

## Diving Medicine

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### Introduction

It could be argued that diving is the most extreme sport of all. Survival in such a hostile underwater environment is dependent on sophisticated breathing apparatus and an understanding of the inherent risks. Diving incidents are well publicised, not least because of the severe injuries that ensue, but remain an uncommon occurrence. Yet despite, or perhaps because of its dangerous and challenging profile, recreational diving is on the increase with more people learning to dive each year. There are currently an estimated 700,000 recreational divers in the UK (1).

Diving within the military may be occupational or recreational. Occupational diving involves a long and rigorous training package designed to provide the diver with the necessary skills to complete their task, be it in mine clearance, underwater engineering or as a combat swimmer. It is Ministry of Defence policy that military diving complies with Health and Safety at Work guidance and with the Diving at Work Regulations as far as reasonably practicable (2).

Diving injuries can be encountered in barracks, on exercise and on operations and Medical Officers need to have an understanding of the presentation and management of conditions associated

with diving. This article aims to review the physics and physiology of diving, discuss the common medical conditions that occur whilst diving, and consider the current thinking in relation to aetiology and treatment.

### A Brief History of Diving

Breath-hold diving has probably been around from before written history. Divers were reportedly used in the Trojan Wars (1194-1184 BC) to disrupt the enemy navies. Aristotle described ruptured tympanic membranes and ear infections in sponge divers, and Marco Polo wrote of pearl divers reaching depths of 27 metres. Alexander the Great is supposed to have made a dive in a primitive diving bell, Roger Bacon described men walking on the seabed in 1240, and in 1535 Guglielmo de Lorena developed the first true diving bell (3).

In 1774 Freminet used a bellows to deliver air from the surface to a diver and Siebe developed the diving suit in 1819 (3). This consisted of a copper helmet and jacket, which was modified in 1839 to resemble the waterproof suit recognised and in regular use to the present day.

Self-contained underwater breathing apparatus (SCUBA) was also developed in the 19th century and in 1943 Jacques Cousteau and Emile Gagnan demonstrated the aqualung (3).

### Physics and Physiology

Air is composed of approximately 21% oxygen and 79% nitrogen. Each of these gases exerts a partial pressure in proportion to their concentration. At sea level man is subject to 1 atmosphere (101.32 kPa). Water, being denser than air, exerts a greater pressure per unit volume such that for each 10 metres of sea water (msw) of descent adds another atmosphere of pressure. A diving cylinder filled with compressed air will contain the same partial pressures of nitrogen and oxygen as environmental air but contain a considerably larger volume, thus allowing a diver to breath air from the tank for extended periods. For example a 12 litre cylinder of air compressed to 232 bar will hold 2784 litres of air (12 x 232). Normal air pressure (1 atm) = 1.01 bar, 760mmHg or 101 kPa.

The pressure increases relating to diving affect the inspired gas and also the gas filled spaces within the diver such as the lungs, sinuses, ears and gastrointestinal tract. The

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Gas Laws make these changes clear.

**Boyle's Law** states that if temperature remains constant, the volume of a fixed mass of gas is inversely proportional to the absolute pressure. As pressure increases by 1 atmosphere for every 10m of descent in water, a given volume of gas will halve in volume at 2 atmospheres. At 20m below the surface (3 atmospheres) the gas will be a third of its original volume and so on. The converse is true on ascent. A volume of gas will double in volume from ascending from 10m deep to the surface. This helps to explain barotrauma.

**Dalton's Law** states that in a mixture of gases, the partial pressure of each gas present is equal to the pressure that gas would exert if it alone occupied the original volume (Table 1). From this it can be seen that with every 1 atmosphere increase in pressure, although the proportions of the gases stay the same the partial pressure increases. The effects of this will be seen when decompression sickness is considered.

**Henry's Law** states that at a constant temperature the amount of gas that will dissolve in a liquid is proportional to the partial pressure of a gas over the liquid. The amount of gas that dissolves will also be affected by Bunsen's coefficient of solubility, time and temperature. This is important in the theory of decompression sickness.

### Physiological Demands of Diving

Being underwater affects many body systems and underwater work creates increased physiological demands on the body. The diver has to move in a medium that is more viscous than air, breathe gases at increased density, cope with an increased hydrostatic pressure, and carry heavy and bulky equipment.

As the diver descends the hydrostatic pressure around his legs may increase the intrathoracic blood volume by up to 700mls. This alters the ventilation perfusion balance and may cause gas trapping. The increased pressures in the right atrium and pulmonary artery stimulates a diuresis, which, compounded by cutaneous vasoconstriction in cold temperatures, causes dehydration.

Vision underwater is altered due to the refraction of light making objects seem larger and closer. Light has a differential absorption in water causing loss of colour (especially red) at depth and many diving environments have poor visibility due to turbid water. Sound travels more than four times faster in water than in air making it much more difficult to localise sound (4). If the diver is using a gas mixture containing helium, the pitch of the voice will increase making underwater voice communication, with wired or wireless systems, almost impossible without the use of expensive electronic equipment (5). Hearing acuity is reduced by around 60dB especially in the higher frequencies, localisation is poorer and there are the thermal and pressure effects due to being under water. There is decreased sensation affecting dexterity, 2-point discrimination, vibration and other functions caused by the cold, the gases used for respiration, pressure and the protective equipment worn to survive in the environment.

The increased density of water leads to an increased heat loss. Water temperatures vary between  $-2$  to above  $30^{\circ}\text{C}$  but are usually below the body's thermo neutral point ( $31^{\circ}\text{C}$ ). Greater depth leads to lower temperatures and water conducts heat 25 times faster than air. Peripheral vasoconstriction occurs to maintain the core temperature, but this can cause loss of manual dexterity, and the alteration in blood flow may contribute to decompression sickness. Protective thermal equipment worn to survive the cold environment causes an additional loss of dexterity.

### The Gases

#### Nitrogen

Nitrogen is an inert gas that constitutes 79% of air and at 1atm has a partial pressure of 0.79atm. It is a weak anaesthetic agent. Diving below 30 metres of sea water (msw) equivalent to 4 atmos and a  $p_{iN_2}$  of 320kPa will lead to a narcotic effect. This is similar to the effects of alcohol, gets worse with depth and causes cognitive and manual impairment. Similar effects will occur with other inert gases including helium and hydrogen (4). While the gas itself is not poisonous, it leads to a condition called nitrogen narcosis ("being narked"), which can be fatal due to the effect of this condition on decision making. Divers can be taught to acclimatise to the milder effects of narcosis by building up to deeper dives gradually and regularly. It is not thought to be a physiological adaptation but a learned response to repeated drills. As nitrogen is inert it is not metabolised by the body. It does, however, enter the body tissues. During a dive the amount of nitrogen that enters the tissues is proportional to its partial pressure in alveolar air. Therefore, as the dive becomes deeper, the amount of

Table 1. Effects of Depth on Lung Volume and Partial Pressures.

Effects of Depth on Partial Pressures				
Depth	Lung Volume	Pressure	pO <sub>2</sub>	pN <sub>2</sub>
Surface	100%	101kPa 1 atmos	21.2kPa	79.8kPa
10m	50%	202kPa 2 atmos	42.4kPa	159.6kPa
20m	33%	303kPa 3 atmos	63.6kPa	239.4kPa
30m	25%	404kPa 4 atmos	84.8kPa	319.2kPa

nitrogen entering the tissues will be greater. Different tissues 'on-gas' at different rates but eventually all the body tissues will become saturated with nitrogen.

On ascent (or decompression) the nitrogen will move out of the tissues into the blood and be eliminated by the lungs. If the ascent is not controlled the nitrogen may form bubbles within the tissue or blood, much like the opening of a carbonated drink. These bubbles may cause damage to their surrounding vessels or block the circulation to a tissue. This manifests as decompression sickness and may result in a wide array of signs and symptoms.



### Oxygen

Oxygen makes up 21% of air and has a partial pressure of 0.21atm at 1atm. It is vital for life and, therefore, must be a part of a diving gas mixture. However, at higher partial pressures it becomes toxic to the lungs and central nervous system. The precise mechanism of this is unknown, but is thought to involve a depression of cellular metabolism by the production of free radicals and inactivation of enzymes (6).

Central Nervous System (CNS) toxicity shows extreme variability between individuals such that time to onset of symptoms cannot be related to a particular depth or time of exposure. Symptoms include facial twitching, nausea and vomiting, tunnel vision, dysphoria, tinnitus and convulsions. Exercise and hypothermia have been shown to hasten the onset of symptoms. CNS Oxygen Toxicity can occur at a  $p_{O_2}$  above 200kPa or 2 atmospheres. The recreational diving limit is set at 1.4atm, the Health and Safety Executive limit to 1.5atm and the maximum limit of 1.6atm to prevent toxicity occurring. Chamber dives providing therapeutic oxygen for treatment of decompression sickness or other conditions are limited to 2.8atm.

Pulmonary Oxygen toxicity occurs after longer duration of exposure or after high inspired partial pressures. Again individual susceptibility varies greatly. Signs and symptoms often start with a tickle in the throat and progress onto a cough and retrosternal pain (5). Dyspnoea, crackles and wheeze may ensue with a loss of lung Vital Capacity. Partial pressures of more than 0.8atm cause acute toxicity which leads to exudative and proliferative changes in the lung. Chronic

exposure to lower partial pressures causes progressive pulmonary fibrosis. A reduction of 10% of Vital Capacity associated with cough and chest pain is usually reversible in a few days. Divers using elevated partial pressures of oxygen in their gas mixtures and performing repeated or long dives calculate their Oxygen Tolerance Units over 24 hours (where 1 OTU is equivalent to exposure to 100% oxygen at 1atm for 1 minute), to prevent toxicity.

Oxygen has been shown to adversely affect vision by causing progressive myopic changes. In 1971 Clark and Lambertsen demonstrated a loss of peripheral vision after breathing oxygen at 3atm for 2.5 hours. Recovery was complete after 45 minutes breathing air (6). The middle ear can also be affected by high concentrations of oxygen as demonstrated by US Navy divers breathing 100% oxygen from a closed circuit system (the rebreather). They experienced a popping sensation in the ear and a mild conductive hearing loss and were found to have fluid in the middle ear caused by absorption of the oxygen. This spontaneously resolved (6).

### Gas Choice

Recreational divers are taught to dive using air, but soon realise the benefits of altering the gas mixture to reduce the risks of narcosis and decompression illness. Divers will either carry their own gas cylinders in open circuit systems (Self Contained Underwater Breathing Apparatus SCUBA) or closed circuit systems ("rebreathers", which utilise soda lime to remove exhaled carbon dioxide, and then recirculate the gas adding small amounts of oxygen).

Military divers use SCUBA and rebreather kits, as well as surface-supplied equipment in which the gas is pumped to them through a long hose. The choice of gas and delivery system is task dependant. SCUBA may be used for under ship inspection tasks, self contained closed-circuit rebreathers for mine clearance operations where the absence of bubbles is beneficial, and surface supplied systems for engineering tasks (7). Pure oxygen systems can greatly increase the maximum duration of the dive but limit the depth to around 7m.

Breathing air and oxygen at pressure causes problems as outlined above. To overcome some of these problems gas mixtures such as Nitrox (a gas mixture that reduces the nitrogen content by replacing it with oxygen) are used. As the absorption of nitrogen is less this lowers the risk of decompression illness and extends the diver's time in the water. For instance a dive to 20m on air for 35 minutes could be extended to 51 minutes using Nitrox 32 without increased risk or decompression time.

Trimix (helium, nitrogen and oxygen) is also used. Helium replaces some of the oxygen and nitrogen in the mixture. Helium is

an inert gas, which does not normally cause narcosis, and so this mixture can be used when diving at greater depths. Oxygen toxicity is less of a problem as the oxygen content is reduced, making a gas safe to breathe at depth although potentially harmful on the surface. Helium enters and leaves tissues up to 2.65 times faster than nitrogen, therefore, causing a different set of issues during ascents.

### **Decompression Illness (DCI)**

DCI is a term that includes decompression sickness (DCS) and arterial gas embolism (AGE). Both conditions are caused by gas bubbles that have either escaped due to barotrauma to the lung or have evolved in the tissues. Data collected by the Divers Alert Network show that 50% of cases of DCI occur within 1 hour of a dive, 90% within 6 hours, and less than 1% present after 36 hours (8).

### **Decompression Sickness**

During a dive, gas dissolves in the body and is eliminated on ascent by the lungs. A large amount of work has been done looking at the optimum rates at which to ascend after each dive to maximise the efficiency of this process. In 1906 John Scott Haldane was commissioned by the Royal Navy to minimise DCS among fleet divers, and published his work in 1908. He concluded that different compartments in the body absorb and release nitrogen at different rates, and that decompression stops should be staged to minimise the chances of decompression sickness. His work produced the first Royal Naval decompression tables. Although the US Navy has further modified these tables, the basic Haldanian model is still used today in recreational dive tables and computers (4).

Decompression sickness is caused by bubbles that have come out of solution in the tissues. Bubbles formed while diving on air are predominantly made of nitrogen and may grow in size to cause arterial obstruction. Intravascular bubbles block blood and lymph circulation, causing ischaemia, endothelial damage and impaired waste removal. Extravascular bubbles distort and compress blood vessels and nerves and may also set up an inflammatory response activating complement and coagulation pathways. It is these pathophysiological changes that create the symptoms and signs of pain, swelling, oedema, rash and paralysis.

### **Risk Factors for DCS**

There are many individual factors thought to play a part in DCS. These can be divided into those relating to the diver, the dive, and post dive situation.

#### ***The Diver***

All military divers are required to undergo regular 'fitness to dive' medicals (7). These

incorporate the need for high levels of physical fitness to accomplish strenuous tasks. The demands on a recreational diver are generally considered to be less and currently self-certification via a questionnaire is all that is required.

Poor physical fitness is a risk factor for DCS. Broome *et al* (1994) experimented on pigs to show that sedentary pigs were more likely to get DCS than those that ran twice a day (9). Increased body fat was presumed to be a risk for DCS due to its slow off-gassing characteristics, but studies have not shown this to be statistically true (10).

Repetitive deep diving increases the likelihood of getting DCS as the surface time for off-gassing is reduced, causing a build-up of residual nitrogen within the body tissues. Both recreational and military diving follows guidelines on safe surface intervals between dives to reduce this risk.

Research into whether women are more or less susceptible to DCS remains inconclusive. The effect of the menstrual cycle on DCS risk was investigated by Lee *et al* (11) and showed an increased incidence of DCS symptoms in the first week of the menstrual cycle and immediately prior to menstruation. This variation in distribution of DCS symptoms across the menstrual cycle differs further between users and non-users of the oral contraceptive pill.

Advancing age, pre-dive dehydration and having a patent foramen ovale are other conditions thought to increase the likelihood of getting DCS. A patent foramen ovale (PFO) is a persistent embryological remnant between the two atria in the heart. It is formed by the failure of fusion of the ostium primum to the ostium secundum after birth. PFO occur in approximately 25% of the population and are usually asymptomatic (12). An association between neurological DCS and the presence of a PFO was observed and various studies have been conducted which have established that paradoxical gas emboli can occur causing neurological defects (13). Divers that present with neurological or cutaneous DCS after a non-provocative dive (that is one with no other obvious predisposing risk factors) should be considered for investigation of a potential PFO.

#### ***The Dive***

The biggest risk factor with the dive is a rapid ascent after a long deep dive. Being cold on ascent and on any decompression stops has also been shown to reduce the nitrogen off-gassing and, therefore, increase DCS risk. Exercising at depth, for example swimming against a current, also requires longer decompression times to avoid DCS. The Divers Alert Network estimate the overall risk of DCS in a diver is 1 in 42000 dives above 30msw, but this reduces to 1 in 7000 if the dive is deeper than 30 msw (8).

### Post dive

Divers should be advised not to exercise, ascend to altitude or have a hot bath after a dive as all these factors will cause a sudden increase in off-gassing and bubble size.

### Types of DCS

Bubbles can form and travel anywhere within the body and therefore DCS may manifest itself in a wide variety of signs and symptoms. Traditionally these were divided into Type 1- relating to musculoskeletal pain, skin rashes and oedema (Figure 1), and Type 2- relating to more serious neurological and cardiopulmonary symptoms. This system was often confusing to use and so is now being replaced by 'The Descriptive Protocol' (Table 2). This divides DCS into 'Manifestation' meaning the site(s) of symptoms and 'Evolution' to assess the development of symptoms over time. Neurological and limb pain are the most common manifestations of DCS. The Evolution of a symptom or sign may be progressive, static, spontaneously improving, or relapsing, and reinforces the important principle of repeated examinations in patients with DCS.

Table 2. Decompression Illness – Descriptive protocol.

Evolution	Progressive, Static, Relapsing, Spontaneously Improving
Manifestation	Pain: Limb, Girdle
	Neurological: Any symptom related to neurological deficit
	Audiovestibular: Vertigo, Tinnitus, nystagmus. May mimic inner ear damage
	Pulmonary: Chest pain, cough, haemoptysis, dyspnoea. Maybe due to AGE
	Cutaneous: Itching, erythematous or marbling rash
	Lymphatic: Tender lymph nodes, oedema
	Constitutional: Headache, fatigue, general malaise
Depth, Type, Duration	Describes dive profile

### AGE

Arterial gas embolus (AGE) originates from alveolar barotrauma when quickly expanding air ruptures the alveolar parenchyma and gas enters the pulmonary venous system. Bubbles are delivered to the left side of the heart and from here to the cerebral circulation. If bubbles block the circulation, irreversible damage to nervous tissue may occur within 10 minutes. Symptoms appear suddenly upon surfacing and may include confusion, vertigo, motor or sensory deficits, frothy sputum, seizures, loss of consciousness or arrest. There may be associated pneumothorax, pneumomediastinum or subcutaneous emphysema.



Fig 1. Cutaneous rash.

### Treatment of DCI

DCS and AGE are often indistinguishable in their manifestations and the treatments are the same in the early stabilising phases.

### Initial Treatment

Management of DCI should start by assessment and management of airway, breathing and circulation. In addition 100% oxygen is administered via a tight fitting mask. The benefits of First Aid oxygen have been shown in several studies, (Divers Alert Network 1996). Divers who received oxygen prior to arriving at a recompression chamber were statistically more likely to have an improved clinical condition (8).

As dehydration is usually a feature of the disease, rehydration should be commenced early, either orally or intravenously. Crystalloids are preferred when intravenous rehydration is required (15). Analgesics may mask the disease process and nitrous oxide (present in entonox) should never be used, as it is highly soluble and will diffuse into any gas bubbles exacerbating the disease.

Care should also be taken to lift an immersed diver in a horizontal position as a sudden loss of the hydrostatic pressure from around a cold diver may induce hypotension and loss of consciousness if vertical. Traditionally patients were placed in the left decubitus head down position but due to concerns of worsening cerebral oedema, the diver is now kept horizontal and supine.

Patients must be transferred to a recompression chamber as quickly as possible, but be aware that if a helicopter is to be used, then the crew must fly at low level (less than 300m) if possible to avoid exacerbating the problem.

### Recompression Therapy

Recompression provides definitive therapy and is best conducted in a purpose built chamber under the supervision of a Diving Medicine Specialist (Figure 2). Recom-



Fig 2. Recompression chamber.

pression is thought to reduce the size of the gas bubbles, and hyperbaric oxygen promotes absorption of the bubbles and increases tissue oxygenation.

Initially casualties are compressed to an equivalent depth of 18msw on 100% oxygen and then reassessed. Treatment continues according to the treatment recompression protocols. At 18msw (2.8 atmospheres) it is possible to see oxygen toxicity in the patient, as previously described, although this is usually easily managed. Patients require careful monitoring and constant attention.

There are many different protocols in existence, but their aim is to compress the bubbles to a smaller size and administer hyperbaric oxygen, which speeds up gas elimination and improves oxygenation to ischaemic tissues, relieving clinical symptoms and preventing secondary damage. The main treatment tables used in the UK are derived from the Royal Navy, the US Navy, and diving companies such as Comex. The choice of table may depend on the facility preference, the gas mixture used and the depth of the dive. Treatments may need to be repeated over many days to achieve complete resolution of symptoms.

Various other adjuncts to treatment have been proposed. Aspirin was introduced in the 1960's by the French after they noticed rouleaux formation in the blood. This appearance is now thought to be due to dehydration and as aspirin may also provoke or sustain haemorrhage into central nervous system lesions, it is not routinely used (6). Non steroidal anti-inflammatory drugs have been shown to improve relief of symptoms and an Australian randomised controlled trial showed a reduction in chamber episodes required to achieve symptom resolution (19).

High dose steroids have been suggested as a way of reducing oedema, and are used in some centres in cases of spinal cord DCS but there is concern that they may reduce the body's tolerance to high partial pressures of oxygen (6). There is a growing body of evidence that intravenous lignocaine has a role to play in the treatment of cerebral arterial gas embolism (15, 16). The exact mechanism of action has not been fully elu-

cidated, but the infusion should start as early as possible after the initial insult and continue for 24-48hrs.

Patients with minor symptoms (limb pain or skin manifestations) should not fly for 24 hours or return to diving for 48 hours (18). If ever in doubt a diving medicine specialist should be contacted

### **Barotrauma**

Barotrauma is trauma caused by pressure in any gas filled space. Boyles Law shows that trapped gas will be compressed on descent and will expand on ascent. If this gas is trapped in a body space barotrauma may occur to the surrounding structures. It can be difficult sometimes to differentiate between the signs and symptoms of barotraumas on ascent and DCS. If this is the case it is safer to treat for DCS while further tests are carried out.

**Otobarotrauma.** The ears are the site most commonly affected by barotrauma. Divers manage this by equalisation of air pressure in the middle ear via the eustachian tubes. On descent the diver equalises the middle ear pressure to the ambient pressure. If the eustachian tube is blocked, preventing equalisation, the lining of the middle ear can swell and blood vessels dilate and rupture. The tympanic membrane will become retracted and eventually rupture. Otoscopic examination of the tympanic membrane after barotrauma shows these epithelial changes and is graded 1-5 in severity with 5 equalling rupture. Tympanic membrane rupture normally heals after 6-8 weeks. Rarely middle ear barotrauma can cause facial nerve palsy due to compromise of its vascular supply. This may be confused with DCS but usually resolves 1-2 hours after decompression of the middle ear.

**Outer ear barotraumas** can also occur on descent, commonly caused by wax in the ear canal, or too tight a hood. These will resolve on removal of the obstruction.

Inner ear barotrauma is a more serious condition, but also less common. It can be caused as a result of middle ear barotrauma or due to the diver's efforts at middle ear equalisation. Increased middle ear pressure causes either the round or oval windows to rupture producing vertigo, tinnitus, vomiting, and deafness. Round window rupture may require surgery, should be considered a surgical emergency, and will probably prevent future diving.

**Sinus Barotrauma** occurs if the ostia are blocked before or during a dive, for example due to a cold. If the sinus pressures are not equalised the mucosa may swell and haemorrhage, leading to pain and epistaxis.

Pulmonary barotrauma occurs on ascent due to expanding gas being unable to escape through the airways. This can be due to the diver holding his breath, a mucus

plug or bronchospasm. The gas may rupture the alveolar membrane and will enter the pulmonary veins causing an arterial gas embolism or enter the pleural cavity causing a pneumothorax, a pneumomediastinum or subcutaneous emphysema. Treatment is by conventional therapies, but recompression may be required for severe persistent emphysema.

Barotrauma may also occur in teeth, in the gastrointestinal tract, from the mask (which can cause conjunctival bleeding and lid oedema), and from the suit causing a characteristic rash or 'suit squeeze'.

## Other Diseases

### *Otitis Externa.*

This is a common condition in diving and is often called Swimmer's Ear. This occurs in those who are repeatedly immersed due to the break down in the epithelial lining of the outer ear. It is treated with appropriate antibiotic drops.

### *Dysbaric Osteonecrosis*

Although this condition has reportedly been found in diving dinosaurs, the first case in modern times was noted in caisson workers in 1888. The condition is more common in those who dive the deepest and rare in those who do not exceed 50msw. The condition can be completely symptomless, or present with joint pain. Over 60% of cases occur in the distal femur, and a further 20% of cases occur in the proximal humerus. Lesions are classified as juxta-articular or head, neck and shaft lesions. They are diagnosed by imaging procedures including radiography, MRI and SPECT scanning. Aetiology is unknown and treatment includes the prevention of further exposure and the reduction of joint loading (6).

## Conclusion

Occupational and sports diving takes place in a hostile environment. Environmental conditions and the effects of pressure on tissues and inspired gases produce a range of conditions that, although not unique to this environment, are associated with underwater work and recreation. The military community has a significant population that works and plays underwater and who are at risk of developing any of the diseases described above.

Medical Officers should have an understanding of the risks of diving, and how diving diseases may present. They also need to be able to provide emergency treatment in cases of diving emergencies, and know where to obtain specialist advice and support.

Diving Medicine advice can be obtained from:

The Royal Navy	0044 7831 151523
Aberdeen Royal Infirmary	0044 1224 81818

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