Neurologic Injuries from Scuba Diving

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KEYWORDS

- Scuba diving • Decompression
- Arterial gas embolism • Decompression illness

Interest in scuba (self-contained underwater breathing apparatus) diving increased in the 1970s, and undersea diving continues to be a popular sport early in the 21st century, with approximately 3 million certified divers in the United States.[1] The Divers Alert Network (DAN), an institution created in 1981 by the Commerce Department, National Oceanic and Atmospheric Administration, has collected diving injury data for US and Canadian divers since 1987 that can be studied to suggest the epidemiologic characteristics of diving.

The 2006 annual DAN Diving Report on Diving Injuries and Diving Fatalities[2] is based on data collected during 2004. This diving report presents information on Project Dive Exploration (PDE), which is a prospective observational study of recreational diving yearly data since 1995. PDE divers are volunteers from the general recreational diving population, but they are not necessarily representative of this population. Most PDE divers are 30 to 50 years of age; 22% are over the age of 50, and 4% are under 20 years of age. Thirty percent of the divers are female. Sixty-six percent of the female divers hold open water, advanced open water, or specialty certification, verses 46% of the males.

The annual number of injury cases reported has increased from 1987 to 2004, with about 600 cases in 1987 and 1100 cases in 2002 (**Fig. 1**).[2] The dramatic dip noted in 2003 represents decreased data collection because of the Health Insurance Portability and Accountability Act (HIPAA). The mean age of divers in the DAN injury population was 39 years (**Fig. 2**).[2] Interestingly, divers with advanced certification had the highest percentage of injuries, but this may be due to unknown diving frequency and may differ between the varying certification categories (**Fig. 3**).

The annual record of US and Canadian diving fatalities, began in 1971 by John McAniff of the University of Rhode Island, was transitioned to DAN in 1989.[2] The number of fatalities reported from 1970 to 2004 ranges from 80 to 120 annually, with a stable number of fatalities (**Fig. 4**).[2] The general diving population seems to be aging, as a larger percentage of divers have been diving for more than a decade

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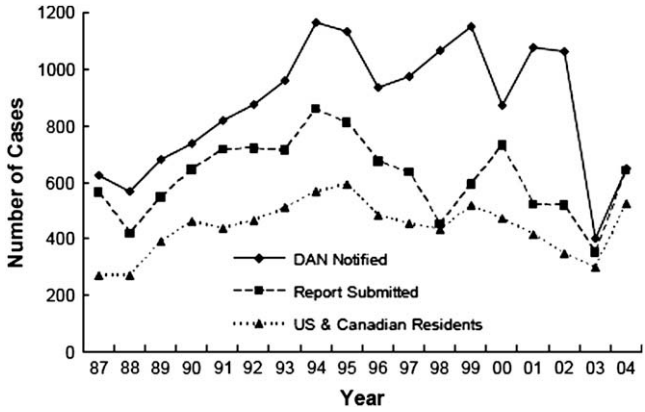

Fig. 1. Annual record of dive injury cases.

since certification. This aging of the diving population may account for the increase in the mean age of diving fatalities from 1989 to 2002. The mean age of divers experiencing injuries also increased from 33 to 39 years during this time.[2]

Medical history (limited) was available in 40% of the fatality cases, and the most frequently reported medical conditions were heart disease and high blood pressure. Most of the fatalities in the DAN Report had open water or advanced certification, and 25% had been certified 10 years or greater, whereas 45% had 1 year or less.[2] This report does not specify the dive frequency among the fatalities. The most common cause of death in the judgment of the DAN pathologist reviewing each case was drowning, whereas the next most common causes were an acute heart condition or an arterial gas embolism. The cause of death was not determined in 10% of the cases, either because the body was not found or the cause was not identified by the local medical examiner.

DECOMPRESSION SICKNESS

Decompression sickness occurs when inert gas comes out of solution, forming bubbles following the reduction of surrounding pressure (decompression). This commonly occurs with breathing compressed air while diving. As the diver descends and is exposed to elevated environmental pressure, increased amounts of inert gas dissolve in the tissues. This is in accordance with Henry's law, which states that the

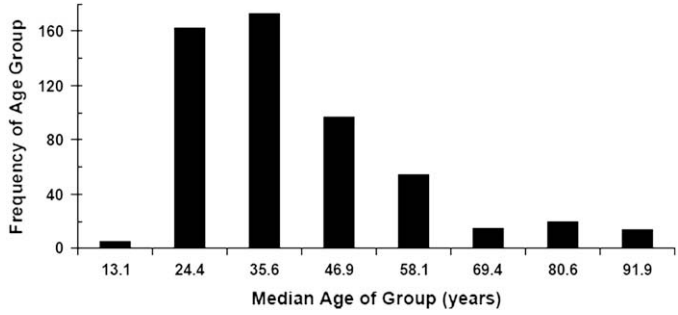

Fig. 2. Median age of group (years).

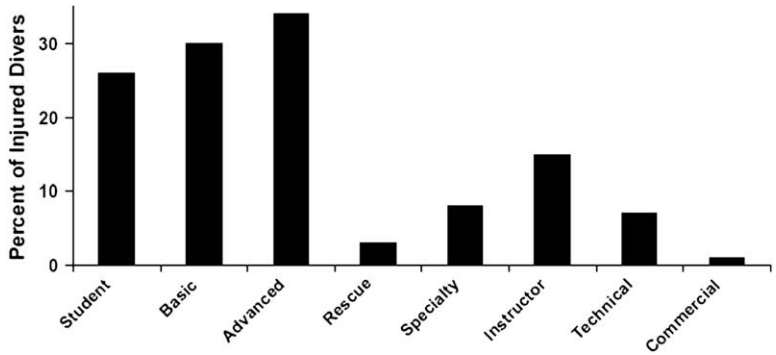

Fig. 3. Certification level of injured divers.

amount of gas dissolved in a fluid is directly proportional to the partial pressure of that gas. The amount of inert gas dissolved depends on the depth and the duration of the dive. If the diver ascends too quickly, the inert gas taken up during the dive exceeds solubility at the reduced pressure, and leads to bubble formation in tissues and in venous blood. The extent of bubble formation depends on the depth and duration of the dive and the rate of the ascent.

The likelihood of decompression sickness is related to the extent of bubble formation. A few bubbles coming out of solution may cause only minor symptoms; however, a large bubble load may result in multisystem failure and death. There are two general types of decompression sickness, Type I and Type II. Small gas loads typically cause Type I decompression sickness, which is characterized by pain in the joints and limbs and itching in the skin (niggles). Decompression sickness Type II is serious, and is characterized by neurologic problems such as weakness or paralysis, limb paresthesias, disturbance of vision, bowel and bladder dysfunction, and vertigo. Most often, the target organ is the spinal cord, usually the thoracic level. The thoracic level is probably targeted because of the vascular anatomy of the spinal cord. The

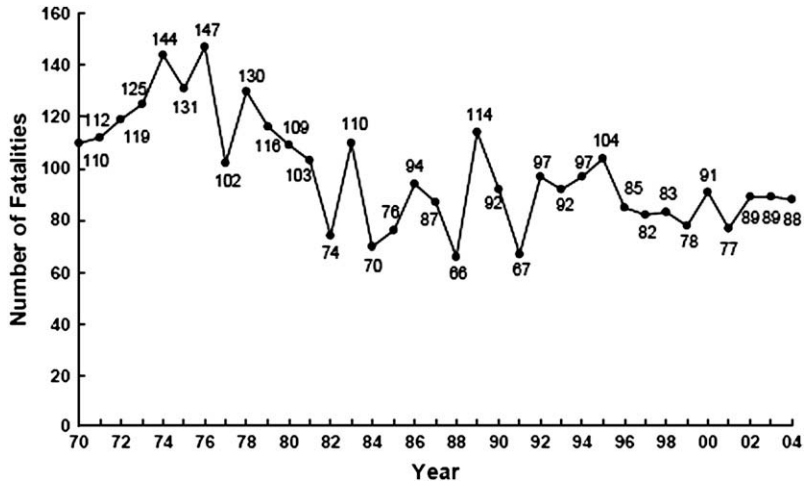

Fig. 4. Annual record of North American diving fatalities.

paravertebral veins (Batson's plexus) allows for bubbles to collect because of stagnant flow resulting in venous infarction in the spinal cord. Cerebral involvement occurs in 30% of the cases of Type II decompression sickness. Divers who have cerebral involvement may complain of confusion, lethargy, mental cloudiness, difficulty with concentration, visual disturbances and dysphagia. Symptoms typically begin within an hour of surfacing, but may be delayed for several hours. An earlier symptom onset may indicate a greater bubble load and a worse prognosis.

The diagnosis of decompression illness is based on the clinical examination, including the neurologic examination, and the dive history. Laboratory and imaging studies sometimes add to the diagnosis. In 2004 Freiberger[3] identified important diagnostic factors using simulated diving injury cases. The top five diagnostic factors in order of importance were: (1) a neurologic symptom as the primary presenting symptom, (2) onset time to symptoms, (3) joint pain as a presenting symptom, (4) any relief after recompression treatment, and (5) maximum depth of the last dive.[3] Age, gender, or physical characteristics were not statistically important.[3]

DECOMPRESSION SICKNESS: CASE ONE

A 32-year-old sport diver with several years experience was in the fifth day of a diving vacation trip to the South Pacific. On each of the previous days, he had made two or three dives, and at least one dive each day had exceeded 100 feet. He was using a decompression computer and was quite sure that he had stayed within the parameters required by the computer throughout his trip. On the fifth day, he made one dive to 150 feet and three dives to 90 feet, each separated by surface intervals that met the requirements of his computer. About 5 minutes after surfacing from his third 90-foot dive, as he was sitting on the bench, he had an aching, pressurelike pain around his flanks and into his groin. The right side was somewhat worse than the left, but as the pain became more intense, both sides were equally affected. A few minutes later, he got to his feet and was unable to walk without assistance. His companions and the boat captain helped him to the bench, where he lay down and was treated with oxygen. Symptoms did not improve. Arrangements were made to evacuate him to a recompression chamber on an island several hundred miles distant. The flight was delayed by darkness. When he arrived at the chamber the next day, about 10 hours after onset, he had moderate weakness in both thighs and virtually no strength in his left foot and lower leg, with his right foot less affected. He had altered sensation and patchy loss of sensation from the umbilicus downward, although he could feel pressure in his feet. He was unable to void without pressing on his abdomen. On urinary catheterization, he had 1500 mL residual urine. Reflexes were hyperactive.

He was treated according to US Navy Table 6. He made some improvement during the first 2 hours and treatment was extended to a full 10 hours. At the conclusion of treatment, his quadriceps and thigh strength had largely returned; his left foot was still nearly flaccid, but his right was only slightly weak. Sensation was improved, and pain resolved. The indwelling catheter was removed on the second day, and he was able to void with a Crede maneuver.

He was retreated on Table 5 (shorter oxygen table) for each of the following 3 days. He made no further improvement after the first treatment.

A week after completion of treatment, he returned to the United States by commercial aircraft. There was no change in his symptoms. In the ensuing year, he reported slow improvement in his mobility and in bladder control. One year after the event, he had persistent spastic paraparesis. He was able to ambulate without a cane or

crutches, but with moderate discernable weakness and bilateral hyperreflexia. Sensation about the perineum was decreased and was nearly normal in the feet.

This case typifies serious decompression sickness with delayed treatment and with partial response. Although this patient was diving "within the rules," he had accumulated a large gas load.[4]

DECOMPRESSION SICKNESS: CASE TWO

A 46-year-old woman, an experienced diver, surfaced after an uneventful dive to 110 feet for 27 minutes, conservatively decompressing for 13 minutes at 10 feet (only 7 minutes normally required). On climbing into the boat, about 10 minutes after the dive, her right foot felt hot, then tingly (as if it were going to sleep); the limb became progressively numb from foot to thigh over 20 minutes while the left leg also became warm and tingly, and she had low back pain. Reaching the shore after 30 minutes, she could not walk. She recovered sensation and strength while breathing pure oxygen for 60 minutes. For several days, her left leg felt strange, and there was some loss of feeling in the left foot, but she felt normal after 1 week. This is a representation of a typical case of mild decompression sickness.[5]

ARTERIAL GAS EMBOLISM

Arterial gas embolism occurs when a diver breathing compressed air at depth ascends without exhaling air from the lungs. This occurs as a consequence of Boyle's law, which states that the product of pressure times volume is constant, or as pressure decreases, volume increases and the alveoli rupture as a consequence of pulmonary overinflation.

When aveoli rupture, air escapes or dissects into the surrounding spaces. Air entering the pleural cavity results in pneumothorax; air escaping into the mediastinum causes subcutaneous or mediastinal emphysema or air dissecting into the pericardium causes pneumopericardium. More dangerous yet, air may enter the pulmonary capillaries, reaching the pulmonary vein in the left heart, and be pumped into the arterial circulation. Emboli that enter the cerebral vessels cause strokelike events that typically occur within minutes of surfacing.

Patients experiencing arterial gas embolism or pulmonary over-inflation will experience pain and respiratory distress, coughing, hemoptysis, headache, unconsciousness, seizures, hemiparesis, quadriparesis, and cortical blindness. The mortality rate is high and immediate treatment is essential.

Pulmonary overpressure accidents occur most commonly in inexperienced divers. Inexperienced divers may simply forget to exhale during ascent. More commonly, this may occur during an emergency ascent, perhaps from an out-of-air situation or equipment failure.

Similar to making the diagnosis of decompression sickness, the diagnosis of arterial gas embolism is entirely based on clinical findings. The top five diagnostic factors for arterial gas embolism cited by Freiberger and colleagues[3] are: (1) the onset time of symptoms, (2) altered consciousness, (3) any neurologic symptoms as a presenting symptom, (4) motor weakness, and (5) seizure as the primary presenting symptom.[3] The National Institute of Health Stroke Scale (NIHSS), when applied to cerebral neurologic diving injuries, is a tool for severity stratification and has adequate predictive ability while providing a more standardized scale.[6]

ARTERIAL GAS EMBOLISM: CASE STUDY

A 28-year-old man made a certification dive in a fresh water lake as part of a primary scuba course. The students were performing an emergency drill in which they made a free ascent from 30 feet, simulating an out-of-air situation. They were instructed to take a full breath and then swim to the surface while exhaling constantly.

He reached the surface, had a generalized convulsion, and lost consciousness. He was towed to shore by his companions and carried to the truck. Oxygen was not available and the patient was transported about 60 miles in the back of a pick-up truck to an emergency medical facility. During this transit, he was conscious, but stuporous and uncommunicative. The emergency department physician found that he had a right hemiparesis and was aphasic, and recommended the likelihood of arterial gas embolism. The patient was treated with oxygen and transported by helicopter to a recompression chamber 200 miles away. Dysphagia and hemiparesis persisted. He was treated in a monoplace oxygen chamber for 90 minutes, at a depth equivalent to 50 feet of sea water. Directly after he was removed from the chamber, he had a generalized and largely right-sided seizure. Oxygen treatment was continued and he was not recompressed. Over the next several hours, the hemiparesis improved and he began to utter simple words. Oxygen was continued. The next morning, he had a minimal persisting hemiparesis and a moderate aphagia. He was discharged after 3 days, substantially improved, but with minimum persisting findings. Two months after the event, his hemiparesis was no longer apparent, his speech had improved to normal, and he was functioning at near his normal level.

This case typifies the sudden onset of a cerebral event in arterial gas embolism. It also illustrates the usually good prognosis in patients who survive the initial insult.[4]

PREVENTION OF DYSBARIC ILLNESS

Divers attempt to protect themselves from decompression sickness by adherence to diving tables. Diving tables were designed by the US Navy and other agencies based on theoretical and empirical data. The tables were created from the theoretical picture of the body consisting of different tissues that accept and relieve gas at different rates, and the decompression tables are designed to allow the diver to surface at a rate compatible with the slowest tissue for the depth and duration of the dive.[7] The tables are not perfect. The US Navy reports incidence of ''the bends'' of approximately 0.5% with strict adherence to the tables.[7] Adherence to the diving tables can be challenging because the diver has no communication with the surface and must keep track of depth and time at the bottom. Decompression computers are used by many divers and provide real-time simulation of body uptake of inert gas.

Several medical illnesses increase the risk of decompression sickness and arterial gas embolism in the diver. The most common is pulmonary dysfunction, including obstructive pulmonary disease, particularly asthma, which is a relatively absolute contraindication to diving.[8] Individuals who have pulmonary dysfunction should be evaluated by a physician. Several neurologic diseases such as migraine, in particular complicated migraine, epilepsy, muscle dystrophy, multiple sclerosis, and spinal disease can increase the risk of dysbaric illness. These should be discussed with a physician knowledgeable with the challenges of diving to assess the risk.

It is generally accepted that inert gas bubbles in the tissue and venous system cause decompression sickness, and the greater the bubble load, the higher the risk of developing decompression sickness. Research has attempted to identify ways to decrease the bubble formation in diving. Duplessis and colleagues evaluated prophylactic Atorvastatin in 16 trained military divers, but this failed to reduce the number of

intravascular bubbles observed following a dry chamber dive.[9] Exercise, both pre- and post-dive, has been studied as a possible means of decreasing inert gas load and bubble formation. Dujic and colleagues studied strenuous exercise after an open sea dive in seven male military divers, and their results suggest that post-dive strenuous exercise reduces post-dive gas bubble formation in well-trained military divers.[10] The current practice is to avoid strenuous exercise after diving because its effect on bubble formation remains controversial. Pre-dive exercise was found to significantly reduce the number of bubbles in the right heart and to protect divers from decompression sickness.[11] In another study, short-acting exogenous nitric oxide before a dive was found to reduce bubble formation.[12] The use of alcohol increases the risk of dysbaric illness in divers because it adversely effects judgment, making adherence to diving tables more difficult.

TREATMENT OF DYSBARIC ILLNESS

Scuba divers breathing from a compressed air source are subject to trauma, hypothermia, asphyxiation, and water aspiration, but may also have decompression sickness or arterial gas embolism. The neurologic outcome of dysbaric illness is greatly influenced by effective early management. Typically, the diagnosis must be suspected and made in the field by a companion or dive supervisor.

Prompt recognition within the diving party is essential to begin on-site treatment. Development of pulmonary or cerebral symptoms on reaching the surface or immediately after leaving the water suggests pulmonary over-pressure injury. If cerebral symptoms such as convulsion, cortical blindness, hemiplegia, or aphasia are present, air embolism must be assumed. Symptoms delayed minutes to hours after surfacing and localizing to the spinal cord implicate decompression sickness. Prompt recompression treatment is necessary in all cases.

Once the presumptive diagnosis of dysbaric illness is established, the most important on-site treatment is administration of 100% oxygen. Most sport dive boats do carry oxygen, and ideally a close-fitting mask and reservoir that will provide high fraction of inspired oxygen and a large oxygen supply to allow for treatment until the patient is delivered to a recompression chamber.

The purpose of oxygen is to increase the rate of nitrogen removal. The damaging lesion is nitrogen bubbles within the tissue; it interrupts blood flow and damages neural structures. The basis of oxygen treatment for dysbaric illness was termed ''the oxygen window'' by Behnke and Shaw, which describes the changing pressure of nitrogen in the alveoli.[7] The administration of 100% oxygen creates a nitrogen gradient that tends to eliminate nitrogen bubbles from tissue. At the surface, the nitrogen bubbles have a calculated pressure of 633 torr and the alveoli air has a pressure of approximately 573 torr, making the off-gassing gradient 60 torr.[7] When the patient breathes pure oxygen, the alveolar nitrogen pressure becomes 0 torr and the gradient is then 633 torr, creating a tremendous treatment advantage, ''the oxygen window.'' This is the critical part of treatment of both decompression sickness and arterial gas embolism. An even greater advantage is achieved by breathing oxygen under pressure.

In the field, the next crucial step is timely transportation to a recompression chamber. This can be exceedingly difficult because diving injuries occur off-shore, frequently at significant distances from emergency medical treatment and recompression facilities. An evacuation plan and communication are essential for a successful emergency evacuation. Air evacuation, when possible, must be done at low altitude, because ascent from sea level further complicates dysbaric illness.

Nitrogen bubbles within tissues and occluding vessels cause ischemia and space-occupying lesions. Bubble volume decreases as absolute pressure increases in accordance with Boyle's law. If pressure is doubled, bubble volume is reduced by half and allows bubbles in vessels to pass downstream, thus reducing the area of ischemia and in other tissues decreasing the space-occupying lesions. The ambient pressure is increased by recompression in a chamber, but there are limits to the use of pressure. Enormous pressures are required to redissolve the nitrogen bubble; however, greater pressure increases allow further absorption of nitrogen, thus 6 atmospheres (165 feet) is the maximum conventional treatment depth.[7,8]

Decompression commonly follows the treatment schedules established by the US Navy in the late 1960s.[7,8,13] The diver is recompressed to 60 feet and breathes pure oxygen, interrupted with air breaks, for 90 minutes. Air breaks are necessary to prevent oxygen toxicity, which can cause pulmonary irritation and loss of compliance. After 90 minutes, the pressure is decreased to 30 feet over a period of 30 minutes, and the diver continues oxygen breathing for 2 additional hours. Finally, the diver ascends to the surface at one foot a minute while still breathing oxygen. The total treatment time is 4 hours and 45 minutes. Treatment schedules may be extended if diver improvement is less than complete. Treatment can be extended to 12 to 13 hours if necessary.

Cianci and Slade[13] present an alternative treatment table more suitable for monoplace chambers that was found to be effective in the treatment of decompression sickness. The use of short oxygen treatment tables, as originally described by Hart and Kindwall, were effective in the treatment of decompression sickness even with long delays to definitive recompression.[13] Bennett and colleagues[14] showed that the addition of an NSAID or the use of heliox (helium and oxygen) was found to reduce the number of recompressions required, but neither improved the odds of recovery.

DIAGNOSTIC STUDIES

The diagnosis of decompression illness is entirely based on clinical findings, because there is no objective "gold standard." The history, especially the details of the dive, assists in understanding the injury; however, in many cases the dive history is innocent. No laboratory test exists that can confirm or reject the diagnosis of decompression illness, and tests rarely assist in guiding treatment.

Divers do develop dysbaric illness in spite of diving responsibly.[6] One possible etiology in these individuals is a patent foramen ovale (PFO) resulting in bubble passage from the right to left side of the heart.[15] When this occurs the bubbles can be arterialized. Divers who have PFO have an increased risk of paradoxical brain embolization. Honek and colleagues[16] examined 28 scuba divers, 15 of whom had decompression sickness associated with the ascent. PFO was diagnosed in 53% of the 15 divers by transesophageal echocardiogram. Among asymptomatic divers, PFO was found in only 8%. The authors concluded that PFO detection is clinically useful after repeated decompression sickness and in all frequent divers and instructors.[16] Transesophageal echocardiogram is the gold standard.

Neuropsychological testing has been studied in commercial and recreational divers with conflicting results, but generally adds little to the clinical examination.[8] Neuroimaging such as CT scanning is not always sensitive enough to detect structural abnormalities associated with cerebral decompression sickness or arterial gas embolism. MRI may be helpful in the diagnosis of decompression sickness in the spinal cord. In a small series, 4 out of 5 patients who had clinical spinal cord decompression sickness had high-intensity lesions in the spinal cord on MRI.[17] In the same series,

there were 15 patients who had clinical cerebral decompression sickness, but MRI failed to identify cerebral lesions in any of the patients,[17] even though it can demonstrate cerebral events in some cases. Cerebral profusion studies (SPECT-HMPAO) have not been found to be helpful in the diagnosis of dysbaric illness.[8]

In the setting of dysbaric illness, electroencephalogram (EEG) demonstrates nonspecific abnormalities in some divers.[17] EEG demonstrated nonspecific abnormalities in only one third of the cases.[17] The value of electronystagmography and evoked potential studies is limited.

LONG-TERM NEUROLOGIC CONSEQUENCES OF DYSBARIC ILLNESS

Fortunately, most deep sea divers are young and healthy, and if treated promptly, ideally in the field, achieve a complete recovery from dysbaric illness. In contrast, when there are long delays to treatment or negligent diving profiles, outcomes may not be as good, as supported by the 2005 Divers Alert Network Report, in which only 70% of the divers obtained complete relief at discharge and 29.7% had residual symptoms.[18]

The residual symptoms and signs may be obvious and debilitating, such as spasticity, urinary incontinence, or weakness, or may be mild, such as peripheral paresthesias.

Similarly, divers who survive arterial gas embolism typically make a full recovery. The youth of the divers and the presence of a healthy circulatory and neurologic symptom likely contribute to their favorable prognosis. The most common residual symptoms reported by divers after arterial gas embolism are difficulties in short-term memory and concentration.[8]

The question of cumulative neurologic damage from asymptomatic diving is less clear. A multivariant analysis demonstrated that divers had significantly more neurologic symptoms and signs than non-divers,[19] with the divers more likely to complain of difficulties with concentration and long- and short-term memory. The most prominent abnormal finding was distal spinal cord and nerve root dysfunction.[19] The occurence of symptoms and abnormal findings was higher in divers who have had a previous recognized decompression sickness episode.

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