Chapter 4
The Underwater World and Diving Physiology

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4.1 History of Diving and Underwater Medicine

The underwater world can be considered as one of the first extreme environments human kind explored. Diving has been utilized in multiple ways for domestic, commercial and military purposes. Recreational diving, especially using scuba (Self-Contained-Underwater-Breathing-Apparatus) became popular in the second half of the twentieth Century. Nowadays, ancient breath-hold diving practices, primarily developed for gathering food and valuable commodities, are slowly giving way to compressed gas diving, and recreational diving has developed into a variety of different modalities. The comprehensive history of diving is far too complex (and fascinating) to effectively summarize in the few pages available; however, the following are a few key events in the development of diving equipment and the field of diving medicine.

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4.1.1 Diving in Ancient Times

The earliest form of diving was breath-hold diving. Archaeological evidence suggests breath-hold dives were used to harvest sponges, food, pearls and corals. These domestic purposes were soon followed by military and salvage efforts [1]. In 900 BC breath-hold divers were used in the Trojan wars to sabotage and attack enemy ships, and Alexander the Great used divers for underwater demolition during the Siege of Tyre around 332 BC [1, 2]. It was said that Alexander the Great descended in a diving bell himself; however, details are sparse and some of the stories sound fanciful.

Many tracts and diagrams describing diving equipment and techniques have survived from ancient and mediaeval times. Many are attributed to famous historical figures such as the Greek philosopher Aristotle in the fourth century BC and the Italian polymath, Leonardo da Vinci. Although it is not known if these sketches were ever realized and developed, they attest the desire to explore the underwater world and remain submersed for an extended time.

4.1.2 Surface Supply Diving

In 1690, Edmond Halley designed his first diving bell with air supplied at atmospheric pressure. He subsequently improved his design in 1719, by cleverly providing air to the bell from two barrels suspended below the bell (and therefore at higher pressure than the bell). Divers used this bell to depths of 18 msw and apparently stayed submerged for up to 1.5 hours [3, 4].

In the 1700s the development of compressors allowed a shift from underwater to surface compressed air supply. This also enabled the development of helmet-hose diving apparatus. Surface-supplied compressed air diving became the prevailing commercial diving technique, and this remains the case up to the present day in many industries [5, 6].

The effectiveness of these new techniques was demonstrated in the salvage operation of the H.M.S. Royal George in 1832. A diving helmet was perfected by Augustus Siebe. The Siebe helmet and closed dress were the common diving equipment for commercial divers [1]. Siebe's diving proved to be so successful, that it established the predominant compressed air surface supply technique in use to this day. The present US Navy Mark V Deep-Sea Diving outfit is only a modernized iteration of Siebe's initial design.

4.1.3 Modern Diving

In 1800 with the then state-of-the-art diving equipment, divers were able to reach depths of 50 m for an extended period. With the venture into depths beyond 30 m divers started to experience the mysterious joint and neurological effects that have become known as decompression sickness (DCS), which caused significant injury and death to many divers.

While diving gear kept developing, the effects of compressed air breathing in environments with elevated ambient pressure also received attention in different setting. During the Brooklyn Bridge project, from 1870–1873, there were 110 cases of DCS reported among the 600 workers employed. Andrew Smith, the physician in charge was the first to use to term "caisson sickness."

In 1878, Paul Bert published his work "La Pression Barometrique" [7], which is considered the founding document of aeronautical medicine. He described theories for both, hyperbaric and hypobaric exposures and their practical applications. His descriptions included that mountain sickness was due to hypoxia and to survive at extreme altitude, oxygen must be breathed, and that higher pressures would require the breathing of lower pressures of oxygen to sustain life. He also described the toxic effects of hyperbaric oxygen in animals, which is now known as acute oxygen toxicity. Bert discovered that rapid decompression can cause nitrogen bubbles to form in tissue and blood. These fundamental findings were the first steps in research towards the understanding of DCS.

It was not until the twentieth century that J.S. Haldane and colleagues [47] derived satisfactory mathematical concepts to describe and design of countermeasures for the physiological problems associated with DCS (1907). The mathematical model reflected the behaviour of inert gas inside the human body and was the initial step towards the development of safe decompression tables. The first tables were designed employing the assumption that DCS could be avoided by not exceeding a 2:1 pressure gradient between tissues and breathing gas during ascent. Although later studies revealed the details of Haldane's model were not entirely correct, his tables had a significant impact preventing many divers from developing DCS—a condition colloquially called 'the bends'.

In the twenty-first century, diving medicine and technology research have together contributed to better addressing the physiological issues and minimize the risk of diving injury. Advances in diving equipment, the development of both large-scale manufacturing and standardized training organizations, have allowed the popularization of diving such that this environment is now accessible to the average man. The design of a proper demand-regulated air supply from compressed air cylinders by Cousteau and Gagnan in 1943 was the founding step towards the scuba equipment as we know today.

4.1.4 Diving Modalities

The following are some of the commonly practiced advanced diving modalities.

Rebreather Diving
The purpose of a rebreather diving set is to recycle a diver's exhaled gas so it can be re-used safely during a dive. Rebreathers may be semi- or fully-closed systems and are increasingly used for recreational, scientific, and technical diving because of both the ability to allow extended time underwater and increased depth capability [8–10].

The traditional scuba set is a fully open-circuit system where no rebreathing occurs. Exhaled gas is simply vented to the surround water. In contrast, while details vary, a rebreather collects exhaled gas in an expandable container (counterlung), passes it through a device to absorb carbon dioxide (scrubber), and replenishes oxygen to the desired concentration before being available for the subsequent inhalation. Thus, the breathing loop is more complex than a simple second stage diving regulator [8, 9].

Mixed-Gas Diving
Often combined with the use of a rebreather, divers employ breathing gases other than air in order to venture to greater depths. Deep diving brings with it a number of physiologic challenges that require close attention to the partial pressures of inhaled gases. Nitrogen (80% of normal air) becomes increasingly narcotic above a partial pressure of about 3.5 atmospheres, and oxygen increasingly toxic at partial pressures above about 1.6 atmospheres. These problems can be addressed by the addition of a diluent in the form of a non-narcotic inert gas (most commonly helium) but involved careful attention to ensure appropriate gases are used throughout a dive—including descent and ascent. Planning the use of 'mixed-gas' diving is complex and potentially hazardous.

The most common mixture is 'Trimax', a combination of oxygen, helium and nitrogen. Mixed-gas diving techniques were developed from commercial and military operations but are increasingly popular in 'technical' recreational diving.

Saturation Diving
When scientific or commercial purposes dictate that long periods (days to weeks) be spent at depth, saturation diving is the obvious solution. It is often complicated, expensive and time consuming to repeatedly dive and resurface while conducting a long-term task—for example, a scientific survey or a construction project. Saturation diving avoids these inefficient repetitive dives and exploits the fact that after a diver is fully saturated with the breathing gas in use at depth, then no further decompression time is required—regardless of the length of time under pressure.

Saturation operations involve the use of a habitat where the divers live between working tasks. Such habitats may be actually compressed vessels placed on board a ship, or submerged at or close to the working depth. Saturation may be undertaken at quite modest depths and yet save many hours of decompression time over the

course of a project. Such diver require long terminal saturations for each diver and take many days if the saturation is at significant depth.

Today, saturation divers are conducted between 30 m and 300 m. A dive to 300 m would take approximately eleven days or more of decompression. Such extensive decompression time required to ascent to the surface is very expensive and a great logistical challenge.

4.2 Diving Physics—The Underwater Environment

4.2.1 Introduction

All matter is composed of atoms or bonded groups of atoms called molecules. These atoms or molecules are in constant motion oscillating around a mean position but continually rebounding off each other in all directions. Each atom and molecule interacts at a close range with all others through weak forces (either attractive or repulsive) known as Van der Waal forces [11]. Van der Waal forces are responsible for much of the observed behaviour of matter including the liquefaction of gases below a certain temperature and the ability of flies to crawl across the ceiling. They are due to fluctuating polarizations as a consequence of quantum dynamics and are distinct from the more powerful forces of covalent or ionic bonding.

Covalent bonding (also called molecular bonding) is a chemical bond that results from the sharing of a pair of electrons between atoms and often results in a more stable configuration—for example the formation of hydrogen (H_2) from two single hydrogen atoms. Ionic bonding similarly involves the sharing of electrons but involves electrostatic attraction between oppositely charged ions—for example common salt is formed from sodium ions (Na^+) and chloride ions (Cl^-) to form NaCl, which has no net electrostatic charge.

The complex interaction between these forces, heat (often expressed as the temperature T) and pressure P are responsible for the manifestation of the three states of matter: solid, liquid and gas. A solid consists of molecules packed together in a lattice construction. If heat is added, the molecules begin to move with greater amplitude and take up more space, thereby weakening the forces between them. At a high enough temperature the forces are no longer strong enough to hold the molecules in the lattice and the solid breaks down to a less structured state usually called liquid. If the temperature is further increased, each molecule gains more kinetic energy and, above a certain temperature, enters the gaseous state. Higher pressures will increase the temperature required for the transformation from solid to liquid and liquid to gas for any substance. Many of the interactions between these elements are described by a series of ‘Laws’, a number of which are highly pertinent to divers.

Table 4.1 Common conversion of pressure units

	ATM	ATA	Bar	mmHg	mmH_2O
ATM	1	1.033	1.013	760	10332.56
ATA	0.968	1	0.981	735.56	10000.3
Bar	0.987	1.02	1	750	10197.44
mmHg	$1.316 \cdot 10^{-3}$	$1.36 \cdot 10^{-3}$	$1.33 \cdot 10^{-3}$	1	13.6
mmH_2O	$9.67 \cdot 10^{-5}$	$9.89 \cdot 10^{-5}$	$9.81 \cdot 10^{-5}$	0.0736	1

Pressure

Pressure P is the most important environmental quantity that a diver is exposed to and is defined as a force F acting on a unit area A:

$$P = \frac{F}{A}. \tag{4.1}$$

Pressure is a highly relevant concept in underwater exposure and diving medicine and can be presented in a potentially confusing myriad of units. Throughout this chapter pressures are presented as atmospheres absolute (ATA) or kilopascals (kPa) depending on the context. For conversion to all commonly used pressure units see Table 4.1.

A depth of approximately 10 msw (metre of sea water) or 33 fsw (feet of sea water) equal to one atmosphere, or one bar or 101.3 kPa (kilo Pascal) of pressure. The ambient pressure acting on a submerged person is the addition of two components: the pressure of a water column and the atmospheric pressure under which the water column lies. Water pressure is the product of water depth and density, while the atmospheric pressure is the pressure exerted by the earth's gaseous atmosphere at the water surface level. Often we accept the air pressure as the average at global sea level, for which we use the term '1 atmosphere absolute' (1 ATA or 101.3 kPa). The density of water is one kilogram per cubic metre ($1\ \frac{kg}{m^3}$), so that at the depth of 10m, the pressure exerted by the water column is $10\,m \cdot 1\ \frac{kg}{m^3}$ $(= 100\,kPa)$. Thus, at 10 m depth, a diver is subject to an ambient pressure of 201.3 kPa—a pressure very closely equivalent to 2 ATA (202.6 kPa).

4.2.2 The Gas Laws

The relationships between pressure, volume and temperature of gases are particularly relevant to divers, so much so that many important practical diving issues rely on an understanding of the consequences of these relationships. They are summarized in a series of 'Gas Laws' outlined here.

Boyle's Law
The relationship between pressure and volume for a given mass of gas at constant temperature.

If the mass of a gas and its temperature are kept constant, the gas volume is inversely proportional to the acting pressure. Boyle's law can symbolically be formulated, denoting initial pressure and volume P and V and submersed pressure, P_{sub} and P_{sub}, respectively:

$$P_i\, V_i = P_{sub}\, V_{sub} = const. \tag{4.2}$$

Charles's Law
The relationship between the volume and temperature, for a given mass of gas at a constant pressure.

If the mass of gas is not changed and pressure is kept constant, then the gas volume will vary linearly with temperature

$$\frac{V_i}{T_i} = \frac{V_{sub}}{T_{sub}} = const. \tag{4.3}$$

Ideal Gas Law
'Ideal gas' is a theoretical gas composed of many randomly moving point particles that are not subject to interparticle interactions. For practical purposes the common gases used in diving can be assumed to be 'ideal'.

The ideal gas law reflects a combination of Boyle's and Charles's Laws and relates to the measurable quantities pressure, volume and temperature. Considering changes in ambient pressure, gases will contract or grow in their volume and can be described with the *Ideal Gas Law*. The law states that the product of absolute gas pressure, P and volume,V must equal the product of the mole fractions, n, the Avogadro's gas constant, R and the absolute temperature T,

$$P\, V = n\, R\, T \tag{4.4}$$

It is the most important law regarding gas systems underwater. For the sake of completeness, it should be noted that at very high pressures and low temperatures, gases no longer obey these gas laws—they can no longer be regarded as 'ideal'. No current efforts of human exposure are anywhere near this threshold.

Dalton's Law
The contribution to total pressure for each gas in a mixture of gases.

Diving gases are rarely composed of a single gas, but rather are a combination of different gases. Each gas exerts a partial pressure that contributes to the total pressure of the gas mixture. Each partial pressure is the pressure that the gas would exert if it occupied the space by itself. The total pressure exerted by a mixture of gases P_{abs} is equal to the sum of the partial pressure of each of the component gases. Put another way, if a mixed gas is composed of three different gases, the

total pressure of the gas will equal the pressure of each of the individual gases added together. The atmosphere at sea level is an example, being composed of 21% oxygen, 78% nitrogen and 1% 'other gases' (principally the noble gases). Thus:

$$P_{abs} = 0.21 \cdot P_{O_2} + 0.78 \cdot P_{N_2} + 0.01 \cdot P_{noble} = 1\,ATA. \tag{4.5}$$

Henry's Law

The relationship between the partial pressure of a gas in contact with a liquid and the amount of that gas dissolved in the liquid.

Whenever a gas is in contact with a liquid, a portion of the gas molecules will dissolve in the liquid. Different gases will dissolve to a greater or lesser extent—each has a unique 'solubility' in every liquid. This factor of solubility is important for underwater exposure and diving because significant amounts of gases are dissolved in body fluids and tissues during exposure to increased pressure while diving. As the pressure increases so does the amount of gas dissolved in a liquid. The relationship between the amount of dissolved gas and pressure is called Henry's Law, which states that the volume of gas dissolved in a liquid is equal to the product of Henry's constant k and the partial pressure of the gas P_{gas}.

$$\frac{P_{gas}}{k_h} = c_{gas}. \tag{4.6}$$

It follows that if the gas partial pressure is doubled, then the amount of gas in solution at equilibrium will double, and conversely, if the gas partial pressure is halved then half of the gas will leave the solution. Some gases are more soluble than others and some liquids are better solvents than others. For example, while nitrogen is five times more soluble in fat than water, helium is not very soluble in fat (this may be relevant in regard to the narcotic effect of Nitrogen but not Helium—see below).

Fick's Law

The relationship of a flux of particles along a concentration gradient.

Gases dissolved in liquids move randomly throughout the liquid in a thermodynamic process and this results in an even distribution of dissolved gas throughout the liquid in a process described as diffusion. When dissolved gas is introduced into a liquid at any particular point, the pressure will be equilibrated throughout the liquid. Another way of describing this phenomenon is that diffusion is the random movement of particles from a region of higher concentration to a region of a lower concentration.

Fick's first law describes that a flux density j is linearly proportional to a concentration gradient $\frac{\partial C}{\partial x}$ with the diffusion constant D:

$$j_x = -D \cdot \frac{\partial C}{\partial x}. \tag{4.7}$$

The flux density j relates to a rate of change of a quantity (e.g. gas particles) with position, describing a transport of the quantity [12]. Hence, a concentration gradient causes a diffusion of particles.

In diving scenarios, increased ambient pressures translate to varying partial gas pressures, creating gradients across anatomical barriers within the body. While the diffusion rates of a gas within a continuous body of liquid is constant, the presence of a barrier within the liquid substantially affect the diffusion rate of the gas. Consequently, it is important to understand the physical laws that govern diffusion of dissolved gas across membranes as they heavily inform the gas exchange process of bubble growth. Fick's law describes the rate at which a dissolved gas diffuses across a barrier given certain properties of the barrier and the gas.

$$\frac{dV}{dt} = D \cdot A \cdot \frac{\Delta P}{d}. \tag{4.8}$$

The equation states that the rate of diffusion $\frac{dV}{dt}$, indicated by volume changes over a period of time, across permeable barrier is determined by the chemical nature of the membrane itself , the surface area of the barrier A, the partial pressure gradient ΔP of the gas across the barrier, and the thickness of the barrier d.

4.3 Diving Medicine

4.3.1 Diving Physiology

The human body is well-adapted to the terrestrial environment, performing best at close to one ATA while breathing air in a gaseous environment. The principal challenges during diving relate to both changing pressure and immersion. Apart from the obvious challenges in successfully supplying the body with oxygen and eliminating carbon dioxide whilst both immersed and dealing with a grossly increased ambient pressure, the much greater density of water compared to air means the body can be uniquely subject to a significant pressure gradient if upright in the water column. Unsurprisingly, significant physiological changes occur when the body is immersed and subjected to increased pressure.

4.3.2 Physiology of Immersion

4.3.2.1 The Diving Reflex

Diving mammals are highly adapted for immersion. One of the most prominent changes on immersion is the rapid onset of a number of physiological changes that extend the period during which oxygen can be supplied to vital organs—

together these are often called the *diving reflex*. These changes include a pronounced reduction in heart rate (bradycardia) and a selective regional constriction in arteries that produce a 'selective ischaemia' resulting in preferential perfusion of oxygen sensitive organs.

Humans retain a vestigial diving reflex, particularly on exposure to cold water on the face [13]. Initially there is a restriction of blood flow to the peripheries (peripheral arterial vasoconstriction) and a reflex bradycardia, probably via a trigeminal nerve–brainstem reflex arc. Efferent parasympathetic pathways mediate bradycardia and efferent sympathetic pathways mediate peripheral vasoconstriction. Similar responses have been noted during neurosurgical procedures close to the trigeminal nerve and have been called the *trigeminocardiac reflex* (TCR).

While there are similarities between the TCR and the diving reflex, the latter is characterized by a degree of hypertension, while in contrast, hypotension is a feature of TCR [16]. The difference may well reflect the prominent venoconstriction secondary to hydrostatic forces on immersion, reinforced by cooling peripheries and resulting in a shift of blood into the central veins and pulmonary circulation [14]. The net result of increased venous return, increased stroke volume and the reflex bradycardia is a modest increase in both cardiac output and arterial pressure.

The increased venous return also stretches the atria, stimulating atrial receptors to release atrial natriuretic factors (ANF) and an associated decrease of anti-diuretic hormone (ADH), resulting in unopposed diuresis [15].

4.3.2.2 Respiratory Effects

Immersion also impacts respiratory mechanical factors [15]. When the diver is upright (particularly with head out of the water), the chest is exposed to a significantly higher pressure than the mouth. At rest, the pressure in the alveoli will be equal to the ambient pressure underwater (1 ATA + water column pressure), while the mouth will be at 1 ATA—encouraging egress of gas from the lung. On inspiration, a great relative negative pressure will need to be generated at the mouth in order to achieve gas flow into the lungs. The work of breathing will increase and the central shift of blood into the thoracic cavity will be enhanced, reinforcing the haemodynamic changes already described by Camporesi [15], Fig. 4.1.

4.3.2.3 Thermal Considerations

Immersion predisposes the diver to hypothermia because of the high thermal conductivity of water compared to air. Hence, except for relatively short, shallow exposures in warm tropical waters, divers need to wear some form of thermal protection. Even mild degrees of hypothermia may threaten the diver through impairment of psychomotor ability.

When immersed, most heat is lost through conduction and convection. Heat is exchanged through the skin into the water immediately surrounding the body and

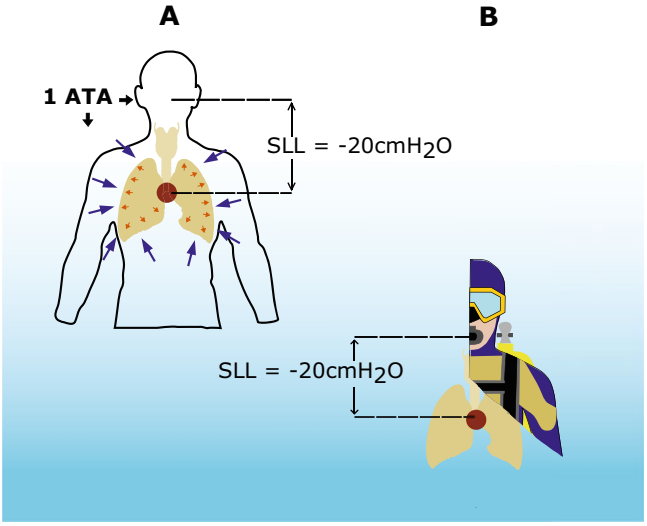

Fig. 4.1 Respiratory consequences of immersion for an individual breathing with head out of water (Panel A) and a scuba diver upright in the water (Panel B). **Panel A**: The distance between the water surface and the centroid of the lung is 20 cm, and a pressure equivalent to that depth is exerted on the thorax and all structures contained in it. This pressure exerted by the water column is $0.2\,m \cdot 1\,kg/m^3 = 1.96\,kPa$. Typically, a quiet inspiration requires a negative pressure of around 5 cm water (0.49 kPa). Immersion requires the individual to increase the negative pressure required to inspire by a factor of five. **Panel B**: A scuba diver upright in the water has a regulator that equilizes the breathing air pressure to that at the mouth. The consequences are the same as Panel A [17]

subsequently carried away through convection. The rate of heat transfer will depend on a number of factors including skin blood flow, the nature of any protection worn and the rapidity of movement of water over the body surface, but primarily depends on the physical characteristics of thermal conductivity and specific heat capacity. Thermal conductivity is a measure of the ability of a substance to transfer heat and is measured as the amount of heat transferred per unit time and surface area, divided by the temperature gradient. For example, at 20°C, the thermal conductivity of water is about $0.59\frac{W}{m \cdot K}$. Specific heat capacity is the amount of energy (joules in the form of heat) that must be added to a standard mass of a substance (1 kg) in order to cause an increase of one-degree Kelvin (1 K). For water at a temperature of 25°C, the energy needed to increase one kilogram of water by 1 K is 4,179.6 joules, meaning that the specific heat of water is $4179.6\,\frac{J}{kg \cdot K}$. Heat is lost much more readily in water than in air because water has a thermal conductivity approximately 23 times that of air and a specific heat about 3500 times that of air.

These differences between air and water mean the thermoneutral range (where an unclothed human can maintain core temperature without an increase in metabolic rate) in water is very narrow (around 34° to 35°) compared to that of air (from about 21° to 30°) [18].

4.3.3 Physiology of Exposure to High Pressure

4.3.3.1 Respiratory Effects

Gases are composed of molecules in constant random motion and separated from each other by relatively large distances. As the pressure is increased the molecules are pushed closer together and the mass of matter per unit volume is increased. We express this increase in matter per volume as density. The density of any breathing gases will be increased in a direct proportional relationship with ambient pressure, such that doubling the ambient pressure will double the density of any single gas if the temperature is held constant Fig. 4.2.

Increased density requires an increase in the work of breathing in order to move the same volume of gas. The practical implications are that the maximum breathing capacity (MBC) is reduced at higher pressures and ultimately the oxygen consumption required to 'shift gas' exceeds the oxygen supplied to the alveoli from that effort. Under those conditions, the increased work of breathing is the limiting factor for survivable depth exposure. The drop in MBC is about 50% at the relatively modest depth of 30 metres of seawater (msw)—4 ATA ambient pressure [15]. These density related problems can be partially ameliorated by good design of breathing apparatus and the use of gases with low density to replace the nitrogen in air (density at 1 ATA = 1.2 $\frac{kg}{m^3}$). Gases in common use to assist with deeper diving include helium (density at 15°C and 1 ATA = 0.08 $\frac{kg}{m^3}$).

Breathing gases at higher densities and where there is a column of water pressure to overcome both contribute to increasing the relative negative pressure within the chest compared to the mouth. This negative pressure may produce problems at the level of the alveolar capillaries through transudation of fluid from capillaries into the alveolar space.

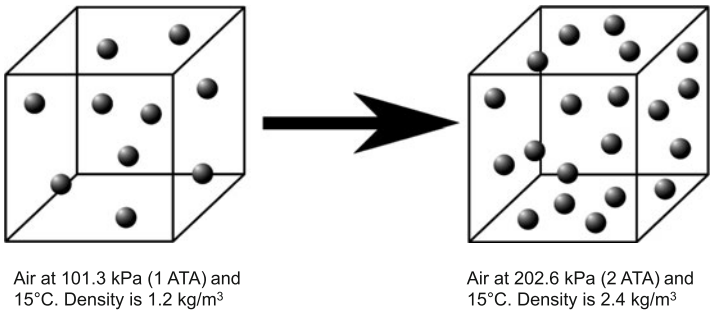

Fig. 4.2 Density and pressure. As ambient pressure increases, so does the density. Here a doubling of pressure from one to two atmospheres (surface to approximately 10 metres of seawater) also doubles the density—there are twice as many molecules in the same volume. Air is of course a mixture of gases but represented here as uniform molecules

The increased work of breathing may also have consequences related to the control of breathing. Under normal conditions, the main determinant of minute ventilation is tight control of arterial carbon dioxide tension ($PaCO_2$) through pH-sensitive chemoreceptors in the carotid bodies. Increased CO_2 production (due to any cause of increased metabolic rate) will rapidly result in an increase in ventilation, thus bringing the $PaCO_2$ back to normal. Neural pathways also have role specifically relating to exercise where ventilation increases almost immediately on commencement of exercise [19]. When the work of breathing increases, more CO_2 will be produced resulting in both increased ventilation and a further increase in $PaCO_2$. Eventually, a new level of ventilation will be found. If the ventilation required is excessive for the individual concerned, the result will be dyspnea (breathlessness) and the diver will be unable to continue the current level of activity for a sustained period. At the extreme, $PaCO_2$ may climb rapidly as the diver is unable to sustain the ventilation required, leading to increasing respiratory distress and eventual unconsciousness from hypercarbia. This sequence of events is a direct result of increased gas density and have been implicated in diving fatalities [20].

Some individuals seem more tolerant of rising $PaCO_2$, exhibiting a 'blunted' ventilatory response. It is not clear if this is a form of acclimatization in experienced divers, an innate variation or a combination of these factors. For whatever reason these 'carbon dioxide retainers' are over-represented in the diving population and may represent a danger to themselves and others if impaired by rising $PaCO_2$ and by increased susceptibility to oxygen toxic seizures or enhancement of nitrogen narcosis (see Sect. 4.3.4.1) [15]. Screening for these individuals in commercial and military diving operations has been advocated by Elliott and others [21].

Overall, immersion and breathing compressed gases tend to push divers towards hypercapnia. In addition to the mechanisms discussed above, there is some evidence that immersion causes the respiratory centre to exhibit a reduced responsiveness to hypercapnia and that this tendency may be exacerbated by inert gas narcosis (usually nitrogen) [22]. Many scuba divers have also learned the technique of reducing their minute ventilation deliberately in order to conserve a limited supply of breathing gas at the cost of tolerating a higher than normal PCO_2. The most common technique involves deliberately long pauses between inhalation and exhalation and is often referred to as 'skip breathing'. Higher $PaCO_2$ may increase the risk of central nervous system oxygen toxicity through cerebral vasodilatation and many of these divers complain of headaches after diving from the same mechanism.

4.3.3.2 Barotrauma

All places in the body where gas exists will be subject to Boyle's Law. This includes the middle ear and sinuses, lungs, gut and potentially any carious teeth. All these sites are potentially at risk of damage as a direct result of changes in pressure and therefore of volume in an enclosed space. The general term for any injury relating to the expansion or contraction of volume (or the increase or decrease in pressure)

is *barotrauma*, although among divers these problems are often referred to as a 'squeeze'—such as 'ear squeeze'.

Middle Ear and Sinus Barotrauma

Most barotrauma is associated with the exposure to increasing ambient pressure (on descent). The most common example is the middle ear (middle ear barotrauma—MEBT). In a normal individual, the middle ear is an air-filled space between the tympanic membrane (eardrum) and the skull including two membranous parts called the round and oval 'windows'. The space is spanned by the three ossicles—small bones articulated so as to amplify the vibration of the tympanic membrane and transmit those vibration to the oval window and thence to the inner ear. The middle ear is connected to the nasopharynx by the Eustachian tube, which serves to keep the middle ear pressure very close to the ambient pressure without conscious effort (Fig. 4.3).

In man, the Eustachian tube is partly surrounded by bone (Fig. 4.3) and partly by the soft tissues of the nasopharynx. Abrupt changes in pressure can easily block the part of the tube within the soft tissues and leave the middle ear at a negative pressure relative to the ambient. This is felt as pressure building in the ear and can become rapidly painful as the pressure differential rises. When the pressure differential is low, the Eustachian tube can be forced open by applying positive pressure within the nasopharynx and pharynx (often called the *Valsalva manoeuvre*) resulting in air being introduced to the middle ear to equalize the pressure with ambient. Some individuals find this easy to achieve with minimal effort, while in others this equalization is difficult or impossible. For everyone, however, past a certain differential pressure the Valsalva no longer works and barotrauma of the middle ear can only be avoided by reducing the ambient pressure (ascending). If a diver continues to descend, the increasing ambient pressure can ultimately result in tympanic membrane rupture and the ingress of water into the middle ear.

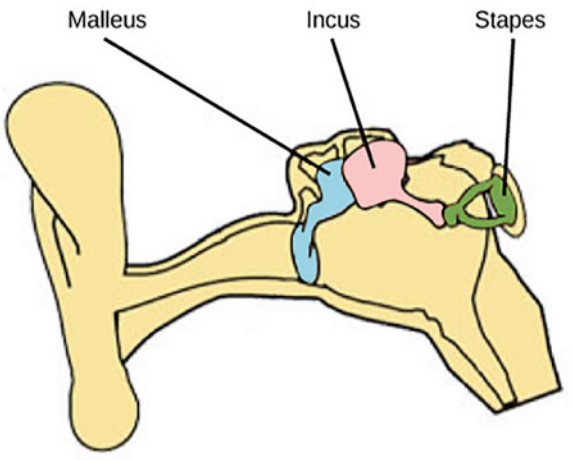

Fig. 4.3 Middle ear anatomy. The middle ear contains three small bones labelled here and is air-filled in health with a connection to the nasopharynx that allows equalization of pressures between ambient and the middle ear space

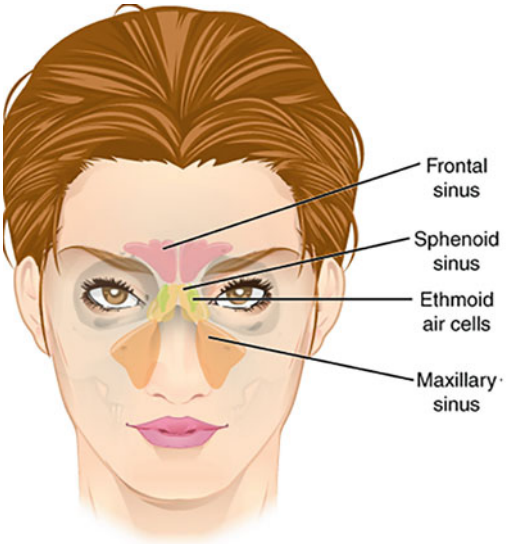

Fig. 4.4 A frontal view of the paranasal sinuses. All communicate with the nasal passage but can be easily blocked by mucus and inflammation of the mucosa lining the passages. In some individuals this may be a frequent occurrence or a chronic state

Lower grades of MEBT result in anything from reddening of the drum, transudation of fluid across the mucosa lining the middle ear to bleeding within the drum and middle ear space. A degree of MEBT is a common complaint of divers and a frequent cause of dive abandonment.

A similar problem occurs in the respiratory (or 'paranasal') sinuses located behind the forehead and cheekbones (Fig. 4.4)

Barotrauma of both the middle ear and paranasal sinuses is most commonly encountered with increasing pressure as a result of descent through the water column. Occasionally problems can arise during a reduction in pressure (barotrauma of ascent or 'reverse squeeze'). These can be highly problematic because of the need for the diver to eventually reach the surface.

Pulmonary Barotrauma

Barotrauma to the lungs (pulmonary barotrauma or PBT) is the one type of barotrauma that is more commonly a problem of ascent than descent. Unlike MEBT or sinus barotrauma, PBT is a potentially fatal problem due to either the entrainment of gas into the pulmonary circulation or to the presence of gas within the chest but outside the substance of the lung.

PBT has been well reviewed by Mitchell in 2016 [23]. As with other barotraumas, it is a direct result of Boyle's Law and occurs when an obstruction to the egress of gas from the lungs occurs during ascent, resulting in a combination of over-pressure and over-distention of the lungs. The pathophysiological relationship between pressure and volume changes is complex and variable between individuals. There is evidence that higher pressures within the lung can be tolerated if the chest is bound so that it cannot expand, suggesting the problem is primarily one of over-expansion

[24]. Gas under pressure disrupts the substance of the lung and is forced out of the alveoli and small airways into the tissues. The gas can track to several different locations, causing distinct pathological syndromes.

Firstly, the gas may track into disrupted capillaries and veins surrounding the alveoli and thence embolize into the pulmonary veins, left heart and be ejected from the left ventricle in the arterial circulation. The most important consequence is the distribution of these bubbles of gas through the cerebral circulation, resulting in cerebral arterial gas embolism (CAGE). CAGE is a true diving emergency and can be rapidly fatal or result in permanent brain injury.

Secondly, the gas may be released into the intrapleural space—the potential space between the lining of the lung and the chest wall. An accumulation of gas here is called a pneumothorax and may restrict ventilation if the volume is large and pressure starts to rise as more gas enters the space and the diver rises through the water column. If the pressure and volume of a gas collection threaten respiration, this is another true diving emergency and can be rapidly fatal unless the gas is released by puncturing the chest wall to facilitate a communication with the outside air.

Finally, the gas may track up the lining of the small airways and manifest in the mediastinum or subcutaneously in the tissues of the neck and face where it is called '*surgical emphysema*'. This manifestation can be impressive but is rarely life-threatening.

4.3.4 Physiology of Exposure to Individual Gases

4.3.4.1 Inert Gas Narcosis

Inert gas narcosis (IGN) refers to a clinical syndrome characterized by impairment of intellectual and neuromuscular performance and changes in mood and behaviour by a gas, which undergoes no metabolic alteration in the body (i.e. it is 'inert'). These changes become more profound with increasing partial pressure of the gas concerned and will ultimately lead to unconsciousness. In compressed air exposure, these changes are due to nitrogen. IGN has been comprehensively reviewed by Bennett and Rostain [25].

The effects of nitrogen breathing at pressure are well-recognized by divers and often called '*nitrogen narcosis*', '*the narks*' or '*raptures of the deep*' (l'ivresse des grandes profondeurs), the latter term coined by Jacques Cousteau. The narcosis, although highly variable, places a practical depth limit to scuba diving with compressed air at approximately 40–50 metres. Effective work at greater depth requires the substitution of a less narcotic respiratory diluent such as helium or hydrogen. It is highly probable that many "unexplained" scuba deaths have been induced by the insidious onset of nitrogen narcosis.

IGN is produced by an increased partial pressure of most inert gases, with helium a notable exception. The effect is highly analogous to that of anaesthetic gases

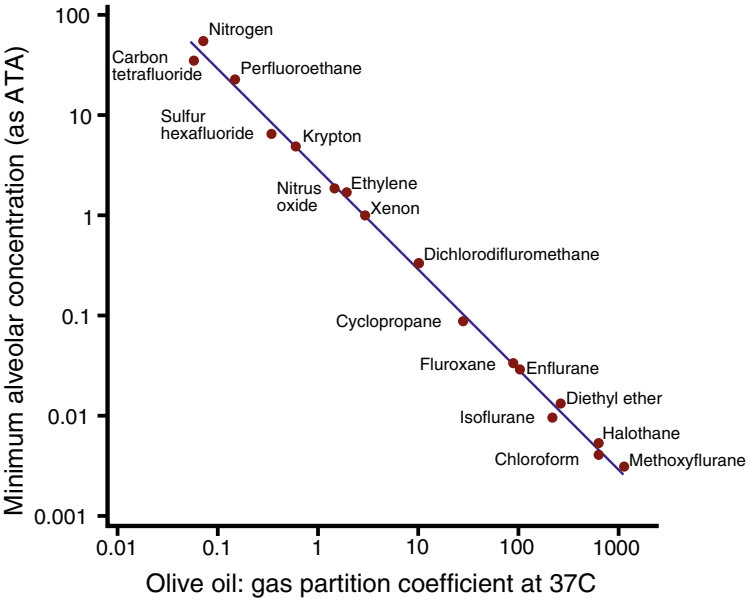

Fig. 4.5 The Meyer–Overton Hypothesis suggests the potency of a gas to produce anaesthesia is directly proportional to the fat solubility as measured by the relative solubility in fat and water—here quantified as the oil:gas partition coefficient. Note nitrogen is highly insoluble compared to useful anaesthetic gases, and so requires great pressures to exert anaesthesia. Only when breathing air at pressures above about 40 msw (5 ATA) are the effects notable. Initial concept from Crowder [29]

and most inert gases display this property—although there are great differences in the partial pressure (dose) required to achieve impairment in function. Xenon is anaesthetic at less than one ATA and is a useful anaesthetic gas in wide use in Europe, with a requirement of about 0.63 ATA (62.2 kPa) to achieve clinical anaesthesia [26]. Although estimates vary, the partial pressures required for other gases vary widely, with nitrogen requiring about 70 to 80 ATA to produce surgical anaesthesia (7,091 kPa to 8,104 kPa or more than 700 msw) [27]. Although recently questioned, the Meyer–Overton hypothesis is a useful concept and suggests the potency of any anaesthetic gas is directly proportional to the solubility in fat as measured using the olive oil-gas partition coefficient (Fig. 4.5). Nitrogen is quite insoluble in fat (low partition coefficient) and thus very high partial pressures are required to produce effect [28].

In practice, IGN from nitrogen is expressed as the insidious onset from about 40 metres depth (5 ATA) of a state of 'euphoria, retardation of the higher mental processes and impaired neuromuscular coordination', which is progressive with increasing pressure so that at 10 ATA (90 msw) most individuals will be unresponsive and in a state of stupefaction. Frank unconsciousness develops beyond that point. In 1939, Behnke and Yarbrough first reported that substitution of helium

for the nitrogen in compressed air eliminates these effects [30]. Although there is marked individual variation in susceptibility to IGN, all divers breathing compressed air are significantly affected at a depth of 60–70 metres.

The higher functions, such as reasoning, judgement, recent memory, learning, concentration and attention are affected first. The diver may experience a feeling of well-being and stimulation similar to the overconfidence of mild alcoholic intoxication. Occasionally an individual will report the experience as terrifying and deeply unpleasant, and this is more probable in the novice who is apprehensive in her new environment. Deeper exposure is characterized by impairment of manual dexterity and progressive deterioration in mental performance, automatisms, idea fixation, hallucinations and finally, stupor and coma. Some divers complain of tunnel vision. They are less aware of potentially significant dangers outside their prescribed tasks (perceptual narrowing) [31]. More recently, abnormal emotional processing has been described, with a suggestion that the emotional responses to threat are muted with increasing IGN [32]. An approximate correlation between depth and both symptoms and signs is shown in Table 4.2.

The narcosis is rapidly evident on reaching the given depth (partial pressure) and is not progressive with time. It is more pronounced initially with rapid compression (descent) and is rapidly reversible upon reduction of the ambient pressure (ascent).

IGN is increased with stress and distraction from other sources including cold, reduced sensory input, and both oxygen and carbon dioxide intoxication. Frequent

Table 4.2 Some observations on the effects of exposure to compressed air at increasing pressure/depth [31]

Pressure (ATA)	Effects
2–4	Mild impairment of performance on unpractised tasks; mild euphoria
4	Reasoning and immediate memory affected more than motor coordination and choice reactions; delayed response to visual and auditory stimuli
4–6	Laughter and loquacity may be overcome by self-control; idea fixation, perceptual narrowing and overconfidence; calculation errors, memory impairment
6	Sleepiness, illusions, impaired judgement
6–8	Convivial group atmosphere: may be terror reaction in some; talkative; dizziness reported occasionally; uncontrolled laughter approaching hysteria in some
8	Severe impairment of intellectual performance; manual dexterity less affected
8–10	Gross delay in response to stimuli; diminished concentration; mental confusion
10	Stupefaction; severe impairment of practical activity and judgement; mental abnormalities and memory defects; deterioration in handwriting; uncontrollable euphoria, hyperexcitability; almost total loss of intellectual and perceptive faculties
> 10	Hallucinogenic experiences; unconsciousness

or prolonged exposure produces some acclimatization, but this may be due to a reduction in psychological stress rather than represent true adaption.

A reliable indication for the presence of IGN is not yet available. Such an indicator would be useful in predicting individual susceptibility (for diver selection); comparing the relative narcotic potencies of different respiratory diluents for oxygen; delineating the role of factors other than inert gas in producing depth intoxication and monitoring the degree of impairment during practical tasks.

4.3.4.2 Oxygen Toxicity

The normal partial pressure of oxygen (PO_2) in air is approximately 0.2 ATA. Although essential for survival, oxygen is toxic at an elevated partial pressure, and the complex systems we have for defending ourselves from oxygen toxicity is a testament to the evolutionary pressure to utilize this highly reactive molecule [33].

A high inspired pressure of oxygen (PiO_2) has several physiological effects on the body. While there is no direct effect on ventilation, there is a reduction in the CO_2 carrying capacity of haemoglobin, a vagally mediated bradycardia and vasoconstriction of intracranial and peripheral vessels. The many manifestations of oxygen toxicity are summarized in Fig. 4.6. In relation to diving, toxic effects on the central nervous system (CNS) and lungs are of prime importance and only these will be discussed in detail.

CNS toxicity (also referred to as the 'Paul Bert effect' after the French physiologist [7]) is an acute phenomenon with a threshold above a PiO_2 of about 1.5 ATA (5 msw) and displaying wide individual variability. Pulmonary effects (also referred to as the 'Lorrain Smith effect' after the Scottish pathologist) are more insidious in onset but are apparent at lower pressures with a threshold of about 0.55 ATA.

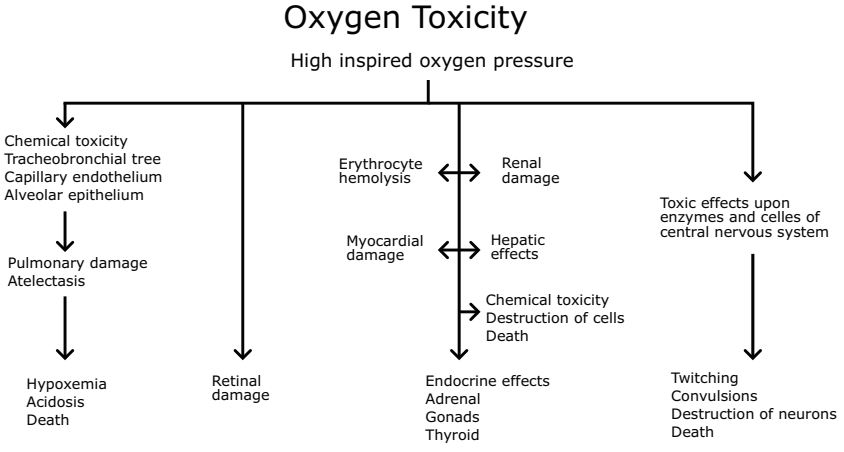

Fig. 4.6 Summary of major manifestations of oxygen toxicity

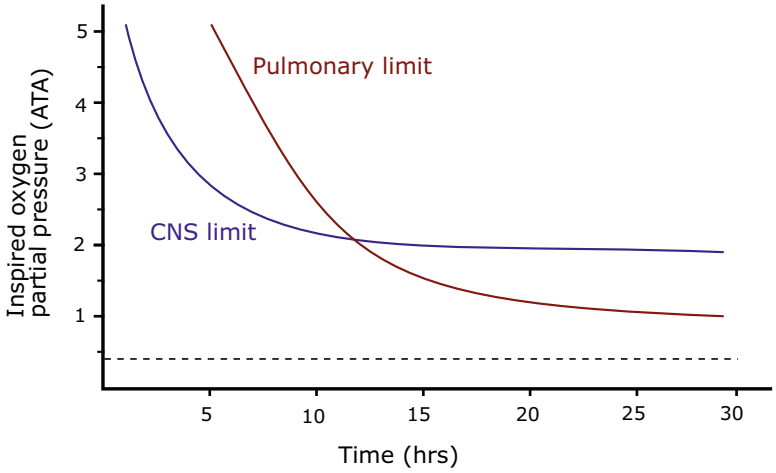

Fig. 4.7 Prediction of pulmonary and CNS toxicity

At lower pressures, pulmonary toxicity is the limiting factor regarding duration of exposure, whereas at higher pressures neurological toxicity is of prime concern, as indicated in Fig. 4.7.

The precise mechanism of oxygen toxicity is unknown. There are a great many sites at which oxygen acts on metabolic pathways or on specific cellular functions. These sites may involve cell membranes, 'active transport', synaptic transmission, mitochondrial or cell nuclei. Many enzymes are inactivated by high PO_2, particularly those containing sulphydryl groups (-SH). It is postulated that adjacent -SH groups are oxidized to form disulphide bridges (-S-S-) thus inactivating the enzyme (this may be important in the development of hyperoxic cataracts of the lens of the eye). Enzymes containing -SH groups, and known to be susceptible, include glyceraldehyde phosphate dehydrogenase (a key enzyme in glycolysis), the flavoprotein enzymes of the respiratory chain and the enzymes involved in oxidative phosphorylation.

The *oxygen free radical theory of toxicity* is widely accepted as an explanation at the molecular level [34]. The production of highly reactive free radicals is a normal consequence of aerobic metabolism, and for this reason, aerobic organisms have developed antioxidant mechanisms to cope with molecular oxygen exposure. Human cells have a system of enzymes to scavenge these radicals called the tissue antioxidant system. Two of these enzymes, superoxide dismutase and catalase, are involved in maintaining adequate supplies of reduced glutathione (containing sulphydryl groups) to deal with the free radicals.

In the face of hyperoxia these mechanisms may be overwhelmed leading to the formation of excess reactive oxygen species and direct cellular toxicity through enzyme inactivation and structural damage (e.g. lipid peroxidation). These radicals are intermediates formed in many biochemical enzyme-catalysed reactions and are

Table 4.3 Physiologic factors known to predispose to oxygen toxicity, from [33]

Physiological states: • Physical exercise • Hyperthermia • Immersion • Stress response	*Gases*: • Carbon dioxide • Nitrous oxide • Inert gases
Pathological states: • Fever • Congenital spherocytosis • Vitamin E deficiency	*Chemicals*: • Paraquat • NH_4CL
Drugs: • Amphetamines • Acetazolamide • Aspirin • Atropine • Disulfiram • Guanethidine	*Hormones and neurotransmitters*: • Insulin • Thyroxin • ACTH • Cortisol • Adrenaline, Noradrenaline • GABA *Trace metals*: • Iron • Copper

the result of the reduction of the oxygen molecule by electrons. For example, the superoxide anion O_2 is formed when oxygen accepts a single electron and hydrogen peroxide (H_2O_2) two electrons. The final reaction is the acceptance by oxygen of four electrons to form water or a stable hydroxyl anion. All these species of oxygen, referred to as oxygen radicals, are highly oxidative [33].

Many factors predispose to the development of oxygen toxicity, and these are summarized in Table 4.3. Several of them have in common the introduction of an added stressor (e.g. fever or exogenous adrenaline), while higher oxygen levels may be tolerated in the presence of enhancers of inhibitory pathways such as lithium (through increasing gamma-aminobutyric acid (GABA) levels).

Central Nervous System (CNS) Toxicity

In diving, CNS oxygen toxicity is the factor limiting depth when oxygen supplementation is used. While breathing compressed air, the effects of increased partial pressure of nitrogen (see Sect. 4.3.4.1) usually prevent the diver from reaching a depth and duration at which oxygen will become a problem. 'Technical' diving, in which a higher FiO_2 than air is commonly used, permits CNS toxicity.

The onset of toxicity may be sudden and develop without warning. A wide range of symptoms and signs has been described, the most dramatic of which is a generalized convulsion, similar to a grand mal epileptic seizure. In practice, only

about half of those affected describe any premonitory symptoms. When present, such manifestations include nausea, vomiting, light-headedness, dizziness, tinnitus, dysphoria, tunnel vision and twitching.

An important aspect of toxicity is the great variation in susceptibility. As well as the wide range of tolerance between individuals, there is marked variation in an individual's tolerance from day to day [35]. In any diver, the time of onset of symptoms cannot be related to a predictable depth or time of exposure. Overall however, it is clear the greater the partial pressure and the longer the time exposure, the more likely is the toxicity to develop. The danger of convulsions prevents divers breathing 100% oxygen in safety when deeper than 8–10 msw.

Pulmonary Toxicity

Clinically obvious pulmonary oxygen toxicity does not manifest in short duration oxygen diving and is of importance only in saturation and long chamber dives and where high partial pressures of oxygen are inspired, such as during therapeutic recompression. Prolonged exposures to partial pressures as low as 0.55 ATA (such as in space flight) have been found to produce significant changes. A PiO_2 0.75 ATA has produced toxicity in 24 hours.

Pulmonary toxicity causes progressive respiratory distress leading to respiratory failure and finally death. In patients receiving high concentrations of oxygen therapeutically, it is sometimes difficult to distinguish between the conditions for which the oxygen is given and the effects of oxygen itself (e.g. shock lung, respiratory distress syndrome).

The factors affecting the degree of toxicity are the PiO_2, the duration of exposure and individual variation in susceptibility. When breathing 100% oxygen at 2.0 ATA, the typical time to produce symptoms in fit individuals is around three hours, but some individuals may remain symptom-free for up to eight hours.

The earliest symptom is usually mild tracheal irritation manifesting as retrosternal discomfort and aggravated by deep inspiration. There may be a dry cough. Chest tightness is often reported and with increasing exposure the discomfort will progress to significant retrosternal pain, dyspnoea at rest and uncontrollable cough. The higher the inspired oxygen pressure, the more rapidly symptoms develop and the greater the intensity. While there are usually no physical signs, some inspiratory extra sounds (rales) may be heard on deep inspiration and low grade fever may develop after prolonged exposure. Eventually, pulmonary infiltrates become increasingly prominent on chest X-ray and a typical adult respiratory distress pattern (ARDS) develops that may be irreversible and fatal.

Forced vital capacity (FVC or VC) is often used to monitor divers at risk and in order to detect early changes before distressing symptoms develop. Reduction in VS is usually progressive throughout the oxygen exposure and may occasionally take several days to return to normal. VC has been used to delineate pulmonary oxygen tolerance limits in normal subjects and the relationship between VC and exposure is shown in Fig. 4.8. Changes in diffusing capacity for carbon monoxide may be the most sensitive indicator of progress.

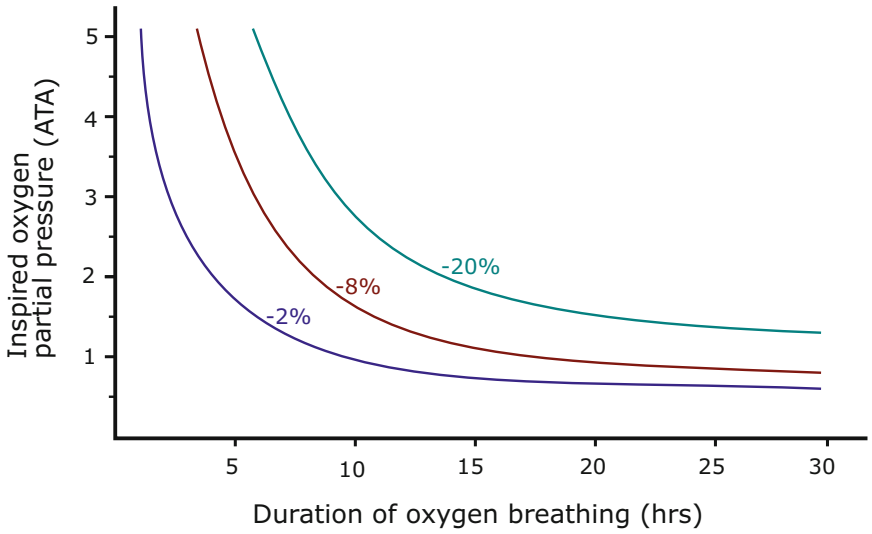

Fig. 4.8 Vital capacity (VC) changes with oxygen breathing, from [36]. Increasing inspired oxygen will hasten the toxic effects of oxygen. Breathing 1 ATA of oxygen for 10 hours will produce approximately a 2% decrement in VC, while breathing 3 ATA of oxygen for 10 hours will result in a more than 20% decrement

No specific therapy is available to delay or modify the pulmonary damage caused by hyperoxia. When toxicity is evident, the oxygen partial pressure should be reduced as soon as practically possible.

In order to assist in the prevention of significant pulmonary toxicity, one concept that has gained popularity is that of the 'UPTD' or unit of pulmonary toxic dose. Using UPTDs, different exposures in time and PiO_2 can be expressed as a 'standard' minutes of exposure at 1.0 ATA. Expected UPTDs can be calculated from any planned exposure and that exposure modified to keep the decrement in VC within acceptable limits (see Clarke [35] for a fuller explanation).

4.3.4.3 Carbon Dioxide Toxicity

Carbon dioxide (CO_2) is produced from the process of cellular respiration and needs to be eliminated via the lungs. Diving neither fundamentally alters the requirement of ventilation to adjust to maintain normal arterial carbon dioxide tension ($PaCO_2$), nor the rate of production. For a hypothetical resting diver the production of CO_2 will be the same except for any additional metabolic requirement for thermal regulation and increased work of breathing. At high work rates, increasing depth or when using breathing circuits with high resistance, there is potential for hypercarbia and the development of toxicity.

The normal $PaCO_2$ is about 40 mmHg and for mixed venous blood is about 46 mmHg. CO_2 in the alveolar gas ($PACO_2$) is in equilibrium with the pulmonary veins and is therefore also about 40 mmHg. Because CO_2 is the product of a metabolic process and we do not produce more of this gas simply because we are at depth, the $PACO_2$ is constant irrespective of depth, unlike oxygen and nitrogen that reflect the pressures in the inspired gas.

CO_2 (along with acidosis) is the primary stimulus for respiration at the central medullary chemoreceptors in the brain. About 200 ml of CO_2 are produced and excreted per minute at rest, but much larger amounts with exercise. As long as ventilation can increase in response, the arterial CO_2 tension will be maintained at a normal level and as a diver descends, the alveolar percentage will decrease.

Divers can manipulate the $PaCO_2$ in both directions. In breath-hold diving, deliberate hyperventilation prior to diving can drive down $PaCO_2$ and extend breath-hold time. For scuba divers, deliberate hypoventilation will allow $PaCO_2$ to rise in order to extend dive time through conservation of air. Both strategies are not without risk. Hyperventilation can allow a breath-hold diver to delay the breathing 'breakpoint' due to rising $PaCO_2$ at the risk of dangerous levels of hypoxia and potential unconsciousness and death prior to reaching the surface. The physiology of breath-hold diving is fascinating and further appraised in Sect. 4.3.7, and in [37].

A rising $PaCO_2$ has widespread effects on the body, most notably the respiratory, circulatory and nervous systems. Where high tissue CO_2 (hypercapnia) produces pathophysiological changes dangerous to the diver, the term '*CO_2 toxicity*' (or '*CO_2* poisoning') is used.

Excluding asphyxia and drowning, there are four main mechanisms of CO_2 toxicity in diving: failure of an absorbent system in closed or semi-closed rebreathing apparatus; inadequate ventilation (flush) of a diving helmet; inadequate pulmonary ventilation (often deliberate) or with increased resistance from the equipment. Whatever the cause, CO_2 toxicity is much more rapid when the diver is exercising and producing large amounts of CO_2.

Inadequate ventilation at extreme depths is a consequence of the combination of equipment resistance and increased gas density. Extreme reductions in ventilation and the increased work of breathing are one of the limiting conditions for achievable depths while immersed. With any given set of equipment there will come a point where the work of breathing dense gas produces more CO_2 than can be eliminated through the minute ventilation that can be achieved. The result is a rapid rise in alveolar, blood and tissue PCO_2 to the point of unconsciousness and death.

Under less extreme conditions the combination of tight wetsuits, harnesses and buoyancy compensators can also restrict thoracic movement and place an increased workload on the diver's respiratory muscles. The extent to which this load is overcome varies greatly among divers, but there is often some elevation of the alveolar PCO_2.

Clinical features depend on the rate of development and degree of CO_2 retention. They vary from mild compensated respiratory acidosis, detected only by blood gas and electrolyte estimations, to rapid unconsciousness and death with exposure to high $PiCO_2$ as a result of absorbent failure in a rebreather unit. As suggested above,

a rising CO_2 is a powerful stimulant to respiration, such that a typical subject breathing air on the surface will double their minute volume when exposed to an inspiratory mixture containing 3% CO_2. There may be some subjective complaint of dyspnea (breathlessness), but at this level, no disturbance of central nervous system function. At a $PiCO_2$ of 5–6% dyspnea will become distressing and is accompanied by further increases in tidal volume and respiratory rate with increased arterial blood pressure and pulse rate. There is sweating of the forehead and hands, and the face feels flushed, bloated and warm. Eventually, mental confusion and lack of coordination may become apparent. At a $PiCO_2$ of around 10% these changes reverse, with a lower pulse rate, hypotension and severe mental impairment. Levels of around 12–14% will cause loss of consciousness and eventually death by central respiratory and cardiac depression. This corresponds with a $PACO_2$ greater than 150 mmHg at 1 ATA. Rapid onset of hypercapnia may be associated with midbrain convulsions, extensor spasms and death.

Removal from the toxic environment usually results in rapid recovery, although some divers complain of nausea, malaise or severe headache for several hours.

It is important to note the inspired concentrations required to produce these effects are progressively lower with increasing depth because toxicity depends on the partial pressure rather than inspired concentration. Breathing 5% CO_2 at 1 ATA delivers a PCO_2 of about 38 mmHg, while the equivalent figures at 2 ATA is 76 mmHg.

There are some divers who tolerate high PCO_2 levels with apparent ease—often called '*CO_2 retainers*'—and this may be particularly so when breathing high oxygen mixtures that can blunt the response to rising CO_2. It is not clear to date whether the impaired response in these divers is inherent or acquired. It may be these divers progressively elevate their CO_2, as an alternative to increasing their ventilation, when the resistance to breathing increases. Under these conditions, it is theoretically possible that toxic levels may eventuate.

Conventional open-circuit equipment using air (scuba) is much less often associated with severe CO_2 retention. Many divers do, however, reduce their ventilation voluntarily in order to conserve air and allow mild elevations of their $PaCO_2$. This practice of long inspiratory and expiratory pauses that reduce minute ventilation is called '*skip breathing*' and often results in a throbbing frontal or bi-temporal headache at the end of a dive and which clears after surfacing. This practice is not generally advised, but common among relatively inexperienced divers trying to extend their dive time. A link with serious diving injury has not been established. It is possible divers develop an adaptation allowing them to tolerate higher levels of CO_2 while diving. Similarly, as 'skip breathers' continue to dive, this breathing pattern becomes habitual and a tolerance to CO_2 may develop.

High Pressure Neurological Syndrome (HPNS)

HPNS is a phenomenon seen with deep diving and usually associated with the use of helium in the place of nitrogen as a diluent for oxygen. As discussed above, high partial pressures of nitrogen cause disabling narcotic effects, but helium is a notably non-narcotic gas and is frequently used in deep diving to avoid narcosis.

HPNS has been described from depths of about 150 to 200 metres (16 to 21 ATA) and is a serious problem that grossly impairs normal neurological functioning and can be associated with unconsciousness and death if allowed to progress. It remains a major limitation to deep diving. Although there were some reports of unexplained neurological dysfunction in experimental animals for about 1880, the first reliable reports of HPNS in humans were made by multiple authors from Britain, Russia, the USA, and France in the 1960s as experimental teams investigated the limits on deeper dives for both commercial and military purposes [38]. The common feature was the use of helium–oxygen mixtures to avoid inert gas narcosis.

The most commonly reported symptoms are tremors (fine at first and becoming coarse), myoclonic jerking, mental clouding and the eventual development of generalized clonic seizures. Divers also report dizziness, headache, euphoria and drowsiness, but the symptoms are notoriously variable. If the pressure is further increased or maintained seizures will result in hypoxia due to the interruption of respiration and death. A reduction in the ambient pressure will relieve symptoms. The addition of a narcotic gas such as nitrogen, hydrogen or even nitrous oxide to the inspired gas mixture will significantly delay or mitigate these symptoms and can be used judiciously to allow further safe descent. For any given gas mixture, the longer the individual has to adapt to rising ambient pressure (slow descent), the less severe the symptoms. There is a marked inter-individual variation in susceptibility.

Gas breathing *per se* does not seem responsible for the development of HPNS and the phenomenon is more likely related to increased hydrostatic pressure. High pressures appear to deform cell wall structures and result in increased excitability in the central nervous system. It is postulated that the reversal of symptoms with narcotic agents (including the gases mentioned above) represents a restoration of normal cell wall function by physical reversal of the pressure changes within cell walls. There is much left to explain about HPNS, which remains an active area of investigation outside diving medicine because of the equally poorly explained phenomenon of the reversal of drug-induced narcosis by anaesthetic agents on the application of pressure [39].

For a more complete description of HPNS physiology including efforts to prevent and treat phenomenon, see Bennett [38].

4.3.5 Cardiovascular Physiology of Submersion

During immersion and diving scenarios, the cardiovascular system is subjected to a number of stresses that may alter cardiovascular functionality. These stresses include temperature, breath-hold diving, reflexes and exercise. Any of these stressors, alone or in combination, can have a significant impact on cardiovascular functions. It is considered that stresses can induce dysfunction in the absence of diseases. Minor cardiac conditions may preface major problems for divers, especially during underwater activities [46].

Stress related changes to cardiac function include tachycardia or bradycardia, hypertension, a reduction of blood volume secondary to submersion-induced diuresis and an overall increase in cardiac work.

Immersion counters the effect of gravity. Venous return is expedited from the vessels of the limbs, causing a redistribution of peripheral blood towards the central thorax, increasing intrathoracic blood volume by up to 700ml. The right atrial filling pressure is also increased by 18 mmHg [43]. Consequently, an increase in stroke volume and cardiac output occurs, due to the increased ventricular preload. Due to the increased cardiac output, the heart's workload is increased by over 30% [43]. This effect can be further intensified when immersed in cold waters. Negative pressure breathing also contributes to the diuretic effect of immersion.

Along with immersion comes a set of reflexes inducing cardiovascular changes. The initial diving response is associated with bradycardia and intense peripheral and selective visceral vasoconstriction. Face immersion exacerbates the haemodynamic effects induced by body immersion.

Over time, the haemodynamic response of the cardiovascular system to full immersion is a reduction in cardiac output, resulting from a decrease in both heart rate and stroke volume, and the reduction of blood volume following diuresis. The reduced blood volume is distributed mainly to the vital organs (heart and brain). The reflexes to immersion result in the effort to maintain blood pressure in a healthy range, reduce heat loss by reducing heat transfer to peripheral regions and by conservation of oxygen for vital organs.

Temperature changes during immersion also have a significant impact on the cardiovascular system, amplifying the previously noted cardiac responses to immersion. Initial cold water immersion causes an increase in sympathetic activity. These sympathetic activities are responsible for an increase in heart rate, systolic blood pressure and ventilation. Furthermore, cold water induces peripheral vasoconstriction that subsequently forces further centralization of blood volume and results in both diuresis and fluid volume reduction.

Exercise on land is commonly associated with an increased heart rate and cardiac output, which do not produce an elevation in blood pressure because muscles and skin become vasodilated. However, during diving vasodilatation is inhibited and thus heavy exercise causes an increased blood pressure and oxygen consumption underwater. Heavy exercise is also considered to aggravate any tendency to cardiac dysrhythmia during immersed conditions.

Heart rate variability (HRV) studies have confirmed a parasympathetic dominance with increased vagal activity and reduced sympathetic tone while diving [44, 45]. ECG abnormalities are frequent during or after the dive (T-wave inversion, premature ventricular excitation, atrial fibrillation) [43], which reflect an inhibition of vagal rhythm and interference with atrio-ventricular conduction.

More detailed information about cardiovascular physiology in diving can be found in Bennett [43].

4.3.6 Physiology of Drowning

Drowning is defined as the process of experiencing respiratory impairment from submersion in liquid [40]. It is the fourth most common cause of injury-related death, accounting for almost half a million deaths annually worldwide [41]. In drowning physiological impairing events are immersion (upper airway above water) and submersion (upper airway under water) [42].

During a drowning event, the victim is unable to ventilate the lungs, resulting in oxygen depletion and carbon dioxide retention. The victim becomes hypercarbic, hypoxemic, and acidotic and will often inhale water leading to death.

During the drowning process, both hypertonic (sea) and hypotonic (fresh) waters cause changes to pulmonary surfactant and the alveolocapillary barrier. These changes in alveolar surface tension and pulmonary compliance cause alveolar instability and atelectasis, negatively affecting the ventilation–perfusion ratio. Prevailing hydrostatic forces during drowning disrupt the integrity of alveolar-capillary membranes, leading to an incapacity for gas exchange. Furthermore, aspiration of either hypo- and hypertonic liquid causes a ventilation–perfusion shift, hypoxemia and metabolic acidosis. Subsequently, these are followed by myocardial depression, pulmonary vasoconstriction and changes in capillary permeability. Loss of consciousness while drowning is associated with asphyxia following liquid aspiration, loss of pulmonary oxygen uptake, brain energy failure and the deterioration of higher brain functions [42].

Although drowning constitutes the primary cause for diving fatalities, it needs to be understood that drowning often occurs as a consequence of other events leading to a reduced level of consciousness and the loss of upper airway reflexes.

4.3.7 Breath-Hold Diving

Breath-hold diving is the oldest form of diving and has been of great importance for commercial and military purposes. Nowadays, breath-hold diving is commonly a sporting activity, including spearfishing, underwater hockey and competitive freediving. For many years it was believed that depth was limited to approximately 30 m, as the lung gas volume diminished with increased depth (Boyle's Law). At this depth, a critical point would be reached where lung tissue would rupture and pulmonary haemorrhage would occur. However, this is not the case, mainly because the human body is able to compensate for this effect by the onset of the diving reflex and shifts of blood volume into the thorax during breath-hold descent. More practically, duration of breath-hold diving in humans is limited by the body's oxygen reserves. At the time of writing the record for breath-hold time on air is over 11 min, whilst the deepest recorded dive is over 200 m.

All diving mammals hold their breath, and their breath-hold time is limited by the amount of oxygen available in the body at the beginning of the dive. In humans, O_2

is mostly stored in the lungs, holding virtually all the oxygen required for the dive. During breath-hold diving O_2 is consumed and carbon dioxide, CO_2, is produced and enters the blood. In the absence of ventilation, the partial pressure of carbon dioxide (pCO_2) is constantly rising. Thus, the breath-hold length is determined by the time it takes for the blood pO_2 or pCO_2 to reach a critical level. The critical pCO_2 level triggers the respiratory system in the brain and immediately forces the diver to take a breath, while the critical pO_2 level will result in loss of consciousness. Either will result in drowning.

As the breath-holding diver descends, the increased ambient pressure squeezes the thorax and gases in the lungs and both, O_2 and N_2 diffuse down the concentration gradient into the blood and tissue. During ascent the chest and the gases in the lungs re-expand diluting any remaining O_2. As a result the blood partial oxygen pressure pO_2 falls more rapidly than would be expected from consumption alone. The diver is now subjected to a greater risk of losing consciousness from hypoxia when approaching the surface. This event is known as '*Shallow Water Blackout*'. The risk of blackout is amplified when the diver hyperventilates before starting the dive. Hyperventilation decreases the initial pCO_2 and allows an extended time to reach the critical pCO_2 limit (breakpoint) when the urge to breathe cannot be resisted. However, also allowing more time for pO_2 to drop at a dangerously low level (Fig. 4.9).

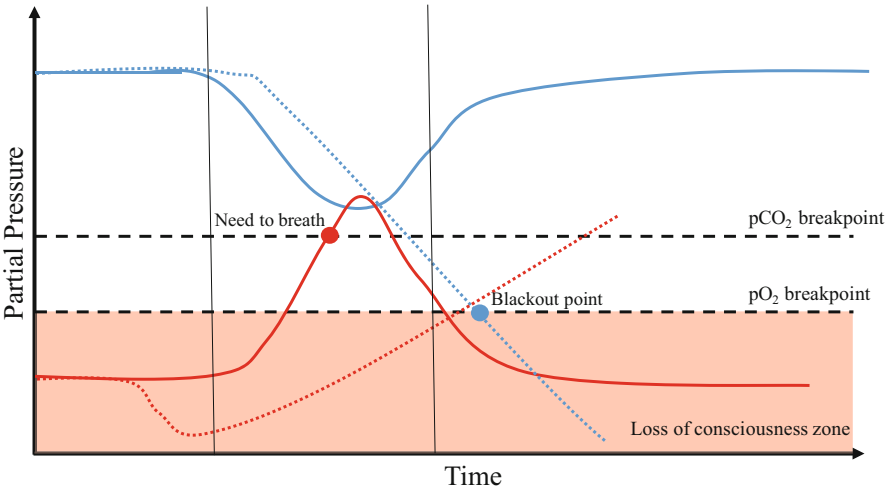

Fig. 4.9 While hyperventilating, the initial pCO_2 is reduced, to extend the time needed to reach the critical breaking point. Thus, pO_2 can continue to fall during this extended time, reaching a dangerously low level in the loss of consciousness zone. Blue lines display the pO_2 trend and red lines the pCO_2 trend. While full drawn lines indicate the trend during normal breathing and diving, the dotted lines represent the trend after initial hyperventilation. When prior dive normal breathing occurred, the pCO_2 breakpoint is reached before a critical low pO_2 can be reached. Hyperventilation shifts the pCO_2 breaking point and a black out scenario can occur

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