Helium and Oxygen Treatment of Severe Air-Diving–Induced Neurologic Decompression Sickness

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Background: The use of helium and oxygen recompression treatment of neurologic decompression sickness (DCS) has several theoretical advantages over the traditionally used air and oxygen recompression tables that have been confirmed by findings from recent animal experiments.

Objectives: To evaluate the outcome of patients with neurologic DCS who had been treated with a helium-oxygen protocol and to compare it with that of a retrospective control group that was treated with air-oxygen tables.

Design: The study and control groups included 16 and 17 diving casualties, respectively. The severity of neurologic DCS was estimated according to a 9-point scale weighting motor, sensory, and sphincter control functions. The study group was treated with a helium-oxygen decompression protocol, and the control group was treated with the US Navy air-oxygen Table 6 or 6A. Persistent residual dysfunction was treated in both groups with daily hyperbaric oxygen sessions, at 2.5 absolute atmospheres for 90 minutes, until no further clinical improvement was noted.

Setting: The Israel Naval Medical Institute (Israel's national hyperbaric referral center), Haifa.

Results: Significant clinical score increments were found for both the helium-oxygen– and air-oxygen–treated groups: 2.8±2.4 (mean±SD) and 7.4±1.1 at presentation vs 7.6±2.1 and 8.1±1.5 at discharge, respectively ($P<.001$ and $P=.005$, respectively). Although the score at presentation was significantly lower for the helium-oxygen–treated group ($P<.001$), no difference was found between the groups' average outcome scores. While most of the improvement in the patients in the study group could be attributed to the helium-oxygen treatment and not to the supplemental hyperbaric oxygen, in the control group, no significant difference could be demonstrated between the scores at presentation and at completion of the air-oxygen recompression table. In 5 patients who were treated with the use of the air-oxygen tables, deterioration was observed after recompression. No deterioration or neurologic DCS relapse occurred in the helium-oxygen–treated group.

Conclusion: The results suggest an advantage of helium-oxygen recompression therapy over air-oxygen tables in the treatment of neurologic DCS.

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RECREATIONAL DIVING enjoys growing popularity throughout the world. In the United States alone, there are more than 5 million people certified as SCUBA (self-contained underwater breathing apparatus) divers. The underwater environment, with its rapidly changing ambient pressures, presents a pathophysiological challenge that may lead to neurologic insults, which necessitate specific treatment measures. When a diver breathes air under increased pressure, his or her tissues are loaded with increased quantities of nitrogen proportional to both the ambient pressure and the solubility of the gas in the specific tissue. When the ambient pressure decreases as the diver returns to the surface, the sum of the gas tensions in the tissue may exceed the absolute ambient pressure. At this point, a state of supersaturation is created, which may lead to the liberation of free gas from the tissues and to the onset of decompression sickness (DCS).[1] Inert gas bubbles first appear in tissues and in the venous circulation.[2] These bubbles may reduce the integrity of the capillary endothelium by physically disrupting the basement membrane[3-5] and by promoting the release of kinins, leading to increased vascular permeability.[6] Thus, a generalized capillary leak develops, and this leak will cause extravasation of plasma and hypovolemia, with increased blood viscosity and reduced tissue perfusion; in severe cases of DCS, this

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PATIENTS AND METHODS

Helium-oxygen recompression treatment is used at the Israel Naval Medical Institute (INMI), Haifa, for every compressed air-diving accident casualty who presents with severe neurologic DCS. The severity of each case is assessed by the diving history (risk factors, potential gas burden, violation of the recommended decompression tables) and by the clinical presentation (time from surfacing to symptoms and objective clinical signs of neurologic involvement). Before recompression treatment and after providing a general and diving history, each patient undergoes a complete physical examination with an emphasis on neurologic and otologic aspects, chest x-ray film, complete blood cell count, blood chemistry and gas studies, and electrocardiogram. After the first recompression treatment, further laboratory evaluation is carried out. This includes urodynamic studies, somatosensory evoked potential (SEP) recordings, single-photon emission computed tomography, cerebral and spinal magnetic resonance imaging, and a contrast-enhanced transesophageal echocardiogram according to the accident presentation and the patient's clinical status.

The treatment protocol begins with recompression according to a treatment table (Comex helium-oxygen treatment table CX-30[41]) that may be extended if no relief is observed (**Figure 1**). Whenever persistence of neurologic dysfunction or worsening of symptoms while under pressure is diagnosed, a helium-oxygen saturation protocol is used at the depth of relief. While the patient is under saturation conditions, his or her tissues are loaded with helium to the point of equilibrium with the ambient chamber atmosphere. Additional daily hyperbaric oxygen sessions are given for residual dysfunction as indicated: 100% oxygen at 2.5 absolute atmospheres (ATAs) for 90 minutes, until no further significant clinical improvement is noted. The helium-oxygen treatment protocol used at the INMI is shown in **Figure 2**.

The degree of neurologic insult at presentation and throughout the course of the treatment is estimated by a 9-point scale weighting the patient's neurologic status (**Table 1**). The study group included 16 divers who had received helium-oxygen recompression treatment of compressed air-diving–induced neurologic DCS at the INMI during the past 7 years. All patients had signs of a neurologic deficit at presentation. In the majority of patients, positive findings in the evaluation of SEPs and results of urodynamic and neuroimaging studies supported the diagnosis.

Until 1988, the US Navy air-oxygen tables constituted the exclusive recompression treatment modality that was practiced at the INMI for the treatment of neurologic DCS. Additional hyperbaric oxygen sessions were provided, as required, and this remains our policy. Of the 65 patients who were treated for neurologic DCS, 17 had less than 9 points according to our scale criteria for severe disease, and they were used as a retrospective control group. The 2 groups were matched for their demographic characteristics, the presence of risk factors for DCS, and the time from the appearance of symptoms to the commencement of recompression therapy.

Statistical analysis was carried out using commercially available software (SAS release 6.04, SAS Institute Inc, Cary, NC) on a personal computer (IBM-compatible). The Kruskal-Wallis test was used for comparisons between the groups, and the 2-tailed t test or Mann-Whitney rank sum statistic was used for a single comparison, as appropriate. Relations between variables were analyzed by calculating the Pearson product-moment correlation coefficients. A P value less than .05 was considered to indicate statistical significance. Unless otherwise indicated, data are expressed as mean±SD.

condition can lead to hypovolemic shock.[5,7-10] Most of the venous gas bubbles that reach the pulmonary arterial system will be trapped in the lung capillary network and will be resolved by diffusion into the alveoli.[11-13] However, where there is a considerable volume or rapid delivery of gas, the bubble-trapping capacity of the lung is impaired, and pulmonary hypertension will develop because of the growing obstruction of the pulmonary arterioles.[14] In fulminant DCS, the combination of venous back pressure caused by bubbles that are trapped in the lung and reduced blood flow caused by hypovolemia and blood hyperviscosity, which also facilitate the backward migration of bubbles from the vena cava, enable accumulation and growth of bubbles in the epidural vertebral venous plexus. Because this venous plexus is a valveless low-pressure system, the formation, accumulation, and growth of bubbles would be further potentiated under such circumstances. Blood-gas interaction brings about structural alterations in plasma proteins with activation of the coagulation, complement, and fibrinolytic cascades; thus, these factors further contribute to venous stasis and finally lead to infarction of the spinal cord.[15-17] Extravascular bubbles have also been demonstrated in the white matter of the spinal cord, causing axon disruption and pressure-induced ischemia.[18] Neurologic manifestations of DCS are also attributed to paradoxical gas embolization in which bubbles escaping pulmonary filtration enter the arterial circulation through a patent foramen ovale or pulmonary arteriovenous shunts.[19]

Symptoms and signs of neurologic DCS include parasthesias and numbness progressing to motor pathway involvement with paraparesis and paraplegia. Urinary bladder and anal sphincter dysfunction is common in severe cases, as well as referred abdominal and back pain.[1] Decompression sickness should be treated with the use of recompression in a hyperbaric chamber. The aim of the various hyperbaric protocols that are used is to eliminate the tissue gas phase by mechanical compression of the bubbles and inert gas washout while supplying the hypoxic tissue with plasma-dissolved oxygen.[20] The role of oxygen treatment tables for air-diving–induced DCS has been well documented, and this type of treatment has been accepted by the vast majority of physicians who treat divers. The use of helium and oxygen as an alternative to air for recompression treatment of DCS was suggested 6 years before the introduction of oxygen treatment tables.[21,22] High rates of incompletely resolved neurologic insults[23-25] and the unexpected deterioration of neurologic DCS during and after treatment using oxy-

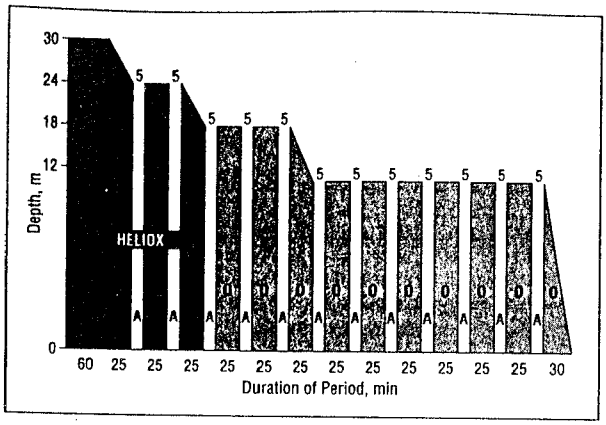

Figure 1. *Helium and oxygen recompression treatment table (Comex CX-30[41]). HELIOX indicates that the breathing mixture is 50% helium and 50% oxygen; A, 5-minute air breaks at the conclusion of helium-oxygen– or oxygen-breathing periods to avoid pulmonary oxygen toxic effects; and O, 25 minutes of oxygen breathing.*

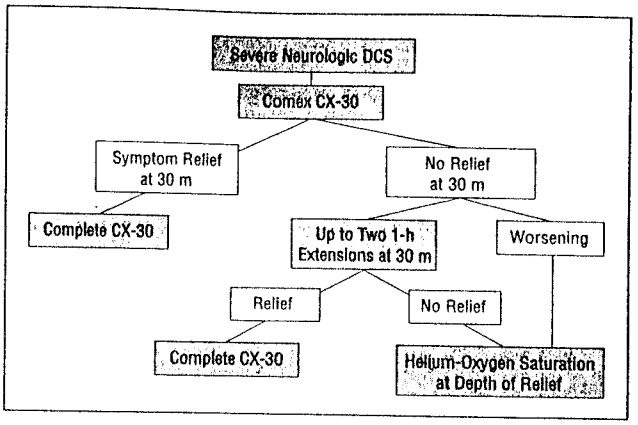

Figure 2. *The helium and oxygen treatment protocol used for severe neurologic decompression sickness (DCS) at the Israel Naval Medical Institute, Haifa, with the use of a helium and oxygen recompression treatment table (Comex CX-30[41]).*

gen tables[26] have revived the interest in helium-oxygen as an alternative treatment.[27]

The advantage of helium-oxygen over oxygen alone as the breathing gas has been attributed to the lower solubility of helium in both blood and fat and to the lower value obtained for the product of the diffusion and solubility coefficients in fatty tissue when using helium, allowing a greater outward flux of nitrogen.[28] Gross involvement of the myelin sheaths in axon bundles has been demonstrated previously in animal studies and in clinical cases of severe DCS.[29]

Myelin sheaths are composed mainly of sphingolipids with a lipid content of 35% to 45%; hence, the use of helium-oxygen instead of oxygen benefits patients who are suffering from neurologic DCS. A further advantage of helium-oxygen is that higher treatment pressures may be maintained because the mixture eliminates the complication of oxygen toxic effects that may arise when pure oxygen is used at similar pressures. Higher pressures will enhance the mechanical reduction of bubble size and increase the diffusion gradient for nitrogen without an additional gas burden.[30]

Despite the theoretical benefits of helium-oxygen, animal studies have presented conflicting results,[31-37] while there has been limited clinical experience with this treatment modality.[23,38-40] The purposes of the present study were to evaluate our clinical experience with the use of helium-oxygen for the treatment of severe air-diving–induced neurologic DCS and to compare the treatment results with those of a retrospective control group that was treated with the use of air and oxygen tables.

RESULTS

The mean depth and bottom time of the accident dives were 33.4±14.8 m and 58±40 minutes for the study group and 28.2±13.4 m and 27±16 minutes for the control group, respectively. The average bottom time was found to be significantly longer for the helium-oxygen–treated group ($P=.007$, 2-sample t test). The dive profile was evaluated according to standard US Navy air decompression tables.[42] For the study group, 12 incidents (75%) of decompression schedule violation were reported by the injured diver or his or her companions, while only 7 (41%) of the patients in the air-oxygen–treated group had failed to conduct the required decompression procedures. The known risk factors contributing to DCS[29] were identified in 8 (50%) and 11 (65%) of the patients in the study and control groups, respectively.

The average time delay from surfacing to the appearance of neurologic DCS symptoms was significantly shorter in the helium-oxygen–treated group: 16±23 (range, 0-90) minutes vs 86±132 (range, 0-420) minutes in the air-oxygen–treated group ($P=.05$, 2-sample t test).

Five of the patients were given recompression therapy before being treated with helium-oxygen at the INMI. An extended US Navy treatment Table 6 was used in 4 patients; an extended Table 6A was used in 1 patient. This treatment was given at the hyperbaric facilities in Eilat on the Red Sea, or at Sharem-El-Sheich, located in the southern part of the Sinai Peninsula. The mean time interval from surfacing to the start of helium-oxygen treatment was 769±540 minutes (12.8±9 hours) (range, 120 to 1500 minutes [2-25 hours]). For the control group, the mean time to the first recompression treatment was 1028±1184 minutes (17.1±19.7 hours) (range, 60 to 4320 minutes [1-72 hours]).

The average clinical score at presentation for the helium-oxygen–treated patients was 2.81±2.45 (range, 0-7) compared with 7.41±1.17 (range, 4-8) in the control group ($P<.001$, Mann-Whitney rank sum test). The clinical score on the 9-point scale for each of the patients at presentation and at discharge, the main recompression treatment, and the number of adjuvant hyperbaric oxygen sessions provided are presented in **Table 2**.

The mean number of hyperbaric oxygen treatments per patient was 11±10.3 (range, 0-30) in the helium-oxygen–treated group and 5.1±7.7 (range, 0-26) in the air-oxygen–treated group.

In the study group, a significant correlation was found between a shorter delay to the appearance of symptoms and the clinical severity of DCS at presentation ($r=0.357$; $P=.04$). The same correlation reached only marginal significance for the control group ($r=0.21$; $P=.06$).

Table 1. Scoring Scale Used for the Weighting of Neurologic Decompression Sickness Severity

Sensory deficit
- 0, no deep or superficial sensation in the body area involved
- 1, no superficial sensation, deep sensation diminished or no deep sensation, hypoesthesia/hyperesthesia of superficial sensation
- 2, hypoesthesia/hyperesthesia of superficial sensation, and/or deep sensation diminished
- and
- 3, intact sensation

Motor deficit
- 0, complete plegia of limbs involved
- 1, voluntary muscle movements, but patient cannot raise the involved limbs against gravitation and/or sit down
- 2, paresis
- and
- 3, intact motor function

Sphincter control
- 0, no urinary bladder and/or anal sphincter function
- 1, urinary bladder and/or anal sphincter dysfunction: urodynamic studies show some sensation and voluntary contraction, but patient is dependent on indwelling catheter or repeated self-catheterization, and patient has anal sensation but cannot control function
- 2, urinary bladder and/or anal sphincter dysfunction: recovery of urodynamic studies; infrequent urinary incontinence and diminished flow, occasional self-catheterization still required, and constipation
- and
- 3, full sphincter function

Table 2. Clinical Scores, Treatment, and Outcome Results for the Study and Control Groups*

Patient No.	Presentation Score	Treatment	Outcome Score	Hyperbaric Oxygen Sessions	Final Score
		Helium-Oxygen–Treated Group			
1	0	CX-30	5	9	7
2	1	CX-30	7	5	8
3	4	CX-30	8	1	9
4	1	CX-30	8	7	9
5	1	CX-30	4	17	8
6	4	CX-30	8	0	9
7	3	CX-30	9	0	9
8	0	Saturation	1	25	2
9	7	CX-30	8	7	9
10	7	CX-30	9	0	9
11	5	CX-30	9	0	9
12	6	CX-30	8	20	9
13	0	CX-30	2	30	3
14	1	Extended CX-30	5	25	8
15	2	Saturation	5	20	8
16	3	Extended CX-30	6	10	7
		Air-Oxygen–Treated Group			
1	7	USN-6	9	0	9
2	8	Extended USN-6	7	6	9
3	8	USN-6	9	2	9
4	8	USN-6	9	0	9
5	4	Extended USN-6A	0	17	5
6	8	Extended USN-6	7	17	8
7	5	Extended USN-6	2	26	4
8	8	Extended USN-6	9	0	9
9	8	Extended USN-6	9	4	9
10	7	USN-6	9	0	9
11	8	Extended USN-6	9	0	9
12	8	USN-6	8	2	8
13	8	Extended USN-6	7	2	7
14	8	USN-6	9	0	9
15	8	Extended USN-6	8	1	9
16	8	USN-6	8	1	9
17	7	USN-6	8	2	8

**CX-30 indicates Comex treatment Table CX-30[41]; USN-6 and USN-6A, US Navy treatment Tables 6 and 6A, respectively.[42]*

The mean final clinical scores for the helium-oxygen– and air-oxygen–treated groups were 7.68±2.1 (range, 2-9) and 8.17±1.5 (range, 4-9), respectively, and these scores were significantly higher than the scores at presentation ($P<.001$ and $P=.005$, respectively, Mann-Whitney rank sum test). No significant difference was found between the final scores of the groups. However, in 5 of the patients in the control group, the neurologic status was found to be worse after air-oxygen recompression. In 2 of these patients, the final clinical score was poorer than that found at presentation (Table 2). No deterioration in patient status or relapse of symptoms and signs occurred in the helium-oxygen–treated group.

To evaluate the contribution of helium-oxygen or air-oxygen recompression treatment alone to the final outcome, comparisons were made among the patients' initial clinical scores, the scores immediately after the helium-oxygen or air-oxygen treatment, and scores at the conclusion of the adjunctive hyperbaric oxygen sessions. The results of these comparisons are detailed in **Table 3**. Most of the improvement in the clinical scores of the patients in the study group may be attributed to the helium-oxygen treatment, whereas the additional benefit gained by the supplemental hyperbaric oxygen sessions did not add significantly to the variance between the initial and final scores. Contrary to this, no significant difference could be demonstrated in the control group between the average scores at presentation and at completion of the air-oxygen recompression treatment table.

COMMENT

The group of 16 patients who were treated with helium-oxygen had severe neurologic DCS with known ominous characteristics that indicated a bad prognosis. All patients included in the study presented with firm evidence of central nervous system involvement, as found on neurologic physical examination and quantified by a strict scoring scale. The time from surfacing to the appearance of symptoms was short (less than 30 minutes in 14 patients). The correlation among short latency, DCS severity, and poor outcome has been reported previously in animal studies[43] and in clinical series.[44] The time from surfacing to the start of any recompression treatment or the helium-oxygen table was no more than 25 hours after the diving accident. Six of the patients, 5 of whom had already been treated using a US Navy air-oxygen table, received helium-oxygen treatment more than 21 hours after the diving accident. However, despite the long delay in commencing treatment, significant improvement was reported in 5 of the 6 patients with clinical scores of 8 and 9 at discharge. One may speculate as to the quantity of nitrogen that is still present in the tissues after such a long interval, particularly when

an air-oxygen recompression table has previously been used, and as to the time delay beyond which it might be anticipated there will be no improvement in patient outcome despite helium-oxygen recompression therapy. Although inert gas elimination from tissues is, for the most part, simply the reverse of gas uptake, when gas bubbles form and clinical signs of DCS appear, it is likely that the rate of gas elimination will be reduced. Gas molecules within a bubble are less available for diffusion out of the tissue than when they are in solution. The presence of bubbles within a tissue will increase the hydrostatic pressure (particularly in tissues with low compliance [eg, myelin sheaths]) and, consequently, reduce tissue perfusion. Intravascular bubbles have been reported even after recompression treatment of DCS.[45] Delayed elimination of the inert gas from the tissues and the suggested presence of gas bubbles long after surfacing from a dive, even after recompression, might explain the clinical response to late helium-oxygen therapy that was observed by us and previously reported even in cases where treatment was delayed up to 7 days after the onset of DCS.[20]

The theoretical benefit of helium-oxygen recompression treatment, when air bubbles inside a tissue with a considerable lipid content (eg, the white matter of the nervous tissue) are considered, may be attributed to the greater outflux of nitrogen under helium-oxygen than when breathing oxygen alone. When gas exchange is limited by tissue perfusion, the lower solubility of helium in blood and lipids compared with that of both nitrogen and oxygen would facilitate quicker bubble elimination. In the case of diffusion limitation, hyperbaric helium-oxygen breathing will still be of an advantage in fatty tissue in which gas exchange is determined by the product of the solubility and diffusion coefficients; this product is lower for helium than it is for nitrogen and oxygen.[27,46] However, when gas exchange is diffusion-limited in aqueous tissue, bubbles would be expected to grow while breathing helium-oxygen, as the product of the solubility and diffusion coefficients in water is greater for helium than it is for nitrogen.[28] Thus, helium-oxygen might not be a good alternative to air-oxygen recompression when inner ear or pulmonary involvement in DCS are being considered.

A significant advantage of helium-oxygen over oxygen at 1 ATA was found when SEP latency was evaluated in rats suffering from spinal DCS.[34] However, no such benefit could be demonstrated when changes in SEP amplitude were used as criteria for the response to recompression treatment in a canine model of spinal DCS while the dogs were breathing helium-oxygen (80:20) at 6 ATAs.[37]

An initial increase in the size of bubbles injected into the spinal cord of rats was demonstrated while the rats were breathing oxygen at 1 ATA or at 2.8 ATAs after decompression, whereas under helium-oxygen (80:20), no bubble enlargement could be demonstrated.[31,32] However, the total time that was taken by bubbles to shrink and disappear at 2.8 ATAs was shorter for oxygen.[31]

The initial growth of air bubbles during oxygen breathing, which was predicted by theoretical deliberations[27] and proved by the animal models, might explain

Table 3. Helium-Oxygen vs Air-Oxygen Protocol: Treatment Results

	Score, Mean±SD		
Protocol	Presentation	Intermediate	Final
Helium-oxygen*	2.81±2.45†	6.37±2.5†	7.68±2.15†
Air-oxygen‡	7.41±1.17§	7.47±2.5	8.17±1.5§

*P<.001 by Kruskal-Wallis 1-way nonparametric analysis of variance.
†P<.001 by Mann-Whitney rank sum test.
‡P=.01 by Kruskal-Wallis 1-way nonparametric analysis of variance.
§P=.005 by Mann-Whitney rank sum test.

the occasional deterioration that was observed by ourselves and others during and after the treatment of neurologic DCS with air-oxygen tables.[26,27,39] Although the symptoms and signs of neurologic DCS might be attributed to extensive bilateral cerebral involvement,[47] in the majority of cases, they are compatible with spinal cord involvement.[25] It has been suggested that whenever the increased pressure caused by bubble accumulation in the white matter of the spinal cord exceeds the feeding arteriolar closing pressure, ischemia will develop in the spinal cord.[18] Even a temporary growth in the size of bubbles might cause the critical increase in tissue pressure that is required for arteriolar occlusion, aggravating spinal cord ischemia and neurologic dysfunction.

A further benefit of helium-oxygen is that higher treatment pressures may be maintained since the mixture eliminates the complication of oxygen toxic effects that may arise when pure oxygen is used at similar pressures. Higher pressures will enhance the mechanical reduction of the size of the bubbles and increase the diffusion gradient for nitrogen without an additional gas burden.[30] When helium-oxygen (50:50) is used at 4 ATAs, the advantages of increased pressure are achieved, while oxygen is still provided at a partial pressure that enables the maximal therapeutic effect on neurologic DCS to take place.[48,49]

Two animal studies of pulmonary DCS that used dog and guinea pig models suggest that helium-oxygen treatment is of no benefit.[35,36] In one of these studies, helium-oxygen breathing caused an 11% to 22% increase in pulmonary vascular resistance; this finding indicated aggravation of the bubble-induced pulmonary vascular obstruction.[35] Indeed, when bubbles in the pulmonary microcirculation cause platelet aggregation, denaturation of lipoproteins, and activation of leukocytes, the coagulation cascade, and the complement system, the resulting endothelial damage and blood vessel occlusion will lead to diffusion-limited gas exchange in aqueous surroundings. Under these circumstances, helium-oxygen would not be expected to be of benefit. A recent study found that air bubbles that were injected into the anterior chamber of the rat's eye, where gas exchange is known to be dominated by diffusion in the aqueous humor, grow in size when the animals breathe helium-oxygen (80:20).[33] Similarly, inner ear DCS caused by bubbles in the inner ear fluids and vessels might not respond to helium-oxygen recompression treatment if blood-bubble interaction has already caused significant compromise of perfusion.

As theoretical considerations and most animal studies have shown that helium-oxygen is of benefit in central nervous system bubble disease, but might be inappropriate to the treatment of pulmonary and inner ear DCS under diffusion-limited circumstances, we use helium-oxygen treatment protocols only for neurologic DCS.[50,51]

Our results suggest an advantage of helium-oxygen recompression over the traditional air-oxygen treatment tables. The air-oxygen–treated control group had several characteristics that should have predicted a better outcome: less severe involvement of the central nervous system, as reflected by a significantly higher neurologic functional score at presentation; significantly shorter bottom times and, thus, a smaller nitrogen burden; and a longer interval from surfacing to the appearance of symptoms known to be correlated with a better treatment outcome. Yet, no differences were found between the groups' final outcome scores. In addition, most of the improvement in the clinical scores of the helium-oxygen–treated group was found to be attributable to the primary helium-oxygen recompression treatment, while the additional benefit of the supplementary hyperbaric oxygen sessions was much less. No such differences could be demonstrated for the relative benefits of the air-oxygen recompression tables and supplementary hyperbaric oxygen therapy. Moreover, clinical deterioration occurred in 5 (29%) of the patients in the control group after completion of the air-oxygen treatment tables. Two of these patients had worse clinical scores at discharge than at presentation.

We could find only a single previous report in which the preliminary results of helium-oxygen and air-oxygen recompression treatment of air-diving DCS were compared.[23,40] In that study, all patients with DCS were initially treated at 2.8 ATAs either by use of US Navy treatment Table 6 or an identical treatment profile using helium-oxygen (50:50) instead of oxygen. No differences in outcome were found between the groups for the first 41 patients who were treated.[40] Unfortunately, no stratification was made of disease severity and the systems involved, and the results were not related to the type of DCS. Differences in gas exchange, differences between the solubility and permeability of helium and oxygen for the various tissues involved, and variance in perfusion make it necessary to distinguish between different types of DCS, as one might expect different responses to helium-oxygen. In addition, the maximal P_{O_2} provided using the helium-oxygen (50:50) mixture was only 1.4 ATAs—far below the reported optimal oxygen pressure of 2.0 ATAs for the treatment of neurologic DCS.[44] No advantage was taken of the higher ambient pressure that can be used with a helium-oxygen (50:50) mixture while avoiding the risk of oxygen toxic effects and increasing the diffusion gradient for nitrogen. The treatment protocol used and the way the data were evaluated might conceal important beneficial effects of helium-oxygen recompression and make a comparison with our results impossible.

We believe that our results support the findings from theoretical and animal studies demonstrating the benefit of helium-oxygen recompression therapy for severe neurologic DCS. The limitations of using a retrospective control group warrant a multicenter prospective study comparing air-oxygen and helium-oxygen protocols. The treatment protocols to be selected should take advantage of all potential advantages of using helium-oxygen mixtures.

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