Spinal Cord Decompression Sickness in Sport Diving

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• **Objective.**—To summarize 16 years' experience in the diagnosis and treatment of spinal cord decompression sickness in Israel.

Design.—The survey data were collected firsthand by physicians trained in underwater diving medicine.

Setting.—The Israeli Naval Medical Institute, Israel's national hyperbaric referral center.

Patients.—Sixty-eight sport divers diagnosed as having spinal cord decompression sickness.

Interventions.—Hydration and 100% oxygen breathing until the patient reached the hyperbaric chamber. All patients received recompression therapy on US Navy treatment tables using oxygen, except for six who were treated by Comex Treatment Table CX-30, which uses helium in addition to oxygen.

Main Outcome Measures.—Neurological examination after the completion of recompression therapy.

Results.—Forty-one percent of the dives were performed within the decompression limits of the US Navy standard decompression tables. Risk factors were fatigue, circumstances suggesting dehydration, and extreme physical effort. The most common presenting symptoms were paresthesias, weakness of the legs, lower back pain, or abdominal pain. Full recovery was achieved in 79% of the patients. Spinal symptoms appeared immediately on surfacing in six of the eight patients who continued to have multiple neurological sequelae.

Conclusions.—United States Navy air decompression tables appear not to be completely safe for sport divers. Even mild spinal symptoms identified on surfacing should be treated vigorously. High-pressure oxygen-helium therapy seems to be a promising alternative in cases of severe spinal cord decompression sickness.

(Arch Neurol. 1993;50:753-756)

D ecompression sickness (DCS) is a clinical syndrome caused by alterations in environmental pressure,[1-3] which result in the liberation into tissue or blood of inert gas bubbles previously loaded within tissues as a soluble phase.[4,5] Sport divers generally use compressed air as the breathing mixture, and in their case, the inert gas that may be liberated during decompression is nitrogen. Pathophysiological changes of DCS are attributable to bubble formation and to the damage caused by the bubbles to tissues and blood supply. Based on clinical manifestations, the generally accepted classification of DCS is type I or II. Type I includes joint pain, skin marbling, small patchy hemorrhages, and lymphatic obstruction, while type II is dominated by injury to the central nervous system, mainly spinal, and inner ear and lung involvement.[6]

In a recent innovative workshop, an effort was made to establish a more accurate classification of type II DCS according to the anatomic localization of the lesion.[7] Spinal cord injury represents one of the most ominous manifestations of the disease, affecting 20% to 50% of cases resulting from air diving and is frequently resistant to recompression therapy.[8-12] Therapy consists of recompression and the concomitant administration of high concentrations of inspired oxygen according to a diverse selection of therapeutic tables.[8] The oxygen partial pressure should be high to increase the inherent unsaturation and hence the driving force for resolution of the bubble. On the other hand, the overall exposure should not precipitate oxygen toxicity. Until the patient reaches a hyperbaric medical facility, the treatment for DCS includes proper hydration and the administration of oxygen at the highest possible concentration.[1-3]

During 1975 to 1990, there were 227 air scuba diving accidents treated at the two hyperbaric centers in Israel and evaluated at the Israeli Naval Hyperbaric Institute, Haifa. We summarize herein our experience in the diagnosis and treatment of 68 cases of spinal cord DCS. All patients were evaluated and treated by our staff, forming, as far as we are aware, the largest series of patients in which a descriptive history of dive profile, risk factors, and time delay to recompression is reported firsthand by physicians with special training in diving medicine.

PATIENTS AND METHODS

Sixty-eight divers (59 men and nine women; mean age, 33.9±9.4 years; range, 20 to 66 years) presented with spinal cord symptoms. Twenty-five percent (17) were inexperienced divers, while 75% (51) were experienced amateur or professional divers. All dives were made in open water, most of them recreational. The typical depth and bottom time were 25 to 30 m and 30 to 40 minutes, respectively. The dive profile was evaluated according to standard US navy air decompression tables,[13] which calculate the amount of nitrogen absorbed
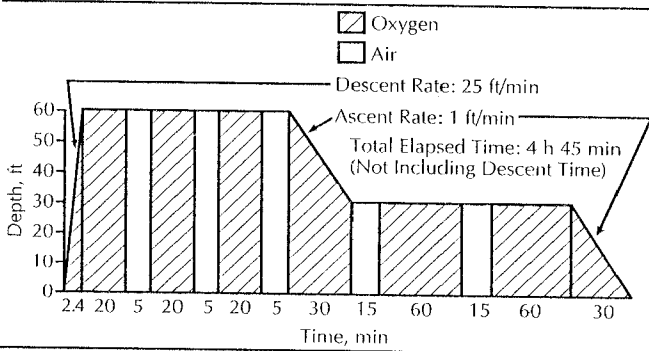
Fig 1.—*United States Navy treatment table 6 using oxygen—recommended for the therapeutic recompression of severe decompression sickness.*
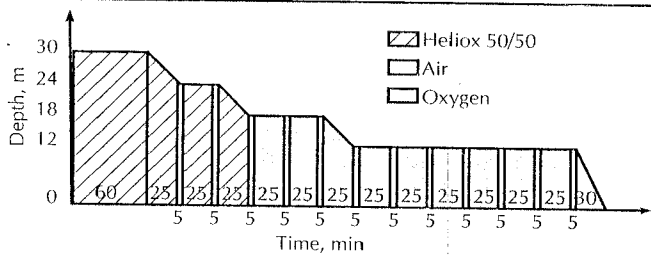

Fig 2.—*Comex Treatment Table CX-30, using 50% helium in addition to 50% oxygen (heliox 50/50) in a new approach to the therapeutic recompression of severe decompression sickness.*

during the dive to plan a safe ascent to the surface without the risk of DCS.

The dive history and possible risk factors[1] were recorded by one of the Israeli Naval Hyperbaric Institute physicians (Y.A.). All patients underwent a comprehensive neurological, physical, and otolaryngological examination. In addition, patients had an electrocardiogram and a chest roentgenogram; a blood sample was obtained. When DCS was diagnosed, intravenous fluids and oxygen were administered at the site of the accident, and the patient was immediately transferred to a recompression chamber. At the start of recompression treatment, all patients were well hydrated and hemodynamically stable. None was suffering from pulmonary manifestations of DCS. Blood cell count, including hematocrit, was normal. The treatment profile most commonly used was US Navy table 6 (Fig 1). If this regimen failed, the table was extended and the treatment was supplemented by hyperbaric oxygen sessions (2.8 atmospheres absolute [ATA], 90 minutes, 100% oxygen) twice daily, either until the patient fully recovered or until no further improvement could be observed. Recently, we successfully treated six patients with 50% helium–50% oxygen as the therapeutic breathing mixture, starting at 4 ATA and followed by 100% oxygen at 2.8 ATA according to Comex Treatment Table CX-30 (Fig 2).

RESULTS

Twenty-eight dives (41.2%) that led to spinal DCS were well within the dive tables, while 58.8% (40/68) deviated from the tables. Of the dives that were carried out within the limits of the dive tables, 64.3% (18/28) had at least one risk factor. The predominant factors for the DCS included fatigue, circumstances suggesting dehydration before the dive (reduced fluid intake, reduced urination, excessive perspiration, and extreme thirst), and exceptional physical effort during the dive (Table 1). Dives within the decompression tables that led to DCS were generally made to a depth of 25 to 30 m. In 29.4% (20/68) of patients, the first

symptoms were present on reaching the surface, and in 41.2% (28/68), symptoms appeared within 30 minutes of surfacing. The average delay from symptoms to recompression therapy was 11 hours (range, 0.5 to 30 hours).

Fifty-five patients (81%) had spinal cord involvement as the sole manifestation of DCS, 10 (14%) had a combination of spinal and cerebral involvement, and three (4%) had spinal and vestibular manifestations. Cerebral involvement was diagnosed when a transient alteration of consciousness, blurred vision, diplopia, dysarthria, or cranial nerve symptoms were present, in addition to spinal signs of DCS. Vestibular manifestations included vertigo, nausea, and nystagmus of peripheral origin. Pure spinal cord involvement was diagnosed when none of the aforementioned symptoms or signs was present. Presenting complaints of spinal involvement included ascending warm or prickly paresthesias that generally progressed to numbness, leg weakness, lower back pain, and abdominal pain (Table 2). Lower back pain or abdominal pain were usually followed by motor weakness and sensory loss. The neurological examination revealed paraparesis in 18 patients (26%) and monoparesis in six (9%). A definite sensory level was observed in 18 patients (26%), in 10 (55%) of them at the level of the D12-L1 dermatome. Bladder disturbances were present in 11 patients (16%). Full recovery was achieved in 53 (78%) of the 68 patients with spinal cord involvement. Fifteen had residual neurological symptoms, and eight still have multiple sequelae. All of the eight were treated within 2 hours of surfacing. In six of them, symptoms appeared immediately on reaching the surface, and after examination of depth and bottom time it became evident that all had gone deeper than 30 m with an extended bottom time.

COMMENT

The pathophysiology of DCS is still a controversial issue. Despite following comparable diving profiles, some pa-

Table 1.—Risk Factors for Dives Within the Limits of the Tables That Resulted in Decompression Sickness	
Risk Factors	**Patients, No.**
Fatigue	7
Dehydration	4
Extreme physical effort	3
Flying after the dive	2
Menstruation	2
Fever	1
Advanced age	1

Table 2.—Presenting Complaints	
Complaints	**Patients, No.**
Paresthesia	59
Weakness in legs	27
General malaise	19
Pain	
Lower back	14
Abdominal	3
Burning sensation in limb	1

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tients make a full recovery while others are left with major neurological deficits.[8,9]

Three theories have been proposed in an attempt to describe the pathophysiological events that lead to DCS. The "arterial" theory claims that the bubble's effect is mediated through arterial embolization of the microcirculation in the spinal cord and local growth of the bubbles by the assimilation of inert gas from the surrounding tissues, finally leading to obstruction of the circulation.[14-17] The "venous" theory proposes that bubbles in the slow-flowing spinal epidural venous bed activate the clotting system and platelet aggregation, leading to stagnation and venous obstruction.[18-21] The "autochthonous" theory suggests that when the tissues are loaded with inert gas and decompression is rapid, bubbles may nucleate within the tissues themselves.[22] As white matter has a high fat content and a poor blood supply, autochthonous bubbles may appear and create sufficient local pressure to occlude blood flow, causing anoxic damage and myelin sheath disruption since bubbles behave like a space-occupying lesion within the nervous tissue. The high fat content of the myelin, together with the high solubility of nitrogen in fat, explain the vulnerability to injury of the spinal cord.

The pathological features of spinal DCS include hemorrhagic infarcts, edema, axonal degeneration, and severe demyelination of the affected areas, even when the clinical manifestations during life were unimpressive.[14,23]

Our data on spinal DCS are similar to previously reported data regarding the symptoms and the distribution of spinal cord injury.[7-9] Early symptoms of lower back pain or abdominal pain were generally followed by aggravation of the disease. As in other reports,[24-27] in our population, the D12-L1 spinal cord segments were most frequently affected, which is believed to reflect the vulnerability of the blood supply in this region of the spinal cord.[28] The clinical presentation of spinal DCS frequently differs from any other acute spinal syndrome in which neurological examination can isolate the level of the lesion. Spinal DCS may produce multifocal lesions at various tracts and levels of the cord, resulting in a clinical picture of combined pain, sensory disturbances (frequently patchy), and motor weakness at multiple sites. Even after a detailed neurological examination, the determination of the level of the injury may be extremely difficult in many cases of neurological DCS.[7]

In six of the eight patients who continued to have multiple neurological sequelae despite recompression treatment within 2 hours of surfacing, symptoms had appeared immediately on reaching the surface. This finding suggests that a short latency to the onset of neurological symptoms may be associated with a poor outcome. A similar conclusion can be reached on the basis of other clinical data[10] and from experimental studies in dogs.[29] In the latter, a short latency to the onset of spinal DCS was significantly correlated with severity and poor outcome. It has also been well established that 20% to 30% of patients with minor manifestations of DCS may progress to more serious neurological involvement.[30] All of these facts stress the importance of rapid recognition and aggressive recompression treatment of the patient with even minor spinal DCS symptoms on surfacing.

Besides depth and bottom time, several risk factors expose divers to DCS. Dehydration, by increasing blood viscosity, and physical stress, by increasing cardiac output and thus loading the tissues with nitrogen, reduce the div-er's nitrogen elimination ability and expose him or her to a greater risk of DCS.[4,5] The appearance of spinal DCS despite adherence to US Navy decompression tables in 41% of our patients is not surprising and has previously been reported in the neurological literature.[9] These tables are based on experiments on fit, well-built, professional Navy divers and seem to be not completely safe when used by sport divers. It should be remembered that in addition to the risk factors mentioned herein, there are many variables associated with individual susceptibility to DCS that may account for its unpredictability in many cases.

The specific treatment for DCS is recompression as rapidly as possible. Proper hydration and 100% oxygen breathing, as well as other support treatment, are indicated until the patient reaches the hyperbaric chamber.

Hyperbaric oxygen therapy given according to US Navy table 6 is the most commonly used initial treatment for neurological DCS.[3,8] The therapeutic effect of a helium-oxygen mixture in treating patients with spinal DCS has been reported in both laboratory and clinical studies.[31-33] Since helium has a lower solubility in the structural fats of the central nervous system compared with nitrogen, treatment with oxygen-helium at 4 ATA allows a higher ambient pressure to be reached without the risk of oxygen toxicity and results in faster elimination of the nitrogen than does treatment with decompression table 6 at 2.8 ATA alone. Our limited but successful experience with 50% helium–50% oxygen at 4 ATA warrants further controlled clinical trials of this mixture in the treatment of spinal DCS.

The conclusions arising from our clinical experience are (1) as the initial symptoms of spinal DCS often fail to reflect the severity of the disease, patients with mild initial symptoms on surfacing should be identified and treated vigorously; (2) divers should be made aware of the risk factors and should take them into consideration when planning the dive profile; (3) the currently available US Navy air decompression tables do not appear to provide sufficient safety margins for sport divers, although they can be used with the addition of safety factors; and (4) based on our experience, we suggest high-pressure oxygen-helium therapy as a promising alternative in the treatment of severe spinal DCS resulting from underwater air diving.

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