In this article, principles of physics and physiology are described and applied to explain the commonly encountered medical problems associated with self-contained underwater breathing apparatus (SCUBA) diving.

**Background**

At sea level, our bodies support a column of air molecules approximately 8 km tall, extending to the upper reaches of the atmosphere. Under gravity, the mass of these molecules exerts a pressure called atmospheric pressure (1 ATA = 1 atmosphere absolute), which amounts to a weight of 10,000 kg m⁻², or 14.7 pounds per square inch. Sea water is approximately 800 times more dense than air at sea level and, under water, the pressure increases by 1 ATA for every 10 m below the surface. Hence, a diver at the surface will be subject to a pressure of 1 ATA due to the atmosphere. If he dives to 10, 20 or 30 m, the pressure on his body will be 2, 3, or 4 ATA, respectively.

**Dalton’s Law**

The partial pressure of a gas in a mixture is proportional to its percentage by volume in the mixture (John Dalton 1801, English chemist)

From Dalton’s Law, the partial pressure of a gas ($P_g$) is equal to the fractional concentration multiplied by ambient pressure (which is proportional to depth). $P_g$ is related to the number of molecules of gas per unit volume by the law of mass action. Hence, the physiological effects of gases under pressure are related to depth and also to the duration of exposure to the gas.

**Boyle’s Law**

At constant temperature, the volume of a gas varies inversely with the pressure (Robert Boyle 1667, English physicist)

There are four types of diving: (i) diving in a thick-walled vessel such as a submarine, bathyscaphe or rigid suit (vessel); (ii) breath-hold diving or snorkelling (breath hold); (iii) diving with compressed breathing gas supplied from the surface, e.g., a diving bell or hookah (surface air); and (iv) diving with compressed breathing gas via SCUBA carried by the diver.

Vessel divers are protected from the increasing ambient pressure by the thick-walled vessel; therefore, their lungs do not undergo any pressure or volume changes during the dive. Breath-hold divers commence their dive with lungs full of air at 1 ATA. As they ascend, the air in the lungs decreases in volume and increases in pressure to match the ambient pressure by Boyle’s Law. Surface air or SCUBA divers breathe compressed gas (usually air) at ambient pressure, which maintains the lungs at normal volume.

**SCUBA**

SCUBA was first developed by Rouquayrol and Denayrouze in 1865 and perfected by Cousteau and Gagnan in 1942. It is an open-circuit two-stage demand valve system; essentially similar to the Entonox apparatus. It supplies the diver with the gas mixture (usually air) at ambient pressure. Closed-circuit systems incorporating CO₂ absorbers are a relatively recent development, enabling greater economy of breathing gas.
**Decompression sickness**

**Pathophysiology**

**Henry's Law**

At constant temperature, the volume of gas going into solution in a given liquid is proportional to the partial pressure of the gas (William Henry 1803, English chemist)

Inert gas (usually nitrogen) becomes dissolved in tissues as a function of time and depth, as dictated by Dalton’s Law of partial pressures and Henry’s Law of gas solubility. For any given ambient pressure and gas mixture, the gas and tissue phases will eventually reach a state of equilibrium (saturation). Rapid ascent results in bubble nucleation and growth in the tissues and blood vessels, analogous to popping the cork on a bottle of champagne, where the sudden reduction in pressure results in bubble formation. Haldane modelled the process using exponential wash-in and wash-out curves and multiple tissue compartments with different half-times in a similar way to models of the uptake and distribution of volatile anaesthetic agents. From this model, decompression tables were calculated (and their algorithms subsequently incorporated into diving computers) which defined limits for the rate and profile of ascent for any given nitrogen load, so that nucleation would not occur and the gas could be safely dissipated without bubble formation. This is analogous to releasing the champagne cork in a slow, controlled manner, or making it slightly permeable, so that the champagne goes flat without any bubbles forming.

However, not all individuals and dive profiles follow the predictions of Haldane’s model and divers may occasionally develop decompression sickness (DCS) even if they keep within the table limits. This has led to a search for new physiological models which more accurately predict bubble formation.

The bubbles exert their damaging effects by mechanical occlusion of blood vessels and disruption of cells. In addition, the high surface tension and electrochemical forces generated at the gas–tissue interface cause disruption of soluble, cellular and endothelial proteins. Activation of mast cells, Hageman factor and complement, coagulation, fibrinolytic and kinin pathways occurs, resulting in a complex acute inflammatory response, which causes further tissue damage.

Depending on the site of bubble injury, DCS can affect the joints (‘the niggles’ or ‘the bends’), skin (mottled rash or itching), central nervous system (psychological changes, intellectual or visual impairment, paraesthesia, paralysis or cerebellar ataxia) and lungs (dyspnoea and haemoptysis). Symptoms may be florid or subtle. Onset may be immediate or delayed, though symptoms usually begin between minutes to 48 h after surfacing. Untreated, the condition may be progressive or relapsing and remitting. Any neurological symptoms within 2 weeks of compressed air diving should be assumed to be DCS until proven otherwise.

Factors pre-disposing to the development of DCS include dehydration (often in association with prior consumption of alcohol), cold, fatigue, obesity and repetitive or ‘reverse-profile’ dives, i.e. maximum depth is reached near the end, rather than the beginning.

**Historical background**

DCS was first described in 1667 by Robert Boyle who observed the formation of bubbles in the aqueous humor of a viper which had been subject to decompression in an evacuated bell jar. DCS was observed in bridge construction workers by Triger in 1841 and Smith in 1873. These men worked in pressurised chambers or caissons, designed to prevent flooding of the foundations. The condition became known as caisson disease. The severe joint pains resulted in a contorted pose that resembled ‘the Grecian bend’, a stooped posture affected by wealthy Victorian ladies. Hence, its alternative epithet of ‘the bends’.

**Management of decompression sickness**

Immediate first aid involves airway management and the administration of 100% oxygen via a face mask and reservoir bag. The patient should be kept in the horizontal or recovery position. Intravenous crystalloid should be given to offset hypovolaemia caused by increased capillary permeability and the effects of immersion- and cold-diuresis. The wet-suit should be removed and passive rewarming commenced to prevent hypothermia. The definitive treatment is recompression and early discussion with and evacuation to the nearest recompression facility is essential. It is important to avoid ascent to high altitude during evacuation as this will exacerbate bubble growth. Flights should be below 500 m unless the cabin is pressurized to sea level and roads over mountains should be avoided.

The patient is recompressed in a chamber to a pressure corresponding to the maximum depth of the dive or until symptoms are relieved. The pressure is then gradually returned to sea-level in a controlled manner over the course of hours or days, as defined by a series of treatment tables. The environment inside the chamber is air (pure oxygen is an unacceptable fire risk) but the patient breathes 100% oxygen at ambient pressure via an aviator face mask, hood or specially modified mechanical ventilator. Periods of breathing oxygen are alternated with periods of breathing air to reduce the risk of acute CNS oxygen toxicity (see below). Recompression causes reduction in bubble volume (Boyle’s Law) and re-dissolves the inert gas in the tissues (Henry’s Law). The high inspired oxygen concentration increases oxygen delivery to ischaemic tissues, provides a low inspired...
nitrogen concentration which facilitates inert gas wash-out, and may have beneficial anti-inflammatory and cerebral vasoconstrictor effects. A number of adjunctive therapies including lidocaine, steroids, non-steroidal anti-inflammatory drugs and heparin are currently being assessed. As the chamber pressure increases, endotracheal tube cuffs may become compressed and leak unless filled with fluid. Intravenous fluids should be carefully monitored to prevent rapid uncontrolled administration.

At sea-level (1 ATA), our tissues are saturated with a certain concentration of nitrogen. In fact, we are all ‘saturation divers’. If ambient pressure is suddenly reduced, for example by failure of an aircraft canopy at altitude, bubble formation will occur in exactly the same way as in a diver, causing ‘altitude DCS’. The symptoms and management are as above.

**Pulmonary over-pressurisation syndrome and cerebral arterial gas embolism**

If compressed gas is breathed at depth and the diver ascends, the gas in the lungs will expand as the ambient pressure falls (Boyle’s law). If gas is unable to escape due to breath holding or bronchospasm, the increase in volume and pressure may cause the lungs to rupture. Gas then escapes into the pleural cavity (pneumothorax), mediastinum and soft tissues of the neck (surgical emphysema), pericardial cavity (pneumopericardium) and pulmonary arterioles (arterial gas embolism). Pulmonary over-pressurisation syndrome (POPS) is the term that describes this sequence of pulmonary overinflation, rupture and escape of gas from the lung.

Most divers ascend head first and the buoyancy of any intra-arterial bubbles causes them to pass cephalad via the carotid arteries. The middle cerebral arteries lie directly opposite the insertion of the common carotid arteries and supply the pre- and post-central cortices which are responsible for somatic motor and sensory function. These areas are, therefore, particularly vulnerable to injury.

The history is usually that of an inexperienced diver who panics or loses control of buoyancy and makes a rapid uncontrolled breath-hold ascent. On surfacing, there is often an audible grunt as air is forced through the glottis from the hyperinflated lungs, followed within seconds or minutes by the onset of profound persistent neurological symptoms, resembling a cerebrovascular accident. Features include paralysis, coma and convulsions, sometimes accompanied by cardiovascular instability or cardiac arrest. POPS and cerebral arterial gas embolism (CAGE) also occur in submarine escape trainees and in breath-hold divers who take a lung full of air from the regulator of a SCUBA diver or submerged air pocket at depth, then ascend without exhalation.

CAGE may also occur when bubbles of venous or tissue origin enter the arterial circulation via a right-left anatomical shunt (paradoxical embolism). Approximately 25% of the population have a probe-patent foramen ovale. If a diver performs a Valsalva manoeuvre to clear the ears or strains to lift a heavy object, the resulting increase in right atrial pressure may cause this vestigial channel to open and embolisation of bubbles into the arterial circulation may result.

It is difficult to distinguish clinically between CAGE and DCS. The two pathologies may co-exist and management is similar, i.e. early recompression and hyperbaric oxygen. Consequently, a new unified diagnosis of acute neurological decompression illness (DCI) has been proposed.

SCUBA divers undertake physical exertion whilst breathing cold dry air from the breathing apparatus and occasionally inhale sea water (hypertonic saline). All of these are potent stimuli for bronchospasm in susceptible individuals. Because of the risks of air trapping at depth with resulting POPS and CAGE, it is generally recommended that asthmatics should not dive. The notion that shallow dives are safer is fallacious in this context, as the greatest proportional change in ambient pressure (and hence lung volume) occurs between the last few metres depth and the surface.

**Oxygen toxicity**

Prolonged exposure to moderate partial pressures of oxygen at sea level \((P_{O_2} > 0.5 \text{ ATA})\) may cause injury to lungs (Lorraine-Smith effect) and retrolental fibroplasia in neonates. However, exposure to high partial pressures of oxygen \((P_{O_2} > 1.2 \text{ ATA}, \ i.e. > 50 \text{ m depth breathing air or > 12 m breathing 100% } O_2)\) may cause acute CNS toxicity (Paul Bert effect). The risk of toxicity depends on the dose, i.e. depth and duration of exposure. However, there is a great range of inter- and intra-personal variability. The symptoms include mood changes, facial twitching, visual and auditory hallucinations, cardiovascular instability, syncope and convulsions. They may occur suddenly and without warning. The mnemonic ‘VENTID’ (vision, ears, nausea, twitching, irritability, dizziness) is commonly used by divers to remain alert to the possible onset of symptoms. If symptoms occur in a chamber during recompression therapy, they may be treated by changing the breathing mix to air and administering benzodiazepines. However, syncope or convulsions under water may be fatal. Hence, although technical divers use cylinders of 100% oxygen to facilitate off-gassing of nitrogen during decompression, it is generally unsuitable as a main breathing gas for all but the most shallow of dives. Susceptibility to CNS oxygen toxicity is increased by hypercarbia (e.g. physical exercise or shivering), catecholamines (e.g. anxiety), steroids and ‘dry’ (chamber)
rather than ‘wet’ (underwater) dives. The mechanism is thought to involve free radical damage, inhibition of the Na+/K+ pump and activation of GABA receptors.

**Inert gas narcosis**

At high partial pressures, inert gases reduce nerve conduction velocity and act like volatile anaesthetic agents. The symptoms are pressure (depth) related and resemble the effects of nitrous oxide inhalation, *i.e.* progression from euphoria to drunkenness, confusion and unconsciousness. Dysphoria and paranoia leading to panic may occur in some subjects. The onset is insidious and results in irrational behaviour, impaired judgement or a false sense of security, which may be fatal at depth. The effects are known colloquially as ‘the narks’ or ‘rupture of the depths’; they are rapidly and spontaneously reversible on ascending.

There is individual variation in susceptibility. However, for nitrogen, symptoms begin at a PN2 of around 3.6 ATA (*i.e.* 35 m for a diver breathing air). Divers quote ‘Martini’s Law’ – each additional 15 m depth on air (PN2 1.2 ATA) is the equivalent to the intoxication of one Martini! The narcotic effects of nitrogen are potentiated by the action of sedative drugs (*e.g.* alcohol and benzodiazepines) and the antimalarial drug mefloquine.

Less narcotic inert gases may be substituted for nitrogen in the breathing mix to enable the diver to go deeper without impairment of judgement. This is the rationale for using helium (which has one-quarter the narcotic potential of nitrogen) as an additive on deep dives in the form of Heliox (He:O2) or Trimix (He:N2:O2). The low density of helium also reduces the work of breathing. However, it is expensive, causes voice distortion rendering communication difficult and may cause an excitatory effect on the nervous system (high pressure nervous syndrome) at depths > 200 m.

**Non-pulmonary barotrauma**

The effects of pressure on the lungs due to Boyle’s Law have been described above but similar effects may occur in any gas-filled cavities in the body or equipment. If pressure in the cavity cannot be equalised on descent, compression occurs (‘squeeze’). If pressurised gas enters the cavity at depth but is unable to escape during ascent, expansion occurs (‘reverse squeeze’). Both may result in tissue disruption and injury.

The most common site of non-pulmonary barotrauma is the tympanic membrane. On descent, increasing water pressure via the external auditory meatus forces the membrane inwards. This may be balanced by the pressure of the gas in the breathing mix via the Eustachian tube. Divers maintain patency of the Eustachian tubes by performing a Valsalva or Frenzel manoeuvre during descent.

However, if they are unable to do so due to inflammation of the Eustachian tube (*e.g.* upper respiratory tract infection), the tympanic membrane is forced inwards and may sustain bruising, haemorrhage or rupture. The sudden ingress of cold water may cause convection currents in the semicircular canals with subsequent vertigo and disorientation. Divers who self-medicate with decongestants may find that the effects wear off during a dive, trapping a pocket of compressed gas in the middle ear, which can expand on ascent, causing the tympanic membrane to bulge outwards and rupture. Ear plugs should never be worn by divers because they create enclosed gas-filled cavities between the plug and the ear drum which cannot be equalised. A tight-fitting wet-suit hood may also create the same conditions.

Gas cavities are also found in the facial sinuses, air space inside the face mask, between the diver’s wet-suit and skin, gastrointestinal tract and within carious teeth. All are potential sites of barotrauma and may result in severe sinus pain, subconjunctival haemorrhage, skin weals, abdominal pain, gastric rupture and painful or exploding teeth.

An extreme form of ‘squeeze’ occasionally occurred in ‘hard-hat’ divers, when the pressurised gas supply to the helmet failed at depth. The greater pressure of the surrounding water forced the diver’s whole body upwards into the rigid helmet.

**Conclusions**

The greatest dangers to divers are panic and failure to follow sensible diving practices. Recreational diving is generally a safe pastime and few experiences can compare with the discovery of the sublimely beautiful ‘silent world’.

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**Key references**


See multiple choice questions 97–99.