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Recreational scuba diving, patent foramen ovale and their associated risks

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Summary

Scuba diving has become a popular leisure time activity with distinct risks to health owing to its physical characteristics. Knowledge of the behaviour of any mixture of breathable gases under increased ambient pressure is crucial for safe diving and gives clues as to the pathopyhsiology of compression or decompression related disorders.

Immersion in cold water augments cardiac pre- and afterload due to an increase of intrathoracic blood volume and peripheral vasoconstriction. In very rare cases, the vasoconstrictor response can lead to pulmonary oedema. Immersion of the face in cold water is associated with bradycardia mediated by increased vagal tone. In icy water, the bradycardia can be so pronounced, that syncope results.

For recreational dives, compressed air (ie, 4 parts nitrogen and 1 part oxygen) is the preferred breathing gas. Its use is limited for diving to 40 to 50 m, otherwise nitrogen narcosis ("rapture of the deep") reduces a diver's cognitive function and increases the risk of inadequate reactions. At depths of 60 to 70 m oxygen toxicity impairs respiration and at higher partial pressures also functioning of the central nervous system. The use of special nitrogen-oxygen mixtures ("nitrox", 60% nitrogen and 40% oxygen as the typical example) decreases the probability of nitrogen narcosis and probably bubble formation, at the cost of increased risk of oxygen toxicity.

Most of the health hazards during dives are consequences of changes in gas volume and formation of gas bubbles due to reduction of ambient pressure during a diver's ascent. The term barotrauma encompasses disorders related to over expansion of gas filled body cavities (mainly the lung and the inner ear). Decompression sickness results from the growth of gas nuclei in predominantly fatty tissue. Arterial gas embolism describes the penetration of such gas bubbles into the systemic circulation, either due to pulmonary barotrauma, transpulmonary passage after massive bubble formation ("chokes") or cardiac shunting.

In recreational divers, neurological decompression events comprise 80% of reported cases of major decompression problems, most of the time due to pathological effects of intravascular bubbles. In divers with a history of major neurological decompression symptoms without evident cause, transoesophageal echocardiography must be performed to exclude a patent foramen ovale. If a cardiac right-to-left shunt is present, we advise divers with a history of severe decompression illness to stop diving. If they refuse to do so, it is crucial that they change their diving habits, minimising the amount of nitrogen load on the tissue.

There is ongoing debate about the long term risk of scuba diving. Neuro-imaging studies revealed an increased frequency of ischaemic brain lesions in divers, which do not correlate well with subtle functional neurological deficits in experienced divers. In the light of the high prevalence of venous gas bubbles even after dives in shallow water and the presence of a cardiac right-to-left shunt in a quarter of the population (ie, patent foramen ovale), arterialisation of gas bubbles might be more frequent than usually presumed.

Key words: diving; pathophysiology; decompression illness; patent foramen ovale

Introduction

In 1943 Emile Gagnon and Jacques Cousteau invented a self-contained underwater breathing apparatus (scuba) with a demand valve, sensing the ambient pressure under water and automatically delivering the amount of air a diver needed at a particular depth. Since then, the popularity of scuba diving as a leisure time activity has been and still is rising. Today, the "Schweizer Unterwasser-Sport-Verband" has 12'000 active members, 150 diving clubs and 55 diving schools. Around 50'000 persons are diving on a recreational basis annually in Switzerland, mostly using compressed air for breathing.

Diving itself and the use of any high density

gas impose several physiological stresses on the body which involve different organs at different stages of a dive. In order to understand most of the morbidity and mortality related to the underwater environment, a brief introduction into its most relevant physical principles will be presented. Thereafter, this review addresses the major medical health problems arising from scuba diving, in the order in which they occur during a dive. Special attention is paid to the occurrence of decompression disorders. The influence of a cardiac right-to-left shunt (mainly across a patent foramen ovale) on decompression events and possibilities for the secondary prevention of such accidents will be discussed.

Diving physics

Water has a higher density (ρ_{water} = 998 kgm^{-3}) and viscosity (η_{water} = 1 × 10^{-3} Nsm^{-2}) than air (at sea level: ρ_{air} = 1.29 kgm^{-3}, η_{air} = 1.82 × 10^{-5} Nsm^{-2}). A change in depth of 10 m in water corresponds to a change in hydrostatic pressure of 1 atmosphere (1 atm = 101 kPa = 1.01 bar), thus at a depth of 30 m of seawater, the ambient pressure has risen to 4 atm (3 atm of hydrostatic pressure and 1 atm atmospheric pressure). This rise in pressure also applies to any breathing gas used by a diver.

The physical properties of a gas are governed by its temperature, pressure and volume. At a constant temperature, the volume (V_i) of a fixed mass of gas varies inversely with the pressure (P_i) exerted on it (*Boyle's Law*):

$$P_1 \times V_1 = P_2 \times V_2$$

This means that at 10 m under water (P_2: ambient pressure = 2 atm), its volume (V_2) is reduced to one half of the original volume (V_1) at the surface (P_1: ambient pressure = 1 atm). Note that the change in volume of a given volume, for example 5 l of gas at the surface between 20 m and 10 m under water is much less than the change between 10 m and the surface during a diver's ascent (ΔV = 0.8 l vs. 2.5 l). Therefore, sudden depth changes in shallow water lead to larger changes in gas volume than equivalent depth changes in deep water.

Dry air is a mixture of roughly 4 parts of nitrogen and 1 part of oxygen. According to *Dalton's law*, the partial pressure ($P_{partial}$) exerted by each single gas of a mixture corresponds to its percentage in volume:

$$P_{total} = \sum Pi_{partial}$$

The partial pressure of oxygen, when using compressed air for diving at 30 m under water can be calculated as $P_{partial}$ = 20% × 4 atm = 0.8 atm or 80 kPa. The amount of gas (C) dissolved in a liquid is directly proportional to the partial pressure of the gas (*Henry's law*):

$$C = P_{partial} \times \alpha$$

The constant of solubility (α) for a specific gas and a specific liquid is influenced by temperature: the lower the temperature, the higher the solubility (open a warm versus a cold bottle of carbonated beverage). The solubility of a gas and its diffusion in the tissue are crucial for the occurrence of decompression events and inert gas narcosis.

Being in a medium with low visibility in addition to the distortion of the optical information due to refraction of light at the face mask can severely impair a diver's performance. In Switzerland with its cold lakes, loss of body heat due to convection (water has a 30 times higher degree of heat conductivity than air) and evaporation is a further severe physiological stress to the body, affecting not only muscular, but also the central nervous system function [1].

Immersion in cold water

Immersion in water at a thermoneutral level rapidly augments cardiac preload due to redistribution of blood from the legs to the core of the body. Intrathoracic blood volume increases by up to 500 ml, resulting in a 30% increase in cardiac output and stroke volume. Right atrial pressure rises up to 16 mm Hg [2, 3]. The increase in central blood volume stimulates diuresis (immersion diuresis and natriuresis) mediated by a reduction of antidiuretic hormone and an increase in circulating atrial natriuretic factor [4]. Cold water reinforces the venous redistribution due to immersion by an additional vasoconstrictor effect. The cold, as a strong peripheral vasoconstrictor, augments cardiac afterload. Arteriolar vasoconstriction increases systemic resistance, blood pressure and left ventricular wall stress [5]. In a few otherwise healthy persons, this vasoconstrictor response can even lead to pulmonary oedema [6, 7]. Interestingly, more than half of them develop arterial hypertension in later life [6], probably reflecting a predisposition for increased vasomotor reactivity.

Immersion in cold water is also accompanied by vagal-tone mediated bradycardia [5, 8]. In diving animals, this reflex phenomenon drastically reduces cardiac output and oxygen consumption and serves to extend the duration of breath-hold dives [9]. In humans, cardiac output decreases only slightly during immersion in cold water [5]. The negative influences of bradycardia and the increase

in afterload on cardiac output are nearly compensated for by the increase in stroke volume due to a higher preload. The degree of bradycardia is dependent on the water temperature: the colder the water, the profounder the bradycardia [10]. In ice water, bradycardia on initial immersion of the face can be so pronounced, that syncope with drowning is possible. In divers with known coronary heart disease, slow sinus bradycardia due to cold water immersion can degenerate into ventricular tachycardia or fibrillation [11]. Tachyarryhthmias (atrial fibrillation with slow ventricular response, ventricular tachycardia or ventricular fibrillation) are mainly observed in healthy divers in the setting of severe hypothermia with a core temperature below 30 ºC [12]. The cardioinhibitory effect of cold water can be used to slow down and convert paroxysmal supraventricular tachycardia [13].

Oxygen toxicity and inert gas narcosis ("rapture of the deep")

Rising ambient pressure during a diver's descent elevates the partial pressure of oxygen. Oxygen toxicity is caused by the production of free radicals which overwhelm the cellular antioxidant defence, resulting in cellular membrane, enzyme and nucleic acid destruction [14, 15]. The respiratory system represents a susceptible tissue for oxygen poisoning. In patients requiring mechanical ventilation, prolonged exposure to hyperoxia causes pathological changes of the lungs, consisting of an acute exudative phase with interstitial and alveolar oedema, merging into a proliferative phase with interstitial fibrosis and hyperplasia of type II alveolar cells [16]. Symptoms of pulmonary oxygen toxicity range from mild substernal discomfort and coughing to dyspnoea at rest. After 12 to 16 hours of exposure to oxygen at 1.0 atm pressure (corresponding to a depth of 40 m when using compressed air) pulmonary symptoms occur in most individuals [17]. Higher partial pressures lead to central nervous system toxicity. Minor symptoms like tinnitus, vertigo and nausea are followed quickly by convulsions [18]. Oxygen convulsions are accelerated by hypercapnia due to cerebral vasodilatation and a higher rate of delivery of oxygen to the brain [19]. Divers are particularly prone to hypercapnia because of the higher density and viscosity of the compressed breathing gas under the increased ambient pressure of the surrounding water. The higher density of the gas is associated with an increase in the breathing resistance, augmenting dead space ventilation and accumulation of carbon dioxide. Furthermore, ventilation can be reduced due to narcotic depression. Therefore, divers should always attempt to keep the partial pressure of oxygen below 1.6 atm or 160 kPa [20]. This prohibits the use of compressed air for dives deeper than 60 to 70 m.

In 1935, Behnke and co-workers described progressive "euphoria, retardment of higher mental processes and impaired neuromuscular coordination" in probands subjected to an ambient pressure of 4 atm while breathing air [21]. They correctly attributed this clinical picture (ie "rapture of the deep"), similar to alcohol intoxication, to the raised partial pressure of nitrogen in the tissue of the nervous system. The narcotic potential of nitrogen is related to its high lipid solubility and its property of interfering physically at synapses or nerve junctions. Current concepts in general anaesthesia suggest a physical effect of the gas on the bilayer permeability of a nerve cell or competing for the binding of neurotransmitters [22]. During dives with compressed air, symptoms may occur at depths deeper than 30 m (ambient pressure 4 atm). At depths deeper than 55 m, a diver's performance is severely impaired. At extreme depths of 90 to 100 m under water, unconsciousness and death results [23], when using compressed air for breathing. Aside from oxygen toxicity described above, nitrogen narcosis further limits the safe use of compressed air to dives not deeper than 40 to 50 m.

The high pressure nervous syndrome, a state of general excitation with marked tremor, dizziness and vomiting, represents the physiological opposite of inert gas narcosis, but is seen only in technical or professional divers, at depths of 150 m or more. In order to reach these depths, nitrogen has to be replaced by helium or neon as breathing gas. As in nitrogen narcosis, a direct effect of the breathing gas (eg, helium) on the lipid membrane of the nervous cell is suspected. Contrary to nitrogen with its high lipid solubility, the less lipid soluble helium might increase the surface tension on the bilayer membrane, resulting in constriction of cell membranes at high ambient pressure with destabilisation and increased excitability of the nervous cells [24].

Decompression related disorders

Most of the medical health problems in recreational scuba diving are consequences of decompression during the ascent. They can be divided according to the physical mechanisms involved (bubble formation from dissolved gas or over-expansion of air-filled cavities with secondary arteriali-

sation of gas bubbles), and according to their clinical relevance with respect to therapy. Because of considerable clinical overlap and common elements in their pathophysiology, the term *decompression illness* is used for describing decompression disorders with bubbles as the initiator. This term encloses *decompression sickness*, resulting from the growth of gas nuclei in predominantly fatty tissue, and *arterial gas embolism* due to gas invasion into the systemic circulation. *Barotrauma* encompasses medical problems particularly related to overexpansion of air-filled spaces (ie, mainly the lung, ear and sinuses, rarely the stomach).

Air embolism due to pulmonary barotrauma ranks second only to drowning as a cause of recreational scuba diving fatalities [25], and is more frequent in novice divers. Unintentional breath-holding, bronchospasm or mucous plugs inhibit deflation of the expanding lungs during ascent. Disruption of the pulmonary parenchyma is a consequence of Boyle's law: due to the decrease in ambient pressure, the gas volume in the lungs proportionally expands. If no expiration of gas takes place, pulmonary parenchyma will over-expand and rupture, with secondary migration of gas into the pulmonary circulation, into the perivascular sheaths, pleural cavity, retroperitoneum or subcutaneous tissues of the neck. Arterial gas embolism, pneumomediastinum or pneumothorax are the common corresponding clinical manifestations. The entrance of air into the arterial system via pulmonary veins and the left chambers of the heart is the most hazardous of these conditions. Most of the time, it occurs at the end of a dive in shallow water, where the rate of gas expansion is greatest and a diver's body is still loaded with a considerable amount of dissolved gas, so that a distinction between arterial gas embolism and decompression sickness may not always be possible.

Cracking joints – pathophysiology of decompression illness

Bubbles are formed in the human body also without changes in ambient pressure. Joints actually do crack when joint surfaces are pulled apart and the flow of liquid into the widening gap is restricted due to its viscosity. The resulting decrease in local pressure leads to the formation of bubbles of water vapour, which audibly crack during normalisation of intra-articular pressure [26]. In recreational divers during their ascent, most bubbles are thought to be formed by expansion of a pre-existing gas phase (*gas nucleus*) [27, 28]. These gas nuclei are found at normal atmospheric pressure in the joints of the limbs and the spine due to gaseous cavitation, as just described. Excessive joint movement before or after a dive increases the risk of decompression illness [28], probably due to the formation of additional intrinsic gas nuclei.

Beside the joints and the spine, bubble formation from gas absorbed into sweat glands and skin pores can be observed only in the superficial layer of the skin [29]. Blood itself seems to be resistant to bubble formation, an observation made already by the grandfather of Charles Darwin [30, 31]. However, tissue bubbles gain access to the capillary or the lymphatic bed by migration. Small volumes of venous gas bubbles are well tolerated and entrapped in and exhaled from the lungs if no cardiac or pulmonary shunt is present (fig. 1). Large amounts of venous gas overload the filter capacity of the lungs and bubbles escape into the arterial

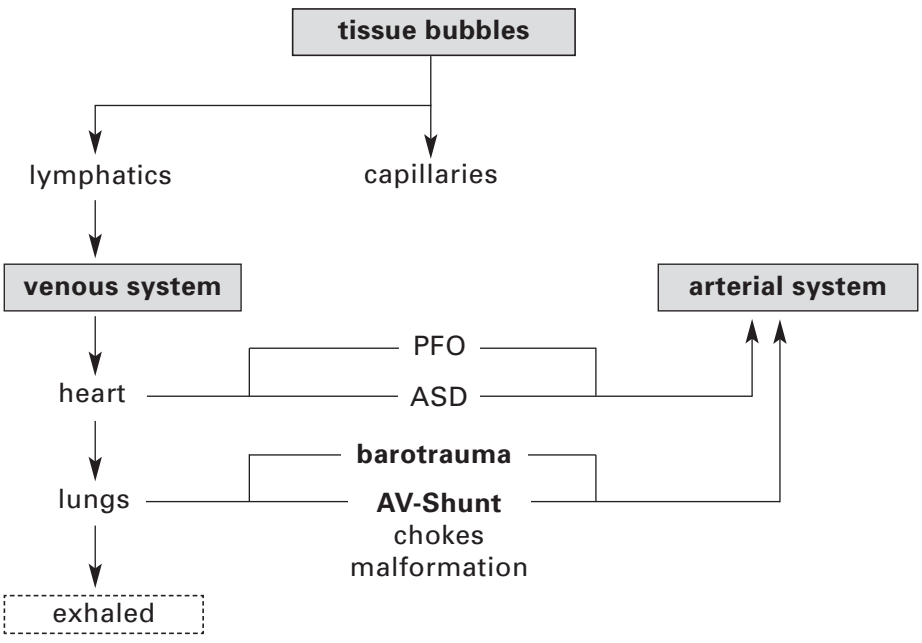


Figure 1

Pathway of bubbles in the body with several possibilities for arterialisation.
Abbreviations:
ASD: atrial septal defect;
AV: arteriovenous;
PFO: patent foramen ovale.

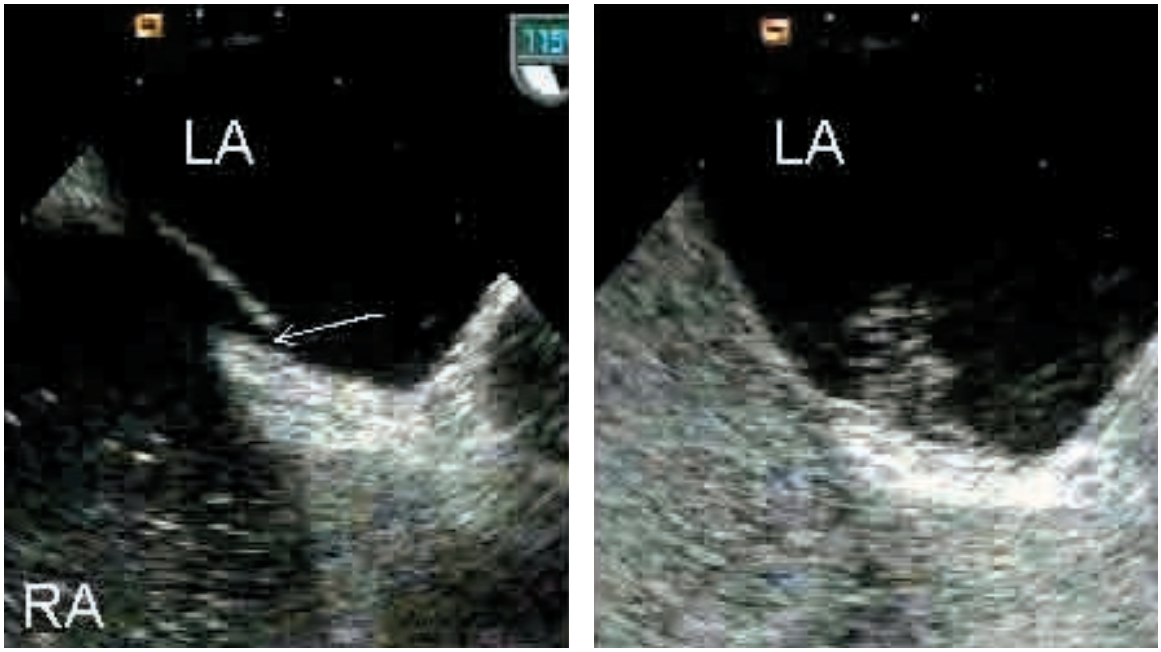


Figure 2

Long axis transoesophageal echocardiographic view in an individual with PFO. Panel A: Interatrial septum with fossa ovalis (thin-walled section between the atria) and region of PFO (arrow). Echocontrast bubbles are just entering the right atrium via superior vena cava (right side of the panel: cranial). Panel B: Identical view as in panel A with the region of the atrium now filled with echocontrast bubbles. A small cloud of bubbles enters the left atrium directly via PFO. The diagnosis of PFO requires crossing of the bubbles within 4 heart beats. Otherwise, appearance of bubbles in the left atrium is considered transpulmonary.
Abbreviations:
LA: left atrium;
PFO: patent foramen ovale;
RA: right atrium.

system [32]. Massive pulmonary air embolism opens arterio-venous shunts in the lungs and raises pulmonary artery pressure, facilitating further transpulmonary passage of bubbles [33].

Of all possibilities for arterialisation of venous gas bubbles, a *patent foramen ovale* is probably the most prevalent pathway. Patent foramen ovale is a persistent opening between the septum primum and secundum at the level of the fossa ovalis. During fetal life, the foramen ovale serves as a flap valve to ensure unidirectional blood flow from the right to the left atrium, bypassing pulmonary circulation. Postnatally, left atrial pressure exceeds right atrial pressure, moving the valve against the limbus of the fossa ovalis, with fibrous sealing of the interatrial channel within the first year of life. Autopsy shows incomplete fusion (ie patent foramen ovale) in 25–30% of human hearts [34]. In 1988, Lechat and co-workers found a relationship between patent foramen ovale in patients under 55 years of age and crypotgenic stroke [35]. Today, contrast transoesophageal echocardiography is part of the assessment of younger patients with crypotgenic stroke because of its superior accuracy for detecting patent foramen ovale compared to other imaging and Doppler methods (fig. 2) [36, 37]. For prevention of stroke recurrence in the presence of a patent foramen ovale, medical therapy with warfarin or antiaggregant medication [38], surgical [39] and percutaneous closure [40] are possible treatment modalities.

Clinical manifestations of decompression illness

The pathological effects of nitrogen bubbles result from their physical presence with mechanical disruption of the tissue concerned (for example nervous tissue [41] or the endothelium [42, 43]), with compression of non-compliant tissue or blood vessels and lymphatics, or from simply obstructing blood vessels. They also evoke a tissue response with activation of platelets and the coagulation cascade [42, 44], complement [45, 46], and leukocyte chemotaxis [47, 48]. The damaged integrity of the endothelium leads to an increase in the haematocrit due to extravasation of plasma volume [49, 50].

These pathological effects are exerted by expanding pre-existing gas nuclei. The rate of growth of these bubbles depends on gas exchange between the bubbles and the surrounding tissue. Blood flow is the primary factor that controls the exchange of dissolved inert gas between blood and tissue. Tissue nitrogen then diffuses into the gas nuclei along a concentration gradient and increases their volume. Tissue perfusion, and the solubility and diffusion coefficients of the inert gases are the major physical determinants of bubble formation beside the rate of reduction of ambient and tissue pressure (ie, the rate of a diver's ascent).

Clinical manifestations of decompression illness are traditionally classified as minor (type I) or major (type II) symptoms [51]. Minor symptoms include musculoskeletal pain ("limb bends") and "skin bends" with rash, pruritus and painless swelling of the skin due to lymphatic obstruction. They usually require no recompression in a pressurised chamber for treatment. By contrast, major decompression illness requires therapeutic recompression and includes neurological and cardiores-

piratory symptoms. It carries the risk of permanent disability or even death.

Musculoskeletal pain is a common symptom of decompression events, typically occurring in the region of the joints of the upper and lower extremities [51]. Limb *bends* occur early after surfacing or even during the ascent. Often, the pain resolves after local pressure to the compromised site [52]. Growing bubbles trapped in the periarticular tissue and tendons are thought to produce pain by compression of nerve endings and induction of a tissue response with release of pain producing inflammatory mediators [29, 47].

Aseptic bone necrosis was first seen in caisson-workers and affects mainly the long bones and juxta-articular regions. Secondary arthritis is a consequence of collapse of the articular surface. Obstruction of nutrient-supplying blood vessels by nitrogen bubbles might be an explanation for the bone necrosis. There is a good correlation between aseptic bone necrosis and either a history of decompression illness or the number of decompressions a diver performed [53]. In recreational divers with short diving times and a limited number of decompressions, bone necrosis is rarely seen.

Large amounts of venous gas emboli reaching the lungs can damage the pulmonary endothelium resulting in dyspnoea, cough and pulmonary oedema *("chokes")* [54]. Retrosternal discomfort is usually the first symptom and can be caused by only 5 ml of air arriving as a bolus [55]. Larger amounts provoke cough and discomfort, leading to right-sided heart failure. Bubble-induced activation of leukocytes leads to an increase in vessel permeability with pulmonary oedema, contributing further to arterialisation of gas bubbles [48].

Neurological decompression illness

In recreational divers, neurological symptoms comprise 80% of reported cases of decompression illness [29]. Bilateral paraesthesia occasionally followed by regional numbness and progressive weakness below a certain spinal level, or impaired bowel or bladder control are symptoms of spinal cord involvement [56]. Unilateral loss of sensory function can be related to local neurapraxia of peripheral nerves [57] or to patchy involvement of the spinal cord during bubble formation. Focal cerebral signs (eg, aphasia, visual disturbances, loss of consciousness, seizures, hemiparesis, hemisensory loss, ataxia, hemineglect) are signs of cerebral decompression illness. In half of the cases, symptoms occur during dives respecting the limits of decompression tables and without identifiable reasons, ie, are *unexpected decompression events* [56]. In an analysis of 1070 cases of central nervous decompression illness, 23% of the cases involved the brain, 66% the spinal cord and 11% both [58].

Current concepts relate cerebral decompression illness primarily to intravascular bubble injury (ie, arterial gas embolism) [53]. Local bubble formation seems unlikely, because of the high cerebral blood flow with effective wash-out of excess inert gas. Results of neuroimaging studies of divers with cerebral decompression illness are consistent with the emboli theory [59, 60]. For spinal decompression illness, Hallenbeck and co-workers favoured epidural venous thrombosis to be responsible [61]. Because of the anatomy of the epidural venous plexus, gas bubbles are thought to accumulate and activate the clotting mechanism, resulting in venous stasis and, ultimately, tissue infarction. Autochthonous bubble formation can further impair spinal cord function by disruption of axons or the myelin sheath and by tissue ischaemia due to compression. Spinal tissue is much more saturated with inert gas than the brain at the end of a dive due to the differences in tissue perfusion, allowing rapid growth of embolic bubbles when they are entrapped in the spinal cord.

Asymptomatic divers

Generally, embolic events occurring in the human body (eg, in bacterial endocarditis, left heart thrombus or fat embolism) far more often cause cerebral than spinal symptoms, which may relate to the corresponding proportion of blood flow. Extending this fact to gas embolism in divers, more cerebral than spinal cord symptoms would be expected. This contrasts with the observed clinical predominance of spinal cord symptoms [58], which is only partially explained by tissue perfusion differences and the resulting decreased wash-out of the spinal nitrogen load. Alternatively, cerebral embolism in divers may frequently be asymptomatic, compared to spinal embolism, which usually results in symptomatic lesions, because of the concentration of neurologically important and eloquent tracts in the spinal cord [62].

Recent neuroradiological studies are consistent with the concept of asymptomatic cerebral embolism in divers. Several investigators observed an increased number of ischaemic brain lesions in asymptomatic divers compared with non-diving controls [63–66]. An investigation in 84 divers with fluorescein angiography of the eye showed patho-

logical changes of the retinal microcirculation independent of a history of decompression illness, indicating central nervous system vasculopathy as a marker for neurovascular injury [67]. Similar results were obtained in a pathoanatomical study [68]. Neurological studies in sport [65] and professional divers [69] indicate subtle functional brain damage even in the absence of a history of decompression events. Correlations between neurological tests and neuroradiological lesions in asymptomatic divers are weak [65], possibly indicating a more complex than simple linear correlation, or simply illustrating the fact, that a pathological change is not always proof of a functional deficit.

Regarding the high prevalence of venous gas emboli even after dives in shallow water [70], a cardiac right-to-left shunt might explain the subtle neuroradiological abnormalities and functional deficit in divers without decompression illness events. In the only study with divers and non-diving controls using cranial MRI and contrast transoesophageal echocardiography, 1.2 and 0.6 ischaemic brain lesions per person were detected in divers with and in divers without a patent foramen ovale, respectively. Among controls, 0.2 and 0.1 lesions per person were detected, respectively ($p < 0.001$ for all groups; fig. 3 and 4). In a regression analysis, diving increased the incidence of one or more ischaemic brain lesion fivefold. Diving itself, even more than diving in the presence of patent foramen ovale was associated with the presence of ischaemic brain lesions [66].

Figure 3

Panel A: The average number of predominantly decompression sickness or arterial gas embolism events per divers (vertical axis, left side) and of decompression sickness events per 10'000 dives (vertical axis, right side) is shown for divers with and without PFO, respectively. Panel B: The average number of ischaemic brain lesions per person depicted by MRI (vertical axis) is shown for the different study groups (horizontal axis). Data from [66], reprinted with the permission from the American College of Physicians – American Society of Internal Medicine.
Abbreviations: PFO: patent foramen ovale.

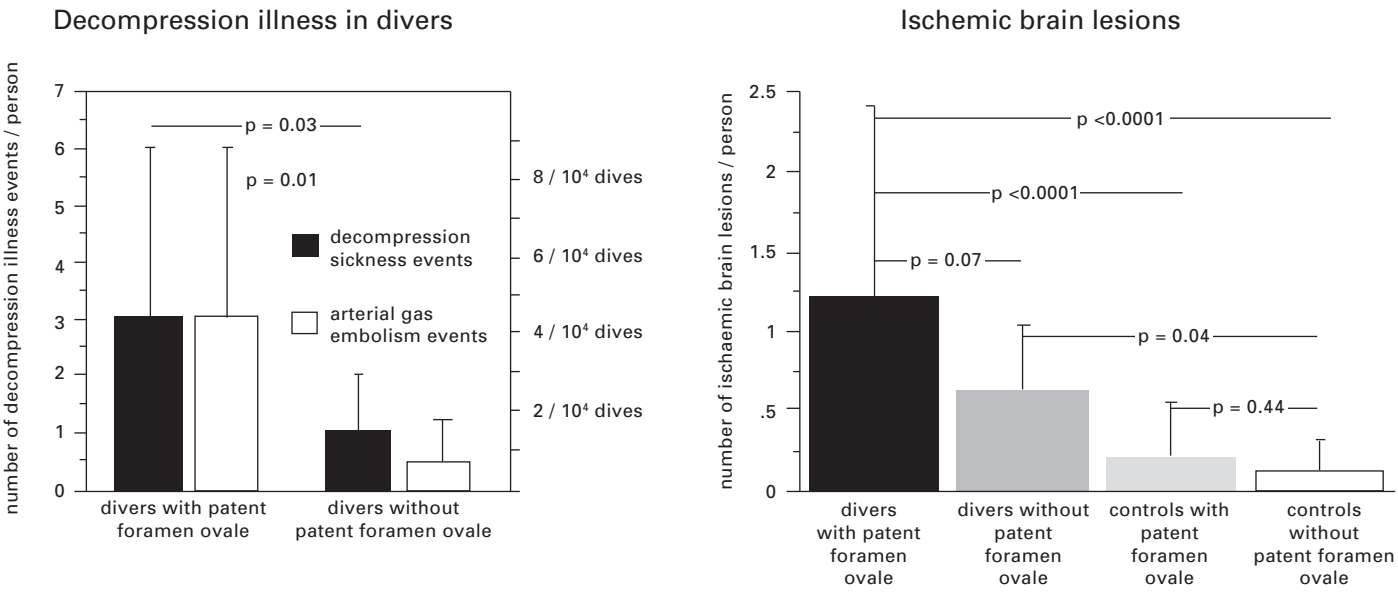
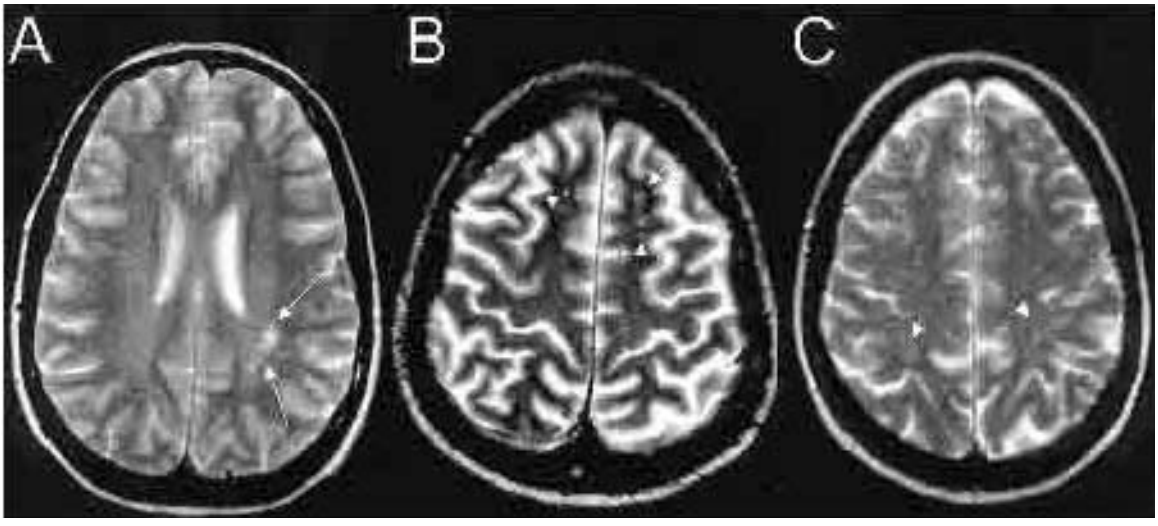


Figure 4

T_2-weighted axial MR images of three divers without symptoms and no history of decompression incidents. Large not well delineated hyperintense lesions corresponding to ischaemic infarcts are seen in the periventricular and subcortial white matter (arrows in panels A, B). Round or linear shaped hyperintensities corresponding to Virchow-Robin-spaces are seen in (B) and (C) (arrowheads).



Patent foramen ovale and decompression illness

Already in 1986, Wilmshurst and associates suggested, that a cardiac right-to-left shunt may be relevant for paradoxical gas embolism among scuba divers [71]. In subsequent studies, the importance of patent foramen ovale for decompression events in sport divers has been repeatedly demonstrated [66, 72–74]. Overall, patent foramen ovale increases the risk for decompression illness 4 to 5 times [66, 75], even in divers strictly adhering to decompression tables. This relationship seems to be stronger for cerebral than for spinal decompression events [66, 74]. However, the absolute frequency of major decompression illness is quite low (fig. 3).

Nevertheless, current examination of divers with a history of neurological decompression illness after a dive within decompression table limits [ie, with unexpected major decompression illness events) should include contrast transoesophageal echocardiography for exclusion of a patent foramen ovale (table 1). For divers with patent foramen ovale and a history of major decompression illness, a paucity of data exists about the risk for future events. Consequently, we advise them to stop diving. If they refuse, they should minimise the load of tissue nitrogen during dives, eg, by refraining from long deep dives, from repeated ascents and descents during the same dive or from diving several times a day (table 1). Some divers try to decrease the amount of nitrogen load by using special nitrogen-oxygen mixtures ("nitrox"), with 40% oxygen and 60% nitrogen as typical example. The reduced nitrogen content compared to air decreases the probability of bubble formation, but 40% oxygen in the breathing gas carries the double risk of oxygen toxicity. These are all but theoretical considerations, and no prospective clinical trial has ever been conducted to establish their utility in daily routine for the prevention of decompression events in the presence of a cardiac right-to-left shunt. Transcatheter closure of patent foramen ovale may reduce the risk for recurrent neurological decompression events [76, 77], but this therapeutic option has not yet been proven to be effective in a randomised clinical trial.

Contrast transoesophageal echocardiography is not mandatory in the routine medical evaluation of asymptomatic sport divers, nor in asymptomatic commercial or professional divers for the primary prevention of decompression illness events. Regarding the lack of clinical data about possibilities for minimising the further risk of decompression events in such divers with right-to-left shunts, they could only be advised to refrain from diving. However, the low individual risk does not justify the exclusion a quarter of the population (ie, with a patent foramen ovale) from diving.

Table 1

Recommendations for recreational divers with a patent foramen ovale.

Asymptomatic diver	routine screening for patent foramen ovale by contrast transoesophageal echocardiography is not mandatory
Diver with a history of unexpected DCI*	contrast transoesophageal echocardiography for exclusion of a cardiac right-to-left shunt is necessary
– with a patent foramen ovale	advise to stop diving otherwise, tissue nitrogen load during dives has to be minimised by: – strict adherance to decompression tables – no repetitive dives during a day – no deep dives (>25–30 m) – reduced rate of ascent during the last 10 m – no Valsalva manoeuvre during ascent, no strenuous physical effort (like carrying heavy diving equipment) shortly after leaving the water – use of nitrox instead of compressed air
– without a patent foramen ovale	consider other possibilities than a patent foramen ovale for arterio-venous shunting
	tissue nitrogen load has to be minimised

* Unexpected decompression illness (DCI) refers to major DCI symptoms after dives within the limits of decompression tables and no evident cause.

Treatment of decompression illness

Treatment for severe decompression sickness and arterial gas embolism is essentially the same. Diving depth and onset of symptoms can give clues to the underlying pathophysiological mechanism. Pulmonary barotrauma and gas embolism can occur during a dive in only a few meters depth, whereas decompression sickness requires a prolonged stay at a certain depth for sufficient loading of tissues with inert gas. Median time of onset for decompression sickness is approximately one hour and for arterial gas embolism 2 minutes after surfacing [78].

Aside from emergency treatment for respiration and circulation, first aid includes administration of fluids, because dehydration and haemoconcentration are common in severe decompression sickness [49, 50, 79]. Administration of 100% oxygen increases oxygen delivery to underperfused areas and secondly, enhances washout of inert gas from the tissue due to the absence of additional inhaled inert gas. Consequently, inert gas diffuses from bubbles to tissue, causing them to shrink. The definitive treatment requires recompression, since increased ambient pressure further reduces the volume of tissue gas. Although bubble volume cannot be reduced to zero, the reduction in volume often leads to reduction in symptoms. For avoiding additional gas loading of tissues during recompression, oxygen is preferably used [79].

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References

1 Coleshaw SR, Van Someren RN, Wolff AH, Davis HM, Keatinge WR. Impaired memory registration and speed of reasoning caused by low body temperature. J Appl Physiol 1983; 55(1 Pt 1):27-31.
2 Hong SK, Cerretelli P, Cruz JC, Rahn H. Mechanics of respiration during submersion in water. J Appl Physiol 1969;27:535-8.
3 Arborelius M, Ballidin UI, Lilja B, Lundgren CE. Hemodynamic changes in man during immersion with the head above water. Aerosp Med 1972;43:592-8.
4 Hong SK. Breath-hold diving. In: Bove AA, editor. Bove and Davis' diving medicine. Philadelphia, PA: WB Saunders; 1997. p. 65-74.
5 Keatinge WR, McIlroy MB, Goldfein A. Cardiovascular responses to ice-cold showers. J Appl Physiol 1964;19:1145-50.
6 Wilmshurst PT, Nuri M, Crowther A, Webb-Peploe MM. Cold-induced pulmonary oedema in scuba divers and swimmers and subsequent development of hypertension. Lancet 1989;1: 62-5.
7 Pons M, Blickenstorfer D, Oechslin E, Hold G, Greminger P, Franzeck UK, et al. Pulmonary oedema in healthy persons during scuba-diving and swimming. Eur Respir J 1995;8:762-7.
8 Brick I. Circulatory responses to immersing the face in water. J Appl Physiol 1966;21:33-6.
9 Irving L, Scholander PF, Grinnel SW. The regulation of arterial blood pressure in the seal during diving. Am J Physiol 1942;135:557-66.
10 Kobayasi S, Ogawa T. Effect of water temperature on bradycardia during nonapneic facial immersion in man. Jpn J Physiol 1973;23:613-24.
11 Wilmshurst P. Cardiovascular problems in divers. Heart 1998; 80:537-8.
12 Mebane GY. Hypothermia. In: Bove AA, editor. Bove and Davis' diving medicine. Philadelphia, PA: WB Saunders; 1997. p. 207-15.
13 Gooden BA. The diving response in clinical medicine. Aviat Space Environ Med 1982;53:273-6.
14 Fridovich I. The biology of oxygen radicals. Science 1978; 201:875-80.
15 Halliwell B, Gutteridge JM. Oxygen toxicity, oxygen radicals, transition metals and disease. Biochem J 1984;219:1-14.
16 Nash G, Blennerhassett JB, Pontoppidan H. Pulmonary lesions associated with oxygen therapy and artifical ventilation. N Engl J Med 1967;276:368-74.
17 Comroe JHJ, Dripps RD, Dumke PR, Deming M. Oxygen toxicity: the effect of inhalation of high concentration of oxygen for twenty-four hours on normal men at sea level and at a simulated altitude of 18'000 feet. JAMA 1945;128:710-7.
18 Donald KW. Oxygen poisoning in man. Br Med J 1947;1:667-72, 712-7.
19 Lambertsen CJ, Ewing JH, Kough RH, et al. Oxygen toxicity. Arterial and internal jugular blood gas composition in man during inhalation of air, 100% O_2 and 2% CO_2 in O_2 at 3.5 atmospheres ambient pressure. J Appl Physiol 1955;8:255-63.
20 Wilmshurst P. Diving and oxygen. BMJ 1998;317:996-9.
21 Behnke AR, Thomson RM, Motley EP. The psychologic effects from breathing air at 4 atmospheres pressure. Am J Phyisol 1935;112:554-8.
22 Franks NP, Lieb WR. Molecular mechanisms of general anaesthesia. Nature 1982;300:487-93.
23 Bennet PB. Inert gas narcosis and high pressure nervous syndrome. In: Bove AA, editor. Bove and Davis' diving medicine. Philadelphia, PA: WB Saunders; 1997. p. 117-30.
24 Moon RE, Vann RD, Bennett PB. The physiology of decompression illness. Sci Am 1995;273:70-7.
25 Russi EW. Diving and the risk of barotrauma. Thorax 1998; 53(Suppl 2):S20-4.
26 Unsworth A, Dowson D, Wright V. 'Cracking joints'. A bioengineering study of cavitation in the metacarpophalangeal joint. Ann Rheum Dis 1971;30:348-58.
27 Weathersby PK, Homer LD, Flynn ET. Homogeneous nucleation of gas bubbles in vivo. J Appl Physiol 1982;53(4):940-6.
28 Vann RD, Thalmann ED. Decompression physiology and practice. In: Bennet PB, Elliott DH, editors. The pyhsiology and medicine of diving. London: WB Saunders; 1993. p. 376-432.
29 Dutka AJ, Francis TJ. Pathophysiology of decompression sickness. In: Bove AA, editor. Bove and Davis' divng medicine. 3rd ed. Philadelphia, PA: WB Saunders; 1997.
30 Darwin E. Experiments on animal fluids in the exhausted receiver. Phil Trans 1774;64:344-9.
31 Ikels KG. Production of gas bubbles in fluids by tribonucleation. J Appl Physiol 1970;28:524-7.
32 Butler BD, Hills BA. The lung as a filter for microbubbles. J Appl Physiol 1979;47:537-43.
33 Niden AH, Aviado DM. Effects of pulmonary embolism on the pulmonary circulation with special reference to arterio-venous shunts in the lung. Circ Res 1956;4:67-73.
34 Hagen PT, Scholz DG, Edwards WD. Incidence and size of patent foramen ovale during the first 10 decades of life: an autopsy study of 965 normal hearts. Mayo Clin Proc 1984;59: 17-20.
35 Lechat P, Mas JL, Lascault G, Loron P, Theard M, Klimczac M, et al. Prevalence of patent foramen ovale in patients with stroke. N Engl J Med 1988;318:1148-52.
36 Cheng WJ, Kuan P, Lien WP, Lin FY. Detection of patent foramen ovale by contrast transesophageal echocardiography. Chest 1992;101:1515-20.
37 Di Tullio M, Sacco RL, Venketasubramanian N, Sherman D, Mohr JP, Homma S. Comparison of diagnostic techniques for the detection of a patent foramen ovale in stroke patients. Stroke 1993;24:1020-4.
38 Bogousslavsky J, Garazi S, Jeanrenaud X, Aebischer N, Van Melle G. Stroke recurrence in patients with patent foramen ovale: the Lausanne Study. Lausanne Stroke with Paradoxal Embolism Study Group. Neurology 1996;46:1301-5.
39 Dearani JA, Ugurlu BS, Danielson GK, Daly RC, McGregor CG, Mullany CJ, et al. Surgical patent foramen ovale closure for prevention of paradoxical embolism-related cerebrovascular ischemic events. Circulation 1999;100(19 Suppl):II171-5.

40 Windecker S, Wahl A, Chatterjee T, Garachemani A, Eberli FR, Seiler C, et al. Percutaneous closure of patent foramen ovale in patients with paradoxical embolism: long-term risk of recurrent thromboembolic events. Circulation 2000;101:893-8.
41 Sykes JJ, Yaffe LJ. Light and electron microscopic alterations in spinal cord myelin sheaths after decompression sickness. Undersea Biomed Res 1985;12:251-8.
42 Warren BA, Philp RB, Inwood MJ. The ultrastructural morphology of air embolism: platelet adhesion to the interface and endothelial damage. Br J Exp Pathol 1973;54:163-72.
43 Hills BA, James PB. Microbubble damage to the blood-brain barrier: relevance to decompression sickness. Undersea Biomed Res 1991;18:111-6.
44 Hallenbeck JM, Bove AA, Moquin RB, Elliott DH. Accerlerated coagulation of whole blood and cell-free plasma by bubbling in vitro. Aerosp Med 1973;44:712-4.
45 Ward CA, McCullough D, Fraser WD. Relation between complement activation and susceptibility to decompression sickness. J Appl Physiol 1987;62:1160-6.
46 Hjelde A, Bergh K, Brubakk AO, Iversen OJ. Complement activation in divers after repeated air/heliox dives and its possible relevance to DCS. J Appl Physiol 1995;78:1140-4.
47 Ogston D, Bennett B. Surface-mediated reactions in the formation of thrombin, plasmin and kallikrein. Br Med Bull 1978;34:107-12.
48 Albertine KH, Wiener-Kronish JP, Koike K, Staub NC. Quantification of damage by air emboli to lung microvessels in anesthetized sheep. J Appl Physiol 1984;57:1360-8.
49 Brunner FP, Frick PG, Bühlmann AA. Post-decompression shock due to extravasation of plasma. Lancet 1964;1:1071-3.
50 Bove AA, Hallenbeck JM, Elliott DH. Changes in blood and plasma volumes in dogs during decompression sickness. Aerosp Med 1974;45:49-55.
51 Elliott DH, Moon RE. Manifestations of the decompression disorders. In: Bennet PB, Elliott DH, editors. The physiology and medicine of diving. 4th ed. London: WB Saunders; 1993.
52 Rudge FW, Stone JA. The use of the pressure cuff test in the diagnosis of decompression sickness. Aviat Space Environ Med 1991;62:266-7.
53 Francis TJR, Gorman DF. Pathogenesis of the decompression disorders. In: Bennet PB, Elliott DH, editors. The physiology and medicine of diving. 3rd ed. London: WB Saunders; 1993.
54 Ence TJ, Gong H. Adult respiratory distress syndrome after venous air embolism. Am Rev Respir Dis 1979;119:1033-7.
55 Melamed Y, Shupak A, Bitterman H. Medical problems associated with underwater diving. N Engl J Med 1992;326:30-5.
56 Dick AP, Massey EW. Neurologic presentation of decompression sickness and air embolism in sport divers. Neurology 1985;35:667-71.
57 Butler FK, Pinto CV. Progressive ulnar palsy as a late complication of decompression sickness. Ann Emerg Med 1986;15: 738-41.
58 Francis TJ, Pearson RR, Robertson AG, Hodgson M, Dutka AJ, Flynn ET. Central nervous system decompression sickness: latency of 1070 human cases. Undersea Biomed Res 1988;15: 403-17.
59 Reuter M, Tetzlaff K, Hutzelmann A, Fritsch G, Steffens JC, Bettinghausen E, et al. MR imaging of the central nervous system in diving-related decompression illness. Acta Radiol 1997; 38:940-4.
60 Warren LP, Djang WT, Moon RE, Camporesi EM, Sallee DS, Anthony DC, et al. Neuroimaging of scuba diving injuries to the CNS. AJR Am J Roentgenol 1988;15:1003-8.
61 Hallenbeck JM, Bove AA, Elliott DH. Mechanisms underlying spinal cord damage in decompression sickness. Neurology 1975;25:308-16.
62 Wilmshurst P. Brain damage in divers. BMJ 1997;314:689-90.
63 Reul J, Weis J, Jung A, Willmes K, Thron A. Central nervous system lesions and cervical disc herniations in amateur divers. Lancet 1995;345:1403-5.
64 Knauth M, Ries S, Pohimann S, Kerby T, Forsting M, Daffertshofer M, et al. Cohort study of multiple brain lesions in sport divers: role of a patent foramen ovale. BMJ 1997;314: 701-5.
65 Tetzlaff K, Friege L, Hutzelmann A, Reuter M, Holl D, Leplow B. Magnetic resonance signal abnormalities and neuropsychological deficits in elderly compressed-air divers. Eur Neurol 1999;42:194-9.
66 Schwerzmann M, Seiler C, Lipp E, Guzman R, Lovbald 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.
67 Polkinghorne PJ, Sehmi K, Cross MR, Minassian D, Bird AC. Ocular fundus lesions in divers. Lancet 1988;2:1381-3.
68 Palmer AC, Calder IM, Yates PO. Cerebral vasculopathy in divers. Neuropathol Appl Neurobiol 1992;18:113-24.
69 Todnem K, Nyland H, Skeidsvoll H, Svihus R, Rinck P, Kambestad BK, et al. Neurological long term consequences of deep diving. Br J Ind Med 1991;48:258-66.
70 Eckenhoff RG, Olstad CS, Carrod G. Human dose-response relationship for decompression and endogenous bubble formation. J Appl Physiol 1990;69:914-8.
71 Wilmshurst PT, Ellis BG, Jenkins BS. Paradoxical gas embolism in a scuba diver with an atrial septal defect. Br Med J (Clin Res Ed) 1986;293:1277.
72 Moon RE, Camporesi EM, Kisslo JA. Patent foramen ovale and decompression sickness in divers. Lancet 1989;1:513-4.
73 Wilmshurst PT, Byrne JC, Webb-Peploe MM. Relation between interatrial shunts and decompression sickness in divers. Lancet 1989/II:1302-6.
74 Germonpre P, Dendale P, Unger P, Balestra C. Patent foramen ovale and decompression sickness in sports divers. J Appl Physiol 1998;84:1622-6.
75 Bove AA. Risk of decompression sickness with patent foramen ovale. Undersea Hyperb Med 1998;25:175-8.
76 Walsh KP, Wilmshurst PT, Morrison WL. Transcatheter closure of patent foramen ovale using the Amplatzer septal occluder to prevent recurrence of neurological decompression illness in divers. Heart 1999;81:257-61.
77 Wilmshurst PT, Nightingale S, Walsh KP, Morrison WL. Effect on migraine of closure of cardiac right-to-left shunts to prevent recurrence of decompression illness or stroke or for haemodynamic reasons. Lancet 2000;356:1648-51.
78 Divers Alert Network. Report on diving accidents and facilities. Durham, NC: Divers Alert Network; 1996.
79 Moon RE. Treatment of decompression sickness and arterial gas embolism. In: Bove AA, editor. Bove and Davis' diving medicine. 3rd ed. Philadelphia, PA: WB Saunders; 1997. p. 184-204.