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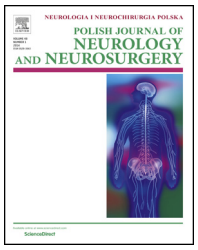

Review article

Neurological complications of underwater diving

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ABSTRACT

The diver's nervous system is extremely sensitive to high ambient pressure, which is the sum of atmospheric and hydrostatic pressure. Neurological complications associated with diving are a difficult diagnostic and therapeutic challenge. They occur in both commercial and recreational diving and are connected with increasing interest in the sport of diving. Hence it is very important to know the possible complications associated with this kind of sport. Complications of the nervous system may result from decompression sickness, pulmonary barotrauma associated with cerebral arterial air embolism (AGE), otic and sinus barotrauma, high pressure neurological syndrome (HPNS) and undesirable effect of gases used for breathing. The purpose of this review is to discuss the range of neurological symptoms that can occur during diving accidents and also the role of patent foramen ovale (PFO) and internal carotid artery (ICA) dissection in pathogenesis of stroke in divers.

1. Introduction

During diving, hydrostatic pressure is exerted on the diver's body in addition to the atmospheric pressure. According to *Dalton's law*, in the course of descent the increase of the ambient pressure also causes an increase of the partial pressures of individual gases in the breathing gas mixture. This contributes to the increased dissolution and transport of gases, mainly nitrogen, in the tissues until saturation in proportion to the ambient pressure, as *Henry's law* states [1,2]. During ascent, when the ambient pressure decreases, the excess gas from the tissue is removed with exhaust air. When the change of the ambient pressure occurs more quickly than removal of nitrogen from the tissues, it may lead to the supersaturation of an inert gas, which is the main cause of the development of decompression sickness. Barotrauma results from the *Boyle–Mariotte law*, according to which gas in the air spaces in the diver's body expands and increases in volume proportionally to the decrease of the surrounding pressure [1,2]. The nervous system is particularly sensitive to the effects of high ambient pressure. The last two decades have seen the increasing popularity of recreational diving, but disregard for basic safety principles resulting from knowledge of diving physiology lead to an increasing number of accidents associated with this activity. Complications of the nervous system associated with diving can result in decompression sickness, pulmonary barotrauma-induced cerebral arterial gas embolism (AGE), ear and sinus barotrauma, high pressure neurological syndrome (HPNS) and the undesired effect of breathing gas in the form of nitrogen narcosis or oxygen toxicity. Differentiation of arterial thrombotic events associated with pulmonary

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barotrauma from decompression sickness is not always possible, and therefore there is frequent reference to the common concept of decompression illness (DCI) [3]. These two are often present together in the same patient. In addition, the differentiation is of little clinical importance because the treatment of both conditions is essentially the same.

2. Neurologic decompression sickness

Neurologic decompression sickness is one of the most serious complications of diving, resulting from the release of inert gas bubbles, usually nitrogen, into the bloodstream and tissues after reduction of ambient pressure. The propensity for the formation of nitrogen bubbles depends on the depth of the dives, the residence time at a given depth and the rate of ascent [4]. Nitrogen bubbles can damage the nervous system as a result of activation of the systemic inflammatory response, involving the cytokines and complement system as well as mechanical disruption or compression of the brain, spinal cord, cranial and peripheral nerves and vascular occlusion, especially large veins [3,5]. White matter of the spinal cord is particularly sensitive to nitrogen bubbles, due to the high solubility of the inert gas in the lipid-rich myelin and relatively poor blood flow in this tissue [6]. Neurologic decompression sickness is more likely to occur in less experienced divers [7]. During descents the diver is exposed to elevated environmental pressure and an increased amount of inert gas dissolved in the body tissue until saturation proportional to the ambient pressure. The desaturation occurs up to several hours after the dive and the presence of gas microbubbles is usually asymptomatic. Repeated exposures contribute to accumulation of these gas bubbles and the emergence of symptoms of decompression sickness. Risk factors of decompression sickness include breaking the principles of decompression, frequent ascents and descents during a single dive (yo-yo diving), repetitive diving in a single day, or plane flights or mountaineering trips within a short time after the dive associated with a reduction in ambient pressure and nitrogen supersaturation of the tissue. Patent foramen ovale (PFO), the persistence of an embryonic defect in the interatrial septum, is present in about 26% of the general population. In patients who have cryptogenic stroke the prevalence of PFO increases to about 40% [8,9]. Patent foramen ovale is one of the risk factors of neurologic decompression sickness, most likely in the mechanism of paradoxical nitrogen emboli, especially in the case of compliance with the proper diving profile [10]. The important role of cardiac right-to-left shunt for paradoxical gas embolism in divers was indicated in 1986 [11]. Approximately 75% of divers who had experienced neurologic symptoms were found to have PFO [12]. Neurological complications and ischemic lesions in the brain assessed by MRI are significantly more frequent among divers with PFO [13]. They also have almost fivefold higher risk of DCI and twice as many ischemic brain lesions than those without PFO. The risk of DCI increases with the size of the leak [13,14] and presence of other factors such as lung diseases, smoking and lung shunts. In patients with PFO the Valsalva maneuver performed during the dive results in increased pressure in the right atrium. Then venous blood containing air bubbles that are produced during the ascent flows directly to the arterial circulation without the specific security filter provided by the pulmonary vessels.

The classification proposed by Golding in 1960 includes two types of decompression sickness. In type I the symptoms are usually mild and may manifest as malaise or fatigue, or may be more specific, involving the skin, joints and muscles. This less severe type of decompression sickness is called *the bends* [5]. Type II decompression sickness is more severe and can affect the lungs, and both the vestibular and the nervous system. Neurologic symptoms of decompression sickness are associated with both spinal cord and rarely cerebral injury. Spinal cord decompression sickness constitutes 50–60% of cases and it involves obstruction of both venous drainage, especially in the epidural vertebral internal venous plexus (Batson venous plexus) [15], and arterial circulation with a predilection to the area supplied by the artery of Adamkiewicz. Changes are mainly located in the lower part of the thoracic spinal cord and lesions of the lumbar or cervical part are rather uncommon. The most severe presentation is partial myelopathy of the thoracic segments [5,16] manifested by rapidly progressive weakness of the lower limbs, paresthesias, dysesthesias and sensory loss in the trunk and lower extremities, pain in the lower back or pelvis and impaired function of the bladder and intestines [4]. Spinal cord decompression sickness may result in specific residual symptoms in the form of sensation loss or pain in the lower back. There are few reports of neurologic decompression sickness manifested as Brown–Séquard syndrome [17,18]. Divers with cervical and thoracic spinal canal stenosis, mostly caused by disk degeneration, are at increased risk for the occurrence of spinal cord decompression sickness [19]. Cerebral decompression sickness constitutes 30–40% of cases and most commonly it involves the arterial circulation [15]. In 90% of cases it occurs within six hours of ascent and more than 50% of patients develop symptoms within one hour of ascent [3,5,20]. Cerebral symptoms occur alone or in combination with spinal decompression sickness [4]. The symptoms of brain damage depend on the location of the emboli. They are often preceded by severe headache associated with increased intracranial pressure, cerebral edema and congestion of blood in the venous sinuses. Perfusion disorders in the area supplied by the anterior and middle cerebral arteries are the most frequent and in consequence multiple ischemic lesions in the frontal and parietal lobes occur. Behavioral and cognitive impairment as a result of cerebral decompression sickness may be persistent or slow to improve.

In decompression sickness the peripheral nervous system is involved rarely and usually the nerves passing through the anatomical spaces where gas bubbles can cause direct mechanical pressure such as the facial, median or trigeminal nerve are affected [21].

The diagnosis of neurologic decompression sickness is based on clinical signs and symptoms and should be suspected in any person with neurological symptoms and a recent history of diving. Neuroimaging studies may serve to confirm the diagnosis; however, implementation of treatment should not be delayed. Magnetic resonance imaging (MRI) is often normal but in 30–55% of cases reveals high-signal lesions on T2-weighted images of the brain and spinal cord which may suggest ischemia, edema and swelling [5,22,23]. The MRI may show an extensive high signal within the central gray matter

of the spinal cord or a patchy high signal on T2-weighted images as well as diffusion-weighted images (DWI) in the dorsal column white matter of the spinal cord. Due to the divergence between the MRI results and the clinical course of the disease, the decision on hyperbaric oxygen treatment should not rely primarily on MRI findings [24]. Neuron-specific enolase (NSE) is a brain-origin protein commonly used to assess the presence and severity of neurological injury. Early measurement of NSE was found to be useful for the diagnosis of neurological DCS with a high specificity. However, its clinical purpose and prognostic value remain to be determined [25].

3. Pulmonary barotrauma

The most severe form of barotrauma is the pulmonary one which occurs during ascent. The most common cause of pulmonary barotrauma among recreational divers is breath-holding ascent [4] or an uncontrolled ascent due to buoyancy equipment failure or loss of consciousness [26]. The pathogenesis of barotrauma results from *Boyle's and Mariotte's law*. According to these, during ascent gas inside the lungs will expand in volume in proportion to the decrease in the ambient pressure. In the case of keeping the air in the airways, the lungs suddenly distend, mechanical strength of the pulmonary parenchyma exceeds the limits and the walls of the alveoli and capillaries in them crack [27]. It may cause pneumothorax, mediastinal and subcutaneous emphysema. The entry of gas bubbles into the vascular system, especially cerebrovascular and rarely coronary, causes symptoms of arterial gas embolism (AGE), which is the most dangerous form of pulmonary barotrauma. The predilection for cerebrovascular complications results from the diving rules according to which the diver usually ascends toward the surface in a head-up position and bubbles located in the blood move upwards. Pulmonary barotrauma and AGE are the cause of almost 25% of all deaths among recreational divers [28]. Pulmonary barotrauma occurs mostly in shallow dives, usually at a depth of up to 10 m, and the dive time is irrelevant. Infection of the lower respiratory tract, pulmonary obstructive disease such as bronchial asthma, bronchiectasis, lung cysts or habitual smoking may predispose to barotrauma. These diseases can lead to airway obstruction by mucous membranes forming a trap for the air retained during ascent, which expands, leading to pulmonary barotrauma. For this reason it is believed that diving can be practiced safely only about a month after bronchitis or pneumonia. The first symptoms such as cough, hemoptysis, sudden, sharp chest pain, shortness of breath, or cyanosis usually occur within 30 min of ascent, most commonly immediately after leaving the water and due to damage of the pulmonary parenchyma. Almost two-thirds of patients with AGE manifest impaired consciousness. Seizures, headache, vertigo and dizziness, as well as focal neurological symptoms such as hemiparesis, quadriparesis or cortical blindness, may also occur [29]. Magnetic resonance imaging may demonstrate multifocal lesions in the brain, with a predilection for frontal and parietal lobes [30]. Arterial cerebral embolism can mimic decompression sickness and the distinction is often difficult. However, spinal cord injury in this mechanism is uncommon.

4. Nitrogen narcosis

Nitrogen narcosis, also known as *rapture of the deep*, occurs during breathing compressed air or a nitrogen–oxygen mixture with the increase in the partial pressure of nitrogen dissolved in the blood from a depth of about 30 m during exposure to pressure of air higher than 3 bar. The origin and mechanism of nitrogen narcosis is not clear-cut. One needs to take into account the interaction of gas molecules with the lipid bilayer of the cellular membrane of nerve endings which leads to increased volume of the hydrophobic zone and hence the disorder of nerve transmission as well as competitive binding to modulation zones of the NMDA (N-methyl-D-aspartate) neurotransmitter receptor [31]. The nitrogen and inert gas theory suggest that there is a parallel between the affinity of narcotic or anesthetic gas for lipid and its narcotic potency. The narcotic effect is consistently greater for gases with higher lipid solubility. The brain areas most affected are the cerebral cortex, the descending reticular formation and the basal ganglia, especially the nigrostriatal pathway, resulting in significant reduction of striatal dopamine and glutamate levels [32]. The occurrence and severity of nitrogen narcosis vary between individuals [33]. In one study symptoms of nitrogen narcosis were observed in 12.1% of recreational divers [34]. Predisposing factors of nitrogen narcosis include physical effort, stress, hypothermia, fatigue, alcohol consumption and limited visibility underwater, as well as carbon dioxide retention, which has a high narcotic potential and also causes increased brain blood flow, increasing the effects of other gases. Symptoms of nitrogen narcosis resemble alcohol intoxication and intensify with increasing depth. It is known as ''*Martini's law*'', the idea that narcosis results in the feeling of drinking 100 ml of Martini, with every 15 m below 30 m depth acting as an additional 100 ml Martini.

The main symptoms of nitrogen narcosis include reduced cognitive performance, temporo-spatial disorientation, memory impairment, euphoria, hallucinations, mood and behavior changes, impaired neuromuscular coordination as well as dizziness, visual effects such as tunnel vision, auditory disorders and significant reduction of pain intensity perception [35]. The most dangerous aspects of nitrogen narcosis are the impairment of judgment and perception or excessive self-confidence and the loss of decision-making ability. The emotional responses become inadequate to the situation. In addition, there are difficulties in performing simple arithmetic tasks, writing becomes rambling and unclear, and speech becomes confused. Impaired performance of acts of precision is due to exaggerated movements. Paresthesia of lips, legs and feet and a characteristic stone face may also occur.

5. High pressure neurological syndrome

High pressure neurological syndrome (HPNS) is a condition of generalized central nervous system stimulation associated with exposure to high pressure in diving beyond a depth of

100 m. This syndrome is encountered only in commercial/technical or professional divers where a helium–oxygen breathing mixture (heliox) is used at depths over 100 m. The most important factors in the pathogenesis of HPNS are the excessive ambient pressure and the effect of helium on the lipid membrane of nerves. Due to the similarity of HPNS symptoms to serotonin syndrome, it seems likely that abnormal serotonin neurotransmission also has pathogenic significance. Manifestations of HPNS include headache, nausea, dizziness, tremor of the arms with a frequency of 8–12 Hz (also called helium tremor), myoclonus, opsoclonus, movement coordination impairment especially dysmetria, moderate cognitive impairment and neuropsychiatric disturbances. The symptoms worsen within increasing depth. It may even lead to partial or generalized seizures. In electroencephalography the reduction of high frequency activity (alpha and beta waves) and presence of delta waves were observed. The symptoms of HPNS are usually reversible after surfacing but some of them such as memory impairment may linger on for long periods [36].

6. Other neurological symptoms related to diving

The use in recreational diving of a breathing mixture with oxygen-enriched air (i.e., nitrox) can result in oxygen toxicity. Nitrox is a breathing mixture that contains more than 21% oxygen and allows the residence time under water to be extended [4]. The toxic effect of oxygen on the nervous system is mainly related to free radical formation and reduced levels of the inhibitory neurotransmitter gamma-aminobutyric acid (GABA). Oxygen inactivates some membrane active transport systems, especially glutamate, which in conditions of hyperoxia is transported out of the cell. Inactivation of membrane transport of brain cells can lead to extracellular accumulation of potassium and glutamate, which can result in increased excitability of neurons and consequently convulsions, because K^+ ions are depolarization factors and glutamate is an excitatory neurotransmitter [37]. Symptoms develop suddenly at depth without warning and include headache, vertigo, nausea and vomiting, paresthesias, facial muscle spasms (in approximately 50–60% of patients), tunnel vision and focal seizures. In 5–10% of patients generalized tonic–clonic seizures or syncope may occur [38]. Depending on the time of exposure and the partial pressure of inspired oxygen, two forms of oxygen poisoning can develop. The acute one involving the brain is called the *Paul Bert effect* and the chronic one involving lungs is known as the *Lorrain Smith effect*. The course of oxygen poisoning depends on the individual predisposition of a diver.

Middle ear barotrauma, which is the most common type of diving complication, occurs mainly during the descent and is associated with insufficient pressure equalization in the tympanic cavity which can be obtained by the Valsalva maneuver (exhaling against a closed nose and mouth), Toynbee maneuver (swallowing saliva with closed nostrils) and Frenzel maneuver (closing the nose and mouth and driving the tongue backwards on the roof of the mouth). The main predisposing factor for this type of injury is obstruction of the Eustachian tube. Symptoms of middle ear barotrauma include sudden pain, vertigo, headache, nausea, vomiting and weakness or conductive hearing loss that lateralizes to the affected side during Weber's test. In severe cases, usually during ascent, increased pressure in the middle ear can cause reversible paralysis of the facial nerve [4] as a result of ischemic neurapraxia. A predisposing factor may be a congenital defect in the canal of the facial nerve [39,40].

Neurological symptoms such as headache and paresthesia in the area innervated by the infraorbital nerve, which is a branch of the trigeminal nerve, may be secondary to paranasal sinus barotrauma, especially frontal or maxillary [41,42]. Risk factors of sinus barotrauma include inflammation of the mucous membrane, nasal or sinus polyps, significant nasal septum curvature and nasal concha hypertrophy. Rare neurological consequences of sinus barotrauma and developing sinusitis are meningitis and brain empyema [43,44]. During the ascent the expanding air can cause damage to the bone wall of the sinus and enter the cranial cavity, resulting in an intracranial edema. This situation often results from sphenoid or ethmoid sinus barotrauma [45,46].

Immersion in extremely cold water can provoke trigeminal neuralgia as well as a tension type headache which can be associated with a contraction of cervical muscles, which lack flexibility, increasing the vulnerability of the neck structures [47].

Long-term occupational diving leads to increased intima-media thickness – a risk factor of cerebrovascular as well as cardiovascular disorders in scuba divers [48].

Internal carotid artery (ICA) dissection is an uncommon mechanism of stroke among divers [49]. ICA dissection can be caused by direct neck trauma as well as hyperextension of the cervical spine or excessive rotation and bending of the head causing mechanical tension of the carotid artery on the bony structures [50], also complicated by the necessity to bear the weight of diving gear and the increasing pressure to which the body is exposed during the descent [51]. ICA dissection results in focal cerebral ischemia within the area supplied by the artery, then the most common symptoms of ICA dissection are hemiparesis, dysphasia and loss of vision. Compression of the neurovascular structures within the neck may result in an ipsilateral pain of the head, face or neck, Horner's syndrome, cranial nerve palsies of the glossopharyngeal, vagus, accessory and hypoglossal nerve and throbbing tinnitus. Dissection of the arteries should be taken into account in the differential diagnosis when neurologic symptoms appear during or after scuba diving, including when the patient does not suffer from headaches [52].

7. Management of complications associated with diving

Management of suspected or confirmed cases of decompression sickness or pulmonary barotrauma includes the fastest transport to the nearest pressure chamber, as the only definitive treatment is recompression and slow decompression. This procedure on the one hand ensures reduction of the diameter of the gas bubbles forming the embolism, allowing them to pass through the capillaries, and on the other hand due to breathing in the atmosphere of increased partial

pressure of oxygen improves oxygenation of ischemic tissues due to congestion [53]. A recovery position with the lower limbs raised above the head is aimed at moving potential gas bubbles to the lower part of the body [27]. 100% oxygen can also be given, to be breathed via a tight-fitting mask. Symptomatic treatment should also be provided. Although PFO might be present in approximately one quarter of divers, DCI in recreational divers is extremely rare, especially after dives to less than 20 m, occurring after only 0.00–0.08% of dives, and the risk of a DCI event correlated with PFO is estimated to be between 0.02 and 0.03% of dives [54]. A screening test of all divers for presence of PFO still seems to be controversial because of the low complete risk of neurological DCI, the unclear relationship between PFO and DCI, and the cost of screening investigations [55], and therefore guidelines are difficult to create. Recommendations for screening for PFO in divers should be based on the absolute risk of decompression illness. Evidence indicates that screening for PFO in average recreational sport divers is not required and the diagnosis of PFO is not a contraindication for diving. Safe diving practice should be performed to reduce the venous bubble. This can be achieved by avoiding diving that requires decompression stops and deeper than 15 m, by limiting bottom time, by staying well hydrated while diving, or by the appropriate use of oxygen-enriched breathing mixes. The Undersea and Hyperbaric Medical Society Best Practice Guidelines [56] state that PFO testing may be considered after severe or repeated neurological DCS, and reduction of decompression stress in future diving activities by more conservative diving practice is probably a better approach than PFO closure.

The Health and Safety Executive specify that examination for the presence of an intracardiac shunt is not a requirement of either the initial or the annual examination in commercial divers. However, examination for PFO should be performed in divers who have suffered neurological, cutaneous or cardiorespiratory decompression illness, especially where there is a history of migraine with aura or where the dive profile was not obviously contributory. A positive finding is not a definite reason to stop diving. However, the opinion of a cardiologist with an interest in diving medicine is recommended [57].

Ischemic stroke related to dissection may be a result of thromboembolism or hemodynamic compromise, although the former seems to be the main mechanism. According to the current American Heart Association/American Stroke Association Guidelines for management of patients with ICA dissection with stroke or transient ischemic attack, antithrombotic treatment for at least 3–6 months is reasonable. In cases of recurrent cerebral ischemic events despite optimal medical treatment, endovascular therapy (stents) may be considered [58].

Management of nitrogen narcosis symptoms includes protection from injury and unreasonable behavior as well as controlled ascent. This results in complete resolution of symptoms. The only consequence may be retrograde amnesia [33]. Prevention of nitrogen narcosis during diving at great depths includes replacing nitrogen with helium and reducing the depth of the dive with air to 50 m. Systematic depth training cause adjustment and less severe symptoms of the disease. Adding hydrogen, nitrogen or nitrous oxide to the breathing mixture reduces the symptoms of HPNS. An interesting solution for prevention of HPNS may be use of 5-HT1A receptor antagonists [59].

Treatment of oxygen toxicity involves reducing the exposure to oxygen, replacing it with air and reducing the depth of the dive. To be allowed to dive in mixed apparatus or using 100% oxygen, a positive oxygen tolerance test is applied which consists of 30 min of breathing at rest with oxygen at a pressure of 2.8 atm in the pressure chamber.

Middle ear barotrauma requires otolaryngological consultation and specialist treatment that involves the use of decongestants (pseudoephedrine, oxymetazoline), nasal and oral antihistamines, analgesics and antibiotics – amoxicillin with clavulanic acid at a dose of 500/125 mg three times daily or clindamycin 300 mg three times daily for 10–14 days in patients with perforation and leakage from the ear [3,60,61]. In the case of facial nerve paralysis paracentesis is used to equalize pressure in the middle ear [62] or oral glucocorticoids are given [39].

8. Summary

Knowledge of diving physiology, diver's health and fitness and diving equipment technical condition are prerequisites for avoiding accidents and diseases associated with diving. Serious complications are diving decompression sickness and pulmonary barotrauma, which is often the result of other diving accidents in the course of which there is a loss of consciousness or sudden change in depth of the dive. Diving-related accidents, and particularly their neurological complications, may present diagnostic and therapeutic problems and therefore the growing number of people engaged in this kind of physical activity is important for understanding the potential complications of the nervous system.

Conflict of interest

None declared.

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Ethics

The work described in this article has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for experiments involving humans; Uniform Requirements for manuscripts submitted to Biomedical journals.

REFERENCES

[1] Brylske A. The gas laws. A guide for the mathematically challenged. Dive Train 1997;(September):26–34.

[2] Dutka AJ, Francis TJ. Pathophysiology of decompression sickness. In: Bove AA, editor. Bove and Davis' diving medicine. 3rd ed. Philadelphia: W.B. Saunders; 1997.
[3] Moon RE. Treatment of diving emergencies. Crit Care Clin 1999;15:429–56.
[4] Newton HB. Neurologic complication of scuba diving. Am Fam Physician 2001;63:2211–8.
[5] Greer HD, Massey EW. Neurologic injury from undersea diving. Neurol Clin 1992;10:1031–45.
[6] Barratt DM, Harch PG, Van Meter K. Decompression illness in divers: a review of the literature. Neurologist 2002;8:186–202.
[7] Newton HB, Padilla W, Burkart J, Pearl DK. Neurological manifestations of decompression illness in recreational divers – the Cozumel, experience. Undersea Hyperb Med 2007;34:349–57.
[8] Sacco RL, Ellenberg JH, Mohr JP, Tatemichi TK, Hier DB, Price TR, et al. Infarcts of undetermined cause: the NINCDS Stroke Data Bank. Ann Neurol 1989;25:382–90.
[9] Meier B, Lock JE. Contemporary management of patent foramen ovale. Circulation 2003;107:5–9.
[10] Moon RE, Camporesi EM, Kisslo JA. Patent foramen ovale and decompression sickness in divers. Lancet 1989;1:513–4.
[11] Wilmshurst PT, Ellis BG, Jenkins BS. Paradoxical gas embolism in a scuba diver with an atrial septal defect. Br Med J 1986;293:1277.
[12] Germonpré P, Dendale P, Unger P, Balestra C. Patent foramen ovale and decompression sickness in sports divers. J Appl Physiol 1998;84:1622–6.
[13] Schwerzmann M, Seiler C, Lipp E, Guzman R, Lövblad KO, Kraus M, et al. Relation between directly detected patent foramen ovale and ischemic brain lesions in sport divers. Ann Intern Med 2001;134:21–4.
[14] Torti SR, Billinger M, Schwerzmann M, Vogel R, Zbinden R, Windecker S, et al. Risk of decompression illness among 230 divers in relation to the presence and size of patent foramen ovale. Eur Heart J 2004;25:1014–20.
[15] Francis TJ, Pezeshkpour GH, Dutka AJ, Hallenbeck JM, Flynn ET. Is there a role for the autochthonous bubble in the pathogenesis of spinal cord decompression sickness? J Neuropathol Exp Neurol 1988;47:475–87.
[16] Aharon-Peretz J, Adir Y, Gordon CR, Kol S, Gal N, Melamed Y. Spinal cord decompression sickness in sport diving. Arch Neurol 1993;50:753–6.
[17] Kimbro T, Tom T, Neuman T. A case of spinal cord decompression sickness presenting as partial Brown–Sequard syndrome. Neurology 1997;48:1454–6.
[18] Louge P, Gempp E, Hugon M. MRI features of spinal cord decompression sickness presenting as a Brown–Sequard syndrome. Diving Hyperb Med 2012;42:88–91.
[19] Gempp E, Louge P, Lafolie T, Demaistre S, Hugon M, Blatteau JE. Relation between cervical and thoracic spinal canal stenosis and the development of spinal cord decompression sickness in recreational scuba divers. Spinal Cord 2014;52:236–40.
[20] Francis TJ, Pearson RR, Robertson AG, Hodgson M, Dutka AJ, Glynn ET. Central nervous system decompression sickness: latency of 1070 human cases. Undersea Biomed Res 1988;15:403–17.
[21] Isakov AP, Broome JR, Dutka AJ. Acute carpal tunnel syndrome in a diver: evidence of peripheral nervous system involvement in decompression illness. Ann Emerg Med 1996;28:90–3.
[22] Dick AP, Massey EW. Neurologic presentation of decompression sickness and air embolism in sport divers. Neurology 1985;35:667–71.
[23] Levin HS, Goldstein FC, Norcross K, Amparo EG, Guinto FC, Mader JT. Neurobehavioral and magnetic resonance imaging findings in two cases of decompression sickness. Aviat Space Environ Med 1989;60:1204–10.
[24] Gao G, Xie L, Wu D, Sun Q, Yang Y, Guan J, et al. The relevance of magnetic resonance imaging in spinal cord decompression sickness: a survey of seven cases. Undersea Hyperb Med 2014;41:105–9.
[25] Gempp E, Louge P, De Maistre S, Emile L, Blatteau JE. Neuron-specific enolase and S100B protein levels in recreational scuba divers with neurological decompression sickness. Diving Hyperb Med 2014;44:26–9.
[26] Siermontowski P, Kozłowski W, Olszański R, Pedrycz A. Znaczenie modelu doświadczalnego urazu ciśnieniowego płuc w badaniu wypadków nurkowych. Pol Hyperb Res 2012;1:7–26.
[27] Siermontowski P, Kozłowski W. Uraz ciśnieniowy płuc niebezpieczeństwem nurkowań płytkich. In: Olszański R, Skrzyński S, Kłos R, editors. Problemy medycyny i techniki nurkowej. Gdański; 1997. p. 231–6.
[28] Cleney TL, Lassen LF. Recreational scuba diving injuries. Am Fam Physician 1996;53:1761–74.
[29] Leitch DR, Green RD. Pulmonary barotrauma in divers and the treatment of cerebral arterial gas embolism. Aviat Space Environ Med 1986;57:931–8.
[30] Gao GK, Wu D, Yang Y, Yu T, Xue J, Wang X, et al. Cerebral magnetic resonance imaging of compressed air divers in diving accidents. Undersea Hyperb Med 2009;36:33–41.
[31] Rostain JC, Balon N. Recent neurochemical basis of inert gas narcosis and pressure effects. Undersea Hyperb Med 2006;33:197–204.
[32] Bennett PB, Rostain JC. Inert gas narcosis. Bennett and Elliott's physiology and medicine of diving. London: Brubbakk A.O., Neuman T.S.; 2003. p. 300–22.
[33] Bennett PB. Inert gas narcosis. In: Bennett PB, Elliott DH, editors. The physiology and medicine of diving. London: Saunders; 1993. p. 170–93.
[34] Nakayama H, Shibayama M, Yamami N, Togawa S, Takahashi M, Mano Y. Decompression sickness and recreational scuba divers. Emerg Med J 2003;20:332–4.
[35] Kowalski JT, Seidack S, Klein F, Varn A, Röttger S, Kähler W, et al. Does inert gas narcosis have an influence on perception of pain? Undersea Hyperb Med 2012;39:569–76.
[36] Talpalar AE. High pressure neurological syndrome. Rev Neurol 2007;45:631–6.
[37] Konarski M, Olszański R. Biochemiczne aspekty zatrucia tlenem. In: Olszański R, Skrzyński S, Kłos R, editors. Problemy medycyny i techniki nurkowej. Gdański; 1997. p. 216–30.
[38] Clark JM, Thom SR. Toxicity of oxygen, carbon dioxide, and carbon monoxide. In: Bove AA, editor. 3rd ed., Bove and Davis' diving medicine, vol. 10, 3rd ed. Philadelphia: Saunders; 1997. p. 131–45.
[39] Whelan TR. Facial nerve palsy associated with underwater barotrauma. Postgrad Med J 1990;66:465–6.
[40] Hyams AF, Toynton SC, Jaramillo M, Stone LR, Bryson PJ. Facial baroparesis secondary to middle-ear over-pressure: a rare complication of scuba diving. J Laryngol Otol 2004;118:721–3.
[41] Murrison AW, Smith DJ, Francis TJ. Maxillary sinus barotrauma with fifth cranial nerve involvement. J Laryngol Otol 1991;105:217–9.
[42] Butler FK, Bove AA. Infraorbital hypesthesia after maxillary sinus barotrauma. Undersea Hyperb Med 1999;26:257–9.
[43] Buisson P, Darsonval V, Dubin J. Sinus infection and diving. Rev Laryngol Otol Rhinol 1990;111:141–3.
[44] Parell GJ, Becker GD. Neurological consequences of scuba diving with chronic sinusitis. Laryngoscope 2000;110:1358–60.
[45] Goldmann RW. Pneumocephalus as a consequence of barotrauma. JAMA 1986;255:3154–6.

[46] Tryqqvasen G, Briem B, Guomundsson O, Einarsdottir H. Sphenoid sinus barotrauma with intracranial air in sella turcica after diving. Acta Radiol 2006;47:872–4.
[47] Cheshire WP. Headache and facial pain in scuba divers. Curr Pain Headache Rep 2004;8:315–20.
[48] Mehrpour M, Rezaali S, Shams-Hosseini NS. Increased carotid intima-media thickness in scuba divers. Iran J Neurol 2014;13:45–7.
[49] Bartsch T, Palaschewski M, Thilo B, Koch AE, Stingele R, Volkmann J, et al. Internal carotid artery dissection and stroke after SCUBA diving: a case report and review of the literature. J Neurol 2009;256:1916–9.
[50] Yair D, Skurnik MD, Zev Sthoeger MD. Carotid artery dissection after scuba diving. IMAJ 2005;7:406–7.
[51] Hafner F, Gary T, Harald F, Pilger E, Groell R, Brodmann M. Dissection of the internal carotid artery after SCUBA-diving: a case report and review of the literature. Neurologist 2011;17:79–82.
[52] Fukuoka T, Kato Y, Ohe Y, Deguchi I, Maruyama H, Hayashi T, et al. A case of anterior cerebral artery dissection caused by scuba diving. J Stroke Cerebrovasc Dis 2014;23:1982–4.
[53] Doboszyński T, Orłowski T. Podstawy terapii hiperbarycznej. WAM Gdynia; 1977.
[54] Bove AA, Moon RE. Patent foramen ovale – is it important to divers? http://www.diversalertnetwork.org/medical/articles/Patent_Foramen_Ovale_Is_It_ Important_to_Divers.
[55] Vann RD, Butler FK, Mitchell SJ, Moon RE. Decompression illness. Lancet 2010;377:153–64.
[56] UHMS best practice guidelines: prevention and treatment of decompression sickness and arterial gas embolism. http://membership.uhms.org/resource/resmgr/dcsandage_prevandmgt_uhms-fi.pdf.
[57] The medical examination and assessment of divers (MA1) http://www.hse.gov.uk/diving/ma1.pdf.
[58] Furie KL, Kasner SE, Adams RJ. Guidelines for the prevention of stroke in patients with stroke or transient ischemic attack. Stroke 2011;42:227–76.
[59] Jain KK. High-pressure neurological syndrome (HPNS). Acta Neurol Scand 1994;90:45–50.
[60] Melamed Y, Shupak A, Bitterman H. Medical problems associated with underwater diving. N Engl J Med 1992;326:30–5.
[61] Farmer JC. Otological and paranasal sinus problems in diving. In: Bennett PB, Eliott DH, editors. 4th ed., The physiology and medicine of diving, vol. 11, 4th ed. London: Saunders; 1993. p. 267–300.
[62] Kamide D, Matsunobu T, Shiotani A. Facial baroparesis caused by scuba diving. Case Report Otolaryngol 2012;2012:329536.