Decompression illness

Richard D Vann, Frank K Butler, Simon J Mitchell, Richard E Moon

Decompression illness is caused by intravascular or extravascular bubbles that are formed as a result of reduction in environmental pressure (decompression). The term covers both arterial gas embolism, in which alveolar gas or venous gas emboli (via cardiac shunts or via pulmonary vessels) are introduced into the arterial circulation, and decompression sickness, which is caused by in-situ bubble formation from dissolved inert gas. Both syndromes can occur in divers, compressed air workers, aviators, and astronauts, but arterial gas embolism also arises from iatrogenic causes unrelated to decompression. Risk of decompression illness is affected by immersion, exercise, and heat or cold. Manifestations range from itching and minor pain to neurological symptoms, cardiac collapse, and death. First-aid treatment is 100% oxygen and definitive treatment is recompression to increased pressure, breathing 100% oxygen. Adjunctive treatment, including fluid administration and prophylaxis against venous thromboembolism in paralysed patients, is also recommended. Treatment is, in most cases, effective although residual deficits can remain in serious cases, even after several recompressions.

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Department of Anesthesiology and Center for Hyperbaric Medicine and Environmental Physiology, Duke University Medical Center, Durham, NC, USA (R D Vann PhD, Prof R E Moon MD)**; United States Army Institute of Surgical Research, San Antonio, TX, USA** (F K Butler MD)**; and Department of Anaesthesiology, University of Auckland, Auckland, New Zealand** (S J Mitchell FANZCA)

Correspondence to:
Dr Richard D Vann, Box 3823, Center for Hyperbaric Medicine and Environmental Physiology, Duke University Medical Center, Durham, NC 27710, USA
richard.vann@duke.edu

Introduction

Decompression illness is caused by bubbles in blood or tissue during or after a reduction in environmental pressure (decompression). It includes two pathophysiological syndromes: arterial gas embolism and the more common decompression sickness. Arterial gas embolism occurs mainly during hyperbaric exposure (eg, diving) and rarely during hypobaric exposure (eg, altitude).

Arterial gas embolism occurs when expanding gas stretches and ruptures alveolar capillaries—pulmonary barotrauma—allowing alveolar gas to enter the arterial circulation (figure 1). This syndrome can occur after ascent from a depth as shallow as 1·0–1·5 m if the starting lung volume is close to total lung capacity.[1] It can be caused by gas becoming trapped as a result of airways obstruction in disorders such as asthma[2] or by the presence of pulmonary blebs, cysts, or bullae.[3] Arterial gas embolism can also arise in the absence of decompression through iatrogenic accidents involving vascular catheters and mechanical ventilation.

Decompression sickness starts with the formation and increase in size of extravascular and intravascular bubbles when the sum of the dissolved gas tensions (oxygen, carbon dioxide, nitrogen, helium) and water vapour exceeds the local absolute pressure. In diving and during compressed-air tunnel and caisson work, this state of supersaturation is made possible by the increase in tissue inert gas partial pressure that occurs when the gas (usually nitrogen, but occasionally helium) is respired at high pressure. Supersaturation arises during decompression if the rate of ambient pressure reduction exceeds the rate of inert gas washout from tissue. Ascent to altitude in aviation and extravehicular activity during spaceflight involves exposure to decreased barometric pressure. In these settings, supersaturation arises as a result of pre-existing dissolved nitrogen at sea level (partial pressure of nitrogen of about 570 mm Hg), which can also cause bubble formation.

Venous gas emboli formed from dissolved gas are easily detected by ultrasonography. In divers, 3·6 m is the minimum dive depth after which venous gas emboli can be seen[4] whereas the decompression sickness threshold, after saturation dives lasting 1–3 days, is about 6 m.[5] During direct decompression from sea level to altitude, the threshold for formation of venous gas emboli is around 3600 m whereas the decompression sickness threshold is about 5500 m.[6,7]

Bubbles can have mechanical, embolic, and biochemical effects with manifestations ranging from trivial to fatal. Clinical manifestations can be caused by direct effects from extravascular (autochthonous) bubbles such as mechanical distortion of tissues causing pain, or vascular obstruction causing stroke-like signs and symptoms. Secondary effects can cause delayed symptom onset up to 24 h after surfacing. Endothelial damage by intravascular bubbles can cause capillary leak, extravasation of plasma, and haemoconcentration.[8] Impaired endothelial function, as measured by decreased effects of vasoactive compounds, has been reported in animals[9] and might occur in man. Hypotension can occur in severe cases.[10] Other effects include platelet activation and deposition,[11] leucocyte-endothelial adhesion,[12] and possibly consequences of vascular occlusion believed to occur in thromboembolic stroke such as ischaemia-reperfusion injury, and apoptosis.[13]

Arterial gas embolism most often affects the brain but can occasionally affect the heart and other organs.

Search strategy and selection criteria

We searched PubMed in English with the search terms "decompression illness", "decompression sickness", and "arterial gas embolism" for reports mostly published in the past 20 years until January, 2010. Bibliographies of selected articles were reviewed for other relevant references. We also relied on our familiarity with key literature. Pertinent review articles, book chapters, proceedings, and papers older than 20 years were used when judged important, but some conclusions are based on anecdotal reports because randomised trials are rare.

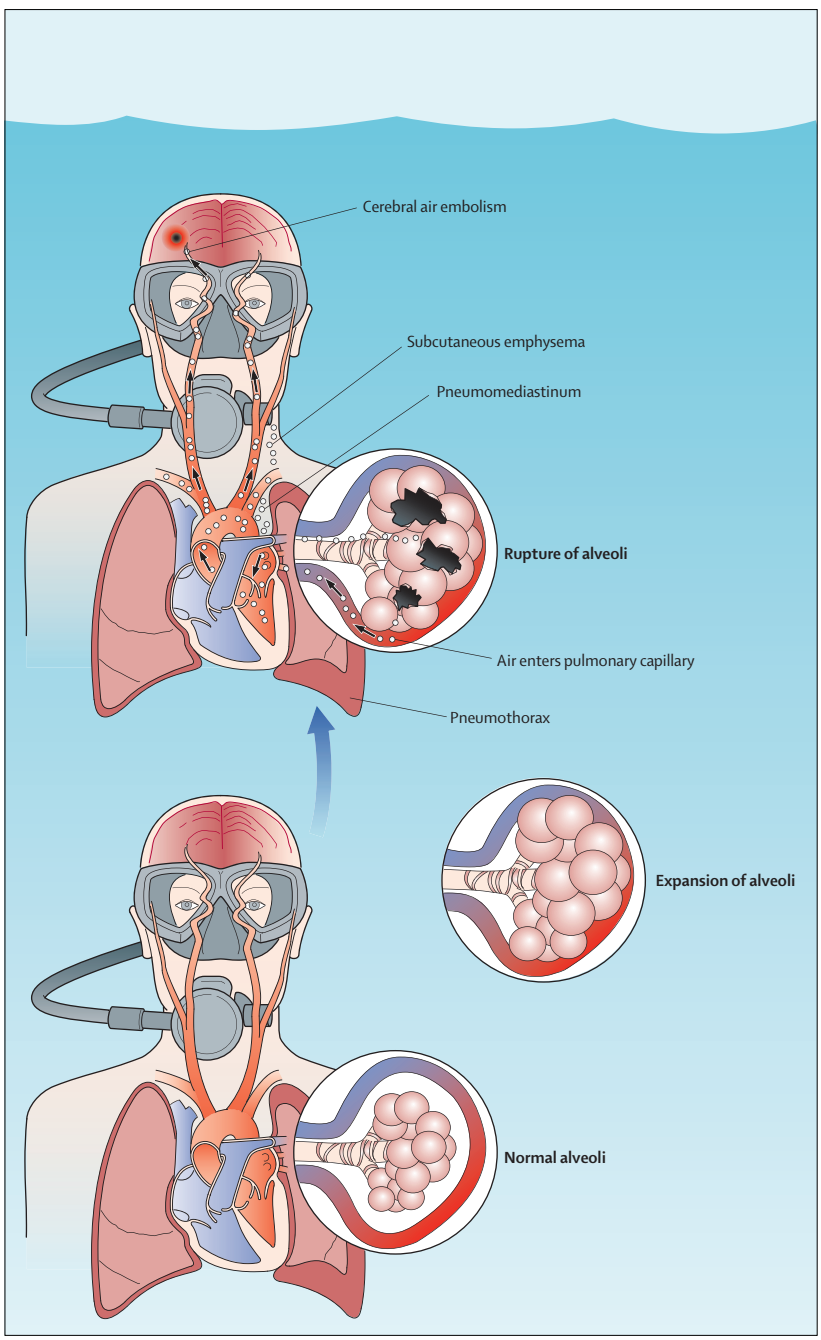

Figure 1: **Pulmonary barotrauma in a diver during breath-hold ascent**

Decompression sickness produces symptoms related to the effects of bubbles on periarticular tissues, spinal cord, brain, lungs, skin, and the audiovestibular system. Concurrent arterial bubbles from arterial gas embolism exacerbate decompression sickness,[14] possibly by reducing tissue perfusion and impairing inert gas washout or by increasing bubble size. Decompression sickness symptoms after recreational diving (figure 2) typically consist of pain or mild neurological manifestations such as numbness or paraesthesias. Most patients with altitude-related decompression sickness have similar manifestations,[15–18] although cerebral symptoms have been reported in U-2 pilots.[19,20]

Small quantities of venous gas emboli are common in diving[21] although they are usually asymptomatic because most of the time they are effectively filtered by the pulmonary circulation. However, large numbers of venous gas emboli can cause cough, dyspnoea, and pulmonary oedema (cardiorespiratory decompression sickness, or chokes)[22] and can overcome the pulmonary capillary filter.[23] Moreover, a patent foramen ovale or other right-to-left cardiac shunt is present in about 27% of the normal population,[24] and theoretically some venous gas emboli could enter the arterial circulation and reach the CNS, where they could grow from the inward diffusion of supersaturated inert gas.[25] Patent foramen ovale has been statistically associated with cerebral, spinal, and vestibulocochlear manifestations,[26–36] and with cutaneous manifestations.[30]

Epidemiology

Arterial gas embolism is usually precipitated by rapid ascent, breath-holding, or the presence of lung disease, and thus is rare with an apparently decreasing incidence. The proportion of cases of decompression illness attributable to arterial gas embolism in recreational divers declined from 18% in 1987 to 8% in 1997.[37] Of 441 confirmed or possible incidents of decompression illness in recreational divers reported to the Divers Alert Network, only 3·9% were classified as possible arterial gas embolism.[38]

If appropriate decompression procedures are followed, decompression sickness is also uncommon. Rate of occurrence (per dive) in operational open water dives from minutes to several hours in duration varies according to the diving population: typically 0·015% for scientific divers, 0·01–0·019% for recreational divers, 0·030% for US Navy divers, and 0·095% for commercial divers.[39,40] The number of active worldwide recreational divers is not known but is likely to be in the millions. The Divers Alert Network took a sample of 135 000 dives by 9000 recreational divers in which the rate of occurrence of decompression sickness was 0·03%. This rate was much higher during dives to wrecks in cold water than during dives in warm water from diving cruise vessels.[38] These numbers are all based on many dives made well within the maximum exposure limits of accepted procedures (decompression tables or computers) and therefore are underestimates of the true rates at the maximum limits. For example, the rate of occurrence of decompression sickness for US Navy dives from 1971 to 1978 at the maximum limits was 1·3%.[41] Moreover, for long exposures under stressful thermal and exercise conditions, US Navy dive trials designed to develop new decompression procedures had an occurrence rate of 4·4 cases of decompression sickness per 100 dives.[42]

Technical diving, a form of recreational diving with deep, long exposures, could be associated with a higher incidence of decompression sickness and more serious manifestations than are the other types of diving, although insufficient data are available for accurate estimates. Commercial diving for construction and offshore oil production is another important diving activity. Decompression sickness is reportedly rare in modern commercial saturation dives (working dives lasting several days, with one, extended decompression) although documentation for commercial diving and for compressed air and caisson work is insufficient. For hyperbaric medicine attendants, the occurrence rate of decompression sickness is reported as 0·02% per exposure.[43,44] In altitude training or flight operations, the rate of occurrence of decompression sickness is typically less than 0·1% per exposure, with most individuals reporting only mild symptoms.[16,17] However, anonymous surveys of high-altitude Air Force pilots indicated higher frequencies,[18] with some cases being quite serious.[19,20]

Risks of decompression sickness that are thought to be acceptable are a matter of subjective judgment. Acceptable risks specified for commercial diving include 0·1% for mild cases and 0·025% for serious cases,[39] whereas for US Navy diving, acceptable risk is 2% for mild cases and 0·1% for serious cases.[45]

Other than depth and time, risk of decompression sickness is affected by other factors that affect inert gas exchange and bubble formation, such as immersion (*vs* dry hyperbaric chamber exposure), exercise, and temperature. Immersion decreases venous pooling and increases venous return and cardiac output.[46,47] Warm environments improve peripheral perfusion by promoting vasodilation, whereas cool temperatures decrease perfusion through vasoconstriction. Exercise increases both peripheral perfusion and temperature. The effect of environmental conditions on risk of decompression sickness is dependent on the phase of the pressure exposure.[39] While under pressure, exercise, immersion, or a hot environment increase inert gas uptake and risk of decompression sickness. During decompression these factors increase inert gas elimination and therefore decrease the risk of decompression sickness.[48] Conversely, uptake is reduced during rest or in a cold environment, hence a diver resting in a cold environment on the bottom has decreased risk of decompression sickness. Rest or low temperatures during decompression increase the risk. If exercise occurs after decompression when supersaturation is present, bubble formation increases and risk of decompression sickness rises.[39,49]

Exercise at specific times before a dive can decrease the risk of serious decompression sickness in animals and incidence of venous gas emboli in both animals and man.[50–54] The mechanisms of these effects are unknown but might involve modulation of nitric oxide production and effects on endothelium. Whether appropriately timed exercise can decrease the probability of decompression sickness in man is unknown. Venous gas emboli and risk of decompression sickness increase slightly with age and body-mass index,[21,39] but the effect of sex is uncertain.[7,55]

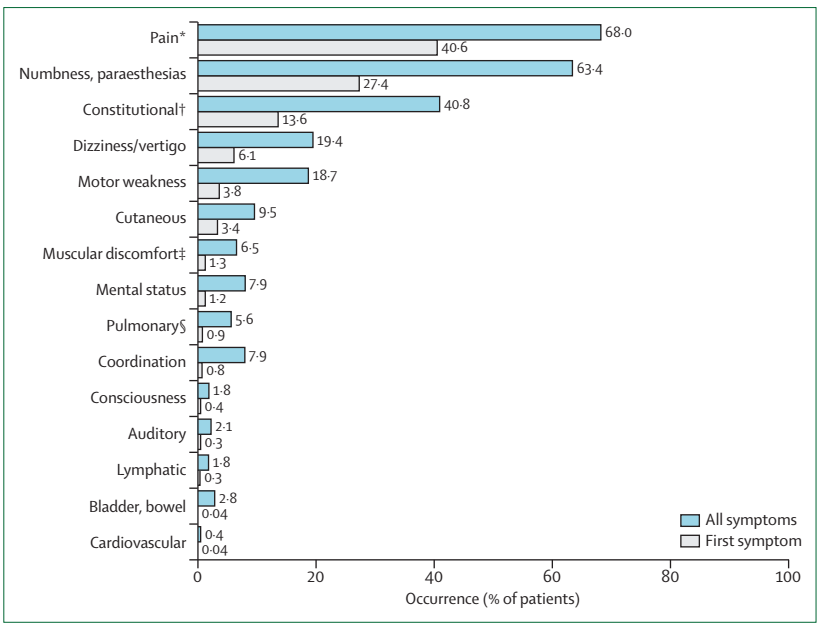

Figure 2: **Classification of initial and of all eventual manifestations of decompression illness in 2346 recreational diving accidents reported to the Divers Alert Network from 1998 to 2004**
*For all instances of pain, 58% consisted of joint pain, 35% muscle pain, and 7% girdle pain. Girdle pain often portends spinal cord involvement. †Constitutional symptoms included headache, lightheadedness, inappropriate fatigue, malaise, nausea or vomiting, and anorexia. ‡Muscular discomfort included stiffness, pressure, cramps, and spasm but excluded pain. §Pulmonary manifestations included dyspnoea and cough.

Diagnosis

The protean nature of decompression illness makes diagnosis difficult. Diagnosis is made on a clinical basis, thus accurate history and physical examination of individuals with symptoms after diving or altitude exposure are crucial.

Arterial gas embolism should be suspected if a diver has a new onset of altered consciousness, confusion, focal cortical signs, or seizure during ascent or within a few minutes after surfacing from a compressed gas dive. If the diver spends much time at depth and might have absorbed substantial inert gas before surfacing, arterial gas embolism and serious decompression sickness can coexist, and in such cases, spinal cord manifestations can predominate.[14] Other organ systems, such as the heart, can also be affected, but the clinical diagnosis of gas embolism is not reliable without CNS manifestations. Arterial gas embolism is rare in altitude exposure; if cerebral symptoms occur after altitude exposure, the cause is usually decompression sickness.

The diagnosis of decompression sickness is based entirely on clinical manifestations. Any new symptom arising shortly after decompression should be considered as possible decompression sickness,

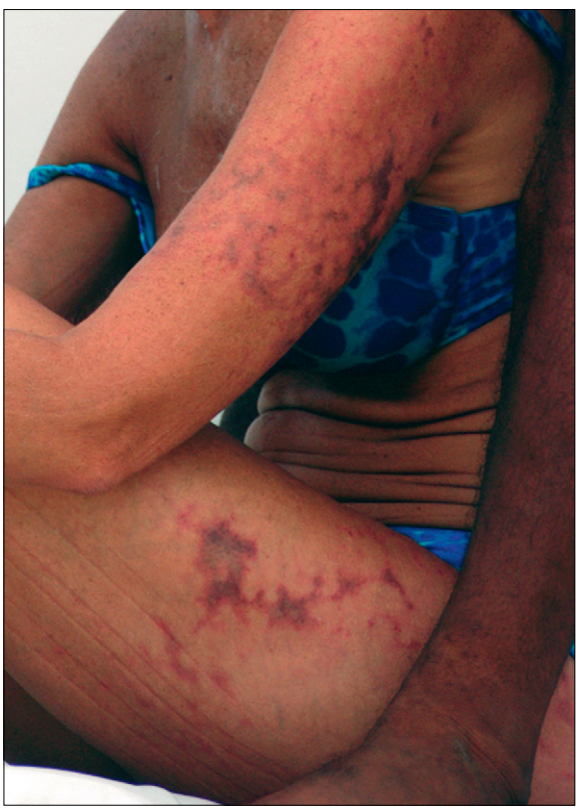

Figure 3: **Livedo reticularis (cutis marmorata) due to decompression sickness in a recreational diver**
This rash occurring after a dive is uncommon but almost pathognomonic of decompression sickness.

especially if the depth-time exposure has approached or exceeded accepted procedures. The diagnosis is more certain after a provocative depth-time exposure (one that is close to or exceeds limits prescribed by dive tables or computers) than after a mild exposure. The *US Navy Diving Manual*[56] is widely used for comparison in assessment of exposure severity.

Although arterial gas embolism can arise after ascent from very shallow depths, decompression sickness almost never occurs after a single dive to depths of less than 6 m, even for an extended time,[5] and is uncommon at depths of less than 10 m. For altitude exposure, decompression sickness is rare unless preceded by diving, or if ascent to 5500 m or higher was rapid (eg, in a military aircraft or hypobaric chamber).

Decompression sickness has a wide range of manifestations.[56–59] The frequency of initial manifestations and all eventual manifestations for recreational divers are listed in figure 2. Constitutional and non-specific symptoms such as malaise, fatigue, headache, and transient periarticular discomfort in isolation are usually thought to be of minor clinical importance but do seem to be related to decompression stress.[60] In the data presented in figure 2, pain, constitutional symptoms, subjective numbness, paraesthesias, or rash initially occurred in 85% of cases, and at least one mild manifestation was present in all cases.

The type of exposure can affect the manifestation pattern. For example, in recreational divers, joint pain tends to be more common in the arms, whereas in saturation divers pain in the knees is more common. Audiovestibular manifestations are more frequent after deep heliox diving than after air diving.[25,58,61] Neurological decompression sickness is more common in civilian treatment centres than after military dive trials,[57,58,62] but whether this difference is due to variations in diving practice or symptom reporting is not clear. Predilection for injury pattern (eg, spinal cord *vs* inner ear) might vary with the characteristics of the divers.[25]

Although some instances of decompression sickness (especially in saturation diving) occur during decompression, most cases present soon after surfacing. For example, in a series of military divers, 42% of symptoms occurred within 1 h after diving, 60% within 3 h, 83% within 8 h, and 98% within 24 h.[56] CNS cases of decompression sickness present more quickly: in 1070 patients, 56% had symptoms within 10 min and 90% within 4 h.[63] Decompression sickness can be considered even several days after diving, especially if the patient has subsequently travelled by air or ascended to altitude.[64–66] Symptoms that develop either during descent or at depth are never due to decompression sickness unless there was previous diving within a few hours.

Neurological examination is essential for all divers with suspected decompression illness, unless re-compression would be delayed during rapid evolution of obvious neurological abnormalities. Cognitive manifestations are often not noticed by patients and can be detected only by direct examination. Generally, the findings in neurological assessment differ from those noted during most common stroke syndromes. Non-dermatomal hypoaesthesia and truncal ataxia are common in neurological decompression sickness and can be missed by cursory examination. Pertinent information includes level of consciousness and mental status, cranial nerve function, and motor strength. Coordination can be affected disproportionately, and abnormalities can be detected by assessment of finger-nose movement, and, with eyes open and closed, ability to stand and walk and do heel-toe walking backwards and forwards. Many of these simple tests can be done on the scene by untrained companions. Physical examination is often normal, especially when symptoms are limited to pain or paraesthesias. Lymphoedema (particularly in the trunk) and rash (figure 3) can result from decompression sickness.

Severe decompression sickness can be accompanied by haemoconcentration due to endothelial leak.[8,10] Thus, measurement of blood haemoglobin or packed-cell volume could help guide fluid resuscitation. Concentration of serum creatine kinase helps

distinguish arterial gas embolism from decompression sickness (enzyme concentration in severe cases can be high in pulmonary barotrauma with arterial gas embolism[67]), although differentiation between these disorders before recompression is unnecessary since recompression is indicated for both. Chest radiography is useful for detection of pneumothorax after a suspected arterial gas embolism. For detection of extrapulmonary air, chest CT is more sensitive but is unnecessary because of its high radiation exposure.

Bubbles are rarely detectable with radiography in joints affected by pain, and are rarely noted in the brain with either MRI or CT.[68] MRI is similarly not useful for detection of abnormalities of the spinal cord related to decompression sickness.[69] Thus, although laboratory and radiological analyses are useful for detection of abnormalities in some cases, imaging studies are not useful for assessment of whether a patient needs recompression treatment and should not delay recompression unless there is a strong suspicion of a non-diving related cause (eg, cerebral haemorrhage). Specific neurophysiological tests (eg, audiometry and electronystagmography for inner-ear decompression sickness) or imaging can usually be delayed until after recompression. Doppler ultrasonography and echocardiography are valuable for research into venous gas emboli[4,21,51–54,70] but not for diagnosis of decompression illness. The differential diagnosis of decompression illness includes acute coincidental illness, especially neurological disorders. Specific disorders that could be confused with decompression illness are listed in the panel.

Decompression injuries have traditionally been classified as arterial gas embolism, and type 1 and type 2 decompression sickness. Type 1 decompression sickness included pain, cutaneous manifestations, and constitutional symptoms, whereas type 2 manifestations included numbness, tingling, paraesthesias, muscle weakness, paralysis, and mental or motor abnormalities.[81] Arterial gas embolism and type 2 decompression sickness were thought to require more aggressive treatment than type 1 decompression sickness. Because the traditional classification system was applied inconsistently and had little predictive value, it has been largely replaced by the inclusive term decompression illness.[82] Although identification of the pathophysiological changes in individuals is useful for epidemiology and recommendations for future diving, the correct pathophysiological changes often cannot be identified and are not usually important for selection of treatment.

Other classification systems that have been proposed were for the creation of severity indices that might be useful to guide treatment and prediction of outcome.[83–89] These approaches showed reasonable associations between severity at presentation and outcome,[69,85,90–94] but need to be more consistent and simpler for their potential to be reached.[95–97]

Panel: **Differential diagnosis of decompression illness**

Inner-ear barotrauma
Inner-ear barotrauma usually occurs during descent and results in tinnitus, hearing loss, and persistent vertigo.[71,72] Conductive hearing loss is seen in middle-ear barotrauma. Both inner-ear and middle-ear barotrauma are usually preceded by difficulty in equalising middle-ear pressure. Transient vertigo during compression or decompression can arise because of asymmetric middle-ear pressure equilibration (alternobaric vertigo).[61]

Middle-ear or maxillary sinus overinflation
This disorder is caused by gas expansion during ascent and an obstructed eustachian tube or sinus ostium, resulting in compression of the facial nerve and unilateral upper and lower facial weakness,[73] or compression of branches of the trigeminal nerve causing hypoaesthesia of the face.[74]

Contaminated diving gas and oxygen toxic effects
Carbon monoxide poisoning due to contaminated breathing gas can cause encephalopathy and convulsions. Toxic effects of oxygen are most common in divers using enriched oxygen breathing mixtures and can cause convulsions at depth.

Musculoskeletal strains or trauma sustained before, during, or after diving[56,58,74–76]
Time of onset and history of trauma or strain are helpful. Pain due to decompression illness is rarely accompanied by tenderness or position-related or motion-related exacerbation physical examination.

Seafood toxin ingestion (ciguatera, puffer fish, paralytic shellfish poisoning)
Ingestion of toxins is often associated with gastrointestinal symptoms and can cause neurological manifestations within hours after ingestion.[77]

Immersion pulmonary oedema
This disorder usually occurs shortly after the start of a dive, while the diver is still at depth, and might be confused with cardiorespiratory decompression sickness, since both cause dyspnoea and cough.[78–80] Symptoms of immersion pulmonary oedema typically begin during descent or at depth, whereas the onset of cardiorespiratory decompression sickness occurs after the dive.

Water aspiration
Water aspiration could be mistaken for cardiorespiratory decompression sickness. Both cardiorespiratory decompression sickness and water aspiration can cause pulmonary oedema, although the diver is usually aware of aspiration.

Coincidental, unrelated acute neurological disorder (eg, stroke, spinal hematoma)
Diagnosis is made with conventional techniques.

Prevention

Arterial gas embolism is rare at altitude and is not related to depth-time exposure in diving. The risk of this syndrome can be decreased by avoidance of breath holding, rapid ascent, and diving with pulmonary infections or disease. Risk of decompression sickness is decreased by reduction of exposure or by elimination of inert gas before (eg, with high oxygen concentrations) or during decompression, but adherence to these procedures does not always prevent the syndrome. 100 years ago, serious manifestations and deaths were frequent in diving and caisson work but they decreased greatly when decompression stops were introduced. These stops delay ascent to the surface and allow inert gases to be eliminated in dissolved form rather than as bubbles.[6,99] A

decompression schedule specifies the stop times typically at 3 m intervals during ascent according to the maximum dive depth and bottom time. In the 1980s, equally effective diver-worn digital computers (dive computers) were developed that continuously track depth-time exposure and specify how slowly a diver should ascend. Gas mixtures with high oxygen partial pressures are sometimes used to decrease inert gas absorption at depth and for faster elimination during decompression. For dives deeper than 45 m, gas mixtures containing helium are used to avoid nitrogen narcosis and to decrease the density of respired gas.

Detection of patent foramen ovale as a way of reducing the risk of serious decompression sickness has been of much interest. Estimates of relative risk for serious neurological decompression sickness associated with a patent foramen ovale range from 2·5 to 6·6.[32] Nevertheless, routine screening of candidate divers for patent foramen ovale does not seem warranted since the absolute risk of neurological decompression sickness is small (<0·02%),[34] and the cost of screening is high.

Decompression sickness is rare after rapid ascent to altitudes lower than 5500 m with increasing risk at higher altitudes,[7] unless the altitude exposure is preceded by diving within several days, in which case decompression sickness can occur at less than 2500 m.[64,99,100] Oxygen breathing at sea level before altitude exposure eliminates dissolved tissue nitrogen and permits higher altitudes and longer flights. For extravehicular activity during spaceflight, protocols have been developed that use oxygen breathing and exercise before the activity to accelerate nitrogen elimination before decompression to space-suit pressures of 0·3–0·4 bar.[39]

Treatment

Decompression illness is rare and only one prospective randomised trial of treatment has been reported so far.[101] The following guidelines are therefore based largely on case reports, case series, animal studies, and clinical judgment, and have empirically changed in the past 60 years, especially with respect to first aid and adjunctive treatment.

The principles of basic and advanced life support apply to any obtunded diver, but manifestations of decompression illness are typically mild and non-specific, although potentially progressive. Appropriate first aid should be applied as soon as possible. Assistance with diagnosis or management can be obtained from various diving medical services, such as the Divers Alert Network or Duke Dive Medicine.

For the **Divers Alert Network** see www.diversalertnetwork.org

For **Duke Dive Medicine** see www.dukedivemedicine.org

The best and primary first aid for decompression illness is 100% oxygen delivered for several hours even if manifestations resolve. Pure oxygen washes inert gas from the lungs and establishes the largest possible inert gas gradient from tissue to alveolar gas. This gradient results in rapid removal of inert gas from tissue to lungs by perfusion and from bubble to tissue by diffusion, and thus removal of bubbles.[102] Another advantage of pure oxygen is amelioration of tissue hypoxia caused by bubble-induced ischaemia, mechanical injury, or biochemical damage. In an observational study, patients with decompression sickness who received oxygen during first aid had symptom resolution after fewer recompressions than did those who did not receive oxygen.[103] Oxygen given under slight pressure while a diver is returned to the water might be appropriate under some circumstances but is controversial because of the increased risk of seizures caused by toxic effects of oxygen on the CNS.[104]

Although the head-down position has been advocated to prevent distribution of arterial bubbles towards the head, it is not effective and can promote cerebral oedema.[105] Consensus is for horizontal orientation in a position that helps care of the patient. Fluid administration is important and intravenous fluids can be beneficial, especially in serious cases, but few clinical data show benefit from a specific type or amount of intravenous fluid. Glucose-containing fluids are best avoided because of the potential for adverse effects of hyperglycaemia in neurological injury, and hypotonic fluids should not be used because they promote intracellular oedema.[106] Substantial fluid resuscitation is not recommended in patients with isolated arterial gas embolism—eg, a patient who suffers gas embolism after a short, shallow dive.[106] Oral rehydration can be used in stable, conscious patients, but is unlikely to help those in greatest need. Nitrous oxide is inappropriate for pain relief or as a component of a general anaesthetic for a coincidental procedure because it can cause an increase in the size of bubbles by inward diffusion.[107]

Typically, recompression is done in a multiplace chamber in which the diver is accompanied by one or more attendants (figure 4). Decompression illness has also been successfully treated in one-occupant monoplace chambers.[108] Recompression while breathing 100% oxygen decreases bubble volume as predicted by Boyle's law and increases the inert gas partial pressure gradients between tissue and alveolar gas. These effects lead to quick resolution of bubbles, relieve mechanical pressure on surrounding tissue, and encourage redistribution of bubbles lodged in the microcirculation.[109] Hyperbaric oxygen also oxygenates compromised tissues and ameliorates inflammatory responses that contribute to tissue injury.[12]

Recompression is usually advised even if manifestations resolve with first aid since untreated decompression sickness can recur days after the initial onset.[110] Evacuation for recompression might be needed, especially from remote locations.[111] For short distances, this evacuation could be by helicopter at low altitude, but for long distances, an air ambulance pressurised to 1 atm is usually recommended in severe cases.[111,112] When pain is the only symptom, short commercial flights seem to have no effect on outcome; when mild neurological symptoms

are present, short flights more than 24 h after diving also seem to be safe.[113] Mild initial manifestations can develop into a more serious form, usually within a few hours after surfacing. In serious cases, delayed recompression is probably less effective, but the time beyond which recompression is pointless is unknown.[94] Decisions about the advisability of recompression should ideally include a physician trained in diving medicine.

Recent decompression and manifestations compatible with decompression illness usually justify emergent recompression with oxygen in a pressure chamber (figure 4) unless another cause is obvious.[56] Recompression should occur as soon as feasible to avoid late recurrence and increased severity.[105,110] Treatment is recommended even in patients with substantially delayed presentation since clinical response often occurs hours or even days after onset.[114–116] Some patients need ventilator support and intravenous drug infusion during recompression (figure 4).

The most common recompression schedule is US Navy Treatment Table 6 (figure 5) or an equivalent procedure such as promulgated by the Royal Navy, in which patients are compressed to 2·8 bar (equivalent to 18 m sea water depth) while breathing 100% oxygen, a pressure with an acceptably low risk of cerebral oxygen-associated toxic effects.[56,117] The time at 2·8 bar and 1·9 bar (equivalent to 9 m sea water) can be extended with additional cycles of oxygen and air if resolution is not complete within the prescription shown in figure 5. If treatment pressures are greater than 2·8 bar, air is used or nitrogen or helium is added to the breathing mix to reduce the risk of oxygen-associated toxic effects. In animal studies, faster bubble resolution has been shown with helium than with oxygen,[102] but this comparison has been inadequately studied in clinical practice. Anecdotally, increased pressures can improve manifestations that are refractory at 2·8 bar,[118] and they have been advocated for both arterial gas emboli (when severe symptoms remain unchanged or worsen within the first 20 min at 2·8 bar)[56] and decompression sickness.[119] However, supporting evidence is weak for depths greater than 18 m for initial recompression without a demonstrated need to go deeper, and no benefit has been shown in animal studies.[120,121] Many recompression strategies ranging from pressures of 1·9–10·0 bar exist, but there are no human outcome studies for comparison of efficacy. Short treatment schedules have been tested, and seem effective.[83,108]

If resolution is complete after one treatment, no additional treatments are needed. With residual manifestations after the first treatment, recompression is typically repeated every day (often with short treatment tables such as the US Navy Treatment Table 5)[56] until complete symptom resolution or no further improvement.[105] Most patients with residual neurological manifestations need only two or three treatments to reach a clinical plateau.[101] Nonetheless, some severe cases

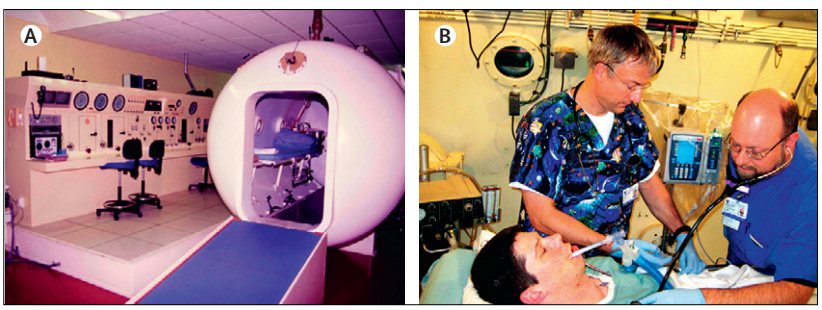

Figure 4: **Recompression chamber**
(A) Multiplace recompression chamber. The chamber can typically be compressed to 6 bar with air. Treatment gas (oxygen, oxygen and nitrogen, or oxygen and helium mixes) is given to the patient via a head tent, tightly fitting mask or, for critically ill patients, through endotracheal tube. Photo from SJM. (B) Critical care in a multiplace chamber. A fluidic or pneumatic ventilator is shown at the left. The infusion pump is contained within a plastic cover, in which 100% nitrogen is used to decrease the fire risk in the event of an electrical problem. The monitor screen is outside the chamber and can be seen through the viewing port. Photo from Duke University Medical Center, with permission.

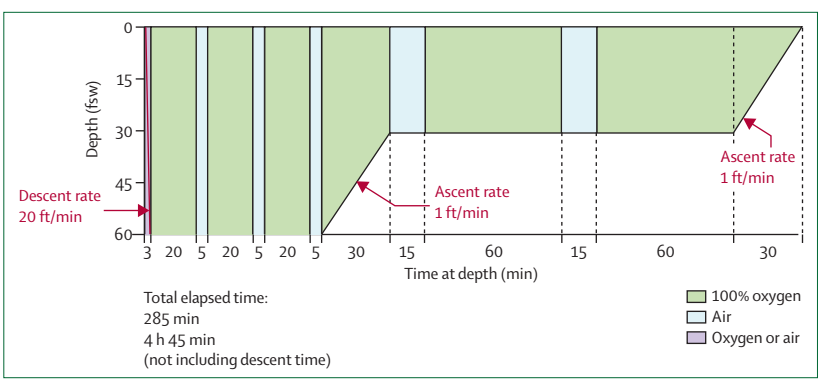

Figure 5: **US Navy Treatment Table 6**
From *US Navy Diving Manual*.[56] fsw=feet sea water.

(eg, important motor weakness) do not reach a plateau until after 15–20 repetitive treatments. In decompression sickness in which the predominant manifestations are sensory or pain, symptoms often wax and wane every day. Unless improvement after each treatment is documented, prolonged hyperbaric treatment is unnecessary.

A few facilities are equipped and staffed to provide saturation therapy, in which the chamber and its occupants are maintained at a constant elevated ambient pressure (eg, 2·8 bar for US Navy Treatment Table 7).[56,122] Decompression starts after clinical resolution or stability is achieved, often in 2–3 days. Saturation treatment is usually reserved for cases of severe neurological impairment, in which resolution was incomplete during initial treatment or deterioration occurred during decompression. Data do not suggest that the outcome for saturation treatment is better than that with repetitive short treatments.

Administration of 100% oxygen at 1 atm is often sufficient treatment for mild decompression sickness after altitude exposure without a preceding dive.[123] Recompression should ideally be offered to all divers with suspected decompression sickness. If recompression facilities are remote and the patient has mild

	n	%
No residual symptoms	34	49·3%
Any residual symptom	35	50·7%
Mild paraesthesias, weakness, or pain	14	20·3%
Some impairment of daily activities	21	30·4%
Difficulty walking	11	15·9%
Impaired micturition	13	18·8%
Impaired defecation	15	21·7%
Impaired sexual function	15	21·7%

Data are taken from 51 men, 18 women; mean age 41·2 years (range 19–70 years). Divers were assessed by phone interview after a median of 6·1 years (range 2·3–9·7 years). All but one had received recompression treatment. (Data from the Divers Alert Network.)

Table: **Long-term outcomes of 69 divers with spinal cord decompression sickness, by manifestation**

decompression illness (defined as static or remitting limb pain, constitutional symptoms, rash, or non-dermatomal sensory changes that remain stable for 24 h with a normal neurological assessment), the outcome is unlikely to be worse without recompression.[111] In a remote location, the risk-benefit ratio of a hazardous evacuation might not be favourable compared with delayed recompression or even no recompression.

Decompression sickness during decompression from a saturation dive (commercial or military) is usually treated with a small recompression to the pressure at which symptoms are relieved and by administration of oxygen-enriched breathing gas (oxygen partial pressure 1·5–2·8 bar).[56]

Although recompression is the primary treatment, especially for serious decompression illness, other aspects of care for seriously ill patients should not be neglected—eg, management of airway compromise, coma, haemodynamic instability, temperature control, metabolic instability, bladder dysfunction, pain, the risks of immobility, and long-term disability. In patients with leg immobility, prophylaxis is recommended because of the substantial risk of venous thromboembolism.[106]

Asymptomatic diving routinely causes slight haemoconcentration suggesting dehydration.[124] In animals, dehydration can increase the seriousness of decompression sickness,[125] and in man, adequate hydration decreases bubble formation after decompression.[70] Severe decompression sickness can cause substantial haemoconcentration and haemodynamic instability, presumably by widespread endothelial damage or inflammation.[10,126] Such findings encourage the routine use of intravenous rehydration with non-glucose isotonic crystalloid fluids. Fluid overload is best avoided because it can contribute to cerebral, spinal cord, or pulmonary oedema.

Specific drugs have been used as adjuncts to recompression. In a randomised trial, the non-steroidal anti-inflammatory drug tenoxicam decreased the number of recompressions needed to achieve symptom resolution or recovery plateau but did not change final outcome.[101] The prevalence of use of non-steroidal anti-inflammatory drugs in this context is unknown. Aspirin has previously been advocated for its antiplatelet effects, but it has not been formally studied in this context and is not recommended.[106]

Interest in intravenous lidocaine for treatment of neurological decompression illness arose out of this drug's apparent efficacy in vivo and anecdotal effectiveness in divers.[127] Neuroprotection has been reported in patients undergoing cardiac surgery,[128] but there are no studies in divers, and results of trials in cardiac surgery did not show repeat of initial success.[129,130] Lidocaine is usually reserved for serious neurological cases with features typical of arterial gas embolism. High-dose steroids worsen outcome in animals[131] and are not recommended in people.[106]

A promising prospect is intravenous perfluorocarbon emulsions[132] because both oxygen and inert gases are very soluble in these compounds. Perfluorocarbon emulsions probably work by enhancement of tissue oxygenation and inert gas transport from tissue to lungs.[133] Tissue bubbles shrank more rapidly with infusion of perfluorocarbon emulsions and oxygen breathing than with oxygen breathing alone in a mouse model of decompression sickness.[134] Moreover, in a pig model of this disease, morbidity and mortality were greatly reduced with infusion of perfluorocarbon emulsions at sea level.[135] Perfluorocarbon emulsions and hyperbaric oxygen have not been studied but hypothetically the combination could increase risk of oxygen-related toxic effects. The availability of a perfluorocarbon emulsion that is suitable for use in man is awaited before trials can be done.

Outcome

When oxygen treatment tables are used with an initial treatment pressure of 2·8 bar and the delay to treatment is not excessive, symptoms are resolved with a high degree of success.[105,108,136,137] 67% of 63 divers with spinal cord decompression sickness had complete resolution at 1 month after treatment. Of 30 patients in the same series with motor weakness, cerebral involvement, or cochleovestibular manifestations, only eight (27%) had severe disability 1 month after treatment.[94] In a study of 268 patients, including both amateur and professional divers, 230 (86%) had complete resolution or minor symptoms at discharge.[93] In a review of 1763 cases of decompression sickness, including several cases with long periods to treatment, 80% showed complete recovery.[136] Of 166 patients with decompression sickness resulting from experimental dives, with recompression facilities immediately at hand, 97% had complete relief after initial hyperbaric treatment, and all patients eventually showed complete resolution, despite some serious cases of neurological and cardiorespiratory disorders.[136] Long-term outcome data of 69 recreational divers with severe spinal cord decompression sickness are listed in the table.

Guidelines suggest that patients treated for decompression illness should be observed within timely reach of a chamber for 2 h when symptoms are mild or 6 h for severe symptoms.[56] US Navy guidelines suggest that patients should be within 1 h travelling time for 24 h after recompression.[56] Hospital admission might be necessary for patients with severe or residual symptoms. Worsening or recurrence suggests a need for immediate re-evaluation. Altitude exposure (eg, commercial airplane flight) after decompression illness can precipitate recurrence of symptoms. Generally, patients who were recompressed with complete relief should not fly for at least 72 h. Consultation with a trained hyperbaric physician is suggested before individuals with residual symptoms are allowed to fly.[56]

Recompression treatment results in complete resolution in most cases, mild residual symptoms in a few cases, and rarely serious residual manifestations. No follow-up diagnostic studies are necessary for patients whose only symptoms are mild (ie, tingling or joint pain) and resolved wholly. Patients whose symptoms are initially serious or are not fully resolved are usually reviewed within a few weeks of discharge. This patient assessment allows documentation of progress and discussion about future diving. Resumption of diving for recreational divers is usually allowed 4 weeks after treatment with complete recovery. Military and commercial diving organisations have similar guidelines for arterial gas embolism or neurological decompression sickness, but allow diving after a few days (7 days for the US Navy) after mild manifestations such as joint pain. Return to flying after pain-only decompression sickness that has fully resolved is also typically allowed after a few days (14 days for the US Air Force). Cautious discussion of risk and benefit is appropriate, especially if decompression sickness occurred after a conservative depth-time exposure (ie, one well within the maximum time allowed by an established decompression table or computer). If arterial gas embolism due to pulmonary barotrauma is suspected, radiological investigation and pulmonary function testing are usually done to exclude underlying pulmonary predisposition.

In severe cases, assessment with MRI of the brain and spinal cord after treatment can show abnormalities, although normal findings do not exclude decompression illness.[58,139] Positive MRI findings are related to severity and outcome.[69] Some individuals with initially normal MRI examinations develop late changes.[139] Similarly, nuclear imaging of the brain (eg, PET) is less sensitive than is clinical evaluation for detection of abnormalities.[140]

For divers with vestibular decompression sickness, compensation and symptom resolution will occur, and physical examination will return to normal, even with residual end-organ damage. Although cochlear involvement (hearing impairment) remains, it is difficult to detect on cursory examination. Therefore, for inner-ear decompression sickness, formal audiometry and vestibular tests (electronystagmography with rotary chair and caloric stimulation) are recommended at 4–6 weeks after injury.

Assessment of patent foramen ovale is often recommended for divers with severe or recurrent neurological decompression sickness.[29,141] For patent foramen ovale to be a precipitating factor for decompression sickness a substantial amount of venous gas emboli should be present, which is unlikely to happen in conservative depth-time profiles.[21,142] Patent foramen ovale or other right-to-left shunts can be detected by bubble contrast injection in conjunction with transcranial doppler, transoesophageal echocardiography, or transthoracic echocardiography. Because intravenous injection of bubbles in the presence of inert gas supersaturation could facilitate endogenous bubble growth, tests of this sort should be done only after completion of all hyperbaric treatments. Although transoesophageal echocardiography is generally the most sensitive test, the relevance of small shunts detectable only with this test is not known. Moreover, new transthoracic echocardiography techniques seem to have similar sensitivity.[143] Detection of a patent foramen ovale could warrant counselling about future diving, aimed at prevention of venous gas emboli.[21,142] Some divers who wish to return to unrestricted diving opt to have the patent foramen ovale repaired with a transvenous septal occluder device, but a careful discussion of risks and benefits is needed before this procedure can be recommended.[138,144,145] Patent foramen ovale is a common anomaly in the general population, and many individuals with decompression sickness do not have it. Therefore, a causal link between patent foramen ovale and an individual case of decompression sickness should not be ascribed.

Conclusions

Decompression illness occurs in a small population but is an international problem that few physicians are trained to recognise or manage. Although its manifestations are often mild, the potential for permanent injury exists in severe cases, especially if unrecognised or inadequately treated. Emergency medical personnel should be aware of manifestations of decompression illness in the setting of a patient with a history of recent diving or other exposure to substantial pressure change, and should contact an appropriate consultation service for advice.

Contributors

All authors contributed equally to the writing of this Review.

Conflicts of interest

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