

Oxy–helium treatment of severe spinal decompression sickness after air diving

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Kol S, Adir Y, Gordon CR, Melamed Y. Oxy–helium treatment of severe spinal decompression sickness after air diving. *Undersea & Hyperbaric Med* 1993; 20(2):147–154.—Spinal cord injury in DCS after air diving is relatively frequent and often has late sequelae. U.S. Navy oxygen tables are sometimes not satisfactory. The advantage of using helium in these cases is based theoretically on its physical properties and has been demonstrated in animal models. We have introduced the Comex-30 (CX-30) oxy-helium table as an integral part of our treatment protocol for severe spinal DCS. We summarize here our clinical experience with seven cases. A case was considered severe if clinical assessment suggested progressive neurologic injury to the spinal cord or roots. Except for one case, the initial treatment was CX-30 followed by HBO sessions as indicated. Of the seven patients treated, five made a full recovery and the remaining two were left with mild neurologic sequelae.

decompression sickness, spinal, oxygen, helium, compressed air diving, scuba diving

In the majority of hyperbaric centers in the world, recompression profiles are based on U.S. Navy recompression treatment tables. The treatment schedule most often used for type II DCS is table 6, in which oxygen is the sole therapeutic gas. The option of using helium under these circumstances was first suggested by the U.S. Navy in 1959 (1).

The theoretical advantage of using helium in the treatment of DCS is based mainly on its lower solubility in fat compared to nitrogen. This is of particular relevance in spinal cord DCS, which has been more clearly defined in the conclusions of a recent workshop (2). The beneficial effect of helium has been demonstrated in laboratory work (3–5) and in clinical experience with saturation recompression therapy (6).

In our experience, CNS involvement in type II DCS resulting from air diving is dominated in two-thirds of cases by injury to the spinal cord or roots. In severe cases, despite aggressive and early therapy using U.S. Navy recompression tables 6 and 6A, patients may be left with neurologic sequelae (7, 8). We have recently used the Comex-30 (CX-30) oxy-helium recompression table (Fig. 1: table profile) in severe cases of spinal DCS after air dives. We report here our preliminary clinical experience.

MATERIALS AND METHODS

In October 1988, we started using CX-30 to treat every diving accident casualty who had severe neurologic DCS. By October 1991, we had treated seven patients. The severity of each case was assessed by the dive history (risk factors, potential gas burden, violation of the recommended decompression tables) and by the clinical presentation (time from surfacing to symptoms, motor or autonomic involvement, objective clinical signs). A case was considered severe if clinical assessment suggested progressive neurologic injury to the spinal cord or spinal roots. The main clinical findings are summarized in Tables 1-3. The patients underwent a thorough clinical evaluation: general and diving history, a complete physical examination with emphasis on neurologic and otologic aspects, chest x-ray, complete blood count, blood chemistry and gases, and ECG. The treatment was done in a multiplace hyperbaric chamber. During treatment the chamber was pressurized with air while the patients breathed the prescribed gas via a face mask. The patients were reevaluated during and after the initial CX-30 treatment. Further hyperbaric sessions were given as indicated: 2 atm abs, 90 min, 100% O₂, once or twice daily until no further significant clinical improvement was noted.

CASE HISTORIES

Case 1

A 39-yr-old fishing diver made a hookah dive to 40 msw for 60 min and ascended without decompression stops. After a surface interval of 10 min he made a second

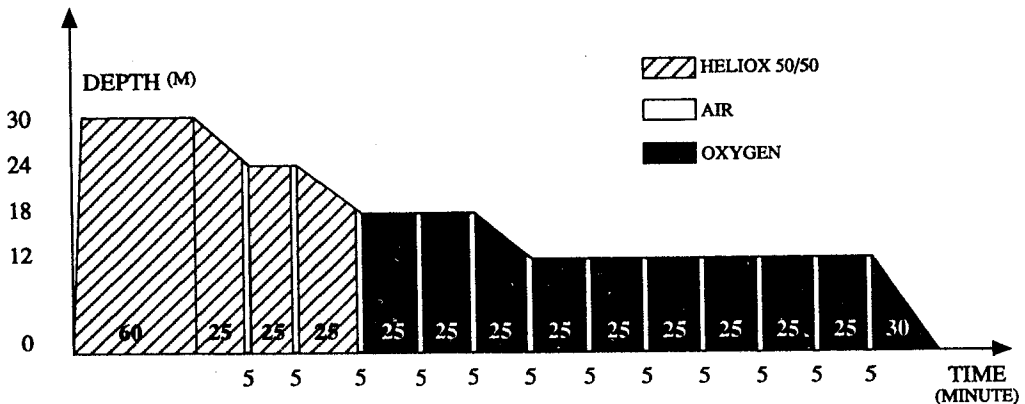


FIG. 1—Comex table 30 (CX-30). A gas mixture of 50:50% He:O₂ is used for the first 150 min of the treatment.

Table 1: Dive History

Case	Age	Type of Dive	Depth, msw	Bottom Time, min	Decompression Stops	Violation of Tables
1	39	fishing	40	60 × 2 ^a	no	yes
2	36	sport	31	35	10 min 3 m	yes
3	46	fishing	42	180	no	extreme
4	37	work	24	49	3 min 3 m	yes
5	35	fishing	34	80	no	yes
6	39	sport	30	25	3 min 3 m	no
7	26	fishing	27	60	5 min 3 m	yes

^aSurface interval between dives: 10 min.

dive to 40 msw for 60 min, again without decompression stops. Ninety minutes later, low back pain appeared, with ataxic gait, numbness and paresthesia of lower limbs, and urinary retention. Examination disclosed impaired sensation below T-12, paraparesis, and neurogenic bladder. Oxy-helium CX-30 was given 4 h after surfacing. After the first 2 h of the table he was able to walk, and the low back pain disappeared. By the end of the 7.5-h table, a complete neurologic examination was normal. However, the patient continued to complain of mild paresthesia below the right knee. This disappeared after a single HBO session the following day, and the patient was discharged.

Case 2

A 36-yr-old sport diver made a dive to 31 msw for 35 min. He made a 10-min decompression stop at 3 msw. Fifteen minutes after surfacing he developed retrosternal tightness, general weakness, headache, and numbness in both feet. Examination revealed tachypnea, hypotension (80/50), hyperreflexia, and a rash. CX-30 was given 4 h after surfacing. Sixty minutes after the commencement of therapy, while still breathing heliox, the headache and weakness disappeared. Thirty minutes later the rash disappeared. By the end of the hyperbaric treatment, physical examination was normal. An additional examination the following day was also normal, and the patient was discharged.

Case 3

A 46-yr-old fishing diver, who had a history of spinal DCS a year previously, made a hookah dive to 42 msw for 180 min, ascending without decompression stops. Fifteen minutes after surfacing he was unable to stand. On examination, severe DCS was diagnosed with loss of sensation below T-11, paraparesis, and neurogenic bladder. Due to the delay in reaching the chamber, CX-30 was given only 24 h after surfacing. During treatment the patient's motor function improved, and he was able to stand

Table 2: Symptoms and Signs

Case	Time From Surfacing to Symptoms	Low Back Pain	General Symptoms and Signs	Neurologic Symptoms	Neurologic Signs
1	90 min	+	no	numbness and paresthesia, lower limbs	ataxic gait, paraparesis, neurogenic bladder, sensory level T-12
2	15 min	-	retrosternal tightness, headache, weakness, rash, tachypnea, hypotension	numbness, both feet	hyperreflexia
3	15 min	+	weakness, vomiting	numbness, both feet	paraparesis, neurogenic bladder, sensory level at T-11
4	90 min	-		lt elbow pain, followed by numbness	motor and sensory injury, C6-C8 roots
5	immediately	+		numbness, lower limbs	sensory level T-10, patellar clonus, paraparesis
6	during ascent	-	groin pain, shoulder pain	weakness and numbness, both legs	sensory level T-8, paraparesis, neurogenic bladder
7	20 min	-	nausea, rt shoulder pain	numbness, rt hand	hypoesthesia, rt arm, interossei weakness

and walk in the chamber. Bladder function returned to normal only after an additional 19 HBO sessions. As no further clinical improvement was noted, the patient was discharged with mild proximal muscle weakness in his right leg.

Case 4

A 37-yr-old professional diver made three dives to 24 msw with a total bottom time of 49 min. On his final ascent he made a decompression stop for 3 min at 3 msw. Possible risk factors were obesity, considerable exertion, and fatigue. Left elbow pain appeared 90 min after the dive, followed by left hand numbness. Neurologic examination revealed total paralysis of the left hand and concomitant sensory loss at the C6-C8 segments. Treatment was started with CX-30 9 h after surfacing.

Table 3: Treatment and Outcome

Case	Time to CX-30	HBO	Outcome
1	4 hours	1	full recovery
2	4 hours	no	full recovery
3	24 hours	19	mild proximal muscle weakness, rt leg
4	9 hours	no	full recovery
5	24 hours (prior—table 6)	5	mild spastic paraparesis sensory level D12 urinary disturbances
6	8 hours	7	full recovery
7	3 hours	no	full recovery

Substantial improvement was noted after 5 min at 4 atm abs, and 30 min later complete resolution was achieved. A neurologic examination carried out on completion of the treatment table was normal.

Case 5

A 35-yr-old fishing diver made a hookah dive to 34 msw for 80 min, surfacing without decompression stops. Immediately on surfacing he experienced numbness of the lower limbs. Neurologic examination 3 h after surfacing revealed loss of sensation below the level of T-10, with bilateral patellar clonus and paraparesis. An initial treatment with table 6, 3 h after surfacing resulted in worsening of proximal left leg weakness. A second treatment was given with CX-30 24 h after surfacing. By the end of the heliox breathing period (150 min) the clonus had disappeared, and for the first time since surfacing he was able to walk freely. After an additional five daily HBO sessions, the patient was discharged with mild spastic paraparesis, sensory level at T12–L1, and intermittent urinary retention.

Case 6

A 39-yr-old sport diver, with a history of severe spinal DCS 2 yr before the present accident, made a dive to 25 msw for 30 min. During his ascent, he felt weakness in both legs with loss of sensation. On the surface he was unable to walk without assistance. Examination revealed sensory level at T8, paraparesis, and neurogenic bladder. Treatment with CX-30 began 8 h after surfacing. Sixty minutes after the beginning of treatment, sensory level was at T12 and motor function was normal. By the end of treatment, neurologic examination was normal, except for patchy areas of hypoesthesia in both legs. After an additional seven daily HBO sessions, the patient made a full recovery.

Case 7

A 26-yr-old fishing diver made a dive to 27 msw for 60 min. Before the dive he had eaten a relatively heavy meal. He planned a 25-min decompression stop at 3

msw, but surfaced after 5 min because of severe and uncontrolled nausea. Twenty minutes after surfacing, right shoulder pain and right hand numbness appeared. Neurologic examination revealed right hand hypoesthesia with weakness of the interossei muscles. A therapeutic trial at 2.8 atm abs on 100% oxygen for 20 min resulted in only partial improvement, and it was decided to switch to CX-30. After 60 min at 4 atm abs breathing 50:50 heliox, all symptoms disappeared and neurologic examination was normal. A follow-up examination the next day was normal and the patient was discharged.

DISCUSSION

It has been suggested that it may be inappropriate to use helium and oxygen mixtures in the therapy of compressed air illness (9), despite a recommendation from the U.S. Navy since 1959 (1). Our results support the use of oxy-helium as the therapeutic mixture in the treatment of severe spinal DCS. It is important to note that none of our patients experienced deterioration of symptoms during recompression on heliox, and we have been unable to find reports of deterioration in the literature. The treatment table we used offers the advantages of a higher initial pressure (4 atm abs compared with 2.8 atm abs in table 6) and helium as the inert gas during the first 150 min of this 7.5-h table.

Given the inherent difficulty in the precise localization of CNS lesions as presented by our patients, it is possible that cerebral decompression injuries were responsible for some of the signs and symptoms described. The problem of localizing the lesion has been discussed extensively (2). However, as the composition of spinal and cerebral tissue is comparable, we suggest that oxy-helium can be employed safely and effectively in cases in which there is insufficient clinical information to obtain a formal and complete localization of the lesion. Moreover, in an emergency situation, when early recompression therapy is crucial, the information required is often unavailable. In such cases, further diagnostic work up must await completion of the first recompression treatment.

The physiologic effect of recompression therapy in DCS is twofold: mechanical compression of the free gas phase (combined with reversal of gas flow from the surrounding tissue to the bubble), and rapid elimination of the inert gas dissolved in body tissues. Oxygen as the sole therapeutic gas has two fundamental limitations: its maximal therapeutic partial pressure in the chamber, which is limited to 3 atm abs, and its flux into fatty tissue. With equal partial pressure differences, the flux of oxygen is twice that of nitrogen and 4 times that of helium (10). This may be responsible for a temporary increase in the volume of nitrogen bubbles, especially in tissues with a low O_2 consumption rate, and hence the deterioration occasionally seen in the patient's condition (10, 11).

The flux of helium into fat is only half that of nitrogen (10). Hyldegaard and Madsen (3) decompressed rats from a 4-h exposure at 3.3 atm abs on air, and observed the behavior of gas bubbles in adipose tissue under various conditions. The bubbles grew during air breathing, whereas they shrank and disappeared during heliox breathing. With oxygen, most bubbles initially grew, then shrank and disappeared. The same consideration may raise concern about the behavior of N_2 bubbles situated in more aqueous tissue. In this case, the product of the solubility and diffusion coeffi-

cients is greater for helium than for nitrogen. It might therefore be expected that if gas exchange is limited by diffusion through a predominately aqueous medium, nitrogen bubbles would grow if the gas surrounding them were quickly changed to helium. However, similar results were obtained from an experiment conducted on N₂ bubbles in the spinal white matter of the rat, which contains only 18% fat (4, 5). These results suggest that gas exchange in spinal white matter is limited by blood perfusion.

Ignoring the controversy regarding their origin, it is generally agreed that bubbles are responsible for the signs and symptoms of spinal DCS. Other clinical manifestations of DCS (e.g., "chokes," inner ear) deserve a separate discussion because of the dependence of the behavior of the bubbles on tissue composition and blood perfusion. For example, Sergysels et al. (12) ventilated animals with He-O₂ during venous air embolism and found hemodynamic changes compatible with the growth of intravascular bubbles due to the rapid inward diffusion of helium.

On the other hand, if we consider bubbles in flowing blood when a gas switch is made, the sole determinant of growth will be the gases' relative solubility in the blood. In this case, a switch to helium should shrink the bubbles because helium is less soluble in the blood than is nitrogen (13, 14).

Using an animal model designed to correspond to an unplanned ascent by a diver with a very large gas burden, Catron et al. (15) found that He-O₂ breathing did not cause dramatic deterioration in cardiopulmonary function. In a similar experiment with guinea pigs, Lillo et al. (16) found that recompression with air seems to be slightly more effective than with He-O₂. These authors suggest that heliox may actually interfere with normal bubble resolution and hence advise caution in the use of heliox in the treatment of DCS resulting from air dives.

These two models focus on nitrogen bubbles in the pulmonary circulation ("chokes"), but clinical experience emphasizes spinal cord injury as the most significant in terms of the number of patients and late sequelae. Hyldegaard et al. (17) used a rat model of spinal DCS to examine the influence of normobaric air, oxygen, and heliox (80/20) on spinal evoked potentials representing spinal cord injury. The results suggest that both oxygen and heliox have a protective effect against DCS. The effect of heliox seems to be superior to that of oxygen alone. However, a more extreme experiment using dogs, with the gases administered under pressure (6 atm abs), failed to demonstrate any advantage in using a heliox mixture as an adjuvant to recompression (18). This latter model is probably associated with considerable edema.

Definite conclusions cannot be drawn from these animal models, and clinical experience with CX-30 has not been published. To the best of our knowledge, only a few hyperbaric centers besides Comex use this treatment table routinely. We believe that both theoretical considerations and experimental evidence (3-5, 14, 17) warrant further clinical trials on the use of heliox in the treatment of spinal DCS to build a database that will allow a strict comparison with O₂ tables.

Manuscript received June 1992; accepted February 1993.

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