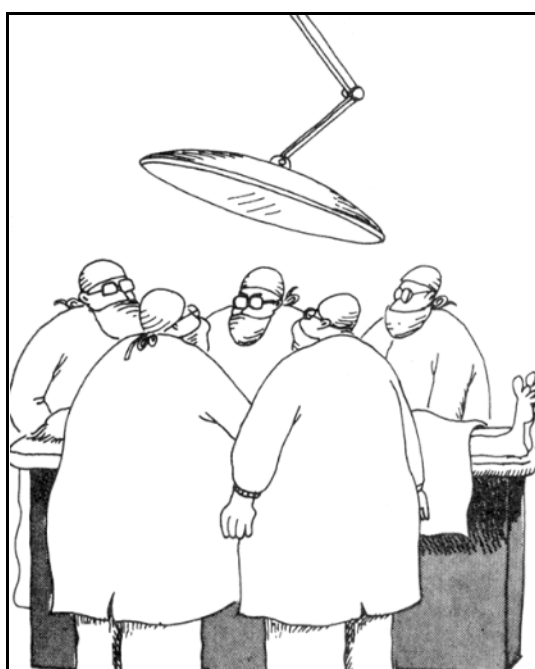


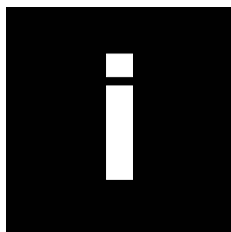
Section 13 — Physiology and Diving Maladies Review



"Okay, Williams, we'll vote.....how many here say the heart has four chambers?"¹

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CHAPTER 1 - DIVING ANATOMY AND PHYSIOLOGY

THE HUMAN BODY

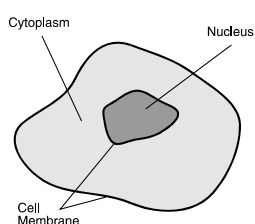


Figure 1. The Cell.

CELLS

To quote the 19th Century French biologist Dutrochet “The cell is truly the fundamental part of the living organism”. The human body is composed principally of simple units called cells. These cells may have different functions and shape but they are all similar in that they all possess the following characteristics.

Structurally they contain a cell membrane, a nucleus and cytoplasm. To function they need oxygen, nutrients and stimuli.

Groups of cells combine to produce tissues such as muscle and blood. Blood contains cells transported in a fluid known as plasma. These cells are called red blood cells, white blood cells and platelets. Tissues combine to form organs within the body such as the heart, brain, lungs and liver.

Groups of organs form the basic systems of the body. Like a computer, the human body is made up of series of complex sub-systems which inter-relate to make the whole. These sub-systems include:

- ✓ Respiratory
- ✓ Circulatory
- ✓ Vestibular
- ✓ Lymphatic
- ✓ Skeletal
- ✓ Nervous
- ✓ Immune

There are a number of other systems in the human body, which are not listed here. However, the ones shown are those, which are impacted by diving the most.

The understanding of many diving ailments discussed throughout the text is dependent upon the knowledge of the basic anatomy and functions of these systems.

NTK!

Knowing how the key physiological systems of the body work will help you to understand how to avoid some of the injuries and maladies associated with diving.



RESPIRATORY SYSTEM

■ STRUCTURE OF THE LUNGS

The respiratory system can be broken up into several major areas. Some of these areas are common to other areas in the body such as the digestive and circulatory systems.

The function of each of these parts is detailed in the following table.



ORGAN	PURPOSES
Mouth and Nose	moisturising, warming, filtering
Pharynx	common airway and food passage, some warming and humidifying
Epiglottis	valve directing air and/or food to the appropriate opening
Trachea (Windpipe)	carry air to the lungs (ringed in cartilage)
Larynx (Voice Box)	produces sounds
Bronchi (Bronchial Tubes)	tubes entering left and right lungs
Bronchioles	further subdivisions of the bronchi leading to alveoli
Alveoli	small sack-like structures where gaseous exchange occurs
Diaphragm	large muscle which contracts and therefore allows lung expansion
Pleural Cavity	sub-atmospheric gap surrounding lungs, keeps lungs inflated
Thorax (Rib Cage)	The thorax is made up of 12 pairs of ribs. Each pair is attached by cartilage to the spinal vertebrae at the back. The other end of the first seven pairs of ribs is attached by cartilage directly to the sternum. The next three pairs join the cartilage above, and the last pair does not attach at all and are called floating ribs. Ribs can break and mend quite easily but the danger of fractured ribs is injury to under lying organs such as the liver, lungs and spleen, with internal bleeding. The muscles in-between the ribs are the intercostal muscles.

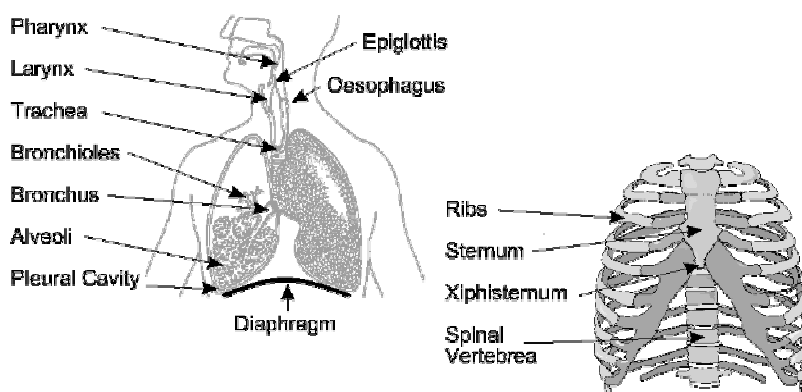


Figure 2. Respiratory System (Left) and the structure of the thorax or rib cage (right)





■ SURFACTANT

Surfactant is the detergent-like substance coating the inner lining of the alveoli. This substance has two essential functions. They are to keep tissues moist and allow gaseous exchange, and to even out surface tension to maintain alveoli shape and volume.

Surfactant is continuously replaced by specific cells within the lungs and has a vital role to play in helping to maintain the healthy nature of the lungs. If the nature of the surfactant changes i.e. when inhaling contaminants such as water or oil, the lungs become inflamed and irritated. The diving ailments related to inspiration of water and oil are drowning, salt water aspiration and lipid pneumonia.

■ PLEURAL CAVITY

The lungs are protected within the chest cavity (thorax) by a series of bones and muscles. The lungs themselves are separated from this protective housing and hang within the thoracic cavity. The outer surface of the lungs and the chest wall are coated by a single membrane called the pleura or pleural membrane.

The only physical connection to the body is via the trachea. The small fluid-filled space between the layers of pleura is at sub-atmospheric pressure. If the lung's pleural membrane is pierced or ruptured, the lung will collapse; such a condition may occur when a diver suffers from a pneumothorax.

The pleura and associated fluids also allow the lungs to expand and contract with minimal friction or interference.

■ BREATHING

THE BREATHING MECHANISM

Breathing is the process whereby gases are moved into and out of the lungs. During this process inspiration of air is achieved when the diaphragm contracts or flattens and the ribs move outwards by intercostal muscles.

The net result is an enlargement of the chest cavity which draws air into the lungs, i.e. the pressure decreases therefore air is drawn in. Stretch receptors located in the intercostal muscles prevent over expansion. During expiration the diaphragm and ribs return to their original positions and air leaves the lungs.

Expiration is a passive operation requiring little energy output. When under stress i.e. during heavy exercise, the muscles of the neck and abdomen also aid in inspiration. During these peak performance periods as much as 4.5 litres of air per breath may be drawn into the lungs. It is only then that the full vital capacity of the lungs is being used.

CONTROL OF BREATHING

The process of breathing is spontaneous. Nerve impulses controlling the diaphragm and intercostal muscles, are produced in the subconscious areas of the brain. This area is referred to as the hypothalamus. These nerve impulses are triggered by cells known as chemoreceptors located in the carotid arteries and aortic arch.



The prime function of the chemoreceptors is to monitor the chemistry of the blood. The major determining factor is the level of carbon dioxide in the blood. High carbon dioxide (hypercapnia) will result in acidosis. This condition will result in vasodilation, increased rate of breathing and increased heart rate (called tachycardia).

Low carbon dioxide (hypocapnia) will result in alkalosis. This condition will result in vasoconstriction, dizziness, reduced urge to breathe and slowed heart rate (bradycardia).



Generally, the body is able to cope with minor alterations in blood chemistry but if the condition is severe, unconsciousness, coma and death may result.

THE BREATHING CYCLE

The normal breathing rate for adults is approximately 12-20 breaths per minute. This rate will increase markedly if any of the following factors are involved; exercise, cold, low oxygen and high carbon dioxide.

Although the requirements for oxygen are greater when the above factors are involved, it is the increased CO₂ which causes the rise in breathing rate. The depth of breathing will also increase when any of the above factors are present. During rest there is a slight pause between each breath but as the demands are increased this pause time becomes less and less until each breath follows on directly from the last.

When referring to breathing on a physiological level it is important to have an understanding of the following terms used for measuring the capacity and activity of the lungs.



TERM	DEFINITION	VOLUME IN LITRES (Approximate)
Total Lung Capacity	lung content when lungs are inflated from total collapse to maximum inflation	6 Litres
Residual Volume	amount of air remaining in lungs after maximal expiration	1.5 Litres
Tidal Volume	amount of air inhaled and exhaled in a normal breath	0.5 Litre
Vital Capacity	the amount of air which can be inhaled after maximal expiration	4.5 Litres
Respiratory Minute Volume	total amount of air moved in and out of the lungs in one minute	25-30 Litres per minute
Inspiratory Reserve Volume	amount of air which can be inhaled after normal inhalation	3 Litres
Expiration Reserve Volume	amount of air which can be exhaled after normal expiration	1 Litre
Respiratory Dead Space (dead air space)	does not take part in gaseous exchange between alveolar air and blood	0.15 Litre

■ GAS EXCHANGE

ROLE OF THE LUNGS IN GAS EXCHANGE

The prime function of the lungs is to allow gas exchange from the body tissues to the atmosphere and vice-versa.

This exchange is a two-way process and only occurs in the alveolar beds of the lungs. All other areas of the lungs such as the bronchioles, bronchi and trachea are termed dead air spaces as no exchange of gases occurs. The diver must understand that an increase in dead air space will require a greater degree of inspiratory and expiratory effort. The dead air space can be increased by the use of masks, regulators and snorkels which can lead to CO₂ build up.



In order for gaseous exchange to occur, several factors must be present. These include a gas pressure difference between tissue and lung, thin moist membrane to allow diffusion, transport agent (blood), and recycling process (breathing).

ROLE OF THE ALVEOLI IN GAS EXCHANGE

It has been estimated that the lungs of an average adult male contain approximately 300 million alveoli. When spread out, these huge numbers of alveoli have a surface area in excess of 70m². (Note: The surface area of the skin is approximately 50 square metres). It is the huge surface area within the confines of the lungs which allows adequate gaseous exchange.

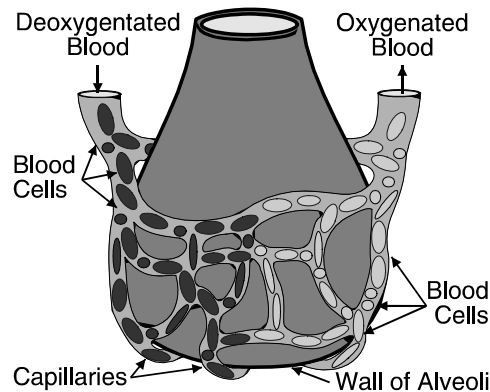


Figure 3. The structure of an alveoli

Another factor essential in allowing adequate gaseous exchange is the thin membrane walls of the alveoli. On average they are less than 0.6 microns across. (Note: 1 micron is 1/1000 of a millimetre). In comparison a single red blood cell is approximately 7 microns in diameter.

The thin walls of the alveoli allow gases to pass through readily by the process known as diffusion. This very thinness, however, can be a problem for divers as over-distension may result in pulmonary barotrauma.

In order to preserve the alveoli walls in a state readily suitable for gaseous exchange they are coated with a material known as the surfactant. This material prevents body fluids from exuding into the alveoli. When the surfactant is removed, as in drowning or salt water aspiration, the fluids are able to build up in the lungs causing further problems such as pulmonary oedema.

CHANGES IN GAS CONCENTRATION DURING DIVING

During the process of breathing, air containing oxygen is taken into the lungs. Immediately upon entering the body, changes to the percentage levels of the gases begin to take place. The most noticeable addition to the air is water vapour.

By the time air has reached the trachea it has taken up 47mm Hg of water vapour. Also, by the time the air reaches the alveoli, it has picked up considerable quantities of carbon dioxide and water vapour.

The carbon dioxide has come from the dead air spaces where it has accumulated after the previous expiration and combines with the newly inspired air. Moisture is created in the nasal passages as well as in the alveoli beds themselves.

Once the newly inspired air reaches the alveoli, oxygen is absorbed through the alveoli walls and into the blood stream. Carbon dioxide is eliminated into the alveoli along with some moisture. Since the tissues cannot produce as much carbon dioxide as oxygen consumed, venous blood returning to the lungs has a lower total gas pressure.



It is obvious that the venous blood is lower in oxygen and higher in carbon dioxide. When under pressure, Dalton's Law becomes of importance with the result that as the total pressure increases, the partial pressures increase proportionally.

This in itself does not cause problems unless the partial pressure of the gases reaches a sufficiently high level to cause the following gas poisonings; carbon dioxide, oxygen and nitrogen narcosis.

Changes in gas levels in the blood will cause some problems and will be discussed in further detail under Circulation.

CIRCULATORY SYSTEM

■ BLOOD

The circulatory system consists of three parts. They are the blood, the heart and the blood vessels.

Blood is responsible for carrying nutrients, oxygen, and waste products to and from the body's tissues. There is approximately 5-6 litres of blood in a human adult, and at rest, this circulates once every minute.

It has 4 major components:



- ✓ Plasma is the fluid part (50%) which carries dissolved nutrients and waste products, as well as the cells listed below.
- ✓ Red blood cells constitute 45% of the blood and carry an oxygen binding substance called haemoglobin.
- ✓ White blood cells help make up the remaining 5% and are mainly responsible for fighting infection.
- ✓ Platelets start the clotting process when a blood vessel is injured.

The most important part of blood as far as respiration and resuscitation is concerned is the red blood cells, known as erythrocytes. Red blood cells contain an iron-based protein known as haemoglobin which gives the blood its red colour. Haemoglobin is responsible for binding oxygen and hence its consequent transport to the tissues. When bound with oxygen it is known as oxyhaemoglobin and has a bright red colour. When free of oxygen, blood has a darker appearance. Haemoglobin plays an important role in carbon monoxide poisoning where carboxy-haemoglobin is formed and anoxia results.

■ THE HEART

The major function of the heart is to serve as a muscular pump, propelling blood to and from all parts of the body via the blood vessels. The heart is a 4 chambered muscular, hollow organ situated between the lungs in the mediastinum. It lies slightly left of the sternum.

The structure of the heart includes myocardium, valves, and arteries which supply the heart known as coronary arteries. The pericardium is a protective sac containing the chambers. It consists of two membranes, between which is a small amount of fluid which acts as a lubricating layer to prevent tissue damage as the heart beats.

The wall of the heart consists of 3 layers of which the middle layer is responsible for the ability of the heart to contract. This middle layer is known as the myocardium, a muscle which can beat continuously throughout life without needing long periods of rest.



The heart is divided into the right and left halves, with two chambers in each. The upper chambers are referred to as the right and left atria and lower chambers are called ventricles. The right atrium is a thin-walled chamber which receives blood from all tissues except the lungs. The vena cava brings blood to the right atrium which then drains into the right ventricle. The pulmonary artery, which carries de-oxygenated blood, leaves the right ventricle.

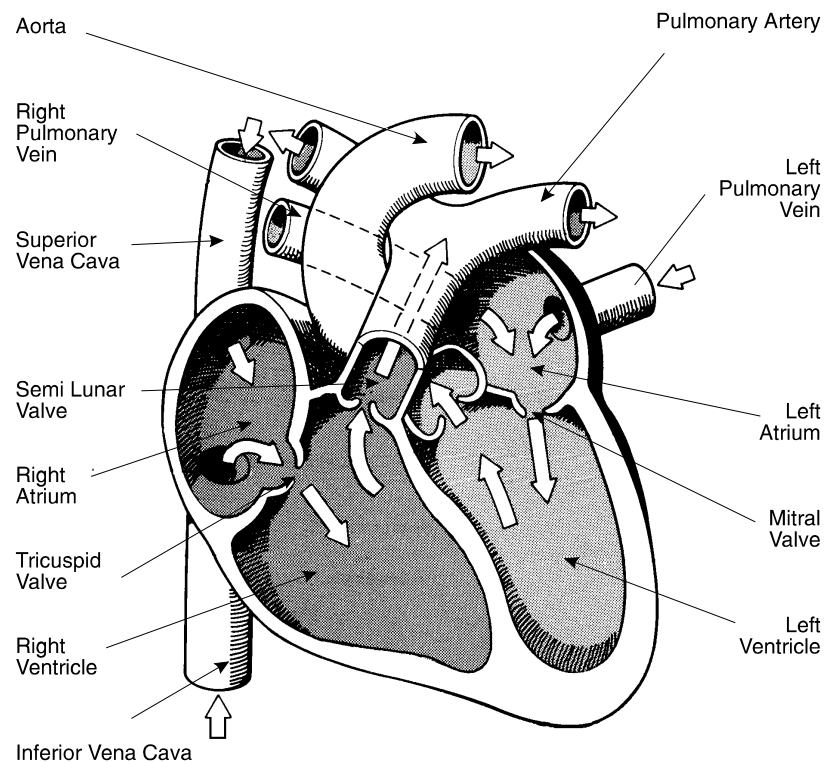


Figure 4. The Heart

The left atrium receives the pulmonary vein(s), draining oxygenated blood from the lungs. Blood then flows into the left ventricle which has walls that are three times as thick as the right ventricle to enable adequate pressure to pump blood throughout the body. From here blood is forced to all parts of the body except the capillary beds of the lungs via the aorta.

There are two types of valves located in the heart to prevent back flow of blood. These are the valves (tricuspid and mitral) located between the atria and the ventricles, and the pulmonary and aortic valves found where the pulmonary artery and the aorta leave the heart.

When the ventricles contract there is an increase in pressure, this is called systolic. As ventricles relax after contraction, the atria fill with blood. The resulting pressure is referred to as diastolic. Hence, when we refer to blood pressure as 120 over 80, it is the systolic over diastolic pressure measured in millimetres Mercury (mm Hg).

■ CARDIAC OUTPUT

As previously stated, under resting conditions, cardiac output is approximately 5 litres per minute.

The output of the heart depends on 3 things:

- ✓ venous return
- ✓ cardiac rate
- ✓ forces of cardiac contraction i.e. stroke volume



The following can affect venous return and hence cardiac output.

INCREASE RETURN	DECREASE RETURN
Hard work	Shock
Breathing in	Continuous standing
Pressure gradients within the circulatory system	Hypothermia
Hyperthermia	

■ BLOOD VESSELS

The blood vessels consist of a closed system of fibres that transport blood to all parts of the body. They consist of arteries and arterioles, capillaries, veins and venules.

Arteries receive blood from the heart at high pressure and are thick walled with elastic tissue and muscle. Arteries branch into smaller and smaller arteries, finally terminating in short, narrow, muscular vessels called arterioles. Blood then enters tiny thin permeable tubes known as capillaries, which serve as both destination and boarding points for gases, nutrients, and waste products. Here the blood moves more slowly and under lower pressure to the venules which converge to form veins which ultimately return the blood to the heart.

Veins generally carry deoxygenated blood to the heart except for the pulmonary vein which carries oxygenated blood. Arteries generally carry oxygenated blood away from the heart except for the pulmonary artery which carries deoxygenated blood to the lungs.

The following table shows the major differences between the blood vessels.



VESSELS	PROPERTIES
Arteries	Carry blood from the heart. High pressure vessels. Thick, strong elastic walls. Muscles in walls expand with pulse.
Capillaries	Very small (0.2mm diameter). Acts as a reservoir (contain 1/6 total blood volume circulating). Allows diffusion of substances. Capable of vasodilation and vasoconstriction.
Veins	Thin walled and less elastic. Low pressure. Returns blood to heart via a system of valves. Greater number of veins than arteries. Closer to surface.

■ THE CIRCULATORY SYSTEM

The circulatory system consists of two systems; the systemic and pulmonary.

SYSTEMIC CIRCULATION

The systemic system carries oxygen and nutrients to the entire body except for the lungs.

The systemic arteries emerge from the left ventricle via the aorta. Other major arteries include the carotid arteries in the neck, the brachial and radial arteries in the arms, and the femoral arteries in the legs.

After oxygenated blood is transported through the body's capillary beds, the deoxygenated blood is returned to the right atrium via the vena cava.



PULMONARY CIRCULATION

The pulmonary system carries blood from the right ventricle via the pulmonary artery to the lungs where gas exchange takes place and the blood is oxygenated. The pulmonary artery branches into the left and right pulmonary arteries. After oxygenation in the capillary beds of the lungs, blood is returned via the pulmonary vein to the left atrium. A pulmonary embolus (blockage of the pulmonary artery) can result from a blood clot, air, fat, tumour, or clumps of bacteria. The most likely outcome of this condition is cyanosis, breathing difficulties and death.

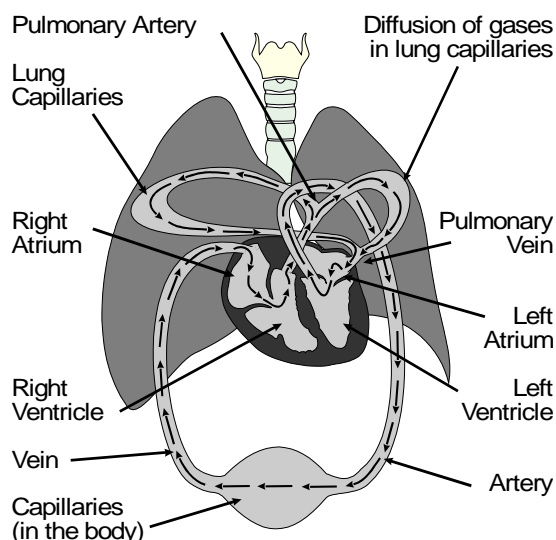


Figure 5. The circulatory system

GAS EXCHANGE

ROLE OF THE BLOOD IN GAS TRANSPORT

The prime function of the blood is to carry O_2 to the cells of the body and to remove the carbon dioxide, water vapour and waste products. For example carbon dioxide is removed via the lungs.

OXYGEN TRANSPORT

Oxygen is picked up from the lungs in two ways. The majority (approximately 19mls of O_2 per 100mls of blood) is chemically combined with haemoglobin. This mechanism produces the compound oxyhaemoglobin.

Less than 0.5mls of O_2 per 100mls of blood dissolves directly into the fluids of the blood. This process is controlled to a large extent by both Dalton's and Henry's Law. As haemoglobin is the major transport agent for oxygen, the amount of haemoglobin present will determine the amount of oxygen to be carried. Conditions such as anaemia will result in low oxygen levels in the blood and therefore tissues.

CHANGES IN GAS CONCENTRATION DURING DIVING

During diving the mechanism for oxygen transport remains virtually unchanged. The only alteration is in the amount of oxygen carried in the blood plasma. This amount will increase according to Henry's Law.

Instead of 0.3 - 0.5ml of dissolved oxygen per 100ml of blood, the amount will increase proportionally so at 10 metres it will be approximately 1ml and at 40 metres it will be approximately 2mls.



At depths in excess of 90 metres the body's minimal oxygen requirements could theoretically be supplied by the blood plasma alone. The body will tend to use the dissolved oxygen in preference to the chemically combined O₂ in the haemoglobin.

■ TACHYCARDIA/BRADYCARDIA



A natural response to changes in carbon dioxide and oxygen levels is a change to heart rate. When the heart rate speeds up it is known as tachycardia. Tachycardia is caused by high CO₂ levels, excessively low O₂ levels and exercise. Tachycardia is also associated with increased blood pressure and vasodilation. When the body suffers respiratory distress or O₂ build up every effort is made to increase blood flow to reduce the problem. When the heart rate slows down it is known as bradycardia.

Bradycardia is caused by low O₂ levels and hyperventilation. A third cause is from repeated breath hold diving resulting in the development of a diving response.

VESTIBULAR SYSTEM



■ INTRODUCTION

The vestibular system refers to the ears, hearing and balance organs.

The ear is physically divided into three major areas; the outer ear, the middle ear and the inner ear, all of which are subject to injury.

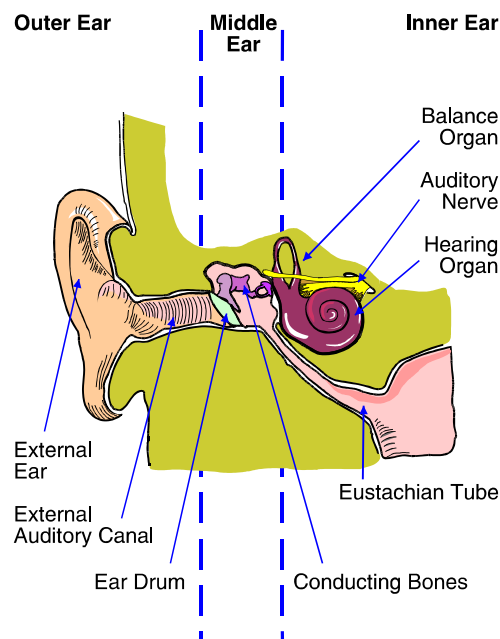


Figure 6. The Ear

The eardrum (called the tympanic membrane) separates the air-filled external ear and middle ear. The fluid-filled inner ear, containing the balance and hearing organs is surrounded by bone, and because it is not an air space it is not subject to volume changes.

The outer ear collects sound waves and directs them down the ear canal to the eardrum (called the tympanic membrane) which is a thin, elastic membrane that vibrates. The vibrations are then transmitted across the three conduction bones in the middle ear to the



hearing organs in the inner ear. Here they are converted into nerve impulses and hence a sound is registered by the brain.

The narrow Eustachian is a soft tube of tissue and connects the middle ear to the back of the throat. This is the passage that allows air to flow into and out of the middle ear to ensure that pressure remains equal.



The opening at the back of the throat does not normally open until we swallow or yawn. These are two methods people use to adjust for changes in ambient pressure when flying or travelling over hills where the pressure change is very slight in comparison to going underwater. The very fact that the pressure changes so quickly when we go underwater is the reason why it is important that equalisation of pressure is achieved before pain is felt.

The aim of equalisation is to open up the Eustachian tube and allow air through to the middle ear. Equalisation can be achieved in a variety of ways: swallowing, wiggling the lower jaw, yawning, or by sealing the nostrils, gently blowing and swallowing (the Valsalva manoeuvre). A sound or pop, similar to that experienced when driving up a mountain, may be heard when equalisation has been achieved. Alternatively, a squeaking noise or no sound at all may be heard but the important thing is that you do not feel pain.

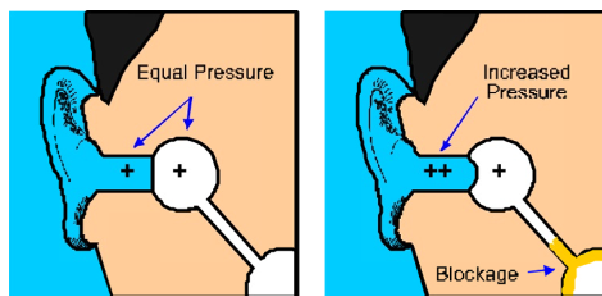


Figure 7. Equalisation (left) normal ear, (right) blocked Eustachian tube

Equalisation is usually easier if carried out in a head up, feet down position, while descending. Any conditions which block up the Eustachian tube, such as colds, flu, allergies, growths or heavy smoking, can hinder equalisation. Trying to equalise with a cold can push infected mucus into the middle ear and cause a painful middle ear infection. Ear infections or injuries automatically exclude a person from diving until completely healed.

THE LYMPHATIC SYSTEM

■ INTRODUCTION

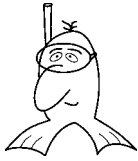
Within the tissue spaces of the body the lymphatics provide an auxiliary vessel system for returning fluids back into the circulatory system. The fluid in this system is called lymph fluid and it originates from the intercellular spaces.

Lymph is similar in composition to plasma, except that it has no high molecular weight proteins, has a large number of white cells, has few platelets, and few red blood cells. The lymphatic system consists of the lymph capillaries, lymphatic vessels, lymphatic ducts, and lymph nodes.

Lymph capillaries carry lymph from the tissue spaces to the lymph vessels. They are thin walled tubes composed of a single layer of cells. Most digested fat is absorbed through the lymph capillaries of the intestine.



All lymph vessels are directed to the thoracic cavity. One-way lymph flow is maintained by valves. The lymph vessels converge into either the right or left (thoracic) lymph duct. Both ducts empty into the venous system.



Lymph vessels have three major functions. They include the return of vital substances to the blood vessels, providing drainage channels into lymph nodes, and aiding in absorption of digested fats. The most important function is the return to the blood of vital substances, particularly proteins which have leaked out from the capillary beds. Without this return, death could result in 12-24 hours due to low plasma osmotic pressure.

Lymph vessels also provide drainage channels into lymph nodes for toxic or malignant products and play an important role in absorption of digested fats. Lymph nodes are small oval bodies found in the lymph vessels.

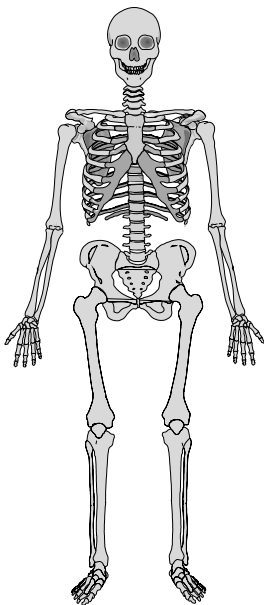
They have several functions. They filter and quarantine noxious products, digest foreign particles, produce some white cells and filter out red blood cells and bacteria. Knowledge of how lymph is transported back to the circulatory system is important in understanding the pressure immobilization technique as a first aid measure for envenomation.

Lymph vessels do not contract and rely on other factors for the movement of lymph fluid. Lymph movement results from new lymph pushing old lymph forward, pulsation of arteries and movements of muscle i.e. breathing movements. The most important mechanism for lymph propulsion is body movement, and the valves maintain a one way flow.

Body movement also increases the amount of lymph fluid formed, hence the need to keep a victim of envenomation still. Although the rate of flow of lymph fluid is slow, approximately 600ml per hour, it is still a major source of return of protein to the plasma. This rate is increased by body movement. The lymphatic system plays an important role in determining the first aid and management for envenomation.

SKELETAL SYSTEM

INTRODUCTION



The skeletal system forms a strong framework for the body. Bones combine remarkable strength with lightness and have the added benefit of being able to repair themselves. Inside bones is bone marrow, which makes blood cells.

The skeleton:

- ✓ gives shape to the body
- ✓ allows movement (muscles pull against bones)
- ✓ protects vital organs (ribs & skull)
- ✓ makes blood cells

The skeleton can be divided into three sections:

- ✓ the skull which consists of the cranium and bones of the face
- ✓ the trunk which consists of the spine, a strong flexible pillar that supports a number of structures of the body
- ✓ the limbs



NERVOUS SYSTEM

INTRODUCTION



The nervous system provides a communication infrastructure for the body. It provides the facility for collecting, interpreting and acting on an enormous amount of information gathered from and projected back in the environment.

The nervous system is particularly important in diving. Decompression illness (DCI) is a disease of the nervous system because nitrogen is very soluble in fat. Pain, which is the main symptom of DCI, may be due to stimulation of pain receptors around muscles and joints, referred pain from the central nervous system (particularly the spinal cord), activation of the immune system or ischaemia/infarction of bone.

THE CENTRAL NERVOUS SYSTEM

The nervous system consists of the central (CNS) and the peripheral nervous systems (PNS). The CNS comprises the brain (inside the cranial cavity), spinal cord (inside the vertebral canal), while the PNS (which lies outside the vertebral canal) consists of 12 pairs of cranial nerves, arising from the brain and 31 pairs of spinal nerves, arising from the spinal cord. There is also the Autonomic nervous system, which will be discussed later but is considered part of the PNS.

The unit of structure of the CNS is the NEURON. A neuron is a single cell, which has been drawn out in several directions into threads or processes. If we think of the nervous system as corresponding to a complicated electrical circuit then the neuron corresponds to a single length of wire and the brain as the main frame computer, which gathers and organises data and determines a response. In a wiring system a single wire is useless unless it is connected to other wires. Similarly, a neuron is unable to function by itself and is joined to other neurons to form a pathway for the transmission of nerve impulses. The junction between the neurons is called a synapse. A chemical transmitter is used to convey the impulse across this junction of synapse.

THE NEURON



Every neuron consists of a cell body and certain processes (dendrites and axons). A collection of cell bodies outside the CNS is called a ganglion.

A sheath of fat called myelin surrounds most axons and is called the myelin sheath. In sections of the brain and spinal cord there are 2 kinds of nervous tissue, one which looks grey and the other white. They are called, respectively, grey matter and white matter. Grey matter consists of an aggregation of cell bodies and therefore, does not contain myelin, while the white matter consists of an aggregation of nerve fibres and therefore will contain myelin. In other words, the grey matter has little fat while the white matter contains fat.

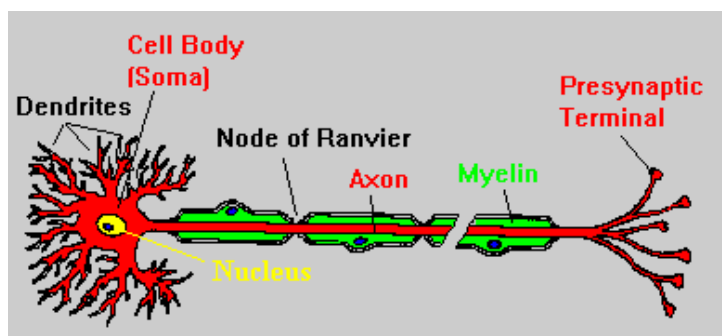


Figure 8. A neuron





THE BRAIN

The brain is in the cranial cavity and is surrounded by membranes called meninges.

It consists of the 2 cerebral hemispheres, the brain stem, the between brain and the cerebellum.

The 2 cerebral hemispheres are the largest part of the brain. They are connected to each other, about half-way down their medial surfaces by the corpus callosum. Each cerebral hemisphere controls the opposite side of the body (the right hemisphere controls the left side of the body). Each cerebral hemisphere is divided into 4 lobes: the frontal, parietal, temporal and occipital lobes.

The interior of the brain contains a series of cavities called ventricles. They are continuous with one another and contain cerebro-spinal fluid (CSF).

THE CEREBRO-SPINAL FLUID

CSF is a watery fluid that occupies the ventricles, central canal of the spinal cord and the subarachnoid space. It is constantly being formed in the ventricles and being absorbed in the subarachnoid space. It has a hydraulic function providing buoyancy for the brain and cushioning any sudden blows to the head.

CEREBRAL BLOOD FLOW

In humans the principal arterial supply to the brain is via the 2 internal carotid and 2 vertebral arteries. Marked fluctuations in regional blood flow occur with neuronal activity. Brain blood flow also varies with changes in arterial oxygen and carbon dioxide levels.

Because the brain and its circulation are enclosed in the skull, changes in volume of either the brain (due to injury), or the cerebral vessels (due to compromised venous drainage), or the cerebral ventricles (due either to obstruction to CSF absorption or circulation or haemorrhage into the ventricles) can produce dramatic changes in the blood flow to the brain which under normal conditions remains constant.

THE BLOOD-BRAIN BARRIER

The blood-brain barrier permits only water, carbon dioxide and oxygen to cross from the circulation into the brain without any difficulty. Other substances cross inversely proportional to their molecular size. The blood-brain barrier is important for maintaining a constant environment for the brain. Disruption of the barrier results in plasma proteins and other substances leaking into the brain producing swelling.



THE SPINAL CORD

The spinal cord, like the brain, developed from the neural tube. It is situated in the vertebral canal. It is a cylindrical structure but tapers at the lower end. It occupies the cervical and thoracic segments of the canal but ends at the lower border of the first lumbar vertebra. Below the termination of the spinal cord is a collection of nerves called the cauda equina (because it looks like a horse's tail). From the spinal cord arise 31 spinal nerves which are the beginning of the PNS.

In a cross section of the spinal cord the grey matter is in the centre of the cord roughly looking like an "H" and is divided into the ventral and dorsal columns. In the centre of the grey matter is the central canal containing CSF. The ventral columns are formed by effector neurons while the dorsal columns are formed by receptor neurons.

The white matter consists of nerve fibres (axons) collected into tracts or fasciculi and are classified as either ascending or descending fasciculi running to or from the brain.



THE PERIPHERAL NERVOUS SYSTEM

A nerve is a cord like structure consisting of a number of units called nerve fibres bound together by fibrous tissue in much the same way a number of wires are bound together to form an electric cable. The function of nerves is to convey messages or nerve impulses to and from the CNS. Nerve fibres can only carry impulses in one direction.

Afferent Fibres are nerve fibres that carry impulses to the CNS from the sensory receptors contained in the sense organs. Sensory receptors modulate energy. Because many variables can be perceived in the environment there are many types of receptors. There are at least 11 conscious senses and a large number of sensory receptors which relay information automatically. A particular amount of energy is needed to stimulate each type of receptor (to stimulate pain receptors requires a different amount of energy than that needed to stimulate temperature receptors, in other words sensory organs are energy specific). Nerve fibres which carry impulses away from the CNS are called efferent fibres.

In general, afferent fibres are sensory fibres, while efferent fibres are motor fibres (they carry the message to the effector organ to perform the determined response). Some venom (see Dangerous Marine Animals) cause paralysis by blocking the motor nerves, they do not block the sensory nerves. In DCI the sensory fibres are stimulated first (pain and the sensation of 'pins and needles'), motor paralysis may occur later (the diver will complain of feeling weak or a 'heavy sensation' in the lower or upper limbs).

THE SPINAL NERVES

There are 2 roots for each spinal nerve, the dorsal root which is afferent and the ventral root which is efferent. These 2 roots join beyond the dorsal root ganglion to form a single nerve.

There are 31 pairs of spinal nerves, which leave the vertebral canal by passing through the intervertebral foramina. These 31 nerves are made up of 8 cervical, 12 thoracic, 5 lumbar, 5 sacral nerves and 1 coccygeal nerve. Except in the cervical region all the nerves leave the vertebral canal below the corresponding vertebra. After leaving the intervertebral foramen each spinal nerve divides into 2 parts called the anterior and posterior primary divisions. These divisions are mixed nerves and supply the anterior and posterior muscles and skin of the thorax, abdomen, limbs and neck. In the thorax these divisions become the 11 intercostal nerves and the sub-costal nerve, in the lower limbs they form the lumbo-sacral plexus, the upper limb the brachial plexus and neck the cervical plexus. The area of skin and muscle controlled by each spinal nerve is called a "dermatome". Any abnormality in the spinal cord will affect the corresponding dermatome, hence examination of these dermatomes will reveal the position of the abnormality.

THE CRANIAL NERVES

There are 12 pairs of cranial nerves. Three of these arise from the brain, the rest from the brain stem. Therefore, any abnormality of the brain stem will affect the majority of these nerves, therefore, an examination of these is important because any abnormality of the "relay station" will affect the quality of impulse reaching the brain.

Some of the cranial nerves are purely afferent, some efferent while others are mixed (see Table following).



<i>The afferent nerves</i>	<i>Olfactory nerve (1st)</i> <i>Optic nerve (2nd)</i> <i>Vestibulo-cochlear nerve (8th)</i>
<i>The efferent nerves</i>	<i>Oculomotor nerve (3rd)</i> <i>Trochlear nerve (4th)</i> <i>Abducent nerve (6th)</i> <i>Hypoglossal nerve (12th)</i>



The mixed nerves	Trigeminal nerve (5th) Facial nerve (7th) Glosso-pharyngeal nerve (9th) Vagus nerve (10th)
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THE ASCENDING AND DESCENDING PATHWAYS

As an example of how neurons are connected to relay nerve impulses let us consider general sensation and its voluntary muscular response. The first set of neurons is the receptor neurons, which transmit the impulse from the peripheral sense organs to dorsal grey column where they may ascend the cord before their termination. The connector neurons cross to the other side and pass upward to end in the thalamus. From there another set of connector neurons pass through the white matter to end at the cerebral cortex. Once the appropriate response has been determined the impulse generated in the cortex passes down an upper neuron through the internal capsule and the white matter of the brain stem and spinal cord to the spinal cord's ventral grey column crossing over to the other side as it does this. In the ventral root the upper neuron synapses with the lower effector neuron, which travels to the muscle or organ and the appropriate response elicited.

THE REFLEX ARC/ACTION

If we touch a hot object our hand is immediately withdrawn. The action is involuntary although we are aware of it when it happened or immediately afterwards. There is no conscious decision to carry out this particular movement and thus it is referred to as a reflex action or act. The nervous pathway concerned is called a reflex arc. The afferent neuron enters the dorsal grey column where it synapses with a connector neuron passing to the ventral grey column. From here the effector neuron passes to the muscle or organ eliciting the response.

REFERRED PAIN

Referred pain occurs where impulses causing pain originate in a structure supplied by a certain nerve but is felt in another place in the body. An example of this is when something is irritating the diaphragm the patient complains of shoulder tip pain.

Several explanations have been proposed for referred pain:

- ✓ It may represent misinterpretation by the cerebral cortex;
- ✓ Two groups of afferent fibres lie next to each other and there is some spill over from one to the other; or
- ✓ The parts of the body developed from the same embryonic tissue and therefore share the same developing neurons.

Referred pain from the spinal cord is one of the causes of pain in DCI.

THE AUTONOMIC NERVOUS SYSTEM



The Autonomic Nervous System presides over matters in the body “that are independent of will” or as it is often referred to as the “independent nervous system”. It is organised on the principle of a reflex arc. Impulses are initiated in the visceral receptors and relayed via afferent autonomic pathways to the visceral effectors. There are 2 component systems - the sympathetic and parasympathetic nervous systems.



THE SYMPATHETIC NERVOUS SYSTEM

The sympathetic nervous system presides over the heart rate, movements of the gut, secretion of the skin's sweat glands and the calibre of arteries and veins. For example, a change in posture from a sitting to a standing position will cause a change in tone of various blood vessels. Some are constricted and others dilated to maintain cerebral perfusion and blood pressure without a change in pulse rate. During hypovolemic shock the sympathetic nervous system is already trying to compensate and so a sudden change in posture may cause a quickening of the pulse and a drop in blood pressure which will cause a drop in the cerebral circulation - the patient will complain of feeling dizzy.

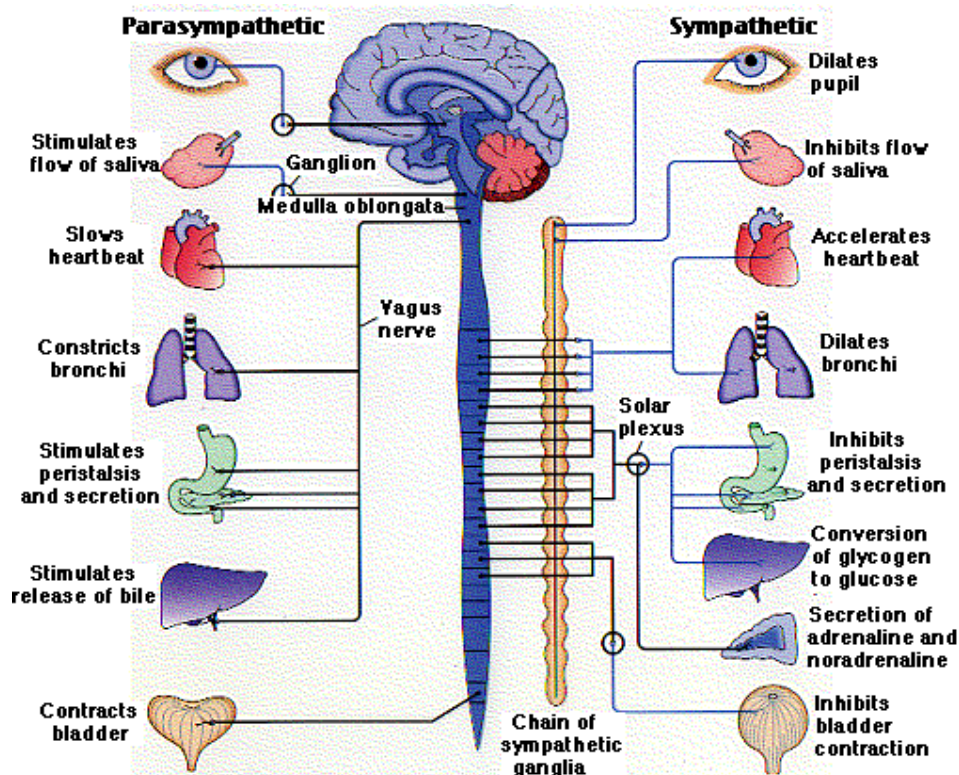


Figure 9. The autonomic nervous system

THE PARASYMPATHETIC NERVOUS SYSTEM

The Parasympathetic Nervous System has two "outflows" - from the 5th, 7th, 9th and 10th cranial nerves and the ventral roots of the sacral nerves. It controls the eye's iris, salivary glands, heart, lungs, alimentary canal (gut motion and the ability to control defecation) and bladder function.

AUTONOMIC PHARMACOLOGY

Various drugs can mimic the action of the transmitters in the autonomic nervous system. They can change the blood pressure, pulse rate, cause diarrhoea, constipation, increase gland secretion and change cerebral function. For example, an overdose of pseudoephedrine (Sudafed) can cause a tachycardia, hypertension, diarrhoea and aggressive behaviour.



THE IMMUNE SYSTEM

INTRODUCTION

The human body has a series of protective processes to guard against invasion from foreign bodies.

- ✓ The lungs filter out airborne foreign material
- ✓ Tonsils act as a filter for disease organisms entering via the mouth
- ✓ Bone marrow produces stem cells, which develop into white cells in the thymus
- ✓ Lymph nodes act as reservoirs for white blood cells which are deployed when foreign cells are detected
- ✓ The liver, spleen and intestines clean the blood of foreign matter

THE HUMAN IMMUNE RESPONSE



This response occurs when a foreign invader's antigen (a substance, which elicits an immune response) alerts the body's immune response.

- ✓ Special white cells produce antibodies which recognise a specific antigen and attach to them
- ✓ Macrophages recognise and surround the antigen/antibody complex, and digest and destroy the invader via a process called phagocytosis
- ✓ Macrophages free the antigens, which act as flags to signal the immune response and aid in the recognition of the next invader

THE IMMUNE RESPONSE AND DIVING

The immune response is invoked whenever the body perceives an invasion by any foreign material.

For the diver, this response will be triggered by:

- ✓ bubbles resulting from decompression sickness or air embolism
- ✓ salt water aspiration
- ✓ carbon monoxide

EFFECTS OF THE IMMUNE RESPONSE TO BUBBLES

The presence of bubbles produces a series of responses in the immune system that complicates the previously held ideas regarding the effects of decompression sickness.

- ✓ Certain protein molecules adhere to the bubbles, and phagocytes (white cells) rush to the site
- ✓ Mast cells, which are usually found on the outside of blood vessels, are attracted also
- ✓ Protein cells release fatty acids from the cell, producing fat emboli



- ✓ Mast cells release histamine which increases blood flow by dilating venules and capillaries
- ✓ It also makes the endothelium sticky. This causes white cells to stick to the walls in a process called margination
- ✓ Margination causes the endothelium cells to separate, leading to leakage of fluid from the capillaries
- ✓ Gas bubbles can also permeate cells walls and finish up in the lymphatic system
- ✓ Injured cells release chemicals that attract platelets, which promote the formation of clots
- ✓ Platelets may also attach themselves to bubbles, creating larger mass, and resulting in further blockage

This will lead to:

- ✓ inflammation
- ✓ loss of blood plasma
- ✓ tendency of clots to form
- ✓ victim feeling unwell
- ✓ fever



2

CHAPTER 2 – DIVING MALADIES

INTRODUCTION

ADAS COMPETENCY

Maintain Health and Safety of Individual and Others.

Identify and explain hazards other than those directly relating to diving affecting individuals of a dive team.

Assist in Treatment of Diving Related Ill Health Conditions/Illness.

Explain the cause and effects of diving related ill health conditions.

GENERAL

This section, deals with the many diving ailments that can affect the occupational diver. Each topic is dealt with in a similar way; cause, signs and symptoms, management (or first aid) and prevention are all discussed.

The occupational diver must have a basic knowledge of the cause of an ailment, how to recognise it, how to treat it and, more importantly, how to prevent it.

The causes of diving accidents are many and varied but can generally be categorized into direct effects of pressure (Boyle's Law), indirect effects of pressure (Dalton's Law and Henry's Law) and exposure.

Signs which the first aider can see, such as bleeding, and symptoms which the victim feels, such as pain, are grouped together to avoid confusion.

Management of a diving related illness is important. If properly administered, first aid will prevent further damage and can save a life. Obviously, the need for first aid will be avoided if the ailment can be prevented. Prevention will result from knowledge and sound dive planning.

GAS SPACES IN THE HUMAN BODY

ADAS COMPETENCY

Demonstrate an understanding of the effects of pressure, volume and temperature changes on the diver and their implications.

List gas spaces in the human body.

Define the term barotrauma.

List some typical barotrauma injuries.

INTRODUCTION

Any gas space will be subject to the effects of pressure changes. This includes those gas spaces in or around the body.

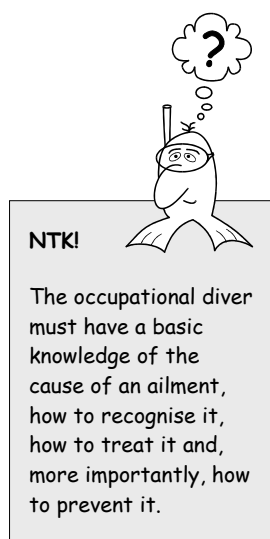
Gas spaces in the body:

- ✓ lungs
- ✓ middle ear
- ✓ sinuses
- ✓ gas in the gut and intestinal tract

Gas spaces around the diver:

- ✓ air inside the mask





- ✓ air inside the dry suit
- ✓ air inside the buoyancy compensator

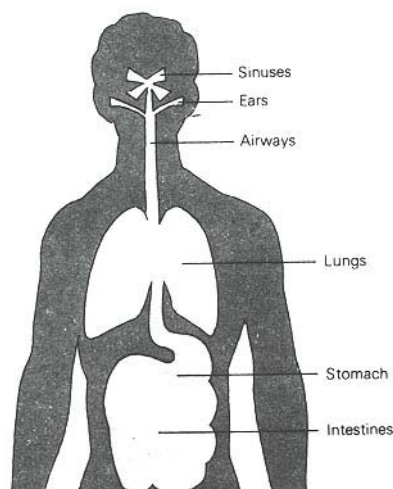


Figure 10. Gas spaces in the body

■ BAROTRAUMA

The pressure related problems which may arise as a diver descends in water, will manifest themselves in one or more of these natural air spaces, or equipment which places an air space next to the diver's body and then cannot be equalised. Natural air spaces have vents which normally allow pressure to equalise. The lungs are ventilated by breathing, the middle ear and all the sinuses are connected by air passages with the throat and nose. If these passages should become blocked, air trapped in the spaces cannot be equalised as the diver descends.

Damage caused by pressure changes is called barotrauma.

- ✓ Baro = pressure
- ✓ Trauma = injury or damage

TYPICAL BAROTRAUMA INJURIES

■ MIDDLE EAR BAROTRAUMA OF DESCENT (MIDDLE EAR SQUEEZE)

CAUSE

This is the most common barotrauma in sport divers. The primary cause is failure to equalize pressure in the middle ear during descent.

SIGN AND SYMPTOMS

Descents should not be continued if pain is felt. Pain indicates abnormal inward bulging of the eardrum and swelling of tissue lining in the middle ear. Pain is experienced by most people at depths greater than 1.5 metres when equalization has not occurred. If descent is



continued, blood vessels will burst in an effort to fill the gap left by compressed air in the middle air. Bleeding will cause a feeling of fullness in the ear and also impair hearing.

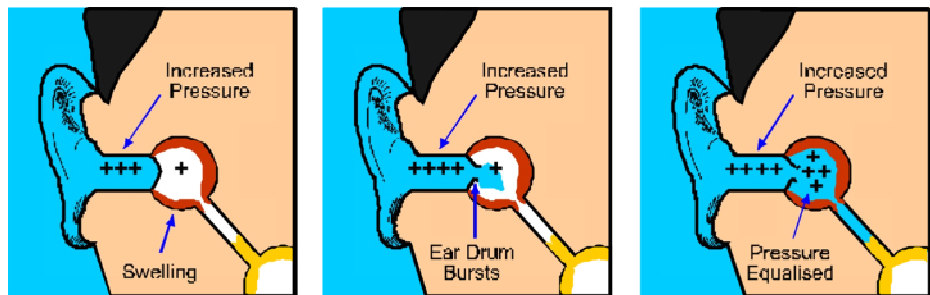


Figure 11. (Left) Swelling of the tissue lining in the middle ear causing blood vessels to rupture

Figure 12. (Middle) Ear drum bursts and cold water enters the middle ear

Figure 13. (Right) Pressure is equalised.

Should a rapid descent continue without equalization the eardrum may burst (rupture), thus immediately relieving pain or feeling of pressure and allowing cold water to enter the middle ear. If the cold water comes in contact with the inner ear the temperature difference may cause a movement of fluid in the balance organs, causing dizziness (vertigo) and sometimes nausea. Bleeding from the ear canal may indicate a burst eardrum.

It should be noted that not all signs and symptoms occur together, nor do they occur immediately. They may take several minutes to manifest themselves.

MANAGEMENT



Should a ruptured eardrum or middle ear barotrauma be suspected, a hand should be placed over the ear (both if you are unsure which is affected), to minimize the entry of water which will cause dizziness. If dizziness occurs, positive buoyancy must be achieved by inflating the buoyancy compensator or by ditching the weight belt. Dizziness usually lasts until the temperature of the water inside the middle ear equalizes with that in the inner ear. This may take about thirty seconds. Hold onto something solid until dizziness subsides.

The diver then must immediately surface, dry the area around the ear and cover it with a clean cloth (e.g. a towel). Nothing should be placed in the ear canal. It is advisable not to equalize on the surface as this may worsen any small perforation which has occurred. A suspected middle ear barotrauma or ruptured eardrum requires medical attention. Consultation with a diving doctor is highly recommended.



MANAGEMENT SUMMARY

- ✓ DRABC
- ✓ Observe and assess the signs and symptoms
- ✓ Cover ear or ears with hand or hands
- ✓ Hold on until dizziness passes
- ✓ Surface
- ✓ Dry and cover ear
- ✓ Do not attempt to compensate ears
- ✓ Do not place anything in ear canal
- ✓ Seek expert medical advice immediately



TREATMENT

Treatment requires that the patient does not dive, swim, or fly for at least six weeks. Antibiotics may help to avoid infection, and decongestants will help reduce excessive fluid.

PREVENTION

To prevent middle ear barotrauma of descent make sure equalization occurs before pressure is felt. If ears do not clear easily on the surface, or if you are suffering from a cold, flu allergies or you are a heavy smoker, do not dive.



■ REVERSE EAR BAROTRAUMA (REVERSE EAR SQUEEZE)

If air pressure in the middle ear area exceeds the pressure in the external ear, the eardrum will bulge outwards and may ultimately rupture.

CAUSE

The outward bulging may be caused in one of two ways. The first is blockage of the Eustachian tube before ascent. This is usually a result of tissue swelling around the opening of the Eustachian tube in the back of the throat. Swelling can occur after the effects of a decongestant have worn off. It is essential to avoid diving while taking any medication.

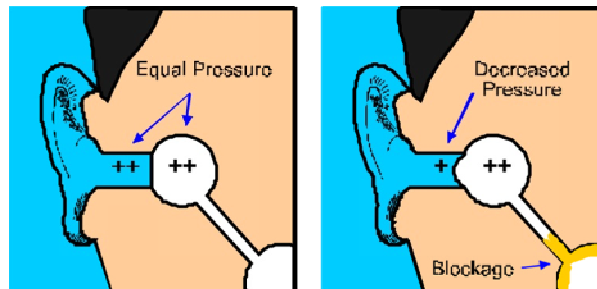


Figure 14. (Left) Normal ear at depth

Figure 15. (Right) Reverse Ear Squeeze of ascent. Note the mucus blockage.

The second is blockage of the outer ear canal which occurs due to ear plugs, excessive wax, or a tight fitting hood while descending. It can be seen in the diagram that equalization cannot occur without causing the eardrum to rupture.

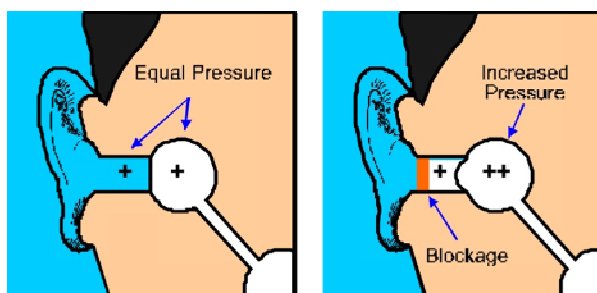


Figure 16. (Left) Normal ear

Figure 17. (Right) Reverse Ear Squeeze of descent. Note the blocked outer ear. i.e. wax

SIGNS AND SYMPTOMS

Signs and symptoms may include difficulty in clearing ears, pain and/or bleeding from the ear.



MANAGEMENT

The ear must be kept dry and a doctor's opinion must be sought.

**MANAGEMENT SUMMARY**

- ✓ DRABC
- ✓ Observe and assess the signs and symptoms
- ✓ Cover ear or ears with hand or hands
- ✓ Hold on until dizziness passes
- ✓ Surface
- ✓ Dry and cover ear
- ✓ Do not attempt to compensate ears
- ✓ Do not place anything in ear canal
- ✓ Seek expert medical advice immediately

**PREVENTION**

To prevent reverse ear squeeze the diver must ensure that the ear canals are clear. Avoid the use of ear plugs. If a tight hood is the problem, small holes about three or four millimetres in diameter may be cut in the hood near the ears to allow free flow of water. Diving with colds, ear infections, or after excessive smoking, must be avoided.

■ INNER EAR BAROTRAUMA (INNER EAR RUPTURE)

CAUSE

Sometimes difficulty in equalizing may be experienced. Should this occur, forceful equalization must not be attempted as serious, permanent damage to the inner ear can occur.

During descent, if equalization is not achieved, the eardrum bulges inwards, therefore pushing the conducting bones out of position. Should equalization be achieved suddenly, the conducting bones will rush back into position, thereby causing a movement of inner ear fluid which may rupture one of the small membranes dividing the middle ear from the inner ear.

SIGNS AND SYMPTOMS

Should a rupture occur, inner ear fluid will leak out resulting in mild to severe deafness. Deafness may be permanent if treatment is not sought immediately.

Signs and symptoms include ringing or buzzing noises, dizziness and disturbed balance, nausea and vomiting.

MANAGEMENT

A diver suspecting this injury must surface immediately. Once on the surface, it is important to prevent further fluid loss by keeping still and upright, and avoid exertion. Do not allow the patient to do the Valsalva manoeuvre. Permanent damage may be avoided by administering 100% oxygen and seeking immediate specialist medical attention via a diving doctor.



**MANAGEMENT SUMMARY**

- ✓ DRABC
- ✓ Observe and assess the signs and symptoms
- ✓ Cover ear or ears with hand or hands
- ✓ Hold on until dizziness passes
- ✓ Surface
- ✓ Dry and cover ear
- ✓ Do not attempt to compensate ears
- ✓ Do not place anything in ear canal
- ✓ **Keep still and upright, and avoid exertion**
- ✓ **Administer 100% Oxygen**
- ✓ **Seek expert medical advice immediately**

TREATMENT

Treatment may involve surgical repair, bed rest and the cessation of further diving or flying.

PREVENTION

Rapid descents and forceful equalizations must be avoided. It is quite common, particularly on second dives the same day that difficulty in clearing the ears is experienced. If ears do not clear easily on the surface, do not dive.



■ ALTERNOBARIC VERTIGO (DIFFERENT PRESSURE DIZZINESS)

CAUSE

During descent or ascent the ears may not equalize simultaneously with the changing water pressure. The result is varying pressures within the middle ear.

SIGNS AND SYMPTOMS

The delicate balance organs are therefore stimulated to a varying extent resulting in vertigo (dizziness) and occasionally nausea.

MANAGEMENT

Should alternobaric vertigo occur on descent, your course of action should be to stop, hang on to something solid such as a rock, anchor, rope or buddy, and wait until the discomfort passes. Then proceed slowly ensuring that adequate equalization of both ears occurs. If the problem persists, surface immediately and seek medical advice. If dizziness occurs while ascending, once again stop and grasp something solid until the symptoms abate.

This condition can occur at any time to anyone. The need for a buddy system, so that assistance can be rendered, is apparent.

Alternobaric vertigo is generally a condition which occurs when inflammation of the Eustachian tube is present, particularly after having had a cold.



■ OTITIS EXTERNA (SWIMMER'S EAR OR OUTER EAR INFECTION)

CAUSE²

Repeated immersion can cause breakdown of the skin which lines the external ear canal and this then allows the bacteria and fungi which are normally present to multiply. It is a condition which most commonly occurs during saturation diving, although frequent immersion of the ears for any reason, such as an intensive diving course or holiday, can promote the condition.

SYMPTOMS AND SIGNS

Itching and/or a wet feeling in the affected ear. This may progress to severe local pain as the external ear becomes swollen and inflamed. There may be a foul smelling, creamy discharge from the ear. Swelling of lymph nodes in the neck, which may make jaw movement painful. Fever in severe cases.

TREATMENT

A temporary cessation of diving so that the ear can be kept dry. Continued use of aluminium acetate drops plus appropriate antibiotic ear-drops if necessary. In serious cases, systemic antibiotics may be necessary.

PREVENTION

This is a completely preventable condition. 7% Aluminium acetate solution, Aqua Ear or Otic Domeboro ear drops will prevent infection if applied after each wet dive. These solutions are bacteriostatic and astringent. Three or four drops of the solution should be poured into each ear in turn and left for a minimum of 5 minutes. During intensive diving operations the drops should be applied at least every morning and night, and after each wet dive or shower.



■ SINUS BAROTRAUMA (SINUS SQUEEZE)

CAUSE

Normally sinuses (hollow areas in the skull opening into the nasal passages) are clear and equalization occurs automatically. However, when a person suffers from a cold, flu, sinus infection, or allergy, blockages can occur thus trapping pockets of air. If diving is attempted, trapped air will expand and compress during ascent and descent respectively, thereby causing sinus barotrauma.

SIGNS AND SYMPTOMS

During descent the volume of air inside the sinuses will decrease. In an effort to equalize, the tissue lining in the sinus will swell, causing pain which will worsen on further descent. If descent is continued, bleeding into the sinus will occur so as to equalize the pressure. On ascent air will expand and force blood and mucus out of the sinus into the face mask. Pain may persist for hours.

MANAGEMENT

A preferred analgesic will relieve pain and no further treatment may be required. However, if pain or condition persists medical advice must be sought.

PREVENTION

Sinus barotrauma may be avoided by not diving with a cold, flu or sinus infection, and by descending slowly.

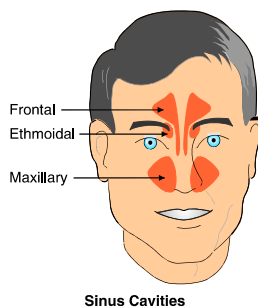


Figure 18. Location of sinus cavities



² From BR20806 Volume II, paragraph 1337.



■ MASK BAROTRAUMA (MASK SQUEEZE, FACIAL BAROTRAUMA)

To enable in-focus vision underwater a face mask must be worn. The mask provides an air space which is subject to volume changes during ascent and descent.

CAUSE

During descent, if the pressure between the face and the face mask is not equalized by blowing high pressure air through the noses, swelling of facial tissues and even of the eye will occur.

SIGNS AND SYMPTOMS

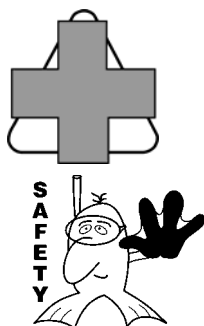
The first symptom of mask squeeze is a feeling of tightness on the face. If descent is continued the face may redden or the eyelids and become bruised and puffy. Red eyes may result from capillary rupture.

MANAGEMENT

Mask barotrauma is generally self-healing but diving must be terminated until recovery. If the eye is damaged seek medical advice.

PREVENTION

When pressure is felt, exhale into the face mask. Avoid the use of goggles that do not allow equalization.



■ TOOTH BAROTRAUMA (TOOTH SQUEEZE)

CAUSE

If tooth decay occurs, it is possible that a small air space will be created. Dental repair will usually eliminate any air spaces. However, should a small volume of air be present between a tooth and filling, a barotrauma can occur during a dive.

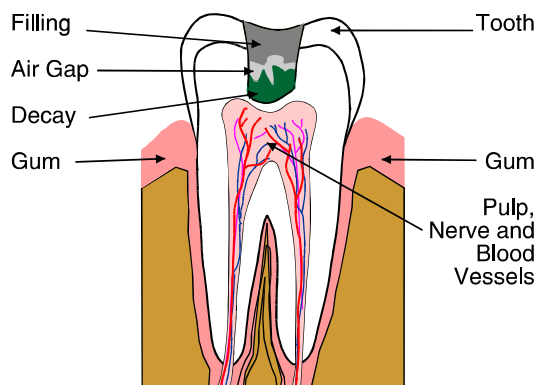


Figure 19. The Tooth

SIGNS AND SYMPTOMS

Pain due to swelling of the tissue and bleeding into the air space will occur during descent as gas volume decreases.

Upon ascent, the compressed air in the tooth will expand, causing mild to severe pain. In some cases the tooth may break due to increased internal pressure.





MANAGEMENT

A preferred analgesic will help ease the pain until the dentist can repair the affected tooth. Regular visits to the dentist will help eliminate the chance of suffering tooth barotrauma.

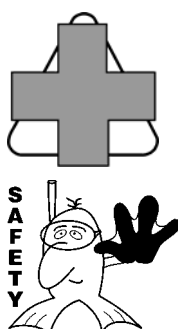
■ GASTRO-INTESTINAL BAROTRAUMA (GUT SQUEEZE)

CAUSE

Foods which produce gas during the digestive process can cause discomfort to the diver. Gas spaces produced in the stomach or intestine expand and compress during ascent and descent respectively.

SIGNS AND SYMPTOMS

Gastro-intestinal barotrauma will cause belching or burping, abdominal discomfort and colicky pains. If severe, fainting and shock may occur.



MANAGEMENT

Divers experiencing gastro-intestinal barotrauma should decrease rate of ascent, stop ascent until pain subsides, or descend to relieve pain. The diver can then slowly ascend.

PREVENTION

Avoid carbonated beverages and heavy meals before diving. Avoid over-breathing which can result in the diver swallowing air.

PULMONARY BAROTRAUMA

■ PULMONARY BAROTRAUMA OF DESCENT (LUNG SQUEEZE)

CAUSE

During a breath-hold dive the lungs become compressed with increasing depth (Boyle's Law). If the lung volume is reduced to a volume below the residual volume, tissue damage may occur. Residual volume is the volume of air left in the lungs after a complete exhalation. The average human lung capacity is 6 litres and the average residual volume is 1.5 litres. If an average breath-hold diver descends to thirty metres then his lung volume becomes 1.5 litres which is equal to the residual volume. Beyond thirty metres his lung volume is less than the residual volume and he may suffer pulmonary barotrauma (lung pressure injury) on descent.

SIGNS AND SYMPTOMS

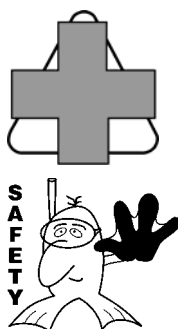
These include chest pain, coughing up blood and even death. Infection may follow due to lung tissue damage.

MANAGEMENT

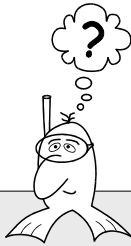
The victim should be treated for shock including administration of 100% oxygen if available. Medical assistance must be sought.

PREVENTION

Avoid breath-hold dives to excessive depths.



■ PULMONARY BAROTRAUMA OF ASCENT (BURST LUNG OR LUNG RUPTURE)



NTKI

Pulmonary barotrauma of ascent is a potentially fatal injury. The professional diver must have a basic knowledge of the cause of PB, how to recognise it, how to treat it and, more importantly, how to prevent it.

Pulmonary barotrauma (lung pressure injury) of ascent is the most serious of all diving ailments. As such any suspected pulmonary barotrauma requires urgent treatment.

It is one of the clinical manifestations of Boyle's Law. It is a rare occurrence in trained divers but the occupational diver needs to be aware of the implications and the need for utmost urgency when dealing with this problem.

CAUSE

If a compressed air diver holds his breath during ascent, expanding gases in the lungs cannot escape and tissue damage will result. The damage may vary from a small pinhole rupture of an alveolus to a larger tear in the lung tissue.

SIGNS AND SYMPTOMS

The onset of signs and symptoms is almost immediate. Most develop within five minutes. Immediate onset is a result of escaped gas expanding rapidly during ascent (Boyle's Law).

The signs and symptoms can be classified under two headings:

- ✓ behavioural
- ✓ physiological

Behavioural signs would include rapid uncontrolled ascent, sudden unconsciousness and appearing confused or disorientated. If a diver exhibits any of the above actions, pulmonary barotrauma should be suspected.

Physiological signs and symptoms can be divided into pulmonary and cerebral involvement. Pulmonary signs and symptoms include chest pains, difficulty in breathing, shortness of breath and occasionally bloody sputum. Cerebral signs and symptoms include confusion, visual disturbances, convulsions, and partial paralysis.

Due to the variety of signs and symptoms confusion with other ailments may occur. If ever in doubt as to the identity of a diving ailment always assume the presence of pulmonary barotrauma.

Reference texts books dealing with the advanced clinical manifestations of pulmonary barotrauma of ascent usually identify the specific problem under four basic headings.

Air Embolism (Or Cerebral Arterial Gas Embolism [Cage])

Air embolism literally means a blockage of the blood stream by an air bubble. The alveolus, a thin air sac embedded in a mass of capillaries, is where gas exchange takes place.

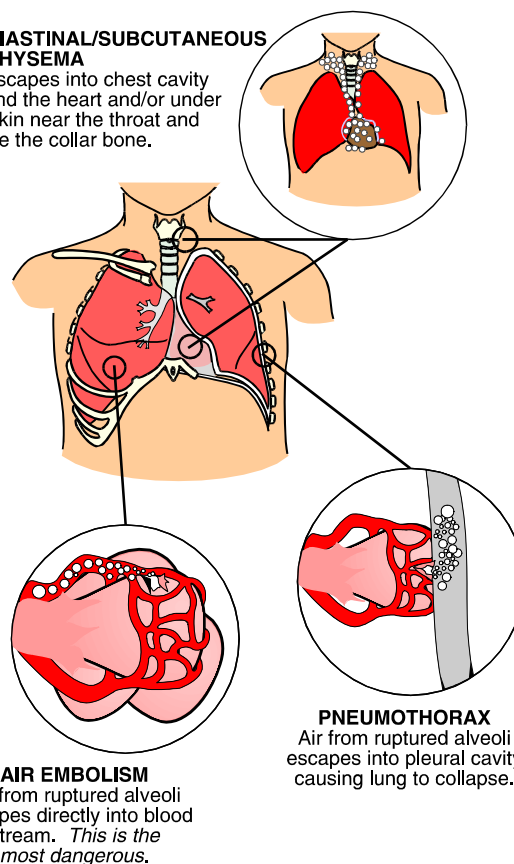
Should a small tear in the alveolus occur, air can escape directly into the blood stream and can be transported around the body. A small tear can occur from a pressure change as little as 0.1 atmosphere absolute (i.e. from one metre in depth to the surface). Of course, as the diver ascends, the bubble will increase in size (according to Boyle's Law).

A bubble in the blood stream will travel until it becomes lodged in a vessel of equal diameter. All tissues beyond the blockage will then be deprived of oxygen and other nutrients necessary to maintain function. Hence the signs and symptoms of air embolism will be determined by the location of the blockage. If the rupture occurs near the surface only a small vessel may be blocked. Therefore, the outcome may not be as serious. However, if the rupture should occur at depth, during ascent the bubble volume will increase and blockage of a major vessel may result, in which case the resultant signs and symptoms would be dramatic.



MEDIASTINAL/SUBCUTANEOUS EMPHYSEMA

Air escapes into chest cavity around the heart and/or under the skin near the throat and above the collar bone.



AIR EMBOLISM
Air from ruptured alveoli escapes directly into blood stream. *This is the most dangerous.*

PNEUMOTHORAX
Air from ruptured alveoli escapes into pleural cavity causing lung to collapse.

Figure 20. Pulmonary Barotrauma of Ascent

Mediastinal Emphysema

If air passes out of the alveoli it can travel through the passages between the lung tissue and lodge in the mediastinum. The mediastinum is the area in the middle of the chest where the heart lies. If air is in this area the condition is known as mediastinal emphysema. Emphysema refers to air in the tissues.

Because of the air pressure against the heart, chest pain, breathing difficulties, and shock are all signs and symptoms of mediastinal emphysema.

Subcutaneous Emphysema

From the central chest area, air bubbles can travel into the upper chest cavity and lodge under the skin in the neck region. The word subcutaneous is derived from sub meaning under and cutaneous meaning skin. Signs and symptoms include breathing difficulties, swelling in the lower neck region, crackling skin, voice changes and difficulty swallowing.

Pneumothorax

Surrounding each lung is a pair of thin, moist membranes called pleura. The area between these membranes is called the pleural cavity.

If an alveolus near the pleural lining ruptures, a tear may also occur in one of the membranes. If air escapes into the plural space it will cause the lungs to collapse. This condition is known as pneumothorax, meaning air in the chest. The word is derived from pneumo pertaining to air, and thorax referring to the chest.



Expanding air in the pleural spaces can also affect circulation. Chest pain, breathing difficulty, reduced chest movements, diver tending to lean to the injured side, shock and cyanosis (blueness) all indicate pneumothorax.

Summary of the Four Clinical Manifestations of Pulmonary Barotrauma



NAME OF AILMENT	LITERAL MEANING, SITE OF TRAUMA, (LOCATION OF AIR)	SIGNS AND SYMPTOMS
Pneumothorax	Air in the chest cavity.	Chest pain, shortness of breath, difficulty in breathing and cyanosis.
Subcutaneous Emphysema	Air below the skin.	Crackling sensation, voice alteration, fullness of the throat, shortness of breath.
Mediastinal Emphysema	Air in the middle of the chest below the sternum.	Heart irregularities, low blood pressure, pain behind breastbone, cyanosis.
Air Embolism	Blockage of blood vessels by air.	Sudden unconsciousness, confusion, visual disturbances, convulsions, pink frothy blood and shock.

Many cases of pulmonary tissue damage show little, if any, signs other than unconsciousness and respiratory arrest. If in doubt assume pulmonary barotrauma and treat as such.

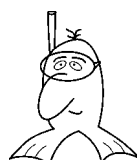
MANAGEMENT



The diver may be confronted with a huge range of symptoms when treating a pulmonary barotrauma case. It is essential that maintenance of breathing and oxygen therapy overrides the tendency to treat individual symptoms.

First aid should follow the following guidelines:

- ✓ Treat for shock by laying patient down flat, reassure, and protect from elements or further exposure, monitoring consciousness/pulse/breathing, and send for medical aid.
- ✓ Administer oxygen if available.
- ✓ If conscious, ascertain relevant details regarding the dive, e.g. depth/time, ascent rate controlled or not, equipment faults, previous activities, buddy comments. This can be carried out even while the patient is breathing O₂.
- ✓ Patient should be postured laying flat or left lateral (coma position) if unconscious.
- ✓ If on-site recompression is available, this should be utilised where appropriate.
- ✓ Arrange transport for further medical attention, preferably where recompression facilities are available. All haste must be made in contacting the local recompression facility and DES.



Remember: If any pulmonary barotrauma is suspected assume air embolism and treat as such. Do not try to recompress the victim in the water. Give pure oxygen.





MANAGEMENT SUMMARY

- ✓ DRABC
- ✓ Observe and assess the signs and symptoms
- ✓ Keep patient still
 - ✎ If unconscious lay in recovery position
 - ✎ If conscious lay down flat, legs level
- ✓ Reassure
- ✓ Administer 100% oxygen
- ✓ Observe and monitor airway, breathing and circulation (pulse)
- ✓ Resuscitate as necessary
- ✓ Protect from exposure
- ✓ Implement company's emergency plan or contact DAN/DES and arrange recompression — DES (Australia) 1 800 088 200
- ✓ Record all details (dive profile, signs and symptoms, first aid given)

TREATMENT

Treatment involves assessment of the patient to ascertain the extent and type of injury. Tests carried out by medical personnel include chest x-ray, blood pressure, electrocardiograms (ECG), lung function test (spirometry), electroencephalograms (EEG).

If the condition warrants recompression then the appropriate therapeutic table is chosen according to symptoms and depth at which symptoms are relieved. The treatment of choice is invariably a Table 62, 100% Oxygen at 18 metres.



PREVENTION

Prevention is simple. Do not hold your breath on ascent; always ascend slowly and breathe normally. Diving with a cold, hay fever, asthma, or if you are a heavy smoker may cause blockage of the alveoli due to excessive mucus or inflammation.

Therefore, even though you may be breathing normally, a rupture can occur on ascent. Be sure to avoid diving until condition has cleared up completely.

ABNORMAL VARIATIONS IN BODY TEMPERATURE



NTKI

Cold water is not only uncomfortable; it can also be very dangerous for those who are not properly protected.

THE PROBLEM OF EXCESSIVE BODY HEAT LOSS

We must now consider why excessive body heat loss (hypothermia) is so important to the diver. We must realise that cold water is not only uncomfortable; it can also be very dangerous for those who are not properly protected.

Hypothermia (loss of body heat to the surrounding environment) is derived from hypo, meaning low, and thermia, pertaining to temperature. Medically speaking, hypothermia is a condition where the body's core temperature drops below normal (37°C/98.6°F). The core temperature refers to the temperature of the vital organs such as brain, heart, lungs, liver and kidneys. It is essential that the core temperature be maintained. Should the body get too hot or cold, the functions of the vital organs will be impaired.



Human beings have evolved different responses to hot or cold stimuli, so that the normal core temperature can be maintained. For instance, when we get hot, the two most noticeable reactions are perspiration and a flushed appearance of the skin.

Perspiring cools the body by evaporation. The flushed appearance, particularly noticeable after a hot shower, results from vasodilation. Vasodilation involves dilation of the surface blood vessels which allows excessive heat to escape from the body. Alcohol and other drugs will also cause vasodilation. In response to cold stimuli, goose bumps, shivering and loss of colour occur. Goose bumps result from erection of hairs on our skin. When human beings were hairy, this hair would trap air and cause a layer of insulation over the skin, helping to prevent heat loss.

Unfortunately this has little effect in modern people. Shivering, however, is quite effective as it generates heat within our body. As we get colder, loss of colour occurs because the surface blood vessels are closed off in an effort to prevent heat loss via the skin. This is referred to as vasoconstriction, which is the opposite of vasodilation.

ADAS COMPETENCY

Demonstrate an understanding of the effects of pressure, volume and temperature changes on the diver and their implications.

Describe the factors affecting heat loss under water and its effect on the diver.

■ HYPOTHERMIA

CAUSE

When our body is immersed, heat is rapidly lost to the water. Water has 25 times the thermal conductivity of air. As a result, even in mild water temperatures (18-22°C/64-72°F), energy production within the body is less than the energy lost to the surrounding water, which can cause a dramatic drop in core temperature, i.e. hypothermia.

Many times the air temperature above the water is considerably colder than the water in which you may be diving. There may also be strong cold winds or water spray blowing over the boat. In such conditions the chill factor is much lower than the actual air temperature would indicate. A wetsuit worn out of the water in the cold wind has just the opposite effect than when being used underwater. Water evaporating from the wetsuit can quickly lead to hypothermia.

SIGNS AND SYMPTOMS

There are three stages to hypothermia. They are referred to as mild, moderate or severe in relation to different core temperatures.

Mild: (35 – 34 Degrees C)

Signs and symptoms of mild hypothermia are maximum shivering; pale, cool skin due to intense vasoconstriction, poor coordination, slurred speech, usually responsive, but with apathy and slowed thinking.

Moderate Hypothermia: (33 – 30 Degrees C)

Signs and symptoms of moderate hypothermia are: most shivering ceases, increased muscle rigidity, consciousness clouded, pulse and respiration slow and become difficult to detect.

Severe Hypothermia: (< 30 Degrees C)

Signs and symptoms of severe hypothermia are: progressive loss of consciousness, cardiac arrhythmias may develop, pupils fixed and dilated, may appear dead.



MANAGEMENT**MANAGEMENT SUMMARY**

- ✓ DRABC
- ✓ Observe and assess the signs and symptoms

COLD:

- ✓ Get out of the water and wind
- ✓ Remove wetsuit and put on dry clothes
- ✓ Warm shower
- ✓ Warm sweet drinks and rest

MILD HYPOTHERMIA:

- ✓ Remove from water
- ✓ Gentle re-warming by body heat - car heater
- ✓ Warm sweet drinks only if patient can swallow and is clear-headed

SEVERE HYPOTHERMIA:

- ✓ Body to body contact within a blanket
- ✓ Immediate transport to hospital
- ✓ Do not use alcohol or external heat

TREATMENT

The treatment for cold is to simply remove the patient from the water, and either a warm shower or dry clothes and warm food will resolve the problem. If the patient is active and aware, then they are only suffering from cold and can be easily treated on the spot.

Treatment of mild hypothermia requires far more care and attention for the prevention of shock. Treatment involves gentle rewarming with external source. Treat for shock and handle patient carefully. Give warm sweetened drinks. The patient should be taken to hospital if recovery is not apparent soon after treatment begins. Tasks which require fine motor co-ordination such as climbing up rocks and driving should be avoided. The patient will usually be exhausted even after the core temperature has gone back to normal. They should be encouraged to rest and sleep until they feel completely well.

Patients suffering severe hypothermia must be hospitalised where intense therapy will be needed if they are to survive. Treatment will largely depend on severity of symptoms and available facilities. Treatment may include use of thermostatically controlled baths, warming inspired air (38°C), intravenous fluids to correct abnormalities.

When dealing with people who are suffering from any degree of exposure, several things **MUST** be avoided.



- ✓ DO NOT apply excessive external heat
- ✓ DO NOT massage arms or legs
- ✓ DO NOT give alcohol
- ✓ DO NOT move patient excessively
- ✓ DO NOT leave patient alone



Excessive external heat may cause a condition known as after drop. This condition results from blood moving to the cold skin area and then flowing back to the core. This causes further core temperature drop. In severe cases care should be taken when removing patient from the water to avoid sudden redistribution of blood. Ideally keep them horizontal or knees to chest position.

GENERAL PREVENTION

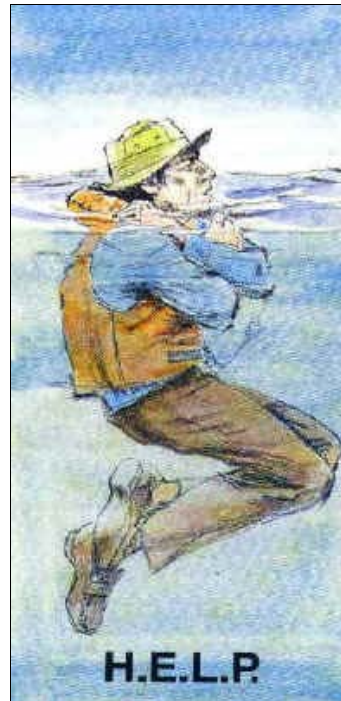


Figure 21. Techniques for keeping warm in the water – HELP – arms close to side of the chest, legs crossed and pulled up closing the groin area. HUDDLE – keep close together and still to keep colder water out.



Good environmental protection suits provide an insulating layer between the body and the surrounding water. Regular exposure to cold water over long periods will aid acclimatisation, therefore reducing reaction to lower temperature. Diet can also affect the onset of hypothermia. An increased deposit of body fat will aid insulation, but this will predispose you to decompression illness. Eating a sensible high calorie meal one to two hours prior to exposure will provide reserve energy. Heat loss can be minimised by reducing the amount of exercise in water. Although exercise does produce heat, the production of heat will not overcome the heat loss. Avoiding currents or excessive movement will enable flow of water over the body to be kept to a minimum.

Alcohol will reverse the body's normal reaction to the cold by causing vasodilation and further heat loss, and worsen the effects of hypothermia. Alcohol must be avoided before and after a dive. Repetitive dives should not be performed until the diver's response to heat and cold is back to normal. A snack and light exercise will achieve this. The signs and symptoms of hypothermia are such that they will often mask signs of other diving related problems. The drowsiness and lack of response, normally associated with carbon monoxide poisoning, will be very hard to determine when in the presence of hypothermia. Similarly, mild symptoms of decompression illness, such as skin rash and itchiness, can often be confused with the blotchy appearance and tingling of a cold diver. A thorough understanding of dive conditions and any associated problems arising from the dive must be sought by thorough, careful cross-examination to eliminate the possibility of any ailments co-existing with hypothermia.

Female Divers and the Cold

Despite the fact that women have a greater proportion of body fat to body weight than men, many women suffer from the cold. This is particularly evident in lean women whose surface



area is large compared to their body weight, therefore, increasing the rate of heat loss. As a result, many women will suffer from diving in cold water sooner than their male counterparts of comparable size and body weight.

While other factors may help, the best solution is a proper fitting, and in a lot of cases, a thicker environmental protection system. If a diver is prone to bad circulation in the hands and feet, gloves and boots will help alleviate these problems.

■ HEAT EXHAUSTION

When subjected to high temperatures, our body reacts by perspiring. Perspiring cools our body by evaporation. However, if excess perspiration occurs due to a priority for temperature regulation rather than maintenance of water and salt balance, severe dehydration and loss of sodium chloride will occur.

Heat exhaustion is characterized by severe muscle cramps, weakness, pallor, vomiting, vertigo, and fainting. These symptoms are due to lack of sodium chloride and can be prevented by taking enough salt tablets and water.

First aid includes resting in a cool place, keep patient cool, and give fluids with sugar, slowly at first. If unconscious, seek medical aid.



■ HEAT STROKE

Heat stroke is caused when persons exposed to excessive heat perspire and the loss of water leads to a decrease in plasma volume. This condition will lead to a decreased cardiac output resulting in a decreased blood flow to the skin. As the blood supply to the skin becomes inadequate, a major pathway for heat loss is decreased.

Body temperature will rise causing heat stroke. This condition can lead to cardiac failure and death. The most striking characteristic of heat stroke is lack of sweat. The skin is dry and flushed. Early signs include headache, blurred vision and vomiting.

Prompt first aid is required and medical help should be summoned urgently. First aid includes cooling and re-hydrating by use of cool packs, cool drinks (if victim can swallow) and a cool place. If unconscious, place the victim in the lateral position. Alcohol must not be given.

Prevention of both conditions includes avoiding prolonged exertion in warm climates, regular fluid intake and recognising early symptoms. Don't remain in wetsuit on hot days.



NTKI

All the gases we breathe on the surface, while causing us no harm, can be potentially dangerous underwater. Here we will identify how these gases can harm us and what we can do to afford the problems associated with gases under pressure.

GAS POISONINGS

■ INTRODUCTION

The earth's atmosphere is a mixture of many gases, most of which are poisonous at various concentrations. Oxygen (O₂), nitrogen (N₂) and carbon dioxide (CO₂) are the three most important gases affecting the air breathing occupational diver. The effect of various concentrations of these gases must be understood to avoid adverse physiological reactions.

■ OXYGEN POISONING

OXYGEN

Approximately 20% of the air we breathe is oxygen (O₂). Oxygen produced in plants by photosynthesis is essential for maintenance of life. The human body requires a partial pressure of oxygen between 0.16 and 0.21 atmospheres absolute (ATA) for normal function. Oxygen poisoning will occur if oxygen is breathed at partial pressures greater than 0.6



atmospheres absolute. Hypoxia, (low oxygen level), occurs when the partial pressure of oxygen breathed is below 0.16 atmospheres absolute. Hypoxia is derived from hypo meaning low and oxia referring to oxygen.

The degree of poisoning by high pressure oxygen depends on the partial pressure of oxygen breathed and exposure time.

A high partial pressure of oxygen can be achieved in two ways: breathing air at depths greater than 20 metres and breathing pure oxygen at any depth.

PULMONARY OXYGEN POISONING (LOW PRESSURE OXYGEN POISONING)

Cause

Progressive breakdown of lung tissue occurs if oxygen is breathed at partial pressures greater than 0.6 atmospheres absolute for extended periods. Breathing pure oxygen on the surface, or air at depths greater than 20 metres, will achieve such partial pressures.

Signs and symptoms of low pressure oxygen poisoning occur after exposure for several hours to days, depending on individual susceptibility. Low pressure oxygen poisoning is generally a problem in subaquatic habitats, and in recompression chambers. Prolonged periods of breathing pure oxygen should be avoided.

The main effects are listed in order of frequency:

- ✓ irritable or sore throat (similar to a cold)
- ✓ soreness behind the breastbone
- ✓ cough (especially with deep breathing, dry and irritating)
- ✓ increasing pain with respiration
- ✓ wheezing and uncontrolled coughing
- ✓ shortness of breath
- ✓ blood stained sputum may be produced
- ✓ death is possible if exposure is prolonged



Management

No specific treatment is available for the toxic effects of oxygen on the lungs. Once signs of oxygen toxicity become apparent, the partial pressure of oxygen breathed must be reduced as much as possible.

CNS OXYGEN POISONING (ACUTE OXYGEN POISONING)

Cause

High pressure oxygen poisoning will occur in individuals when the partial pressure of oxygen breathed exceeds 1.6 atmospheres absolute (ATA). Diving, using air to depths in excess of 70 metres, or diving in excess of 6 metres using pure oxygen, will achieve an oxygen partial pressure greater than 1.6 atmospheres absolute (ATA).

Signs and Symptoms

The onset of high pressure oxygen poisoning is usually rapid. Early warning signs and symptoms such as nausea, dizziness, incoordination, muscular twitching (particularly of facial muscles), may not even be noticed. The most obvious sign is usually convulsions.



Management

A reduction of oxygen partial pressure must be achieved by ascent. To avoid pulmonary barotrauma of ascent it is important to maintain depth until the convulsion, which may cause blockage of the airway, ceases and the victim is relaxed. This usually takes up to 30 seconds.

Recovery will be rapid unless some water aspiration occurs. In all cases, medical attention is required.

Prevention

It is important to ensure that all scuba cylinders are filled only with pure breathing air, not oxygen, and excessive depths on scuba are avoided.



MANAGEMENT SUMMARY

- ✓ DRABC
- ✓ Observe and assess the signs and symptoms
- ✓ Keep patient still
 - ✎ If unconscious lay in recovery position
 - ✎ If conscious lay down flat, legs level
- ✓ Reassure
- ✓ Administer 100% oxygen
- ✓ Observe and monitor airway, breathing and circulation (pulse)
- ✓ Resuscitate as necessary
- ✓ Protect from exposure
- ✓ Implement company's emergency plan or contact DAN/DES and arrange recompression — DES (Australia) 1 800 088 200
- ✓ Record all details (dive profile, signs and symptoms, first aid given)

■ HYPOXIA

Maintenance of normal levels of oxygen is important for normal functioning of the human body. Underwater, if the level drops below 0.16 atmospheres absolute (i.e. the diver becomes hypoxic), results may be fatal.

CAUSE

The individual causes of hypoxia are many and varied. Generally, there are two major categories:

- ✓ failure of sufficient oxygen reaching the lungs, e.g. equipment failure, exhaustion of air supply, contamination and drowning
- ✓ failure of sufficient oxygen reaching the tissues, e.g. cardiac arrest, air embolism, and carbon monoxide poisoning

Individual discussion on each cause will be dealt with throughout the text.

SIGNS AND SYMPTOMS

When the partial pressure of oxygen in the body falls, an oxygen debt is incurred; the amount of oxygen required exceeds the amount available.



As this debt increases a feeling of fatigue may be replaced by confusion, incoordination and panic while, in very rare cases of hypoxia, a final stage of euphoria (a feeling of wellbeing) may be reached, followed by unconsciousness.

MANAGEMENT

It is imperative to remove the unconscious diver from the water to avoid drowning. Upon reaching the surface administer 100% oxygen if available. Should respiratory or cardiac failure occur, resuscitation must be commenced immediately and medical aid sought.



MANAGEMENT SUMMARY

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PREVENTION

Regular equipment servicing and the use of a contents gauge should eliminate the failure of air supply. Contamination will be avoided by getting scuba cylinders re-filled at a reputable dive store. Correct training in the practical aspects of diving, good dive planning and maintenance of a good physical condition will ensure that a situation involving hypoxia will not occur.

If the total supply of oxygen to the diver is removed, a condition known as anoxia will result. Anoxia is a state of no oxygen as opposed to hypoxia which is a state of low oxygen.

CARBON DIOXIDE

Carbon dioxide (CO₂) is produced by combustion and respiration, consequently the concentration of carbon dioxide in the atmosphere varies. In heavily industrialized areas the level of carbon dioxide would be high compared to the open ocean. Carbon dioxide is also produced as a waste product of respiration in animals. In mammals, carbon dioxide is exhaled via the lungs. If proper ventilation does not occur, a build-up of carbon dioxide in the respiratory system follows and will eventually cause carbon dioxide poisoning.

Physiologically, carbon dioxide is very important since it is the primary stimulant of the respiratory system. Chemoreceptors, located in the major arteries, monitor the level of carbon dioxide in the blood and send information to the respiratory centre located in the brain. If the concentration of carbon dioxide is high the respiratory centre stimulates breathing. The depth and rate of breathing will be increased in an effort to rid the system of excess carbon dioxide.



Carbon dioxide can be blamed for one of the most common complaints in diving - headache.

CAUSE

Although contamination and poor regulator function can be blamed for carbon dioxide poisoning, the main cause lies with the diver. Poor ventilation resulting from "skip breathing" or diving too deep, will cause carbon dioxide build-up in the system. Skip breathing is the act of infrequent or shallow breathing during a scuba dive.

Tight fitting wetsuits and over tightened straps will also cause poor ventilation and hence a build-up of carbon dioxide. Over exertion, particularly when diving where there are strong currents, can lead to carbon dioxide build-up.

SIGNS AND SYMPTOMS

Generally a build-up of carbon dioxide will result in a stimulus to breathe. The most noticeable symptom of carbon dioxide poisoning may be a slight increase in breathing rate during the dive. Upon reaching the surface, the diver may suffer a throbbing headache with associated nausea, vomiting and confusion.

MANAGEMENT

If loss of breathing control should occur during the dive it is important to try and restore a normal breathing pattern by ceasing all muscular activity. If normal breathing is not restored, ascend and breathe fresh air. In severe cases, administer 100% oxygen if available. If improvement is not fast seek medical advice.

PREVENTION

Skip breathing in an effort to conserve air is false economy and should be avoided. Avoid the use of poor quality or tight fitting equipment, and remember to breathe out fully, particularly when diving deep. Diving supervisors should be alert at all times for any signs of CO₂ toxicity being exhibited by the diver over the communications system.



■ HYPERVENTILATION

Hyperventilation (hyper refers to over and ventilation means breathing) is most dangerous when combined with breath-hold diving. Taking more than three or four breaths before a breath-hold dive constitutes dangerous hyperventilation (over-breathing). Traditionally, hyperventilation has been practiced by spearfishermen in an effort to prolong the time of a breath-hold dive. By hyperventilating, the partial pressure of carbon dioxide level is lowered leading to a delayed urge to breathe. Although the carbon dioxide levels are lowered, oxygen levels are not significantly increased. This is because most oxygen is carried by red blood cells which are normally 97% saturated.

It is because the oxygen levels do not increase as the carbon dioxide levels decrease that makes the practice of hyperventilation dangerous.



HYPERVENTILATION ANOXIA - (LATENT ANOXIA, SHALLOW WATER BLACKOUT)

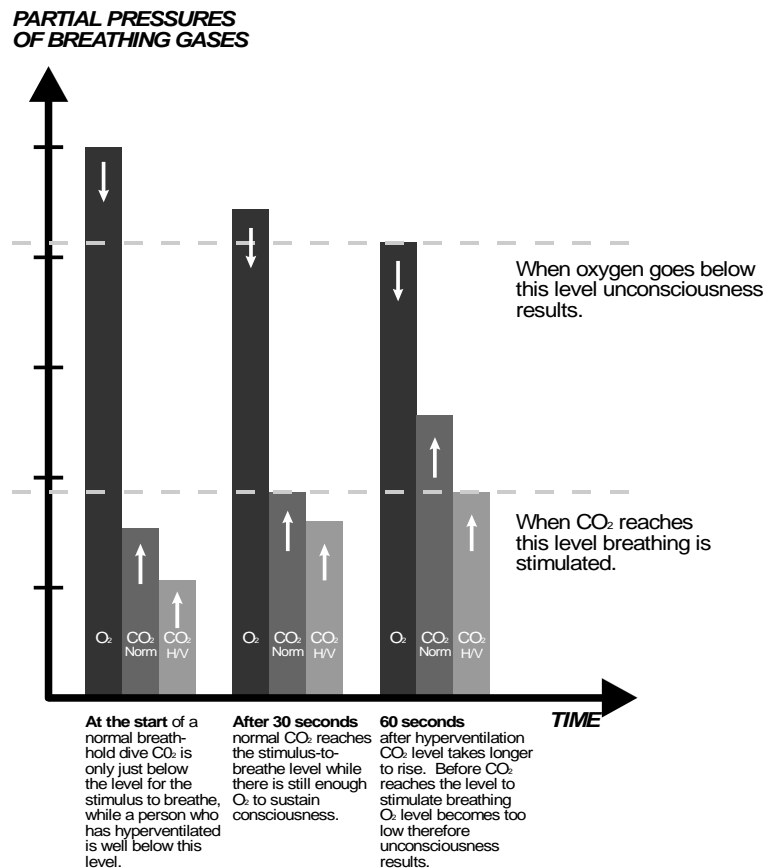


Figure 22. Gas levels during a breath hold dive

Hyperventilation is not restricted to spearfishermen. Many underwater swimmers have fallen victim to it.

Cause

Dangerous hyperventilation is the cause. During a breath-hold dive without hyperventilation, the urge to breathe occurs normally, allowing enough time to reach the surface safely. If the breath-hold diver hyperventilates, although the urge to breathe is delayed, oxygen consumption remains the same. Upon ascent, as total pressure lowers, the oxygen partial pressure drops to below the level required to sustain consciousness.

Signs and Symptoms

Unconsciousness usually occurs without any warning while diver is ascending.

Management

Drowning is obviously inevitable unless the victim is quickly recovered. Expired air resuscitation (EAR) should be commenced immediately to aid re-establishment of normal carbon dioxide levels. When breathing has stabilized, administer 100% oxygen for treatment of possible salt water aspiration. It is essential to continue observation of the patient as respiratory failure may recur due to the low level of carbon dioxide in the system. Medical advice and observation for at least 24 hours are required.



**MANAGEMENT SUMMARY**

- ✓ DRABC
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 - ☞ If conscious lay down flat, legs level
- ✓ Reassure
- ✓ Administer 100% oxygen
- ✓ Observe and monitor airway, breathing and circulation (pulse)
- ✓ Resuscitate as necessary
- ✓ Protect from exposure
- ✓ Implement company's emergency plan or contact DAN/DES and arrange recompression — DES (Australia) 1 800 088 200
- ✓ Record all details (dive profile, signs and symptoms, first aid given)

**Prevention**

Hyperventilation, whether intentional or not, is a very dangerous practice and should always be avoided. The need for a good buddy system is once again emphasised.

■ NITROGEN NARCOSIS - (NARCS: RAPTURE OF THE DEEP; INERT GAS NARCOSIS)

Nitrogen, approximately 80% of the atmosphere, is not used by the body. However, when breathed at high partial pressure, it has a narcotic effect on the diver known as nitrogen narcosis.

Nitrogen narcosis, although in itself not physiologically damaging, has been the indirect cause of many diving fatalities.

CAUSE

The reasons why breathing high partial pressures of nitrogen causes nitrogen narcosis are still not completely understood, however, it can be clinically detected in all divers at 30 metres and some are affected at shallower depths. Beyond this depth the influence of nitrogen will worsen, depending on individual susceptibility. It becomes hazardous at depths beyond 40 metres.

SIGNS AND SYMPTOMS

The signs and symptoms of nitrogen narcosis will occur within minutes of reaching a particular depth and will worsen as depth is increased. Nitrogen narcosis can affect divers in many different ways and the signs and symptoms can be quite obvious or very subtle. When diving greater than 20 metres divers must be on the alert for signs and symptoms not only in themselves but also in their buddy.

Subtle signs can include a delayed response in answering hand signals or slowness in interpreting gauges.

Some divers may develop anxiety at depth while others may become over confident. Either is a warning that nitrogen narcosis may get worse and indeed dangerous.



More serious signs and symptoms include light-headedness, idea fixation, lack of reasoning, poor judgement i.e. changing the dive plan while at depth or calculation errors e.g. trying to work out decompression tables at depth. Amnesia or memory loss after the dive is a common symptom of nitrogen narcosis. Lack of co-ordination and concentration are obvious.

However, a diver may not be aware of narcosis until serious signs and symptoms occur. Seemingly simple tasks such as mask clearing, turning on a torch or focusing a camera become difficult.

Should descent be continued, mental confusion, hallucinations and unconsciousness may develop.



MANAGEMENT

If nitrogen narcosis is evident, immediate ascent to shallower depths will eliminate its effects. It should be noted that both divers in a buddy system may be affected. Therefore, the symptoms may pass unnoticed and no attempt may be made to remedy the situation.



PREVENTION

By restricting sport divers to above 30 metres, nitrogen narcosis should not be a problem. Constant assessment during any dive below 30 metres is essential. Diving below 30 metres should only be attempted with adequate training and with suitably experienced and qualified divers.

PREDISPOSING FACTORS



Any factors that will increase the likelihood of nitrogen narcosis are termed predisposing factors. Increased carbon dioxide retention resulting from rapid descent or hard work at depth will increase the effects of narcosis until proper ventilation is re-established. Poor visibility usually predisposes to narcosis or apprehension. Inexperience may develop anxiety which, combined with dives greater than 30 metres, will result in greater susceptibility to nitrogen narcosis. The chances of a diver experiencing narcosis are markedly increased by cold water, hangover, alcohol and other drugs. The onset of nitrogen narcosis varies within individuals as well as from dive to dive and day to day. Strong motivation and willpower are marginally beneficial in overcoming narcosis.

CARBON MONOXIDE POISONING

CAUSE

Carbon monoxide is produced in the combustion process when the supply of oxygen is limited (such as in an automobile engine). Carbon monoxide is a colourless, odourless gas which has the ability to combine with red blood cells. The red blood cell/carbon monoxide combination serves no useful purpose. Not only does carbon monoxide reduce the blood's oxygen carrying capacity but seems to also have a direct poisoning effect on the cell itself. This reduced red blood cell function leads to hypoxia.

Even if an intake hose is correctly positioned, carbon monoxide contamination can still occur if the compressor is poorly maintained. In a well maintained compressor the cylinder rings will prevent oil from entering the head area. If the rings become worn, oil will be left on the cylinder wall during the piston's downstroke. When the piston comes up the oil will be in a high pressure area and can spontaneously ignite ("flash") producing carbon monoxide.

Most compressor filtration systems do not filter out carbon monoxide; therefore contamination of the scuba air supply will occur even though pure air is entering the compressor.



If the oil in a compressor is not changed regularly or if the wrong type of oil (e.g. vegetable oil) is used, then this thinner, less viscous oil can get into the cylinder head. If this oil “flashes” carbon monoxide will be produced.

SIGNS AND SYMPTOMS

Carbon monoxide poisoning results in headaches, confusion, dizziness if severe, shortness of breath, nausea, vomiting, weak pulse, and unconsciousness. The most distinguishing feature of carbon monoxide poisoning is the cherry red appearance of lips and fingernail beds. This arises because the red blood cell/carbon monoxide combination results in a bright red colour. Therefore, areas close to the surface of the body appear flushed. Unfortunately, this symptom usually manifests itself after death because a high level of contamination is needed.

It is worth noting that the effects of carbon monoxide poisoning will decrease with increasing depth. More oxygen will dissolve in the blood plasma and transport of oxygen to tissues can be maintained.

MANAGEMENT

Remove the victim from the source of contamination. Fresh air, if necessary 100% oxygen may relieve the signs and symptoms but because of a possible relapse it is vitally important that the Diver Emergency Service be contacted.

Recompression therapy using oxygen under medical supervision may be necessary for complete recovery. Monitor vital signs and resuscitate as necessary.



MANAGEMENT SUMMARY

- ✓ DRABC
- ✓ Observe and assess the signs and symptoms
- ✓ Keep patient still
 - ☞ If unconscious lay in recovery position
 - ☞ If conscious lay down flat, legs level
- ✓ Reassure
- ✓ Administer 100% oxygen
- ✓ Observe and monitor airway, breathing and circulation (pulse)
- ✓ Resuscitate as necessary
- ✓ Protect from exposure
- ✓ Implement company's emergency plan or contact DAN/DES and arrange recompression — DES (Australia) 1 800 088 200
- ✓ Record all details (dive profile, signs and symptoms, first aid given)



TREATMENT

Minor cases of carbon monoxide often need only bed rest and treatment for the general nausea and headache associated with the problem. For severe cases, hyperbaric (high pressure) oxygen in a recompression chamber may be needed.

PREVENTION

It is important to ensure a clear air way supply by taking care when positioning the air intake hose of a compressor and to keep the compressor well maintained.



Air that has an oily odour or taste may be contaminated with carbon monoxide and should not be used.

DECOMPRESSION ILLNESS



NTKI

Decompression is a major limiting factor to all diving operations and DCI is a significant hazard.

A basic knowledge of the cause of DCI, how to recognise it, how to treat it and, more importantly, how to prevent it is essential.

INTRODUCTION

The clinical consequences of Henry's Law, in conjunction with Dalton's Law of partial pressures and the principle of gas diffusion, can be seen in the diving ailment known as decompression illness.

Every time a diver descends underwater the increase in ambient pressure will cause more air to dissolve in the blood stream (Henry's Law). The increased levels of oxygen and carbon dioxide in the blood will pose very little problem because they are consumed via the body's normal physiological processes. Nitrogen is not used by the body and increased levels will be distributed by the blood throughout the body. Different tissues will absorb nitrogen at different rates. For instance, bone tissue will absorb nitrogen slowly due to its poor blood supply whereas brain tissue will absorb nitrogen more readily due to its prolific blood supply. As dive time progresses, more and more nitrogen will diffuse from the lungs into the blood stream and hence into various tissues. Saturation of all tissues can take more than 12 hours.

CAUSE

Depth and time will determine how much nitrogen actually dissolves in the tissues. When ascending from depth, time must be allowed so that excess nitrogen can be eliminated via the lungs. If ascent is too rapid, and the pressure drop is too great, nitrogen molecules will form bubbles in the tissues.

BUBBLE FORMATION

It has been known for a long time that decompression illness is an illness related to bubbles in the blood and tissues, but it still remains uncertain exactly when and where these bubbles begin to form.

Most researchers believe that the partial pressure of inert gas dissolved in a tissue has to exceed the ambient pressure by some critical amount before gas bubbles form.

THEORIES OF BUBBLE FORMATION

There are a number of theories, which attempt to describe bubble formation. To date, however, no theory has been indisputably proven. The truth may lie in a combination of various aspects of these theories.

One popular theory supposes that tiny bubble "nuclei" (microscopic pockets of gas) are present within our bodies, and that excess gas diffuses into these nuclei, expanding them and creating bubbles.

If you look closely into a glass of coca-cola or some other carbonated drink, you might notice that the bubbles only originate from the walls or bottom of the glass. This is true as long as the drink is held still, but if the drink is shaken, new bubbles form from within the liquid itself, rather than only at points of contact with the glass.

In the still drink, bubbles only form at tiny cracks or imperfections in the glass. It is believed that a small "nucleus" of gas is trapped in each of these cracks, and this nucleus resists being crushed.



When the lid was removed from the bottle (i.e. the ambient pressure was reduced) the excess gas that was dissolved in the solution comes out and some of the gas diffuses into these nuclei. Each nucleus grows and eventually a bubble forms and breaks away, leaving the nucleus free to form other bubbles.

Certain theories propose that vessel walls in the human body retain small pockets of gas which could act as nuclei into which excess gas could diffuse when we ascend from a dive. Other researchers propose that bubble nuclei are circulating continuously, stabilized by elastic skins through which gas diffuses at normal diving pressures. However, these explanations alone do not fully explain why bubbles form in the human body. Some research indicates that the non-wettable nature of the surface of the blood vessels plays an important role in allowing bubbles to form.

BUBBLE GROWTH

For a bubble to grow from “nothing” to a finite size it must pass through two distinct phases:

- ✓ formation of the initial small bubble
- ✓ growth of the small bubble into much larger bubble

For a bubble to grow the total pressure within the bubble must be greater than the restricting/crushing pressures. The constricting pressures are made up of three main components.

- ✓ **Ambient pressure** – pressure at the given depth
- ✓ **Tissue pressure** – the nature of body tissue is such that it resists being deformed by bubbles
- ✓ **Surface tension** – inward attraction due to intermolecular forces

Thus, the necessary condition for bubble growth is:

$$P_{\text{Bubble}} > P_{\text{Ambient}} + P_{\text{Tissue}} + P_{\text{Surface Tension}}$$

This condition is also known as supersaturation. The main question is the extent of supersaturation required before bubbles begin to have prolonged existence.

Site of bubble formation

Bubbles form at places where conditions favour their development for example regions where:

- ✓ lower pressure conditions exist
- ✓ a suitable surface exists



Regions of lower pressure – these promote the level of supersaturation and thus enhance bubble formation. These occur where

- ✓ turbulence exists in blood vessels
- ✓ shearing forces, e.g. within muscle tissue

Surface influence nucleation – It appears that bubble formation is significantly affected by the presence of pre-existing gas nuclei. (i.e. A microscopic pocket of gas).

- ✓ The walls of a crevice are hydrophobic, therefore surface tension has little effect in collapsing the micro bubble
- ✓ Super-saturation occurs, bubble grows
- ✓ Crevice reduces the surface tension effect, i.e. it is protected



- ✓ Bubble breaks away and a new nucleating effect commences

NOTE: Silent bubbles may form – these are asymptomatic.

THE EFFECTS OF BUBBLE FORMATION

Once bubbles form they often join together to form larger pockets of gas which may become trapped in the tissue or vessels. Bubbles can also damage the linings of blood vessels. If bubbles do lodge in tissue or in blood vessels, they can put pressure on nerves, damage delicate tissue and block the supply of blood to vital organs.

The presence of bubbles in the blood and tissues causes complex biochemical reactions within the body. The clotting mechanisms are activated and blood platelets become sticky and begin to stick to the bubbles and to each other. Recent experiments have indicated that the presence of gas bubbles in the blood activates certain complement proteins which appear to play an important role in the development of decompression illness.

As previously mentioned, the presence of bubbles slows down nitrogen elimination. This may be due to both the reduction in circulation through the lungs due to the bubbles blocking lung capillaries, and the changes in the blood, precipitated by the bubbles.

DETECTION OF BUBBLES

Ultrasonic Doppler flowmeters are often used to detect bubbles. These devices emit ultrasonic waves which are reflected off moving bubbles. The flowmeters are usually aimed at the heart to detect bubbles in the venous blood entering that organ. However, they can only detect bubbles that are larger than a certain size, (40 microns) and which are circulating. These devices do not detect bubbles trapped in the tissues, which are the ones believed to cause many of the symptoms of DCI.



It is important to realise that the fact that no bubbles can be detected by a Doppler flowmeter does not necessarily mean that no bubbles are present. This is relevant when testing dive tables and computers.

THE ILLNESS

In the seventeenth century Robert Boyle used a pump to decrease the pressure within a vessel which contained a viper. When he released the pressure he noticed that a bubble had formed in the snake's eye and it writhed in pain. He believed the snake to have decompression sickness (DCS).

In the nineteenth century, caisson workers, who worked in tunnels digging through wet soil, often complained of pains and other disorders after coming out from the tunnels. The pressure of the air in the tunnels had been raised in order to prevent the water entering into and filling the tunnels and these workers would spend many hours breathing the compressed air.

Later that century, divers began to dive on compressed air and they would often complain of similar symptoms. Early this century, aviators who had flown in unpressurised aircraft would at times get similar problems.

What these sufferers all had in common was that they had developed symptoms after their environmental pressure had been reduced, that is, after decompression. This ailment was initially called the "bends", since sufferers would often adopt a bent gait because of pain in the hips and knees.





NOTE: Decompression sickness has been classified in various ways over the years. However, relatively recently, a new and more general classification has been introduced and is now commonly used. Decompression illness (DCI) is a term used to describe decompression sickness and cerebral arterial gas embolism (CAGE). Because the manifestations of DCI and CAGE are often identical it is usually impossible to differentiate between them. In addition, the recommended first aid management and treatment for the two disorders is usually the same.

Decompression illness results from the development of gas bubbles within the body. Excess gas is dissolved in our blood and body tissues when we dive due to the higher partial pressure of the gases in our lungs. When we dive breathing air, the extra gases are oxygen and nitrogen. The oxygen is easily used up by the body functions and generally does not create bubble problems, but the extra nitrogen is not used and much of it is stored in the tissues. When we ascend, the excess gas is released from our tissues and, if the ascent is slow enough, this excess gas will stay dissolved and will leave our body in a safe and orderly way. If, however, we ascend too quickly, bubbles of excess gas will form in some of our tissues and at times in our blood during or following the ascent. These bubbles can distort and disrupt tissues and can block and damage blood vessels. When the bubbles cause symptoms it is called decompression illness.

Bubbles can occur without creating symptoms or causing any damage to our body. Bubbles, which do not cause symptoms, are called “asymptomatic” or “silent” bubbles. Some experts fear that at times some silent bubbles can still cause damage.

■ SIGNS AND SYMPTOMS

A common symptom of DCI is a feeling of extreme fatigue or malaise after the dive. We are often tired after the dive due to the effort of lugging equipment around, or due to a hard swim. The fatigue of DCI is fatigue beyond what you would expect from the amount of exertion done. The cause of these symptoms is unconfirmed but it has been suggested that they may result from bubbles in the central nervous system and/or a reaction precipitated by the presence of bubbles. Although these symptoms often disappear, some doctors fear that at times they may have serious implications, especially if untreated.

The most commonly noticed manifestation of DCI is pain, somewhere in the body.

The dive profile influences, to some extent, where bubbles form and, therefore, the type of symptoms which might occur. Theoretically, a short, deep dive produces a high nitrogen load within the “fast” tissues, but a smaller load within the “slow” tissues, which need more time to saturate. If bubbles form during or after the ascent they may be more likely to form in the “fast” tissues such as blood and the central nervous system (brain and spinal cord), causing symptoms such as paralysis, numbness, loss of bladder function, mental changes, or difficulty in breathing (a symptom of a bubble overload in the lungs). Rapid ascent makes bubble formation in the blood more likely, since there is even less time for the lungs to rid the blood of its nitrogen load.

A long dive to a shallower depth will put more nitrogen in the “slow” tissues so this type of dive profile tends to produce pain at or near joints. However, close examination often shows neurological signs/symptoms as well.

Most divers suffering from DCI will feel symptoms within six hours of having dived, the majority showing some signs or feeling symptoms within the first hour. However, some divers have become symptomatic as long as one to three days or even longer after the dive. As a general rule, the earlier the onset of symptoms of decompression illness, the more serious the illness is likely to be.

The location and volume of the bubbles determines the type of symptoms and the severity of DCI. Bubbles blocking vessels within various large organs cause no pain and often cause no



damage if there are other blood vessels supplying the organ which can provide enough oxygenated blood to replace the supply from the blocked vessels. If blood does not get through, part of the organ may be damaged.

Initial determination of decompression illness revolves around accurate determination of the dive profile. Factors to be considered include depth, time, ascent rate, activity/cold, and previous activities. If these factors are not within safe limits, then decompression illness should be considered. Signs and symptoms of decompression illness can be related to each major system of the body.

System of the Body	Signs and Symptoms	Common Name
Skin	skin rash – shoulders, back etc, marbling effect, red weals and itchiness	itches
Musculoskeletal	joint pains, dull ache, numbness	niggles, bends
Neurological	loss of co-ordination, partial paralysis, confusion, fits, blurred vision	staggers
Vestibular	vertigo, nausea, vomiting	
Gastrointestinal	nausea, vomiting, abdominal cramping, diarrhoea	
Cardio-respiratory	irregularities of heartbeat, shortness of breath, chest pain, coughing	chokes

■ FIRST AID FOR DECOMPRESSION ILLNESS



The longer the interval between the onset of symptoms of decompression illness and the commencement of hyperbaric oxygen treatment, the more likely is the possibility of permanent damage. Therefore, it is essential to get a diver to a recompression chamber as soon as possible.

The first aid procedure is designed to minimise the damage to the diver during this interval. The first aid procedure and its rationale is as follows:

- ✓ **Monitor the consciousness, breathing, pulse, and resuscitate if necessary.** Supporting life is the most urgent requirement and if resuscitation is indicated, it takes precedence over other first aid measures.
- ✓ **Lay the casualty down.** The diver should normally be positioned horizontally without the head or legs elevated. However, if the diver feels faint, has a thready pulse or low blood pressure, elevation of the legs may raise the blood pressure. However, if the diver's condition appears to deteriorate as a result, the legs should be lowered.
- ✓ **Unconscious or nauseated divers** should be placed on their side to prevent aspiration of vomitus and to facilitate an open airway.
- ✓ **Administer oxygen.** Breathing oxygen will increase the oxygenation of any hypoxic body tissues and also help to flush out any dissolved nitrogen and nitrogen present in bubbles. The concentration of inhaled oxygen should be as near to 100% as can be reached in order to achieve the maximum benefit.

Oxygen administration should begin as soon as the injury is recognised and should generally continue uninterrupted until the supply is exhausted or until the diver reaches the recompression chamber.



Previously, it was recommended that oxygen administration be interrupted periodically to provide "air breaks". However, air breaks are now not normally recommended prior to recompression since the injured diver usually doesn't breathe 100% oxygen for prolonged periods. At present, the only time an air break may be recommended is if the injured diver was to breathe 100% for longer than about 5 hours prior to recompression.

The most efficient and reliable way to deliver 100% oxygen is by demand valve and mask. However, this is only possible with a casualty who is breathing effectively enough to trigger the demand valve. A breathing diver who is having difficulty with a demand valve can use a constant flow mask with reservoir bag, with a flow rate of at least 15 litres per minute.

The breathing of oxygen is a first aid measure and must never be substituted for the recompression required for decompression illness, regardless of the response.

- ✓ **Treat for shock.** The first aid for shock includes laying the patient down and administering oxygen and hopefully these will have been initiated already. The diver should be kept thermally comfortable but not rewarmed rapidly, since rapid rewarming may encourage more bubbling. He should be reassured and kept still and quiet. This will maximise the blood supply to vital organs. Remaining still will discourage further bubble formation due to movement.
- ✓ **Implement company emergency procedures** or contact the Divers Emergency Service (DES) on 1 800 088 200. DES will give advice about any further first aid treatment that might be necessary.



THE DIVING EMERGENCY SERVICE (DES)

The Diving Emergency Service (DES) is a toll free 24-hour Australia-wide telephone number that provides emergency advice on diving and pressure-related accidents to callers in Australia. Calls to this number will initiate all contacts necessary to enable hand-over management by the geographically nearest hyperbaric facility. The DES is based at the Hyperbaric Medicine Unit of the Royal Adelaide Hospital in Adelaide, South Australia. It works in close liaison with other Australian hyperbaric units. The DES calls are promptly directed to the on-duty DES consultant. Advice will be given on the management of the diving casualty, and will include the likely diagnosis and necessary first aid measures.

- ✓ Callers within Australia: 1 800 088 200
 - ✓ Callers from outside Australia: 61 8 223 2855
 - ✓ Non-emergency enquires: 08 8224 5116
- ✓ **Seek medical aid.** The casualty should be taken to the nearest medical facility as soon as possible so that proper medical treatment can be initiated. Arrange for the attending medical person to contact DES, if this has not yet been done.
 - ✓ **Provide fluids to a diver who is not acutely ill.** Fluids are given to rehydrate the diver. They increase the blood volume, thin the blood and reduce shock. Thinner blood is less likely to clot or sludge thereby obstructing circulation. Intravenous fluids such as normal saline provide the greatest benefit.

Where IV fluids are not available, it is generally recommended to give oral fluids such as isotonic fluids or water to a diver suffering from DCI, provided that the diver is fully conscious, alert, has control of his airway, is cooperative, his condition is stable and he is not suffering from nausea, vomiting, severe pain, urinary retention or paralysis (unless a urinary catheter is used).



IV fluids are preferred because:

- ☞ there is less likelihood of vomiting or regurgitation
- ☞ a diver in shock may not absorb oral fluids effectively
- ☞ it is difficult for the diver to drink without lifting his head
- ☞ oxygen administration need not be interrupted for the diver to remove the oxygen mask in order to drink
- ☞ divers occasionally begin to fit due to the elevated oxygen partial pressures in a recompression chamber, it is possible that a diver with a stomach full of fluids could aspirate his stomach contents in the event of a fit



- ✓ **Record details of the dive profile, first aid given and casualty's response to first aid.** This information will be useful in confirming the nature of the injury and in its subsequent treatment. It will also be important during any subsequent investigation by Worksafe.



MANAGEMENT SUMMARY

- ✓ DRABC
- ✓ Observe and assess the signs and symptoms
- ✓ Keep patient still
 - ☞ If unconscious lay in recovery position
 - ☞ If conscious lay down flat, legs level
- ✓ Reassure
- ✓ Administer 100% oxygen
- ✓ Observe and monitor airway, breathing and circulation (pulse)
- ✓ Resuscitate as necessary
- ✓ Protect from exposure
- ✓ Implement company's emergency plan or contact DAN/DES and arrange recompression – DES (Australia) 1 800 088 200
- ✓ Record all details (dive profile, signs and symptoms, first aid given)



■ TREATMENT FOR DECOMPRESSION ILLNESS

The definitive treatment for decompression illness is recompression in a chamber breathing as high a concentration of oxygen as possible.

Recompression reduces the size of the bubbles and so may restore the circulation to the affected area. The higher oxygen concentration serves two purposes. First, it provides greater oxygenation to any hypoxic tissue. Second, it accelerates nitrogen elimination by increasing the pressure difference (pressure gradient) between the nitrogen in the bubble and in the surrounding tissues. This encourages nitrogen to diffuse out of the bubble, into the tissues and eventually to the lungs.

Intravenous fluids are given to rehydrate the diver and to restore electrolyte levels in the blood. Various drugs are sometimes given to reduce any swelling in the brain or spinal cord or to combat the chemical changes in the blood.

A diver who is suspected of suffering from DCI is often recompressed to 18msw (metres sea water) breathing 100% oxygen (i.e. O₂ at 2.8 ATA). If the symptoms are reduced, decompression illness is assumed and the full treatment is carried out. If no relief of symptoms



occurs after 20 minutes at this pressure, the diagnosis of DCI may be questioned, but the treatment is often continued in the event that bubbles are present.

Experience has shown that repeated treatments with hyperbaric oxygen (HBO) often cause improvements in patients who are left with neurological problems after the initial treatment. The repeated treatments are usually given 12-24 hours apart.

However, despite multiple recompression treatments, many divers are left with residual impairment for an extended period of time after the treatment has finished. Some are left with permanent injury.

■ PREDISPOSING FACTORS



Some factors increase the likelihood of a diver getting DCI. Factors that may influence the occurrence of DCI are listed below.

- ✓ **Time Underwater:** The shorter the time the lower the risk. Obviously, ignoring depth considerations, shorter dives allow less time for nitrogen uptake.
- ✓ **Repetitive diving:** Repetitive dives are associated with more DCI than single dives.

DAN USA data indicate that in 1987, 64% of all the divers treated for decompression illness in the USA had done repetitive dives, whether using computers or tables. This increased to 78% by 1991. The overall increase in DCI after repetitive diving may indicate that more divers are doing repetitive dives. However, it may partly be a consequence of divers attempting to maximise dive time on multi-level and repetitive dives by using decompression systems such as dive computers and certain tables which are less conservative than traditional tables for repetitive diving.

The high incidence of DCI after repetitive dives is not surprising since the mathematics of gas elimination in all tables and computers are only vague approximations to what happens in the body. So a diver starts a repetitive dive with an unknown load of extra nitrogen in his body. Gas elimination during the surface interval will depend largely on the amount of bubbling resulting from the first dive, the greater the bubbling, the slower the off-gassing. Therefore, when a diver starts a repetitive dive, the actual amount of extra nitrogen in his body is, in reality, unknown. Redescending will add gas to any existing bubbles, may increase the rate of nitrogen uptake and may also allow some bubbles, trapped in the lung capillaries, to pass into the arterial circulation.

In addition, the same overpressure ratios used to avoid symptoms on the first dive do not apply to subsequent dives. Bubbling appears to occur far sooner on succeeding dives. Most tables and dive computers fail to accommodate this.

- ✓ **Deeper diving:** A growing bank of data from throughout the world indicates that a greatly increased risk of DCI is associated with diving deeper than 24-30m. High nitrogen loads can quickly build up during relatively short periods at depth, and it is easy to approach or exceed No-Stop Limits. In addition, the greater ambient pressure drop during ascent from a deeper dive can further increase the likelihood of DCI.
- ✓ **Dehydration:** During a dive we may dehydrate considerably because of the dry air we breathe and the effects of immersion. If we are dehydrated before a dive our blood volume can be reduced substantially. The reduction in blood volume reduces the rate at which nitrogen can be removed from the tissues and puts the diver at a much higher risk of DCI. In addition, as the blood thickens it becomes stickier and more likely to clot around any bubbles that form, impeding their movement to the lungs.

Always ensure that you are well-hydrated before and after diving. Because of the severe dehydrating effect of alcohol, it should not be consumed the night before diving. Drink fluids (other than diuretics such as alcohol, coffee and other caffeine sources) before and after diving.



- ✓ **Rate and frequency of ascent:** In general, the slower the ascent rate the lower the risk. Multiple ascents may increase bubble formation and thus the risk of DCI.
- ✓ **Flying after diving:** Flying or otherwise ascending to a higher altitude after having dived can predispose a diver to decompression illness. The lower atmospheric pressure at altitude will allow any existing bubbles to expand and can also cause new bubbles to form.
- ✓ **Exercise:** Exercise during the dive increases the demands for oxygen. As the breathing and circulation rates increase, more nitrogen is delivered to the tissues. Exercise also raises carbon dioxide levels in the blood. Exercise after the dive increases turbulence and agitation of blood and tissues and encourages bubbling. Exercise immediately before the dive may influence nitrogen uptake at the start of the dive if the increased circulation has not yet returned to normal and if excess carbon dioxide has been retained. Physical activity may also increase the number of bubble nuclei. During decompression, exertion may increase or decrease the likelihood of DCI depending on the degree of physical activity. In general, divers are discouraged from doing any exercise during safety or decompression stops.
- ✓ **Presence of a patent foramen ovale:** A patent foramen ovale is a defect between the atria of the heart which allows some blood to flow directly from the right atrium to the left atrium. Divers with this defect appear to have a higher risk of suffering severe DCI.
- ✓ **Excess carbon dioxide:** High carbon dioxide levels dilate blood vessels thereby increasing nitrogen delivery. Dissolved carbon dioxide may diffuse into and expand existing nitrogen bubbles.
- ✓ **Cold:** Low environmental temperature has been shown to predispose to DCI. The distribution of nitrogen throughout the body is altered as various blood vessels constrict from the cold. In addition, since nitrogen is more soluble at lower temperatures more may dissolve in the colder tissues.
- ✓ **Diving at altitude:** Most decompression tables apply to a diver who surfaces to the normal atmospheric pressure of 1 ATA. When we ascend from a dive at altitude, we ascend to an atmospheric pressure which is less than 1 ATA. If appropriate adjustments have not been made to the sea level schedule, the chance of DCI occurring will be greatly increased. Some dive tables are designed for diving at altitude; others must be converted to do so. Certain dive computers automatically compensate for altitude, while others don't.
- ✓ **Lack of fitness:** Inadequate circulation may not remove nitrogen from the tissues to the lungs fast enough during the ascent. An unfit diver may breathe more rapidly therefore absorbing more nitrogen than a fit diver.
- ✓ **Age:** Predisposition to DCI increases as we age. The risk has been reported to increase sharply at middle age and beyond, possibly because of reduced circulation efficiency and increased body fat.
- ✓ **Fatigue:** Fatigue is associated with reduced cardiac performance, which gives the same results as the lack of physical fitness.
- ✓ **Illness:** Chronic illnesses often reduce one's fitness and so may be a contraindication to diving. Acute illnesses all decrease a person's fitness.

The early stages of many illnesses are associated with circulatory changes. These usually include an increased heart rate which will result in a faster circulation. This leads to greater nitrogen uptake early in the dive. Later in the dive, the effects of cold reduce the blood flow through the arms and legs, thereby inhibiting the release of extra nitrogen. This increases the risk of DCI.

- ✓ **Physical injury or previous decompression illness:** Damage to the circulation around the site of the injury may delay the release of nitrogen and may predispose to DCI.



- ✓ **Anxiety:** Anxiety increases the breathing rate, speeds up the heart, and increases the circulation. This increases the uptake of nitrogen and production of carbon dioxide during the dive and may lead to an increased likelihood of DCI.
- ✓ **Alcohol:** Alcohol depresses the brain and dehydrates the body. Together the two effects lead to changes in the circulation and in the diver's behaviour and may lead to a higher risk of DCI.
- ✓ **Medications and other drugs:** There is a lack of substantial data on how various drugs may affect the risk of DCI. However, any drugs for the heart or for hypertension (high blood pressure) are likely to affect the heart's output and hence the circulation, producing the same changes as lack of fitness. A further complication is that pressure alters the effects of some drugs in an unpredictable manner.
- ✓ **Obesity:** Obesity has been suggested as a predisposing factor for DCI but supporting evidence is inconsistent. Fat has a high affinity for nitrogen and has poor circulation. After a long dive fat will hold a lot of nitrogen and needs quite some time to unload it. If given insufficient time to unload, fat may bubble. If bubbles form in the fat of the bone marrow or spinal cord the results may be serious.
- ✓ **Nitrogen narcosis:** Nitrogen narcosis, by distorting a diver's thinking, creates anxiety and causes deviations from the dive plan, which may indirectly increase the diver's chances of developing DCI.

ADAS COMPETENCY

Maintain Health and Safety of Individual and Others.

Explain, in general terms, the use and effect of Nitrox in surface orientated diving operations using SCUBA.

THE USE AND EFFECT OF NITROX

Divers have used air as a breathing gas since the beginning of diving. Its principal advantage is that it is readily available and inexpensive to compress into cylinders or use directly from compressor as with surface-supplied diving equipment. Air is not the "ideal" breathing mixture for diving because of the effects of nitrogen narcosis at deeper depths and the decompression liability it imposes. Since decompression obligation is dependent on exposure to inspired nitrogen partial pressure, this obligation can be reduced by replacing a portion of the nitrogen content of divers' breathing gas with oxygen, which is metabolised by the body. This is the fundamental benefit of nitrogen-oxygen diving.

"Nitrox" is a generic term that can be used for any gaseous mixture of nitrogen and oxygen. Using nitrox can significantly increase the amount of time a diver can spend at depth without incurring additional decompression.

ADVANTAGES

Nitrox breathing mixtures are beneficial in helping to prevent decompression sickness. Using a gas mixture with less nitrogen in it effectively lowers this risk. This does not mean that a diver will never get decompression sickness; it means with proper management, the already small risk of DCS can be even smaller when diving with oxygen-rich mixtures.

Nitrox mixtures are of special value in the multi-day, repetitive-dive series. No-stop dive incident statistics show that almost 80 percent of recreational cases of decompression sickness are a result of repetitive air dives (Divers Alert Network 2000). If a diver were to dive the same time and depth profiles with nitrox instead of air, not only would the decompression be less, but there would also be an overall reduction of nitrogen in the body, thereby lowering the risk of decompression sickness.

DECOMPRESSION ILLNESS – UNIT SUMMARY

- ✓ A diver is said to be "saturated" when the partial pressure of nitrogen in all of his body tissues is the same as the partial pressure of nitrogen in his lungs



- ✓ Some body tissues absorb (and release) nitrogen far more rapidly than others and thus become saturated sooner. The differing rate of nitrogen absorption depends on the fat content of the tissue and the amount of blood flow (perfusion) to the tissue.
- ✓ The opening and closing of capillaries affects the rate of nitrogen uptake and release, as do temperature, exercise and various other factors
- ✓ A scuba diver is essentially saturated after 24 hours
- ✓ When the partial pressure of nitrogen in a tissue is greater than that in the lungs, the tissue is said to be “supersaturated”
- ✓ If the blood or tissues become supersaturated, bubbles may begin to form
- ✓ Most of the bubbles become trapped in the lungs, where they eventually resolve
- ✓ Nitrogen elimination is slowed down when bubbles are present
- ✓ Bubbles can damage blood vessels and other body tissues
- ✓ Bubbles will form when the partial pressure of inert gas in a tissue exceeds the ambient pressure by some amount
- ✓ There are a number of theories describing bubble formation in the body. However no theory has as yet been indisputably proven
- ✓ Bubbles can block and/or damage blood vessels, put pressure on nerves and damage tissue
- ✓ Bubbles activate various biochemical reactions in the body, which have been shown to play an important role in the development of DCI
- ✓ The presence of bubbles slows down nitrogen elimination
- ✓ Doppler ultrasonic bubble detectors can only detect circulating bubbles and not those trapped in the tissues
- ✓ Decompression illness (DCI) results from the development of bubbles within the body
- ✓ These bubbles can distort and disrupt tissue, block and damage blood vessels and precipitate biochemical reactions in the blood which can cause damage
- ✓ Bubbles can occur without creating symptoms - “silent bubbles”
- ✓ The lungs act as filters and trap most of the bubbles in the bloodstream
- ✓ The dive profile may influence where bubbles form and the type of symptoms which might occur
- ✓ Rapid ascent makes bubbles more likely to form in the blood and the other “faster” tissues (e.g. brain)
- ✓ Symptoms usually occur within six hours of diving but can take as long as one to three days or more to show
- ✓ The location and volume of the bubbles determines the type of symptoms and the severity
- ✓ Common signs and symptoms of DCI include pain, extreme fatigue, numbness and tingling sensations
- ✓ First aid for DCI includes life support, laying the casualty down horizontally, administration of 100% oxygen, contacting the DES, arranging medical aid and possibly providing fluids



- ✓ Treatment for DCI includes recompression in a recompression chamber, hyperbaric oxygen and intravenous fluids. The recompression reduces the size of the bubbles and restores circulation. The hyperbaric oxygen provides greater oxygenation to hypoxic tissue and encourages nitrogen elimination. The intravenous fluids rehydrate the diver and restore electrolyte levels in the blood.
- ✓ Despite breathing oxygen and rapid recompression, some divers are left with serious neurological problems
- ✓ It is feared that even though a treatment may appear to have been completely successful, damage may still have occurred
- ✓ It is also possible that “silent bubbles” may cause cumulative neurological damage in divers
- ✓ The following predisposing factors may increase a diver's likelihood of getting decompression illness: deeper diving, repetitive diving, rapid or multiple ascents, dehydration, flying after diving, age, obesity, lack of fitness, exercise before, during or after the dive, ingestion of alcohol or various drugs and medications before the dive, cold water, illness or injury, fatigue.
- ✓ Allowable bottom times should be shortened to cater for predisposing factors to DCI
- ✓ Rapid ascents appear to produce more cases of neurological DCI. Severe DCI is less likely to follow a slow ascent.
- ✓ An ascent rate of about 10m/minute (from depths of about 24-30m to the surface) seems to be reasonable rate for recreational divers; a faster rate of about 15-18m/minute may be beneficial while deeper than about 24-30m.
- ✓ It is often difficult to determine and control the rate of ascent. Using an ascent line and monitoring depth and time throughout the dive assists in controlling rate of ascent.
- ✓ Multiple ascents, in the course of the same dive, increase the risk of DCI and should be avoided
- ✓ A safety stop can reduce the amount of bubbling within us
- ✓ Deeper and long dives carry a higher DCI risk
- ✓ DCI most often occurs after repetitive dives. Repetitive dive schedules must be calculated conservatively, especially for multi-day repetitive dives.
- ✓ Depth gauges should be monitored and checked periodically for accuracy. A depth gauge which reads too shallow may cause a decompression accident.
- ✓ Certain dive profiles appear less likely to cause DCI. Probably the safest profile is to descend to the maximum depth early in the dive and then gradually work shallower throughout the rest of the dive.

INHALATION OF WATER

■ SALT WATER ASPIRATION SYNDROME

CAUSE

This condition may result when a fine mist of salt water is inhaled. The mist will need to be almost as fine as that produced from pressure cans such as fly spray. Salt water as droplets



may cause the epiglottis to close so directing the water to the stomach. This can also lead to a laryngeal spasm.

Once into the lungs, the fine mist of salt water causes an acute inflammation of the alveoli. It is thought that the surfactant is affected thus reducing the ability of the lungs to allow gas transport.

SIGNS AND SYMPTOMS

The most commonly occurring signs and symptoms include shortness of breath, coughing, pain behind the breastbone and uncontrolled shivering. Others include hot and cold flushes, loss of appetite, nausea, vomiting and bronchospasm (patient appears to have asthma or an allergic reaction restricting respiration).

Some of the symptoms may take several hours to develop and the first aider must keep the patient under constant supervision. When confronted with a suspected case of salt water aspiration syndrome, pulmonary barotrauma cannot be ruled out.

MANAGEMENT



MANAGEMENT SUMMARY

- ✓ DRABC
- ✓ Observe and assess the signs and symptoms
- ✓ Keep patient still
 - ☞ If unconscious lay in recovery position
 - ☞ If conscious lay down flat, legs level
- ✓ Reassure
- ✓ Administer 100% oxygen
- ✓ Observe and monitor airway, breathing and circulation (pulse)
- ✓ Resuscitate as necessary
- ✓ Protect from exposure
- ✓ Implement company's emergency plan or contact DAN/DES and arrange recompression — DES (Australia) 1 800 088 200
- ✓ Record all details (dive profile, signs and symptoms, first aid given)

Management should follow the checklist below.

- ✓ Treat for shock by laying patient down flat, reassure, protect from elements or further exposure, monitor consciousness/pulse/breathing; send for medical aid
- ✓ Administer oxygen if available.
- ✓ If conscious, ascertain relevant details regarding dive, e.g. depth/time, ascent rate controlled or not, equipment faults, previous activities, buddy comments. This can be carried out even while the patient is breathing O₂.
- ✓ The patient should be postured laying flat or left lateral (coma position) if unconscious
- ✓ Arrange transport for further medical attention, preferably where recompression facilities are available





PREVENTION

This ailment is easily preventable as long as the cause is understood. Consideration should be given to preventing inhalation of water. This can be done by checking perforated mouthpiece, exhaust ports, diaphragm, or using poor buddy breathing or snorkelling technique.

■ NEAR DROWNING

CAUSE

Near drowning occurs when water is able to enter the lungs. The quantity and composition of water inhaled as well as duration of immersion will determine the resulting signs and symptoms.

When quantities of water enter the lung, whether it be fresh or salt water, major physiological changes occur. The major areas affected are blood chemistry, cardiac performance and respiratory performance.

In laboratory animals it has been shown that in fresh water osmosis causes haemodilution and in salt water, osmosis causes haemoconcentration. Haemodilution means water moves from the lungs to the blood, diluting it. Haemoconcentration means fluids move from the blood to the lungs causing a concentrating effect.

SIGNS AND SYMPTOMS

Near drowning will be characterized by cyanosis, unconsciousness, frothy sputum, noisy rasping breathing or lack of breathing.

Recovery from prolonged submersion can be aided by the following:

- ✓ diving reflex causes bradycardia therefore reducing oxygen demand
- ✓ hypothermia reduces oxygen demand by slowing body metabolism (concentrates blood to vital organs)
- ✓ laryngeal spasm prevents water entering the lungs

Drowning can be said to be the unsuccessful conclusion of near drowning through either prolonged immersion or irreversible biochemical damage, which results in death.

MANAGEMENT

Bring the patient to the surface and if they are not breathing immediately commence expired air resuscitation (EAR) on the surface. Assess vital signs and resuscitate as necessary. Administer 100% oxygen and treat for shock.

Medical advice must be sought immediately. It is important to note that even if the patient appears to have recovered fully he must be admitted to hospital for observation and vital blood tests. A relapse is highly probable.





MANAGEMENT SUMMARY

- ✓ DRABC
- ✓ Observe and assess the signs and symptoms
- ✓ Keep patient still
 - ✎ If unconscious lay in recovery position
 - ✎ If conscious lay down flat, legs level
- ✓ Reassure
- ✓ Administer 100% oxygen
- ✓ Observe and monitor airway, breathing and circulation (pulse)
- ✓ Resuscitate as necessary
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- ✓ Implement company's emergency plan or contact DAN/DES and arrange recompression — DES (Australia) 1 800 088 20
- ✓ Record all details (dive profile, signs and symptoms, first aid given)

MARINE ANIMALS AND THEIR DEFENCE MECHANISMS

■ INTRODUCTION

Most marine animals have evolved a means of protection from potential predators, or the means to be efficient predators. Spines, teeth, speed, cryptic colourations, and a huge range of irritants and toxins which can act either externally or internally, are examples of adaptations to these ways of life.

Naturally one or more of these attributes can be equally effective against people. Consequently, there are certain animals of which we need to be aware, respect and avoid. If, due to mismanagement or ignorance they are not avoided, it is important to know what to expect as a result, and what to do after the encounter.

The type of injury resulting from an unfavourable encounter can be divided into five categories.

- ✓ Bites
- ✓ Puncture wounds
- ✓ Stings
- ✓ Internal poisoning
- ✓ Electric shocks

■ BITES

These may result from a number of animals.

NON-VENOMOUS BITES

Some marine animals, such as grouper, killer whales, seals and sharks have been known on rare occasions to injure divers.



The wounds from bites are generally extensive with ragged edges. Tissue injury and haemorrhaging are usually severe. Blood loss, shock and secondary infection are the main features causing concern. In severe cases, quick and efficient first aid is of vital importance.

It is important to note, however, that the most likely cause of this type of injury will be boat propellers, broken glass or divers falling and hurting themselves.

MANAGEMENT



Management Summary

- ✓ DRABC
- ✓ Observe and assess the signs and symptoms
- ✓ Broad pressure bandage (see pressure immobilisation)
- ✓ Splint
- ✓ A second broad pressure bandage

Prevention

Sharks

Despite the fact that there are very few instances of sharks attacking a fully submerged diver, it is wise to avoid large sharks at all times. If you encounter sharks, leave the water as soon as possible. If immediate exit is impractical, remain submerged and appear aggressive. Banging on the cylinder with knife handle or rock and shouting into the water has been effective in driving sharks away. When diving in an area where dangerous sharks are known to exist, be extremely cautious with speared fish or blood in the water, as these conditions will attract them. Here are a few facts to help dispel the myth that sharks are an enemy of the diver:

On average there are 1,000,000 dives conducted in Australia each year. The statistics over the last few years average out at about one shark attack every 2-2.5 years. The odds of a diver being attacked/killed by a shark are roughly 1:2,000,000. This is not bad; according to the latest statistics, the average person has a 1:8,500 chance of being killed in a car accident. You are, in fact, statistically more likely to die from a bee sting or driving to the dive site than a shark attack!

Orcas

Are very rarely seen underwater by divers and there are no recorded attacks. However, due to their size, speed, intelligence, and voracious appetites, divers should leave the water immediately if orcas are sighted in the area.

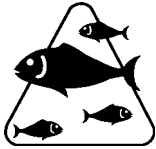
Eels

Moray, conger, and spotted eels are all of the same family and their behaviour patterns are usually the same. They live in caves or holes and are rarely aggressive. If disturbed, they will bite. Their bite is not poisonous but is extremely susceptible to infection. Divers should never stick their hands into a hole or cave without a preliminary inspection first.

Seals, Sea Lions and Walrus

Are usually playful in the water, although the males tend to be aggressive near rookeries, if an aggressive male appears, it is wise to leave the water.





NOTE Avoid trying to hand feed animals underwater. Although this can give a feeling of interaction with them, it can sometimes result in the animal mistaking the divers hand for food and getting bitten. Hand feeding can also lead to unnatural behaviour patterns being established which may be detrimental to the animal's survival. Well meaning divers have unintentionally caused the death of some creatures by feeding them food which they cannot digest, or by passing on infections when stroking or handling them. These are wild creatures and divers need to carefully consider their motives and the effects of establishing relationships with underwater marine animals.

VENOMOUS BITES

There are principally two animals that are known to bite causing injection of venom into the wound. They are blue-ringed octopus and the sea snake. However, Cone Shell (which provides a venomous puncture wound) is included in this group as the venom and the first aid management is the same as for sea snake and blue ringed octopus.

Blue-ringed Octopus

A blue ringed octopus grows to a maximum of 20cm from tip to tip of its tentacles. When undisturbed, this animal has a yellowish-brown colour with dull ringed markings on the tentacles and stripes on the body. When disturbed, these dull markings become iridescent blue. The initial bite is almost painless. After about 15 minutes the bite becomes swollen, resembling a mosquito bite. A few minutes after the bite a rapid, painless paralysis results. The symptoms progress as follows:

- ✓ Numbness around the mouth and neck, which may cause difficulties in swallowing
- ✓ Nausea and vomiting
- ✓ Breathing difficulties arise fairly quickly
- ✓ The eye muscles gradually become paralysed
- ✓ General weakness and difficulty with coordination leads to complete paralysis which persists for 4 to 12 hours

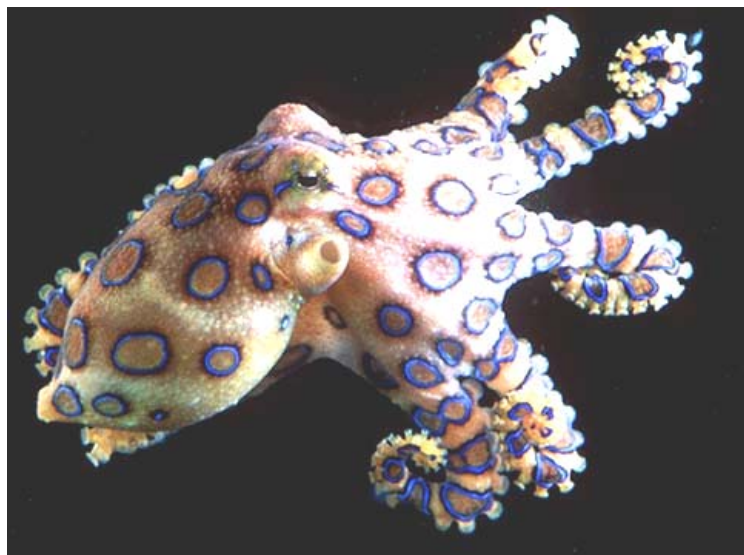


Figure 23. Blue Ringed Octopus



Sea Snakes

There are a number of different species of this marine reptile, some being more dangerous than others. The toxins interfere with nerve and muscle function. The initial puncture is noticed but is not painful. Symptoms may take 10 minutes to several hours to manifest themselves. Restlessness, nausea and vomiting may occur, followed by general stiffening and aching. Weakness develops into paralysis and spreads. This may be preceded by muscular twitching and spasms. Respiratory and cardiac distress and failure may also occur.



Figure 24. A Sea Snake on the beach, note the flat tail at the left of the picture

Cone Shells

Although more prevalent in tropical waters, cone shells are found in temperate waters. These animals have a minute, rasp-like feeding appendage (radula) which can thrust out of the narrow end of the shell. The radula has a series of 1-20 teeth which penetrate the skin and inject the venom. These specialised teeth are generally used for immobilising prey, but may also be used for protection. The shell should never be picked up by divers.

The toxins injected affect the nerve muscle function. The initial puncture may be painless or associated with excruciating pain. Numbness and tingling may extend from the bite to involve the whole body, particularly the mouth and lips. This process takes around 10 minutes. Mild to severe muscular paralysis may result within 10-30 minutes, including respiratory paralysis. Cardiac arrest may also occur. The patient's general condition deteriorates for 1-6 hours, after which improvement is likely.



Figure 25. A cone shell



MANAGEMENT



Management Summary

- ✓ DRABC
- ✓ Observe and assess the signs and symptoms
- ✓ Broad pressure bandage (see pressure immobilisation in chapter 4)
- ✓ Splint
- ✓ A second broad pressure bandage



PREVENTION

Wear protective clothing, boots and gloves when diving in areas where these types of sea life are present. Avoid collecting bottles and shells which could house the tiny blue ringed octopus.

PUNCTURE WOUNDS

These generally result from spines or darts. They may be complicated by toxins or irritants.

PUNCTURE WOUNDS WITHOUT VENOM

This type of wound is probably the most common injury resulting from contact with the marine animal. These wounds often occur while cleaning fish, exploring reefs, and while handling specimens.

Fish with Spines

A common example is the Old Wife. These fish have long, strong spines as part of their fins. The presence of venom is doubtful. The puncture takes the form of a sharp prick. This may develop into an ache, or severe pain extending from the site of the injury to include the whole limb. Bleeding is variable depending on the wound. The ache may last for several hours.

Sea Urchins

Sea urchins are the most common marine animal encountered by divers that cause a puncture wound. The spines of these animals are long, sharp and brittle. The spines tend to break off within the tissues. Pain occurs immediately after penetration. Swelling or inflammation usually occurs. The area of inflammation may increase and begin to ache. This ache can extend to involve the whole limb. The lymph glands become tender and swollen. General weakness and shock may result. With sea urchin spines, removal should not be attempted unless it can be accomplished without breaking the spine.

MANAGEMENT



Management Summary

- ✓ DRABC
- ✓ Observe and assess the signs and symptoms
- ✓ Remove spine if possible
- ✓ Local anaesthetic (spray type is better)
- ✓ Bath wound in warm water (50 °C/122 °F - test first)
- ✓ Antivenene if available





Prevention

Wear protective clothing and gloves when diving in areas where these types of sea life are present.

PUNCTURE WOUNDS WITH VENOM

Fish with Spines

Many fish are particularly well endowed with spines. The butterfly cod (lionfish), stonefish, and catfish are common examples found within tropical waters.

In these fish, the spines are covered by a sheath which is pierced and displaced when contact is made with the victim. Venom then passes along the spine into the wound. Severe pain is experienced at the affected site. This gradually increases in intensity. The intense pain lasts for several hours and may persist as a dull ache for a few days. The wound is susceptible to secondary infection.

Pain may extend to the lymph glands of the groin and armpit. Distress as a result of the pain may lead to mild delirium. Nausea, vomiting, sweating, fever and shock may also result. Respiratory failure may occur. Weakness and exhaustion may last for several days.

It is unlikely to be fatal except in the case of the stonefish. These fish have short stout spines covering the head and gill covers which may inflict a simple puncture wound.

Stingrays

Stingrays have a large serrated spine situated towards the posterior end of the tail. The "stinging" response of the animal occurs when pressure is applied to the posterior section of the animal's back. The tail is thrust upward and forward, driving the spine into the victim. A sheath over the spine is ruptured during the process. Some stingrays have associated venom which passes down the spine and into the wound.

The wound may vary from a puncture to a laceration up to 15cm/6 inches in length. Pain is immediate and increases over the first hour or two. There is some easing after 6-12 hours but pain may persist for some days. Bleeding is variable depending on the type of wound.

Nausea, vomiting, diarrhoea, fever, fainting and excessive salivation may occur. Pain may extend to the lymph glands of the groin and armpit. Respiratory depression, coughing and pain on inspiration may complicate matters, particularly in those cases where the venom has been injected.

MANAGEMENT SUMMARY



Management Summary

- ✓ DRABC
- ✓ Observe and assess the signs and symptoms
- ✓ Remove spine if possible
- ✓ Local anaesthetic (spray type is better)
- ✓ Bath wound in warm water (50 °C/122 °F - test first)
- ✓ Antivenene if available

Prevention



When sting rays strike, it is a purely defensive action. A painful encounter can be avoided by shuffling ones feet while wading in shallow water. This action disturbs the ray, causing it to swim away. Do not touch cone shells. Wear protective clothing and gloves.



■ STINGS

Animals that have tentacles coated with stinging cells (nematocysts) belong to a group of creatures called the cnidarians (hollow gutted animals) or coelenterates and include corals, anemones, hydroids, fire urchins and sea jellies.

Sea Jellies

Examples of the better known species are; blue-bottle, mauve stinger, jimble, and the most dangerous of all, the box jelly. Portuguese Man-of- War is a compound hydroid rather than a sea jelly but gives similar symptoms.

Generally, a few seconds after contact, a stinging sensation is felt and this increases in intensity for a few minutes. A red coloured reaction surrounds the area of contact and may rise up in the form of a pimple. Lesions, welts and blisters often result, the shape of which may be characteristic of the animal responsible. The pain may extend to the lymph glands of the groin and armpit.

General symptoms include; abdominal pains, cramps, muscular aches, respiratory distress, sweating, anxiety and restlessness. Nausea and vomiting may also occur. Symptoms generally diminish within 4-12 hours. Convalescence may take up to a week. In the case of the box jelly, if death occurs, it usually occurs within the first 10 minutes.

Anemones (Including Corals and Hydroids)

The initial symptoms vary from a prickly sensation over the affected area to a severe burning pain. This occurs immediately on contact and may increase in the next few minutes. It may extend up to the lymph and involve the regional lymph glands in the groin and armpit. Pain generally lessens in a few hours but a residual ache or itch may persist for weeks or months.

Small blisters may develop. In severe cases, ulceration may result. Secondary infection may be a problem. Nausea, fever, chills, thirst, abdominal pain and cramps may also result. In severe cases, shock and respiratory distress may occur.

MANAGEMENT

Box Jellyfish



Priority lies with immediate and appropriate resuscitation. Vinegar inhibits the discharge of all known box jellyfish nematocysts. So it should be used to douse all known box jellyfish stings. The stings can then be scraped off with a knife. Apply vinegar first and then a pressure immobilisation bandage. Soak the bandage in vinegar if practicable. Administer antivenom if available. Vinegar plays no role in pain relief therefore pain relief methods must be used together with treatment for shock.



Figure 26. Box Jelly Fish



Other Jelly Fish Stings

Vinegar may actually discharge other jelly fish stings. Therefore, for these painful but not life threatening stings, nematocyst inhibition is not as high a priority. The stings can be scraped off with a knife or picked off using the thicker pads of the fingers. Ice packs or cold packs can then be applied for pain relief, much like treating a burn. Ice should not be applied directly as the ice melting may cause the nematocysts to discharge.



Management Summary

- ✓ DRABC
- ✓ Observe and assess the signs and symptoms
- ✓ Remove tentacles by gently shaving the area with a diver's knife
- ✓ Apply ice packs
- ✓ Do not rub
- ✓ Local anaesthetic (spray kind)



- ✓ Do not rub the stung area, especially with sand
- ✓ Do not apply pressure immobilisation over stung area
- ✓ Do not apply tourniquet or ligature
- ✓ Do not apply fresh water
- ✓ Do not move seriously affected patients
- ✓ Do not interrupt or stop CPR
- ✓ Do not put vinegar on blue bottle stings or sea jellies south of Bundaberg on the east coast or south of Geraldton on the west coast



Prevention

Avoid touching because stings are painful. When diving in an area where large concentrations of jelly fish or anemones are present, wear an environmental protection system. Most sea jellies can cause problems if they touch you when you are not wearing protective clothing. Avoid laying or leaning on the reef.



INTERNAL POISONING

Many fish are known to be poisonous when eaten. The most common belong to the family of fish called Tetradons. These include toadfish, puffers, porcupine fish and sunfish.

The onset of symptoms varies according to the toxin ingested. They include general weakness, dizziness, incoordination, numbness and cramps. Sweating, chest pain, headache, nausea, vomiting and diarrhoea may be noted. Respiratory distress may also occur.



PREVENTION

Only eat what you know to be safe. Use a good fish identification book.



■ ELECTRIC SHOCKS

Electric rays or numbfish are slow and ineffective swimmers. The electric discharge varies from 8 to 220 volts. The ray can deliver a successive series of shocks. The electric shock may be disabling, a major threat coming from drowning. Recovery is uneventful and treatment is not usually required.

■ MARINE ANIMAL INJURIES IN REMOTE AREAS



In the situation where medical aid is unavailable for some hours, extra care must be taken to avoid the potential injuries from marine organisms. Dive supervisors have the responsibility to know the location and relevant telephone numbers of the nearest medical facilities and emergency services. They should also ensure the presence of a comprehensive first aid kit which should be put together in consultation with a doctor who is aware of the added problems associated with diving and the marine world.

Finally, the responsibility rests with the individual to avoid any situation which may lead to injury. This is particularly important in remote areas. Animals which are unknown should be left alone and not touched or eaten. Similarly, the diver should stay well within their capabilities.

MISCELLANEOUS PROBLEMS

■ UNDER WATER BLAST INJURIES

During an underwater explosion the blast wave or pressure wave is carried over greater distance in the water than in air. Therefore the danger area is greater. Standing near a small explosion in air may result in no injuries to the diver. The same explosive charge in water over the same separation could be fatal.

Injury from underwater blasts occur mainly in the gas/tissue interfaces of the body as in barotrauma - lungs, ears, sinuses, gut etc. One of the most common symptoms is abdominal pain. The diver may not appear to be seriously injured in the early stages and there may be no external signs of injury.

Management of a victim caught in an underwater explosion is the same for any other severe body trauma. Regardless of the apparent lack of injury or pain the victim must seek medical advice as abdominal injuries may take some time to manifest themselves.

Prevention of this problem is straightforward. Divers must exit the water before any underwater blasting. If diver can't exit the water elevate chest and abdomen out of the water (Float on the surface on your back).

■ CAROTID SINUS SYNDROME

The Carotid Sinus is the point in the carotid arteries where they branch to the brain and facial tissues. This enlarged area has several nerve endings called baroreceptors and chemoreceptors. These receptors monitor pressure on the throat and blood chemistry respectively.

If pressure is exerted over the carotid sinus area by tight fitting wetsuits the diver may become aware of an uncomfortable feeling of nausea and will tend to show signs of agitation. This external pressure has artificially stimulated the baroreceptors and causes vasodilation, bradycardia and hypotension. These are natural responses to high blood pressure. If allowed to continue, Carotid Sinus Syndrome may cause confusion and disorientation.



The treatment is to remove the cause i.e. loosen or remove hood.

■ ASEPTIC BONE NECROSIS (DYSBARIC OSTEONECROSIS)

CAUSE

The cause of aseptic bone necrosis is not fully understood. However, deep diving and untreated decompression illness may cause the blood vessels supplying the bones to become blocked. Generally, the long bones of the upper arm and upper leg are most affected. If the blood supply to the bone tissue is blocked, oxygen and other essential nutrients will not be supplied and the bone tissue will die. This condition is known as aseptic (without infection) bone necrosis (death), i.e. non-infectious death of bone tissue. It is not common in recreational divers.

SIGNS AND SYMPTOMS

Aseptic bone necrosis can only be detected by X-ray examination. Manifestation can take up to nine months. In more serious cases the joint will become immobile and limb movement will be severely hindered. Commonly affected joints are shoulder and hip.

PREVENTION

The correct use of decompression tables, conservative use of diving computers and avoiding deep or repetitive dives will help to prevent aseptic bone necrosis. Early recognition of the condition is imperative so that further damage can be prevented by stopping diving activities. Long bone x-rays are recommended three to 6 months after any incidence of DCI or if minor joint pain persists.

