	6	1.2
Modified USN Table 6A ¹⁷	6	3
USN Table 8 ¹⁹	7.8	1.4

The logical suggestion that the maximum pressure needed to treat arterial gas embolism should be 4 bar was not implemented by the US Navy because it was felt that fleet diving medical officers would not accept any table with a maximum depth less than 6 bar. Since then, Des Gorman and colleagues, using a rabbit model, have examined a range of pressures and made two interesting observations. First, bubbles often passed directly through the vascular network without becoming trapped, even without recompression. Second, bubbles that ended up in communicating vessels, through which there was very little blood flow, did not resolve, even after recompression to 6-10 bar.¹¹

Other studies have examined the effect of recompression on brain electrical function. In one series of experiments, Leitch and colleagues examined the effect of various ambient pressures on in dogs with gas embolism. Somatosensory evoked response amplitude was used as an end point, and no differences in efficacy among ambient pressures between 2.8 to 10 bar were observed.¹² McDermott, in two feline studies published in 1992, examined recompression to 6 bar on air followed by 2.8 bar on oxygen versus 2.8 bar on oxygen without deeper recompression, and found no differences,¹³ nor were there were differences when an enriched O₂ mixture was administered at 6 bar.¹³

Similar lack of effect of pressures greater than 3 bar has been observed in a canine model of spinal cord decompression sickness.¹⁴ No advantage to recompressing to 7 bar breathing air was observed.

Table 2 illustrates the partial pressures of the different component gases in inspired and alveolar gas, arterial blood, tissue, and bubble. At 1 bar breathing air here is a slight difference in partial pressure of nitrogen from the bubble to the tissue of about 150 mm Hg. For this reason a tissue bubble eventually resorbs because the inherent diffusion gradient facilitates gas diffusion from the

TABLE 2

PARTIAL PRESSURES OF NITROGEN IN ALVEOLAR GAS AND BUBBLE.

The calculated partial pressure gradient from bubble into surrounding tissue, assuming tissue PN_2 = alveolar PN_2 , is shown.

Pressure (bar)	FiO_2	Alveolar PN_2 (mmHg)	Bubble PN_2 (mmHg)	Bubble PN_2 -Tissue PN_2 * (mmHg)
1	0.21	571	713	142
1	1.00	0	713	713
2.8	0.21	1664	2096	432
2.8	1.00	0	2096	2096

* For this calculation it is assumed that tissue PN_2 = alveolar PN_2 .

The inherent difference between bubble and tissue PN_2 , which increases during O_2 breathing, is known as the "oxygen window".

bubble to surrounding tissue, from where it is transported away by the blood.

After recompression to 18 m (2.8 bar), in addition to reduction in bubble volume, the partial pressures of oxygen and nitrogen are raised, and the N_2 diffusion gradient increases from 150 to about 440 mm Hg. However a disadvantage is that the increased partial pressure of nitrogen loads up other tissues with inert gas, which subsequently has to be washed out.

Breathing oxygen at the surface (1 bar) also increases the nitrogen diffusion gradient, from about to about 700 mm Hg, illustrating one advantage of breathing oxygen at the surface compared with recompression breathing air. Recompression to 18 m (2.8 bar) breathing 100% oxygen raises the gradient for diffusion to over 2,000 mm Hg. This is the basis for modern treatment of decompression illness: recompression while breathing supplemental O_2 .

The question of the optimal PO_2 has been examined in several animal studies. Leitch and colleagues, using a canine model of spinal cord DCS in which the animals were recompressed to 5 bar, observed that the optimal PO_2 was between 2 and 2.5 bar.¹⁵ However the investigators used somatosensory evoked responses to monitor recovery, which in our experience in human DCI, do not correlate well with clinical response. The optimum PO_2 for humans has not been established, but clinical experience suggests that it is in the range 2.5-3 bar.

The conclusion of these experiments on the treatment of DCI is that if pressure and increased partial pressure of oxygen are administered, the differences among different pressures and PO_2 values are fairly small, at least in animal studies using direct observation of bubbles or electrophysiological function as measures of outcome.

Which table to use

Although a wide range of pressures and PO_2 values are used with current treatment tables (Table 1, page 225), the most commonly used recompression table is US Navy Table 6 (Figure 1). It consists of administration of oxygen and air cycles at 18 m (2.8 bar, 60 ft), then decompression, breathing oxygen, to 9 m (30 ft) over 30 minutes, then a number of cycles of oxygen and air, followed by a 30 minute decompression, breathing oxygen, to the surface.

It is very flexible, as the number of cycles can be extended at both 18 and 9 m. US Navy Table 5 (Figure 2, page 225), designed for pain only bends, is a shortened version, which consists of only two cycles of oxygen at 18 m 60 feet and a reduction in the time at 9 m. US Navy Table 6A is a Table 6 preceded by 30 minutes at 50 m (6 bar, 165 ft) breathing air (Figure 3, page 226). It was initially recommended as initial treatment for arterial gas embolism. A subsequent modification of it by civilian practitioners substitutes 40-50% O_2 for air at 50 m,^{16,17} but today it is rarely used.

The Table 6 two-step "paradigm" has been taken perhaps to its ultimate limit in the Catalina Marine Science Center Treatment Table, which appeared in the SPUMS Journal 12 years ago.¹⁸ Divers taken to the chamber on Catalina Island had to be shuttled between the hospital on the mainland in Los Angeles and the chamber on the Island. This table was therefore designed to administer as much oxygen to the diver during the initial treatment as practically feasible. The Catalina Treatment Table is shown in Figure 4 (page 227).

Current US Navy¹⁹ and recreational²⁰ guidelines recommend (for treatments initiated from the surface) an initial treatment depth of 18 m, with deeper recompression

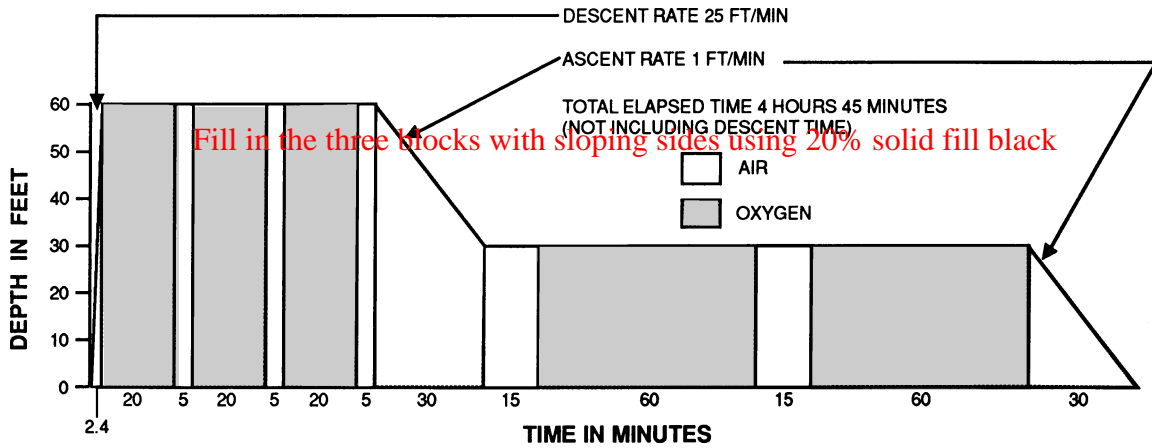


Figure 1. USN Treatment Table 6. Table 6 is used for treatment of neurological decompression illness and patients with pain-only or mild cutaneous symptoms that are not relieved within 10 minutes of reaching 60 ft breathing oxygen. Table 6 can be extended at 60 ft and at 30 ft (9 m, 1.91 bar) if symptoms have not been relieved within the first three oxygen cycles. (Drawing reproduced from Moon⁵⁵).

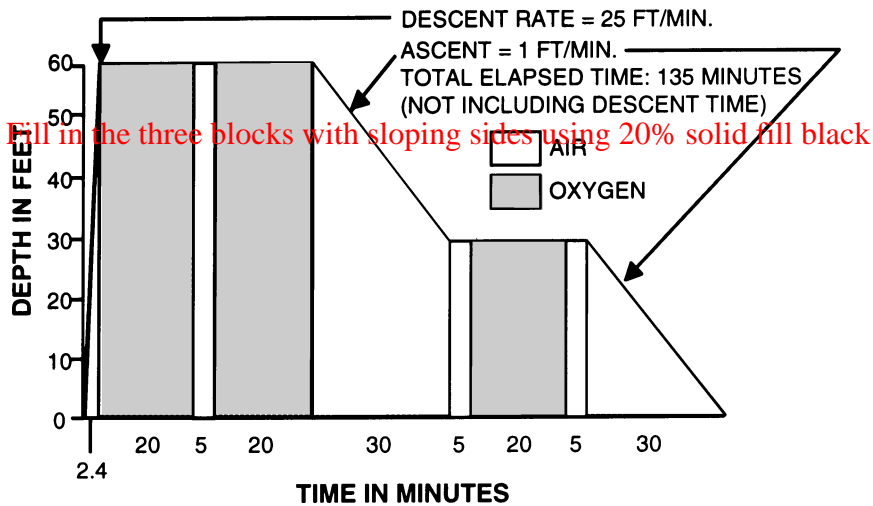


Figure 2. USN Treatment Table 5. This treatment table is recommended for pain-only or mild cutaneous symptoms with no neurological symptoms or signs. If complete relief of symptoms has not occurred within 10 minutes of compressing the patient to 60 ft (18 m, 2.8 bar), then Table 6 is recommended. (Drawing reproduced from Moon⁵⁵).

as an option available for instances of inadequate clinical response. Indeed, the vast majority of cases of decompression illness will respond satisfactorily to one or more applications of an 18 m treatment table.

Table 3 (page 228) shows a number of published series, largely from military experience, of divers with DCI including a total of 1,763 patients. After the first treatment around 80% had complete relief. Most ultimately attained asymptomatic status after one or more extra treatments. Objective evidence indicates the high degree of success of these treatment tables in military practice, and supports their wide acceptance. However, in recreational diving, where it is rare to have immediate access to a chamber, the success rate is less than optimal. In Ball's series²¹ (Table 3) the

success rate in civilian divers was poor, seemingly attributable to treatment delay. In recreational divers in the Divers Alert Network (DAN) database delay to treatment is common. Of 483 cases of DCI reported in the 1998 DAN Report only 17% received recompression therapy within 4 hours of symptom onset, and 43% within 12 hours.²² Delay to treatment is a factor that is associated with poor outcome.²³

Shallow or short recompression

Short tables, such as those designed for use in monoplace chambers, also appear to be effective.^{24,25} The monoplace table designed by Hart specifies 100% oxygen

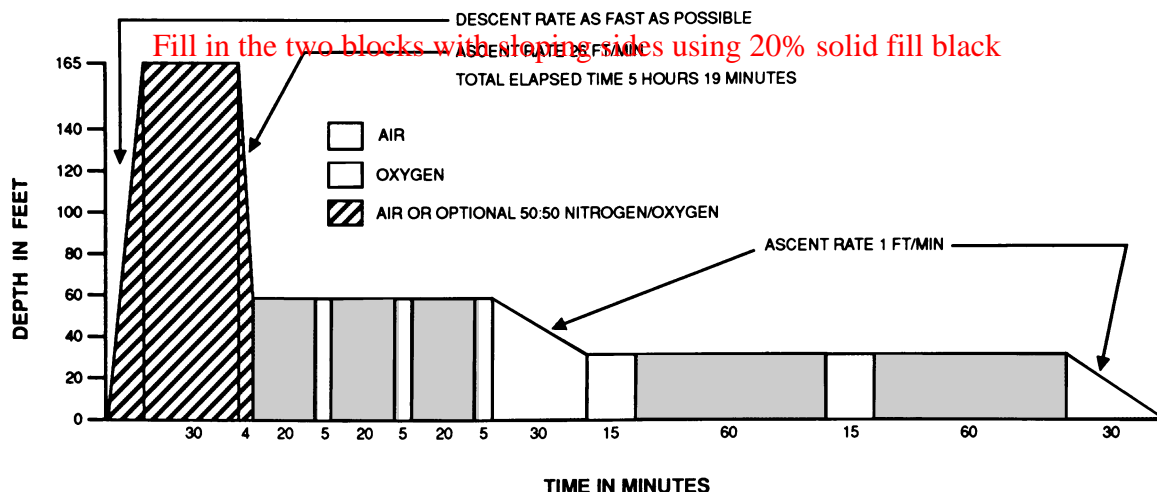


Figure 3. USN Treatment Table 6A. This table consists of a 30 minute excursion to 6 bar (50 m, 165 ft) while the patient breathes air, followed by an oxygen breathing portion identical to USN Table 6, and was initially recommended for treatment of arterial gas embolism. While the U.S. Navy still recommends air as the appropriate breathing gas at 6 bar, others have used nitrogen-oxygen mixtures (usually 60:40 or 50:50). Some animal studies have indicated that compression beyond 2.8 bar provides no additional benefit. Nevertheless, in clinical experience a small percentage of patients may respond to treatment at 6 bar, but fail to do so at 2.8 bar. (Drawing reproduced from Moon⁵⁵).

administration at 3 bar for 30 minutes followed by 2.5 bar for 60 minutes.^{24,26} These shorter tables appear to be effective in most cases, although they have not been prospectively compared with the more commonly used schedules such as USN Table 6, and their equivalence to the longer oxygen tables in severe decompression illness is questionable.²⁵

Data presented by Imbert²⁷ suggest that for pain only bends a 12 m (40 ft; 2.2 bar) recompression for two hours is adequate. In his retrospective review of DCI treatments in commercial divers he reported 91% success in 407 cases. Few details are provided, however, and whether this treatment modality can be applied to recreational divers, in whom long delays to treatment are common, is uncertain.

Deeper recompression

There are several published tables incorporating initial recompression to depths exceeding 18 m. Comex Treatment Table 30,²⁷ for example, incorporates an initial recompression to 30 m for 60 minutes, breathing either 50-50 N₂-O₂ or He-O₂ (see Figure 5, page 228); USN Treatment Table 6A was described earlier. A modification of Table 6A has been described by Lee and colleagues,¹⁷ who reported a series of divers who had very severe disease, with long delays to treatment. In their modified treatment tables, the divers spent 60 minutes at 50 m (6 bar, 165 ft) breathing 40% oxygen and were then decompressed according to Table 6A. The published results were remarkable: 70% of 99 divers were cured and 29% were improved. It was not a randomised trial, however, and it is

possible that improvements attributed to the modified table were due to general supportive procedures such as fluid resuscitation.

To examine the issue of deeper recompression after an initial period at 18 m (2.8 bar), in 1985 Leitch and Green retrospectively reviewed a number of cases of DCI in naval divers. Fourteen of their cases were recompressed to 50 m (6 bar) breathing air. Six cases were cured, however two were already improving at 2.8 bar and only one had motor abnormalities. Five cases had marginal improvement, and in three cases there was no effect. Compression beyond 6 bar was implemented in 10 cases, in whom there were two cures in divers with sensory problems only, and 8 instances of incomplete or no improvement. Four of these 8 relapsed during decompression. Their retrospective data review did not suggest that this was hugely successful. Recompression to greater than 2.8 bar or greater than 18 m should be an available option, the information that is in the literature suggests that it is rarely useful.

Figure 6 (page 229) shows data from 3,899 cases of decompression illness reported to DAN, illustrating the relationship between probability of complete relief and delay to recompression. The probability of complete relief is greater if recompression is administered early rather than late. However, even divers treated after 12 hours or more delay have some relief of symptoms. Response to recompression treatment of DCI, even after several days' delay, has been reported.²⁸⁻³¹

Recently there has been some interest in using helium as a component of a treatment gas for divers with

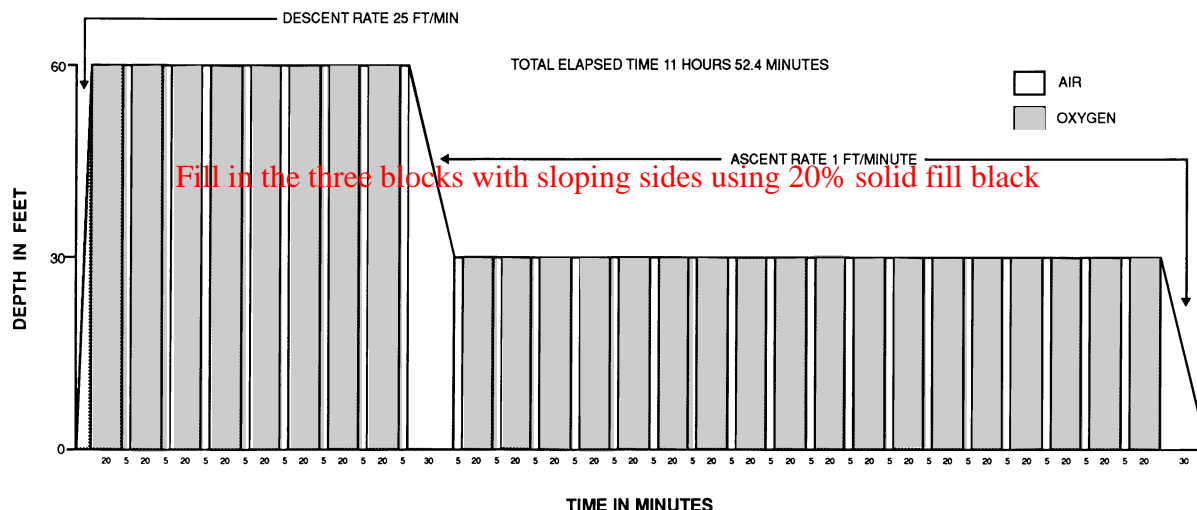


Figure 4. Catalina Treatment Table. This is a modified version of USN Treatment Table 6. All oxygen breathing cycles are of 20 minutes duration, followed by 5 minutes breathing air. In its current implementation, shorter versions of this table may be used as follows. After 3 oxygen cycles at 60 ft, a minimum of 6 cycles are required at 30 ft (equivalent to USN Table 6); after 4 cycles at 60 ft, 9 cycles are required at 30 ft; after 5 to 8 cycles at 60 ft, a minimum of 12 cycles are required at 30 ft. Up to 18 cycles at 30 ft (as shown above) can be used. Tenders must breathe oxygen for 60 minutes at 30 ft and during the decompression to the surface (total 90 minutes). If there have been fewer than 4 oxygen cycles at 60 ft and fewer than 9 cycles at 30 ft, then only 30 minutes of oxygen breathing is required for the tender at 30 ft in addition to the decompression time (total 60 minutes). Further treatments can only be started after 12 hours of air breathing at the surface. Further details of this treatment table have been published by Pilmanis. (Drawing reproduced from Moon⁵⁵).

DCI after air or nitrox dives. There is some reason to believe that recompression with helium might offer some advantage. The movement of a gas through a liquid depends upon its solubility in the liquid and its diffusivity, a function of molecular weight. The rate of flux of gas along a partial pressure gradient is related to the partial pressure difference and the gas permeability: the product of solubility and diffusivity. The permeability of helium in oil is less than that of nitrogen. Therefore, in fatty tissues it might be possible that breathing helium-O₂ could allow nitrogen to diffuse out of a bubble faster than helium diffuses in. Indeed, in a study by Hildegaard and colleagues, when bubble size in rat spinal cords at 1 bar was measured as a function of time, the rate of bubble diameter shrinkage was faster when the animals were administered He-O₂ than when they received 100% O₂.³² In another rat experiment, breathing He-O₂ prevented the development of spinal cord DCS after a chamber air dive to 3.8 bar for one hour, and appeared to be superior to 100% O₂.³³ It is also conceivable that there are pharmacological effects of helium unrelated to gas diffusion. In an animal study, using tissue oxygen electrodes in the cerebral cortex, cortical PO₂ was highest when the animals breathed He-O₂ compared with air or 100% O₂ at the same inspired PO₂.³⁴

Two small uncontrolled series of human DCI suggested that He-O₂ recompression is effective.^{35,36} Imbert reported the results of Comex Table 30 (Figure 4). He found when using nitrox during the 60 minute period at 30 m, four out of 25 divers required an additional

treatment, whereas when heliox was used none of 11 required further treatment.²⁷ These observations are consistent with an advantage of heliox, although the difference between the two treatments is not statistically significant.

However, in a guinea pig model of severe DCI Lillo and colleagues³⁷ observed that recompression with He-O₂ resulted in a slower recovery from tachypnoea than when air was used as the treatment gas. A retrospective review of cases treated in the US Navy indicated no advantage of tables using He-O₂ vs. those using N₂-O₂/O₂.³⁸

A randomised trial³⁹ currently (1997) underway in Auckland may decide the issue. In the meantime there is no compelling reason to switch from using O₂ treatment tables to those incorporating He-O₂.

How many treatments ?

Most diving physicians recommend repetitive treatment with hyperbaric oxygen until the patient's symptoms have resolved, or until there is no further improvement after a treatment (clinical plateau). The vast majority of cases of DCI will respond to a single recompression treatment. Although a small minority of divers with severe neurological injury may not reach a clinical plateau until 15-20 repetitive treatments have been administered, formal statistical analysis of approximately 3,000 DCI cases in the DAN database supports the efficacy of no more than 5-10 repetitive treatments for most injured

TABLE 3
SINGLE RECOMPRESSION SUCCESS RATE OF USN OXYGEN TREATMENT TABLES
 (from Thalmann³⁸)

Source	Number of cases	Complete relief (%)	Substantial relief (%)	Comments
Workman ⁴⁶	150	85	95.3 (after 2nd treatment)	
Erde & Edmonds ⁴⁷	106	81		
Davis ⁴⁸	145	98		Altitude DCS
Bayne ⁴⁹	50	98		
Pearson & Leitch ⁵⁰	28	67	83	
Kizer ⁵¹	157	58	83	Long delays
Yap ⁵²	58	50	84	Mean delay 48h
Gray ⁵³	812	81	94	
Green ⁵⁴	208	96		All pain only, USN Table 5
Ball ²¹	14	93 (mild cases)		{
	11	36 (moderate cases)		{ Many cases with long delays
	24	8 (severe cases)		{
TOTAL	1763	81		

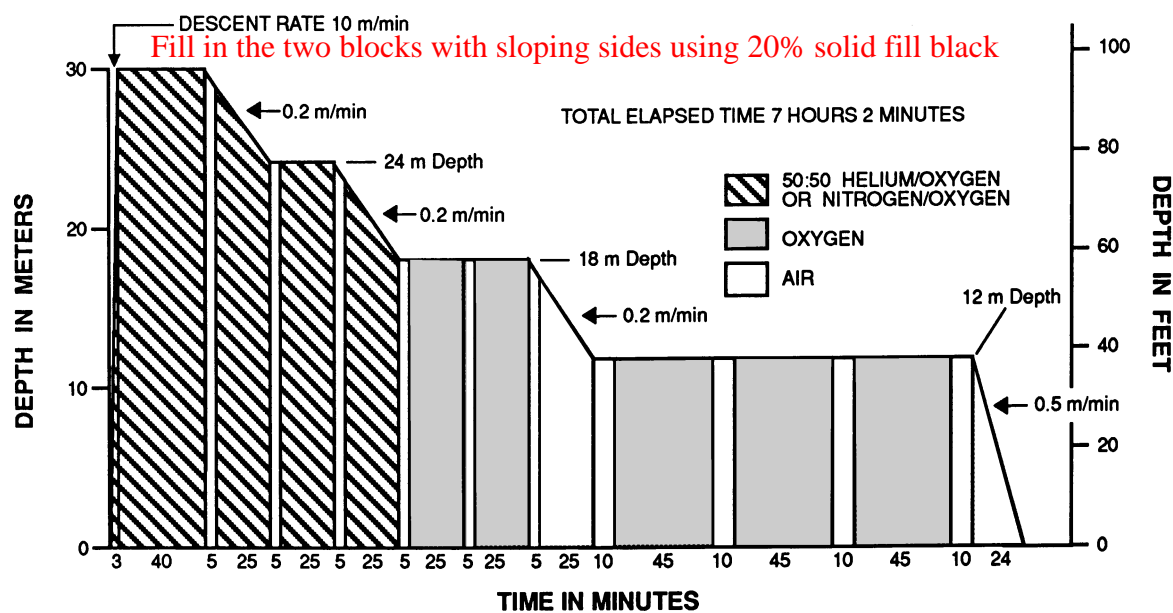


Figure 5. Comex Treatment Table 30. This table is an option for the treatment of DCI, and can be implemented using either N₂-O₂ or He-O₂ for the period at 30 msw (approximately 100 ft). In European practice, this table is frequently implemented as the initial treatment of decompression illness. (Drawing reproduced from Moon⁵⁵).

divers.⁴⁰⁻⁴² Symptoms and signs that are unresolved at the end of hyperbaric treatment usually continue to resolve spontaneously for months or even years.

availability of more definitive data, there is no incontrovertible basis upon which to recommend any particular follow-up table.

There are few data that address the issue of which treatment table to use for follow-up treatment. A retrospective review by Wilson and colleagues⁴³ suggested that follow-up tables at 18 msw (2.8 bar, 60 ft) were associated with a lower relapse rate than those at 14 msw (2.4 bar, 46 ft). The analysis was subject to the potential pitfalls of a retrospective review, and pending the

Saturation treatment

Saturation recompression treatment is a technique in which the chamber remains for a prolonged period at treatment pressure until the patient's symptoms are resolved or maximally improved. Saturation treatment can be

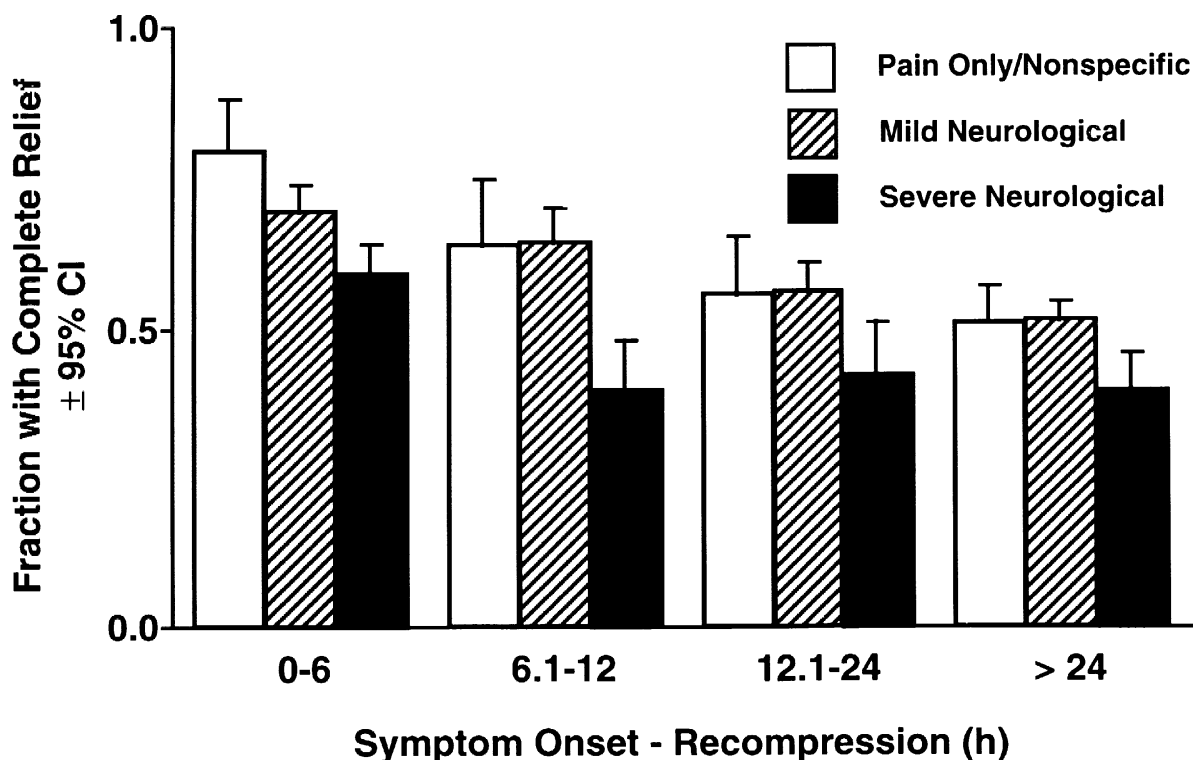


Figure 6. Residual Symptoms after Recompression Treatment. Data represent 3,899 cases of DCI in recreational divers reported to DAN, and are stratified by severity: Severe neurological symptoms include convulsions or abnormalities of vision, gait, urinary/anal sphincter function, motor strength or consciousness.

routinely implemented in commercial diving practice, and has been used occasionally in the treatment of recreational diving casualties.¹⁶ It requires a chamber in which the atmospheric carbon dioxide and oxygen can be accurately monitored and controlled. Saturation treatment optimally requires at least two tenders and a chamber large enough to care for a critically ill person comfortably. Treatment depth is typically 18-30 m (2.8-4 bar); depths exceeding 18 m require reduction of ambient PO₂ to maintain a mixture that is not toxic (PO₂ typically 0.4-0.6 bar). USN Treatment Table 7 is one of the easiest saturation treatment tables¹⁹ The saturation depth for this table is 2.8 bar (18 m, 60 ft), and hence air can safely be used for the chamber atmosphere.

The Duke University experience with saturation treatment in 16 divers since 1977 has been reported previously.⁴⁴ The 15 divers who failed an initial USN Table 6 treatment were compared with a similar group of 10 who subsequently received multiple short O₂ treatments (USN treatment tables followed by twice daily treatments at 2 bar for 120 minutes). Following treatment, gait tended to be better in the divers treated with saturation tables. One week post accident 5 of 15 divers who received saturation treatment could walk with or without assistance, vs. none of 10 in the short O₂ table group. At hospital discharge one third of the divers treated with saturation tables could walk

independently, vs. only one in the short O₂ table treatment group.

Indications for considering saturation treatment are severe neurological DCI, and either continued improvement at 18 msw even after a maximum number of oxygen cycles has been administered, or significant deterioration during decompression.

Summary

Definitive treatment for DCI is recompression using an oxygen enriched breathing mixture. Treatment protocols (“tables”) have been empirically designed, and using an initial treatment pressure and PO₂ of 2.5-3 bar have a high degree of success. Use of a standard treatment table is recommended, with follow-up treatments administered until resolution or clinical plateau. Treatment pressures in excess of 3 bar are rarely required. Advantages of administration of a breathing gas other than O₂ or N₂-O₂ (e.g. He-O₂) have not yet been substantiated.

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