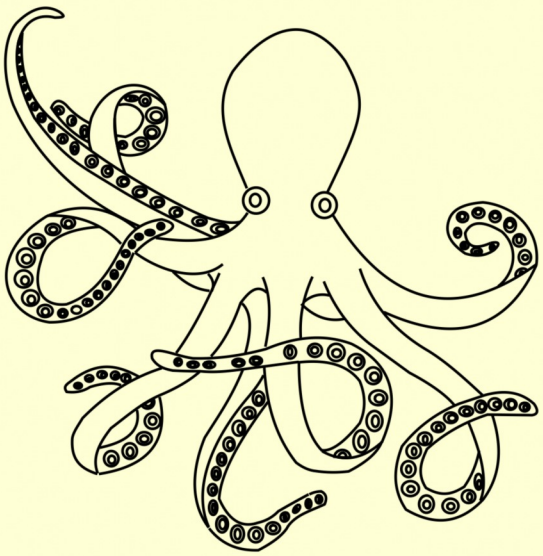

Diving & ROV specialists

Surface supplied diving using DCIEM tables

Book #1

Description and prevention of diving accidents

September 2024

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Diving & ROV Specialists

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This document is book number one of the ensemble of three books constituting the "Surface-supplied diving handbook" described underneath.

Books	Description
Book #1: Description and prevention of diving accidents	This document describes the accidents linked to surface-supplied diving and the procedures to solve and avoid them.
Book #2: Definition and elements for preparation	The document describes the scope of surface supplied diving procedures, the DCIEM decompression tables, and some elements to consider when organising a surface supplied diving project such as the necessary personnel, organization of the maintenance of the diving system, weather conditions, surface supports, systems of communications, work procedures with ROV, documents that must be available, etc.
Book #3: Air and nitrox procedures using in-water & surface decompression	This document describes procedures for safe air and nitrox dives using in-water air decompression, in-water nitrox decompression, in-water oxygen decompression at 6 m, and surface oxygen decompression.
Complementary books that have not yet been published but are planned shortly.	
Book #4: Air & nitrox diving procedure using scuba replacement	Diving using SCUBA replacement systems has widely evolved throughout the years. A particular organization is necessary for these operations, whose limitations are more stringent than normal surface-supplied diving operations and require specific diving systems. In addition, these procedures include the conception or the organization of relevant surface supports, and this aspect of the organization is essential. For these reasons, it appears logical to describe the organization of such operations in a separate book.
Book #5: Air & nitrox procedures using O2 decompression in wet bell	Wet bells provide numerous advantages over diving baskets when they are well-designed. That includes bells that are sufficiently light and as compact as baskets to be easily operated from lightweight surface supports, which is not the case for many units currently in use. In addition to setting up the elements for designing adequate wet bells, the document will provide procedures for using them and their limitations.
Book #6: Heliox diving procedure with basket and wet bell	Even though heliox surface-supplied diving procedures provide the advantage of diminishing the effects of narcosis, they imply numerous precautions and have some disadvantages, such as longer decompression stops than air diving, limiting their possibilities of use. These specific precautions and the limitations of these procedures will be discussed in this separate book

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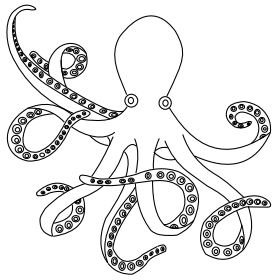
Important Note

This book is written with the only aim of informing people interested in diving activities of elements to take into account to prepare safe and successful diving operations.
I express my sincere thanks to the people who are supporting this project and provided me with useful documents and advice, particularly Jean Pierre Imbert (Divetech) and doctor Jean Yves Massimelli.

Kindly note that this document does not replace specialized diver medical courses.

Christian CADIEUX - Author

Revision	Date	Elements modified or added
Revision 1	20/11/22	Document published
Revision 2	09/08/23	Updated the topic “Adverse effects of hyperbaric oxygen” with the Equivalent Surface Oxygen Time (ESOT), a concept based on the revision of Arieli’s K concept by doctors Risberg and van Ooij, also published in DMAC 35.
Revision 3	05/09/24	Modified the text for diving accidents



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Part A - Summary of diving accidents

Purpose

This summary identifies accidents and illnesses linked to diving activities by their main symptoms.
Note that diseases or accidents that are not of the same origin can have similar symptoms. For this reason, symptoms should not be used alone but associated with other elements, such as the working conditions, the dive profile, the victim's physical condition, etc., to try identifying their origin. That highlights the importance of the diving medical specialist, who must be contacted in priority for any abnormal condition.

More details about these accidents are developed in point “C - Accidents linked to saturation diving”.

Symptoms linked to diving accidents.

Symptoms	*Possible accident*	*Possible causes*
- Speech impairment - Fixed ideas - Sluggish reactions - Confusion - Irritability - Amnesia - Muscular rigidity - Collapse , fainting - If nothing is done: death	Hypothermia	- Body T° equal or less than 35° (death occurs at 25°) - Too long time in cold water with an inappropriate suit, or insufficient hot water delivery to a hot water suit.
- Rapid breath rate and spasmodic respiration when reaching the water or suddenly at depth in cold water	Cold shock	- Due to insufficient thermal protection when launching the dive or the suit opening suddenly at depth. - Not to be confused with hyperventilation due to worrying, which often starts before launching the dive.
- Headache - Dizziness - Dry skin - Hot and flushed (red skin) - Dilated pupils - Confusion - Shallow and fast breathing - Weakness - Convulsions - Rapid and weak pulse - Collapse and loss of consciousness	Hyperthermia	The victim's core temperature is equal to or greater than 39°, usually as a result of a too-warm environment.
- Pain in the forehead - Pain in the cheek area - Bleeding nose - Inability to clear the ears	Sinus Barotrauma	Infection or irritation of the sinuses
- Pain in the ear - Red color eardrum - Impaired hearing	Barotrauma of the external ear	- Bad equilibration during compression - Blockage of the eustachian tube
- Pain under the ear - High-frequency noises - Loss of hearing	Barotraumas of the middle ear	- Bad equilibration during compression - Blockage of the eustachian tube - Oval or/and round windows damaged

Symptoms linked to diving accidents. (continuation)

Symptoms	*Possible accident*	*Possible causes*
- Upset balance - Vertigo , Nausea - Affected hearing - If using face mask with the ears exposed to the water: Sudden vertigo followed by stabilization	Eardrum rupture	- Bad equilibration during compression - Blockage of the eustachian tube - Water intrusion in the middle ear
- Pain under the ear - High frequency noises (tinnitus) - Dizziness - Nausea - Loss of hearing	Barotraumas of the inner ear	- Bad equilibration - Blockage of the eustachian tube - Oval or/and round windows damaged or ruptured - Cochlea damaged
- Pain in the maxillaries, mostly during ascent	Teeth barotrauma	- Breathing gas trapped in the teeth
- Chest pain - Bleeding lungs - Difficulty breathing - Pain in the neck and the shoulders - Bleeding ears - Hemorrhages of the eyes and the nose - Bruised and puffy cheeks	Squeeze	- Suction into the helmet in case of no return valve failure resulting in depression in the helmet - Often associated with continuous flow helmets *(Can lead to Pulmonary barotraumas associated with fractures of the ribs and /or collar bones in severe cases)*
- Bruising of the skin	Nips	- Due to the external pressure on the suit, creating folds which will trap and pinch the skin - Often associated with dry suits
- Visual disturbance (tunnel vision) - Nausea - Hearing problems - Twitching - Irritability - Dizziness - Convulsions	Acute oxygen poisoning *(Paul Bert effect)*	- Too elevated oxygen partial pressure - Diver suffering from intolerance to oxygen
After a long time under oxygen : - Dry irritated throat - Tight feeling in the chest - Dry, painful cough - Painful fingertips - Shortness of breath - Mild tingling in the lungs followed by burning sensation.	Chronic oxygen poisoning *(Lorrain Smith effect)*	- Oxygen breathed at a partial pressure above 0.5 bars during long periods
In the water or in chamber: - Exaggerated euphoria or anxiety - Lack of memory - Irritability - Hallucinations - Lack of coordination - Sudden panic - 1st visible symptoms around 30 m with air diving.	Narcosis	- Too elevated partial pressure of nitrogen (air diving). - Narcotic gas in the mix (nitrogen, argon, etc.)
- Headache - Increased breathing - Superficial breath - Feeling apprehension - Panic	Hypercapnia	- Too elevated CO_2 partial pressure in the blood (over 0,059 / 45 mm hg)

Symptoms linked to diving accidents. (continuation)

Symptoms	*Possible accident*	*Possible causes*
- Cyanosis, bluish tinge - Poor coordination - Breath increased - Increased heart rate	Hypoxia	- Oxygen Partial pressure below 160 mb
- Loss of conscious - Death follows quickly	Anoxia	- No oxygen in the breathing mix
- Increased respiratory rate. - Casualty vomiting - Pulmonary oedema - Unconsciousness	Drowning	- Airways invaded by a liquid The symptoms are linked to catastrophic events during the dive
- Difficulty breathing - Pain behind the breastbone - Sensation of fullness	Mediastinal emphysema *(Pulmonary barotrauma)*	- Gas trapped in the space between the lungs
Symptoms in addition to those listed above: - Gas trapped below the skin near shoulders and around the neck - Hoarse voice - Sensation of fullness of the throat	Mediastinal emphysema with subcutaneous emphysema *(Pulmonary barotrauma)*	- Gas trapped in the mediastinal space also trapped under the skin of the shoulders and neck
- Chest pain - Restricted chest movements - Difficulties breathing - Chest moving without venting - Blood vessels of the neck may be swollen	Pneumothorax *(Pulmonary barotrauma)*	- Gas trapped within the pleura NB: In slight cases can be only detected by X rays
Generally, 5 to 10 min after surfacing: - Headache , dizziness - Visual disturbance - Confusion, hallucinations - Loss of coordination - Abnormal gait - Paralysis - Collapse - Symptoms similar to infracts	Arterial Gas Embolism *(AGE)*	- Gas bubbles entering the blood vessels affecting the nervous system and the circulatory system
- Skin itching - Rash under the skin	Skin only decompression accident *(classified type 1)*	- Pathogen bubbles in the small vessels under the skin
- Localized non-radiating pain, generally in a joint (elbow, wrist, knee, etc.)	Pain only decompression accident *(classified type 1)*	- Bubbles trapped in or around joints
Radiating pain - Radiating pain in the shoulders or the limbs - Girdle Pain in the lumbar area - Numbness, tingling - Decreased sensation to touch - Paraplegia/paralysis - Loss of bladder and bowel control	Spinal decompression accident *(classified type 2)*	- Bubbles trapped In the spinal cord . The affected area depends on the location of the bubble in the spinal cord
- Vertigo - Loss of hearing - Nausea - Nystagmus (also called dancing eyes)	Vestibular decompression accident *(classified type 2)*	- Bubbles in the cochlea, the vestibulocochlear nerves, the cerebellum or cortical pathways.
- Headache , dizziness *See the continuation on the next page*	Cerebral decompression accident *(classified type 2)*	- Pathogen bubbles trapped in the brain

Symptoms linked to diving accidents. (continuation)

Symptoms	*Possible accident*	*Possible causes*
Continuation of the previous page - Visual disturbance - Confusion, hallucinations - Loss of coordination - Abnormal gait - Paralysis of one side of the body - Collapse	Cerebral decompression accident *(classified type 2)*	- Pathogen bubbles trapped in the brain
- Deep aching pains - Gritting and popping within the joints - Symptoms may occur around 30 metres at rapid compression rates - At a slow compression rate, symptoms are not present before 90 metres - The symptoms usually disappear in reverse order during the ascent.	Compression arthralgia	- It is thought to result from the sudden increase in tissue gas tension surrounding the joints causing fluid shifts and interfering with joint lubrication. - Often linked to too fast compression
- Burning sensation on swallowing - Nausea and vomiting - Abdominal cramps - Weakness - Anesthesia - Hallucinations - Changes in color perception - Blindness - Seizures - Coma	Hydrocarbons intoxication	Ingestion or breathing of hydrocarbons
- Eye and respiratory tract irritation - Loss of smell - Headache and nausea - Loss of reasoning - Loss of balance - Unconsciousness - Breathing will stop within minutes	Hydrogen sulfide (H_2S)	- Hydrogen sulfide breathing
- Headache - Dizziness - Nausea - Loss of consciousness - Death if nothing is done	Carbon monoxide poisoning	- Carbon monoxide breathing
following maintenance of the air/gas supply system - Nausea, vomiting and abdominal cramps. - Weakness - Anaesthesia - Blindness - Coma	Cleaning fluids	Contaminants into the chamber, gas piping, and/or the gas storage..
- Upper respiratory flu or cold like symptoms - Coughing - Vomiting - Diarrhea - Pneumonia - Headaches - Dizziness - Skin lesions *See the continuation on the next page*	Contamination by chemicals	Some chemical contaminants easily blend with the water and can enter in the body by ingestion, inhalation, or contact with the skin

Symptoms linked to diving accidents. (continuation)

Symptoms	*Possible accident*	*Possible causes*
Continuation of the previous page - Headache - Visual disturbances - Paralysis, - Convulsions - Loss of conscious (When diving in rivers, lakes, ports or near some polluted facilities)	Contamination by chemicals	Some chemical contaminants easily blend with the water and can enter in the body by ingestion, inhalation, or contact with the skin
- Rash - Itchy skin. - Fever or chills - Muscle aches - Allergic reaction, - Cough / asthmatic attack. - Nausea & vomiting, - Diarrhea, - Blood in urine - Loss of weight - Jaundice - Nervous manifestation	Contamination by parasites	Most parasites are acquired by ingestion, but some gain entry into the body by skin contact.
- Dry mouth - Difficulties swallowing - Nausea & vomiting - Abdominal cramps/diarrhea - Jaundice - Faeces with blood - Difficulties in breathing - Fever & dehydration - Nose bleeds - Vision troubles/hallucinations - Ear canal pains and swellings - Fickle mood - Fatigue/lethargic feeling - Muscle weakness/cramps - Collapses	Contamination by Bacteria	Bacteria can be found in almost any environment and sometimes concentrate in a thin layer on the water surface, or a thin layer on the top of sediment.
- Gastrointestinal disturbances - Weight loss - Headache - Fever, - Delirium, - Seizures - Difficulty breathing - Sore throat - Cough - Fatigue & Lethargy - Myalgia - Paralysis - Death	Contamination by Virus	Viruses are the smallest pathogen agents. They are difficult to detect and treat.
- Nausea & vomiting - Diarrhea - Skin burns (skin reddening) - Weakness - Lethargy and fatigue - Loss of appetite (anorexia) - Fainting *See the continuation on the next page*	Exposure to radiations	

Symptoms linked to diving accidents. (continuation)

Symptoms	*Possible accident*	*Possible causes*
Continuation of the previous page - Dehydration - Inflammation of tissues (swelling, redness or tenderness) - Hemorrhages under the skin - Bleeding from your nose, gums or mouth - Anemia - Hair loss (usually from just the scalp) - Decrease in platelets - Death can happen quickly	Irradiation	Direct exposure to radioactive materials
- Tinnitus - Difficulty of hearing in noisy areas - Sensation of muffling - Difficulties understanding words, especially when there is background noise - The need to ask others to speak more slowly, clearly and loudly - Memory disturbances - Intellectual impairment - Depression - Permanent sensorineural hearing loss - Dizziness - Nausea - Symptoms occur after diving in noisy surrounding	Hearing loss	Exposure to: - Noisy tools - Noisy surrounding - Sonar
- Blurred vision - Lightheadedness - Vibratory sensations in hands, arms and legs - Tremors in upper extremities . - Pain cochleo-vestibular - Change in rate of respiration - Skin tension	Harmful noises	Diving operations in noisy surroundings or in the vicinity of boats using low frequency sonar
- Sudden acute abdominal pain like a kick in the stomach. - Transient paralysis of the lower limbs - Nausea, vomiting (with or without blood) - Sensation of an electric current passing through the body - Testicular pain - Chest discomfort followed by expectoration of blood or sputum (hemoptysis) and hiccups - Tachycardia - Cyanosis - Mild to severe shock - Loss of hearing - Disorientation - Delirium - Unconsciousness - Amnesia - Rectal bleeding may be apparent. - Death can occur quickly	Underwater explosion	In an underwater explosion, the surrounding water doesn't absorb the pressure but moves with it.

Symptoms linked to diving accidents. (continuation)

Symptoms	*Possible accident*	*Possible causes*
- Hypertension - Tachycardia - Dysrhythmias, ventricular fibrillation, Asystole - Impaired ventilation - Hypo - Fractures (due to muscular contraction) - Motor deficit - Sensory deficit - Problems hearing - Shock - Loss of conscious - Coma - Death	Electric shock	Exposure to electricity and electrical fields
- Cuts or abrasion of the skin	Coral cuts	- Insufficient skin protection/ Inappropriate diving suit
- Intense pain - Nausea and vomiting for two to three hours - Small welts on the skin with red lesions - Swelling, blisters, and pus-filled incrustations - Itching or welts remaining on the skin	Contact with fire coral	- Insufficient skin protection / Inappropriate diving suit
- Small linear eruptions that develop rapidly at times surrounded by a raised inflammation - Blisters or even necrotic ulcers - Weakness - Nausea - Headache - Muscle pain and spasms - Abundant tears and nasal discharge - Increased sweating - Changes in pulse rate - Chest pain	Contact with anemone	- Insufficient skin protection / Inappropriate diving suit
- Dermal irritation - Abnormal increase in sensitivity to stimuli of the sense, and sensation of tingling, tickling, prickling, pricking, or burning - Redness of the skin due to increased blood flows of the capillaries, with or without papule and vesicle development	Contact with sponge	- Insufficient skin protection / Inappropriate diving suit
- Spines with pain	Contact with ea urchin	- Insufficient skin protection / Inappropriate diving suit
- Inflamed and swollen region, sometimes white and ischaemic (Insufficient blood supply), with a cyanotic area surrounding it - Numbness and tingling in the whole body, and especially the mouth and lips (10 minutes to develop) *See the continuation on the next page*	Poisoning by cone shell (Sting)	- Insufficient skin protection/ Inappropriate diving suit - Inappropriate handling

Symptoms linked to diving accidents. (continuation)

Symptoms	*Possible accident*	*Possible causes*
Continuation of the previous page - Skeletal muscular paralysis that may spread from the site of injury - Difficulty with swallowing and speech. - Double and blurred vision (paralysis of voluntary muscles and pupillary reactions) - Respiratory paralysis with shallow rapid breathing and a cyanotic appearance preceding apnea, unconsciousness and death. - Cardiac failure - If the patient survives, he/she is active and mobile within 24 hours.	Poisoning by cone shell (Sting)	- Insufficient skin protection/ Inappropriate diving suit - Inappropriate handling
- Pain increasing in intensity, often coming in waves. - Multiple interlacing whiplash lines red, purple, or brown, 0.5 cm wide - Patient confused, acting irrationally - Consciousness disturbance. - Hypotension, tachycardia, and a raised venous pressure - Coma and death - Clinical state oscillating from hypertension episodes, tachycardia, rapid respiration, and normal venous pressure to those of hypotension, bradycardia, apnea, and elevated venous pressure. - Respiratory distress, pulmonary congestion, oedema, and cyanosis may be due to cardiac effects or a direct midbrain depression. - If death occurs, it usually does so within the first 10 minutes; survival is likely after the first hour. - Amnesia - Paralysis and abdominal pains.	Contact with Box jelly fish	- Insufficient skin protection/ Inappropriate diving suit
Symptoms visible after a latent period from 10 minutes to several hours - 4 to 20 teeth marks - Euphoria, anxiety, or restlessness. - Thirst, dry throat, nausea, and vomiting - Generalized stiffness and aching - Muscle weakness - Paralysis that extends centrally from the area of the bite - Muscular twitching, writhing, and spasms - Difficulty to speak and swallow - Paralysis may extend to the bulbar areas - Facial and ocular paralysis may develop. - Necrosis of muscles and damage in the kidneys that may develop in several hours - Respiratory distress, shortness of breath, cyanosis, and finally, death.	Bitten by sea snake	- Insufficient skin protection - Improper behaviour

Symptoms linked to diving accidents. (continuation)

Symptoms	*Possible accident*	*Possible causes*
- Excruciating pain that increases in severity and sometimes comes in waves. - unconsciousness - Ischemia of the area is followed by cyanosis - Area swollen and edematous, often hot, with numbness in the centre and extreme tenderness around the periphery - Paralysis of the adjacent muscles immobilizing the limb. - Pain likely to spread proximally - Cardiac failure like bradycardia, or cardiac arrhythmias - Pallor, sweating, hypotension, and syncope - Cardiac arrest is possible. - Respiratory failure - Cardiac arrest. - Delayed healing, necrosis, and ulceration	Poisoning by stone fish	- Insufficient skin protection - Improper behaviour - Animal not seen
- Local pain that increases in intensity - Anaesthetized puncture wound with hypersensitivity to the surrounding area. - One or more puncture wounds, with an inflamed and sometimes cyanotic zone. - Area that is pale and swollen, with pitting oedema surrounding the cyanotic zone - Foreign body reaction, chronic localized inflammation, edema, necrosis, and severe disability - Casualty distressed, he may fall into a delirious state. - Malaise, nausea, vomiting, and sweating - Respiratory distress may develop - Cardiovascular shock state may supervene and lead to death	Pricked by a small fish	- Insufficient skin protection - Improper behaviour - Animal maybe not seen
- Bite that results in minimal pain for the first 5-10 minutes, and then the bitten area gets numb. This condition spread to the rest of the limb. There may be some bleeding - Nausea, vomiting, and difficulty swallowing. - Double vision, blurred vision, fixed dilated pupil - Numbness or loss of feeling around the lips and mouth. After approximately 10 minutes, the victim may have general weakness, difficulty breathing, and paralysis. - Respiratory failure may occur, which may lead to unconsciousness, cardiac arrest, and death.	Bitten by octopus (Blue ringed)	- Insufficient skin protection - Improper behaviour - Animal not seen

Symptoms linked to diving accidents. (continuation)

Symptoms	*Possible accident*	*Possible causes*
- Wound ragged with massive bleeding. - Casualty sweaty (cold and clammy) with a rapid pulse, hypotension - Syncope may happen. - Secondary infection in the area is common.	Bitten by moray eel	- Insufficient skin protection - Improper behaviour - Animal not seen
- Immediate pain increasing over 1 to 2 hours and easing after 6 to 10 hours - Swollen and pale area, with a bluish rim, spread around the wound after one or two hours. - Pain may be constant, pain pulsating, or lancinating - Possibility of massive bleeding - Spine integument may be visible - Anorexia, nausea, vomiting, diarrhea, salivation with frequent urination. - Muscular cramps, tremors, tonic paralysis with fever, and sweating. - Difficulty in breathing, cough, and pain on inspiration - Fainting, palpitation, hypotension, cardiac irregularities, and ischemia - Confusion, or delirium - Recurrence of symptoms with aggravation of the pain and secondary infection with necrosis and ulceration within days or weeks - Fatalities are possible	Stingray spine	- Improper behaviour - Animal not seen
- Injuries are from a single bite with no deep cuts to tissue loss, limbs ripped off and death due to the massive bleeding and traumas - Associated with the wounds there may be the following symptoms: • Emotional shock • bruising and rubbings • Massive hemorrhages • Foreign objects in the wounds (teeth) • fractures • Internal organs may be injured • Internal hemorrhages • Hypovolemic shock • Cardiogenic shock • Vasodilatation shock • Septic shock • Death may occur quickly, depending on the hemorrhages and traumas…	Bitten or amputated by big predator	- Improper behavioural - Animal not seen

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Part B - Elements of anatomy and physiology

Note:

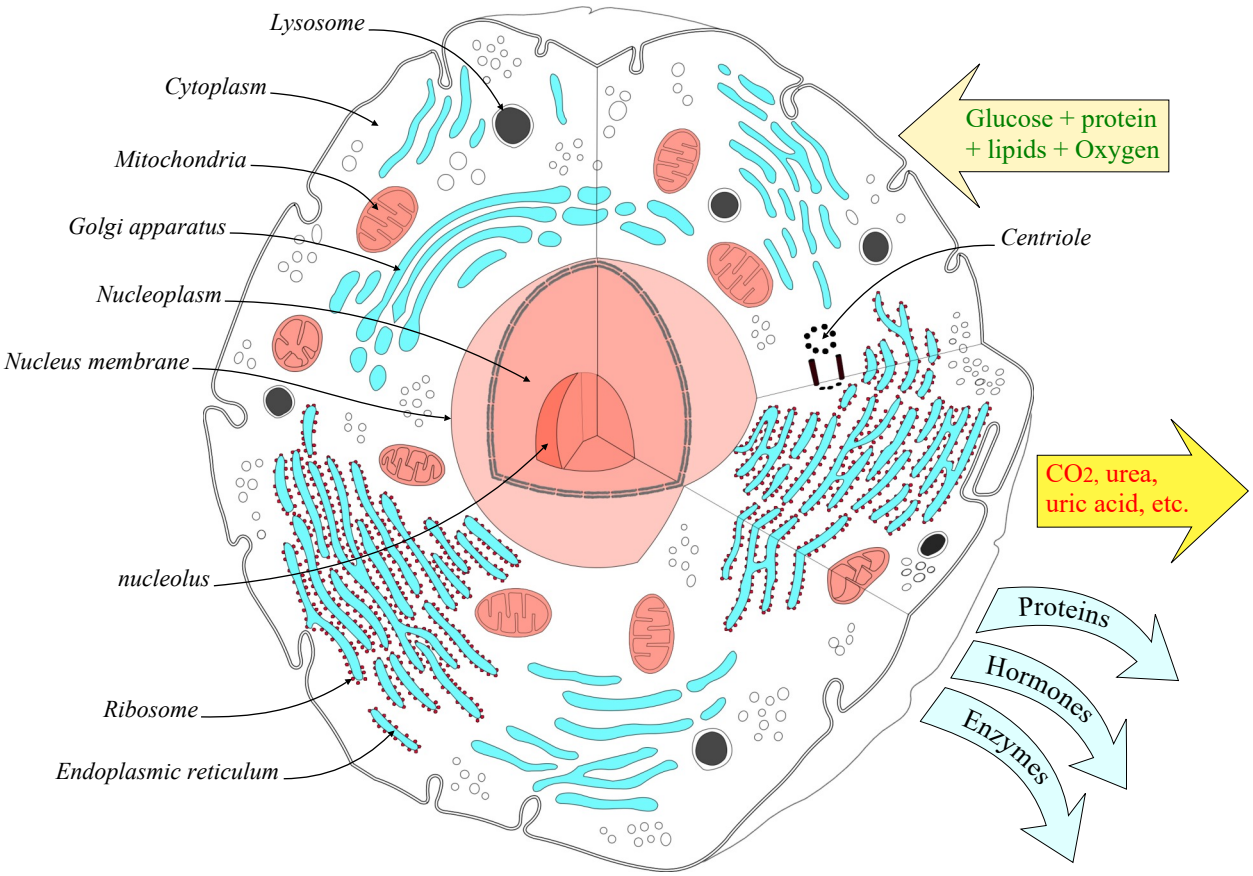
This part gives a quick overview of some of the main systems that compose the human body and may be involved in several types of accidents or diseases.
These systems are all interconnected and work in symbiosis. For example, the digestive system that is fundamental to feed the cells that compose the body is vascularized, and its activity is controlled by the sympathetic nervous system. It is maintained in position and partially protected by the skeletal, and activated by the action of muscles. These organs need oxygen, which the respiratory system provides. They are isolated from the external by the skin, which is also innervated and vascularized. Also, blood cells are generated in the long bones, etc.…
As a result, it isn't easy to give an order of description, and the one used in this chapter corresponds to those commonly found in medical books.
Note that auditory organs are also involved in equilibrium and space reference functions of the body. However, they are not described here, but in the next chapter, with the accidents in which they are involved. It is also the case of organs that have only one function.

These descriptions can be used as complementary references to comprehend the complex processes triggered in an accident or a disease.

Cells

Description

The human body is composed of several hundred different types of cells of various shapes and functions. Despite this great diversity, common characteristics are found.
Cells are formed of an internal substance called cytoplasm and an external membrane. The internal parts of the cell also comprise the nucleus and several organs described below.



- The membrane is a soft, half porous unit of 50 to 100 angstroms (1 angstrom = 1 mm/10000), composed of a layer of lipids between two layers of proteins. It comprises pores of 6 to 8 angstroms. The membrane controls the cell's exchanges with the external that occurs either through the membrane or through the pores, using the principles of osmosis, oncotic pressure, ionic exchanges, and hydrostatic pressures. Note the following:
  - Osmosis is a process of liquids movement through a semi-permeable membrane from a region of lower solute concentration to higher solute concentration to equalize the solute concentrations of the two sides.
  - The oncotic pressure is the osmotic process across the venules and capillaries. This pressure is due to the difference in protein concentration between plasma and interstitial fluid. It counteracts capillary and venule hydrostatic forces in determining net fluid flux across the blood vessels' inner cellular lining (endothelium).
  - Ion-exchange is a chemical reaction between two substances, each composed of electrically charged atoms or molecules. The positively charged ions are called cations, and the negatively charged ions are called anions.
  - Hydrostatic pressure is the pressure exerted by a fluid. As an example, 1 bar equals a column of 10 m of seawater.
- The cytoplasm is a liquid medium containing dissolved substances (proteins, glucose, some lipids, ions), and the following elements:
  - The "Endoplasmic reticulum" a continuous membrane system that forms a series of flattened sacs that serve multiple functions such as the synthesis, folding, modification, and transport of proteins.
  - Ribosome particles are sites of protein synthesis. They can be free particles or be attached to the membranes of the endoplasmic reticulum.
  - The Golgi apparatus or Golgi complex is involved in the processing and packaging of the macromolecules like proteins and lipids that are synthesized by the cell.

- Mitochondria are membrane-bound cell organelles (mitochondrion, singular) that generate most of the chemical energy needed to power the cell's biochemical reactions. Chemical energy produced by the mitochondria is stored in a small molecule called adenosine triphosphate. Mitochondria contain their own small chromosomes. Generally, mitochondria, and therefore mitochondrial DNA, are inherited only from the mother.
- A Lysosome is a membrane-bound cell organelle that contains digestive enzymes that break down excess or worn-out cell parts. They may be used to destroy invading viruses and bacteria.
- Centrioles are two cylindrical, rod-shaped, microtubular structures near the nucleus that are involved in the cell division.

- The nucleus is made up of a liquid medium called “nucleoplasm”, separated from the cytoplasm by the nucleus membrane. One small spherical body called nucleolus is present in the nucleoplasm. Note that sometimes there are several nucleoli (plural of nucleolus). A granulous substance called chromatin is also found in the Nucleoplasm.
  - Nucleoplasm main function is to store “deoxyribonucleic acid (DNA)” and facilitate an isolated environment where controlled transcription and gene regulation is enabled.
  - Deoxyribonucleic acid (DNA) is a molecule carrying genetic instructions for the development, functioning, growth and reproduction of all known organisms and many viruses.
  - Chromatin is a combination of DNA and protein whose primary function is packaging long DNA molecules into more compact, denser structures. That prevents the strands from becoming tangled and reinforces the DNA during cell division.
  - The nucleolus is producing and assembling the ribosomes. A ribosome is a complex molecular assembly that produce proteins from amino acids during the process called protein synthesis or translation, which is the primary function of living cells. Following assembly, ribosomes are transported to the cell cytoplasm, where they serve as protein synthesis sites.

Cell process

Cells behave like small factories: They use fuel and raw materials for given jobs and export or store the products of their activity. They also reject waste and need energy for their activities, which is produced by using fuel and an oxidizer. These activities are regulated, monitored, and directed by their nucleus.

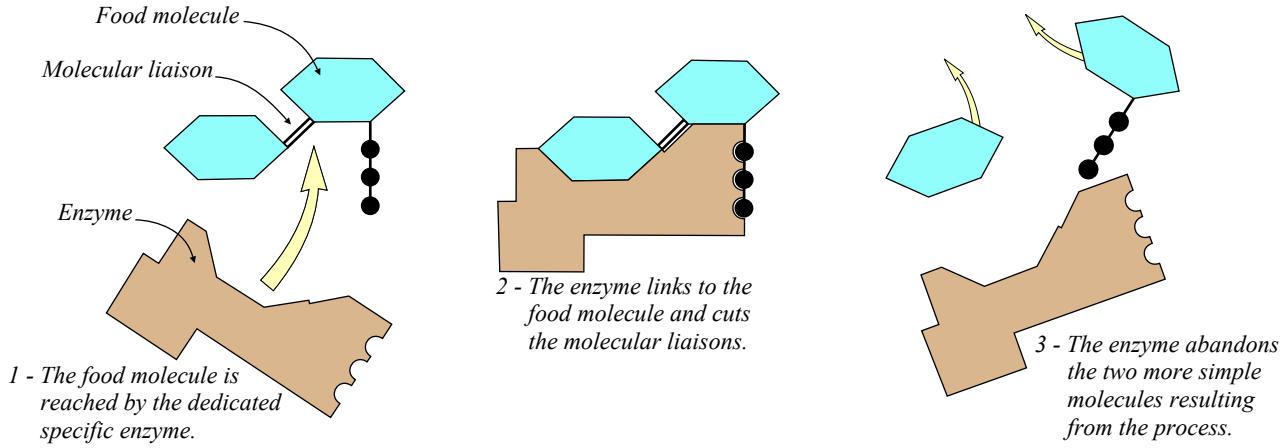
The cell draws the food and the oxidizers it needs from the extracellular environment. These different elements enter the cell in three ways:

- Passive transport or diffusion, without energy consumption:
  The extracellular elements enter the cell through the pores. The ease of penetration depends on the size of the pores, which are 8 angstroms, and the fact that they have a positive electrical charge. As a result, substances with a negative electrical charge or no electrical charge and which size is smaller than 8 angstroms can easily pass through the pores, while substances with a positive electrical charge and a size larger than to 8 angstroms cannot.
- Active transport with cellular work and, therefore, energy consumption.
  Extracellular fluids that are fat-soluble can enter the cell by passing through the membrane, regardless of the size of the pores. It is the case with fatty acids, oxygen, and carbon dioxide. On the opposite, water is stopped by the lipid layer. Note that some non-fat-soluble substances, such as carbohydrates, can take the same path with the help of a lipoprotein molecule, which combines with sugar to constitute a liposoluble compound that can cross the cell membrane.
  Also, there are cases where the cell forces certain substances to circulate at the opposite of osmosis law.
  It is the case for sodium and potassium ions, where sodium concentration is low in the cell, and the potassium concentration high, where it is the opposite outside the cell. Therefore, the cell continually expels sodium from a weakly concentrated medium (the cell) to a highly concentrated medium (the extracellular medium). It is said that this means of transfer consumes energy.
  Note that active transportation mechanisms are still under investigation.
- Cytoplasmic incorporation or “endocytosis”.
  Endocytosis is a mechanism of cellular absorption by which the cell gradually incorporates the particle to be absorbed. By deforming the cell membrane envelops the particle, which is then incorporated into the cytoplasm. The particle is then subjected to the action of diastases or digestive enzymes of the cell.
  Note that the “endocytosis” process is called “pinocytosis” for food molecules and “phagocytosis” for bacteria or biological debris by white blood cells.

Cell digestion consists in separating the complex organic elements drawn into the cell to simple organic elements. Diastases or enzymes are biochemical protein substances, which have the property of making possible certain specific biochemical reactions, allowing to break-down complex molecules into simpler elements.
There are nearly 2000 different kinds of enzymes that only act on a certain type of biochemical reaction. They are not modified by the chemical reaction which they activate and cannot modify it.

In addition, the enzymes produced by the cell are accumulated and packaged in the “Lysosomes” to separate them from other cellular elements (see in the previous point). The digestion, therefore, occurs by attaching one or more Lysosomes to the “endocytosis vacuole”. The lysosomes release their enzymes inside the vacuole, which allows for the degradation of complex food molecules into simple organic elements. Unusable residues remain in the vacuole, which evacuates them outside the cell.

Note that the digestion described above occurs at the level of specialized tissues (digestive tract) and within any cell. It always involves enzymes that are synthesized by the cell (role of the plasma reticulum), and then finished and packaged in the “Golgi apparatus”, to be finally stored in the lysosomes. They can either be excreted outside the cell (glandular cells) or stored in lysosomes inside the cell to digest the food contents of endocytosis vacuoles.



Cells need energy for their various activities. The interaction of fuel and oxidizer provides this energy:

- The oxidizer is oxygen, for the majority of the reactions which occur aerobically.
- Fuel is food elements or nutrients: glucose (carbohydrates), fatty acids (lipids), and amino acids (proteins).

Numerous complex biochemical reactions take place to release energy by oxidation of glucose and fatty acids with carbon dioxide rejection. At the end of all these reactions, the released energy is fixed on a transporter called “adenosine triphosphate (ATP)”, containing three phosphate radicals. The last two phosphate radicals are linked to the rest of the molecule by energetic bonds requiring a large amount of energy to build up and releasing a large amount of energy when they break. This is how ATP transforms into “adenosine diphosphate (ADP)” when releasing energy, and vice versa, depending on the reaction: ATP = ADP + P ions + energy.

The complex reactions resulting from the breakdown of nutrients, in the presence of oxygen and enzymes, and the release of energy occurs in the “mitochondria” *(see the drawing in the previous point)*. The energy released in this way is used in the mitochondria to convert ADP into ATP. The mitochondria is, therefore, the place of cellular energy reactions and that of storage of the energy produced in the form of ATP.

The ATP in the mitochondria is used by the cell according to its needs as follows:

- Cellular absorption and excretion.
- Mechanical labour, which is most often a contraction. It may be the contraction of muscle fibrils resulting in muscle contraction. It may be the localized contraction of certain portions of the cytoplasm, such as the white cells of the blood. It may be the asymmetric contraction of the filaments that constitute the vibratile cilia with are provided to the borders of certain cells and whose undulation can be a factor of movement and advancement such as flagellum of the sperm.
- The synthesis of new products. Synthesis means the manufacture of a more or less complex molecule by assembling simple constituent elements (from the Greek synthesis = put together). Plants synthesize the molecules they need using the energy of sunlight: this is photosynthesis. Animal cells synthesize elements necessary for their life using the energy provided by ATP: this is biosynthesis.
  - The biosynthesis of carbohydrates or sugars takes place from glucose molecules not used for energy. The attachment of several hundred glucose molecules makes it possible to synthesize glycogen, which is stored in the liver.
    - The biosynthesis of fats, also called lipid synthesis, is complex. It starts with “acetyl-coenzyme A”, which comes from the breakdown of glucose. Multiple reactions transform this chemical body into fatty acids, which can then be attached to glycerol to form lipids.
    - It is important to note that sugar is the starting point for the biosynthesis of fats. In the case of overweight, an effective weight loss diet should reduce all forms of sugar.
    - Protein biosynthesis is complex since a protein is a three-dimensional structure comprising an extremely complicated assembly called an amino acid. There are twenty different kinds of amino acids, which assembly one behind the other in a particular number and order constitutes the characteristic sequence of a given protein (primary structure). Also, the assembly of these amino acids in space (secondary structure), and then the more or less complicated twisting of the whole on itself (tertiary structure) defines the protein.
      Ribonucleic acid (RNA) is a non-protein matrix with specific sequences that determine the code that corresponds to each particular amino acid, so that only the sequence of amino acid programmed can be

used. It controls all cell life because it controls the way to protein synthesis. Also, enzymes, which control all cell metabolism, are protein substances synthesized by the cell. The key to protein enzymatic synthesis is the key to all cellular life, in all its forms, at all times.
RNA is found in the nucleus and is the replica of another basic nucleic acid called “Deoxyribonucleic acid (DNA)”, which contains all the protein manufacturing codes.
DNA transmits the key to making a particular protein to the RNA that exits the nucleus through orifices in the nucleus membrane to attach to the ribosomes that are on the endoplasmic reticulum. This transmission is performed by the “transfer RNA”, which is a short nucleotide RNA chain.

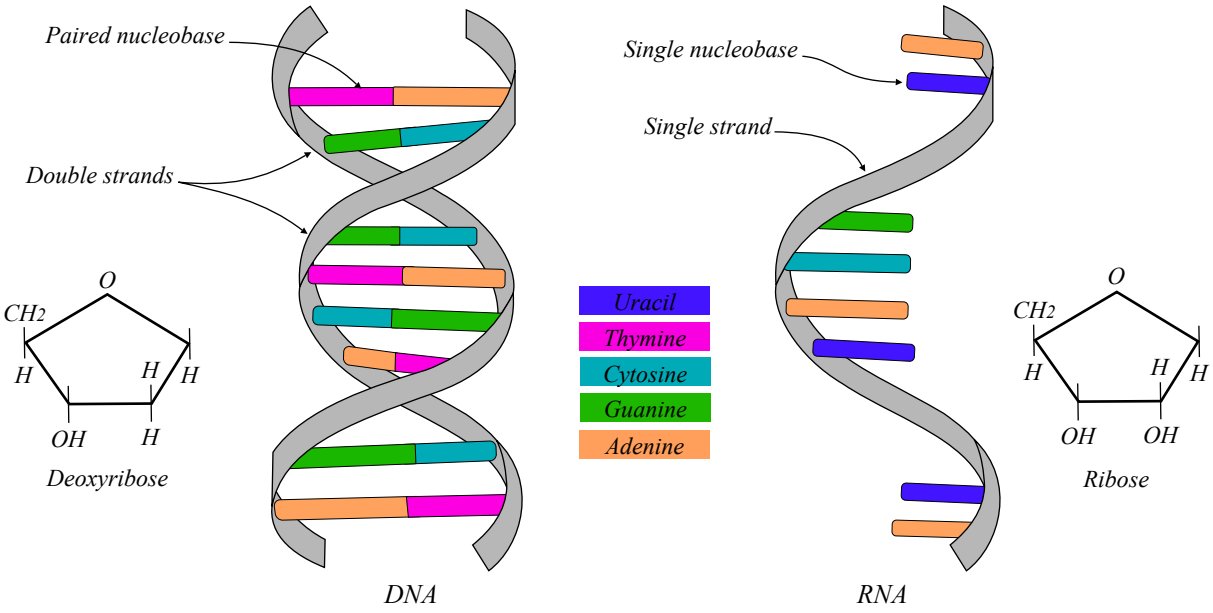
As previously indicated previously, the nucleus contains nucleic acids called “deoxyribonucleic acid or DNA” and “ribonucleic acid or RNA”.
A nucleic acid is made up of the succession of basic structures called nucleotides, which are made up of the combination of three molecules:

- A phosphoric acid molecule.
- A sugar, ribose, or deoxyribose, hence the name given to nucleic acid.
- A nitrogen base.

Nucleotides attach by the alternation of phosphorus and sugar molecules, forming a chain, a ribbon, on which the nitrogenous bases are connected perpendicularly.
The nitrogenous bases play the fundamental role, determined by their succession the code of the nucleic acid.
DNA is the central library where the memory of cells is stored. It is responsible for hereditary transmission and directs the synthesis of proteins. More than a library, it is a real computer, regulating the cellular life and the transmission of that life.

The differences between DNA and RNA can be summarized as follows:

- DNA contains the carbon sugar molecule called “deoxyribose”, while RNA contains the carbon sugar molecule called “ribose”. The difference between ribose and deoxyribose is that ribose has one more -OH group than deoxyribose,
- DNA is a double-stranded molecule, while RNA is a single-stranded one.
- DNA is stable under alkaline conditions, while RNA is not.
- DNA is responsible for storing and transferring genetic information to make other cells and new organisms, while RNA directly codes for amino acids and acts as a messenger between DNA and the ribosomes to transmit genetic information and make proteins.
- DNA uses the bases thymine, adenine, cytosine, and guanine; RNA uses uracil, adenine, cytosine, and guanine.

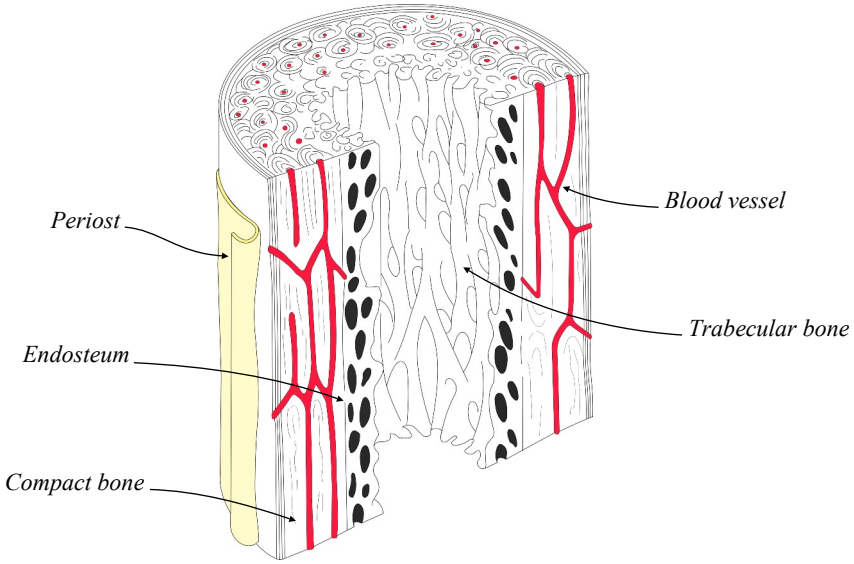


Skeletal system

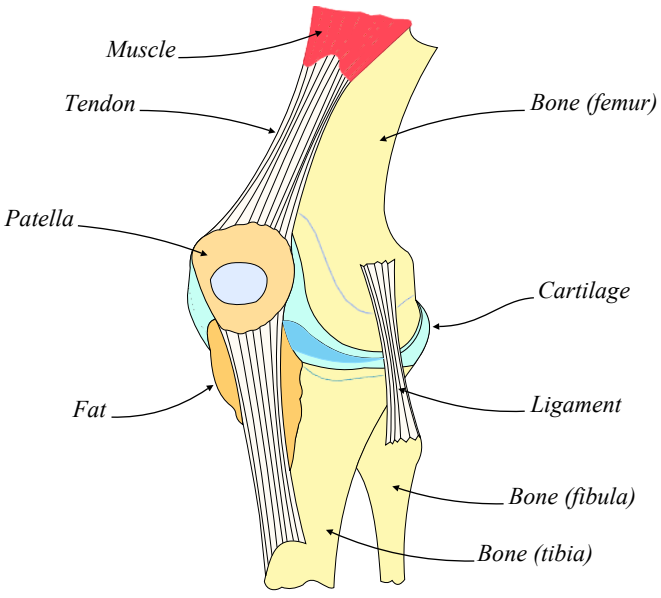
Overall description

The human skeletal system provides support and protection of the body. In addition, it allows for movements and produces blood cells. It is made of bones, cartilage, tendons, and ligaments of the body.

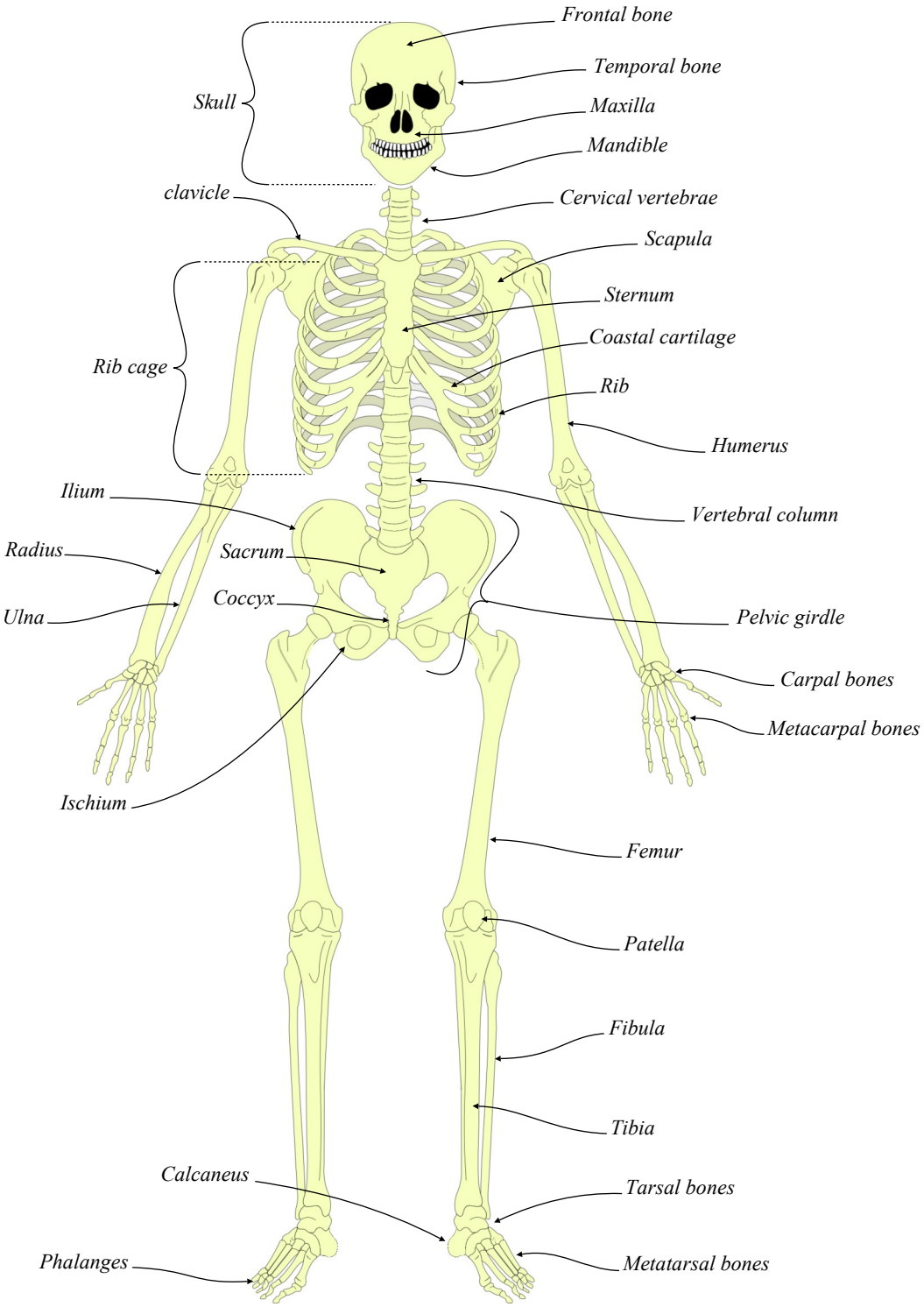
- Bones are rigid tissues consisting of cells embedded in an abundant hard intercellular material. The two principal components of this material are collagen and calcium phosphate.



  - Trabecular bone, also called spongy bone, is a porous part of bones enclosing numerous large spaces with a spongy appearance. The bone matrix is organized into a three-dimensional latticework of bony processes, called trabeculae, which spaces between are often filled with marrow and blood vessels.
  - The “endosteum” is the layer of vascular connective tissue lining the medullar cavities of bones.
  - Compact bone, also called cortical bone, is the denser material of the skeleton that form the outer shell of most bones in the body.
  - The “periost” is the thick fibrous two-layered membrane covering the surface of bones.
- Cartilage is an elastic smooth tissue, that covers and protects the ends of long bones at the joints and nerves. It is also a structural component of the rib cage, the ear, the nose, the bronchial tubes, the inter-vertebral discs, etc. Note that sharks have their skeletal exclusively made of cartilage.
- Tendons are tissues that attach muscles to bones and transmits them the mechanical force of muscle contractions. They are made of connective tissues firmly connected to the muscle fibres at one end and the bone at their other end.
- Ligaments are bands of tough elastic tissue that connect bones, give joints support, and limit their movement. They are present around knees, ankles, elbows, shoulders, and other joints.



An adult's skeleton contains 206 bones, which 6 of them are those of the ear. It represent approximately 20% of the body weight. Males have a higher bone mass than females, which skeletons are provided with a broader pelvis to accommodate pregnancy and childbirth. Also, children's skeletons have more bones because some of them fuse with age.



The bones that constitute the skeleton are classified according to their shapes:

- Long bones are those where the length predominate on width and thickness (example: Femur).
- Short bones are those where the 3 dimensions are nearly equal (example: Carpal bones).
- Flat bones are those with thickness inferior to the other dimensions (example: frontal bone of the skull).
- Irregular bones have complex shapes that cannot be classified into the categories above (example: vertebras).
- Sesamoid bones are embedded within a tendon or a muscle (example: Patella).

The skeleton is divided into two 2 groups: The axial skeleton, and the appendicular skeleton.

1. The axial skeleton forms the central axis of the skeletal system and provide posture, balance, and stability. It is composed of :
    - The skull, which includes the bones of the cranium, face, and ears (auditory ossicles).
    - The hyoid bone, which is the U-shaped bone or complex of bones located in the neck between the chin and larynx.

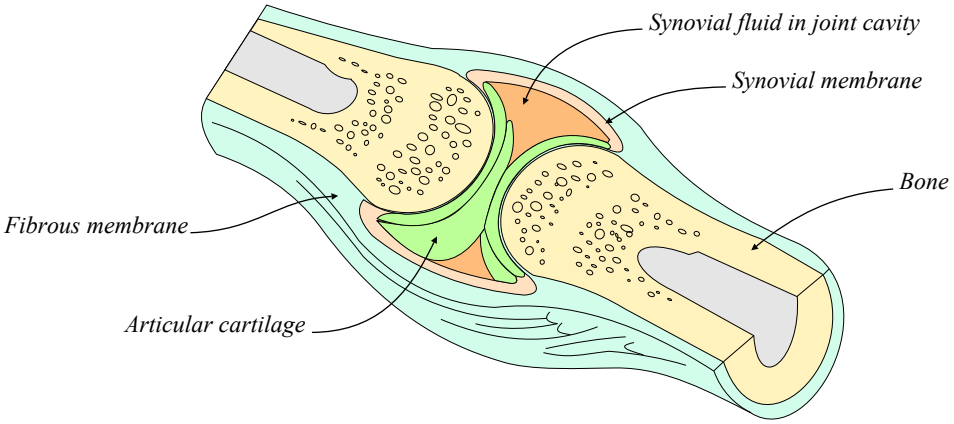
- The vertebral column, which is composed of the vertebras.
- The thoracic cage, which is composed of the ribs and the sternum (breastbone).

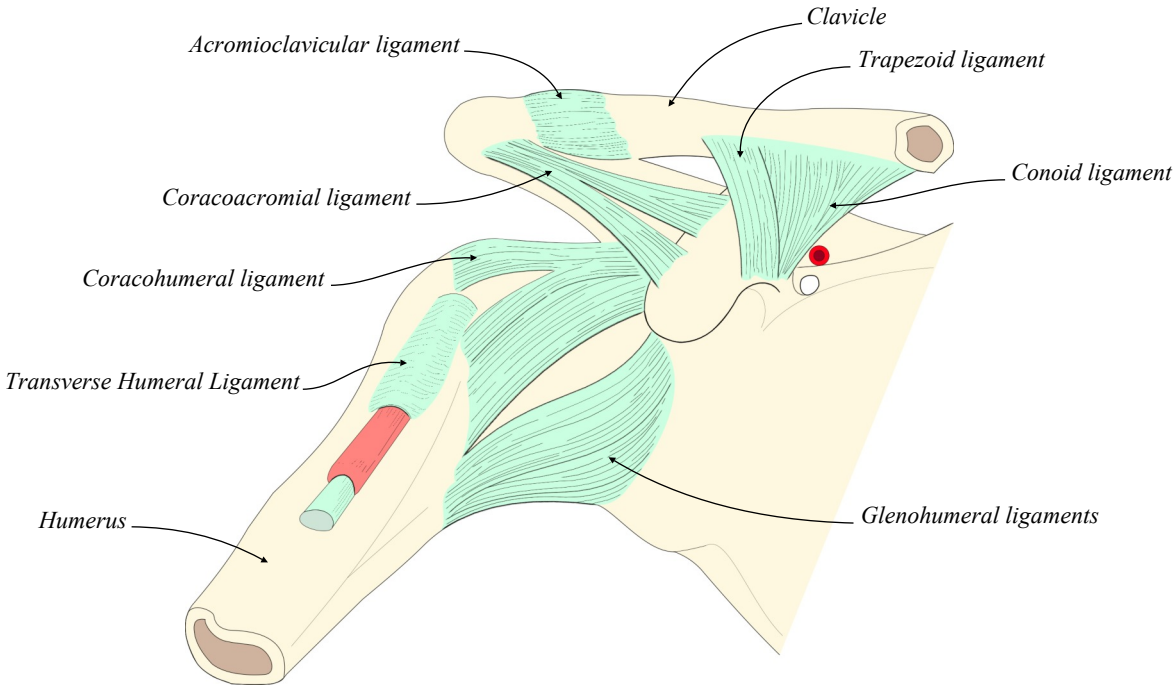
1. The appendicular skeleton is composed of body limbs and structures that attach limbs to the axial skeleton. the primary function of the appendicular skeleton is for bodily movement, it also provides protection for organs of the digestive system, excretory system, and reproductive system. It is composed of:
   - The pectoral girdle, which is composed of the clavicle and scapula.
   - The upper limbs, which are the clavicle, scapula, humerus, radius, ulna and hand bones.
   - The pelvic girdle, which is made of three fused bones: the “ischium”, the “ilium”, and the “pubis”.
   - The lower limbs, which are the bones of the legs and feet.

Articulations

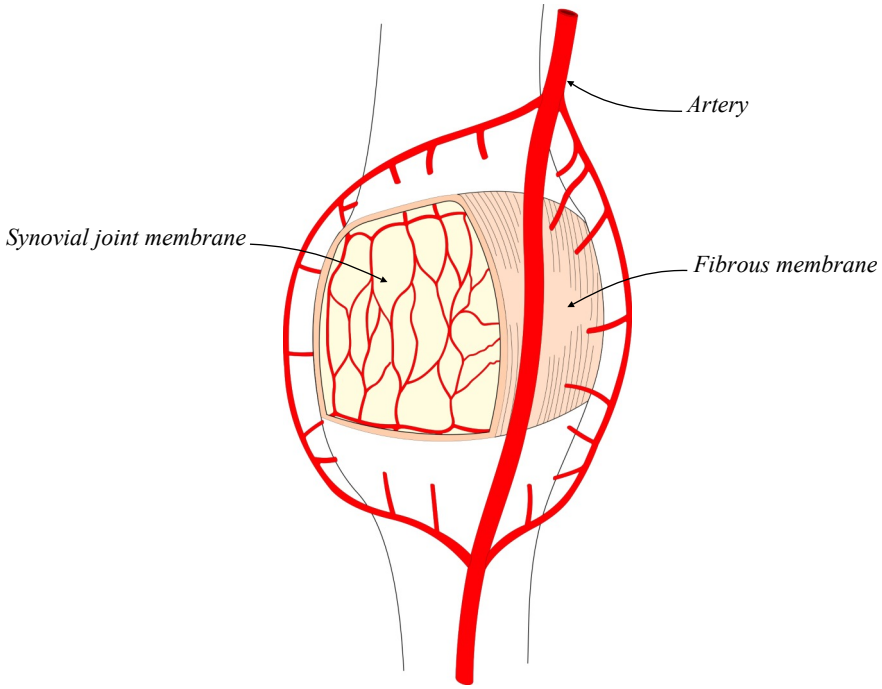
An articulation, or joint, is where two bones come together. They are classified into immovable, slightly movable, and freely movable joints.

- Immovable joints, also called synarthroses, include skull sutures, the articulations between the teeth and the mandible, and the joint found between the first pair of ribs and the sternum. These bones come in very close contact and are separated only by a thin layer of fibrous connective tissue.
- Slightly movable joints are also called amphiarthroses (singular form is amphiarthrosis). This type of joint is made of bones connected by hyaline cartilage or fibrocartilage. It is that case of ribs, which are connected to the sternum , the symphysis pubis, and the joints between the vertebrae and the intervertebral disks.
- Freely movable joints are also called diarthroses (singular form is diarthrosis). They have the ends of opposing bones covered with hyaline cartilage, and they are separated by a space called the joint cavity. The components of the joints are enclosed in a dense fibrous membrane. The outer layer of the capsule consists of the ligaments that hold the bones together *(see the drawing of the shoulder below)*. The inner layer is the synovial membrane that secretes synovial fluid into the joint cavity for lubrication. Because all of these joints have a synovial membrane, they are sometimes called synovial joints.





The vascularization of freely movable joints is very rich and highly organized: Some tissues are highly vascular with a high density of vessels, whilst other tissues are avascular. Vascularization comes from arterial and venous periarticular circles, with some arteries entering the bone a little bit outside the capsule and others being part of the ligaments and the capsule.



The joint capsule and ligaments of freely movable joints are also richly innervated:

- Fibrous ligaments have a rich nerve plexus and various specialized and unspecialized nerve endings, most of which are somatic in origin.
- The synovial membrane contains a more delicate nerve network and various nerve endings, most of which are autonomic in origin. But a substantial number of somatic nerves enter the synovial membrane, some of which terminate in nerve loops, globular endings, or simple unspecialized endings.

The joints' nerves also supply the motor muscles connected to the joint and the skin covering the insertion of these muscles. These nerves participate in the reflex regulation of movements and postures.

Joints are subjected to varied stresses, which are essentially pulling, twisting, or pressing:

- The pressures are significant at the level of the spine and the lower limbs. Poor pressure distribution leads to wear and tear of the cartilage and then to bone condensation.
- Tractions tend to move the joint surfaces away. The capsule, ligaments, periarticular muscles, and intra-articular pressure, which is slightly negative, oppose to this dislocation.

Note that the fact that freely movable joints are highly vascularized and innervated may impact them in an inappropriate decompression process. Also, professional or sports microtraumas can lead to lesions responsible for osteoarthritis.

Blood making

Almost 1 percent of the body’s red cells are generated each day. The rate of blood cell formation varies depending on the individual. A typical production might average 200 billion red cells, 10 billion white cells, and 400 billion platelets per day for an adult.

Blood cells are made in the bone marrow that produces:

- All of the red blood cells.
- 60 - 70 percent of the white cells.
- All of the platelets, which are formed from bits of the cytoplasm of the giant cells (megakaryocytes) of the bone marrow

The lymphatic tissues, particularly the thymus, the spleen, and the lymph nodes, produce the lymphocytes (comprising 20 - 30 percent of the white cells).

The reticuloendothelial tissues of the spleen, liver, lymph nodes, and other organs produce the monocytes (4 - 8 percent of the white cells).

Muscular system

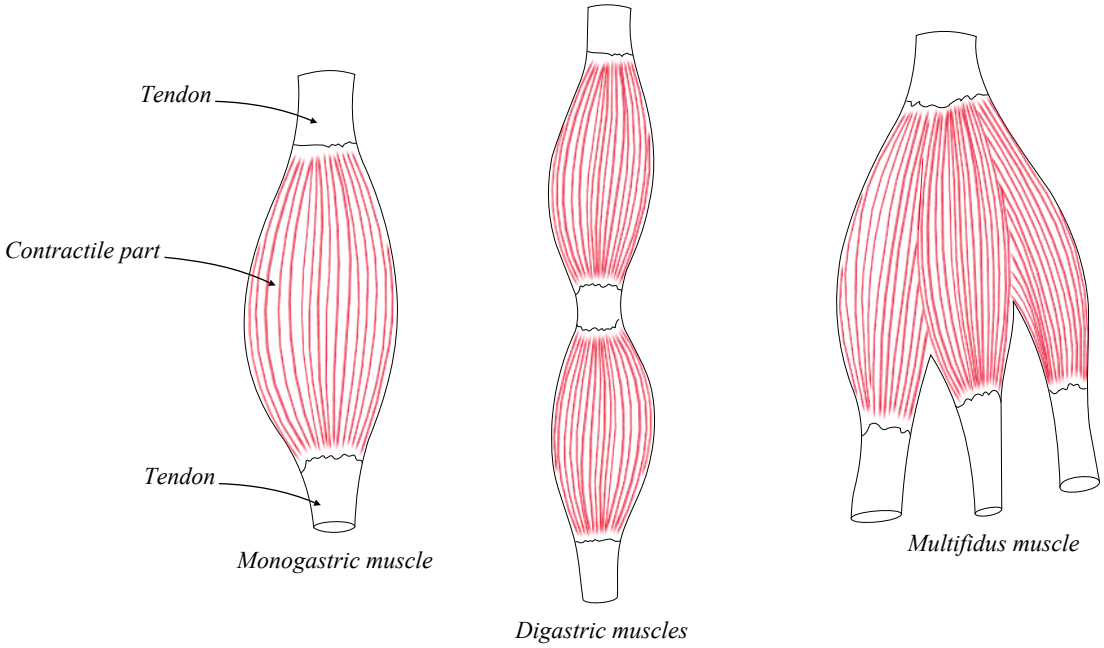
The muscles allow for moving the body, and create work and heat. They can be classified as follows:

- Skeletal muscles are streaked muscles that can contract under the influence of the will. They act on the skeletal system.
- Smooth muscles are unstreaked muscles that escape the influence of the will. They are localized in the viscera, vessels, and skin.
- Mixed muscles are striated muscles that are independent of the influence of the will. As an example, the muscles of the heart.

Skeletal muscles

The skeletal muscles represent approximately 40% of the body's weight.
Note that passed 25 years, the muscle mass decreases progressively and continuously. This reduction is a function of physical activity.
Skeletal muscles are generally composed of a thick body that is contractile and of two narrower white and resistant extremities called tendons. Depending on their shape, they can be:

- Monogastric muscles, which are shaped like a belly.
- Polygastric muscles, having more than two bellies. These can be successive (digastric muscles) or juxtaposed (multifidus muscles).
- Long muscles have their length clearly greater than their thickness and width. They are predominant in limbs.
- Flat muscle have predominant length and width.
- Short muscles are reduced in all their dimensions.
- Annular or circular muscles surround orifices. Example: the sphincter muscles.
- Square, pyramidal muscles are similar to the homonymous geometric figure.



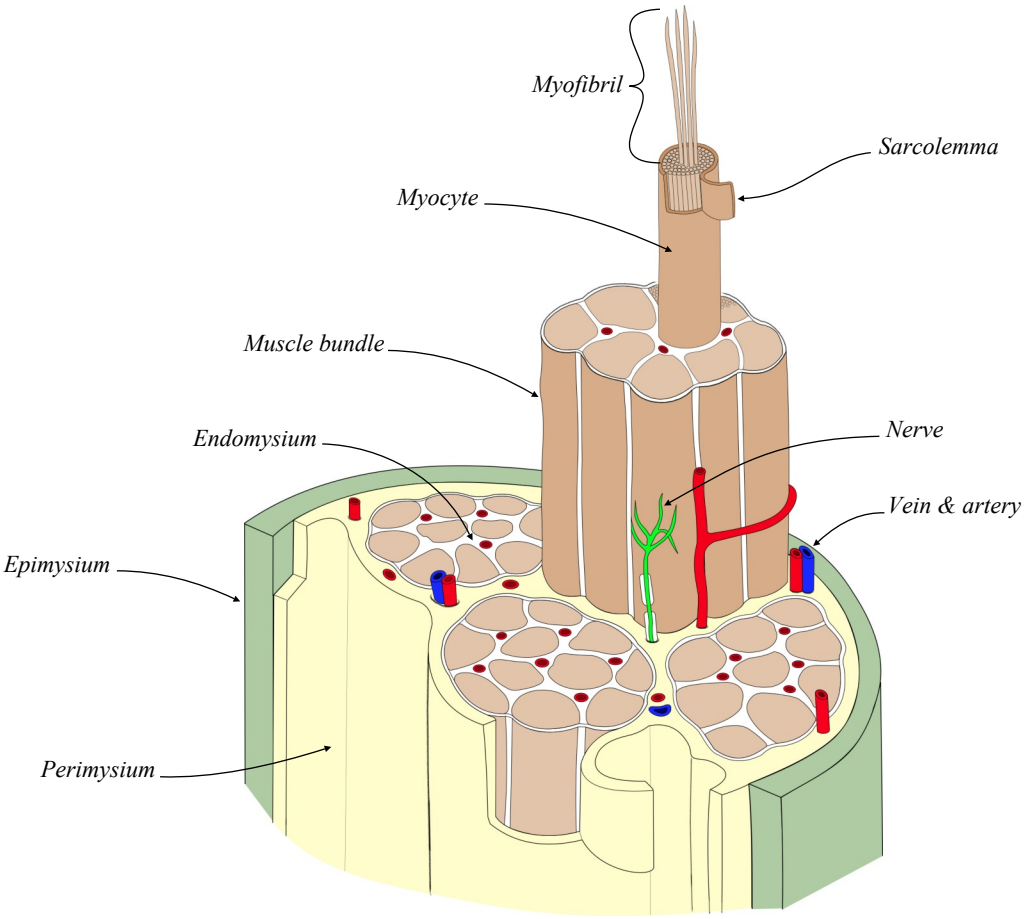
Muscles attach to various surfaces such as bones, cartilages, dermis (skin muscles), mucous membranes (tongue), fascias, fibrous arches. This attachment can be done either by fleshy fibres, tendons, or broad fibrous laminas called aponeurosis.

The morphological and mechanical unit of the muscle is the “muscle fibre”, which is made of parallel myofibrils, striated transversely and enveloped by a connective membrane called “sarcolemma”. This envelope contains several peripheral nuclei, and the “sarcoplasm” containing a red pigment called “myoglobin”.

Muscle fibres are separated from each other by delicate, vascularized connective tissues called “endomysium”. Also, several fibres are grouped in bundles surrounded by a conjunctive lamina called “perimysium”, and the entire muscle is covered with a dense fibrous tissue called “epimysium”.

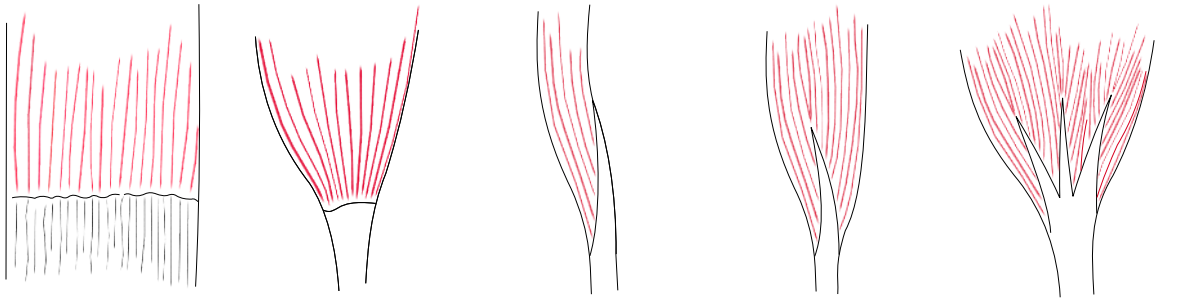
Tendons and aponeurosis are made up of bulky collagenous fibres organized in the same direction. There is no continuity between the collagenous fibres and the myofibrils because the sarcolemma separates them.
The fibers group together in bundles separated by longitudinal planes of loose connective tissue containing the vessels.

This connective tissue is called “endotendineum” around primary bundles, “peritendon” around secondary bundles, and “epitendineum” around the tendon.
Note that tendons attach to the bone's periost and penetrate the bone tissue (perforating fibers), which explains bone tearing in some lesions.
In the elderly, the tendon may ossify over a certain length. The tendon sometimes presents fibrous expansions constituting secondary attachments.



Muscle bundles can:

- Have the same parallel direction as the tendon bundles (flat muscles)
- Converge towards the end of the tendons (spindled muscles)
- Attach to the lateral side of a tendon (unipennate muscles)
- Attach to both sides of a tendon (bipennate muscles)
- Attach to the sides of the subdivisions of a tendon (multipennate muscles)



Elements annex of the muscle:

- Muscle fascia is a tissue that surrounds muscles. It is located either under the skin (superficial fascia) or in contact with the muscles (deep fascia). Note that tearing of the fascia promotes muscle herniation through the fascia breach.
- The inter-muscular septum is a connective septum separating muscle groups.
- Interosseous membranes are the septa stretched between bones.
- The synovial bursa is a membranous bag filled with synovia that promotes the sliding of a muscle against a bone or another muscle.
- Vinculum and mesotendon are connective formations that are independent or in continuity with each other, and in this case, confused. The vinculum is a formation uniting the tendon with the underlying bone. The

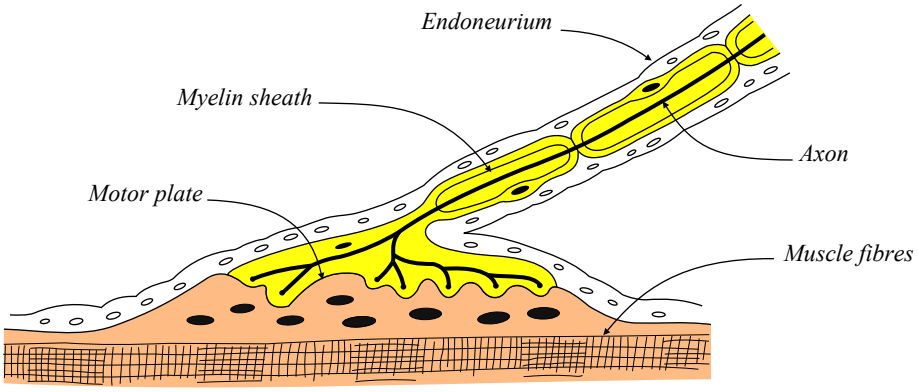
mesotendon unites the tendon with the synovial sheath and contains vessels and nerves for the tendon.

- The muscular trochlea is a fibrous or fibro-cartilaginous ring serving as a reflection pulley for a tendon.
- Tendons retinaculum are large fibrous lamina holding the tendons. They are surrounded by their synovial sheaths that slide under a retinaculum, stretched between two bones.

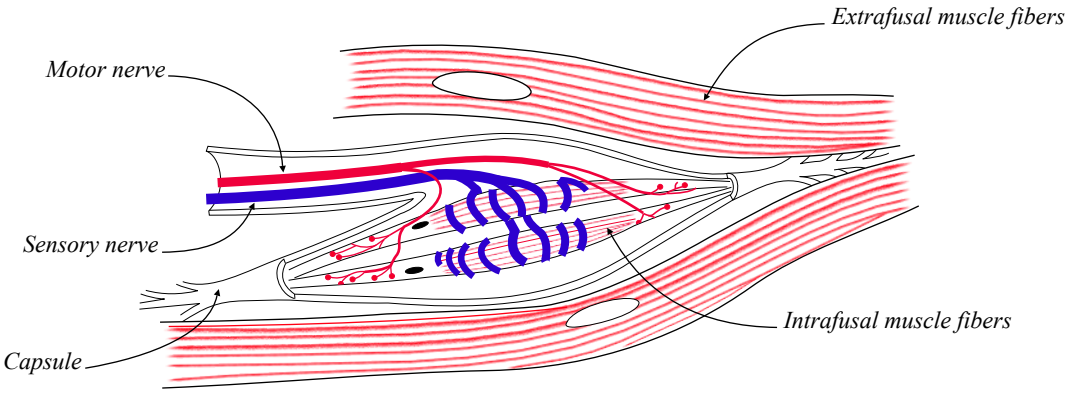
Numerous arteries vascularize the skeletal muscles. They come from the neighboring arterial trunks, and muscles with the same function are often vascularized by arteries originating from the same trunk.
The main artery entry point is often constant. This penetration can be done at the same point as the nerve (polarized muscle) or at a different point (non-polarized muscle).
Arteries are divided into multiple capillary branches those whose direction is parallel to the muscle fibers are from these branches. Note that the veins are provided with numerous valves. Also, the belly part of the muscle is more richly vascularized than the tendon.
Note that a hypovascularized muscle gets tired very quickly and shows disorders such as cramps.

Many muscles have multi-segment innervation, which receives neuro fibers from several spinal nerves. Muscles with a similar function are innervated by the same nerve.

- Motor innervation is provided by myelinated fibers. Each neuro fibres branches at its distal end, and each branch terminates at a muscle fibres through the motor plate *(see the drawing below)*. The number of muscle fibers per motor unit is inversely proportional to the precision of movements.



- Sensory innervation is provided by myelinated fibers whose receptors are the neuromuscular spindles. It informs the nerve centres on the degree of tension and stretching of the muscle.
- Vasomotor innervation is provided by sympathetic unmyelinated fibers intended for muscle vessels.
- The tendon corpuscles are located at the musculotendinous junction. They are the starting point for provoked tendon reflexes.
- Note that the destruction of the nerve makes the muscle atrophic and flaccid.

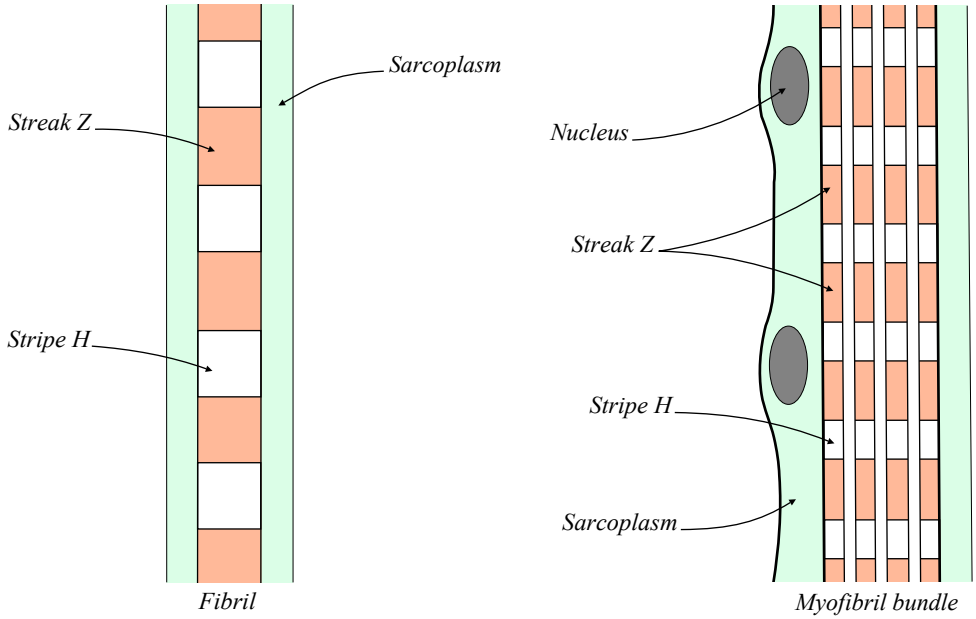


A muscle fibre is a modified cell comprising a cytoplasm, the sarcoplasm (from the Greek sarkos = flesh, muscle), a nucleus, and muscle fibrils or myofibrils grouped in parallel bundles, all encased in a cell membrane or sarcolemma.
Microscopic examination of the myofibril bundles shows that they are made of a type of protein micro-fibers called “myosin”, and another type which is thinner and is called “actin”. So, the “actin” fibres can slide between the “myosin” fibres.
At rest, the spaces between the stripes of actin microfibres correspond to the stripes of myosin fibers, and the spaces between the stripes of myosin microfibres correspond to the stripes of actin microfibres. That explains the tonal stripes that can be observed (light stripe H and streak Z)
The passive elasticity and the muscle's active work are related to the possibility of sliding, and therefore of moving away or coming together of the actin fibers within the corridors delimited by the myosin fibers, similarly a cylinder slides in a piston.

- Muscle elasticity is due to the separation of the ends of the actin fibers, which has the effect of enlarging the white stripe H and separating the two concomitant Z streaks from each other. This results in an elongation of the

myofibrils, therefore of the bundle of myofibrils, therefore of the muscle fibre, and therefore of the muscle itself, since all these structures, from the simplest to the most composed, work together.

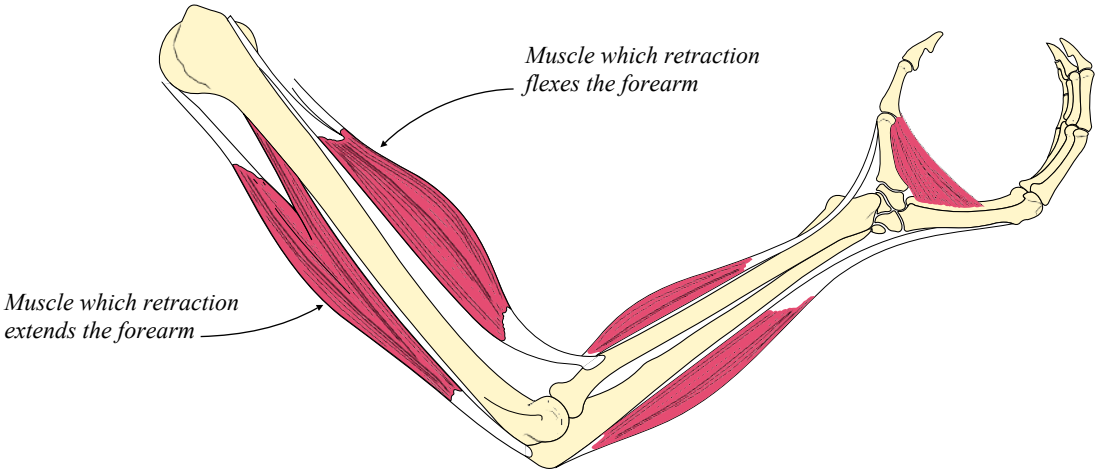
- Muscular work is caused by bringing together the ends of the actin fibers, which may even overlap. That reduces and then eliminates the white stripe H, which is finally replaced by a dark stripe (overlap). There is an approach of the associated Z streaks, which can result sin:
  - Muscle tension, if the ends of the muscles do not bring their bony insertions.
  - Contraction if the ends of the muscle bring their bony insertions together. This contraction results in a shortening of the muscle and a movement.



Since most of the actions of the body require opposite movements (for example, flexion and extension of the forearm), each articulated segment must be provided with two muscles to ensure the movements. The two muscles or the two groups of muscles must work in opposition to obtain opposite movements *(see the scheme below)*:

- Forearm flexion is achieved by flexing muscles located on the anterior surface of the elbow joint.
- The extension of the forearm onto the upper arm is achieved by the extensor muscles located on the elbow's posterior surface.

Therefore, it is the location of the muscles and especially the position of their bony insertions that determine the result of their action, and it is the coordination of antagonistic muscles that allows for precise and effective movements.



Smooth muscles

Smooth muscle fibers are made up of spindle-shaped cells, non-striated, with a single nucleus, that are smaller than those of striated muscle fibers. They are grouped by a conjunctive-elastic web, forming a more or less thin, elastic, and contractile layer. Note that their contraction is slower than skeletal muscles.
These muscles constitute the walls of almost all the hollow organs of the body (Vesicle, bladder, uterus, digestive tract, etc.), and pipes (vessels, bronchi, ureter, glandular ducts, etc.).
These muscles also constitute the smooth sphincters controlling the emptying of many organs (bladder, rectum).

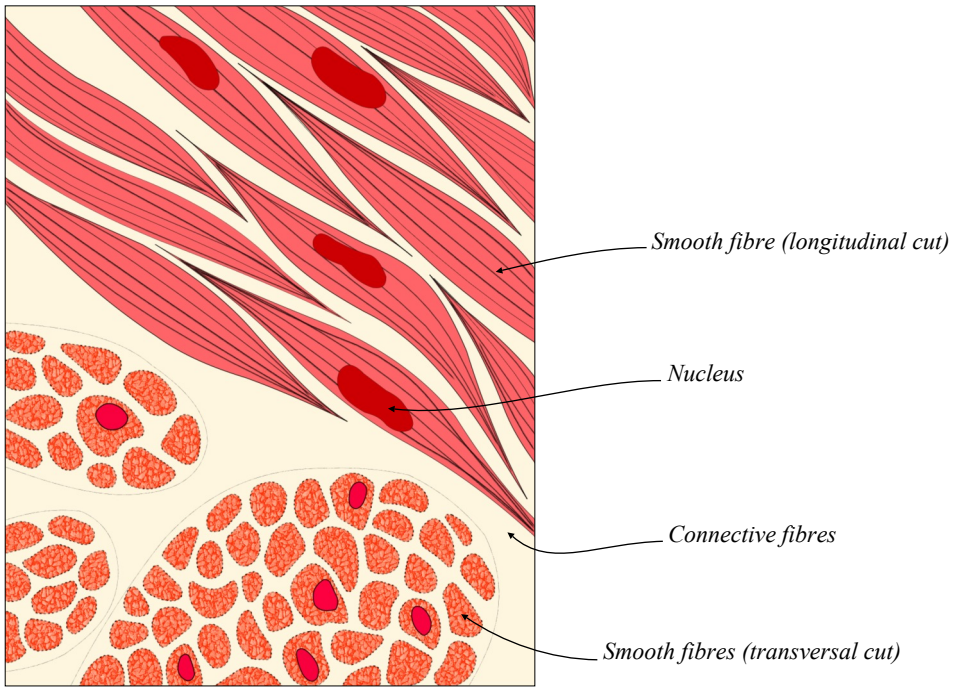
Unlike striated fibers, smooth fibers seldom constitute muscle spindles with separate insertion points, except for the small muscles attached to the hairs (muscles that make the hairs crinkle).
More numerous than skeletal muscles, smooth muscles are classified according to their shape:

- Flat smooth muscles. For example, the dartos muscle.
- Annular smooth muscles. For example, the constrictor muscle of the iris.
- Tubular smooth muscles. For example, the muscular tunic of the intestine.
- Vascular smooth muscles.

Smooth muscle tissues are provided with a rudimentary intrinsic nervous system made up of plexuses of nerve fibers and nerve ganglia, all belonging to the sympathetic system. It provides the smooth muscle with a certain autonomy, such as a muscle tone independent of innervation coming from the outside and the possibility of contracting in the absence of external nerve stimulation. These automatic contractions account for certain peristalsis *(radially symmetrical contractions and relaxations of muscles that propagate in a wave down a tube such as the intestine)* made possible by the intrinsic nervous system's coordinating action. Also, there is no degeneration when the external nerves are severed.
However, while it can retain some of its properties in the absence of stimulation, the smooth muscle tissue is not really physiologically isolated and left on its own as its activity is controlled by the vegetative nervous system, which can accelerate, slow down, facilitate, or inhibit its activity.
This vegetative system is made up of two antagonistic systems, more described in the chapter “Nervous system”, called “sympathetic system” and “parasympathetic system”.
Every smooth muscle tissue receives fibres from both systems. However, their terminations are different from those of striated fibers because there is no motor plate. Therefore, there is no depolarizing wave passing from the nerve to the muscle via a chemical mediator. Instead, the chemical mediator is released at the end of the nerve fibre and diffuses into the smooth tissue and changes its state (relaxation or contraction). This diffusion, together with the lack of myofibrils, explains the slowness of the smooth muscle contraction and the length of its recovery time.
The mediators consist of:

- Adrenaline for the terminal fibers of the sympathetic system, which prepares the body for stressful or emergencies.
- Acetylcholine for the terminal fibers of the parasympathetic system, which regulates body processes during ordinary situations.

Smooth muscles are poorly vascularized. Some are even avascular. and feed by imbibition.



Skin

Overall description

The skin is the membranous part of the integumentary system covering most of the surface of the body.

- The estimate of the body surface areas of an adult is based on the “rule of nines” where":
  - An upper limb represents 9%
  - the head and neck represent 9%
  - A lower limb is equal to 18%
  - Each half-torso represents 18%
  - The perineum *(region between the pubic arch and the coccyx)* is equal to 1%
- Its thickness is from 1 to 2 mm. The thickest parts are the plantar and dorsal regions. Note that this thickness diminishes with age. The skin is also thinner in women.
- Its weight is between approximately 6 kg - 9 kg for an adult, depending on the size and weight of the subject.
- Its temperature is between 32 C° to 36 C°, depending on the region, the toes being the coldest regions.
- Its elasticity that decreases with age is important and allows surgical plasties.
- Its electrical charge is negative (on its surface).
- Its colouring varies according to the races. Its pigmentation is not uniform, with very pigmented regions such as the areole of the breasts and less pigmented areas such as palmar and plantar regions.

The surface of the skin presents pores from which emerge hairs and pores where cutaneous glands open. There are visible transverse furrows at the flexion folds level and crisscrossing cracks visible or discreet at the level of palmar and plantar regions (used as elements of identification of individuals).

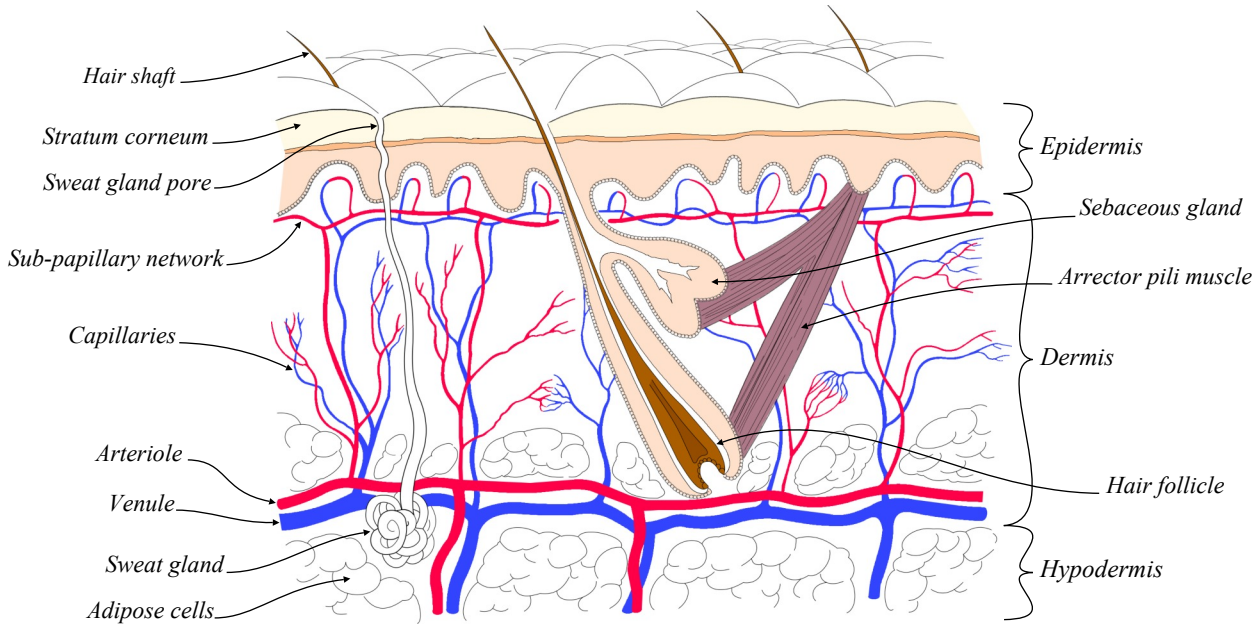
- The epidermis is a multilayered stratified epithelium of 0.04-0.4 mm thickness. It contains the melanocytes responsible for the skin staining.
- The dermis is underlying the epidermis and sits on the subcutaneous web. It contains the skin glands, terminal nerve corpuscles, capillaries, muscles, and hair follicles. Its thickness is from 0.5 to 2.5 mm, and includes two layers:

a) The papillary stratum is made up of delicate connective tissue and presents numerous papillae towards the basal stratum, to which it is closely united.

b) The reticular stratum is thicker and consists of a dense connective tissue containing many collagen and elastic fibers.

The skin is very vascularized, with numerous blood vessels located in the dermis. However, the epidermis is devoid of vessels.

- Arteries form a network in the derma from which vertical branches form the sub-papillary arterial network.
- Veins have the same arrangement as arteries with a dermal venous network and a sub-papillary venous network.
- Between the terminal arterioles of the sub-papillary network exist numerous simple connections between blood vessels (anastomosis).
- The lymphatic vessels form a network under the dermis that is difficult to isolate.



Innervation is provided by the cutaneous branches of the spinal nerves and certain cranial nerves. These nerves constitute subepidermal and dermal plexuses. Note that the dermis is rich in nerve ending, and the epidermis contains only free nerve endings.
Sensory neurofibres ensure the perception of touch and pain, and sympathetic neurofibres control vasomotor skills, and sweating. Note that a cutaneous territory innervated by a spinal nerve constitutes a dermatome.

Functional anatomy

The skin provides the following functions:

- It is a sensory organ that allows one to feel and recognize pain, touch, pressure, and temperature due to its richness in tactile, thermal, and pain receptors.
- It isolates the organism from the external environment due to its resistance, elasticity, and secretions (sweat, sebum, keratinized cells, and provitamin D). The mechanical resistance depends mainly on the stratum corneum and the dermis. The skin also interacts with the immune system of the body and help destroys microorganisms. The cells within the skin, like Langerhans cells, phagocytic cells, and epidermal dendritic cells, help with immunity. In addition, melanocytes protect against solar radiation.
- It is involved in the thermo-regulation of the body. This function results from many nerve receptors collecting barometric and thermal stimuli and of a dense vascularization. Thermolysis takes place in the skin by convection, conduction, radiation, or evaporation.
  - Convection is the movement of molecules away from the area of contact.
  - Conduction is the heat exchange between two objects in contact with each other.
  - Radiation is a transfer of heat by high-frequency waves from one object of a higher temperature to another.
  - Evaporation is the process of turning from liquid into vapour.
- It is a purifying organ, particularly for CO2 and urea. Note that the quantity of water that is eliminated by perspiration (Sweating) is slightly greater than that of the lung (500 to 700 g per day).
- It is an organ of absorption of water and gas, except for carbon monoxide: This function is used in therapy.
- It is a metabolic organ that participates in the synthesis of certain vitamins (A, B, C, D) and is involved in the mechanisms of immune-allergy.

Notes

- Vital prognosis is engaged in the event of the destruction of a skin surface greater than 10%.
- With 1st degree burns, such as sunburns, the surface layers are destroyed. They are characterized by redness of the skin (erythema). Healing starts from the deepest layer of the epidermis (stratum basal). It ends with the peeling of the dead skin.
- With a 2nd degree burn, the basal layer of the epidermis is affected. It is characterized by blisters, which are circumscribed elevations of the epidermis due to a clear fluid collection in a neo-formed cavity. The repair starts from the epithelium. It results in an indelible scar but not retractable.
- With a third-degree burn, the entire dermis is affected. It is characterized by the formation of skin ulcers that are persistent deep ulceration due to necrosis resulting from the interruption of the vascularization of the area. Skin regeneration is slow and uncertain. It starts from the basal stratum and ends in a scar.
- Note that prolonged stop of cutaneous blood circulation leads to the formation of bedsores.
- Water exposure is an influential factor in some common dermatoses. It has also been shown that water has an effect on barrier function and biophysical properties of skin.

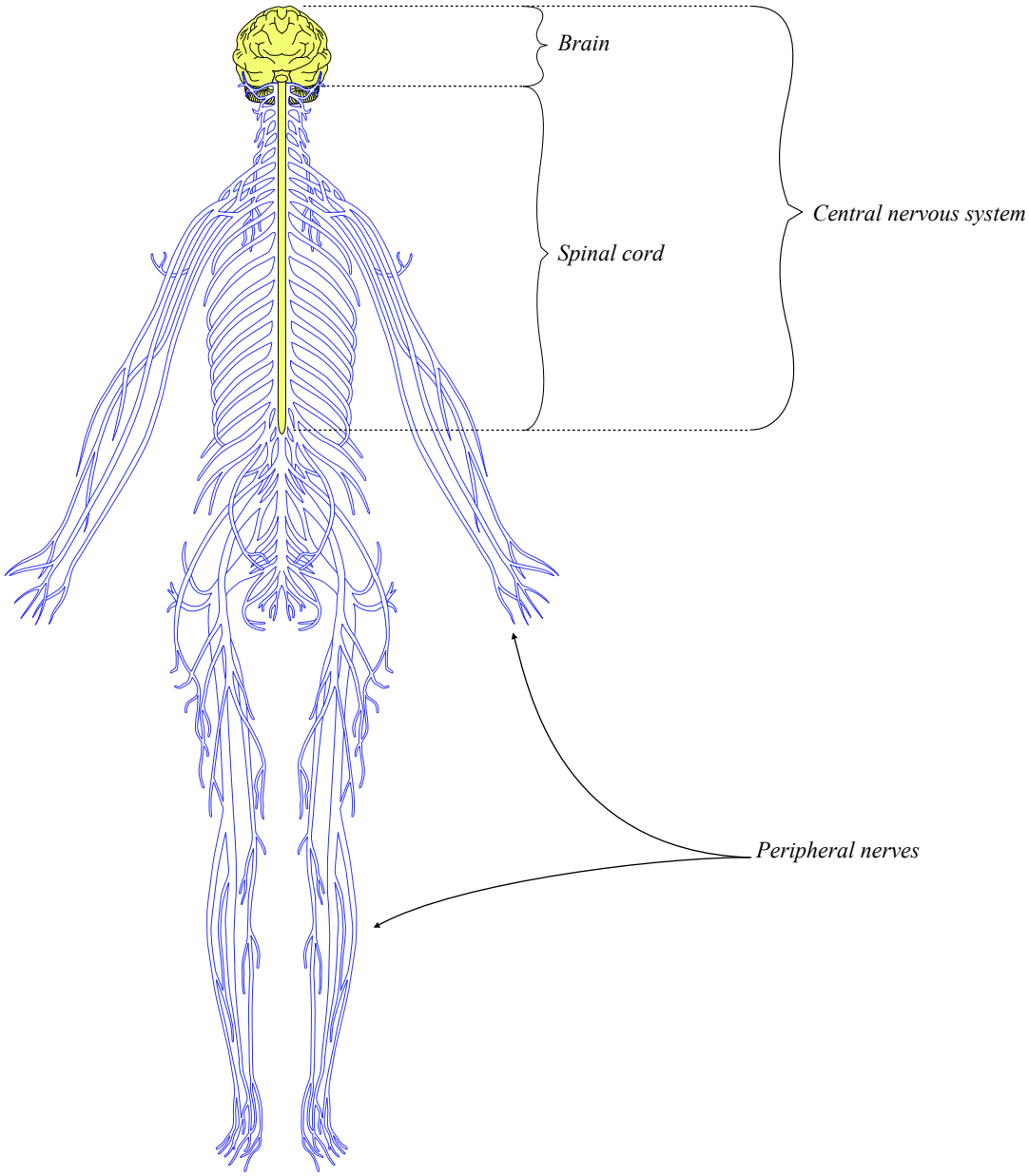
Nervous system

Overall description

- Central and peripheral nervous systems

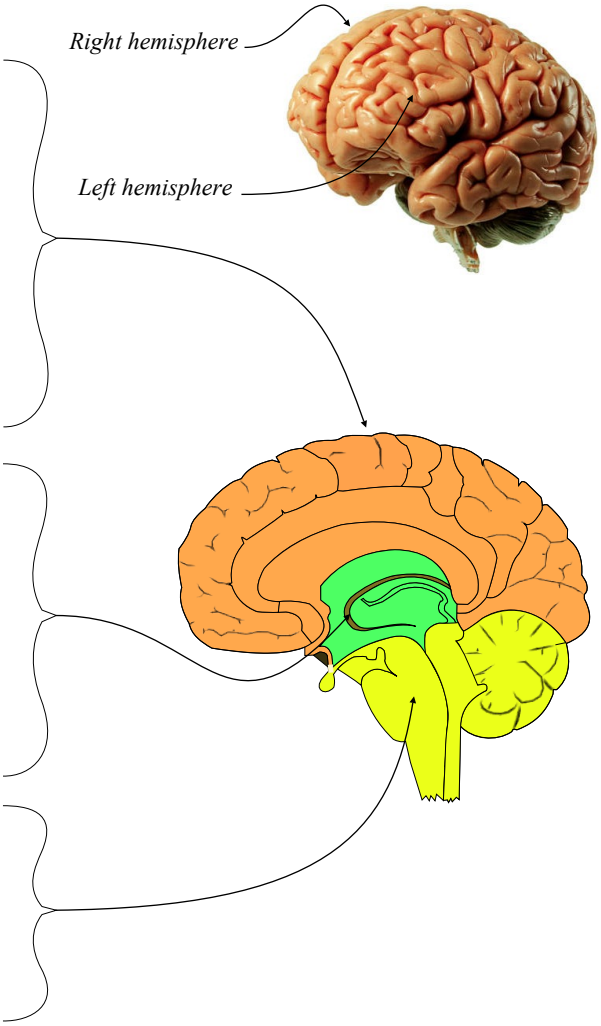
The nervous system has two parts, called the “central nervous system” and the “peripheral nervous system”.

- The central nervous system (CNS) includes the nerves in the brain and spinal cord that are contained within the skull and vertebral canal of the spine.
  - The brain is the centre of higher mental functions such as consciousness, memory, planning, and voluntary actions. Also, it controls autonomic functions such as the maintenance of respiration, heart rate, blood pressure, and digestion.
  - The spinal cord consists of nerves that carry incoming and outgoing messages between the brain and the rest of the body. It is also the centre for reflexes and some autonomic functions.
- All of the other nerves in the body are part of the peripheral nervous system (PNS).
  - Sensory nerves, also called an afferent nerves, carry sensory information toward the central nervous system.
  - Motor nerves transmit the messages for movement and necessary actions to the skeletal muscles.



- The brain and the spinal cord

The brain is divided into 3 parts:



The fore brain *(also called procencephalon):*
It is in the top part of the cerebrum. It is the area where complex reasoning are elaborated.
It includes the telencephalon that contains the cerebral hemispheres, and then the diencephalon under them. The diencephalon contains:

- Thalamus (organization of sensory information),
- Hypothalamus (endocrine system and thermo regulation),
- Epithalamus (secretion of melatonin, regulation of motor pathways, and emotions) and sub-thalamus (correlation center for optic and vestibular impulses).

The midbrain *(also called mesencephalon):*
It is the region of the brain that contains the cranial nerves that stimulate the muscles controlling eye movement, lens shape, and pupil diameter. The midbrain is located in between the forebrain and the hindbrain. It is made up of the tectum and the tegmentum.
The tectum is contrasted with the tegmentum and responsible for auditory and visual reflexes.
The tegmentum is where several cranial nerve nuclei are located.

The hindbrain *(Also called Rhombencephalon):*
It is the lower portion of the brain which controls functions such as respiration and heart rate. The hind brain is made up of the brain stem and the cerebellum.
The brain stem that is made up of medulla, reticular formation, and pons. It controls functions such as breathing and swallowing. The brain stem also connects the brain to the spinal cord.

The brain is composed of several areas each of them has a particular function and works in connection with the other areas to effect the voluntary and involuntary actions.

#1 - Medulla
It is part of the “brain stem” *(see previous page)*. It controls the autonomic (involuntary) functions such as the cardiac, respiratory, vomiting and vasomotor centres and therefore deals with the autonomic functions of breathing, heart rate and blood pressure.

#2 - Pons
It is also part of the “brain stem”. It conducts signals from the brain stem to the cerebellum and medulla, and carries the sensory signals to the thalamus.

#3 - Hypothalamus
It regulates the autonomic nervous system that governs temperature regulation, response to effort, thirst, hunger, sleep, mood, and sex drive. It is also involved in endocrine functions, such as metabolism and growth.

#4 - Thalamus
It is located just above the brain stem between the cerebral cortex and the midbrain and has extensive nerve connections to both. The main function of the thalamus is to relay motor and sensory signals to the cerebral cortex.

#5 - Cerebellum
It receives input from sensory systems of the spinal cord and from other parts of the brain, and integrates these inputs to fully controlled motor activity. Disorders in fine movement, equilibrium, posture, and motor learning result from the cerebellum being unable to perform its function.

#6 - Limbic system
It is an ensemble of nerves that connects the hypothalamus with other areas of the frontal and temporal lobes and controls and memorises the emotions and the expressions associated, as well as some automatic functions.

#7 - Visual areas *(visual cortex)*
They are situated above the cerebellum and interpret the signals from the eyes transmitted through the thalamus. They allow recognizing elements such as complex shapes and colors. Note that the visual cortex in the left hemisphere receives signals from the right eye, and the visual cortex in the right hemisphere receives signals from the left eye.

#8 - Posterior parietal cortex
It is above the visual areas and synthesises multiple sensory inputs to create a complete comprehension of the object being felt.

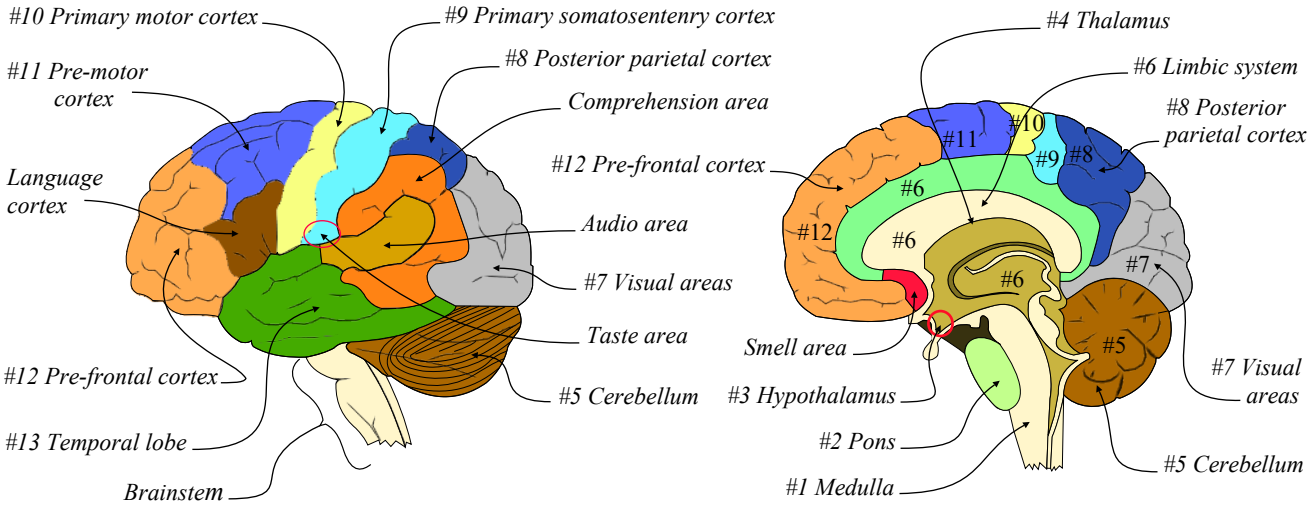
#9 - Primary somatosentenry cortex
It is the region of the brain to which the information received by the body through its five senses is transmitted.

#10 - Primary motor cortex
It works in association with other motor areas including pre-motor cortex, the primary somatosentenry cortex, posterior parietal cortex, and several sub cortical brain regions, to plan and execute movements.

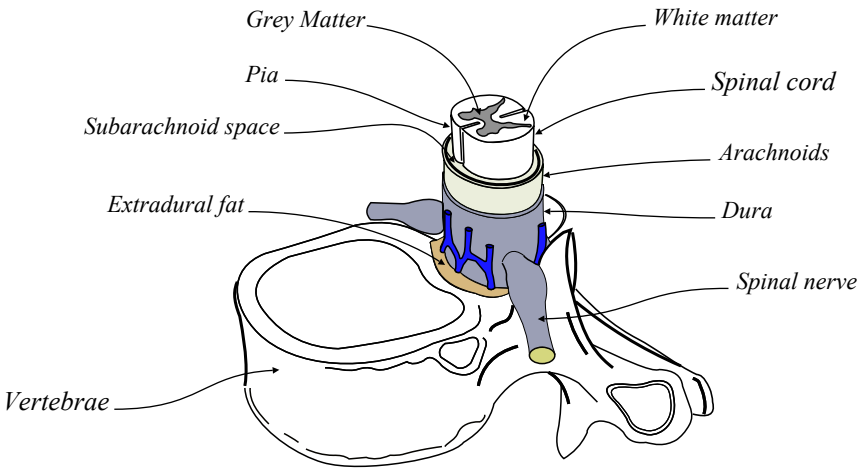
#11 - Pre-motor cortex
It may play a role in the direct control of the trunk muscles of the body. It may also play a role in planning movement, in the spatial guidance of movement, in the sensory guidance of movement, in understanding the actions of others, and in using abstract rules to perform specific tasks.

#12 - Pre-frontal cortex
This part of the brain is the centre of higher cognitive and emotional functions.

#13 - Temporal lobe:
It manages many cognitive functions, including hearing, language, memory and vision of complex forms.



The spinal chord is the continuation of the brain to which the peripheral nerves are connected. It transmits the information from the organs, muscles, and sensory cells to the brain, and send the appropriate orders to adapt the body to the environment or perform a voluntary action. It is protected by the vertebrae and the meninges (dura, arachnoids, subarachnoid space, pia). The subarachnoid space is the interval between the arachnoid membrane and the pia.



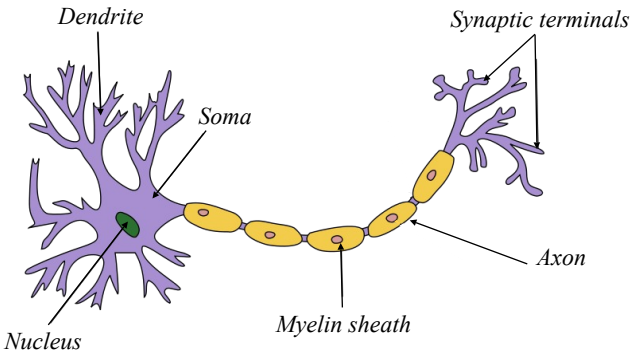
- Neurons, grey and white matters

The central nervous system is made of grey and white matter which are composed of neurons. A neuron, also called nerve cell, receives, processes, and transmits information through electrical and chemical signals. These nerve cells can connect to each other and form chains and bundles. The signals are transferred between neurons via specialized connections called synapses. A neuron is composed of the following main elements:

- The soma, or cell body, that is usually compact
- The axon and dendrites that are filaments that extrude from the soma. signals to other neurons are transmitted by

- The dendrites branch profusely, getting thinner with each branching. They are terminated by synapses.

Neurons have never more than one axon. They are designed according to their function; Some may have numerous dendrites, where some others lack dendrites, or can be without axon, or have a long axon *(approximately 1.5 m max)* protected by a myelin sheath when some others have a short axon without protection.



The difference between grey matter and white matter is that grey matter contains numerous cell bodies and relatively few myelinated axons, while white matter contains relatively few cell bodies and is mainly composed of long axons protected by a myelin sheath.
A lot of neurons forming the grey matter are characterised by short axons. They control senses and functions such as elocution, hearing, feeling, view, memory, and also the muscles.
The white matter composes structures at the centre of the brain such as the thalamus and the hypothalamus. It is found between the brainstem and the cerebellum. It allows communication to and from grey matter areas, and between the grey matter and the other parts of the body. It is involved in the control of functions such as temperature, blood pressure, heart rate, control of food, as well as the intake of water and the expression of emotions.

There are two categories of synapses which do not make body contact with each other when conveying messages:

- Chemical synapses
  In a chemical synapse, electrical activity in the pre-synaptic neuron is converted into the release of a chemical called a neurotransmitter. The neurotransmitter may initiate an electrical response or a secondary messenger pathway that may either excite or inhibit the post-synaptic neuron. Because of the complexity of receptor signal transduction, chemical synapses can have complex effects on the post-synaptic cell.
- Electrical synapses
  The pre-synaptic and post-synaptic cell membranes of an electrical synapse are connected by special channels called gap junctions or synaptic cleft that are capable of passing an electric current, causing voltage changes in the pre-synaptic cell to induce voltage changes in the post-synaptic cell.

Note that because a neurotransmitter can stimulate or inhibit neurons that produce other neurotransmitters, the disruption of one neurotransmitter can have secondary impacts on others and then corrupt the chain of information or command of the central nervous system.

- Transmission of signals to and from organs

The transmission of signals to and from organs is performed through nerves and ganglia.

- Nerves are collection of neurofibres visible to the naked eye that are classified according to:
  - Their distribution with the cranial nerves that emerge from the brain, and the spinal nerves that originate from the spinal cord.
  - Their function, with the motor nerves, sensory nerves, and mixed nerves (which are both motor and sensory).
- Ganglia. are nodular formations located on the path of the nerves.

Twelve pairs of cranial nerves emerge from the inferior surface of the brain. Most of them have both sensory and motor components.

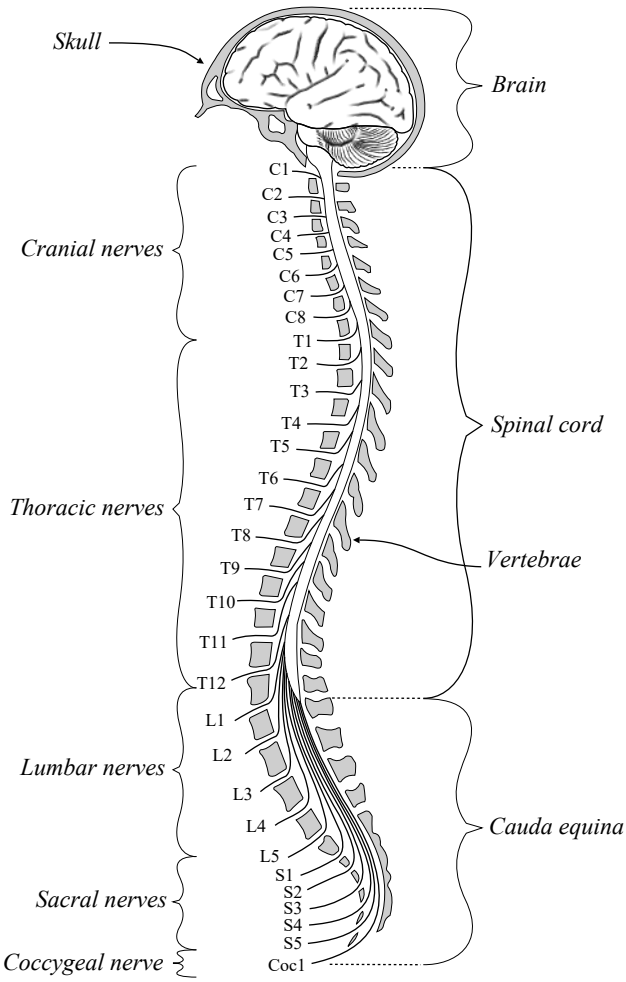
- Three of the nerves are associated with the special senses of smell, vision, hearing, and equilibrium and have only sensory fibers.
- Five other nerves are primarily motor in function but do have some sensory fibers.
- The remaining four nerves consist of significant amounts of both sensory and motor fibers.

Thirty-one pairs of spinal nerves emerge laterally from the spinal cord. Each pair of nerves corresponds to a segment of the cord and they are named accordingly. Thus, eight cervical nerves, twelve thoracic nerves, five lumbar nerves, five sacral nerves, and one coccygeal nerve.

- Each spinal nerve is connected to the spinal cord by a dorsal root and a ventral root.
- The cell bodies of the sensory neurons are in the dorsal root ganglion, but the motor neuron cell bodies are in the grey matter.
- The two roots join to form the spinal nerve just before the nerve leaves the vertebral column.
- Because all spinal nerves have both sensory and motor components, they are all mixed nerves.

The connection of the peripheral nervous system to the central nervous system through the spinal cord is organised as follows:

Nerve roots	*Parts of the body innerved*
C1 to C4	*- Intracranial blood vessels, eyes, lacrimal glands, parotid glands.* *- Base of the skull, neck muscles, diaphragm.*
C5 to C8	*- Neck muscles, shoulders, elbows, arms, wrists, hands, fingers.* *- Oesophagus, heart, lungs, chest.*
T1 to T4	*- Arms.* *- Heart, lungs, chest.* *- Oesophagus, trachea, larynx.*
T5 to T10	*- Gallbladder, liver, diaphragm, stomach, pancreas, spleen, kidneys, small intestine, appendix, adrenals.*
T11 to T12	*- Small intestines, colon.* *- Uterus, buttocks.*
L1 to L5	*- Large intestines, colon,* *- Reproductive organs.* *- Buttocks, groin, thighs, knees, legs, feet.*
S1 to S5	*- Reproductive organs, bladder, prostate gland.* *- Buttocks, legs, ankles, feet toes.*
Coccygeal	*- Anus & rectum*



- Autonomic nervous system

The autonomic nervous system regulates the involuntary processes of organs such as:

- Blood pressure
- Heart and breathing rates
- Body temperature
- Digestion
- Metabolism
- The balance of water and electrolytes (such as sodium and calcium)
- The production of body fluids (saliva, sweat, and tears)
- Constriction of the pupil in bright light and dilation of the pupil in dim light.
- Urination
- Defecation
- Sexual activity

It responds to information sent by a receptor cell and processed in the brain using stimulating processes through the sympathetic division, or inhibiting process through the parasympathetic division.

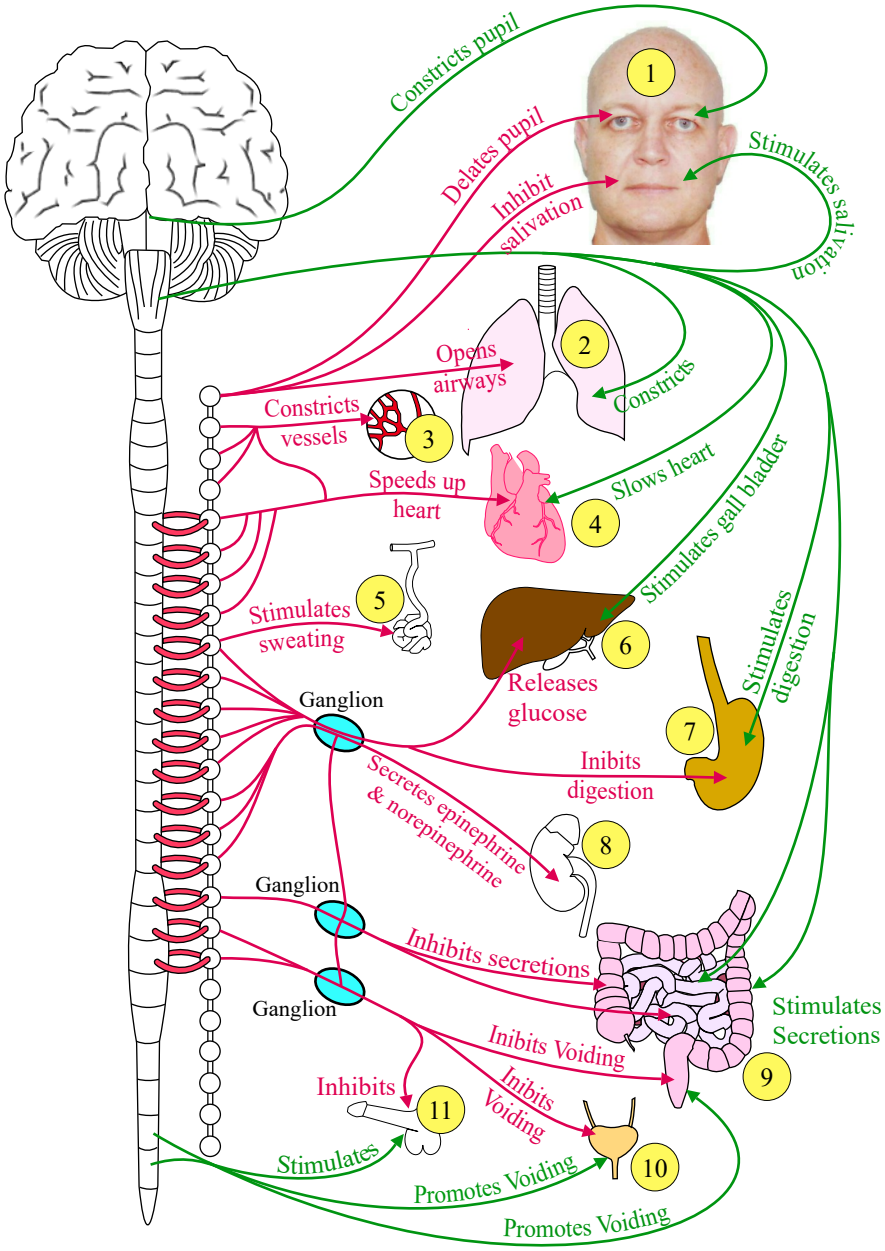
- The sympathetic division prepares the body for stressful or emergency situations. As an example, it increases heart rate and the force of heart contractions and dilates the airways to make breathing easier.
- The parasympathetic division regulates body process during ordinary situations. As an example, it slows the heart rate and decreases blood pressure. It stimulates the digestive tract to process food and eliminate wastes

The process selected is effected through one cell located in the brain stem or the spinal cord that is connected to the other cells through the “autonomic ganglions” which are clusters of nerve cells.
Most ganglions for the sympathetic division are located on both sides of the spinal cord. However, they are not part of it.
The ganglia involved in the actions from the parasympathetic division are located near or in the organs.

If a person is affected by an accident, a disease, or a drug, the reaction of the autonomic nervous system shows symptoms that can be interpreted by the medic to evaluate which parts of the nervous system are affected.
Also, disorders of the autonomic nervous system can lead to death if nothing is done in time.

Actions of the sympathetic division (in red) and of the parasympathetic division (in green) on various organs:

#1 - Head
#2 - Lungs & airways
#3 - Blood vessels
#4 - Heart
#5 - Sudoriparous glands
#6 - Liver + pancreas
#7 - Stomach
#8 - Kidneys
#9 - Intestines
#10 - Bladder
#11 - Sex



Elements that may affect the nervous system

The nervous system is vulnerable to various disorders such as those listed as follows:

- Trauma
- Infections
- Degeneration
- Structural defects
- Tumors
- Blood flow disruption
- Auto-immune disorders

Nervous system disorder can be detected by the following symptoms:

- Persistent or sudden onset of a headache
- A headache that changes or is different
- Loss of feeling or tingling
- Weakness or loss of muscle strength
- Loss of sight or double vision
- Memory loss

- Impaired mental ability
- Lack of coordination
- Muscle rigidity
- Tremors and seizures
- Back pain which radiates to the feet, toes, or other parts of the body
- Muscle wasting and slurred speech
- Language impairment (expression or comprehension)

Circulatory and respiratory systems

Circulatory system

The circulatory system, also called cardiovascular system, consist of the blood, heart, and blood vessels *(arteries and veins)*, and includes the lymphatic system.

- ***The blood***

The blood is a fluid consisting of plasma *(55%)*, red blood cells, white blood cells, and platelets, carrying oxygen, nutrients *(such as amino acids and electrolytes)*, hormones, and cellular waste products away from the tissues. The body contains approximately five litres of blood.

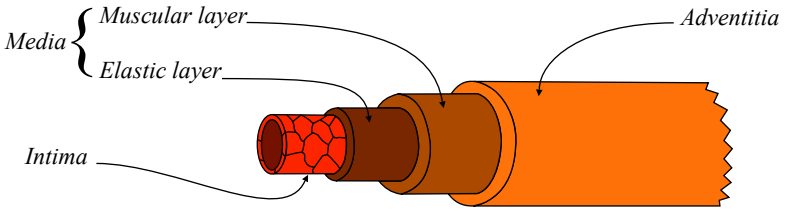
- The plasma is constituted of
  - water *(91%)*,
  - proteins *(albumin, globulin, fibrinogens)*,
  - nitrogenous organic substances *(Urea, uric acid, ammonia, creatinine, bilirubin*
  - lipids, carbohydrate, cholesterol, and lactic acid
  - Minerals that are divided in "cations" *(elements with positive electric charge such as sodium, potassium, calcium, and magnesium)*, and "anions" *(elements with negative electric charge, such as chlorine and phosphate)*.

  The electrical balance of the plasma is neutral, and it's acid-base balance nearly neutral *(7.37 instead of 7)*. Anions are the acid elements and cations the base elements. The cellular metabolism tends producing ions H+ that increase the acidity of the blood. This acidity is eliminated through the kidneys and the lungs. However, in the case that this elimination is not sufficient "buffer substances" such as proteins, hemoglobin, and bicarbonate capture the ions H+ to reestablish the ideal balance.
- Red blood corpuscles, also called red cells or erythrocytes, are bi-concave disk shaped cells without a nucleus that transport oxygen through hemoglobin. As they have no nucleus red blood cells do not contain DNA and cannot repair themselves once damaged. They are produced inside of red bone marrow from stem cells. Hemoglobin is a substance made of "hem" *(5%)*, which is composed of iron and "porphirin"*(*1)*, that is combined with a protein called "globin" *(95%)*.

  *(*1) Porphyrins are organic aromatic compounds composed of four pyrrole rings interconnected to each other and to the Fe2+ ion. Pyrrole is a basic, cyclic substance, obtained by destructive distillation of various animal substances whose formula is C4H4NH.*
- White blood cells, also called leukocytes, that are less numerous than red cells *(6 to 8000/mm³)* are parts of the body's immune system protecting the body against infectious disease and foreign invaders. They are produced and derived from multipotent cells in the bone marrow called hematopoietic stem cells. They are divided into two classes:
  - "Granular Leukocytes", also called "granulocytes" are a class of leucocytes with a granular cytoplasm that contain digestive enzymes neutralizing bacteria that invade the body. It is composed of the "Neutrophils", "Eosinophils", and "Basophils".
    - Neutrophils are polymorphonuclear leukocytes that defend the organism against bacterial or fungal infection. They represent 70% of the leucocytes.
    - Eosinophils have a bi-lobed nucleus. They deal with parasitic infections and inflammatory cells in allergic reactions.
    - Basophils release histamine and heparin. Histamine triggers the dilatation of the vessels to increase the flow of blood to injured tissues. Heparin is an anticoagulant that inhibits blood clotting and promotes the movement of white blood cells into an area.
  - "Agranular leukocytes", also called "agranulocytes" or "mononuclear leukocytes", are characterized by the absence of granules in their cytoplasm. This class is composed of "Lymphocytes" and "Monocytes"
    - Lymphocytes, which are much more common in the lymphatic system than in blood, include T cells and natural killer cells that fight off viral infections and B cells that produce antibodies against infections by pathogens.
    - Monocytes develop into cells called macrophages that engulf and ingest pathogens and the dead cells from wounds or infections.
- "Platelets", also called "thrombocytes", are small cell fragments without nucleus, that are responsible for the clotting of blood and the formation of scabs. Platelets form in the red bone marrow from megakaryocyte cells. They survive in the blood system for up to a week before macrophages capture and digest them.
  Low platelet concentration is called "thrombocytopenia" and is due to either decreased production or increased destruction. This can trigger mild to serious bleeding.
  Elevated platelet concentration is called "thrombocytosis" and is either congenital, reactive (to cytokines), or due to unregulated production. Effects of thrombocytosis can include stroke, heart attack, and blood clots due to blood vessel blockage.

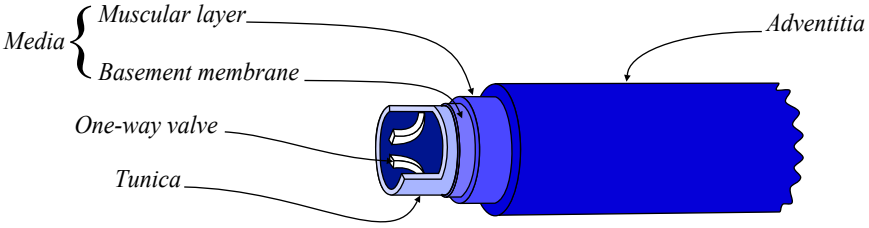
The blood is carried through the arteries which are the blood vessels that deliver oxygen-rich blood from the heart to the tissues of the body, and the veins that return the deoxygenated blood and the wastes dissolved in it to the heart.
Each artery is a muscular tube lined by smooth tissue that has three layers:

- The intima, which is the inner layer lined by a smooth tissue called endothelium.
- The media, composed of an elastic layer and a muscular layer that handle high pressures.
- The adventitia, which is a connective tissue anchoring arteries to nearby tissues.



Arterioles are narrower arteries that branch off from the ends of arteries and carry blood to the capillaries. The capillaries carry the blood close to the cells of the tissues. Their walls consist of only a thin layer of endothelium which acts as a filter which keeps the blood cells inside of the vessels while allowing liquids, dissolved gases, and other chemicals diffusing into or out of the tissues. The arterial pressure decreases in the arterioles and the capillaries, from 116 - 120 mm Hg *(considered normal tension)* in the arteries to 15 mm Hg at the end of the capillaries. The capillaries are connected to the venules that return the blood to the veins and the heart.
A vein is also composed of three main layers:

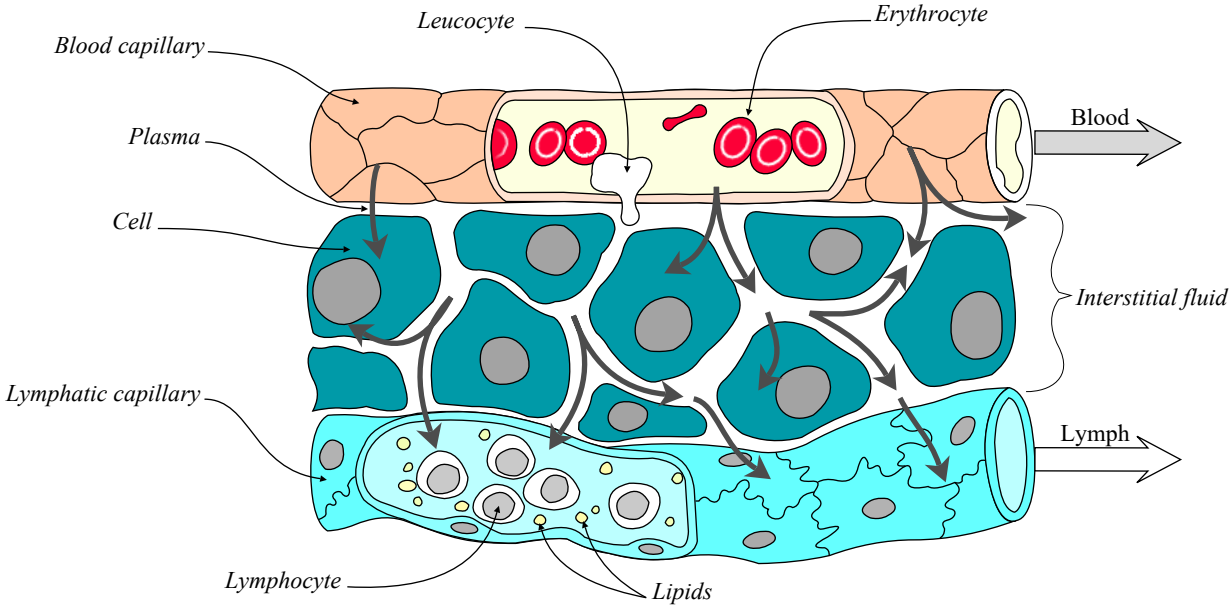
- The inner layer lined with endothelial cells called “tunica”. One-way valves are present in some veins.
- The middle layer, called the tunica media which is composed of smooth muscles.
- The adventitia, which is a connective tissue anchoring arteries to nearby tissues.



The blood pressure in the veins is low *(15 mm Hg max and decreasing)*. For this reason, the blood in the veins is pushed to the heart by gravity, inertia, the force of skeletal muscle contractions, and one-way valves that prevent it from flowing back.

- The lymphatic system

The lymph is a clear/white fluid made of white blood cells, especially lymphocytes *(cells that attack bacteria)* and fluid from the intestines called “chyle”, which contains proteins and fats.
The lymphatic system is the circulatory system that filters the interstitial fluid before putting it back into the bloodstream. Tiny lymphatic capillaries arise between the cells and unite each other to gradually constitute the network of lymphatic vessels. They are connected to the lymph nodes which are grouped in clusters at the level of the roots of the limbs, the upper abdomen level (epigastric), and at the supraclavicular level. Blood plasma leaks into tissues through the thin walls of the capillaries. The portion of blood plasma that escapes is called interstitial or extra-cellular fluid. It contains oxygen, glucose, amino acids, and other nutrients needed by the cells.



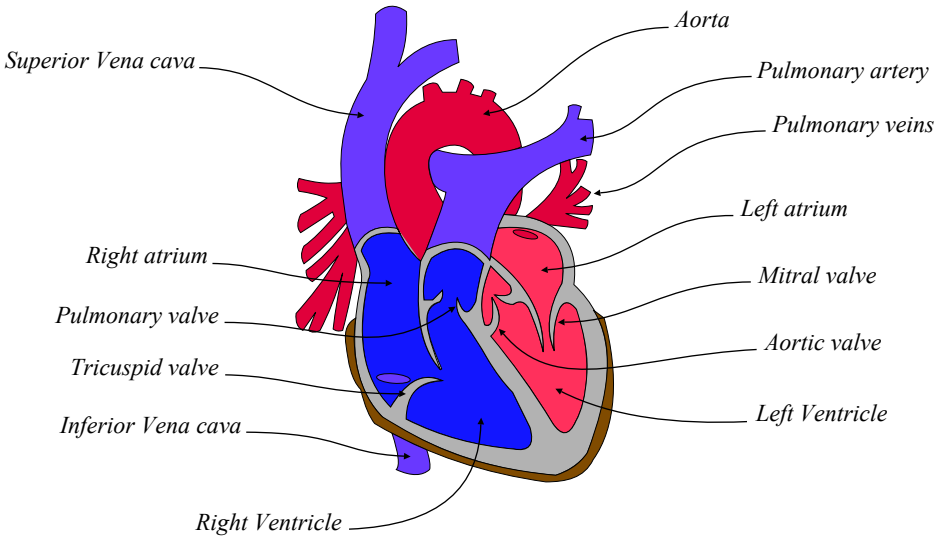
The lymphatic system has three main roles:

- It removes proteins from the "interstitial fluid compartment" *(brought back into the bloodstream)* to maintain a significant gradient of "oncotic pressure" *(*2)* between the interstitial compartment *(low in proteins)* and the "plasma compartment" *(rich in protein)*, which is necessary to maintain liquids in the bloodstream. Disruption of this mechanism leads to an excess of fluid from the plasma compartment to the interstitial compartment, which is one of the causes of oedema *(the other cause is capillary blood pressure)*.
  *(*2) Oncotic (or colloid osmotic) pressure is a form of osmotic pressure exerted by proteins that usually tends to pull water into the circulatory system.*
- It filters the lymph throughout its return to the subclavian veins. It is the role of lymph node reticuloendothelial cells *(*3)* that clear the lymph from harmful bacteria, toxins and protein debris.
  *(*3) Reticuloendothelial cells (also called macrophage cells) are phagocytic cells having the ability to take up and sequester inert particles and vital dyes.*
- Finally, due to the action of the lymphocytes it plays a vital role in the immunological defences of the organism.

- ***The heart***

The heart is a muscular pumping organ located between the lungs along the body's midline in the thoracic region.
The wall of the heart is made up of three layers: epicardium, myocardium, and endocardium. They are supplied in oxygen and nutriments by the coronary arteries. enclosed in a protective tissue, the pericardium, which also contains a small amount of fluid. The deoxygenated blood returns to the inner heart through the coronary sinus. The rhythm of the heart is determined by a group of cells in the sinoatrial node.
The heart is divided into four chambers:

- Upper left and right atria
- Lower left and right ventricles.



The heart has four valves, which separate its chambers. One valve lies between each atrium and ventricle, and one valve at the exit of each ventricle.

- The right atrium and ventricle which are referred "the right heart" receives deoxygenated blood from the systemic veins and pumps it to the lungs for oxygenation. The right atrium receives blood almost continuously from the superior and inferior vena cava. A small amount of blood from the coronary circulation drains into the right atrium via the coronary sinus, which is immediately above and in the middle of the opening of the inferior vena cava.
  The internal surface of the right atrium is smooth. An oval-shaped depression which is a remnant of an opening in the fetal heart called the "foramen oval" is in the wall between the right and the left atrium. Studies show that this depression is not fully closed in 25% of people.
  The right atrium is connected to the right ventricle by the tricuspid valve. The walls of the right ventricle are lined with trabeculae carneae, which are ridges of cardiac muscle covered by endocardium. They prevent the inversion of the tricuspid valve and the aspiration of the walls that would impair the ability of the heart to pump efficiently , which could be the case with a flat surfaced membrane.
  In addition to these muscular ridges, a band of cardiac muscle, also covered by endocardium, called "moderator band" reinforces the thin walls of the right ventricle and plays a role in cardiac conduction. The right ventricle tapers into the pulmonary trunk, into which it ejects the blood when contracting. The pulmonary trunk branches into the left and right pulmonary arteries that carry the blood to each lung. The pulmonary valve lies between the right heart and the pulmonary trunk
- The left counterparts, called the left heart, receives oxygenated blood from the lungs and pumps it through the systemic arteries to the tissues of the body. The left atrium, and the left ventricle are separated by the mitral valve. As in the right atrium, the left atrium is lined with pectinate muscles. It receives oxygenated blood back from the lungs via the pulmonary veins.

The left ventricle is thicker than the right ventricle because a greater force is needed to pump the blood to the entire body. As for the right ventricle, the left ventricle also has trabeculae carneae, but there is no moderator band. The left ventricle pumps the blood to the body through the aortic valve and into the aorta. Two small openings above the aortic valve carry the blood to the coronary arteries.

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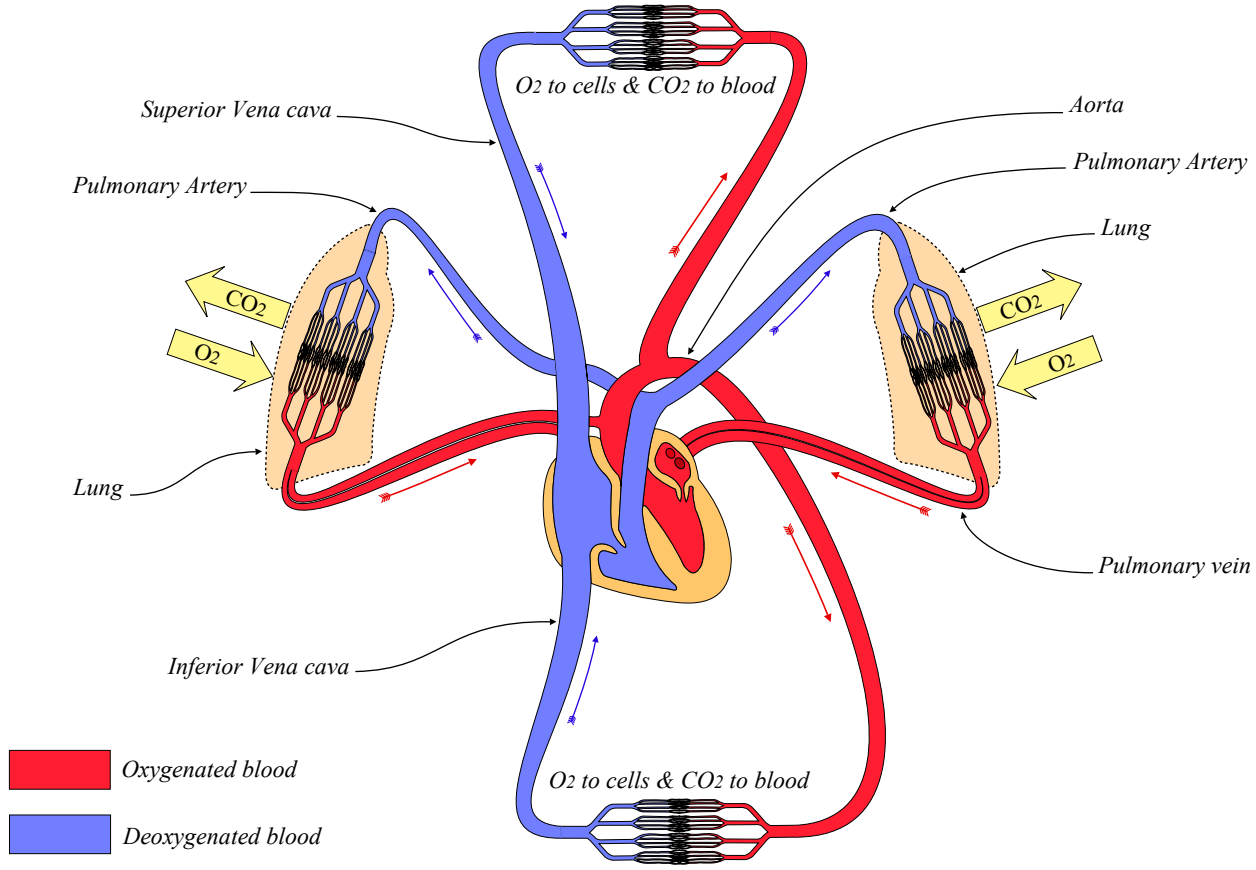
Note that pulmonary and aortic valves close when the pressure in the arteries is above the pressure in the ventricles and tricuspid and mitral valves close when the pressure in the ventricles is above the pressure in the atrium, which allows only a one-way blood circulation.

The circulation of the blood to and from the heart is divided into “pulmonary circulation” *(also called small circulation)*, and “systemic circulation” *(also called large circulation)*:

- Pulmonary circulation begins in the right ventricle from which the deoxygenated blood is pushed into the pulmonary artery that is connected to the lungs where the red cells are oxygenated. The oxygenated blood returns to the left atrium and then enters the left ventricle where the systemic circulation starts.
- The systemic circulation begins in the left ventricle from which the oxygenated blood is pushed into the aorta and is then distributed into the arteries, arterioles, and capillaries to transfer nutrients and oxygen to the cells. Then, the blood returns to the right atrium through the capillaries, venules, and veins. From the right atrium, it is pushed to the right ventricle from which the pulmonary circulation starts again.



As any muscular tissue, the cardiac muscle contracts only when it is stimulated by an excitation which, in this case, is a depolarisation. The origin of the stimulation resides in certain regions of the cardiac muscle that are part of the “Electrical conduction system”.

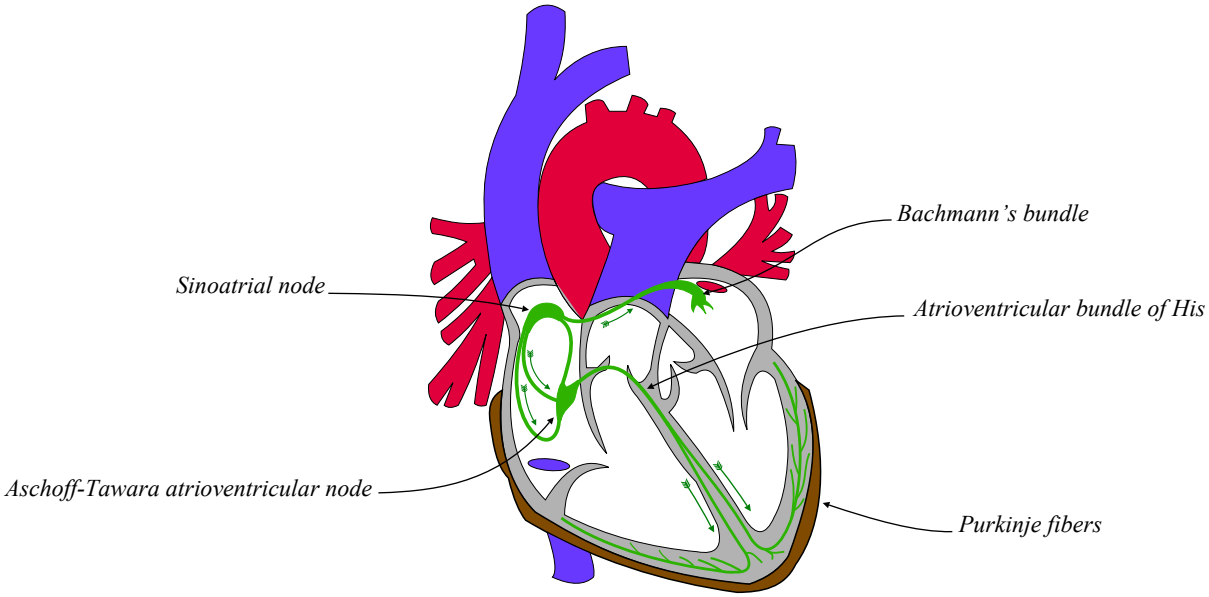
In a particular point of the electrical conduction system called “sinoatrial node”, a spontaneous modification of the ionic permeability of the membrane of the cells gives rise to a brief depolarization followed by long repolarization with a prolonged period during which the muscle is unresponsive *(refractory period)* that propagates in all the electrical conduction system and the neighbouring cardiac muscle tissues. The electrical conduction system is not a nervous network, but particular points of the cardiac muscle called “pacemaker cells”, from and to which the automatic and rhythmic beats of the cardiac muscle originate and transfer.

- The Sinoatrial node is located in the right atrium. It gives the heart rhythm (pacemaker).
- The Aschoff-Tawara atrioventricular node is located in the upper part of the interventricular septum of the right heart and transmits the cardiac impulse initiated by the sinoatrial node.
- The atrioventricular bundle of His descends into the inter-ventricular septum and divides into a right and left arm for each corresponding ventricle. Each arm leads to the “Purkinje fibers”, which provide electrical

conduction to the ventricles, causing the cardiac muscle of the ventricles to contract at a paced interval.

- The Bachmann's bundle activates the left atrium

The stimulation wave from the sinoatrial node propagates through the muscle and the electrical conduction system. It reaches the Aschoff-Tawara atrioventricular node within 13/100 of a second and then progresses through the atrioventricular bundle of His. As a result, there is 10/100 of a second difference between the contraction of the atrium and the beginning of the ventricular contraction. This delay allows the atriums to complete their contraction before the ventricles start moving. As the electrical conduction system consists of cardiac muscle cells and conducting fibers, any lesion of the wall reaching the myocardium and/or resulting in the death of myocardial cells (myocardial infarction) can generate troubles of the cardiac rhythm.

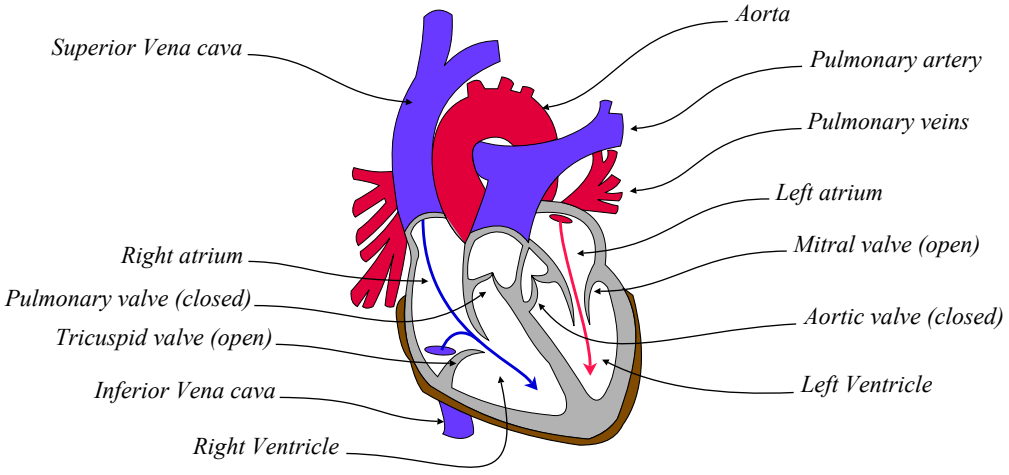


The cardiac cycle is the sequence of events that occur when the heart beats. Each side of the heart operates in two steps

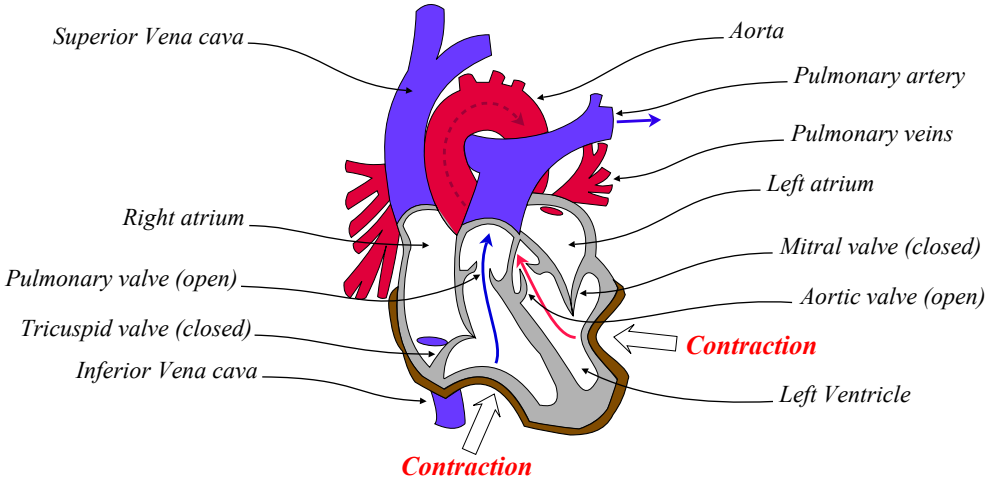
- A step called diastole *(from Greek diastole = dilation)*. It is the time where the muscle fibers are relaxed and the heart fills with blood.
- A step called systole *(from Greek systole = contraction)*. It is the muscular contraction that pushes the blood out of the cavity of the heart *(atrium of ventricle)*.

The cardiac cycle phases explained below are based on the blood circulation explained previously and starts when the deoxygenated blood from the systemic circulation and the oxygenated blood from the pulmonary circulation enter into the heart

1. The atrium and ventricles are relaxed and the atrioventricular valves are open *(diastolic phase)*.
2. Oxygen-depleted blood returning to the heart from the body passes through the superior and inferior vena cava and flows to the right atrium. At the same time, the oxygenated blood from the pulmonary veins fills the left atrium *(diastolic phase)*.
3. The open atrioventricular valves *(tricuspid and mitral valves)* allow the blood to pass through the atriums to the ventricles. Impulses from the sinoatrial (SA) node travel to the atrioventricular node that sends signals that trigger both atrium to contract *(systolic phase)*.
4. As a result of the contraction, the right atrium empties its content into the right ventricle, and the left atrium empties its content into the left ventricle.
5. The tricuspid valve, located between the right atrium and right ventricle, prevents the blood from flowing back into the right atrium. Simultaneously, the mitral valve, located between the left atrium and left ventricle, prevents the oxygenated blood from flowing back into the left atrium.



6. The ventricles are filled with the blood from the atriums *(Diastolic phase)*.
7. The ventricles receive impulses from the Purkinje fibers, which carry the electrical impulses to the ventricles causing them to contract *(systolic phase)*. As this occurs, the atrioventricular valves close and the "semilunar valves" *(pulmonary and aortic valves)* open.
8. The ventricular contraction pushes the deoxygenated blood from the right ventricle to the pulmonary artery. At the same time, the oxygenated blood in the left ventricle is pushed to the aorta. The pulmonary valve prevents the blood from flowing back into the right ventricle, and the aortic valve prevents the oxygenated blood from flowing back into the left ventricle.
9. The pulmonary artery carries the deoxygenated blood to the lungs, where it picks up oxygen . Simultaneously, the oxygenated blood is provided to all parts of the body through the systemic circulation.
10. Then the oxygen-depleted blood is returned to the heart via the vena cava, and the oxygenated blood is returned from the lung to the left atrium of the heart by the pulmonary veins.

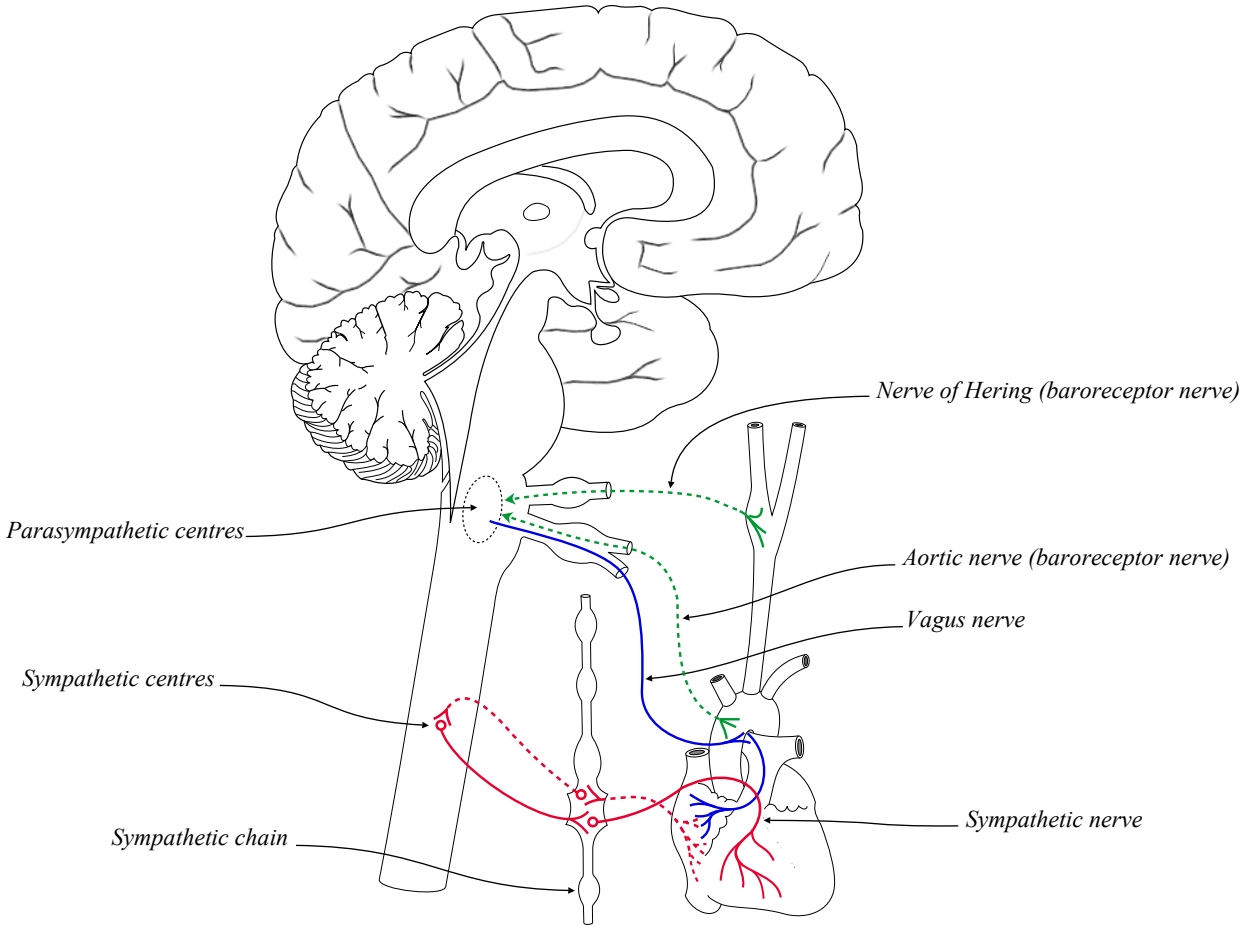


The cardiac adaptation to the needs of the organism is ensured by the autonomic nervous system which acts essentially on the rhythm and the force of the cardiac contractions through the sympathetic and parasympathetic divisions described previously.
It is obvious that this adaptation is possible within certain physiological limits and must be coupled with an adaptation of the respiratory rhythm.

- The parasympathetic division, whose chemical mediator is acetylcholine, acts on the nodal tissue as well as on the myocardial cells. It increases the membrane permeability of the cells of the sinoatrial node, so inhibits the depolarization wave and therefore the muscle contraction. As a result:
  - It slows down the heart by lengthening the diastole.
  - It decreases the power of the atrial systole.
  - It lengthens the atrioventricular conduction time.
  - It weakens the tone of the myocardium.

  The parasympathetic division slows the spontaneous heart rhythm from 120-130/min for an isolated heart to 60-80 (or slower).
  The parasympathetic regulatory centres of the heart are located in the brainstem. Their motor nerve is the vagus nerve. This nerve has fibers that branch out to almost all heart tissues.
- The sympathetic system, whose chemical mediator is norepinephrine *(also called noradrenaline)* leads to an acceleration of the depolarization of the membrane of the cells of the sinoatrial node, which leads to an acceleration of contractions. As a result:
  - It accelerates the heart by shortening its diastole.
  - It increases the power of the atrial systole.
  - It shortens the atrioventricular conduction time.
  - It strengthens the tone of the myocardium.

  Sympathetic centres are located in the spinal cord at the level of the lower cervical spine and upper dorsal. Their motor nerves are the sympathetic nerves that leave the spinal chord at each vertebra and connect in the heart.
  Sympathetic nerves constantly impose an acceleration to the heart rhythm. However, this acceleration is constantly reduced by the actions of the parasympathetic system.
- The parasympathetic and sympathetic centres are also connected to indicators of blood pressure (baroreceptors) located in the aortic arch and at the bifurcation of the carotid.
  - A drop in blood pressure detected by the baroreceptors results in the action of the parasympathetic division being reduced and leads to an acceleration of the heart rate.
  - At the opposite, a rise in blood pressure results in a cardiac slowdown.



Also, as described previously, the sympathetic and parasympathetic divisions control the blood pressure in the circulatory system by the vasodilatation and vasoconstriction of the blood vessels.

- Vasodilation refers to the widening of blood vessels resulting from the relaxation of the muscles of the vessels walls. When the blood vessels dilate, the flow of blood is increased due to a decreased vascular resistance. Therefore, the blood pressure decreases.
- Vasoconstriction is the narrowing of the blood vessels resulting from the contraction of the muscular walls of the vessels, in particular, the large arteries and small arterioles. When the blood vessels constrict, the flow of blood is restricted or decreased, thus retaining body heat or increasing vascular resistance.

Respiratory system

- The Nasal cavity

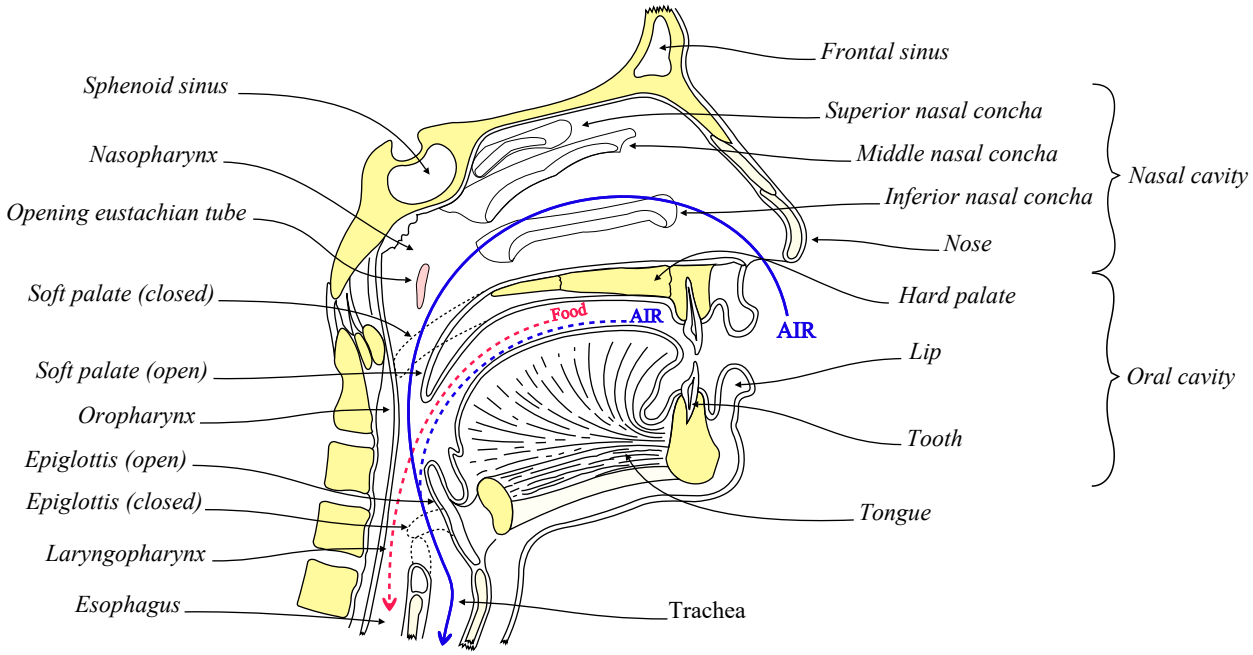
The nasal cavity plays a role in filtration and humidification of the breathed air.

- Hairs and mucus lining the nasal cavity trap dust and other environmental contaminants before they reach the trachea.
- Humidification of the air is provided by the mucous membrane, which is kept moisten by the lachrymal glands. Several scroll-shaped thin bony elements, covered by the mucous membrane called “Superior nasal conchae”, “middle nasal conchae”, and “inferior nasal conchae” *(Conchae = plural of concha)*, that project from the wall of the nasal cavity create a turbulence of the air breathed in, increasing its contact with the mucous membrane.
- Note that the opening of the “Eustachian tubes” are at the end of the nasal cavity (nasopharynx). The Eustachian tubes which are also called “auditory tubes” or “pharyngotympanic tubes”, link the nasopharynx to the middle ears. they adjust the pressure of the air within the middle ears to that of ambient air.
- Also, as for the eustachian tubes, the sinus openings are arranged in the nasal cavity.
  - The openings of the sphenoid sinuses are situated above the superior nasal conchae.
  - Posterior ethmoidal sinuses are linked beneath the superior conchae.
  - The frontal, anterior ethmoidal, maxillary and middle ethmoidal sinuses are linked beneath the middle nasal conchae.
- In addition to the sinuses, the openings of the nasolacrimal ducts arrive beneath the inferior nasal conchae.

- The oral cavity and the pharynx

The pharynx is the part of the throat that is behind the mouth and nasal cavity and above the esophagus and the larynx. It

is the aero-digestive junction of the digestive tract and the tracheobronchial tree, also called respiratory tree, which is connected to the lungs. It is composed of the “Oropharynx” which is the part at mouth level, and the “Laryngopharynx” which is at throat level.
Except when eating, the respiratory tree is open, as breathing is the priority. When food or saliva is swallowed, the soft palate rises and closes the communication between the oropharynx and the nasal cavity. The epiglottis, a flap of elastic cartilage that acts as a switch between the trachea and the esophagus, closes the trachea and prevents the food from entering the airways. These complex mechanisms are triggered by the contact of the alimentary bolus with the mucous membrane of the soft palate, the uvula and the walls of the Laryngopharynx. The nerves coordinating the swallowing are located in the medulla oblongata.
However, local anesthesia of the mucus membrane prevents normal swallowing mechanism, and the food can enter into the nasal cavity and the trachea. In the case of entry of food particles into the respiratory tract, a discharge reflex triggering coughing and vomiting usually happens to evacuate the foreign body. Nevertheless, if the foreign body is not removed, it can trigger death by asphyxiation, or, if its volume is sufficiently small to follow the respiratory tract without being ejected, it will be the cause of infectious complications.
Note that,the opposite of the air breathed through the nasal cavity, the air breathed through the mouth is not filtrated and moistened, which quickly gives a sensation of dryness, and exposes the airways to potential infection.



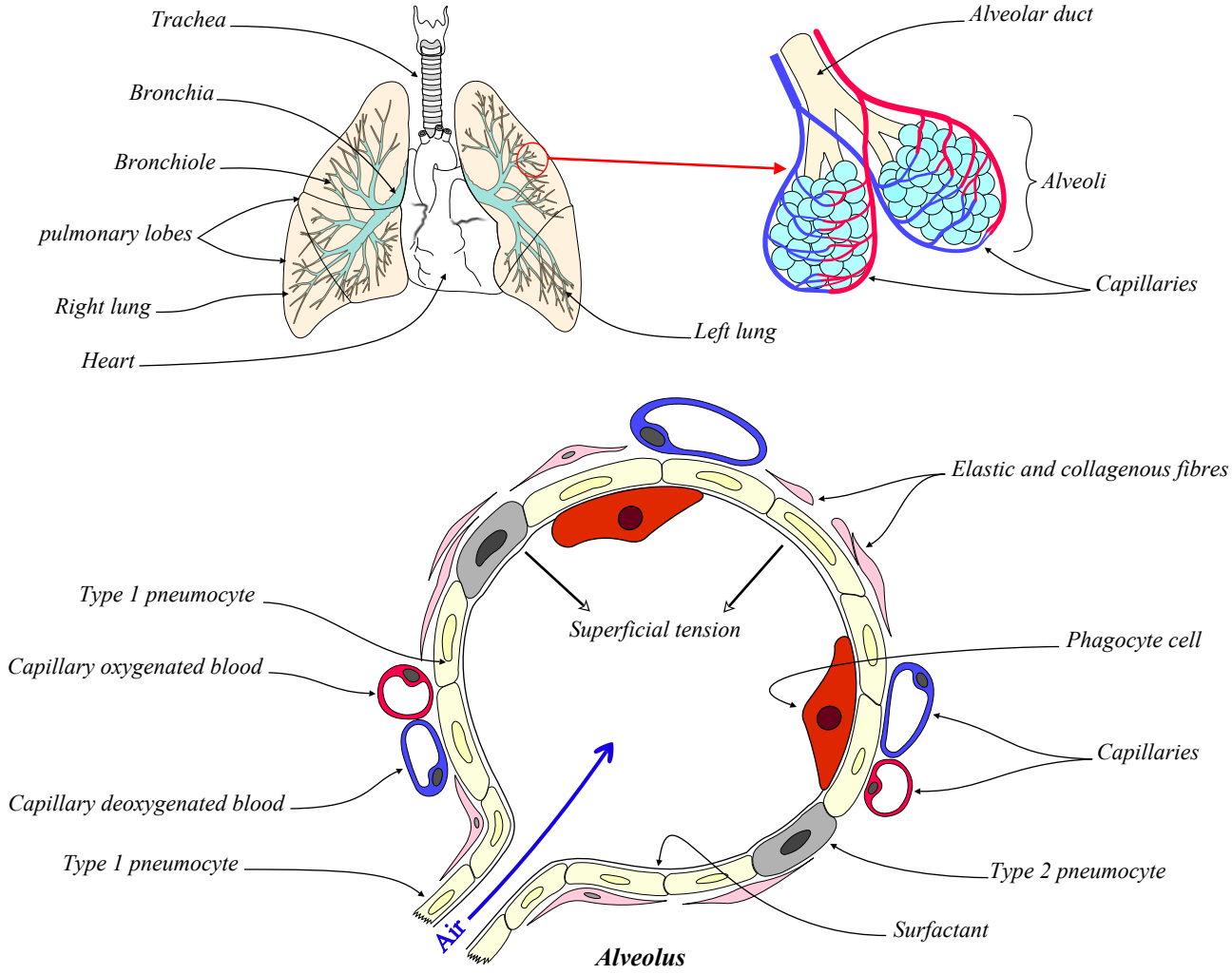
- ***The trachea, bronchi, and bronchioles***

The trachea, also called windpipe, is a pipe approximately 20 cm long and 20 mm diameter, composed of sixteen to twenty cartilaginous rings. The back part of each ring is made of muscle and a moist connective tissue called mucosa which lines the inside of the trachea. The mucosa produces a mucus that traps dust and other contaminants and prevents them from reaching the lungs. The cilia at the surface of the mucosa cells move this mucus toward the pharynx where it is swallowed and digested in the gastrointestinal tract.
The tracheal bifurcation is done by a cartilage shaped in Y at the level of the bronchial strains.
The left and right bronchi run into each lung before branching off into smaller secondary bronchi that carry air into the lobes of the lungs these bronchi split in smaller bronchi, then in bronchioles that guide the air to the alveoli.
The bronchi and bronchioles are provided with rings of circular muscles that permanently contract or relax to adjust the size of the bronchi and, therefore, the amount of air breathed. These movements are controlled by the autonomic nervous system which continues its action when we are sleeping or in the case of a coma. The sympathetic division, which uses a neurotransmitter called adrenaline, dilates the bronchi. The parasympathetic division, which uses a neurotransmitter called acetylcholine, has an antagonistic action.

- ***The lungs***

The lungs are the zones of exchange between the inhaled external air and the blood carried by the pulmonary vessels. They are situated within the thoracic cavity of the chest.
The right lung is bigger than the left which shares space in the chest with the heart. The lung situated on the right-hand side has three lobes instead of two for the lung situated on the left.
The pulmonary alveolus represents the basic unit of the lungs where the exchange is performed. Each lung is composed of approximately 400 million pulmonary alveoli, grouped in clusters in the pulmonary lobes. The cumulated surface of alveoli gives approximately fifty square meters of exchange surface per lung which varies according to the distention of the alveoli.
The structure of an alveolus consists at 90 - 95% of a thin coreless cytoplasmic membrane made of type 1 pneumocytes (cells). This membrane is permeable and enables gas exchange between the air in the alveolus and the blood. Note that type 1 pneumocytes cannot replicate themselves.

The alveolus is also lined with type 2 pneumocytes which are cells that secrete a substance composed of proteins and lipids called “surfactant”. This substance reduces the superficial tension of pulmonary fluids and contributes to the elastic properties of the lungs. Note that type 2 pneumocytes can replicate and replace damaged type 1 pneumocytes.
A few nucleated phagocyte cells that eliminate the dust and the various inhaled particles are disseminated on the internal side of the alveolus.
Numerous capillaries are running on the outer surface of the alveolus. Also, elastic and collagenous fibres that help to maintain the shape of the alveolus are weaving between the capillaries. The elastic fibers give the membrane its elasticity and the collagenous fibers its rigidity.

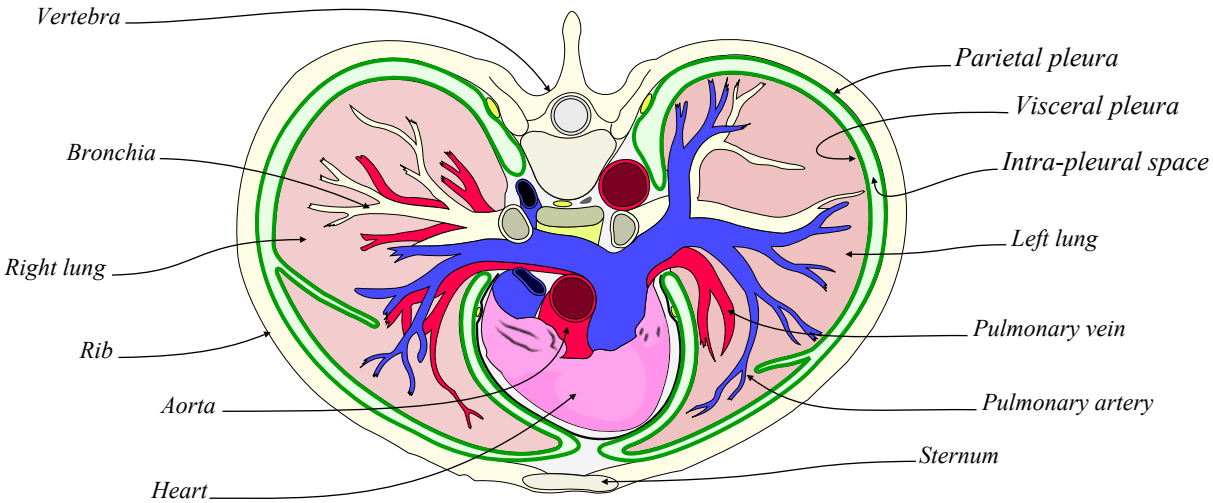


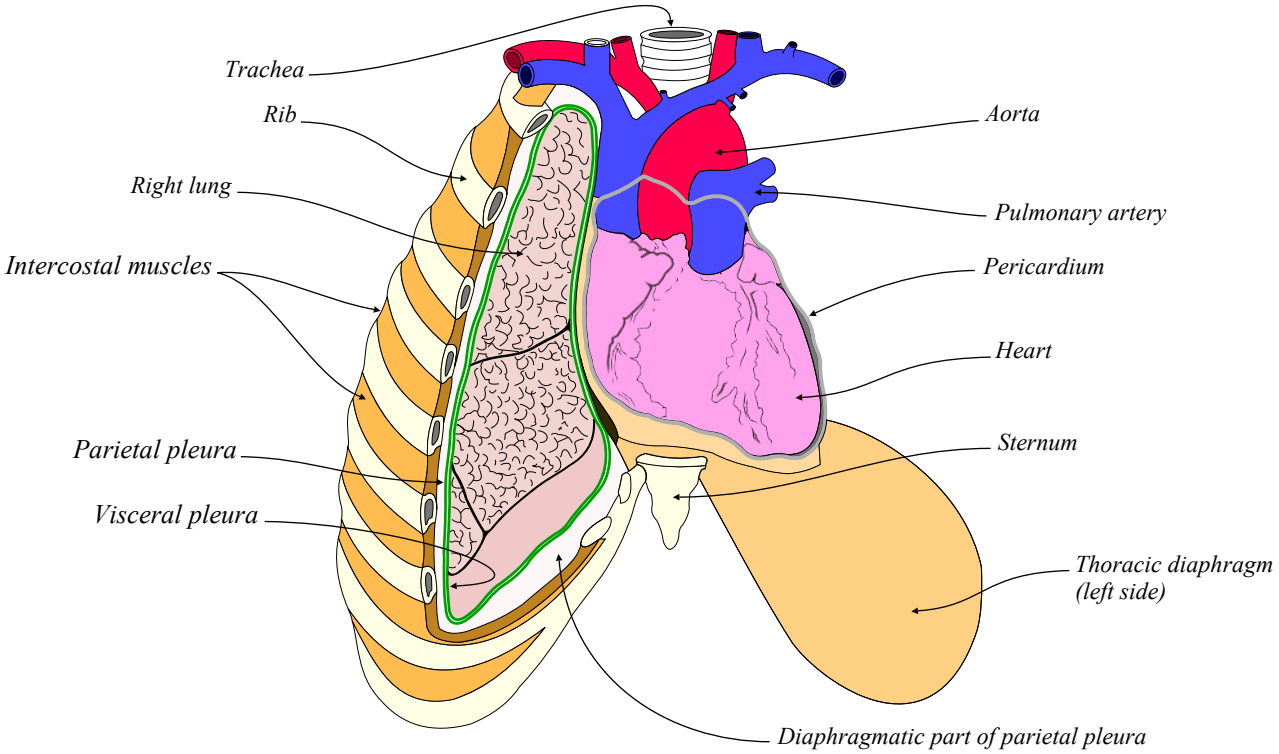
The elasticity of the lungs has for effect that they naturally retract on themselves. As a result, if the atmospheric pressure would be exerted inside and outside the lungs, they would only follow their natural inclination to collapse, and respiration would be impossible.
For this reason, the lungs are separated from the walls of the rib cage by the pleurae, which form a sealed space with an internal pressure below the atmospheric pressure *(negative pressure)*, perfectly surrounding the outside of the lungs. The outer membrane of this bag, called “parietal pleura”, adheres to the inner walls of the thoracic cavity, and the inner membrane, called “visceral pleura”, adheres to the outer surfaces of the lungs, which results in the following situation:

- The atmospheric pressure exerts on the thorax.
- The atmospheric pressure also exerts inside the pulmonary alveoli which are connected externally by the respiratory tracts.
- The negative intra-pleural pressure allows the lungs to inflate and to expand in the entire thoracic volume. Also, it enables the perfect transmission of movements from the rib cage to the lungs and vice versa. That makes possible the mechanisms of inspiration and expiration:
    - Inspiration
        1. The volume of the rib cage increases due to the action of the respiratory muscles *(see their description in the next paragraph)*.
        2. The leaflets of the parietal pleura adhering to the rib cage walls follow this thoracic expansion.
        3. The internal pressure is higher in the lungs than in the intra-pleural space surrounding them. As a result, the lungs expand until contact with the chest walls. This expansion increases their volume, which causes the suction of the outside air.
    - Expiration
        1. The cessation of the action of the respiratory muscles causes the reduction of the volume of the ribcage by a spring-back effect due to the crushing effect of the atmospheric pressure and also

because the lungs exercise their natural retractive force which applies through the pleural cavity to the thoracic cage itself. Indeed, the retractive force of the lungs obviously applies to the visceral pleura which adheres to them. As a result, two different pressures are exerted on both sides of the wall of the thoracic cavity:
   - The atmospheric pressure outside.
   - The intra-pleural negative pressure inside.
2. Therefore, the thoracic cage decreases in volume until its limit of elasticity is reached. During their retraction, the lungs reject the air inhaled during the inspiration phase.

Note that a chest wound penetrating the pleura and thereby equalizing the intra-pleural pressure with atmospheric pressure causes the immediate collapse of the lung on the injured side (pneumothorax).



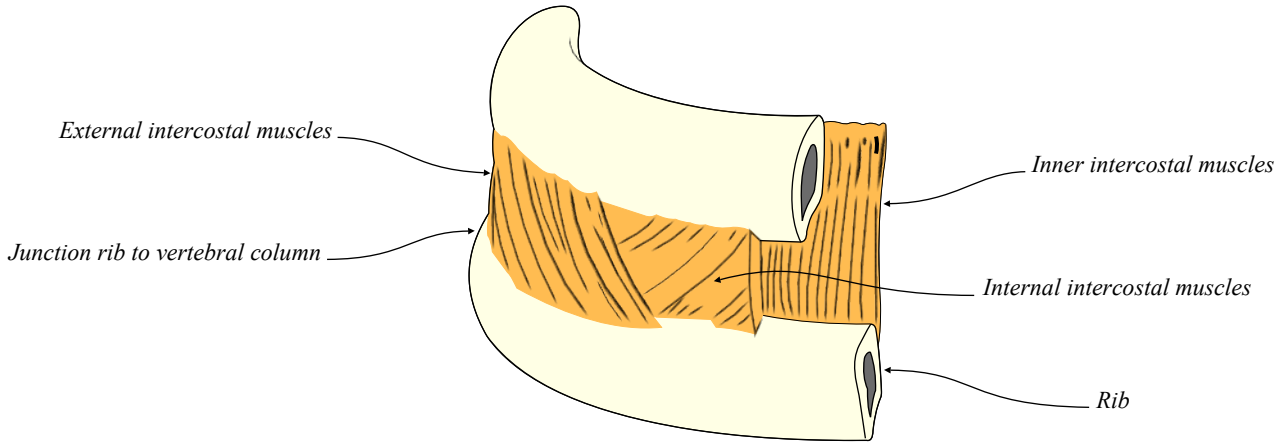


The rib cage is a semi-rigid bone cage which dimensions can vary as the consequence of the articulation of the ribs and sternum on the vertebral column. The rib cage is moved by the respiratory muscles, mainly composed of the diaphragm and the intercostal muscles.

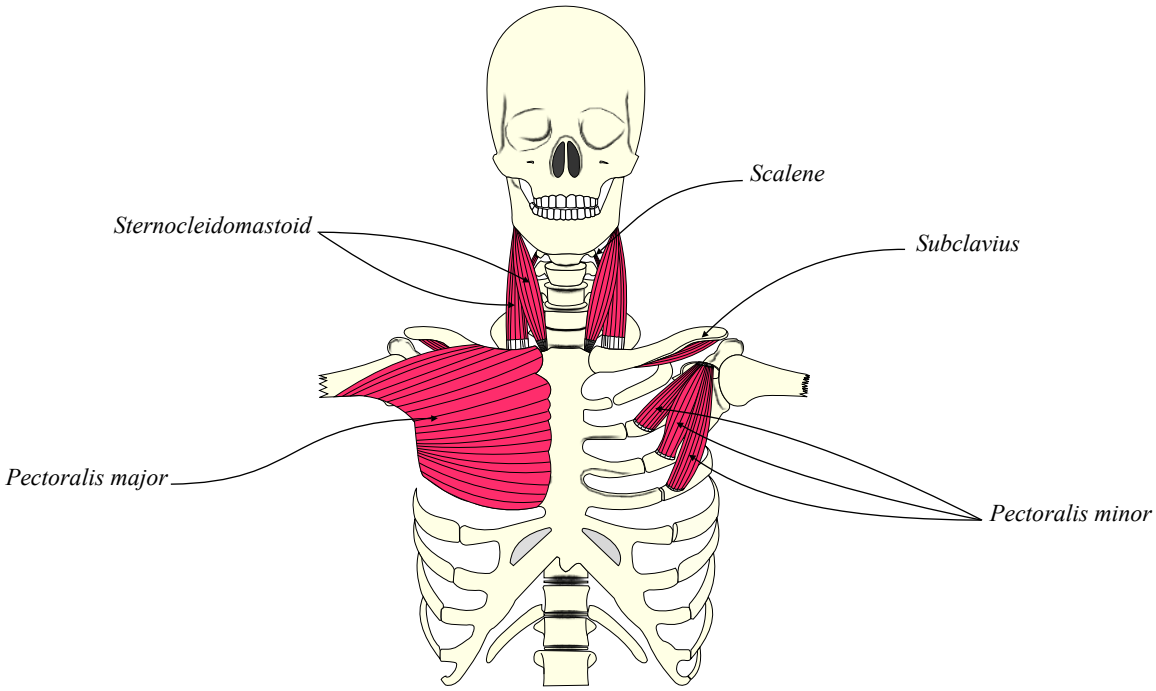
- The diaphragm is a thin, dome-shaped muscular membrane that separates the abdominal cavity from the thoracic cavity. It protrudes inside the thoracic volume, diminishing it accordingly. The contraction of this membrane erases its convexity, which increases the volume of the thoracic cage.
- The intercostal muscles are attached between the ribs and are arranged in three layers.
  - The external layer is arranged in diagonal and is the most involved in respiration. The contraction of these muscles horizontalizes the sides of the rib cage which has in effect the increasing of its volume.
  - The internal intercostal muscles are arranged diagonally in opposite path to the external layer. They are involved in the expiration where as a result of their contraction, the ribs bend inward and decrease the

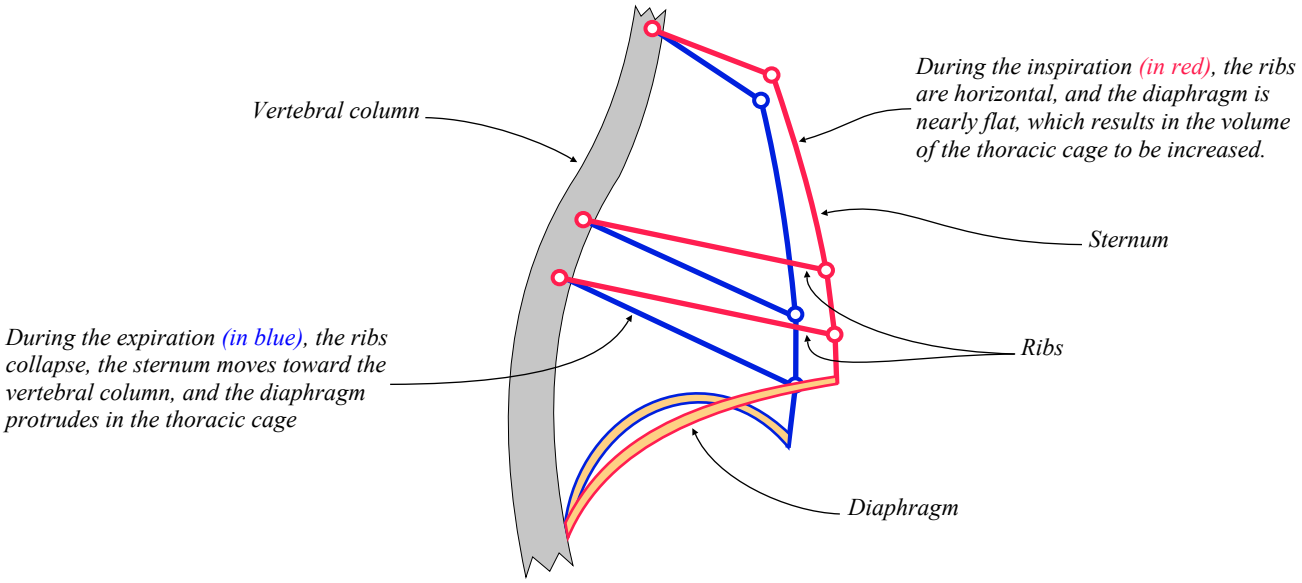
volume of the thoracic cage.

- The inner intercostal muscles are arranged vertically. Note that the diaphragm is the continuation of these muscles.



- Forced inspiration may be necessary. In this case, subclavius, scalene, sternocleidomastoid, and pectoralis are muscles whose contraction provides traction on ribs and move them closer to the horizontal may be involved. The additional muscles that are involved in forced expiration are the anterior abdominal wall.



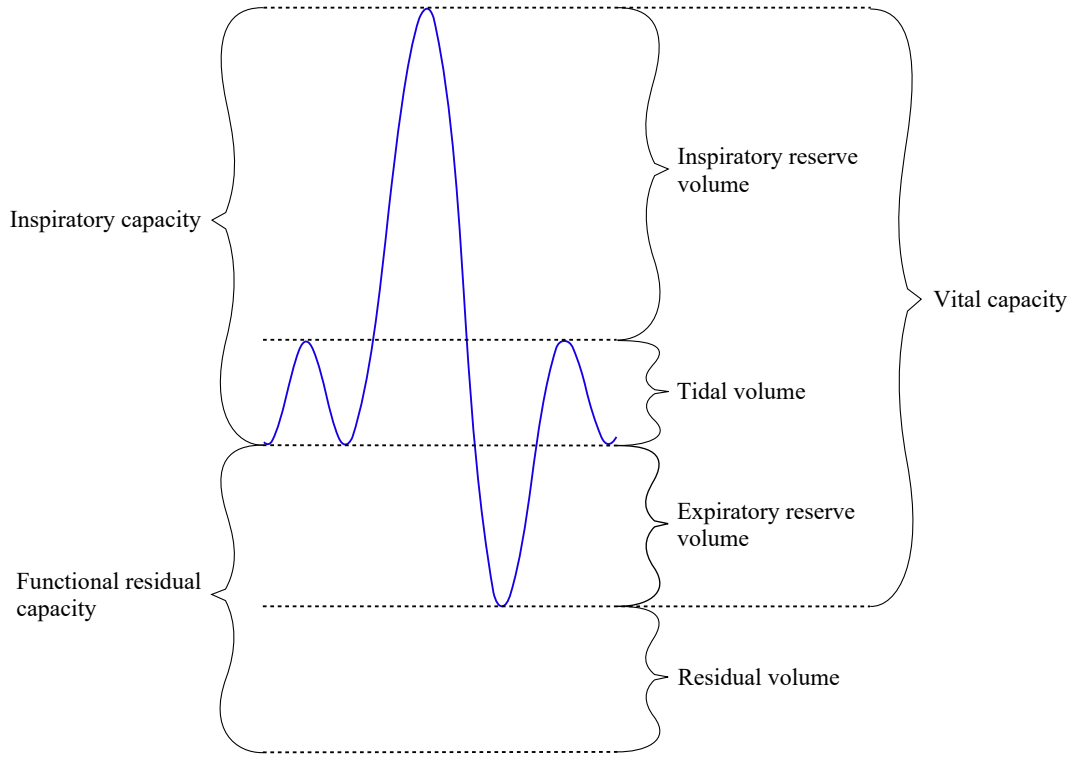


The purpose of the respiratory movements are for the periodic renewal of the air contained in the pulmonary alveoli.
During a normal inspiration followed by a normal expiration, a certain volume of air is inhaled and then exhaled, this volume is called “tidal volume”.
If forcibly inhaled, the additional volume of air is called “inspiratory reserve volume”
If forced expiration is performed, the volume of exhaled air in addition to the tidal volume is called “expiratory reserve volume”.
The air which remains in the lungs at the end of a forced exhalation is called the “residual volume”.
The average figures for the normal adult man are as follows:

- Tidal volume, 0.5 litres
- Inspiratory reserve: 3 litres
- Expiratory reserve: 1.3 litres
- Residual volume: 1.3 litres

Studies of these volumes allow appreciating the ventilatory capacities of a patient. Note that they will vary according to the sex, height, and age.

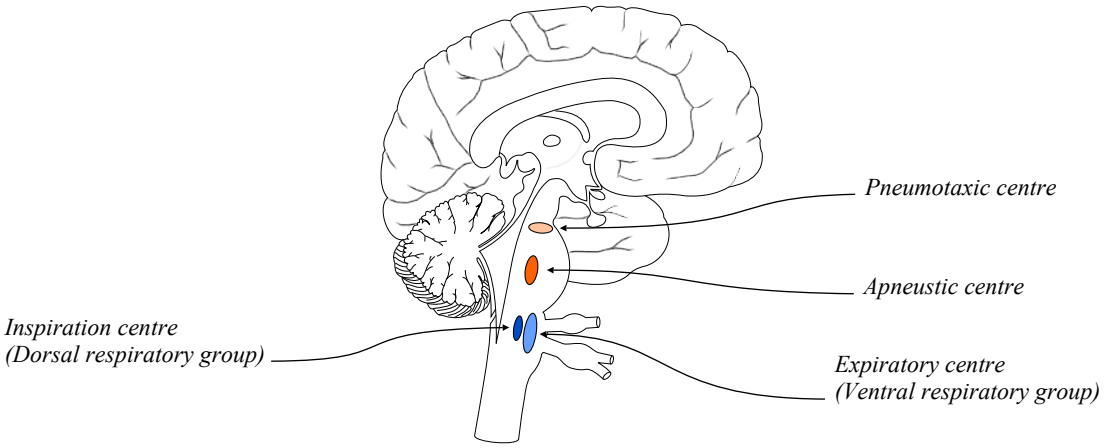
- “Inspiratory capacity” is the tidal volume + the inspiratory reserve volume (= 3.5 litres).
- “Functional residual capacity” is the expiratory reserve volume + the residual volume (= 2.6 litres).
- “The vital capacity” that is commonly checked during medical checkups is the addition of the tidal volume + inspiratory capacity + expiratory reserve volume (= 4.8 litres).



Due to the residual volume and also the volume of air contained in the airways, there is only a partial renewal of the air of the lungs.
At rest, the ventilatory flow rate depends on the tidal volume and the breathing rate, which is the number of inspirations and expirations that take place in one minute under normal conditions. As the average number of breathing movements at rest is 16 per minute and the tidal volume is 0.5 litres, we have a ventilation rate of 8 litres/min.
The ventilation flow can vary with the lung capacity and the frequency. However, note that the acceleration of the frequency may result quickly in breathlessness (fast but short and superficial breathing). Lung capacity can be expanded through flexibility exercises, breathing exercises, and physical activity.

Hematosis *(conversion of venous blood to arterial blood by oxygenation)* and pulmonary ventilation are constantly adapted to the needs of the organism by the respiratory centres which are located in the medulla oblongata and the pons. However, note that the vegetative functions that are controlled by the autonomic nervous system can be modified by the will (which is not the case for other vegetative functions).

- The inspiratory centre is located within the dorsum of the medulla.
- The apneustic centre and the pneumotaxic centre coordinate in the involuntary control of respiration:
  - The pneumotaxic center, located in the upper portion of the pons, provides inhibitory impulses on inspiration and thereby prevents overdistension of the lungs and helps to maintain alternately recurrent inspiration and expiration.
  - Apneustic centre in the lower pons inhibit the signal from the pneumotaxic centre and prevent inhalation neurons from being switched off. Thus, it promotes inhalation.
- Expiratory Centre is located in the ventrum of the medulla

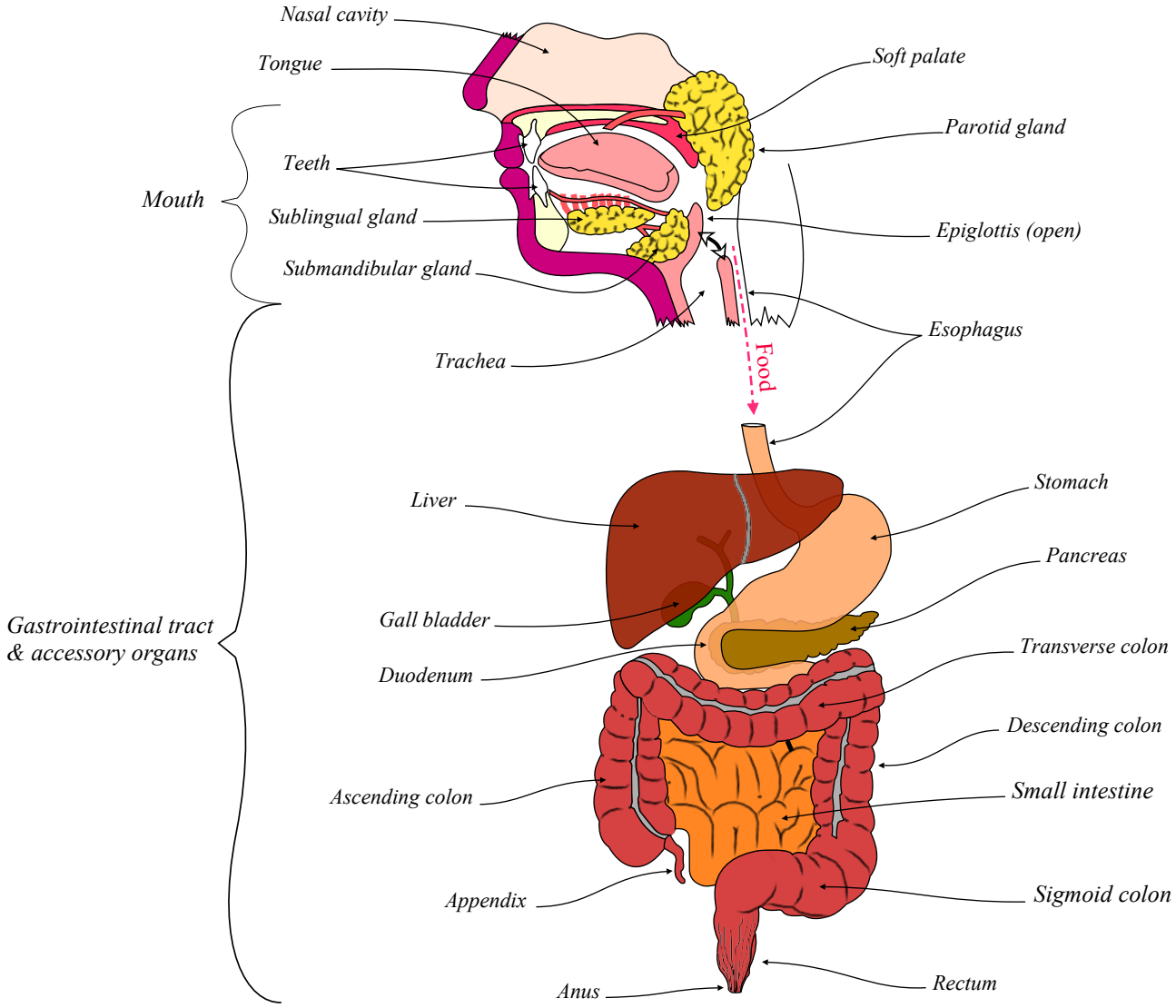


The respiratory centres receive input from chemoreceptors, mechanoreceptors, the cerebral cortex, and the hypothalamus to regulate the rate and depth of breathing. Input is stimulated by altered levels of oxygen, carbon dioxide, pH, hormonal changes, anxiety from the hypothalamus, and also by signals from the cerebral cortex that allow a conscious control of respiration.

Digestive and urinary systems

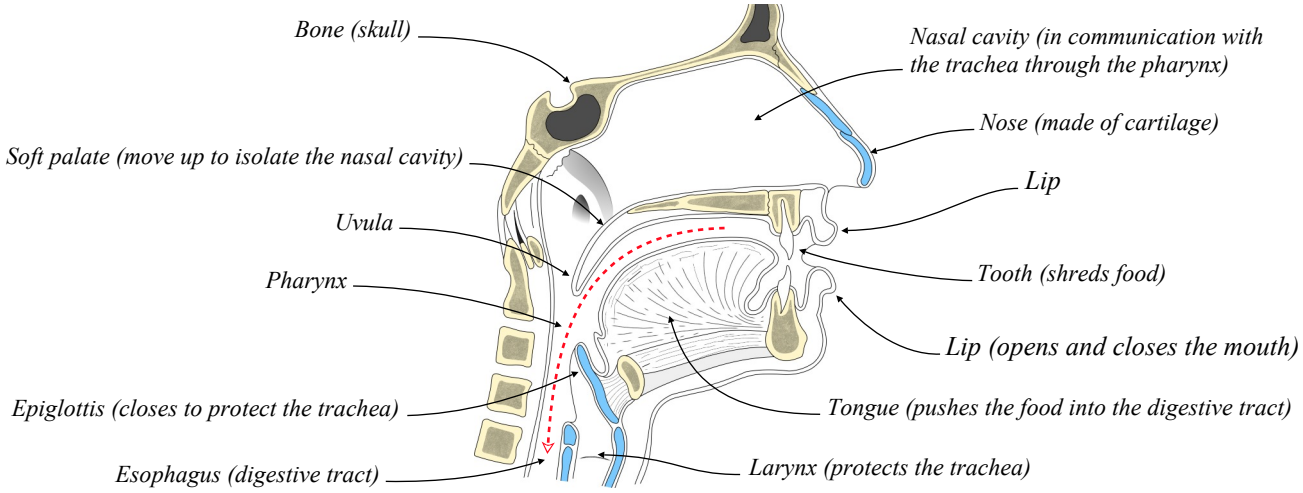
Digestive system

The digestive system is the group of organs working together to convert the food into energy and basic nutrients. It consists of the organs of the mouth (tongue, salivary glands), the gastrointestinal tract (esophagus, stomach, intestines), and the accessory organs (pancreas, the liver, and the gallbladder).



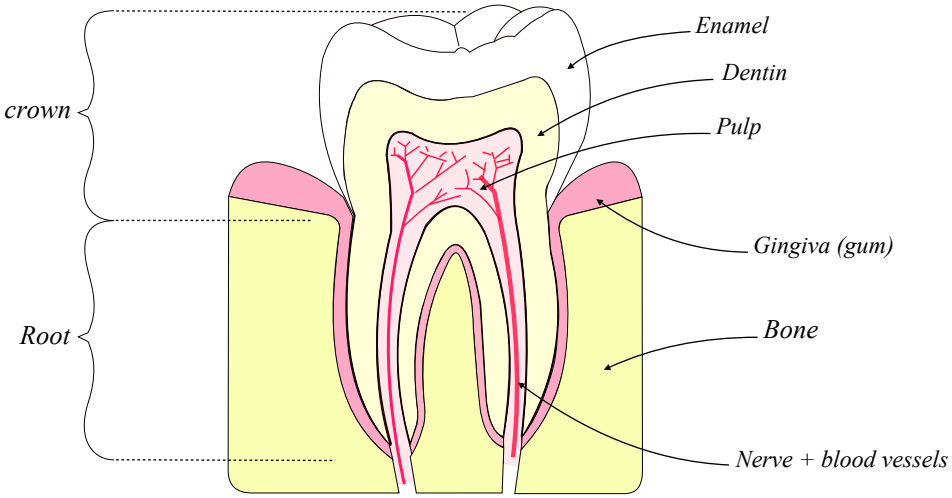
- ***The mouth***

It is the place where the food is broken down into smaller components, that are then sent into the esophagus.



Organs of the mouth involved in the process of digestion which are the tongue, the teeth, and the salivary glands are classified accessory organs.

- There are thirty two teeth that are made of a bone-like substance called dentin and covered in a layer of enamel. They contain blood vessels and nerves under the dentin in a soft region known as the pulp. Gingiva, also called the gums, are the soft tissue that covers and protects the roots of the teeth.



- The tongue, which is located on the inferior portion of the mouth *(see scheme previous page)*, is made up of several pairs of muscles covered by a thin, bumpy, skin-like layer. The outside of the tongue contains many rough papillae for gripping food. The taste buds on the surface of the tongue detect taste molecules in food and connect to nerves in the tongue to send taste information to the brain. The tongue also helps to push food toward the posterior part of the mouth for swallowing.
- Three sets of salivary glands produce a secretion called saliva that helps to moisten food and begins the digestion of carbohydrates. Also, saliva lubricates the food as it passes through the mouth, pharynx, and esophagus. The sublingual glands are under the tongue. The parotid glands, which are the largest glands, are situated on either side of the mouth and in front of the ears. The submandibular glands are located beneath the floor of the mouth. Note that saliva reduces the quantity of pathogen agents in the mouth and protects the teeth and tissues from excessive acidity.
- The pharynx, also called throat, connects the posterior end of the mouth to the esophagus, where the masticated food is sent. Also, its other function is to connect the nasal cavities with the larynx and then the trachea and the lungs. For this reason, it contains a valve made of soft tissue called epiglottis that closes when the food routes to the esophagus and opens when air passes to the larynx.
- Above the tongue, there is the hard palate, which is a thin horizontal bony plate that is lined with soft tissue. It is continued by a muscular extension called soft palate *(see scheme previous page)* which isolates the nasal cavity from the pharynx when the nutrients are sent to the esophagus.

- ***The esophagus***

It is a muscular tube that carries the food from the pharynx to the stomach. It is terminated by a muscular ring called the lower esophageal sphincter or cardiac sphincter which function is to close its end and trap the food in the stomach.

- ***The stomach***

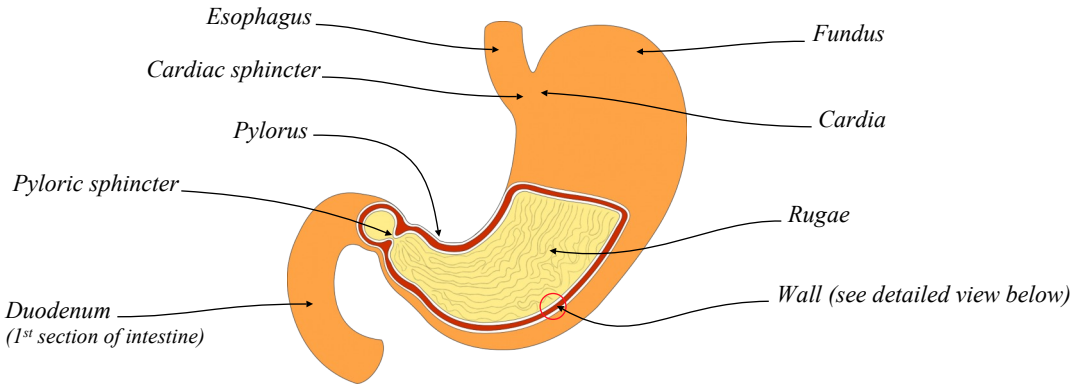
It is a crescent-shaped enlargement of the gastrointestinal tract situated below the diaphragm on the left side of the abdominal cavity and is between the esophagus and the small intestine. It can be divided as follows:

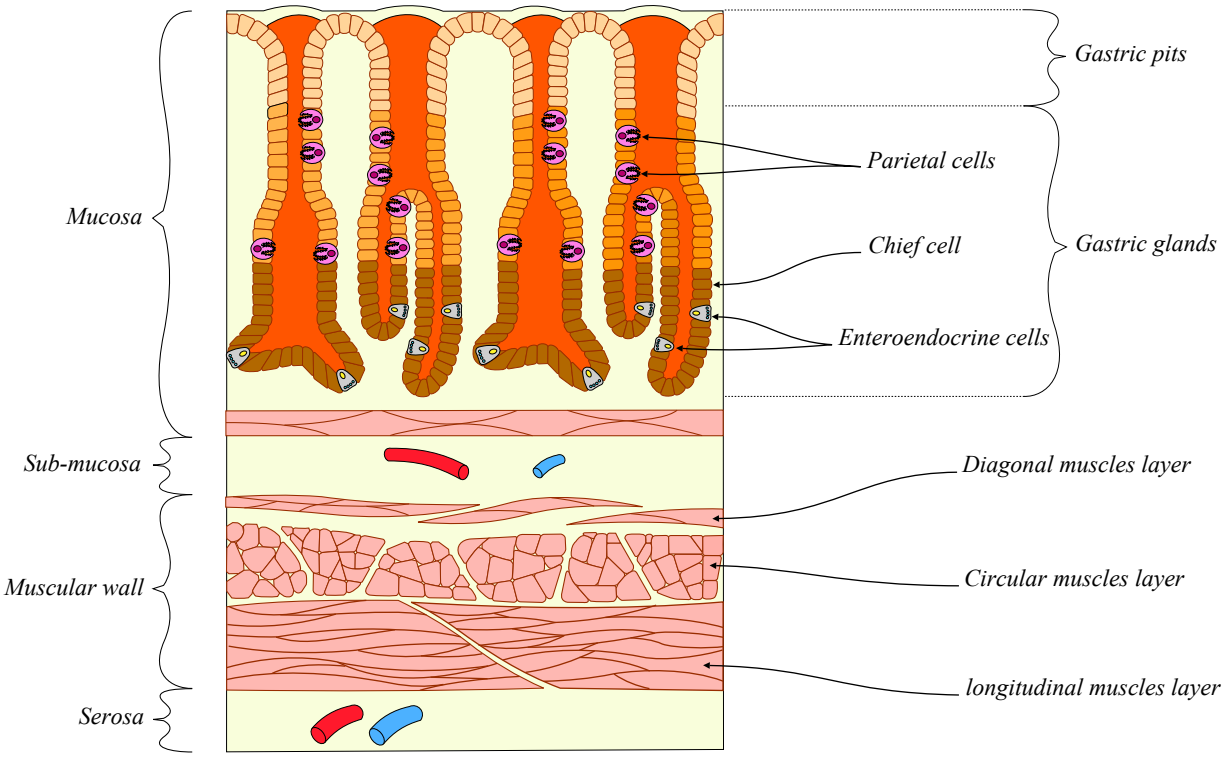
- The cardia that is the region where the esophagus connects. It is a narrow, tube that is opened and closed by the cardiac sphincter. It opens up into the wider regions of the stomach.
- A dome shape called “fundus” is above the cardia.
- At the bottom of the stomach, there is a funnel shaped region called pylorus that connects the stomach to the duodenum and contains the pyloric sphincter. The pyloric sphincter controls the flow of partially digested food *(which is called chyme)* out of the stomach and into the duodenum.

The walls of the stomach are composed of four layers:

- The “mucosa”, is the innermost layer and contains gastric glands that secrete the digestive juices. It is covered by a layer of column-shaped (*columnar*) epithelial tissue.
- The “sub-mucosa” is made of dense connective tissues and has blood vessels, lymphatic vessels, and nerves. It is between the mucosa and the muscular wall
- The “muscular wall” is made of inner diagonal, circular, and outer longitudinal smooth muscles which contracts and relaxes to help decompose the food and propel it forward.
- The “serosa”, which is the outermost layer, is an epithelial layer and connective tissue that connects to the surrounding organs.

The mucosa and the sub-mucosa are present as folds termed “rugae” that allow the stomach to stretch for accommodating large meals and help to grip and move the food during digestion. When the stomach is distended with food, the rugae are flattened out and appear smooth. When the stomach is empty or almost empty, its mucosa contracts by forming folds. It was previously thought that stomach contractions in the absence of food were the cause of the feeling of hunger. It is now known that it is mainly due to the decrease in blood glucose. However, the contractions and gurgling of the stomach can often be felt as precursors to the feeling of hunger.





The stomach produces and secretes several substances to continue the process of digestion. These substances are produced by the exocrine or endocrine cells situated in the mucosa.

- Exocrine cells product the gastric juice that contributes to digest the food and is mainly composed of:
  - Hydrochloric acid which denature the proteins *(digestion)* and kills pathogenic bacteria. It is secreted by the parietal cells.
  - A glycoprotein, called “Intrinsic factor”, that binds to the vitamin B12 in the stomach and allows it to be absorbed in the small intestine is also secreted by the parietal cells. Note that B12 vitamin has a role in the synthesizing of myelin (that protects the axons of neurons) and the formation of red blood cells.
  - Digestive enzymes such as “pepsin” which breaks proteins into amino acids, and “gastric lipase” which digest fats. They are produced by the “chief cells”
- Specialized exocrine mucous cells secrete a mucus into the lumen of the stomach and into the gastric pits. This mucus which is rich in bicarbonate ions that neutralizes acid, spreads across the surface of the mucosa and coat the lining of the stomach with a thick, acid and enzyme-resistant barrier.
- G cells are endocrine cells that secrete in the blood a hormone called “gastrin”. Stimulation of this hormone leads to increased secretion of gastric juice, stronger contractions of the stomach, and the opening of the pyloric sphincter to move the chyme into the small intestine. Gastrin also binds to receptor cells in the pancreas and gallbladder where it increases the secretion of pancreatic juice and bile.

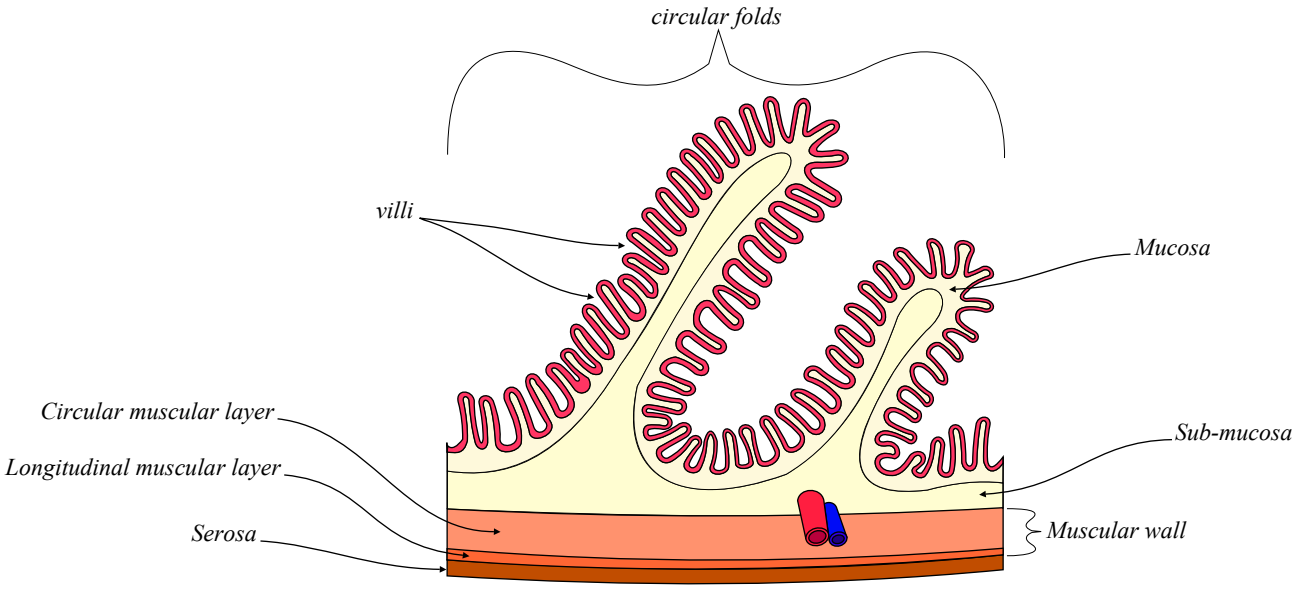
- ***Small intestine***

The small intestine *(also called small bowel)* is between the stomach and the large intestine. It absorbs nutrients and minerals from the chyme, using small finger-like protrusions called “villi” that extend into the lumen and increase the internal surface area of the intestinal walls making available a greater surface area for absorption. The small intestine is composed of three distinct regions:

- The duodenum, which is approximately 20 cm long, receives gastric chyme from the stomach, the bile *(a fluid which aids the digestion of lipids)* from the liver, and digestive enzymes *(juice)* from the pancreas. The digestive enzymes break down proteins and the bile and emulsify fats into micelles. The duodenum contains glands that produce a mucus-rich alkaline secretion containing bicarbonate. These secretions, in combination with bicarbonate from the pancreas, neutralize the stomach acids contained in the chyme.
- The jejunum is the midsection of the small intestine connecting the duodenum to the ileum that contains villi which are arranged in circular folds (also called, plicae circulares). Sugars, amino acids, and fatty acids are absorbed into the bloodstream at this level. This section is approximately 2.5 m long
- The ileum, which walls are covered with villi similar to those of the jejunum, absorbs vitamin B12 and bile acids, as well as any other remaining nutrients. it connects to the cecum of the large intestine at the ileocecal junction.

The walls of the small intestine are very similar to those of the stomach:

- The “mucosa”, is the innermost layer that is covered by villi.
- The “sub-mucosa” is made of dense connective tissues and has blood vessels, lymphatic vessels, and nerves. It is between the mucosa and the muscular wall
- The “muscular wall” is made of inner circular, and outer longitudinal smooth muscles. Thus, there is no diagonal muscles as with the stomach with the small intestine
- The “serosa”, which is the outermost layer, is an epithelial layer and connective tissue that connects to the surrounding organs.

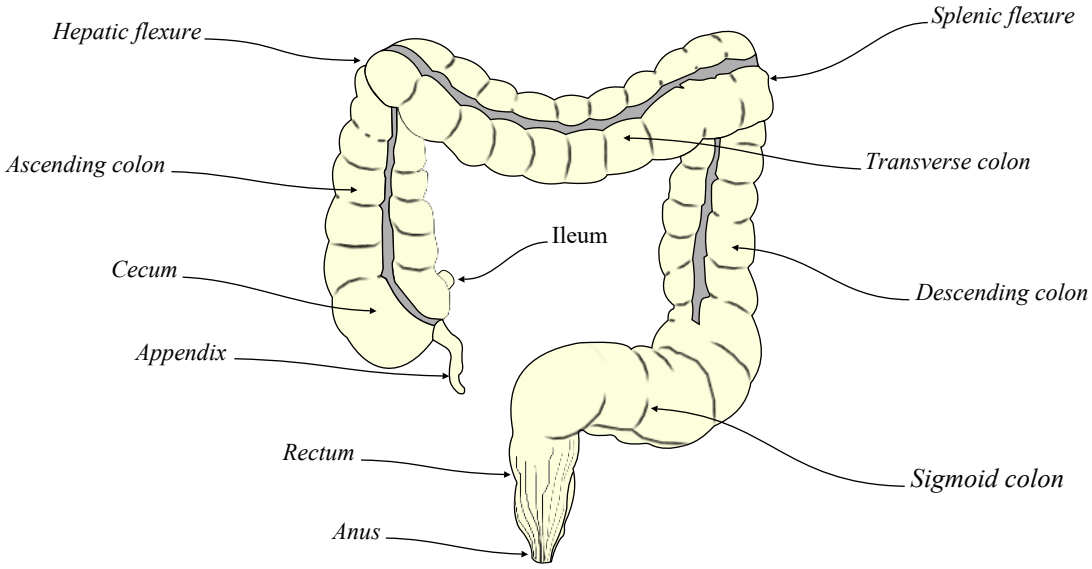


- ***Large intestine***

It is also called “large bowel”, or “colon”, and is approximately 1.5 m long and 6 -7 cm diameter. It is the final section of the gastrointestinal tract which function is to absorb water, salts, and vitamins while converting the chyme previously digested in the small intestine into faeces. It is divided into the following sections:

- The cecum is the first section of the colon that receives chyme from the small intestine. It is separated from the ileum (the final portion of the small intestine) by the ileocecal valve (also called Bauhin valve) which limits the rate of food passage into the cecum and prevents material from returning to the small intestine.
- The appendix which develops from the cecum, is a small closed structure finger-shaped not involved in digestion which function is uncertain; some sources believe that it has a role in housing a sample of the colon's micro-flora. Note that acute inflammation of the appendix is a common abdominal surgical emergency which results of its ablation (removal).
- The ascending colon is the continuation of the cecum. The process of extraction of water, salts, and vitamins starts. The unwanted waste material is moved upwards toward the transverse colon by the action of peristalsis.
- The transverse colon begins at the “hepatic flexure”, which is a sharp bend at the superior end of the ascending colon and moves horizontally to the splenic flexure, which is the sharp bend to the descending colon. It mixes feces by contractions in a process known as segmentation. During this process, bacteria ferment the waste material to release vitamins and a few trace nutrients remaining in the waste. Liquids, nutrients, and vitamins are absorbed through the walls and then used by the tissues of the body. The colon then uses slow longitudinal waves of muscle contraction known as peristalsis to push the feces along its length.

- The descending colon is the continuation of the transverse colon. Its primary function is the storage and accumulation of faeces prior to defecation. However, absorption of liquids and vitamins continues as long as the faeces are stored in it. During defecation, the descending colon helps to propel faeces toward the sigmoid colon and rectum and eventually out of the body by contraction of its smooth muscle tissue.
- The sigmoid colon is the final segment of the colon. It transports faecal matter from the descending colon to the rectum and anus. Faeces are stored in the sigmoid colon until they are ready to be eliminated from the body through the anal canal. During this time, the absorption of liquids, nutrients and vitamins from faeces continues.
- The rectum is the final segment of the large intestine that connects the colon to the anus. It stores faecal matter produced in the colon until it is eliminate through the process of defecation. This process is triggered by the increasing pressure of the faeces which stimulates stretch receptors that send nerve impulses to the brain. It results in a feeling of discomfort and the need to empty the rectum through defecation. Then, the process triggers the relaxation of the smooth muscle of the internal anal sphincter to allow defecation to proceed.



The gastrointestinal canal of the large intestine is made of four tissue layers that have the same names and are similar to those encountered in the small intestine:

- The “mucosa” is made of columnar epithelial tissue similar to those encountered in the small intestine. However, there is no villus in the mucosa of the large intestine which is smooth. A lot of mucous glands secrete mucus into the hollow lumen of the large intestine to lubricate its surface and protect it from rough food particles. Also, this membrane absorbs liquids, nutrients, and vitamins
- As in the small intestine, the “sub-mucosa”, which is between the mucosa and the muscular wall, is made of dense connective tissues and has blood vessels, lymphatic vessels, and nerves.
- The muscular layer that surrounds the sub-mucosa contains a layer of circular muscles surrounded by a layer of longitudinal muscles. It is involved in the segmentation and the peristalsis processes (see above)
- The serosa, which is the outermost layer. is an epithelial tissue which secretes a watery serous fluid that protects the large intestine from friction to the surrounding organs, muscles, and bones.

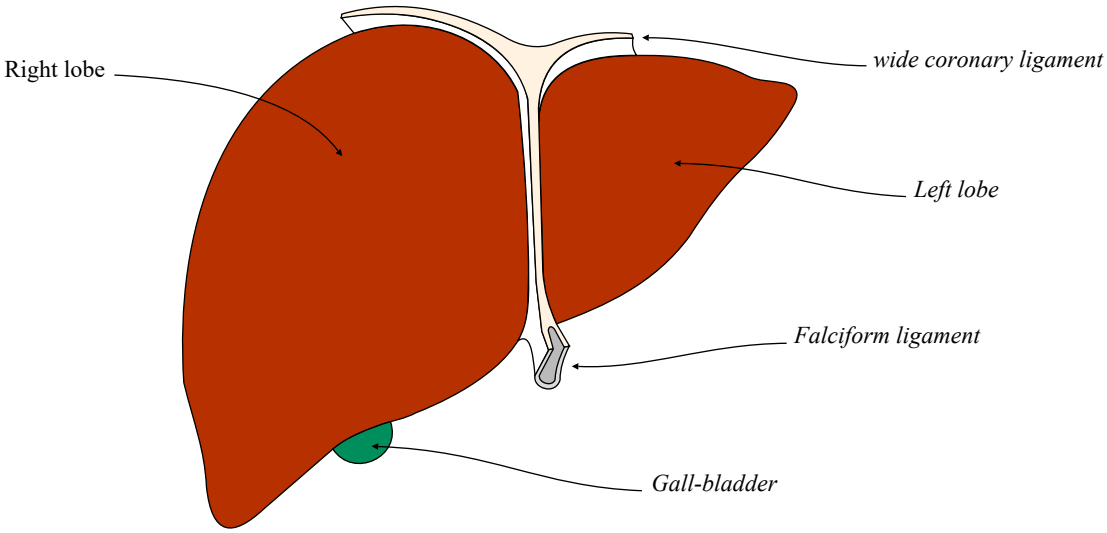
- The liver

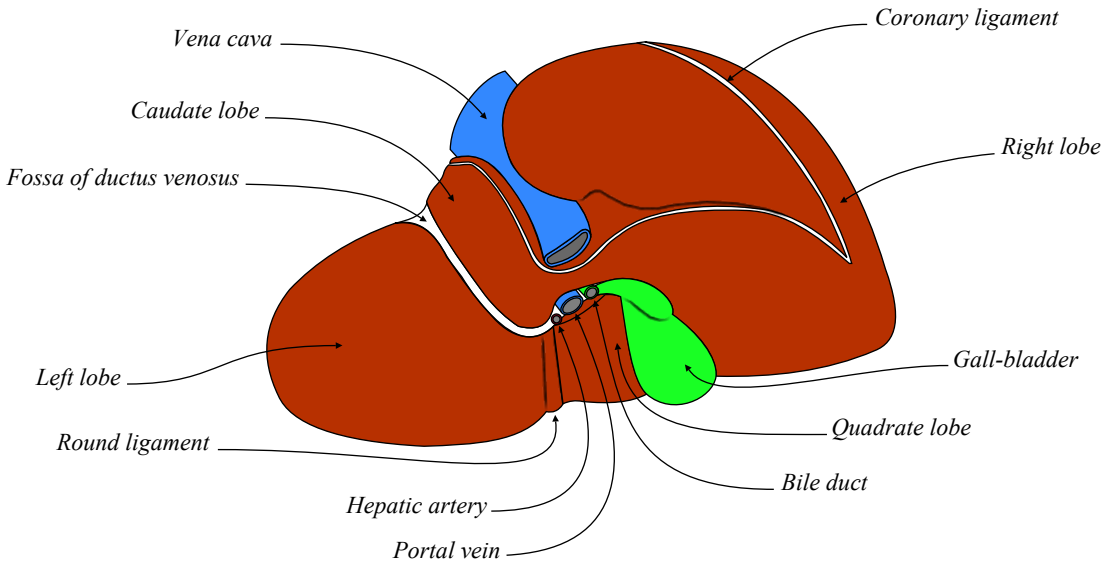
The liver is a vital organ which performs functions related to digestion, metabolism, immunity, and the storage of nutrients within the body. The gallbladder sits under the liver, along with parts of the pancreas and intestines. The liver and these organs work together to digest, absorb, and process food.
The liver is made of soft, brown tissues enveloped by a connective tissue. This tissue is reinforced by the peritoneum which attach it to the diaphragm and the anterior abdominal wall and helps to reduce friction against other organs:

- The “wide coronary ligament” connects the central superior portion of the liver to the diaphragm.
- The left and right “triangular ligaments” connect the superior ends of the liver to the diaphragm.
- The “falciform ligament” attaches the liver to the anterior (ventral) body wall, and separates the left and right lobe of the liver.
- Note that what is called “round ligament” is a remnant of the fetal umbilical vein that has no function when the fetal period is over.

The liver has four lobes that can be seen looking to its back as only two are visible looking to its front.

- The “left and right lobes” are the largest lobes are those visible when looking at the front of the liver. They are separated by the falciform ligament.
- The “caudate lobe” extends from the posterior side of the right lobe and wraps around the inferior vena cava.
- The “quadrate lobe” is below the caudate lobe and extends from the posterior side of the right lobe and wraps around the gallbladder.





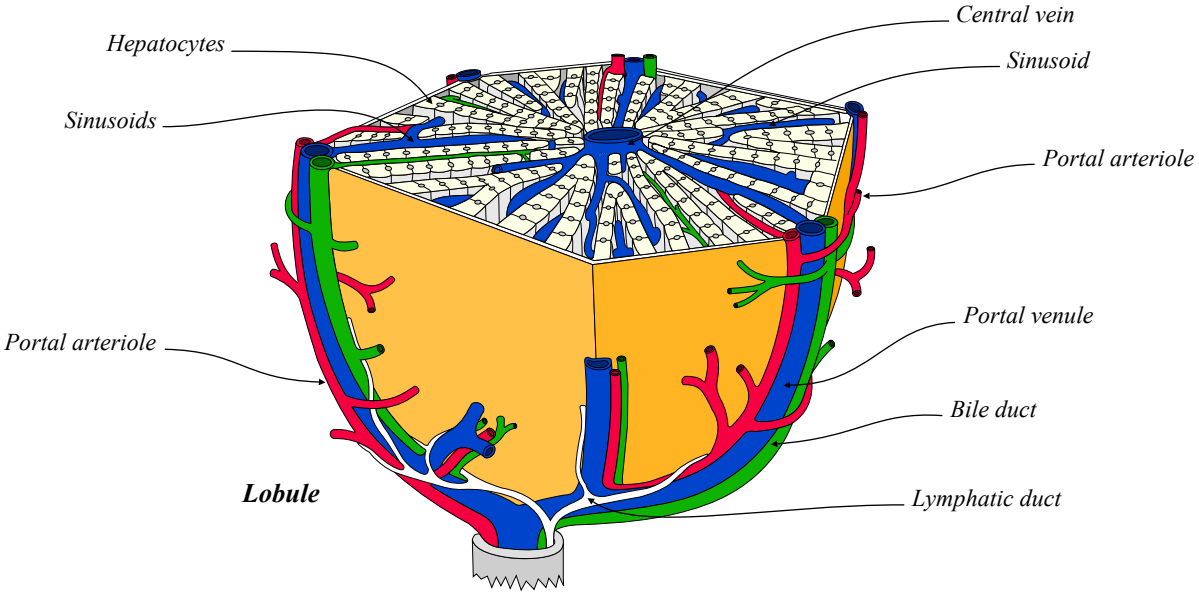
The liver is connected to the “hepatic artery” which carries oxygenated blood from the aorta, and the “portal vein” which carries blood rich in digested nutrients from the gastrointestinal tract, the spleen, and pancreas. These blood vessels subdivide into capillaries which then lead to the lobules, which are the basic metabolic cells.
The lobules, which have hexagonal shapes, are made up of millions of hepatic cells (hepatocytes). They are built around a “central vein” surrounded by six portal venules and six arterioles which are connected to numerous small vessels called "sinusoids" which extend from the little portal veins and arteries to connect to the central vein. Each sinusoid passes through liver tissues containing two main cell types:

- "Kupffer cells" are macrophage cells that capture and break down the old red blood cells passing through the sinusoids.
- "Hepatocytes" are epithelial cells that line the sinusoids and perform functions such as metabolism, storage, digestion, and bile production.

The lobules are held together by a connective tissue, called “Glisson's capsule”, which extends into the structure of the liver and ensheathes the hepatic artery, portal vein, and bile ducts within the liver. Note that there are around 100,000 lobules in the liver.
The blood that has been treated by the hepatic cells is collected into the hepatic veins that lead to the vena cava and returns to the heart. Also, the liver has arteries and arterioles that provide oxygenated blood to its tissues as for another organ.
The bile *(a mixture of water, salts, cholesterol, and the pigment bilirubin)* that is produced by the liver cells is carried to the gallbladder by a branched structure called "biliary tree":

1. The bile drains into microscopic canals called "bile canaliculi" which run parallel to the sinusoids.
2. The numerous bile canaliculi join together into larger bile ducts. These bile ducts which are parallel to the venules and arterioles arranged around the central vein, join to form the left and right hepatic ducts, which carry bile from the left and right lobes of the liver.
3. Those two hepatic ducts join to form the common hepatic duct that drains all the bile away from the liver.
4. The common hepatic duct finally joins with the "cystic duct" from the gallbladder to form the common bile duct, carrying bile to the duodenum of the small intestine.
5. The bile in excess is stored in the gallbladder until it is needed for digestion.

6. The bile is released from the gallbladder into the duodenum. the process is triggered by a hormone called "cholecystokinin" that is sent by the cells of the duodenum when the chyme arrives. The bile acts as a detergent that dissolves fats that are normally not soluble in the water. As a result, fats are broken into small pieces that can be digested by the organism.
   Note that the bile transports the "Bilirubin" to the small intestine. Bilirubin results from the dead red blood cells that have been destroyed by the Kupffer cells and transferred to the hepatocytes where components that can be employed by the body are further transformed and those that cannot be used are transformed in "Bilirubin" and released in the bile and then in the small intestine. Bilirubin, which contains iron, gives their colour to the bile, and following the action of bacteria, to the feces.



The liver intervenes in the food metabolisms. Also, it plays an important role in coagulation and against toxic products.

- Glycogen, also called animal starch, is a substance fabricated and stored in the liver. It is made from food glucose and also lactic acid and certain amino acids. The liver can easily convert this glycogen into glucose *(glycogenolysis)*, which allows the liver to provide for the needs of the body *(as an example, muscle activity)* and to ensure the stability of blood glucose.
- Deamination of amino acids *(separation of the amino radical NH2)* causes the formation of ammonia, which is a toxic substance. This process occurs mainly in the kidneys and liver. In the kidneys, the ammonia is eliminated by the urine. In the liver, ammonia is transformed in urea, following a complex biochemical process. The urea is then eliminated by the kidneys in the urine.
- The liver is a place of storage of excess fat resulting from hyper alimentation. There is also a liver lipid "catabolism", which is a metabolic process that breaks down large molecules *(such as polysaccharides, lipids, nucleic acids and proteins)* into smaller units, and "anabolism", which is the process by which the body utilizes the energy released by catabolism to synthesize complex molecules.
- The liver plays a major role in blood coagulation by ensuring the formation of fibrinogen, the absorption of vitamin K, which is essential for the synthesis of prothrombin *(a plasma protein)*, and the manufacture of heparin (anticoagulant).
  Also, it is involved in the formation of blood:
  - Formation of plasma proteins, especially serum albumin.
  - Storage and regulation of iron metabolism which is essential for the synthesis of hemoglobin.
  - Development of blood cells in the foetus and newborn.
  - Destruction of too old red blood cells (see the description of Kupffer cells) as in other hematopoietic organs (bone marrow, spleen).
- The liver has an antioxidant function that allows it to filter and stop substances that are toxic by themselves or because of an excessive blood level. It then turns these substances into less toxic products that are eliminated by the bile and in uric acid. Chemical reactions allow the formation of new molecules which reduce the toxicity of certain chemical substances produced by the digestion in the intestine. This antitoxic function also works against exogenous toxins (microbes, industrial toxins, alcohol, poison, etc.)

Note that the liver is the only human internal organ capable of natural regeneration of lost tissue. However, it is not true regeneration as the growth of the liver is a restoration of function, not its original form.

- Pancreas

The pancreas sits across the back of the abdomen, behind the stomach. Its head is on the right side of the abdomen and is connected to the duodenum through a small tube called the pancreatic duct. The narrow end of the pancreas, called the tail, extends to the left side of the body. It is about 15 cm long. Glandular tissue that makes up the pancreas gives it a loose, lumpy structure. This tissue surrounds many small ducts that drain into the central pancreatic duct. The

pancreatic duct carries the digestive enzymes produced by the endocrine cells to the duodenum.
the pancreas is a mixed gland which has an internal hormonal role (endocrine) and an external digestive role (exocrine).
The endocrine part is composed of hormonal tissue distributed along the pancreas in discrete units called islets of Langerhans. These cells secrete insulin.
The exocrine pancreas represents the rest of the pancreatic parenchyma *(The functional or specialized tissue of an organ)* which is constituted by the juxtaposition of pancreatic acini. Each acinus is constituted by a base of pyramidal cells delimiting a central cavity where the secretory product accumulates which is led outwards by a small excretory canal. The acini are grouped into lobules and lobes, the convergence of the excretory ducts lead to the major pancreatic duct *(also called duct of Wirsung)* and the accessory pancreatic duct *(also called duct of Santorini)*.

Pancreatic juice *(exocrine pancreas)* contains mineral salts *(notably bicarbonates)* and many enzymes.

- Proteolytic enzymes, (also called protease) which split the polypeptide molecules *(chains of amino acids and essential portions of proteins)* into simpler elements: amino acids and short peptides. Among these many enzymes, include trypsinogen *(activated trypsin by duodenal enterokinase that is released in significant amount when required for protein digestion)*.
- Lipolyptic enzymes including cholesterol esterase which converts esterified cholesterol into free cholesterol, the lipase that cleaves triglycerides into simpler elements: fatty acids and glycerol.
- Glycolytic enzyme or amylase, which hydrolyzes starch into maltose.

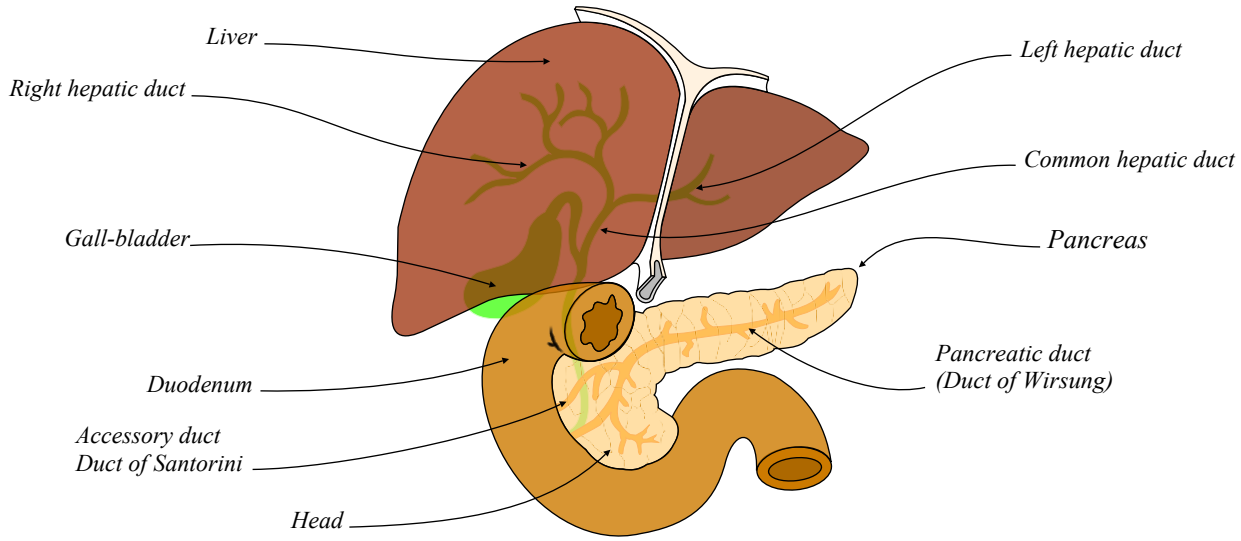
The secretion of pancreatic juice is not continuous but triggered by meals. It is stimulated by gastric hydrochloric acid via two hormones: Secretin and cholecystokinin of duodenal origin, the first stimulant especially the hydrobicarbonated secretion of the pancreas and the second especially the enzymatic secretion . Incidentally, gastrin developed by the gastric antrum would also play a role.

Islets of Langerhans of the endocrine pancreas consist of two types of cells: Beta cells (75%) and Alpha cells (25%).

- Beta cells secrete insulin. Insulin is a hypoglycemic *(low blood sugar)* hormone that acts at two different levels:
  - In the liver, it promotes the transformation and storage of circulating glucose in the form of hepatic glycogen and slows down the reverse process .
  - In tissues, it promotes the penetration and cellular consumption of glucose.

  Pancreatic insulin secretion is mainly controlled by changes in blood glucose levels. However, there is also a neurovegetative regulation *(the pneumogastric excito-secretor)*.
- Alpha cells of the endocrine pancreas secrete another hormone, glucagon, which has schematically opposite properties of insulin and is therefore hyperglycemic *(increasing hepatic glycogenolysis)*.

The overall action of the endocrine pancreas is hypoglycemic *(and the destruction of the pancreas causes diabetes with hyperglycemia)* since it is formed mostly of Beta cells (insulin).



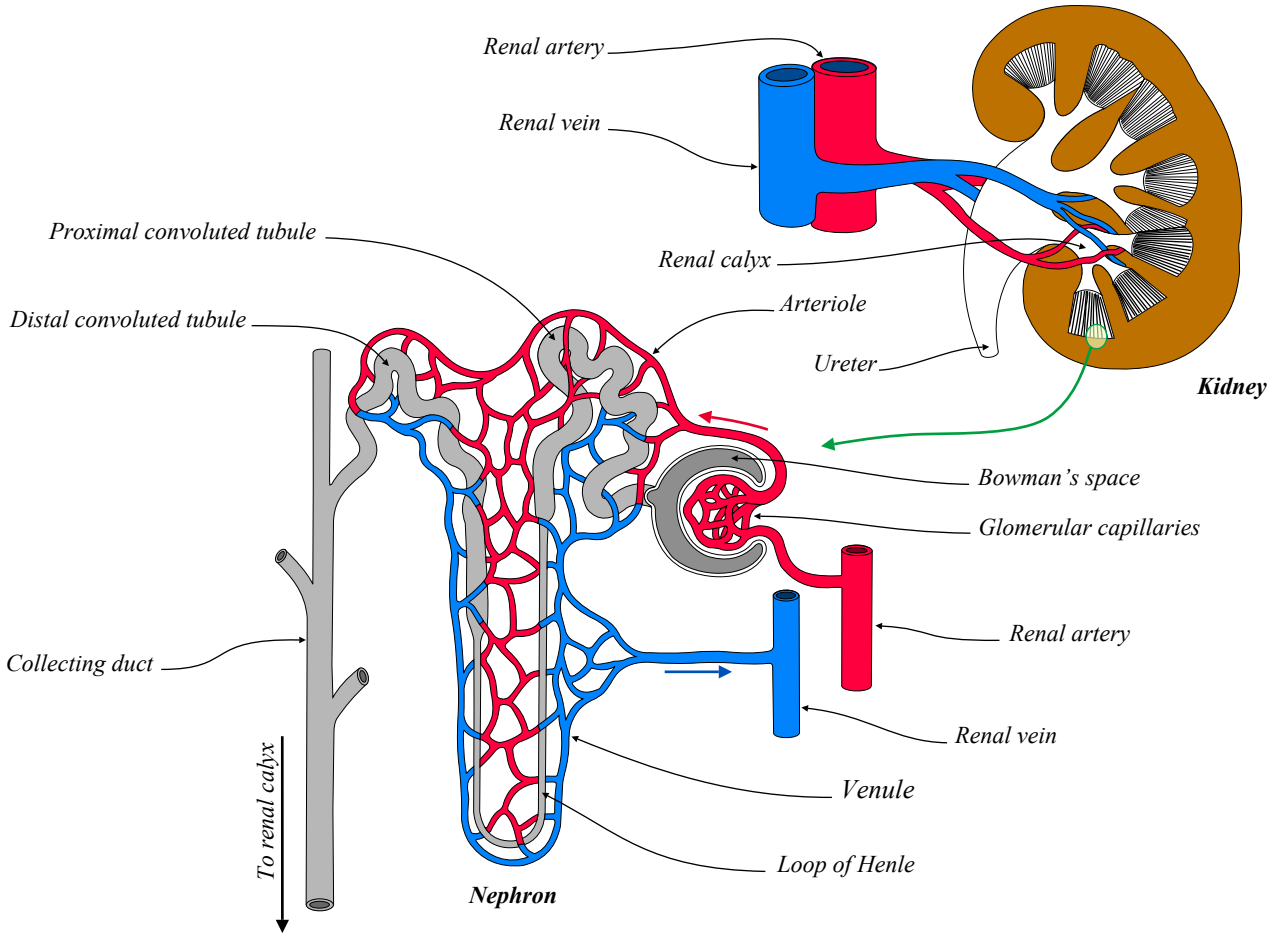
Urinary system

The urinary system consists of the kidneys, ureters, bladder, and the urethra. This system eliminates wastes from the body, regulates blood volume and blood pressure, controls levels of electrolytes and metabolites, and regulates blood pH.

- Kidneys

There are two kidneys which have an extensive blood supply via the renal arteries which leave the kidneys via the renal vein. Each kidney consists of millions of functional units called “nephrons” that removes waste from the body through urine, and reabsorbs elements that are necessary for the body. A nephron is a tube of complex shape formed of only one layer of cells. There are about one million nephron in each kidney. Each nephron is made up of two parts:

- The “renal corpuscle “ is the head of the nephron and it is where urine first forms, and consists of the gromerulus which is surrounded by the bowman’s capsule. Bowman’s capsule is made of a thin porous membrane. Gromerulus are networks of capillaries. The blood is filtered across the glomerular capillaries into Bowman’s space. Filtrates then exits the Bowman’s space into the renal tubules.
- The tubule begins at the Bowman’s capsule and consists of the following elements each has a dedicated function:
  - Proximal convoluted tubule:
    - Reabsorb 2/3 of the filtered Na (65-80%) and water
    - Reabsorb all the glucose and amino acids,
    - Reabsorb a fraction of the bicarbonate, potassium, phosphate, and calcium
    - Secrete ammonia *(to neutralize acid in excess)*
    - Secrete creatine *(that facilitates the recycling of adenosine triphosphate (ATP), the energy currency of the cell, primarily in muscle and brain tissue)*
  - Loop of Henle:
    - Water and salts are reabsorbed into the blood
    - Water passes to the interstitial fluid
  - The distal convoluted tubule function is:
    - Reabsorb sodium and chloride
    - Secrete ammonium ions and hydrogen ions.
    - Concentrate the urine
  - The collecting duct
    - Reabsorbs sodium and water
    - Secretes potassium



As described above, the kidneys maintain the balance and stability of the cells of the body (homeostasis) by controlling the excretion and reabsorption of substances.

- When potassium, sodium, calcium, magnesium, phosphate, and chloride ions, reach a higher concentration than normal, they are eliminated into the urine. When they are at a lower concentration than normal they can be reabsorbed into the blood during filtration.
- Hydrogen ions (acid), which are produced by the metabolism of proteins, accumulate in the blood over time. As a response, hydrogen ions in excess are excreted in the urine. Also, bicarbonate (base) is conserved or reabsorbed to balance the PH of the blood.
- The kidneys control the amount of water in the body. Excess water in the blood is excreted in urine, which results in diluted urine. When the body is dehydrated, the kidneys reabsorb water into the blood. As a result,

urine is highly concentrated in excreted ions and wastes.

- The kidneys monitor the blood pressure: When blood pressure is elevated, the reabsorption of water into the blood is reduced and the excess is eliminated in the urine. When blood pressure becomes too low, the kidneys can produce the enzyme “renin” to constrict blood vessels, which allows more water to remain in the blood. Urine expelled is concentrated.
- The kidneys stimulate the production of red blood cells through the hormone Erythropoietin.

- ***Ureters***

The ureters are the tubes that carry urine from the kidneys to the urinary bladder. They are 25 to 30 cm long and run on the left and right sides of the body parallel to the vertebral column. Urine is moved toward the urinary bladder by the movements of the muscles walls. The ends of the ureters extend slightly into the urinary bladder. They are sealed at the point of entry to the bladder by the ureterovesical valves. These valves prevent urine from flowing back towards the kidneys.

- ***Bladder***

The urinary bladder is a hollow muscular and elastic organ that collects and stores urine from the kidneys before disposal by urination. Urine enters the bladder via the ureters and exits via the urethra. The urinary bladder plays an important role in delaying and controlling urination so that the average person only has to urinate several times each day instead of constantly leaking small amounts of urine.
The urinary bladder is made of several distinct tissue layers:

- the mucosa layer which lines the hollow lumen is lined with transitional epithelial tissue that is able to stretch in the case of large volumes of urine and provides a protection from acidic or alkaline urine.
- The sub-mucosa, is a layer of connective tissue with blood vessels and nervous tissue that supports and controls the surrounding tissue layers.
- The muscular layer surround the sub-mucosa and provide the urinary bladder with its ability to expand and contract. It contracts during urination to expel urine from the body. Also, it forms the internal urethral sphincter, which is a ring of muscle that surrounds the urethral opening and holds urine in the bladder. During urination, the sphincter relaxes to allow urine to flow into the urethra.

- ***The urethra***

The urethra is the tube through which urine passes from the bladder to the exterior of the body. The flow of urine is controlled by the internal and external urethral sphincter muscles.

- The internal urethral sphincter is made of smooth muscle. It opens involuntarily when the bladder reaches a certain level of distention. This process results in the sensation of needing to urinate.
- The external urethral sphincter is made of skeletal muscle and may be opened to allow urine to pass through the urethra or may be held closed to delay urination.

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Part C - Accidents linked to air & surface gas diving

Hypothermia

Description:

Hypothermia is a physical condition that occurs when the body's core temperature falls below the normal 37° C to 35° C or cooler.

Cold water dangerously accelerates the onset and progression of hypothermia since body heat can be lost 25 times faster in cold water than in cold air.
The danger is considerably greater for mixed gas divers because of the very high conductivity of helium. The respiratory heat loss is enormous due to the fact that heat loss under heliox is six times faster than in air.

Hypothermia affects the body's core (brain, heart, lungs, and other vital organs).

Hypothermia diminishes the victim's physical and mental abilities, even in a mild case. Thus, it increases the risk of inappropriate decisions and accidents. .

Also, hypothermia increases the risk of a decompression accident

Severe hypothermia may result in unconsciousness and possibly death.

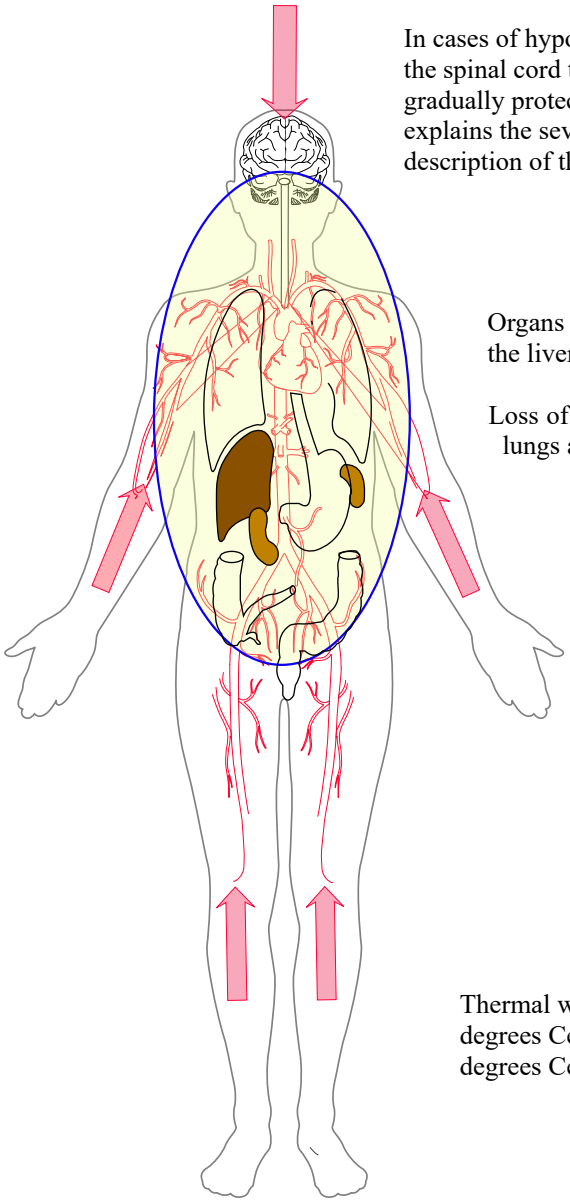
Symptoms

Internal Body temperature	Symptoms
36°C Mild case	Uncontrollable shivering; cold extremities; needs to urinate. Note that not all people shiver, and some can become hypothermic without any warning
34°C Moderate case	Impaired judgement; fixed ideas; confusion; irritability; amnesia; slurred speech.
32°C Severe case	Shivering decreasing and replaced by muscular rigidity. Movement becoming erratic and jerky.
28°C Critical case	Irrational behaviour increasing; stupor; muscular rigidity increasing; cardiac rate and respiration slowed; casualty becoming semiconscious.
27°C	Unconsciousness; loss of reflexes; fixed and dilated pupils, low or undetectable pulse. Ventricular fibrillation may occur.
25°C	Failure of cardiac and respiratory systems; ventricular fibrillation; death.

Hypothermia rates of an unprotected person in the water

Water temp. (°C)	*Loss of dexterity & intense shivering*	*Expected time before unconsciousness*	*Expected time of survival*
> 27° C	*2 to 12 hours*	*indefinitely*	*indefinitely*
27° C	*1 to 2 hrs*	*3–12 hours*	*3 hours – indefinitely*
21° C	*30 to 40 min*	*2–7 hours*	*2–40 hours*
16° C	*10 to 15 min*	*1–2 hours*	*1–6 hours*
10° C	*< 5 min*	*30–60 minutes*	*1–3 hours*
4° C	*< 3 min*	*15–30 minutes*	*30–90 minutes*
1° C	*< 2 min*	*Under 15 minutes*	*Under 15–45 minutes*

Note:
Cold currents with lower temperatures than the surrounding waters can be encountered at depths close to the surface and affect the divers in tropical areas.



In cases of hypothermia the brainstem and the spinal cord trigger a vasoconstriction to gradually protect the vital organs. This explains the several steps indicated in the description of the symptoms

Organs producing heat are the liver and the muscles

Loss of heat is through the lungs and the skin.

Thermal well being is 24 to 25 degrees Celsius in air and 33 to 34 degrees Celsius in water

Treatment

Internal Body temperature	Treatment
36°C Mild case	Prevent further heat loss - Allow body to re-warm itself - Warm sweet drinks (no alcohol) - Apply gentle heat source - Keep the victim warm for several hours, with head and neck covered. - Keep the casualty near the Deck Decompression Chamber.
34°C Moderate case	Offer warm sweet liquids only if victim is fully conscious, begins to re-warm, and if he is able to swallow (no alcohol) - Seek medical advice - Keep the victim warm for several hours, with head and neck covered - Observe for possible decompression accident. The principle of re-warming is the inverse process of the one established by the vasoconstriction: Re warming the core 1st, and then gradually the limbs. It must be very gradual.
32°C Severe case	Get medical advice/help as soon as possible - Move the casualty carefully (rough handling may cause cardiac arrest or ventricular fibrillation of heart) - If equipment available, give hot saline by irrigation through a naso-gastric tube - When the victim is able to swallow give hot liquids by mouth - Treat for shock – Keep temperature from dropping, while avoiding too rapid a temperature rise. Rapid re-warming can cool the blood as it flows through the cold outer tissues.
28°C Critical case	As above, medical support is absolutely necessary - If breathing and pulse are no longer detectable, start CPR, and re-warm gradually as indicated above. Give hot saline irrigation by naso-gastric tube. De-fibrillation and O2 assistance to be started as soon as possible. Never assume that a hypothermic victim is dead until completely re-warmed and still not responsive. If the patient still exhibits no vital signs (pupils not reactive, no heart beats and no breath), the decision to stop will be made by the doctor.

Prevention

- It is common today to use coveralls as diving suits in tropical waters. But, even in tropical waters, cold currents can be encountered at certain depths, especially after events like tropical storms or similar. A good practice is to wear specific diving clothes (wet or dry), except for shallow dives where the water temperature can be easily controlled.

- The diver may be suffering from hypothermia and not feel it. The supervisor must be very careful and look for any signs of hypothermia such as changes of behaviour and slurred speech . For the diver the need to urinate and shivering will be the 1st signs … Any sensation of cold must be reported to the supervisor.

- In case of cold during the dive, the diver must be removed from the water. The chamber and the surface team must be ready to carry on surface O2 decompression all the time. In addition, due to the vasoconstriction, hypothermia is able to trigger a decompression accident: To avoid such problems, a good practice is to reinforce the decompression procedure and keep the diver under observation after the completion of the stops.

- In case of suspected cold currents, a good practice is to reduce the bottom times.

Cold shocks

Cold shocks are the effects of sudden exposure of unprotected parts of the body to cold water.

Caloric vertigo:

This is due to the exposure of the external ear canal of one side and not the other side to cold water.
The effects are disorientation and nausea
Because the divers are wearing helmets, they will not be affected in normal working conditions. But a rescue diver (bellman) may be affected if wearing a damaged or badly adjusted hood.

Diving reflex:

This is due to the sudden exposure of the face to cold water.
The result is bradycardia (heart rate slowing down) and constriction of the vessels of the face.
The divers wearing helmets are not affected, except in the case of a loss of helmet… Appropriate training is generally sufficient for prevention.

Hyperventilation:

This is due to improper thermal protection of the diver during the launching of the dive, or an unexpected opening of the suit in cold water.
Sudden immersion in cold water results in an involuntary gasp, followed by 1 to 3 minutes of involuntary hyperventilation (respiratory rate increasing).
This hyperventilation results in a profound lowering of blood carbon dioxide levels and raising of blood pH levels, which causes a large risk of ventricular fibrillation (“cardiac arrest”), muscular cramps, and cerebral vasoconstriction which starves the brain of oxygen, causing disorientation and confusion. In this case, the diver cannot coordinate his breathing and swimming movements, and that makes his survival unlikely. This can also lead to panic with illogical actions like tearing off the helmet.
In normal diving conditions from a bell, the diver remains under control and can be immediately recovered into the bell. If the diver becomes incapacitated, the rescue diver must be sent to recover him immediately.
Thermal protection is essential to avoid such effects…

Hyperthermia

Description:

- Hyperthermia is defined as a temperature of the body greater than 38°C; the average normal human body core temperature being 37°C. Body temperatures above 40 °C can be life-threatening.

- Hyperthermia may occur in water to divers using hot water suits if the system is improperly set up or divers performing dives in tropical waters with diving suits designed for cold water.
  In chambers, it may result from a cooling system failure or be caused by the heat resulting from excessive pressurization rates. It may also result from the improper adjustment of the temperature of the chamber or the bell that should normally be as follows:

Depth (msw)	*Temperature range (C°)*
0- 50	*22 - 27*
50 - 100	*25 - 29*
100 - 150	*27 - 30*
150 - 200	*28 - 31*

Depth (msw)	*Temperature range (C°)*
200 - 250	*29 - 31*
250 - 300	*30 - 32*
300 - 350	*31 - 33*

- Note that Hyperthermia also affects people working on deck under direct sun or in too hot rooms.

Symptoms

Severity	Symptoms
Moderate case	- Abdominal cramps - Muscle cramps - Nausea - Headache - Dizziness - Weakness - Heavy sweat - feeling uncomfortable
Severe case	- Odd or bizarre behaviour - Irritability - Delusions - Hallucinations - Increased pulse rate - Disorientation, confusion - Vomiting
Critical	- Collapsing - Coma - Death

Treatment

Cases	Treatments (diver medic or medical support)
Moderate case	- Have the patient resting. As he rests, he cools gradually and may not need any other treatment since he is no longer producing heat by working. - Cool the patient by removing clothing, fanning, and applying ice packs or cool compresses to the face, neck, underarms, and groin. - If not nauseated, give ordinary fluid by mouth, starting slowly. - If the patient is nauseated, hypotensive, or faints upon sitting or standing, give 500-1000 cubic centimetre of "IV fluid" for 30-40 minutes. - If IV fluids are not available, let the patient rest and cool down for 20-30 minutes, lying down, then place ice chips in the mouth or give small sips of liquid, increasing the intake as tolerated. - Have the patient rest for 8-24 hours. He may be sensitive to heat for a period of time.
Severe case	- Cool the patient as fast as possible: Apply ice to the neck, arm pits and groin. Soak the victim in ice water. Continue until a deep rectal temperature is about 38.5 C or until the victim feels like someone with an ordinary fever. Then stop active cooling and place the patient in a cool area. - If the patient is hypotensive or shocked, do not give vasopressor drugs. Support the blood pressure with "IV Ringer's lactate" or normal saline sufficient to maintain a systolic pressure of 100-110. Avoid sudden infusions of large volumes of IV fluid. It is better to give 200-300 cc. amounts and observe the effect over 5 minutes, then repeat as necessary. - When the blood pressure is stable, change to "IV D5W" (or oral fluids if the patient is conscious) and observe the urine output. - If available, insert a urinary catheter (Foley) and give sufficient fluid (IV fluid or orally) to maintain a urine volume of at least 60 cc. (2 ounces) per hour to avoid the possibility of kidney damage. - MEDEVAC to be organized
Critical case	- If the patient is unconscious or delirious, convulsing, or has skin that is very hot and dry, it means that the heat has damaged the temperature and blood pressure centers in the brain. This heat stroke is a medical emergency with a high mortality rate. - Complications-convulsions, stroke, or myocardial infarction may all occur in heat-stroke. Treatment is the same as in the usual setting. - Urgent MEDEVAC to be organized.

Definitions:

- Hypotension: Low blood pressure

- "IV fluid" : Supply fluids when casualties are unable to take in an adequate volume of fluids by mouth. Provide salts needed to maintain electrolyte balance. Provide glucose (dextrose), the main fuel for metabolism. Provide water-soluble vitamins and medications.

-Vasopressor: Increases the blood pressure.

- "IV D5W": Sugar solutions, such as with glucose (also called dextrose), to be injected intravenously, have the advantage of providing some energy, and may thereby provide the entire or part of the energy component of nutrition by injection.

Prevention

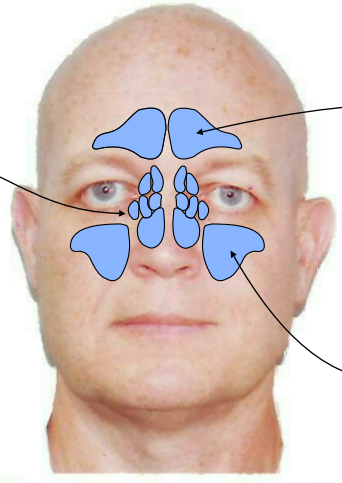
- The divers must never be kept for a long time under direct sun before launching
- Avoid exposures to direct sun, to the personnel working on deck and the standby diver (shelters to be installed)
- Chamber in designated refrigerated container.
- Pressurization of the chambers should not be too fast
- Acclimate personnel who are not used to tropical countries
- Distribute sufficient fluids
- If using hot water clothes, make sure that the adjustment of the heat is optimal…

Sinus Barotrauma

Description:

- Sinuses are rigid filled airspaces within the skull's bone and are mostly connected to the upper nasal passages. The largest are the bone's frontal sinuses over the eyes and the maxillary sinuses in the cheekbones. There are other smaller sinuses elsewhere within the skull. Sinuses appear to serve no useful purpose other than to reduce the total weight of the skull.

The ethmoid sinuses are small pockets in the ethmoidal bone, which is the bone that separates the eyes from the nose. These sinuses are separated from the eye by a thin bone thickness.



The frontal sinuses are over the forehead and above the eyes. These are absent at birth, and in approximately 5% of adults

The maxillary sinuses are pockets near the checks. They are located in the maxilla bone under the eyes. They contain three recesses and are shaped like pyramids.

- The sinus spaces are connected to the respiratory airway by fine passages.
- If the connecting passages are blocked, an imbalance of pressure will cause acute pain.
- If the pressure is not relieved by equalizing or by reducing the ambient pressure, the linings of the sinus cavity will bleed, flooding the cavity to balance the pressure. A slight nosebleed during or after a dive is a common sign of a mild sinus blockage.
- A cold or hay fever will cause inflammation and swelling of the tissues making up the nasal tract, Eustachian tubes, sinus cavities and airways, and the secretion of mucus. All of which will lead to blockages of the airways and the inability to clear ears and sinuses.

Symptoms:

- Inability to clear the ears
- Bleeding of the nose, and nose plugged
- Intense pain in the forehead or the cheeks

Treatment:

- Decongestant medication should only be used under medical guidance.
- Seek medical advice if the diver suffers persistent difficulty with ears and sinuses.

Prevention:

DO NOT DIVE WITH NASAL INFECTION OR CONGESTION.

Ear Barotrauma

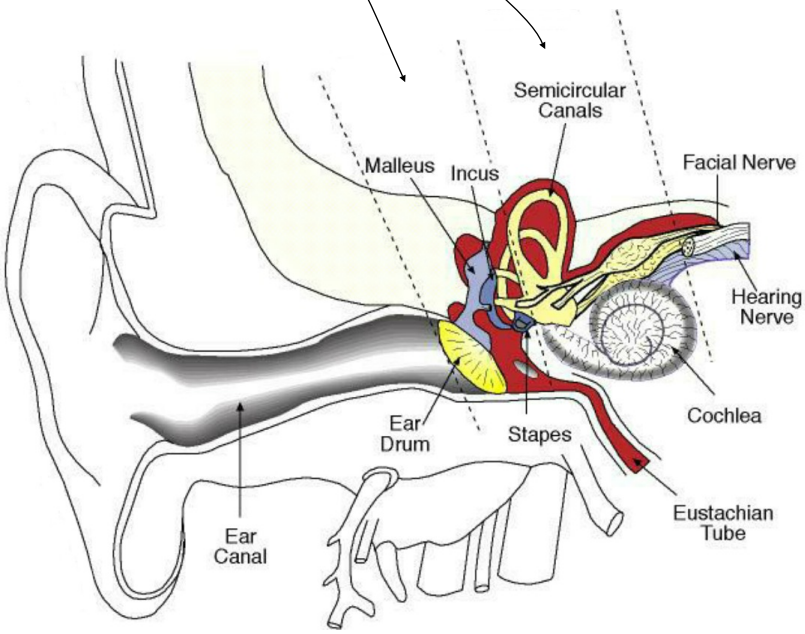
Description:

The inner ear *is filled with fluid and is embedded in the skull. Vibrations picked up at the eardrum and transmitted to the inner ear are converted into nerve impulses sent to the brain and perceived as sounds.*

The semicircular canals are considered part of the inner ear but play no part in the hearing. They are of major importance for a sense of balance and position, and their function can be upset by certain conditions affecting the middle ear.

The middle ear *is a rigid air-filled space, surrounded by bone (skull). The eardrum forms an outer wall, and the airspace is connected to the rear of the throat by the Eustachian tube.*

The middle ear comprises a series of bones, which transmit vibrations from the eardrum to the inner ear.



The external ear *consists of the pinna and the ear canal, which is closed off at its inner end by the eardrum.*
The outer ear is open to the air, and its purpose is to collect sound waves and direct them to the eardrum.

- The ears are very sensitive to changes in pressure and will be affected around 2 metres below the surface.

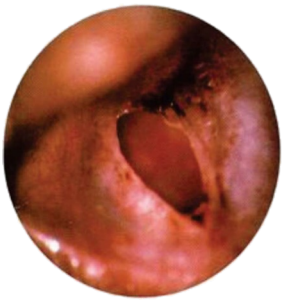
- One common reason of imbalance is the blockage of the eustachian tube if the diver descends too quickly without equalization, or if the Eustachian is plugged due to an infection or the obstruction of the ear canal by a plug of wax.

- On the descent, the diver's inability to equalize causes a vacuum in the middle ear, sucking the eardrum and tissues in the middle ear and eustachian tubes inwards. Pain is felt and increases with pressure.
  On ascent, the inability to equalize the middle ear air space can cause a build-up of excessive pressure, flexing the eardrum outwards.

- If the imbalance of pressure is not relieved, the eardrum will rupture. If the diver is wearing a face mask, some cold water may enter the middle-ear cavity and upset the balance and hearing organs.

- The inner ear is responsible for both hearing and balance. The round and oval windows separate the inner ear from the middle ear. These openings are covered by some of the thinnest, most delicate tissues in the human body. The oval window is connected directly to the eardrum by a chain of bones called the ossicles.
  As the eardrum flexes inwards, the pressure is transferred directly to the oval window via the ossicles, causing the oval window to flex inward in conjunction with the eardrum. At this point, the ossicles either press through the oval window (perforating it), or the increased pressure in the inner ear from the oval window pressing in causes the round window to bulge out and burst.

Symptoms:

State	Symptoms
External ear squeeze	- Discomfort and pain on descent - Outward bulging of the eardrum - Swelling and bruising of the skin lining the ear canal.
Middle ear barotrauma	- It can be recognized after a dive by the feeling of "fullness" or "water in the ear" that cannot be relieved. This sensation is caused by the accumulation of blood and body fluids in the eardrum and middle ear, - Type I: Portions of the eardrum are red, possible distortion of the eardrum (in or out)  - Type II: Completely red eardrum, possible distortion of the eardrum (in or out) - Type III: Type II, but with blood and fluid in the middle ear - Type IV: Perforated eardrum with other accompanying symptoms 
Inner ear barotrauma	- Immediate feeling of vertigo, possibly accompanied by nausea or vomiting. - Disorientation - Hearing loss and tinnitus (buzzing or ringing ears) are also common signs.

Prevention:

- Do not dive when sick or congested.

- The Divers should never equalize forcefully.

- Equalization at the very beginning of the descent.

- Descend feet first and head up allow for easier equilibration.

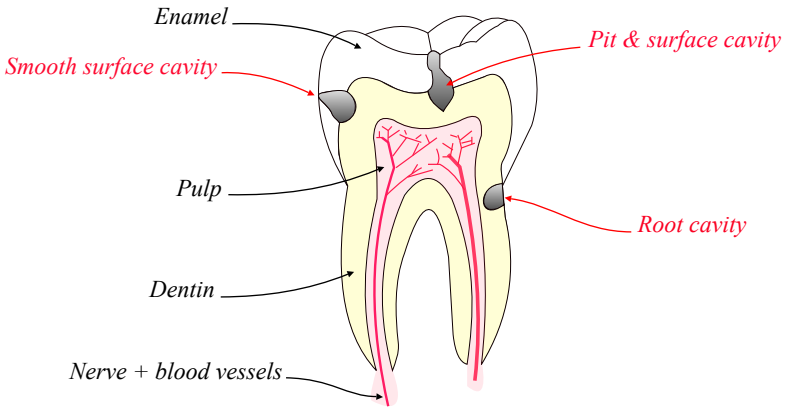
- The dive is to be stopped in case of problems of equalization.

- Remember that those who have equalization problems in the water will have similar problems in a chamber.

Teeth Barotrauma

Description

Gas spaces may exist in the roots of infected teeth, along with dying nerves, in necrotic areas of the pulp, and alongside or associated with fillings that have undergone secondary erosion (it is one of the main teeth barotrauma causes).



- The gas may enter around the edge of the filling, adjacent to the tooth, or through micro-fractures of the enamel and dentine. Teeth with full cast crowns may be susceptible to air being forced into the cemented material between the crown and the tooth. During the descent, the gas space is replaced with the soft tissue of the gum or with blood and effusion.
- Another presentation of dental barotrauma occurs in cases involving a carious tooth with a cavity. As pressure differences across the cementum develop, the tooth may cave in (implode) on the descent or explode on the ascent. Fast rates of ascent or descent will tend to precipitate this.

Symptoms:

- Pain may happen during the pressurization. If, because of slowed descent, symptoms are not noticed, the gas expansion on ascent may be restricted by the blood in these spaces, resulting in distension and severe pain.
- Explosion of the teeth causes considerable pain.

Treatment:

- A pressure applied to the casualty‘s teeth may cause pain and identify the affected tooth. Sensitivity to cold may also localize the affected tooth.
- A mouth mirror and a dental pick can be used to identify the tooth affected and the extends of damages.
- An analgesic can diminish pain during a limited period. However, the person must consult a dentist as soon as possible.
- The diver affected should not dive as long as the problem is not definitively solved.
- If the problem is detected during the saturation, the diver affected must be closely observed during the ascent to ensure that no pressure builds in the tooth, which may result in an explosion. That may result in a slower ascent.

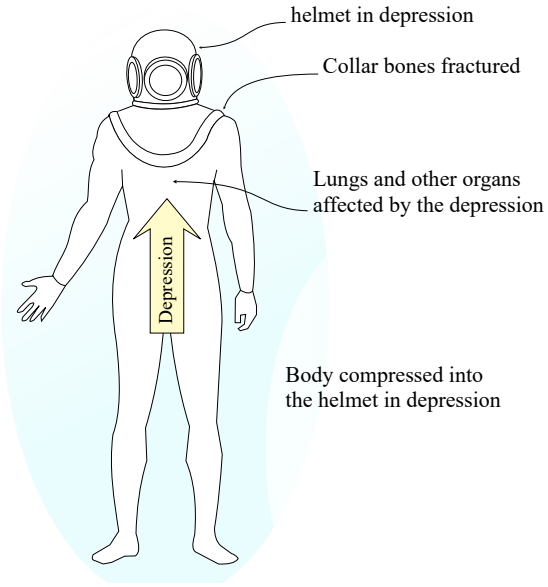
Prevention:

- Biannual dental checks (including X-ray examinations). Also, avoidance of hyperbaric exposure after a tooth extraction or surgery until complete tissue resolution has occurred.
- Buccal hygiene and self-examination are essential.

Helmet squeeze and nips

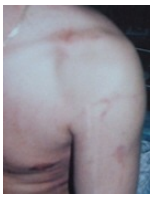
Description & effects

This accident was common in the old days when the divers were using suits communicating with continuous flux helmets.
Such accidents have happened because the diver, wearing lead shoes and unable to swim, was falling deeper than expected with a gas supply unable to compensate for the surrounding pressure.
Another reason was the rupture of the supply hose at or near the surface.
The most common effects were the fracture of the collar bones, the rib cage crushed, and severe internal hemorrhages, so the accident could be fatal. Descriptions of divers compressed into their helmets have been published, sometimes with exaggeration.

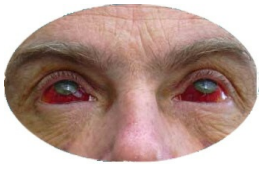


Commercial divers working in the offshore industry are not using the equipment described above nowadays.
However, continuous flux helmets such as the “AH 5” produced by “Divex” are still in use for particular operations, mostly by companies working in unhealthy surroundings or the nuclear industry, as they have the advantage to be perfectly waterproof. To protect the diver from the accidents described above, they are often separated from the suit by a “neck dam”, and must be fitted with a non-return valve. Nevertheless, an accident may happen if these protections are defective.

Another squeeze effect is called “nips”. This can happen if the diver uses a dry suit without an inflator or with a deficient inflator. In this case, the surrounding pressure will compress the suit on the body, creating folds that will trap and pinch the skin and will create bruising on the skin.



Mask squeeze occurs in scuba diving when the diver fails to equalize the internal mask's pressure with the surrounding pressure by blowing into it, which results in the mask acting like a suction-cup. The eyes can be affected by hemorrhages as they are composed of very soft tissues.
Because only helmets are used during saturation, this problem should not happen except in



Treatment

Because the lungs and other organs may be damaged, the diver suffering from helmet squeeze must be evacuated from the work site to a proper hospital as soon as possible. That poses the problem of long decompression and medical support during this phase for the divers saturated. For this reason, external medical support must be triggered.

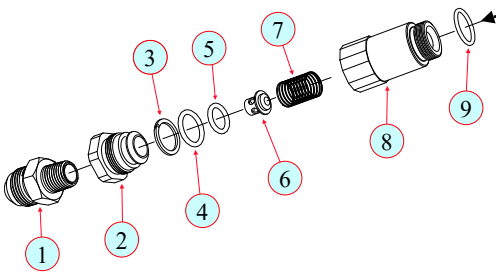
Note that a diver using helmet suffering of bloody eyes may be affected by more severe damages.

Nips are painful but generally are no serious injuries, and they can be easily treated using dedicated skin creams.

Prevention

- All modern helmets are fitted with a non-return valve and this valve must be tested before the launching and at the completion of all dives.
- The integrity of the umbilical and particularly the fittings is to be checked as well as the condition of the compressors and panels

Non return valve



1 - Connector umbilical
2 - Seat
3 - Wiper
4 - O ring
5 - O ring
6 - Poppet valve
7 - Spring
8 - Valve body
9 - O ring

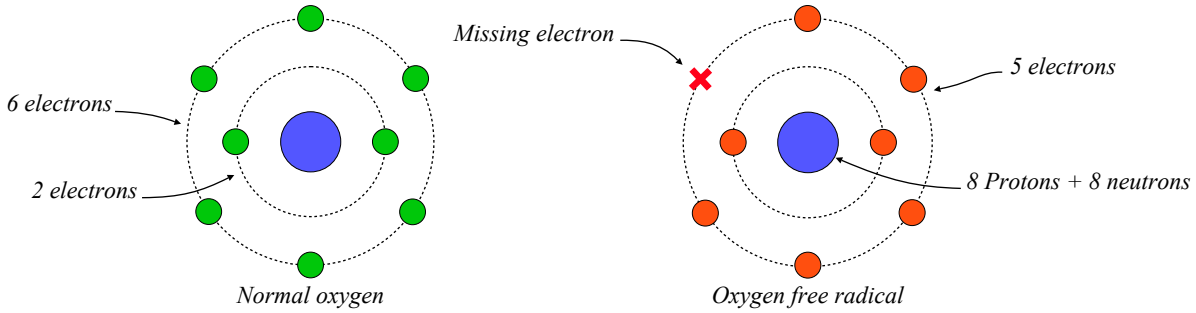
Adverse effects of hyperbaric oxygen

The role of oxygen has been simultaneously demonstrated by Joseph Priestley (1733-1804) and Antoine Lavoisier (1743-1791). Oxygen is necessary for life, and the papers taken as references in the previous section prove its benefic role when used in association with hyperbaric conditions. However, oxygen is poisonous at high doses, with toxicity that depends on both the partial pressure (PPO2) and exposure time. Acute O2 poisoning is linked to too high oxygen partial pressure and may result in a violent seizure crisis, while chronic oxygen poisoning results in the inflammation of the lungs after long exposures to pressures not sufficient to trigger acute poisoning but elevated enough to impair the lungs (>0.5 bar). In addition, it has been discovered that repetitive and long exposures to hyperbaric oxygen, even at sufficiently low pressures not to trigger the two effects above, may lead to diseases not immediately detectable.
For convenience, these three adverse effects, which may act simultaneously, are explained separately.

Acute oxygen poisoning *(Paul Bert effect)*

Description

Acute oxygen poisoning, also called "Central Nervous System (CNS) oxygen toxicity", was discovered first by Paul Bert (1833 - 1886), who, through a series of experiments described in his book *"Barometric pressure"*, clarified the role of oxygen in the air. He discovered and demonstrated that oxygen has adverse effects at high pressure through experiments on animals, where he observed that this gas could lead to convulsions, often resulting in the subject's death, and compared these convulsions to the effect of strychnine, a deadly poison causing muscle contractions and asphyxia. He also concluded that exposure to pure oxygen at pressures of 20 atmospheres and above triggers immediate convulsions that quickly lead to death, that this phenomenon of immediate convulsions can be observed at 6 atmospheres, and that birds submitted to pure oxygen at 3.5 atmospheres had onset of convulsions after appropriately 5 minutes exposure. As a reminder of this scientists, this phenomenon is also called the "Paul Bert effect" (These experiments are detailed in chapter IV - Subchapter #1, "Toxic action of oxygen at high tension" of the book *"Barometric pressure"*).
Studies of this undesirable phenomenon continued after Paul Bert and are still undertaken. They have not yet resulted in complete comprehension of the mechanism that triggers it. However, they have identified the conditions that favour its onset and the signs allowing us to predict it. Thus, they have allowed us to implement precautions to avoid triggering it and the counter-measures to control it from arising.
Even though not fully clarified, there is a consensus to link the onset of CNS oxygen toxicity to the action of some Reactive Oxygen Species (ROS). For example, in an article entitled *"Central Nervous System Oxygen Toxicity and Hyperbaric Oxygen Seizures"*, doctor Edward P. Manning says that the human body has several equilibrated safeguards that minimize the effects of reactive species on neural networks, believed to play a primary role in CNS O_2 toxicity. The increased partial pressure of oxygen (PO_2) appears to saturate protective enzymes and unfavourably shift protective reactions in the direction of neural network over-stimulation. Certain regions of the Central Nervous System (CNS) appear more susceptible than others to these effects. In another study called *"Oxygen Toxicity and Special Operations Forces Diving: Hidden and Dangerous"*, Doctors Thijs T. Wingelaar, Pieter-Jan A. M. van Ooij, and Rob A. van Hulst say that the most plausible explanation is related to an overflow of reactive oxygen species (ROS) in the brain after an increase in cerebral blood flow.
Reactive Oxygen Species (ROS) are molecules capable of independent existence, containing at least one oxygen atom and one or more unpaired electrons. This group includes oxygen free radicals, as well as free nitrogen radicals. Free radicals are atoms that have one or more unpaired electrons. They can have positive, negative or neutral charge. Oxygen has eight electrons in two separate orbitals and is especially susceptible to radical formation. Radicals derived from oxygen are those of most concern in biological systems.



Oxygen-derived radicals are permanently produced during the normal biological process and are necessary intermediates in various biochemical reactions. For example, they are involved in eliminating invading pathogens through white blood cells, such as neutrophils. However, due to their high chemical reactivity, they can damage many macromolecules when generated in excess or not appropriately controlled. That can be the case when the body is confronted with abnormal

situations such as hypoxia or hyperoxia.
The main toxic effect of oxygen radicals is damage to cellular membranes. It is initiated by a process called “lipid peroxidation”, which consists of attacks against lipids, particularly the fatty acids of the membrane. That results in an altered permeability of the membrane and a decreased activity of membrane-bound enzymes, which are the catalysts that carry out chemical reactions and are responsible for the maintenance of cellular functions such as ion transport, secretion, and uptake of a variety of substances, as well as cell to cell interactions. Thus, the activity of the cell membrane and its various receptors such as, for example the synapses, is detrimentally changed.
It must be noted that in a paper called *“Effects of prolonged oxygen exposure at 1.5, 2.0, or 2.5 ATA on pulmonary function in men”*, doctors J M Clarck, Chris Lambertsen, R Gelfrand, N D Flores, J B Pisarello, M D Rossman, & J A Elias, suggest that the magnitudes of measured effects vary widely at different pressures and among different individuals. Thus we can conclude that some people are more affected by this phenomenon than others.
Also, in a study called *“Acclimatization and Deacclimatization to Oxygen: Determining Exposure Limits to Avoid CNS O2 Toxicity in Active Diving”*, doctors Ran Arieli and Ben Aviner suggest that there is a process of acclimatization and possibly of de-acclimatization to oxygen. Doctor Arieli also says that the two principal modulators affecting CNS oxygen toxicity are metabolic rate and CO_2 load.

Symptoms and triggering parameters

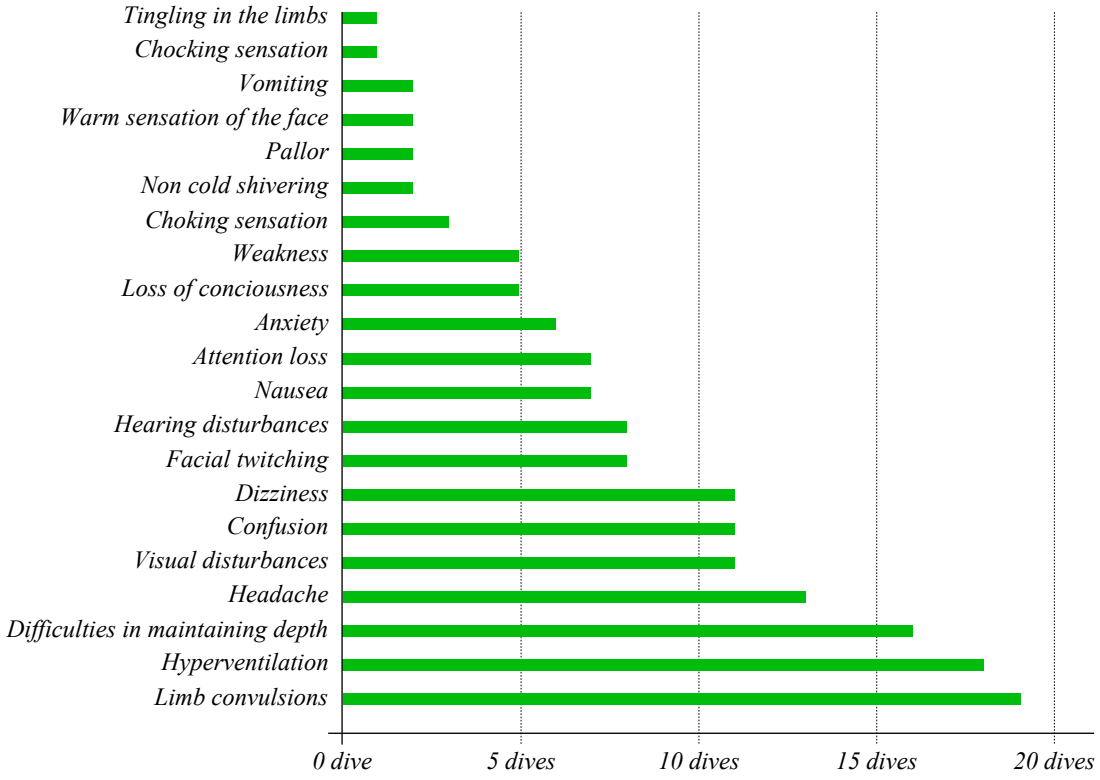
Evaluating the onset of a crisis is of primary importance to stop it in time. Based on the above, a list of warning symptoms has been published that can be remembered by the mnemonic “VENTID” (Vision, Ears, Nausea, Twitching, Irritation, Dizziness). It is in force within most diving organizations:

1) Vision: Tunnel vision, dim or blurred vision, flashes of light or patterns in the air.
2) Ears: Hearing-bells, music, knocking, ringing or humming.
3) Nausea: There may be sudden vomiting without actual nausea, retching, indigestion, or stomach discomfort.
4) Twitching: It typically involves the lips, but can be any part of the face or body. It is said that it is not a reliable warning if taken alone.
5) Irritability: The diver becomes nervous, not listening to instructions from the surface. This is followed by sleepiness, depression, feeling happy or "high", sudden fear or feeling of danger, restlessness, and fidgeting.
6) Dizziness: The diver may feel faint, dizzy, or light-headed.

If no action is undertaken during the warning phase, the symptoms evolute as follows:

1) Respiratory: A choking sensation, panting, grunting, hiccups, or spasms of the diaphragm may be seen.
2) Unpleasant taste or smell.
3) Convulsion: Usually a sudden, unexpected grand mal seizure, identical to epilepsy. There may be only jerking or twitching of one part of the body with the diver remaining conscious. The onset of the convulsion is often very fast, giving little time to react.

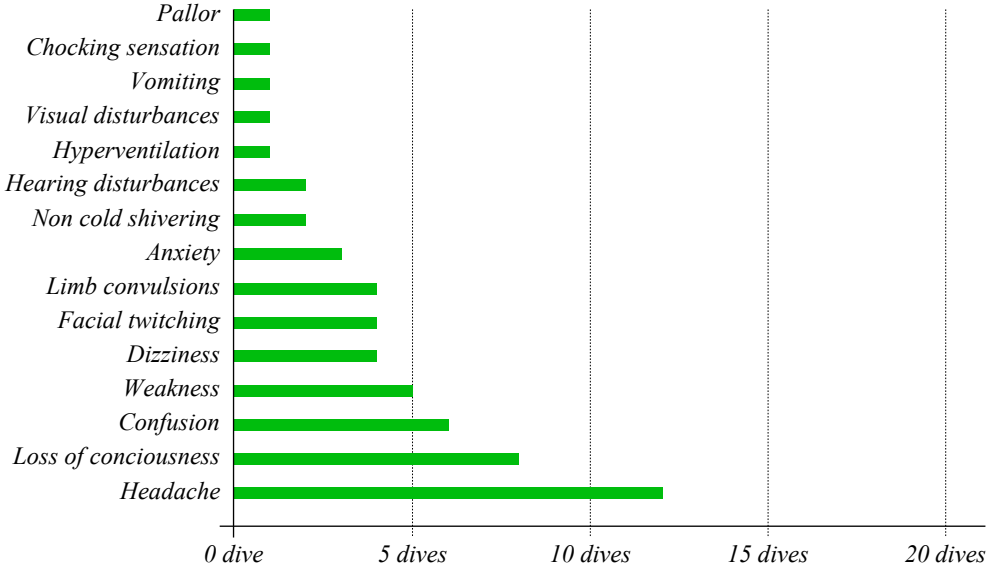
The list above can be completed by the evaluation of the frequency of symptoms appearing during the dive in the table below, published by doctors Ran Arieli, Yehuda Arieli, Yochanan Daskalovic, Mirit Eynan, and Amir Abramovich in a paper called *“CNS Oxygen Toxicity in Closed-Circuit Diving: Signs and Symptoms Before Loss of Consciousness”*.



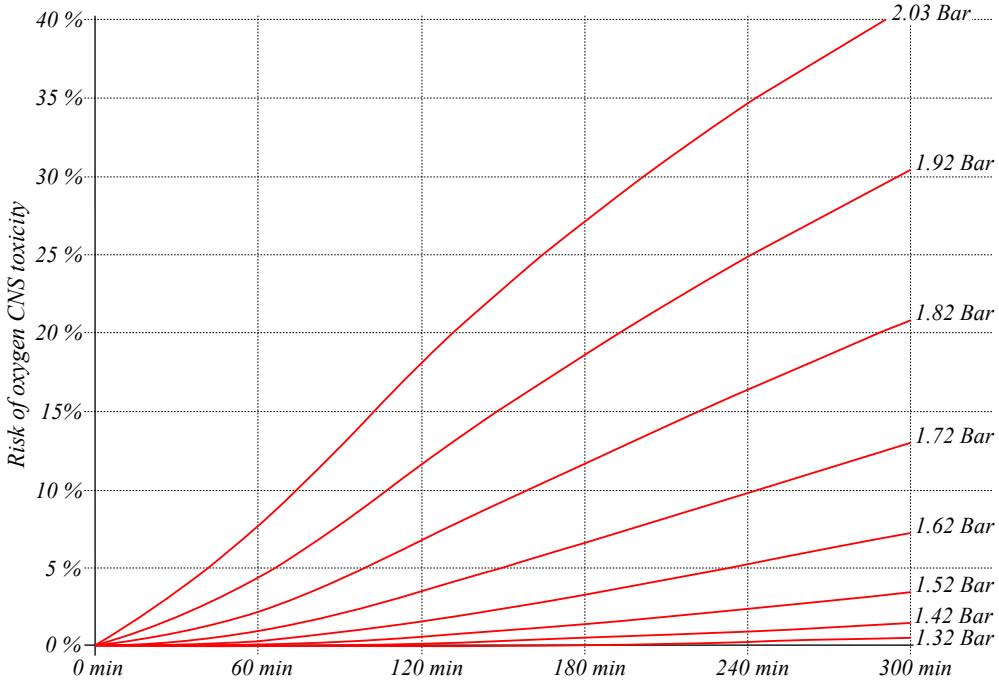
This study aimed to report the sensations and symptoms accompanying CNS oxygen toxicity accidents and evaluate whether loss of consciousness can occur without any warning signs. The authors documented 36 CNS oxygen toxicity accidents in closed-circuit oxygen diving to achieve this task. Their analysis included the evaluation of the first reports from the diving units and of the interviews of the victims and their buddies. The examinations of the diving equipment by the investigation teams were also taken into account.
Regarding the symptoms that appeared before termination of the dive, the authors say the following:

- In a large number of dives, there were limb convulsions (uncontrollable tremors), hyperventilation, difficulty in maintaining a steady depth, and headache.
- In about one-third of the dives there were visual disturbances, confusion, and dizziness.
- In about one-quarter of the dives there was facial muscle twitching, nausea, hearing disturbances, attention loss, anxiety, weakness, and loss of consciousness.
- In a smaller number of dives other symptoms were reported: A choking sensation; non-cold shivering; pallor; a warm sensation in the face; vomiting; vertigo; and tingling in the limbs.

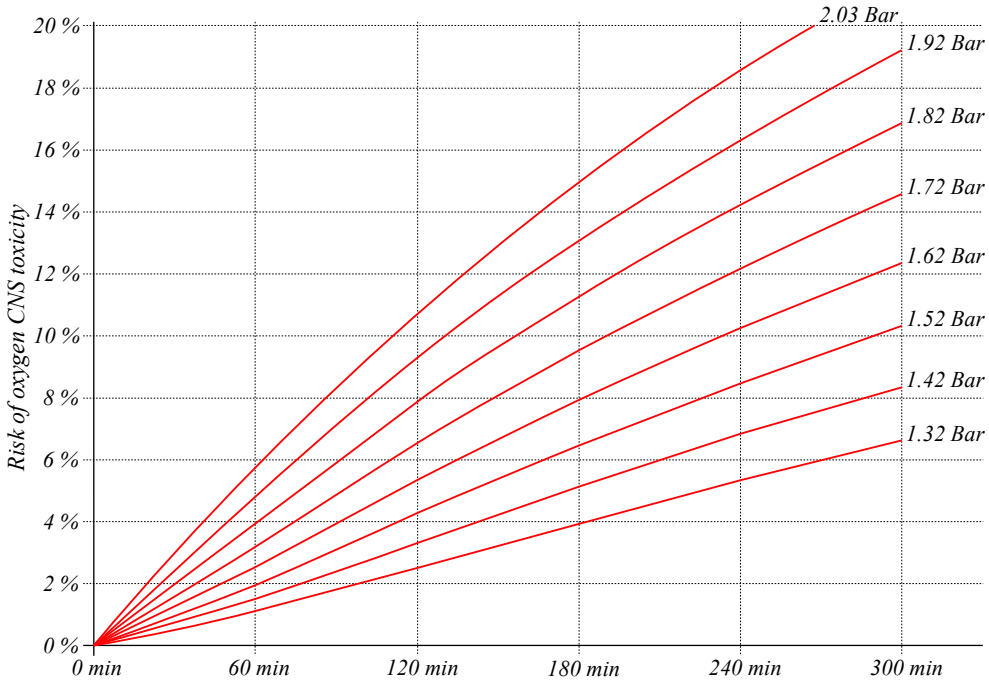
The authors also say that a number of symptoms were experienced immediately after detachment from the mouthpiece and breathing atmospheric air. These are presented in the table below. It shows that the most frequent symptoms after the termination of a dive were headache, confusion, weakness, facial muscle twitching, and limb convulsions. It also shows that loss of consciousness occurred more often immediately after the dive completion.



A lot of experiments have been done to highlight the parameters favouring CNS oxygen toxicity. From these studies, mathematic equations and empiric safety curves indicating the general limits of most divers have been published.
In the paper *"Modeling pulmonary and CNS oxygen toxicity and estimation of parameters for human"*, doctors R Arieli, A. Yalov, & A. Goldenshluger say that a method used for selecting the parameters for equations allowing calculating the CNS oxygen toxicity can be derived from the maximum likelihood method from official observations. It is the case of the following curves that represent the percentages of risk of CNS toxicity in the function of time and PO2, which calculations were derived from human hyperbaric exposures.

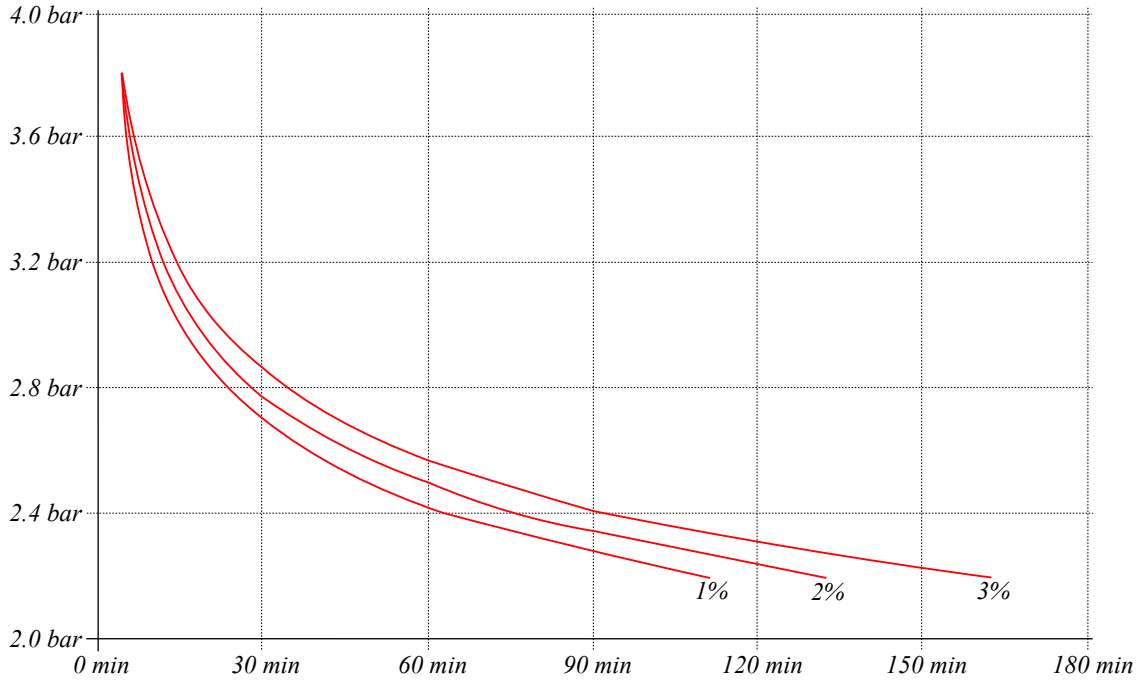


Doctors R Arieli, A. Yalov, and A. Goldenshluger based the curves displayed on the previous page on the studies published by A L Harabin during the nineties. To reinforce this model, they conducted experiments on rats to quantify the effects of metabolic rate and CO_2 load. That resulted in the publication of complex equations that allow quantifying both phenomenons. In addition, they gathered reports of 2,039 closed-circuit oxygen dives and took the data from their experiments together with the diving data to issue an equation taking into account the phenomena indicated above with the maximum likelihood. The curves below are the result of this work. We can see that they are more pessimistic than those on the previous page.

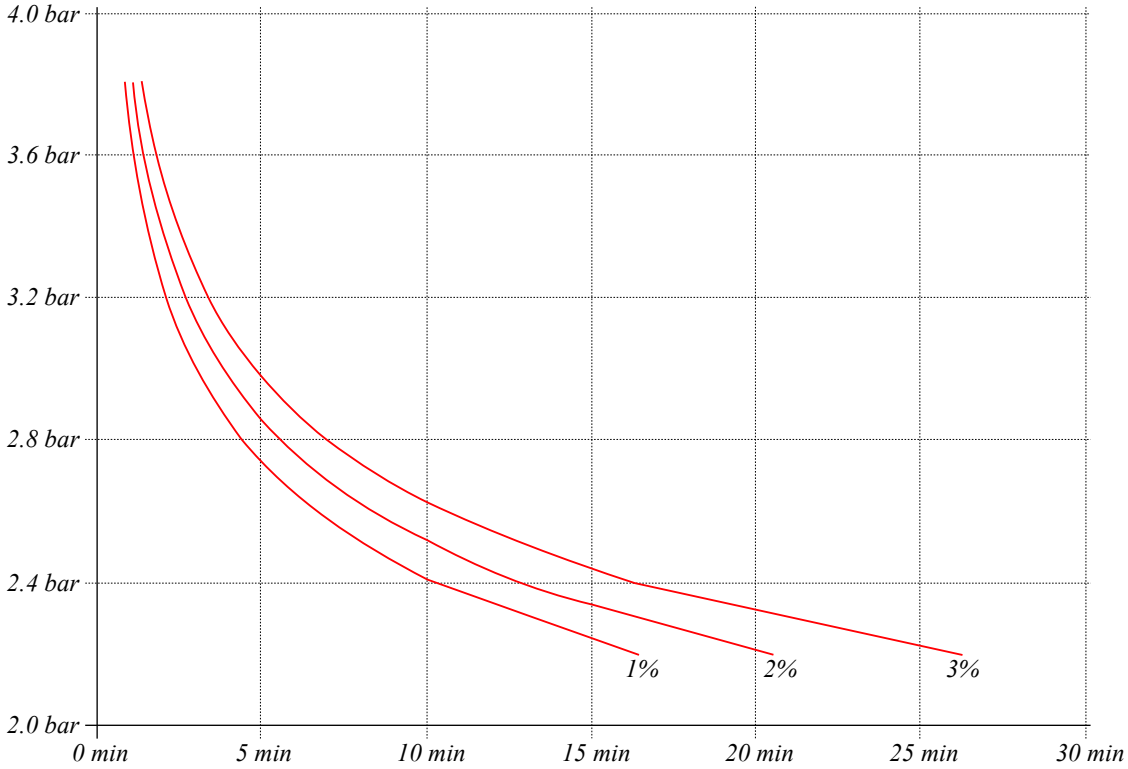


Of course, curves derived from mathematic models have to be considered with caution due to the considerable individual variation in susceptibility to O_2 toxicity. Also, other works are currently ongoing that may result in new models. For example, in another document called *"Model of CNS O_2 Toxicity in Complex Dives with Varied Metabolic Rates and Inspired CO_2 Levels",* doctor Arieli describes a development of the model above that allows taking various metabolic rates into account. This development was done with data from rats but not tested with humans. For this reason, he concluded this study by saying that it was impossible to derive a predictive equation for humans due to the lack of data. Nevertheless, it is probable that such a procedure will be tested on humans one day.

In addition to the effect of the metabolism on O_2 toxicity, it has been discovered that the risk of CNS oxygen toxicity at rest is greater during immersion than in dry conditions. We can see the difference between the two sets of curves below extracted from the document *"Power Equation for Predicting the Risk of Central Nervous System Oxygen Toxicity at Rest"*, published by doctors Ben Aviner, Ran Arieli, and Alexandra Yalov, and which purpose was to create a predictive algorithm for use in hyperbaric oxygen therapy and rest periods during saturation diving, as indicated in the title.



Calculated risk of CNS oxygen toxicity at rest for dry exposure as a function of time and PO_2



Calculated risk of CNS oxygen toxicity at rest for immersed exposure as a function of time and PO_2

When comparing the curves of 1 % of risk of toxicity, we can see that at 2.4 bar this level is reached within approximately 10 minutes during an immersed exposure and a little bit more than 60 minutes during a dry exposure.

In addition to what is said above, in their study *"Oxygen Toxicity and Special Operations Forces Diving: Hidden and Dangerous"*, doctors Thijs T. Wingelaar, Pieter-Jan A. M. van Ooij, & Rob A. van Hulst say that no oxygen-induced convulsions have been described with a PO_2 lower than 1.3 bar in humans, even though susceptibility to oxygen toxicity has a high interpersonal and intra-individual variability.
They also say that reports on the incidence of CNS toxicity vary greatly, ranging from 1 in 157,930 close circuit rebreather military dives to approximately 3.5% of the dives. They attribute this considerable variation to different definitions of the symptoms or because the nature of the operations precludes the reporting of precise incidences. We should also consider these facts when analysing similar events in commercial diving. However, we can assume that the frequency of CNS oxygen toxicity incidents is below 3.5% of hyperbaric oxygen exposures.
In addition, based on the procedures from doctor Arieli described above, they say that an estimation of the likelihood of CNS toxicity in diving, as Z-value in a normal distribution can be made with the time " t " in minutes and the PO_2 in kPa, using the formula Z = (ln " t " − 9.63 + 3.38 × ln "PO2") / 2.02. Note that in the formula "ln" means "natural logarithm". They also say that there is no consensus regarding a maximum acceptable risk.

In summary, Central Nervous System (CNS) oxygen toxicity is unpredictable and has considerable variability:

- Some people have great tolerance to oxygen, and others very little: According to some medical manuals, the dose of oxygen required to produce a reaction in a given person can vary as much as eight times.
- The sensitivity is greater at work than at rest and in water than in a chamber.
- Factors such as emotion, fatigue, illness, and hangover may also increase the sensitivity to oxygen.
- Reducing the partial pressure of oxygen and the exposure time allows controlling the phenomenon.

Treatment

For any symptoms of acute O_2 poisoning, the procedure is to remove the O_2 supply and continue on a normoxic mix (A normoxic mix is a mix having a normal oxygen concentration, typically 21% for surface-supplied diving).

If the symptoms happen in the chamber or in the bell during normal O_2 stops:

- Remove the O_2 mask, the diver breathes air for 15 minutes, then resume the decompression at the point of interruption. Generally, it will not happen again, but the diver must be followed . In the case a 2nd crisis starts, the decompression will have to be completed on air.
- Convulsions may occur during oxygen breathing and a few minutes after oxygen breathing has been stopped. If a convulsion occurs, the attendant must prevent the diver from injury himself (guide but not restrict movements). The attendant must not try to maintain the mouth of the casualty open. If the opportunity arises, a knotted handkerchief or similar is installed between the jaws to prevent the tongue from being bitten. Breathing must be continuously monitored. The diver will normally recover from the coma unharmed.

- Important: DO NOT attempt to decompress a diver during a convulsion: The casualty will be unable to exhale with the high risk to create a pulmonary barotrauma. The ascent to the next stop must begin only after full recovery, and the patient is relaxed.
- If the decompression has to be completed on air, use the " Standard air table" (in-water air) or double the O_2 stops.

If the symptoms happen during the dive or the in-water stops

- The O_2 or nitrox supply must be stopped, and the helmet flushed with air.
- If the diver is at work, he ascents to the basket which should be stored above him (that should reduce the partial pressure of O_2). Example : At 20 msw with a mix 40% O_2 the PPO_2 is 1.2 bar, if the diver ascents to 15 msw, the PPO_2 is 1 bar
- To move from the "in water O_2 deco" table to the air table, the procedure is similar to the switching from the "surface O_2 deco" to "Standard Air table ".
- The stand by diver must be sent to assist the diver.
- If the diver was using nitrox and has been passed on air when at depth. The decompression table to apply is the air decompression table for the actual depth of the diver. If the diver has been passed on air when the "equivalent air dive" level has been reached or passed, the decompression to apply is the one corresponding to the equivalent air dive level.
- For minor symptoms, wait for the symptoms to subside then wait 15 more minutes, and recommence O_2 at the point of interruption. Or, switch immediately to the "Standard air table" and resume the decompression using this table.
- Important point: Surface decompression must be envisaged instead of switching to air, even for trivial cases. It must be organized for all cases which could become more serious. In the eventuality that the incommoded diver is vomiting in his helmet, or has a deep crisis, the things can very quickly become unmanageable with additional risks like drowning or injuries for the casualty in addition to the problems posed by the O_2 poisoning... Prudence must be the rule! Because the Air stops and a part of the O_2 stops have normally been completed, switching from the in water or wet bell O_2 decompression to the surface O_2 decompression table is easy and does not pose any problems. The in water air stops prior to the deco time in the chamber of the surface O_2 decompression table are the same as for the "in water Air O_2" decompression tables, allowing to jump from one to the other. What is important is to be sure that the deco time corresponding to the air stops of the surface decompression table are fully completed before ordering the transfer to the chamber.
- Another essential point: In the eventuality that the air stops in the water cannot be completed, the diver will have to be treated for omitted decompression after the transfer in the chamber. Before starting the treatment on O_2, the team must ensure that the casualty is relaxed and the interval of 15 min without breathing O_2 is completed . For safety reasons the diving doctor must be consulted without delay.

If the symptoms happen during hyperbaric treatment for Arterial Gas Embolism, or Decompression Illness:

The procedure indicated by the designers of the US Navy medical tables are the following:

- For Treatment Tables 5, 6, and 6A:
  1) Remove the mask
  2) After all symptoms have completely subsided, decompress 10 feet at a rate of 1 fsw/min. For convulsion, begin travel when the patient is fully relaxed and breathing normally.
  3) Resume oxygen breathing at the shallower depth at the point of interruption.
  4) If another oxygen symptom occurs after ascending 10 fsw, contact the hyperbaric physician appointed by the company to have appropriate modifications to the treatment schedule.

  In the eventuality that the medic cannot be contacted and O_2 breathing cannot be restored within 2 hours, the procedure will be to switch to the comparable air table at current depth for decompression if 60 fsw or shallower (table 7 to be considered). The rate of ascent must not exceed 1 fpm between stops. If symptoms worsen and an increase in treatment depth deeper than 60 feet is needed, Treatment Table 4 shall be used.
- For Treatment Tables 4, 7,and 8:
  1) Remove the mask.
  2) Consult the hyperbaric physician appointed by the company before administering further oxygen breathing. No compensatory lengthening of the table is required for interruption in oxygen breathing. The same procedure as above has to be applied in the eventuality the hyperbaric doctor cannot be contacted and the O_2 breathing cannot be restored within 2 hours.
- Note: The procedure for table 5, 6, 6A can be applied with COMEX tables Cx12, Cx18, and Cx 30. The procedure for tables 4, 7, and 8 can be applied with COMEX tables Cx30 sat.

Prevention

Based on the studies discussed above and many others, and as already suggested, two strategies are implemented conjointly: Reducing the oxygen partial pressure and limiting the exposure times

In his book "Barometric pressure", Paul Bert already suggested that a limitation of the oxygen partial pressure is

necessary to avoid the effects of Central Nervous System (CNS) oxygen toxicity.
A lot of experiments have been then undertaken to discover the best operational limits to not being incommoded by the effects of acute oxygen poisoning, which resulted in the studies already discussed and many others.
This research was mainly conducted by militaries who used closed-circuit oxygen rebreathers for their operations.
As a result of the early experiments, it has been a long time considering that 1.6 bar was a suitable maximum pressure at work and that in-water stops at 1.9 bar/ata and in-chamber stops at 2.8 bar/ata were acceptable. However, for a few years, there has been a consensus that 1.3 bar is the limit allowing long exposures without triggering acute oxygen poisoning.
An example already discussed is the study *"Oxygen Toxicity and Special Operations Forces Diving: Hidden and Dangerous"*, where doctors Thijs T. Wingelaar, Pieter-Jan A. M. van Ooij, & Rob A. van Hulst say that no oxygen-induced convulsions have been described with a PO_2 lower than 1.3 bar in humans.
In another study called "*Pulmonary effects of repeated six-hour normoxic and hyperoxic dives"*, doctors Barbara E. Shykoff & John P. Florian examined differential effects of immersion, elevated oxygen partial pressure, and exercise on pulmonary function after a series of five daily six-hour dives at 130 kPa (1.3 bar) that did not result in acute oxygen poisoning. Note that Barbara Shykoff did other experiments at 1.35 ata that also did not result in CNS toxicity.
These discoveries are reinforced by the publications of scientists such as Ran Arieli, already discussed, whose estimation curves issued from his papers are displayed in the description of this phenomenon.
Based on these studies, the US Navy has limited the maximum partial pressure at work of surface-supplied diving operations to 1.4 ata, and those with Electronically Controlled Closed-Circuit Underwater Breathing Apparatus (EC-UBA) to 1.3 ata. However, the US Navy has kept the in-water oxygen stops at 30 and 20 feet.
A lot of organizations have also adopted the limitation at 1.4 bar, such as the Diving Medical Advisory Committee (DMAC) through the guidance *"Oxygen content in open circuit bail-out bottles for heliox saturation diving"*, or IMCA that, in addition to recommending 1.4 bar as the upper limit for partial pressure of oxygen in the nitrox mix breathed by the diver when at depth if using surface-supplied diving techniques, says that higher partial pressures than 1.4 bar can be used for the decompression stops. Also, in its *"Diving Standards & Safety manual*", NOAA (National Oceanic and Atmospheric Administration - USA) says that the PO_2 of any gas mixture breathed during a dive must not exceed 1.4 absolute atmospheres (ata), except during the decompression phase when a PO2 of 1.6 is allowed. For information, this limitation of the in-water stops to 1.6 ata or bar is not new, as it was already in force with COMEX offshore since the seventies. It is the limitation adopted in this handbook.

Paul Bert has also demonstrated the effects of the exposure time according to the oxygen pressure in his book *"Barometric pressure"*.
Studies have then been undertaken to correlate oxygen pressure with a maximum exposure time. They have resulted in equations that can be used to elaborate safety curves, such as those indicated previously.
These studies have also resulted in the discovery that periods of normoxic intake in between periods of oxygen breathing allow to diminish the effects of oxygen toxicity, and thus, is an efficient technical approach for increasing the total time of exposure to hyperoxia. As an example, in an article called *"Extention of oxygen tolerance in man (predictive studies VI)"*, doctors Lambertsen and Clark tested several variations of intermittent exposures at 2 ata. Doctors Lambertsen and Clark conducted a lot of other studies regarding the safe extension of oxygen exposure, such as the study called *"Optimization of oxygen tolerance extension in rats by intermittent exposure"* (J. M. Clark, C. J. Lambertsen, R. Gelfand, and A. B. Troxel). As also said by doctor Bitterman in a paper called *"CNS oxygen toxicity"* or doctor Manning in his study *"Central Nervous System Oxygen Toxicity and Hyperbaric Oxygen Seizures"*, there is a consensus to consider that intermittent exposure procedures prolong the time of oxygen exposure without being affected by O_2 poisoning.
Periods of oxygen and normoxic breaks vary according to the tables.

A method of evaluation that can be used for controlling the duration of exposure is the one described below, which has been published by NOAA *(National Oceanic and Atmospheric Administration - USA)*.

PPO2	*Max. single exposure (min)*	*Maximum per 24 hr (Min)*
1.6	45	150
1.55	83	165
1.5	120	180
1.45	135	180
1.4	150	180
1.35	165	195
1.3	180	210
1.25	195	225
1.2	210	240
1.1	240	270
1	300	300
0.9	360	360
0.8	450	450
0.7	570	570
0.6	720	720

NOAA says that these estimated oxygen exposure limits, which were 1st published in the 1991 version of their Diving Manual, were developed with the help of experts.

- These limits, displayed in the table on the previous page, are intended for a diver doing dives for research, sampling, inspection, observation, and light to moderate work at the higher PO_2 levels. The lower levels can be used for heavier and more stressful types of work.
- For each level of oxygen, the chart shows an allowable time for a single exposure and also an accumulated time at that level over a full day.
- If more than one dive is made to the maximum exposure of a PO_2 of 1.6 ata, a suggested surface interval of at least 90 minutes is advised between dives (three dives of 45 minutes each would theoretically be possible within the 150-minutes daily total allowed at 1.6 ata PO_2). This helps lower the accumulated oxygen dose. This only applies to the exposure at 1.6 ata, because only one maximal dive can be done in a single day with lower oxygen exposure levels.

If, however, one or more dives in a 24-hour period have reached or exceeded the limits for a normal single exposure, the diver should spend a minimum of two hours at a normoxic PPO_2 (such as on the surface breathing air) before resuming diving. If diving in a 24-hour period reaches the Maximum 24-hour Limit, the diver must spend a minimum of 12 hours at normoxic PPO_2 before diving again.

NOAA exposure limits are also published in percentages of the allowable limit *(see below)*.
This table allows calculating the percentages of the maximum limit for a nitrox dive, or when oxygen decompression is used. The percentages for one minute are the result of 100% divided by the maximum allowable minutes. This result is then multiplied by the bottom times to obtain the desired percentage.
To enter in the table the PPO2 of the mix used must be calculated. The table below can be used:

% mix	*Depth*														
	3 m	*6 m*	*9 m*	*12 m*	*15 m*	*18 m*	*21 m*	*24 m*	*27 m*	*30 m*	*33 m*	*36 m*	*39 m*	*42 m*	*45 m*
25	0.33	0.40	0.48	0.55	0.63	0.70	0.78	0.85	0.93	1.00	1.08	1.15	1.23	1.30	1.38
30	0.39	0.48	0.57	0.66	0.75	0.84	0.93	1.02	1.11	1.20	1.29	1.38			
35	0.46	0.56	0.67	0.77	0.88	0.98	1.09	1.19	1.30	1.40					
40	0.52	0.64	0.76	0.88	1.00	1.12	1.24	1.36							
45	0.59	0.72	0.86	0.99	1.13	1.26	1.40								
50	0.65	0.80	0.95	1.10	1.25	1.40									
100	1.30	1.60	1.90	2.20	2.50	2.80									

When the PPO_2 is calculated, find the corresponding value in the table below (column PPO_2) and follow the line to the crossing with the bottom time duration. As an example, 45 minutes with a PPO2 of 1.6 bar is 100% , so the maximum limit. The values obtained for the bottom times and the stops are added to give an estimation of the percentage of the maximum allowable limit that is planned to be used.

PPO2	*Single dive limit (min)*	*Bottom times*											
		5	*10*	*15*	*20*	*25*	*30*	*35*	*40*	*45*	*50*	*55*	*60*
1.6	*45*	11	22	33	44	56	67	78	89	100	111	122	133
1.55	*83*	6	12	18	25	30	36	42	48	55	61	67	73
1.5	*120*	4	8	13	17	21	25	29	33	38	42	46	50
1.45	*135*	4	7	11	15	19	22	26	30	33	37	41	44
1.4	*150*	3	7	10	13	17	20	23	27	30	33	37	40
1.35	*165*	3	6	9	12	15	18	21	24	27	33	33	36
1.3	*180*	3	6	8	11	14	17	19	22	25	28	31	33
1.25	*195*	3	5	8	10	13	15	18	21	23	26	28	31
1.2	*210*	2	5	7	10	12	14	17	19	21	24	26	29
1.1	*240*	2	4	6	8	11	13	15	17	19	21	23	25
1	*300*	2	3	5	7	8	10	12	13	15	17	18	20
0.9	*360*	1	3	4	6	7	8	10	11	13	14	15	17
0.8	*450*	1	2	3	4	6	7	8	9	10	11	12	13
0.7	*570*	1	2	3	4	4	5	6	7	8	9	10	11
0.6	*720*	1	1	2	3	4	4	5	6	6	7	8	8

Note that NOAA says that no specific laboratory validation of this technique has been undertaken. In addition, such a procedure is not necessary if the policies recommended in the latest *"Diving Standards & Safety manual NOAA"* (1.4 ata at work & 1.6 ata during in-water stops) and also recommended by this handbook are implemented: 1.4 ata/bar is close to 1.3 bar that is a PO_2 recognized not to trigger acute oxygen poisoning, and allow for longer exposures than those allowed by the tables. In addition, when the recommended UK-HSE operational limits (from doctors Shield & Lee) are implemented, the O_2 stop times at 20ft/6m usually are short enough not to trigger oxygen poisoning.

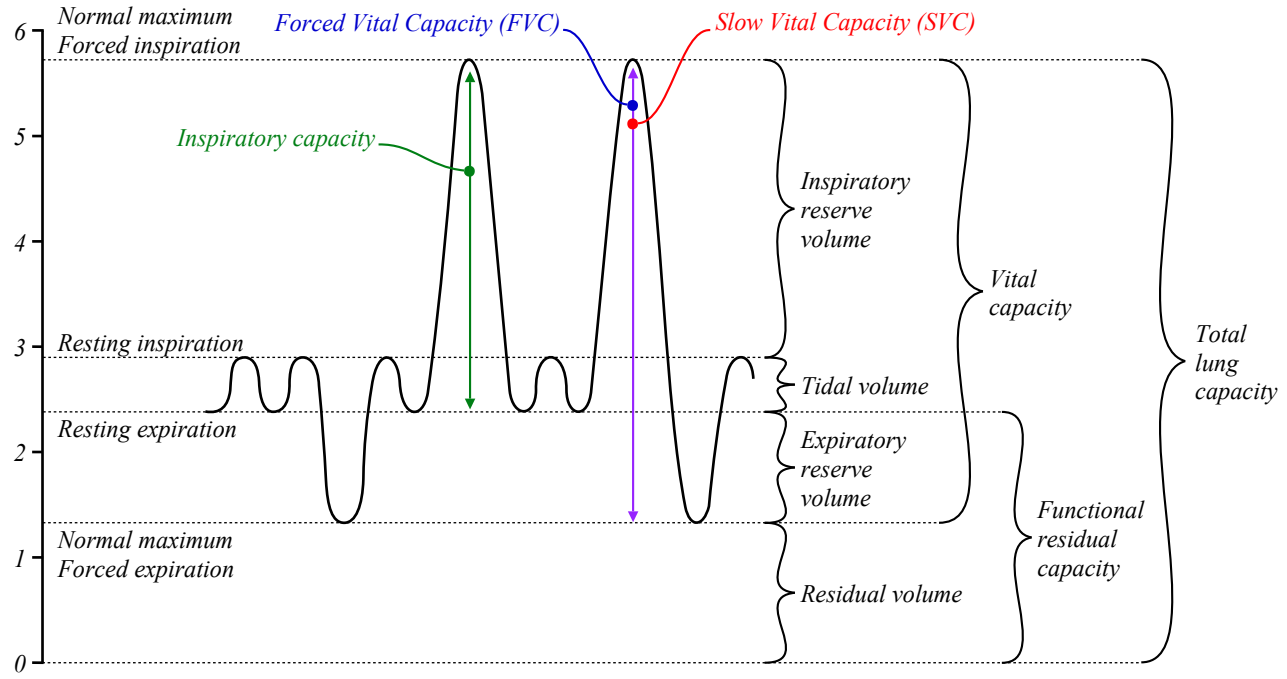
Chronic oxygen poisoning *(Lorrain Smith effect)*

Description

"Chronic Oxygen Poisoning", also called the "Lorrain Smith Effect", or "Pulmonary Oxygen Toxicity", or "Low-pressure oxygen poisoning", is due to prolonged exposure to oxygen at a partial pressure not sufficient to trigger acute oxygen poisoning but sufficient to result in the inflammation of the lungs and loss of breathing capacity. This phenomenon was described 1st by James Lorrain Smith (1862 - 1931), who conducted research on respiration in collaboration with Sir John Scott Haldane (The creator of the 1st decompression tables). As Sir Haldane, James Lorrain smith started his research from the discoveries of Paul Bert he often refers to in his book *"The pathological effects due to increase of oxygen tension in the air breathed"*, and developed the investigations on oxygen described in the book *"Barometric pressure"* in addition to correcting some assumptions emitted by Paul Bert at his time. It is commonly considered that this form of oxygen poisoning starts when the partial pressure of oxygen is above 0.5 bar. However, some specialists believe this value should be reduced.
Investigations on chronic oxygen poisoning have continued with those on acute oxygen poisoning after James Lorrain Smith and are still on the way. As for Central Nervous System (CNS) toxicity, some mechanisms are still unclear.
The decrement of pulmonary capacity has been documented in many documents. As an example, in the study *"Effects of prolonged oxygen exposure at 1.5, 2.0, or 2.5 ATA on pulmonary function in men (Predictive Studies V)"*, doctors J M Clarck, C Lambertsen, R Gelfrand, N D Flores, J B Pisarello, M D Rossman, J A Elias, said that the average Forced Vital Capacity (FVC) and Forced Expiration Volume in 1 second (FEV_1) decrements near the end of O_2 exposure were:

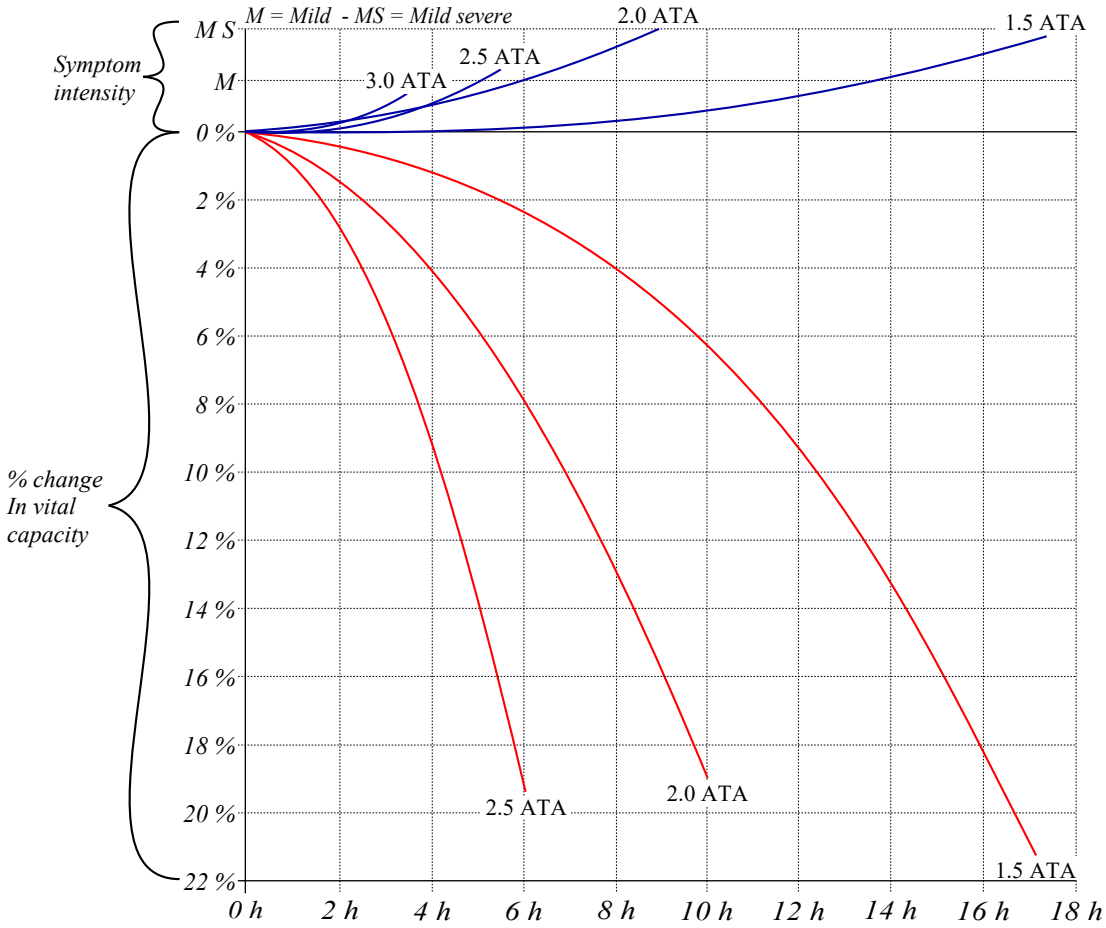
- 20.4% and 14.0% for 17.5 h of O_2 breathing at 1.5 ATA,
- 21.0% and 22.2% for 8.8 h at 2.0 ATA,
- 13.8% and 12.8% for 5.7 h at 2.5 ATA.

For clarification, the Forced Expiratory Volume (FEV1) is the maximal volume of air a person can expel as hard and fast as possible in one second from the point of maximal inspiration. The Forced vital capacity (FVC) is the maximal volume of air a person can expel as fast and hard as possible in one expiration from the point of maximal inspiration to the end of the Expiratory Reserve Volume (ERV). In opposition to FVC, Slow Vital Capacity (SVC) is the maximal volume of air a person can expel in a relaxed manner in one maximal expiration from the point of maximal inspiration to the ERV.
The rate FEV1/FVC is essential in assessing a pulmonary condition. An individual test result is compared to the predicted value based on age, height, and sex. For simplification, we can say that this ratio, called the “Tiffeneau ratio”, is expected to be 70 to 80% in adults and 85% in children.



This report also highlights that most people exposed to 1.5 or 2.0 ATA experienced pulmonary symptoms that included chest pain, cough, chest tightness, and dyspnea at regular intervals during O_2 breathing. Although the combination and severity of pulmonary symptoms varied among individuals, their average intensities were moderately severe by the end of the O_2 exposures at these pressures. In contrast, people who breathed O_2 at 2.5 ATA for 5-6 hours or at 3.0 ATA for 3.5 hours had relatively mild symptoms.

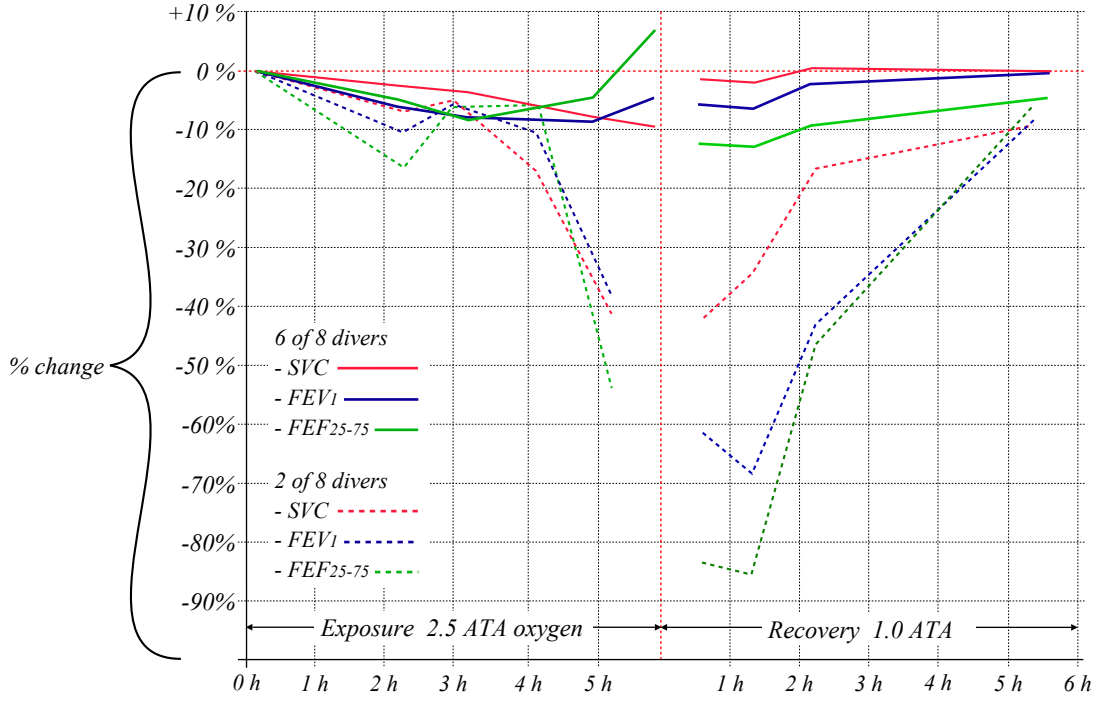
The scheme on the next page shows the rates of development of pulmonary symptoms and Slow Vital Capacity (SVC) decrements during continuous O_2 exposures at 3.0, 2.5, 2.0, and 1.5 ATA. Nevertheless, SVC was not measured during 3.0-ATA exposures. These curves were drawn through the average SVC data and symptom scores. At each O_2 pressure, a decrease in SVC started before the onset of symptoms and became significant while symptom intensity was still mild. Although average rates of symptom development correlated with average rates of SVC reduction, this did not always occur in individual subjects. These results are slightly different but correlate those with FVC discussed above.



Note that doctors J M Clarck, C Lambertsen, R Gelfrand, N D Flores, J B Pisarello, M D Rossman, J A Elias, said that some recovery rates were about 5.7 h after breathing O_2 during 5.7 h at 2.5 ATA and 8.4 hours after 8.8 h exposures at 2.0 ATA. However, they also said: *"It is important to be aware that continuous exposures beyond the PO2 duration limits selected for this study could produce more severe effects of O_2 toxicity that are not completely reversible".*

The scheme below, also from the paper *"Effects of prolonged oxygen exposure at 1.5, 2.0, or 2.5 ATA on pulmonary function in men (Predictive Studies V)"*, shows the pulmonary function changes during and after O_2 breathing at 2.5 ATA for 5 - 6 h, and the comparison of the average changes in SVC, 1-s forced expired volume ($FEV_{1.0}$), and maximal mid-expiratory flow rate ($FEF_{25–75}$) for 2 of 8 subjects who had unusually large deficits, compared with the 6 other subjects who had much smaller changes. The team calculated the changes according to control measurements performed during pre-exposure and at the start of exposures. Note that at the end of exposure, the $FEF_{25–75}$ comes above the initial value.

Note: The Forced Expiratory Flow (FEF_{25-75}) is the rate of airflow recorded to measure the Forced Vital Capacity (FVC), it is usually calculated as an average flow over a portion of the expiratory curve. Thus, 25-75 shows the portion in percentage of the curve considered. 25-75% of the FVC is called the "Maximal Mid-expiratory Flow".



Regarding the duration of the recovery phases presented in this scheme, we must take into account that doctors J M Clarck, C Lambertsen, R Gelfrand, N D Flores, J B Pisarello, M D Rossman, J A Elias also said that recovery from pulmonary O2 poisoning has to be considered a complex process that involves several overlapping sequences that include rapid reversal of early intracellular biochemical events, slower recovery from cellular structure and function alterations, and more severe exposures may involve the repair of irreversible structural damage.
Even with the fully reversible degrees of pulmonary O2 poisoning to which the people involved in the experiments mentioned above were exposed, the rates and patterns of recovery varied among different components of measurable pulmonary functions.

Based on the fact already mentioned that 1.3 and 1.4 bar are, depending on the time of exposure, considered safe limits regarding acute oxygen poisoning, numerous experiments with a partial pressure of oxygen limited to 1.3 bar or slightly above have been performed by militaries to investigate the possibilities of exposing divers to these values during war operations without irremediably affect them with chronic oxygen poisoning. As an example, in the US Navy report *"Repeated 4 hours dives with PO2 = 1.35 atm"*, doctor Barbara Shykoff describes multiple experimental four-hour resting dives with a partial pressure of 1.35 atmosphere, performed with surface intervals of 44 hours for 18 divers and 20 hours for 17 divers. Note that the divers were permitted to surface, breathe room air, and eat or drink for no more than five minutes per hour. The following maximum decreases in pulmonary functions were logged:

- Forced Expiratory Volume in 1 second (FEV_1) = 11.2%
- Forced Vital Capacity (FVC) = 16.2%
- Forced Expiratory Flow (FEF) = 22%

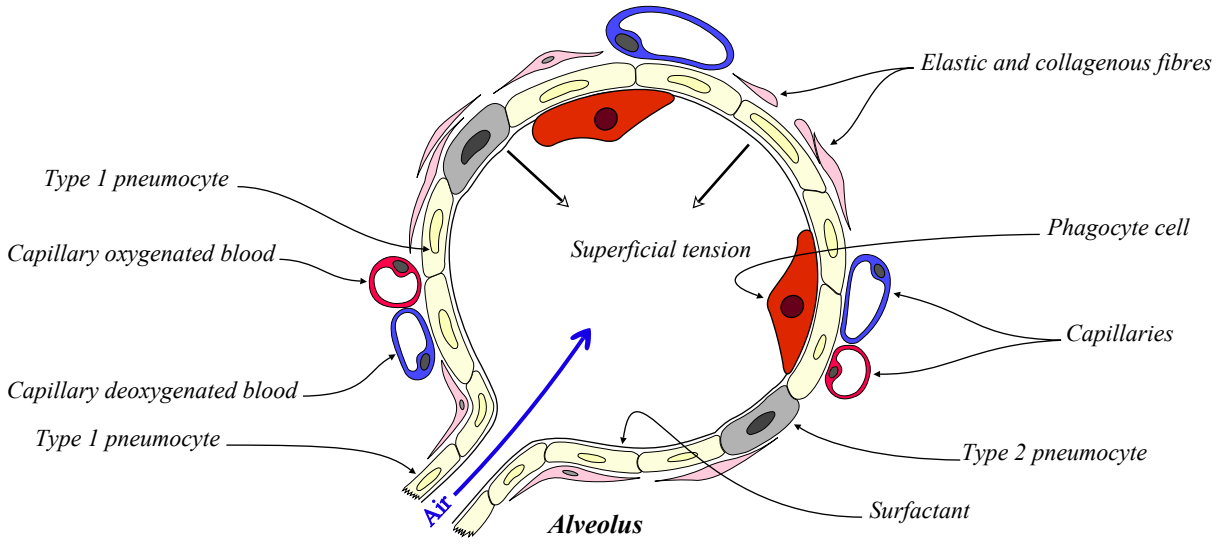
Chest tightness, cough, and inspiratory burning were the main symptoms recorded. Their intensity varied from "moderate" to "mild", with a majority of reported severities classified "Mild".
In the discussion regarding these experiments, doctor Shykoff said the following:

- *All pulmonary function values were within the normal range around baseline by ten days after diving; we were unable to measure daily to follow the time course of recovery. For most subjects, variables were within the normal range around baseline by three to four days after diving, when all symptoms had resolved.*
- *We were surprised to see no accumulation of pulmonary injury with even five days of diving. The 20 hours between dives may be sufficient for recovery. In fact, divers commented that the diving seemed more comfortable after the third dive of the five, and that a second dive (with a 20-hour surface interval) removed the symptoms caused by the previous dive. None of the measurements is sensitive enough to detect an acclimatizing effect, but such an effect is plausible. By increasing the production of protective materials after repeated exposures, the body can protect itself from many mild injuries.*
- *Repeated four-hour dives did not cause significant change in visual refraction. One diver showed a definite, small refractive change, and one a short-term change in visual acuity without a change in refraction.*

To conclude Doctor Shykoff said:
Although single four-hour dives at PO_2 = 1.35 atm can provoke mild to moderate respiratory symptoms and changes in pulmonary function variables, repeated dives with a 20-hour surface interval do not appear to increase the incidence of respiratory problems in comparison to that of single dives. A 44-hour surface interval is not notably better than a 20-hour interval. Middle ear and skin problems must be considered, and, until hyperoxic myopia in the water is better understood, vision should be monitored if dives are to be repeated more than twice in three days.

Even though the mechanism of Chronic oxygen poisoning is not fully identified, its evolution is precisely documented. As an example, in their paper *"Oxygen Toxicity and Special Operations Forces Diving: Hidden and Dangerous"*, already mentioned to describe acute oxygen poisoning, doctors Thijs T. Wingelaar, Pieter-Jan A. M. van Ooij, and Rob A. van Hulst, say that the evolution pulmonary oxygen toxicity can be divided into two phases:

1. The "exudative phase" is marked by local inflammation with capillary and endothelial* edema*, a decrease of type I pneumocytes (alveolar cells)*, and an influx of inflammatory cells. These changes are reversible and the lung returns to its normal state if the inspired oxygen pressure is reduced below 0.5 ATA.



Definitions*:

- Edema: Medical term for swelling caused by fluids trapped in tissues.
- Endothelial cells: They form a single cell layer that lines blood vessels and regulates exchanges between the bloodstream and the surrounding tissues (See in the chapter "Circulatory and Respiratory systems' at the beginning of this book).
- Type 1 pneumocytes: The alveolar cells that make the thin coreless cytoplasmic membrane that composes the structure of the alveolus at 90-95%.

2. In the following proliferative phase fibroblasts and type 2 pneumocytes (alveolar cell) infiltrate the inflamed endothelia. With continuing inflammation, this ultimately leads to alveolar fibrosis and a four to fivefold increase of thickness of the air-blood membrane and, as a consequence, loss of diffusion capacity. These changes are irreversible. The rate at which these changes occur is directly related to the inspired PO_2 and can occur as early as 3 h at a PO_2 of 3 ATA during a dry dive.
   Definitions*:
   - Fibroblast: Fibroblasts are cells contributing to the formation of connective tissue. These cells secrete collagen proteins that help maintain the structural framework of tissues.
   - Type 2 pneumocytes: They are cells that secrete a substance composed of proteins and lipids called "surfactant". This substance reduces the superficial tension of pulmonary fluids and contributes to the elastic properties of the lungs. Note that type 2 pneumocytes can replicate and replace damaged type 1 pneumocytes. (see the description at the beginning of this book).

As a summary it is considered that exposures to the conditions mentioned in the examples described above result of recoverable decrements of the Vital Capacity (VC), however the variability of people regarding oxygen is to be considered as well as the conclusions from doctors J M Clarck, C Lambertsen, R Gelfrand, N D Flores, J B Pisarello, M D Rossman, & J A Elias.

The visible symptoms are linked to the exposure to oxygen and the degrees of damage :

- Mild tickling or irritation in the trachea and bronchi, usually with a slight cough
- Severe, constant burning in the chest
- Uncontrollable cough
- Shortness of breath, even at rest.

Means of control

Similarly to the strategy developed for acute oxygen poisoning, the means of control of chronic oxygen poisoning consist of limiting the oxygen partial pressure and the times of exposure.

Partial pressure limitation

Since the 1st experiments, there has been a consensus to consider that 0.5 Atmospheres absolute (or 0.5 bar) is the partial pressure of oxygen below which the effects of chronic oxygen poisoning described above are not noticeable. However, many scientists recommend lower values for long exposures. For example, in a paper published in 1972 called "*Use of the University of Pennsylvania Institute for environmental medicine. Procedure for calculation of cumulative oxygen toxicity*", doctor W. Brandon Wright, a scientist who worked with doctor Christian Lambertsen said:
"The lower level of inspired oxygen pressure which will cause progressive lung toxicity, or the maximum PO_2, which will cause no toxicity for very long exposures, is not known with certainty. Normal men have breathed oxygen at 0.5 ATA for several days without detectable pulmonary changes. Therefore, this assumption cannot be used as evidence to choose 0.5 ATA of oxygen as a safe pressure for very long exposures such as might be encountered in deep saturation diving. Thus, we will continue to recommend a PO2 of 0.30 to 0.35 Ata for saturation dives. However, a PO2 of 0.5 ATA should be a reasonable asymptote for exposures of the durations encountered in hyperbaric therapy procedures". Regarding the recommendations above, it must be noted that the present oxygen partial pressure of the US Navy saturation procedures is 0.44 - 0.48 ATA during the rest periods at the storage depth. By contrast, the values we suggest with the saturation procedures Norman 15 are close to those initially recommended by doctor Wright.

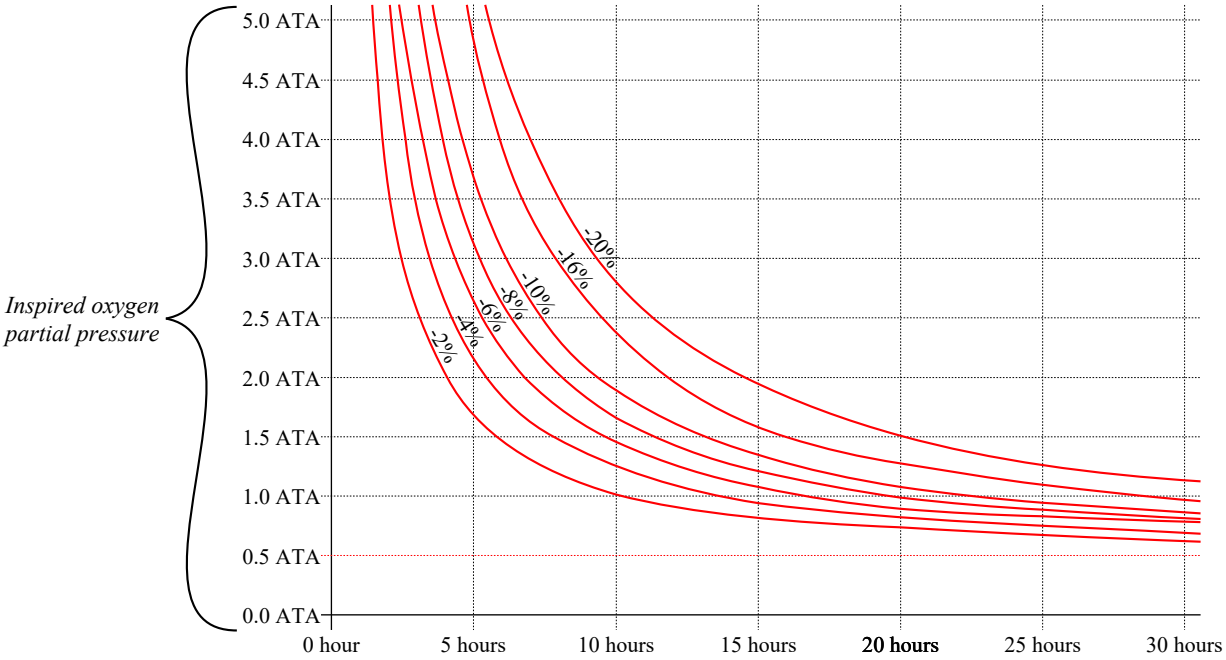
Limitation of exposure - The Unit Pulmonary Toxic Dose (UPTD) concept

In the document *"Use of the University of Pennsylvania Institute for environmental medicine. Procedure for calculation of cumulative oxygen toxicity"*, mentioned above, doctor W. B Wright said that the severity of lung changes caused by hyperoxia depends on the dose of oxygen, which can be described in terms of partial pressure and duration of breathing. Based on this fact, he considered that it would be relatively easy to establish a table stating the exposure to oxygen in minutes at given partial pressures that are considered safe, and imagined a method for calculating the cumulative pulmonary toxicity that may be expected following exposure to oxygen at various pressures.
Note that doctor W. B Wright did not start his work from scratch, and based on the following studies:

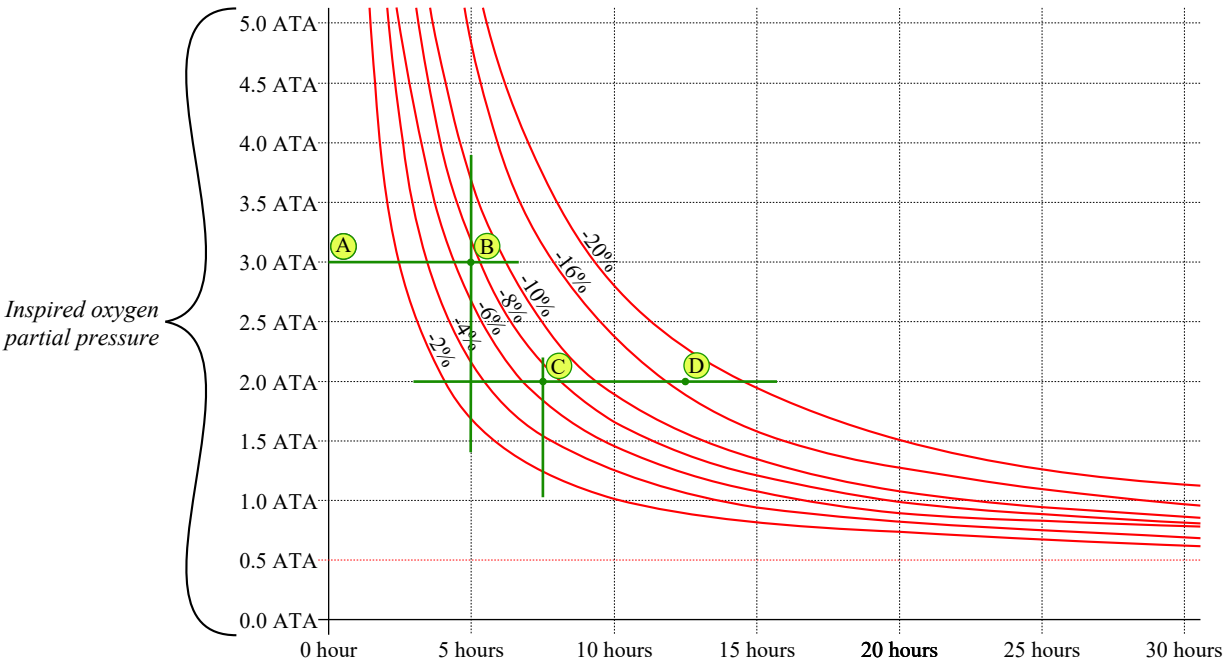
- *"The Rate of Development of Pulmonary Oxygen Toxicity in Man"* - Authors: J. M. Clark and C. J. Lambertsen - Published in 1967.

- *"Basic Requirements for Improving Diving Depth and Decompression Tolerance"* - Author C. J. Lambertsen - Published in 1967.
- *"Alveolar-Arterial O2 Differences in Man at 0.2, 1.0, 2.0 and 3.5 Ata Inspired PO2"* - Authors: J. M. Clark, C. J. Lambertsen - Published in 1971.
- *"Rate of Development of Pulmonary O2 Toxicity in Man during O2 breathing at 2 0 Ata"* - Authors: J. M. Clark, C. J. Lambertsen - Published in 1971.
- *"Quantitative Definition of Pulmonary Oxygen Toxicity in Normal Men in Continuous Multilevel Exposures to High Oxygen Pressure"* - Authors: J. M. - Published in 1971.
- *"Pulmonary Oxygen Toxicity A Review"* Authors: J. M. Clark, C. J. Lambertsen - Published in 1971.
- *"Derivation of Pulmonary and Central Nervous System 0] Tolerance Curves in Man"* - Authors: C. J. Lambertsen , & J. M. Clark - Published in 1972.

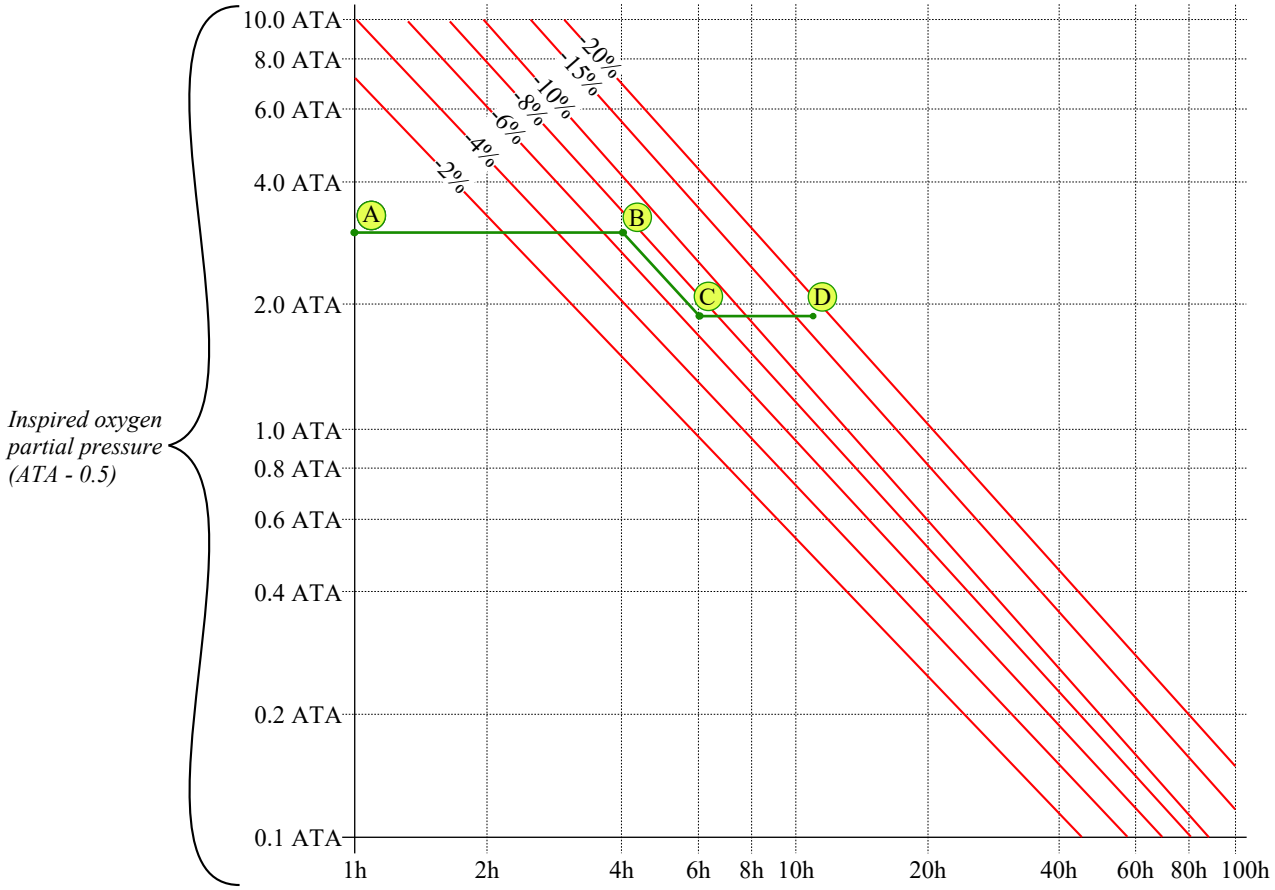
Doctor Wright's research was based on measuring the subjects' decreases in pulmonary function. He said he used the Vital Capacity (VC) assessment because it could be rapidly and accurately measured, was repeatedly reproducible, and reflected the onset, degree, and rate of pulmonary involvement. These measurements made it possible to assess the degree of pulmonary toxicity from a given exposure using curves like those shown below.



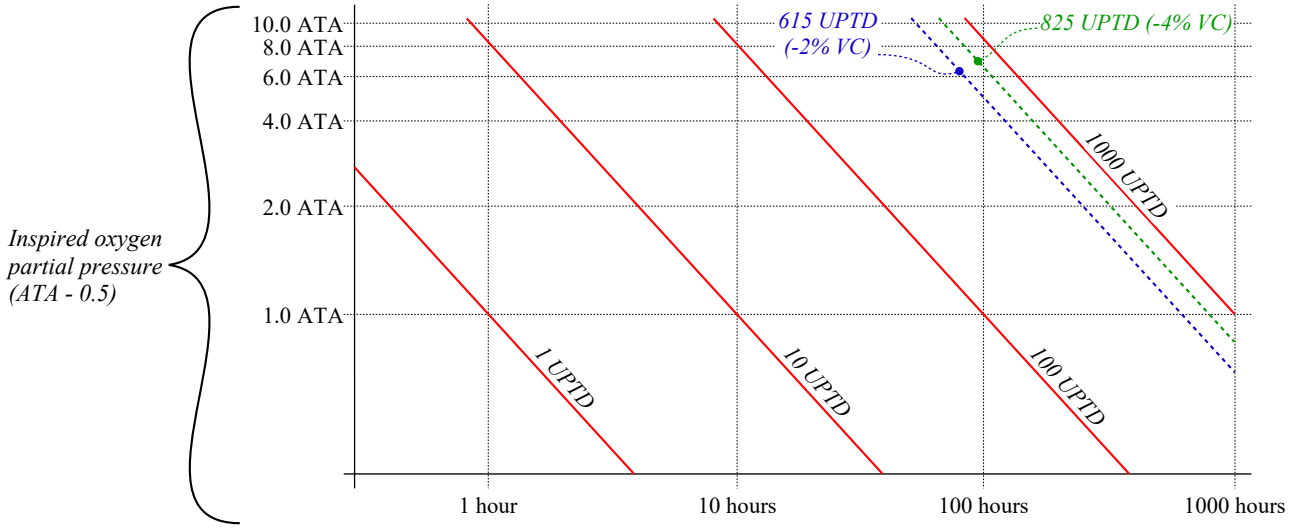
However, he also said that using this graph in estimating the cumulative damage incurred by uninterrupted exposure to several different partial pressures of oxygen is complex. For example, if a subject is exposed to 3 ATA oxygen pressure for five hours and then to oxygen at 2 ATA for five more hours, he would incur a more significant pulmonary detriment than a subject who breathed oxygen continuously at 2 ATA for ten hours: After five hours at 3 ATA, the subject has 7% decrease (*see B below*). When the PO2 is reduced to 2 ATA he still has a 7% decrement (*See C*). Thus the 5 hours of 3 ATA are equivalent to 7 or 8 hours at 2 ATA. The subsequent 5 hours at 2 ATA gives 17% decrement *(see D).*



This approach assumes that pulmonary oxygen damage produced at one pressure is additional to toxicity at other pressures. In summary, the point describing a person's exposure moves along a horizontal line as the duration of exposure continues at a constant PO2. It moves up and down as PO2 changes and is not moved along a vertical line, and thus, is not easy to calculate.
For this reason, it has been considered that a logarithmic transformation of these curves would result in linear curves that are easier to establish. This method for estimating the total oxygen dose, the lung damage that may be occurring, and the approximate safe limit of exposure is called the "Unit Pulmonary Toxic Dose (UPTD) concept".
One UPTD is defined as the degree of pulmonary toxicity incurred by breathing 100% oxygen at a partial pressure of 1 ATA for one minute. It is assumed that this same toxicity can be achieved by exposures to various other combinations of PO2 and duration of exposure. Doctor Wright set the lowest PO2 which will produce any pulmonary toxicity within a finite period of time to 0.5 ATA, with the reserves mentioned previously regarding saturation diving.
In this situation, all combinations of PO2 and time which have a toxicity of 1 UPTD fall along the equation below:
$\log (P - 0.5\ ATA) = m \log t + \log b$, which may be rewritten as: $P - 0.5\ ATA = bt^m$,
where " P " = inspired PO2 in ATA, " t " = time in minutes, " b " = intercept constant for t = 1, " m " = slope constant.
This logarithmic formula allows plotting the pulmonary oxygen tolerance lines to graphically determine the cumulative pulmonary toxicity, such as the one below that reuses the example on the previous page.



In complement, doctor Wright said that breathing 1 ATA PO2 for two minutes produces 2 UPTD. Thus, a curve has the same slope as the I UPTD curve but with a larger intercept constant which includes all combinations of PO2 and time, which have a toxicity of UPTD can be drawn as the example below.



Since all points along a line represent the same pulmonary toxic dose, the points on one curve are also assumed to represent an identical influence of oxygen upon pulmonary function. Thus the O_2 tolerance lines shown in the scheme can express in terms of a certain number of UPTDs corresponding to decrement in the Vital Capacity (VC). Regarding these decrements, doctor Wright provided the following values:

UPTD	*Decrement Vital Capacity (VC)*	*UPTD*	*Decrement Vital Capacity (VC)*	*UPTD*	*Decrement Vital Capacity (VC)*	*UPTD*	*Decrement Vital Capacity (VC)*
615	*2%*	*1035*	*6%*	*1425*	*10%*	*2190*	*20%*
825	*4%*	*1230*	*8%*	*1815*	*15%*		

To continue his demonstration, doctor Wright said the following:
The lines of equal pulmonary toxicity are linear, parallel and have the same asymptotes of zero time and 0.5 ATA. Therefore, any exposure in terms of PO_2 and time can be expressed in UPTD units. It is assumed that these units may be added to describe a total exposure pattern.
To convert an exposure of any PO_2 = P2 and any time = t2 to a dose of equal toxicity at some P1 and t1, simply perform the division:

$$\frac{P1 - 0.5\ ATA}{P2 - 0.5\ ATA} = \frac{bt1^m}{bt2^m}$$

Which can be solved for t1 to give:

$$t1 = t2 \sqrt[m]{\frac{P1 - 0.5\ ATA}{P2 - 0.5\ ATA}}$$

where *" P "* = inspired PO_2 in ATA, *" t "* = time in minutes, *" b "* = intercept constant for t = 1, *" m "* = slope constant. When t2 is expressed in minutes, P2 in Ata, and P, is set equal to 1 Ata; then t1 will be the number of UPTD units incurred by exposure (P2, t2).

$$t1 = t2 \sqrt[m]{\frac{0.5\ ATA}{P2 - 0.5\ ATA}}$$

On the basis of empirical evidence currently available the value of m = -1.2 has been selected as the pulmonary index of toxicity in man. Therefore to calculate the pulmonary toxic dose of oxygen for any exposure to oxygen at PO2 = P2 and duration t2 , solve only the following equation:

$$UPTD = t2 \sqrt[-1.2]{\frac{0.5}{P2 - 0.5}} = \frac{t2}{\sqrt[-1.2]{\frac{0.5}{P2 - 0.5}}}$$

These equations are complex for those who are not used to mathematics. For this reason, and to simplify the implementation of the procedure, doctor Wright published tables based on the function above that have been further developed later by other scientists and are still in force with organizations such as NOAA. They are explained on the next page. However, we have published these mathematical formulas as it is essential to understand the process that triggered this concept's creation and, thus, the exposure limitations tables commonly used by many diving teams.

To explain the practical applications and the limits of this concept, doctor Wright said the following:
"It is routine and reasonable in surface supplied helium-oxygen helmet diving and in treatment of decompression sickness to increase the speed and safety of decompression by using elevated oxygen pressures. One can sensibly accept minor degrees of pulmonary toxicity if they are completely reversible and asymptomatic.
The degree of pulmonary toxicity equivalent to a two percent decrease in vital capacity is completely reversible, asymptomatic and even impossible to measure under ordinary circumstances. A UPTD of 615 will produce this degree of pulmonary toxicity. Therefore, a UPTD of 615 or less is a reasonable maximum limit of oxygen exposure for treatment of uncomplicated decompression sickness or other treatment procedures.
When elevated pressures of oxygen are used in the treatment of more serious diseases such as severe decompression sickness or gas gangrene, it may be reasonable to access a greater degree of pulmonary toxicity in order to better treat the illness.
The primary requirement of any therapy is that the treatment not be worse than the disease. Since high pressures of oxygen are so useful in treating serious decompression sickness it is reasonable to use a greater dose of this agent than would be sensible to hasten decompression.
The degree of pulmonary oxygen toxicity which produces a ten percent decrease in vital capacity is associated with moderate symptoms of coughing and pain in the chest on deep inspiration.
This degree of impairment of lung function has been shown experimentally to be reversible within a few days following cessation of exposure to elevated oxygen pressures. However, symptoms and signs of pulmonary toxicity can progress for a few hours following the termination of the elevated oxygen exposure. Greater oxygen exposures may not be reversible. Therefore, it is suggested that a 10% decrement in vital capacity or a UPTD of 1425 be chosen as the extreme limit for hyperbaric oxygen therapy procedures.
It is possible that oxygen exposures which produce reversible changes in pulmonary functional measurements may be associated with chronic histologic changes in the lungs. Whether this is true or not is unknown, but the possibility should council caution against the indiscriminate use of elevated PO2."

Tabular method for calculation of the Unit Pulmonary Toxic Dose (UPTD):

With this method, the UPTD is calculated by using tables given for oxygen exposures from 0.6 atmospheres to 5.0 atmospheres in increments of 0.1 Ata. *Note that these tables are displayed at the end of this book.*

The times are expressed in minutes with the corresponding UPTD, with a maximum of sixty minutes. If an exposure is longer than sixty minutes, one may convert the time of the stop to hours and minutes and enter the table twice, once for the number of hours to get UPTD in hours and once for the remaining time in minutes to get UPTD in minutes.

1. Convert the partial pressure of oxygen breathed at each depth to PO_2 in atmospheres.
2. Select the appropriate PO_2 table *(see the table below for 0.6 ATA)*.
3. Enter the table in the "time" line at the time corresponding to the duration stop.
4. Read the corresponding UPTD.
5. Add the UPTD's for each depth together to get the total UPTD for the exposure.

Unit Pulmonary Toxic Dose - PP oxygen 0.6 ata

Time (minutes)	***1***	***2***	***3***	***4***	***5***	***6***	***7***	***8***	***9***	***10***
UPTD	*0.26*	*0.52*	*0.78*	*1.05*	*1.31*	*1.57*	*1.83*	*2.09*	*2.35*	*2.62*
Time (minutes)	***11***	***12***	***13***	***14***	***15***	***16***	***17***	***18***	***19***	***20***
UPTD	*2.88*	*3.14*	*3.4*	*3.66*	*3.92*	*4.18*	*4.45*	*4.71*	*4.97*	*5.23*
Time (minutes)	***21***	***22***	***23***	***24***	***25***	***26***	***27***	***28***	***29***	***30***
UPTD	*4.49*	*5.75*	*6.02*	*6.28*	*6.54*	*6.8*	*7.06*	*7.32*	*7.58*	*7.85*
Time (minutes)	***31***	***32***	***33***	***34***	***35***	***36***	***37***	***38***	***39***	***40***
UPTD	*8.11*	*8.37*	*8.63*	*8.89*	*9.15*	*9.42*	*9.68*	*9.94*	*10.2*	*10.46*
Time (minutes)	***41***	***42***	***43***	***44***	***45***	***46***	***47***	***48***	***49***	***50***
UPTD	*10.72*	*10.98*	*11.25*	*11.51*	*11.77*	*12.03*	*12.29*	*12.55*	*12.82*	*13.08*
Time (minutes)	***51***	***52***	***53***	***54***	***55***	***56***	***57***	***58***	***59***	***60***
UPTD	*13.34*	*13.6*	*13.86*	*14.12*	*14.38*	*14.65*	*14.91*	*15.17*	*15.43*	*15.69*

It is also permissible to break a single exposure to the same PO2 down into partial times small enough to be found in the tables and then add each corresponding UPTD to find the total UPTD of the stop.

Simplified arithmetic method for calculation of the Unit Pulmonary Toxic Dose (UPTD):

This procedure can be use in place of the previous one. It has the advantage of using only one table. To explain it, Doctor Wright referred to the linear function for calculating UPTD, which formula,can be expressed as follows:

$$UPTD = t \sqrt[-1.2]{\frac{0.5}{P - 0.5}}$$

This formula can be reduced to

UPTD = KP x t When " P " is held constant

As a result, at any PO2, a factor " KP "exists, which, when multiplied by the time of exposure to that PO2, yields the UPTD for that exposure.

$$KP = \sqrt[-1.2]{\frac{0.5}{P - 0.5}}$$

The table displayed on the next page lists the KP factors from 0.5 ATA to 5 ATA. To make the explanations of Dr. Wright more simple each KP factor correspond to 1 UPTD on the previous table. Thus KP 0.6 ATA equal 0.26.

The procedure for finding the UPTD for a given exposure is as follows:

1. Convert the partial pressure of oxygen breathed at each depth to PO_2 in atmospheres.
2. Select the corresponding KP value from the table
3. Multiply the time of exposure at that PO_2 by the corresponding KP to get the UPTD for that depth.
4. Add the UPTDs for each PO2 in the complete exposure to get the total exposure.

Unit Pulmonary Toxic Dose - Table of KPs

P O_2	***0.5***	***0.6***	***0.7***	***0.8***	***0.9***	***1***	***1.1***	***1.2***	***1.3***	***1.4***
KP	*0*	*0.26*	*0.47*	*0.65*	*0.83*	*1*	*1.16*	*1.32*	*1.48*	*1.63*
P O_2	***1.5***	***1.6***	***1.7***	***1.8***	***1.9***	***2***	***2.1***	***2.2***	***2.3***	***2.4***
KP	*1.78*	*1.93*	*2.07*	*2.22*	*2.36*	*2.5*	*2.64*	*2.77*	*2.91*	*3.04*
P O_2	***2.5***	***2.6***	***2.7***	***2.8***	***2.9***	***3***	***3.1***	***3.2***	***3.3***	***3.4***
KP	*3.17*	*3.31*	*3.44*	*3.57*	*3.7*	*3.82*	*3.95*	*4.08*	*4.2*	*4.33*

An example of implementation of the procedure is provided with the medical table USN 6:

Time in minutes	***%***	***Depth (feet)***	***PO2 (ATA)***	***KP***	***UPTD***
60	*100*	*60*	*2.8*	*3.57*	*214.2*
15	*21*	*60*	*0.6*	*0.26*	*3.9*
120	*100*	*30*	*1.9*	*2.36*	*283.2*
30	*21*	*30*	*0.4*	*0*	*0*
30	*100*	*45*	*2.4*	*3.04*	*91.2*
30	*100*	*15*	*1.5*	*1.78*	*53.4*
				Total UPTD	*645.9 (= 646)*

Note that the procedure includes the air breaks already mentioned for acute oxygen poisoning.

Limitation of exposure - The Repex method

The “Repex method” is an evolution of the UPTD concept that allows doses to be calculated or tabulated the same way using the same equation but calls the single dose unit, “OTU or Oxygen Tolerance Unit”. This system was developed as a means of dealing with daily hyperoxic exposures over a mission duration of several days or longer. It was described by Rw Hamilton, D. Kenyon, Re Peterson in a document called *“REPEX habitat diving procedures: Repetitive vertical excursions, oxygen limits, and surfacing techniques”*, published in 1988, and adopted later by NOAA. Note that the value of the OTUs are similar to the KPs mentioned by Doctors Wright , Clark, Lambertsen, Bardin, and J. Field. The main difference with the initial UPTD concept is that this method provides daily limits for the diving operations undertaken.
To justify the adoption of this system, in their diving manual, NOAA says that tracking OTUs is not of great importance when the dives are of a no-stop nature. It is when the diver will be conducting many dives over more than three days, and where the exposures get lengthy, that OTU tracking will be of significant value.
The 1st phase of the procedure is as for finding UPTDs using the simplified arithmetic method described above:

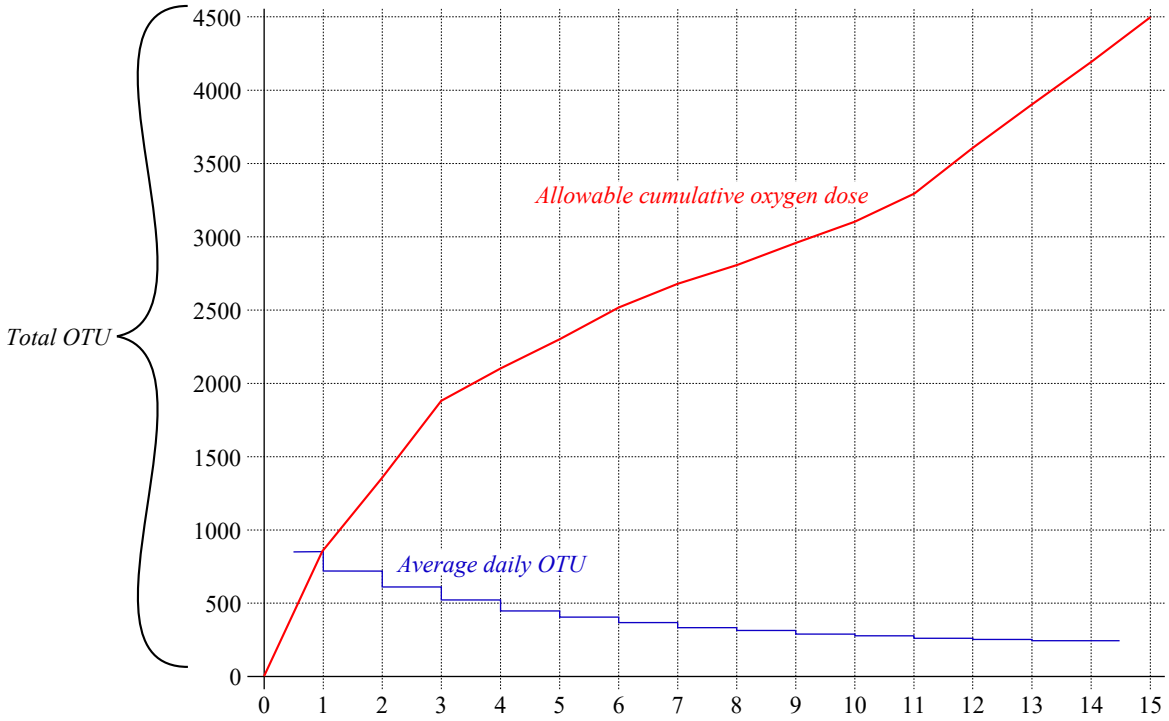
1. Convert the partial pressure of oxygen breathed at each depth to PO_2 in atmospheres.
2. Select the corresponding “OTU” value from the table below (They are of the same values as the ”Kps” above).

PPO_2 (ATA)	***OTU***	***PPO_2 (ATA)***	***OTU***	***PPO_2 (ATA)***	***OTU***	***PPO_2 (ATA)***	***OTU***
0.5	0	1.1	1.16	1.7	2.07	2.3	2.91
0.6	0.26	1.2	1.32	1.8	2.22	2.4	3.04
0.7	0.47	1.3	1.48	1.9	2.36	2.5	3.17
0.8	0.65	1.4	1.63	2.0	2.64	2.6	3.31
0.9	0.83	1.5	1.78	2.1	2.64	2.7	3.44
1	1	1.6	1.93	2.2	2.77	2.8	3.57

3. Using the table below, check whether the diver is within the tolerable limit. Note that the allowable daily dose is gradually decreasing from 850 to 300 and is then maintained at this value.

	Days of exposure														
	1	***2***	***3***	***4***	***5***	***6***	***7***	***8***	***9***	***10***	***11***	***12***	***13***	***14***	***15 - 30***
Average daily dose	*850*	*700*	*620*	*525*	*460*	*420*	*380*	*330*	*330*	*310*	*300*	*300*	*300*	*300*	*300*
Total dose	*850*	*1400*	*1860*	*2100*	*2300*	*2520*	*2660*	*2800*	*2970*	*3100*	*3300*	*3600*	*3900*	*4200*	*As required*

The scheme below represents the total allowable cumulative oxygen dose in OTUs in red and the average daily dose in bleu according to the table above. The corresponding days are on the horizontal line at the bottom. Note that the cumulative oxygen dose curve increases exponentially as the daily dose line becomes flatter.



Limitation of exposure - Advantage of the UPTD / Repex system and elements to consider for its implementation

The UPTD concept is easy to implement, which is its main advantage. Although it is based on a theoretical assumption, it has provided a simple means to estimate the planned effects of prolonged hyperbaric oxygen exposures for years. That explains why divers and diving medical specialists still commonly use it instead of more modern concepts.

Based on what is said in the previous points, a UPTD of 615 is a reasonable maximum limit of oxygen exposure for the treatment of uncomplicated decompression sickness or other treatment procedures. Also, in case a longer exposure is necessary, it is suggested that a 10% decrement in vital capacity or a UPTD of 1425 has been shown experimentally to be reversible within a few days following cessation of exposure to elevated oxygen pressures. In his study, doctor Wright recommended a 10% decrement as the ultimate limit.
For information, the USN treatment tables 5, 6 and 6A have the following UPTD values.

Treatment table	***Total UPTD***	***Treatment table***	***Total UPTD***
USN 5	*333*	*USN 6 extended at 60 and 30 min*	*860*
USN 6	*645*	*USN 6A*	*690*
USN 6 extended at 30 min	*718*	*USN 6A extended at 30 min*	*763*
USN 6 extended at 60 min	*787*	*USN 6A extended at 60 min*	*833*

Some specialists suggest that a dose of around 2190 UPTD that would cause a 20% decrement would be acceptable on the first day for treatments requiring lots of oxygen, as the lung damage would be tolerable and recoverable. However, we cannot confirm this point because no official guidance as straightforward as the one of doctor Wright has, according to what we know, been published, even though data from several studies may suggest it could be possible. Thus,

exposures to such decrement levels are medical decision and should not be the fact of the diving team, whose responsibility regarding the treatment to apply to a victim ceases when the Diving Medical Specialist intervenes, which must be at the very beginning of the treatment. Also, in their study *"Oxygen toxicity and special operations forces diving: Hidden and dangerous"*, doctors Thijs T. Wingelaar, Pieter-Jan A. M. van Ooij, and Rob A. van Hulst confirm exposure to 1425 UPTD as the absolute maximum limit, and this only in exceptional circumstances with sufficient medical support available. They also consider that oxygen exposure should be limited to 450 UPTD per day and 2250 UPTD per week.

Concerning the "Repex method", as for the procedure regarding the prevention of acute oxygen poisoning recommended in the latest *"Diving Standards & Safety manual NOAA"*, it must be noted that this method is not necessary when the recommended UK-HSE operational limits (from doctors Shield & Lee), explained throughout the handbook are implemented, as the accumulated OTUs are far from the daily limits when this recommendation is implemented. Thus, those applying it should regard the Repex method as a backup procedure in case of an undesirable event.

Limitation of exposure - Inconvenient of the UPTD / Repex system

The limitations of the UPTD concept have been known for a long time, and the following reserves have been published in various diving medical books:

- The system attempts to describe biological function with mathematics, which can never be more than a close approximation.
- The system cannot allow for variations in sensitivity between people: The method calculates a theoretical dose that will produce a given amount of damage in an "average" person.
- It is not possible to allow for oxygen exposure prior to treatment: The effect of this pre-treated exposure can only be estimated.
- The method does not take into account the recovery between oxygen exposures.

In addition, in the article mentioned above, published in May 2017, and referring to other scientists, doctors Thijs T. Wingelaar, Pieter-Jan A. M. van Ooij, and Rob A. van Hulst emit the following reserves regarding the UPTD system:
The main flaw in the UPTD concept and the derived equations is the change in Vital Capacity (VC) as the sole indicator to determine oxygen stress.
Vital Capacity (VC) has a circadian rhythm, and there is a strong intra and interpersonal variability when measuring lung volumes. Ventilation during anaesthesiology with a high PO_2 is known to influence VC, possibly due to absorption atelectasis (Note: atelectasis is the complete or partial collapse of the lung).
Whether this also occurs in divers, or how long this endures after diving, is unknown. Recent findings have proven that immersion itself alters VC regardless of oxygen stress.
Since the UPTD model was derived from dry dives, the above-mentioned factors are not taken into account. Although the original authors recognized the limitations of the UPTD model, more advanced diagnostic measurements were either too difficult to perform or were unavailable in the 1960's/1970's.

Also, in another article called *"UPTD calculations should not be used"*, published in May 2017 on Shearwater's website, doctor Barbara Shykoff said the following critics, which should be considered:

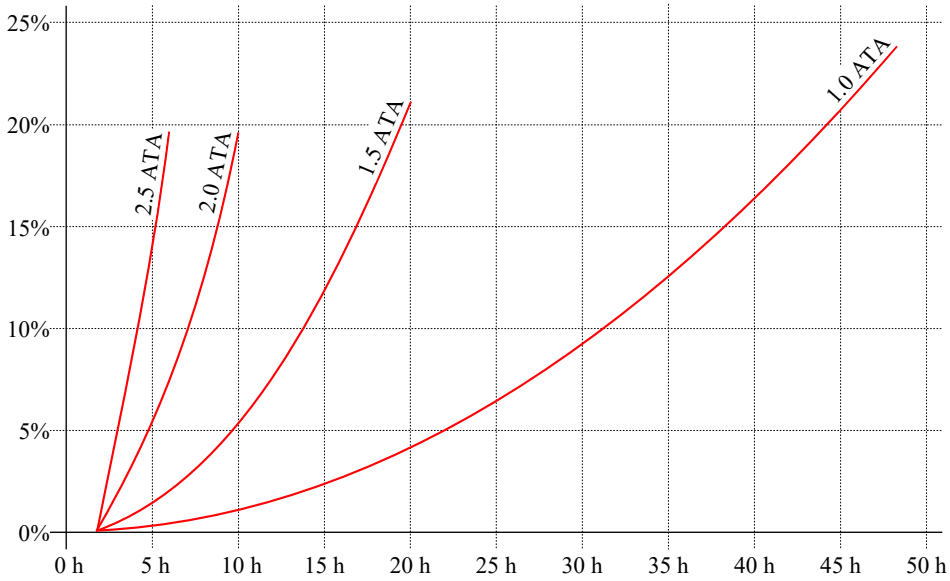
1. *The group at the University of Pennsylvania who proposed the UPTD model continued to collect hyperbaric chamber data about the development of pulmonary oxygen toxicity. The U.S. Navy Experimental Diving Unit (NEDU) found that the results for PO_2s from 1.5 to 2.5 bar deviated importantly from the UPTD predictions. In fact, the model fit well only to the PO_2 = 2 bar study used in the development of the UPTD model (but not to a later PO_2 = 2 bar study) and to PO_2 =1.5 bar data.*
2. *Researchers from Duke University showed that the underlying injury to the lungs differs depending on the exposure PO_2 even though the signs and symptom are similar for a large range of PO_2. Thus, a single model cannot be expected to fit for all PO_2s.*
3. *There is no "unit dose" that can be multiplied by time to find the effect across all PO_2 VC changes approximately as a function of time for PO_2 <1.5 bar,4 but approximately as a function of time squared for higher PO_2s.*
4. *The U.S. Navy Treatment Table 6 (TT6) provides 633 UPTDs. This is predicted to cause a 2% (median) decrease in VC, a negligible change. However, among 18 healthy subjects who underwent a TT6 exposure,7 three divers had decreases in Vital Capacity (VC) ranging from 8% to 12%, decreases that lasted from 1 to 3 days. Changes in other pulmonary function variables were also notable in those three and in one other diver. One of the divers reported mild symptoms. Clearly, UPDTs do not provide the full picture.*
5. *Because Vital Capacity (VC) changes cannot be detected until there is swelling of lung tissues and/or liquid in some of the air spaces (interstitial and/or pulmonary oedema), other markers of pulmonary oxygen toxicity that occur earlier with exposures to low PO_2s, for example, symptoms, are preferable. UPTDs have not been correlated to symptoms or to other changes in pulmonary function.*
6. *We need a model that includes recovery if we are to assess "pulmonary O_2 dose" over multiple dives and/or recompression treatments.*

Science progresses through the proposal of models and hypotheses that are tested initially with one set of data, retested with new data, and either disproven or not disproven. The UPTD model that showed promise in 1970 has been displaced by later evidence. It is time for the diving community to pay attention to the last 40 years of evidence. UPTDs are not a useful measure.

Limitation of exposure - Arieli K concept

In October 2022, Dr. Pieter-Jan van Ooij, vice chairman of the DMAC, who is also one of the authors of documents taken into a reference in this chapter, proposed to the members of this organization a document titled *"Exposure index for pulmonary oxygen toxicity in diving"*, he wrote with doctor Risberg, which has been accepted as a DMAC guideline.
This document is based on the works of Ran Arieli and his team, who, in a document titled *"Calculated risk of pulmonary and central nervous system oxygen toxicity: a toxicity index derived from the power equation",* provides the following clarifications to explain his new concept:
The concept of the unit pulmonary toxic dose (UPTD), which is based on a modification of the rectangular hyperbola, was proposed in response to a request for oxygen exposure limits based on a very small amount of research data: A point at four atmospheres absolute (atm abs) (405.2 kPa) and the absence of known injury at an inspired partial pressure of oxygen PO2 of 0.5 atm abs (50.6 kPa). It was merely descriptive, without any basis in physico-chemical or physiological mechanisms.
In light of all this, it was clear that a different model was required to fit outcome data. The power law approach was adopted for this study. The power equation derived from the chemical reactions related to PO2, which produce Reactive Oxygen Species (ROS) and Reactive Nitrogen Species (RNS), was shown to have a good predictive capability.
The main difference between the power equation and the rectangular hyperbola is the high power of PO2 in the former: 6.8 for Central Nervous System Oxygen Toxicity (CNS-OT); and 4.57 for Pulmonary Oxygen Toxicity (P-OT). At a high PO2, the rectangular hyperbola will lose its predictive power.

Doctor Arieli bases the Toxicity Index on the following power equation: Toxicity Index = K = t^2 x PO_2^c , where *" t "* represents time in hours or minutes, *" PO_2 "* is expressed in atmosphere absolute, and *" c "* represents the power term for Pulmonary Oxygen Toxicity: so 4.57. Thus, the toxicity index formula can be K = t^2 x $PO_2^{4.57}$, and the expected relative reduction in VC is set to 8.2 x 10^{-4} of the K value.
In the document mentioned above, doctor Arieli says that the US Navy recommended oxygen exposure limits that will result in a 2% change in Vital Capacity (VC), maximum exposure being expected to produce a 10% decrement. Thus, inserting ΔVC = 2% or ΔVC = 10% in the power equation will set the PO_2 and time limits (Note: delta (Δ) is used to represent "difference" or "change" in mathematics). For these two values of ΔVC, the Pulmonary Oxygen Toxicity index t^2 x $PO_2^{4.57}$ should not exceed 244 and 1,220, respectively, both at constant pressure and for a complex exposure.
Doctor Arieli also used complex equations to calculate the recovery from oxygen toxicity.
He concludes his study by suggesting this index is as a replacement for the UPTD concept. However, he also suggests that for the most common exposures, the Pulmonary Oxygen Toxicity (P-OT) index limit can be set at 250.
Arieli's formula allows drawing the following curves of VC reduction.



Doctors Risberg and van Ooij say that Arieli K concept should replace UPTD for tracking pulmonary oxygen toxicity in surface-oriented diving for the following reasons:

- Arieli K is a more accurate estimate of pulmonary oxygen toxicity for such exposures.
- Arieli K is expected to better predict pulmonary oxygen toxicity development after multiple exposures to various PO_2.
- The estimation of pulmonary oxygen toxicity recovery is much better validated in the equation for Arieli K than the Repex model.

Doctors Risberg and van Ooij say that there are two main drawbacks of replacing UPTD will Arieli K concept:

- Firstly the calculus of Arieli K concept is more complex for multiple PO_2 exposure levels.
- Secondly, UPTD is accepted as a de-facto standard for hyperoxic exposure.

They also suggest a "pulmonary oxygen toxicity limit" of K=120 for individual dives and two consecutive days of diving.
For multi-day diving with two days off per week, they suggest that the daily exposure level should not exceed K=70, while the P-OT should not exceed K=50 if only one day is allowed off diving after five consecutive days of diving.

Equivalent Surface Oxygen Time (ESOT)

The Equivalent Oxygen Time (ESOT) is a concept based on Arieli's K concept, reviewed by doctors Risberg and van Ooij and published by the Diving Medical Advisory Committee in the document DMAC 35.
Due to the complexity of Arieli's equations it has been considered That hyperoxic exposure monitoring can be facilitated using an alternative index termed Equivalent Surface Oxygen Time (ESOT):

$$ESOT = t \times PO_2^{2.285}$$ *Where, t = time in minutes, and PO_2 is expressed in atmospheres*

ESOT=1 is the hyperoxic exposure reached after 1 min of breathing 100% O_2 (Fi O_2 =100%) at surface pressure. ESOT is in this respect comparable to UPTD. In a multi-PO_2 segmented dive, the ESOT from each segment can simply be summed up to reach the total hyperoxic exposure for the dive.
Based on the studies made by doctors Risberg and van Ooij, the DMAC suggests the following:

- A maximum exposure level of ESOT of 660 for a maximum of two consecutive days of diving. Also, two days off diving should be allowed after this exposure.
- A maximum of five consecutive days of diving, followed by two days off if the daily (24 h) exposure level is ESOT=500 or less. The daily exposure may reach ESOT=420 if two days off diving are allowed after ten consecutive days of diving. Intermittent breathing of compressed air ("air break") has been shown to delay the development of pulmonary oxygen toxicity and recovery of POT will take place faster in resting divers.

Thus, the DMAC considers it acceptable to relax these limits in resting divers breathing hyperoxic gas with intermittent air breaks. There is a lack of studies on pulmonary oxygen toxicity after successive multiday surface-oriented dives, particularly dives with short exposures to PO_2 <1.3 Atm. For this reason, the DMAC says that it may be acceptable to allow more than ten successive days of diving for such exposures, but the advice of a diving physician should be sought in each case.
In addition, as long as UK-HSE bottom time restrictions are adhered to, there is no need to calculate ESOT for air dives with in-water decompression, as such dives are not expected to cause any relevant pulmonary oxygen toxicity. The DMAC also says that air in-water decompression dives can even be done on the two days prescribed "off diving".
The table below shows the hyperoxic exposure limits for commercial surface-oriented diving adhering to the UK-HSE bottom time limitations.

- The maximum number of successive diving days and minimum surface interval for repetitive exposures depend on ESOT after the preceding dive.
- Shorter surface intervals are allowed but require residual ESOT calculation (see the next tables). †: K exceeds recommended threshold values even for single dives. "Daily" should be interpreted as a 24 h period.

Daily maximum ESOT	*Maximum number of successive days of diving*	*Minimum surface interval (h)*
> 660	†	24
501 - 660	2	12
420 - 500	5	12
< 420	10	12

Subject to UK=HSE bottom time limitations, DMAC says that the practical consequences of this guidance would mainly be limitations on the number of successive nitrox oxygen surface decompression and nitrox transfer under pressure dives.

- If more than five consecutive days of nitrox diving with in-water decompression are planned, some limitations will apply if PO_2 exceeds 1.3 Atm and bottom time exceeds 180 min. Inert gas load (repetitive group designator) rather than hyperoxic exposure will tend to restrict bottom time for a repetitive dive following a dive with high hyperoxic exposure.
- Open-circuit nitrox diving adhering to the UK-HSE bottom time limitations will only be a concern with a Fi O_2 > 40% , irrespective of decompression mode. These guidelines will not restrict the number of consecutive days of air dives with staged in-water decompression.
- The guideline should be considered by occupational divers using rebreathers (e.g., scientific divers), but dives planned by technical, recreational divers, and military divers using rebreathers with high Fi O_2 or fixed PO_2 are beyond the scope of their guidance.

The table #2 on the next page can be used for dives with a constant or variable PO_2. It does not take into account the risk for CNS Oxygen toxicity that should be considered independently of pulmonary oxygen toxicity.
For example, consider a dive with oxygen surface decompression with compressed air to 15 msw for 180 min, where after surfacing, the diver is recompressed to 15 msw for 15 min followed by 30 min O_2 breathing at 12 msw (US Navy protocol):

- The hyperoxic exposure in the bottom phase with a PO_2 of 0.5 Atm is ESOT=37.
- The 15 msw exposure will give ESOT=122. Note that air breaks can be ignored.
- The 12 msw exposure adds additional ESOT=182.
- The total exposure burden will be 37+122+182=341.

PO2 (Atm)	K	Time (h:min)							
		15	30	60	90	120	150	180	240
0.5	0.21	3	6	12	18	25	31	37	49
0.6	0.31	5	9	19	28	37	47	56	75
0.7	0.44	7	13	27	40	53	66	80	106
0.8	0.6	9	18	36	54	72	90	108	144
0.9	0.79	12	24	47	71	94	118	141	189
1	1	15	30	60	90	120	150	180	240
1.1	1.24	19	37	75	112	149	186	224	298
1.2	1.52	23	46	91	137	182	228	273	364
1.3	1.82	27	55	109	164	219	273	328	437
1.4	2.16	32	65	129	194	259	324	388	518
1.5	2.53	38	76	152	227	303	379	455	606
1.6	2.93	44	88	176	263	351	439	527	702
1.9	4.33	65	130	260	390	520	650	780	1040
2.2	6.06	91	182	364	545	727	909	1091	1454
2.5	8.12	122	243	487	730	974	1217	1461	1948

Alternatively, the ESOT may be calculated using the k-column in Table 3. During the bottom phase (pO 2 =0.5 Atm) k=0.21. Multiply with exposure time to reach ESOT bottom phase = 0.21 x 180 = 38. ESOT 15 msw = 8.12 x 15 = 122 and ESOT 12 msw = 6.06 x 30 = 182. The grand ESOT=38 + 122 + 182 = 342.

Table #2 allows calculating Arieli K for a dive with multiple segments with different PO2 levels
Each cell holds the required exposure time to reach a certain K (first row) for a given PO2 (first column).
Table #3 is the continuation of Table #2 for calculating K for calculating K in a dive with segments of different PO2 for exposure levels exceeding those listed in Table #2

P O2 (Atm)	K Value						
	140	160	180	200	220	240	260
1	11:43	12:38	13:24	14:08	14:49	15:29	16:07
1.1	09:31	10:10	10:47	11:22	11:55	12:27	12:58
1.2	07:48	08:20	08:50	09:19	09:46	10:12	10:37
1.3	06:29	06:56	07:22	07:45	08:08	08:30	08:51
1.4	05:29	05:51	06:13	06:33	06:52	07:10	07:28
1.5	04:41	05:00	05:18	05:35	05:52	06:08	06:23
1.6	04:02	04:19	04:35	04:49	05:04	05:17	05:30
1.7	03:31	03:45	03:59	04:12	04:24	04:36	04:47
1.8	03:05	03:18	03:30	03:41	03:52	04:02	04:12
1.9	02:43	02:55	03:05	03:15	03:25	03:34	03:43
2	02:25	02:35	02:45	02:54	03:02	03:10	03:18
2.1	02:10	02:19	02:27	02:35	02:43	02:50	02:57
2.2	01:57	02:05	02:12	02:20	02:26	02:33	02:39
2.3	01:45	01:53	02:00	02:06	02:12	02:18	02:24
2.4	01:36	01:42	01:48	01:54	02:00	02:05	02:10
2.5	01:27	01:33	01:39	01:44	01:49	01:54	01:59

Tables #2 & #3 are intended for use in a dive with multiple segments of varying PO2.
The principle is based on transforming K from a previous segment into the exposure time needed to reach the same K for a succeeding segment.
Example: For a dive with 3 hours bottom time, breathing a mix 1.3 atm, and oxygen decompression of 17 min at 1.9 atmosphere (30 fsw/9 msw) and 96 min at 1.6 atmospheres (20 fsw/6 msw):

1. Using Table #2 , follow the row for 1.3 atmospheres until you reach a number equal of exceeding 3:00 (with this example: 03:00). 03:00 on line 1.3 corresponds to the K-Value = 30
2. The next segment is the 1.9 atmospheres exposure: Use the K-value = 30 column and read the time in the PO2 = 1.9 row. You find 01:15.
3. This value should be added to the actual bottom time (17 min) at 1.9 atmosphere (30 fsw/9 msw), giving a total a total equivalent exposure time of 1:32. The closest cell in the 1.9 atmospheres row contains 1:37 , and is placed in the K=50 column. The segment at 1.9 atm (30 fsw/9 msw) has added K=20 to the Pulmonary Oxygen Toxicity (POT) exposure burden.
4. Follow the column to PO2 = 1.6 atmospheres (20 fsw/6 msw), and read 2:24 in the crossing cell. At this stop, the diver should stay 96 min (1:36). This exposure time (2:24 + 1:36) equals 4:00, which is in Table #3.

P O2 (Atm)	*K Value*											
	10	*20*	*30*	*40*	*50*	*60*	*70*	*80*	*90*	*100*	*110*	*120*
1	03:09	04:28	05:28	06:19	07:04	07:44	08:22	08:56	09:29	10:00	10:29	10:57
1.1	02:32	03:35	04:24	05:05	05:41	06:13	06:43	07:11	07:37	08:02	08:26	08:48
1.2	02:05	02:56	03:36	04:10	04:39	05:06	05:30	05:53	06:15	06:35	06:54	07:13
1.3	~~01:44~~	~~02:27~~	03:00	03:28	03:52	04:15	04:35	04:54	05:12	05:29	05:45	06:00
1.4	01:27	02:04	02:32	02:55	03:16	03:35	03:52	04:08	04:23	04:38	04:51	05:04
1.5	01:15	01:46	02:10	02:30	02:47	03:04	03:18	03:32	03:45	03:57	04:09	04:20
1.6	01:04	01:31	01:52	02:09	02:24	~~02:38~~	~~02:51~~	~~03:03~~	~~03:14~~	~~03:25~~	~~03:35~~	~~3:~~
1.7	00:56	01:19	01:37	01:52	02:06	02:18	02:29	02:39	02:49	02:58	03:07	03:15
1.8	00:49	01:10	01:25	01:39	01:50	02:01	02:11	02:20	02:28	02:36	02:4	02:51
1.9	00:43	01:01	01:15	~~01:27~~	01:37	01:47	01:55	02:03	02:11	02:18	02:25	02:31

Go to 04:00 in table #3

5. Follow the 1.6 Atm row until the equal or higher cell to 4:00 is reached. In this example, this cell is 04:02 in table #3, and the corresponding column is K = 140, which will be the total exposure for this dive.

P O2 (Atm)	*K Value*						
	140	*160*	*180*	*200*	*220*	*240*	*260*
1	11:43	12:38	13:24	14:08	14:49	15:29	16:07
1.1	09:31	10:10	10:47	11:22	11:55	12:27	12:58
1.2	07:48	08:20	08:50	09:19	09:46	10:12	10:37
1.3	06:29	06:56	07:22	07:45	08:08	08:30	08:51
1.4	05:29	05:51	06:13	06:33	06:52	07:10	07:28
1.5	04:41	05:00	05:18	05:35	05:52	06:08	06:23
1.6	04:02	04:19	04:35	04:49	05:04	05:17	05:30
1.7	03:31	03:45	03:59	04:12	04:24	04:36	04:47
1.8	03:05	03:18	03:30	03:41	03:52	04:02	04:12
1.9	02:43	02:55	03:05	03:15	03:25	03:34	03:43

Notes:

- The segment at 1.6 atmospheres has added K=90 to the Pulmonary Oxygen Toxicity (POT) exposure burden, which is somewhat higher than the true result and is due to rounding. This inaccuracy is due to a conservative approach in the second step.
- Air breaks will reduce the K-value, but the authors have not tabulated them as the absolute magnitude of a 5-10 min air break is relatively small.

Relevant formulas applicable for accurate calculation of ESOT and expected VC reduction provided by DMAC 35

- ESOT is a simple transformation of "Arieli K". Transformation of Arieli K to ESOT: $ESOT = 60 \times \sqrt{K}$
- ESOT can be calculated according to the following equation as a function of exposure time (t) in minutes and PO2 in atmospheres: $ESOT = t \times PO_2^{2.285}$ *Where, t = time in minutes, and PO2 is expressed in atmospheres*
- Multiple successive exposures/pO2 segments can be calculated as a summary of all individual segments according to the equation above. In other words, accumulated ESOT *(ESOTacc)* for successive dive segments

can be calculated as the sum of ESOT for each dive segment: $ESOT_{acc} = ESOT_1 + ESOT_2 + \dots ESOT_n$

- Recovery of ESOT can be calculated according to residual ESOT (ESOT rec) after a normoxic recovery period of t rec (h) depending on pO 2 (Atm) of the proceeding exposure. Use pO 2 =1.1 Atm for exposures subceeding this level: $ESOT_{rec} = ESOT_i \times e^{(0.21 - 0.192 \times PO_2) \times t_{rec}}$
- Decrement in Vital Capacity can be estimated based on the following equation: $\Delta VC = 0.0082 \times (ESOT/60)^2$

Limitation of exposure - To conclude on UPTD and the Arieli K concept as proposed by doctors Risberg and van Ooij

The limitations provided by the Arieli K concept and tabulated by doctors Risberg and van Ooij propose a better estimation of Pulmonary Oxygen Toxicity (POT) and, thus, should be considered a safer approach.
Using the tabulations provided in this new DMAC guidance makes it as simple to use as the UPTD concept; thus, there is no reason not to implement this new procedure.
Also, based on what scientists such as doctors Arieli, Shykoff, Thijs T. Wingelaar, Pieter-Jan A. M. van Ooij, Rob A. van Hulst, and Risberg mentioned about what they consider the obsolescence of the UPTD/Repex system, we can assume that Arieli K concept should be implemented in replacement.
However, we have explained and kept the Unit Pulmonary Toxic Dose (UPTD) concept for the following reasons:

- Even though it is based on a different approach and more complex equations Arieli K concept should be considered an evolution of the old UPTD concept. Thus, understanding the UPTD concept allows a better understanding of the Arieli K Concept. We also consider that publishing the two systems allows comparing them.
- Linked to above, explaining the UPTD concept allows us to remember the scientists who created it according to the knowledge available to them when they emitted this idea. As suggested previously regarding the work of Doctor Wright, scientists rarely develop ideas from scratch. Thus, new concepts are often created because predecessors originated a previous one, etc. Arieli K Concept will probably be developed further in the future and perhaps replaced by a new idea generated from the data it provides today.
- To our knowledge, the DMAC is the 1st organization to publish a public release document with tabulations for calculating Pulmonary Oxygen Toxicity (POT) based on Arieli K Concept. We do not know whether other organizations will adopt this guideline or whether these will continue to recommend the Unit Pulmonary Toxic Dose (UPTD) concept to their affiliates.

Normoxic mix intake between periods of oxygen breathing is another way to diminish the effects of chronic oxygen toxicity.

As already mentioned for acute oxygen poisoning, it has been discovered that periods of normoxic mix intake in between periods of oxygen breathing allow diminishing the effects of oxygen toxicity, and thus, is an efficient technical approach for increasing the total time of exposure to hyperoxia. As an example, in an article called *"Extention of oxygen tolerance in man (predictive studies VI)"*, doctors Lambertsen and Clark tested several variations of intermittent exposures at 2 ATA. Regarding pulmonary toxicity, they concluded their study by saying, "*Our results show that pulmonary oxygen tolerance can be extended significantly at 2.0 ATA by the systematic alternation of oxygen and normoxic exposure periods. They also show clearly in man or animal that early toxic effects on the lung can be stabilized or reversed at least partially during continued intermittent exposure with an appropriate combination of oxygen exposure and normoxic recovery periods"*
Periods of oxygen and normoxic breaks vary according to the tables.

Based on this principle, the organization of breaks when intensive diving operations are in process is recommended. For example, it is a recognized good practice to stop the diver one day a week. Also periods without diving activities should be organized following long periods of nitrox diving.

Diseases resulting from long exposure to hyperoxia

Acute oxygen poisoning and chronic oxygen poisoning are effects from hyperbaric oxygen described and studied since the end of the 19th century. As explained in the previous points, they show visible symptoms. More recent studies have highlighted that repetitive and long exposures to hyperbaric oxygen may lead to diseases not immediately detectable.

Oxidation reactions are crucial for life, but on the other hand, they can be involved with mechanisms of cells destruction: "Oxidation" is a chemical reaction that transfers electrons or hydrogen from a substance to an oxidizing agent. As already discussed to explain acute oxygen poisoning, oxidation reactions can produce free radicals. In turn, these radicals can start chain reactions. When the chain reaction occurs in a cell, it can cause damage or death to the cell.
To control this phenomenon, the body maintains complex systems of multiple types of antioxidants. Antioxidants are molecules that inhibit the oxidation of other molecules. These systems are influenced by diet and genetic factors. It is said that the ability to produce antioxidants decreases with the age, nevertheless the specialists do not currently know the capacity for antioxidant defence.
Many studies have linked the decreased production or the inhibition of antioxidants to diseases such as cancer, insulin resistance, diabetes mellitus, cardiovascular diseases, atherosclerosis and others.
It has been proved that at sufficient pressure and exposure duration, oxygen can inhibit the antioxidant defense, and cause functional impairment. The severity of effects that occur in different tissues are dependent upon interactions between the oxygen dose and relative susceptibilities of the exposed tissues.

In an article named *"Saturation diving; physiology and Pathophysiology"*, doctors Alf O. Brubakk, John A.S. Ross, and Stephen R. Thom say that the regulation of these highly reactive molecules and the defense mechanisms must be kept under tight control.
Surface-supplied divers are less concerned than saturation divers with this phenomenon as they are not permanently submitted to oxygen pressures higher than normal. However, it must be recognized that daily diving operations will expose them to accelerated oxidation periodically.
Of course, the procedures discussed previously to control oxygen poisoning will contribute to reducing these effects. However, another strategy is to control the food quality and ensure sufficient antioxidants are provided to the divers. Thus divers' dietetics should be closely monitored by the diving medical specialists of the diving company and people in charge of the team, as several investigations made on vessels prove that although food quantities are usually sufficient, the quality of the menus in terms of dietetics is often poor. At the discharge of the cooks, it must be noted that the food is usually served in self-service and bad menus are often the fact of the consumers, so it is often linked to an educational problem.
Nevertheless, people in charge of the team should ensure that the following categories of aliments are promoted and always available:

- Fresh fruits are high in antioxidants and vitamins.
- Dried fruits have an antioxidant ratio higher than fresh fruits as the water is removed, and they are, therefore, more concentrated. They have the advantage of being easy to conserve and carry.
- Vegetables are as rich in antioxidants as fruits.
- Spices and herbs are also reputed to be rich in antioxidants.
- Antioxidants also come from beverages such as fruit juices, tea, and also real coffee. However, note that adding milk to coffee or tea blocks antioxidants.

Air pollution is also known to trigger the formation of excessive free radicals. For this reason, in addition to controlling pollutants in the compressed air dedicated to divers, the dive station should be installed far from engine exhausts and other pollution emissions.

Narcosis

Description

Narcosis *(also known as nitrogen narcosis, inert gas narcosis, raptures of the deep, Martini effect)* is a reversible alteration in consciousness that occurs while diving at depth with air or mixes using nitrogen or other narcotic gasses.

As soon as he leaves the surface and descends, a diver breathing air is exposed to increasing partial pressure of nitrogen. At the same time, the effects of nitrogen narcosis begin. The effects are mild at shallow depths, but as he descends, the effects increase, altering his awareness of events and his own behaviour. The condition is completely reversed by ascending to a shallower depth with no long-term effects.
The precise mechanism of narcosis is not well understood. Still, it appears to be the direct effect of gas dissolving into nerve membranes and causing a temporary disruption in nerve transmissions. While the effect was first observed with air, other gases, including argon, krypton, and hydrogen, cause very similar effects at higher than atmospheric pressure. One particular group of nerve cells in the brain appears particularly sensitive to nitrogen and other narcotic agents. They are in the reticular formation, which is one of the brain's exchange area situated in the brain stem. The reticular formation receives messages from one part of the brain and transmits them to other parts. If the reticular formation is disrupted, all brain functions linked to it become disrupted, and the subject becomes narcotised or loses consciousness.

The narcotic effect of gases has been studied since the beginning of the diving industry, and what is known is derived from the study of anesthetic gases.
At the beginning of the 20th century, two German scientists, Meyer (1853-1939) and Overton (1865-1933), independently discovered the correlation between lipid solubility and anesthetic potency. The presumed mechanism is related to the hypothesis that agents could act at the cell membrane's lipid layer, particularly those of neurons. Further experimentations highlighted the role of proteins in this process. These observations are known as the Meyer-Overton hypothesis, which says that the anesthetic potency of a gas is inversely related to its lipid solubility. In other words, lipid-soluble gases produce narcotic effects at lower concentrations than less soluble gases. Note that according to this hypothesis, oxygen is considered a narcotic gas.

Gas	***Solubility coef. in olive oil***
Helium (He)	*0.015*
Hydrogen (H2)	*0.042*
Nitrogen (N2)	*0.052*
Oxygen (O2)	*0.11*
Argon (Ar)	*0.15*
Krypton (Kr)	*0.44*
Carbon Dioxide (CO2)	*1.34*

The most dangerous aspects of narcosis are the impairment of judgment, multi-tasking and coordination, and the loss of decision-making ability and focus, which increases the risk of an accident and, at the same time, decreases the ability to cope with an emergency.

Symptoms

The analogy between alcohol and narcosis is commonly used to describe the effects of narcosis:

- If taken in substantial amounts, alcohol can be fatal because it poisons the brain cells, and the person who takes it passes into a coma, stops breathing, and dies. However, most alcohol deaths occur when people have much less alcohol than this in their blood but make irrational decisions and cannot react in an unplanned event.
  Like alcohol, narcosis may be fatal at a high partial pressure of a narcotic gas, but this is very rare. Much more frequently, narcosis causes divers' death in indirect ways due to lack of reactivity, hallucinations, and bizarre behavior that may occur.
- Similarly to alcohol, there is individual variation in susceptibility to narcosis. In addition, the same individual may be susceptible to the effects of narcosis on some occasions more than on others.
- It is recognized that alcohol affects the brain even at small doses, and there is no doubt that, if measurements are made, everyone shows some evidence of narcosis at depth.

However, unlike alcohol, which takes time to be absorbed from the stomach into the blood, the partial pressure of a narcotic gas in the blood changes quickly with the depth. Thus, the effects of narcosis occur with short delay as the partial pressure of the narcotic gas changes. As indicated in the previous point, narcosis disappears as the diver ascent, and there are no residual effects such as hangover.

The effects of nitrogen narcosis are also compared to the effects of some anaesthetic agents used during surgery. These agents and nitrogen all have one thing in common: They are more soluble in fats than they are in water.

Depth in m	*Depth in ft*	*Visible Symptoms*
10 to 30	*33 to 100*	*- Mild impairment of performances* *- Mildly impaired reasoning* *- Mild euphoria possible*
30 to 50	*100 to 165*	*- Delayed response to visual and auditory stimuli* *- Reasoning and immediate memory affected more than motor coordination* *- Calculation errors and wrong choices* *- Idea fixation* *- Over-confidence and sense of well-being* *- Laughter and loquacity (in chambers) which may be overcome by self-control* *- Anxiety (common in cold murky water)*
50 m / 165 ft is the limit commonly admitted for air diving		
50 to 70	*165 - 230*	*- Sleepiness, impaired judgment, confusion* *- Hallucinations* *- Severe delay in response to signals, instructions and other stimuli* *- Occasional dizziness* *- Uncontrolled laughter, hysteria (in chamber)* *- Terror*
70 to 90	*230 to 300*	*- Poor concentration and mental confusion* *- Stupefaction with some decrease in dexterity and judgment* *- Loss of memory, increased excitability*
> 90	*> 300*	*- Hallucinations* *- Increased intensity of vision and hearing* *- Sense of impending blackout, euphoria,* *dizziness, levitation, manic or depressive states* *- Disorganization of the sense of time, changes in facial appearance* *- Unconsciousness, Death*

Note that other factors like sedative drugs; alcohol; fatigue; heavy exertion; apprehension; poor visibility, can increase an individual's susceptibility to nitrogen narcosis.

In addition to the above, repeated exposure to depth allows some degree of tolerance to develop. How this occurs is still unclear. It may be that repeated exposure enables some divers to function with a high level of nitrogen narcosis in the way chronic alcoholics can function with blood alcohol levels which would put most people to sleep.

Treatment and Prevention

As the effects of narcosis are linked to the depth, the only solution is to ascend to a shallower depth where there should be nearly immediate improvement.
If the diver does not respond to orders, the standby diver will have to be sent for immediate recovery.
In any case of severe narcosis the dive will have to be stopped.

There is no way of eliminating these effects, unless a gas mixture is used that has less narcotic effects than nitrogen in air. It is the purpose of using tri-mixes (helium = Oxygen + Nitrogen) or heliox (helium + Oxygen), either for surface gas or saturation diving. However, mix gasses such as heliox may be incorrect and require proper gas management procedures, as indicated in Book #3 of this handbook , and Book #2 and #3 of our saturation handbook.

It is impossible to lay down precise limits of acceptable depth. That should depend on the dive's objective and the divers' experience in the prevailing conditions. The term "limit" itself implies that at a particular depth, things are safe, while at a metre or a foot deeper, they are unsafe. Clearly, the danger increases progressively with depth, and safe cut-offs are never precise. However, as they had to set a limit for their national rules, most European governments have limited air diving to 50 m/165 ft. In the USA, the National Oceanic and Atmospheric Administration (NOAA) promotes a limit of 185 fsw (56.4 m) for surface-supplied diving, which is also the case for associations such as ADCI. As a remembering, the maximum limit of air diving recommended in this handbook is 50 m/165 ft.

Hypoxia - Anoxia

Description

- Hypoxia:

Hypoxia, is a condition in which the body is deprived of adequate oxygen supply that occurs when the PPO_2 is less than 160 Mb (0.16 bar). However, it is said that a completely inactive person can survive for a time on less than 100 Mb.

A severe hypoxia impedes the normal function of cells and eventually kill them. The brain is the most vulnerable organ in the body to the effects of hypoxia.

Lack of oxygen can have various causes:

- Improper line up of breathing gases supply resulting in a too low partial pressure of oxygen in line.
- Mechanical failure of the air/gas supply
- Foreign object blocking the normal gas supply
- Helmet supply blocked due to diver sick and vomiting.
- Paralysis of the respiratory muscles due to spinal cord injury…
- Pneumothorax or reduction of the O2 exchange at alveoli/capillary membrane tissue area due to decompression illness, oedema…
- Undetected chronic disease like: Anemic hypoxia *(oxygen content of the blood is reduced due to a decreased ability for hemoglobin to carry it)*; "ischemic hypoxia" *(due to decreased blood flow)*; histotoxic hypoxia *(due to cells unable to use the oxygen effectively)* and others…
- Also, a diver holding his breath too often during a dive can have the PPO2 in the lungs falling progressively and becoming hypoxic without feeling it sufficiently early.

- Anoxia:

What is called Anoxia is a Hypoxia in which there is complete deprivation of oxygen. The result of anoxia is nearly immediate death. The main cause of anoxia is the breathing of gasses without oxygen such as pure helium, pure nitrogen, etc., which are often used for gas mixing, calibration of analysers, or other purposes

Symptoms

- Hypoxia:

- 1st visible symptoms:
  - Increased pulse rate, blood pressure, and breathing (body trying to compensate hypoxia)
  - Cyanosis (It cannot be seen when the diver is in the water)
  - Lack of concentration
  - Lack of muscle control
  - Inability to perform tasks requiring sharp skills
  - Weakness, agitation, euphoria

- Symptoms when the victim is in a state of deep hypoxia:
  - Loss of breath control
  - Drowsiness
  - Loss of consciousness

- Important point:
  There is no reliable warning of the onset of hypoxia. It can occur unexpectedly, making it a particularly serious hazard.

- Anoxia

Anoxia happens immediately without any visible symptoms and result in nearly immediate death. The victim collapsing without having the time to understand what happens.

Treatment

There is nothing possible with anoxia. Regarding hypoxia, the following procedures should be applied:

- Shift the gas supply to back up.
- Flush the hat.
- Send the standby diver and recover the diver as soon as possible (and safely).
- Pure oxygen is to be provided through a mask.
- Resuscitation may be necessary.
- Maintain a close observation of the victim and call the diving medical specialist.

Prevention

Prevention is linked to the management of gasses and the close monitoring of the diver:

- Gas containers must be color-coded, and the exact composition of their content marked and logged.
- Analysers with high and low alarm must be used during the dives, the fabrication of mixes, and the transfer of gasses. Analysers with alarms must also be in place in storage rooms.
- Regading the use of helium, DMAC 5 *"Minimum level of O2 in helium supplied offshore"* says: *"DMAC endorses the recommendation that an oxygen and helium mixture should be used in place of pure helium supplied to offshore diving installations. It is recognised however that contractors may need to use pure helium as a calibration gas. The choice of mixture supplied should be left to the diving contractor but a minimum of perhaps two percent of oxygen should present no problems operationally from 50 to 150 metres, and from 150 metres a smaller percentage may be appropriate".*
- Pure gasses used for calibration and other purposes must be separated from breathing gasses and be stored in a ventilated area.
- Backup supply to be ready all the time
- The standby diver must be ready to intervene at all times.
- Good diving practices like avoiding breath holding should be in force.
- Close monitoring of the divers and gas supplies should be in place. Regarding this point, DMAC #2 highlights the importance of monitoring the diver through communications. In addition, pictures provided by an ROV allow the supervisor to assess a situation and react on time.
- Oxygen and appropriate breathing masks must be ready at all times.

Hypercapnia

Description

Hypercapnia, also called "Carbon dioxide toxicity" is due to accumulation of carbon dioxide (CO_2) through excess production or inadequate ventilation (breathing).
The excess production of CO_2 is usually due to metabolism from increased exertion. Whereas only 0.5 litres/minute of CO_2 is produced at rest, that can rise to over 3 litres during maximal exercise.

- The phenomenon can be caused by:

- Mass density of the gas increased at depth *(at 37°C, one litre of air at the surface = 1.133 g, and at 60 m =8 g)*
- Breath-holding
- Strong effort during heavy work at depth
- Excessive dead space and inadequate ventilation of some models of helmets
- Failure of gas supply system
- Inappropriate setting of the regulators on surface panel or in the bell
- Excessive breathing resistance of the helmet regulator
- Inappropriate flushing of the air chambers or, if installed, inefficient scrubber
- Air intakes in vicinity of source of pollution (The CO_2 is 13% of the total of the exhaust gas of a diesel engine)
- Recycling system failure (saturation)
- Failure to control the level of CO_2 in the chamber or in the bell.

- The mechanism can be explained as follows:

The pressure of carbon dioxide is about 40 mm hg in the alveolus, and about 45 mm in the venous blood reaching the lungs while the normal pressure of CO_2 in the atmosphere is 0.3 mm hg.

	Atmospheric air			*Alveolar Air*		
	P. mm Hg	***P. ATA***	***%***	***P. mm Hg***	***P. ATA***	***%***
N_2	*597*	*0.78*	*78.62 %*	*569*	*0.749*	*74.9 %*
O_2	*159*	*20.9*	*20.8 %*	*104*	*0.136*	*13.6 %*
CO_2	*0.3*	*0.00039*	*0.04 %*	*40*	*0.052*	*5.3 %*
H_2O	*3.7*	*0.0048*	*3.7 %*	*47*	*0.62*	*6.3 %*
Total	*760*	*1*	*100 %*	*760*	*1*	*100 %*

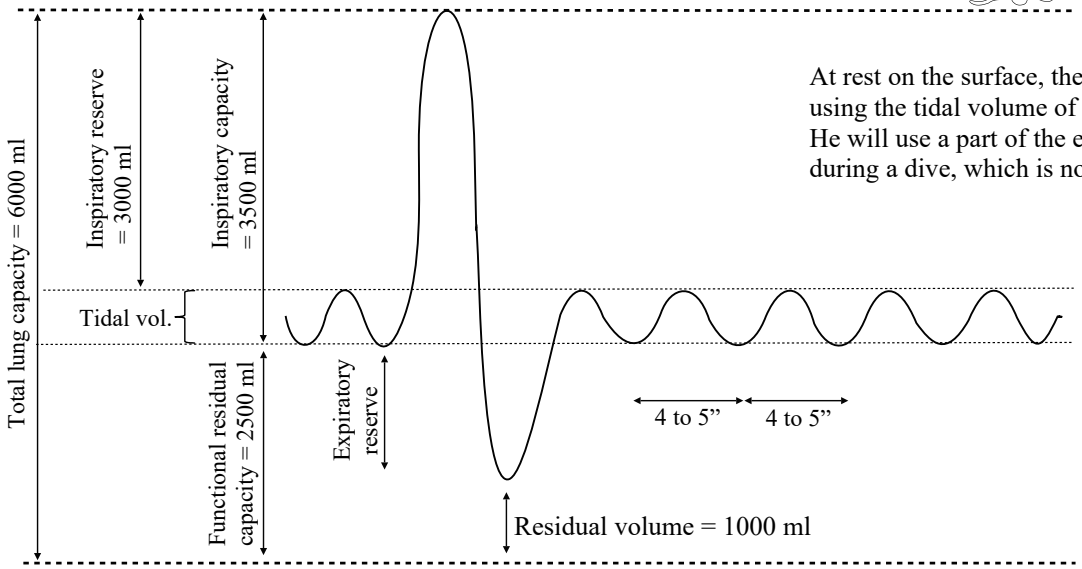
Note:
Millimetre of mercury (mm of hg) is the unit commonly used in medicine to measure the pressures:

- *760 mm hg = 1 Atmosphere*
- *760 mm hg = 1013 Mb*
- *40 mm hg = 53.33 Mb*
- *0.3 mm hg = 0.4 Mb*

The oxygenation and pulmonary ventilation is adapted to the needs of the organism by the respiratory muscles which are innervated by motor nerves *(see "Circulatory and respiratory systems" in part 2 of this book)*.
Breathing is a vegetative function which can be modified by the will.
The adaptation of the vegetative respiratory system is dependent on nerve centres, which provide management, synchronization, and control of the entire ventilation. These respiratory centres are situated in the brainstem and are independents from the rest of the brain, operating automatically without the concurrence of the will. The will intervenes only for the forced inspiration and expiration.
The respiratory centres adjust their actions based on the content of oxygen and carbon dioxide in the blood. Any elevation of the CO_2 level in the blood leads to an increased ventilatory flow. The rising of the ventilation automatically triggers the frequency of the heart to eliminate the excess of CO_2 as soon as possible.

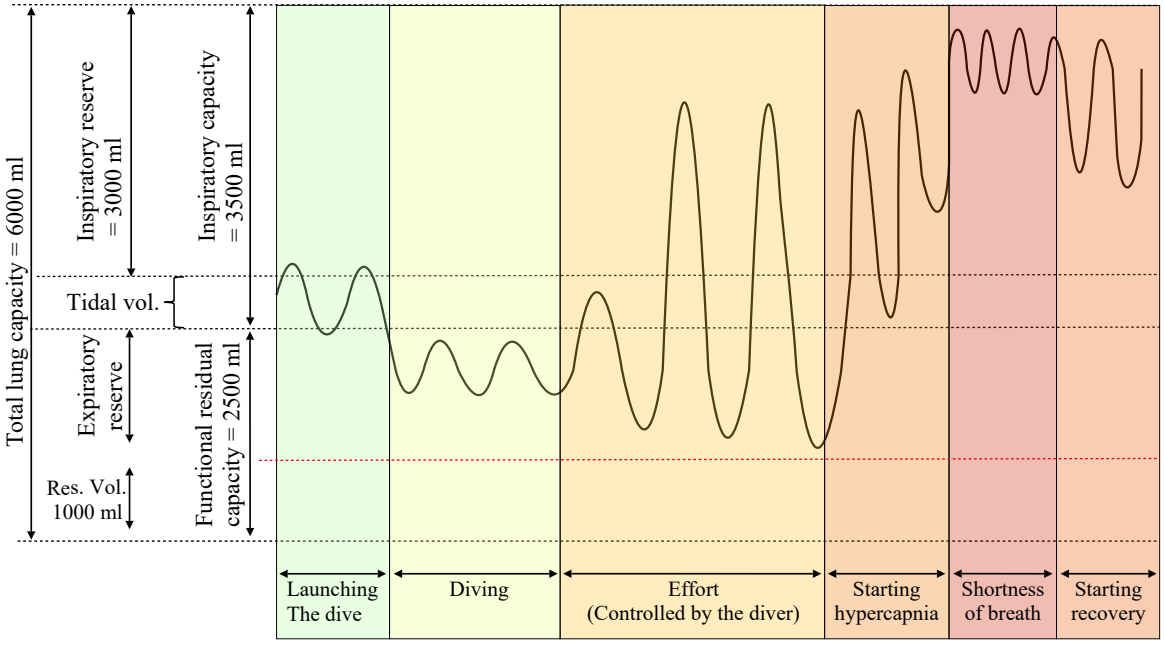
During the diving operations, the partial pressure of CO_2 in the gas supply increases due to the absolute pressure at depth.

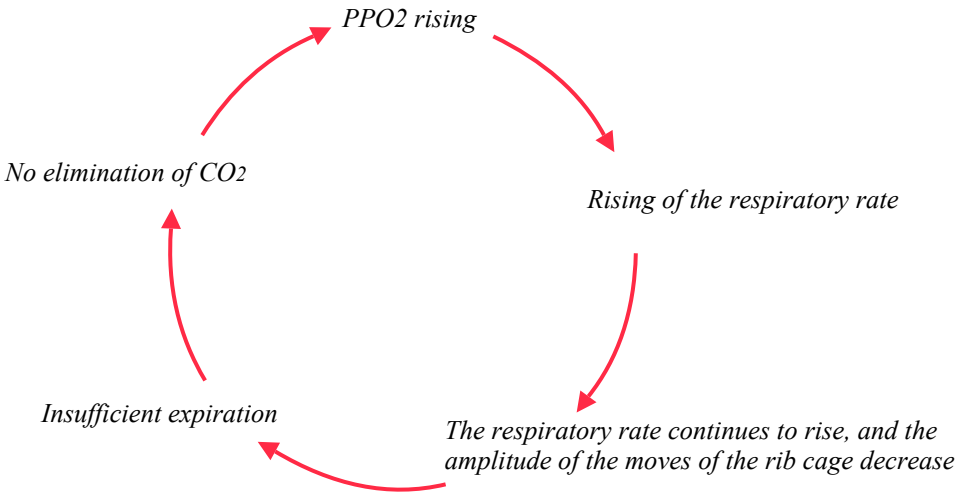
Depth	***Absolute pressure (bar)***	***% CO_2***	***PPO_2 (bar)***	***PPO_2 (mb)***	***PPO_2 (mm hg)***
0 m	*1*	*1*	*0.01*	*10*	*7.5*
10 m	*2*	*1*	*0.02*	*20*	*15*
30 m	*4*	*1*	*0.04*	*40*	*40*
50 m	*6*	*1*	*0.06*	*60*	*45*



At rest on the surface, the diver breathes using the tidal volume of the lung capacity. He will use a part of the expiratory reserve during a dive, which is not requiring efforts.

If the excess of CO_2 cannot be eliminated, the partial pressure of CO_2 continues to rise. As a result, the ventilation and the cardiac rate rise and the amplitude of the movements of the thoracic cage as well. At this moment, the body uses the inspiratory and expiratory reserve. The diver can control this process when it starts, because he still has the possibility to perform forced expiration and inspiration using the expiratory and inspiratory reserves.
However, if the CO_2 is still not eliminated, the respiratory and cardiac rates increase again, but the thoracic cage is not able to follow the frequency requested and the amplitude of the movement decreases quickly with the elimination of the CO_2 becoming inefficient.
At last, only one part of the inspiratory reserve is used and no more expiration is performed, allowing the partial pressure of CO_2 to rise again. That can lead to a fatality if nothing is done.





Symptoms

As described previously, the symptoms are linked to the partial pressure of carbon dioxide (CO_2).

The chart below indicates the possible symptoms for a mix with 1% CO_2.

Note that 5 mb is the maximum partial pressure of carbon dioxide agreed in a diving system

Depth	*Absolute press (ATA)*	*% CO2*	*PPO2 ATA*	*PPO2 mb*	*Visible symptoms*
Surface	*1*	*1*	*0.01*	*10*	*- Nothing visible* *- Note that 10 Mb was the maximum agreed in "emergency" in previous times.*
10 m	*2*	*1*	*0.02*	*20*	*- Slight hyperventilation*
30 m	*4*	*1*	*0.04*	*40*	*- Shortness of breath associated with headaches* *- Possible panic*
50 m	*6*	*1*	*0.06*	*60*	*- Strong shortness of breath associated with narcosis* *- Panic* *- Irrational behavior*
60 m	*7*	*1*	*0.07*	*70*	*- Uncontrolled shortness of breath associated with strong narcosis* *- Hallucinations* *- Dizziness* *- Vomiting* *- Strong panic*
70 m	*8*	*1*	*0.08*	*80*	*- Strong dizziness* *- Stupor* *- Loss of conscious*

Note that:

The effects of narcosis and hypercapnia can add to each other: A diver under the influence of narcosis will probably not note the warning signs of carbon dioxide intoxication. Hypercapnia, in turn, will intensify the symptoms of narcosis.

Carbon dioxide in excess dilates the arteries of the brain. This may partially explain headaches often associated with carbon dioxide intoxication, though these headaches are more likely to occur following the exposure than during it.

The increase in blood flow through the brain, which results from dilation of the arteries, can explain the reason carbon dioxide in excess speeds up the onset of CNS oxygen toxicity.

Excess carbon dioxide during a dive is also believed to increase the likelihood of decompression sickness, but the mechanism is not fully clarified.

Treatment

The strategy consists of reducing the excess of carbon dioxide as soon as possible.

- During the diving operation:
  - The diver affected should stop the job undertaken and flush his helmet. In complement, the diver should ascend several meters and ensure that the phenomenon is fully stopped.
  - If the procedure above is not sufficient, the diver must return to the basket/bell.
  - The dive must be stopped as soon as possible in case of severe symptoms.

- The supervisor should switch to the backup gas supply for precaution.
- The rescue diver should be sent to assist if the diver is not able to return in the bell.

- In the chamber
  - The divers should be on BIBS until the return to the normal situation.
  - The Life Support Technician should switch on the backup supply, and flush the chamber.
  - The gas supplies should be checked and the soda lime of the cartridge of the scrubber be changed.
  - The analysers and their calibration should be verified.

Prevention

The prevention is linked to gas management procedures and the control of the efforts during the operations.

- The divers should be trained to control their efforts and detect the 1st signs of hypercapnia.
- The gasses planned to be in line must be checked.
- Chambers must be provided with CO_2 analyzers that must be must be in line: PP CO_2 < 0.005 bar (5 mb) in chamber.
- The diving equipment should be regularly maintained and checked by competent personnel. Helmets and gas supply equipment not in perfect condition should not be used.
- A proper checklist should be performed prior to dive.
- Continuous communications with the diver should be in place with adequate monitoring.
- Task plans should be designed to avoid unnecessary efforts.

Drowning

Definition

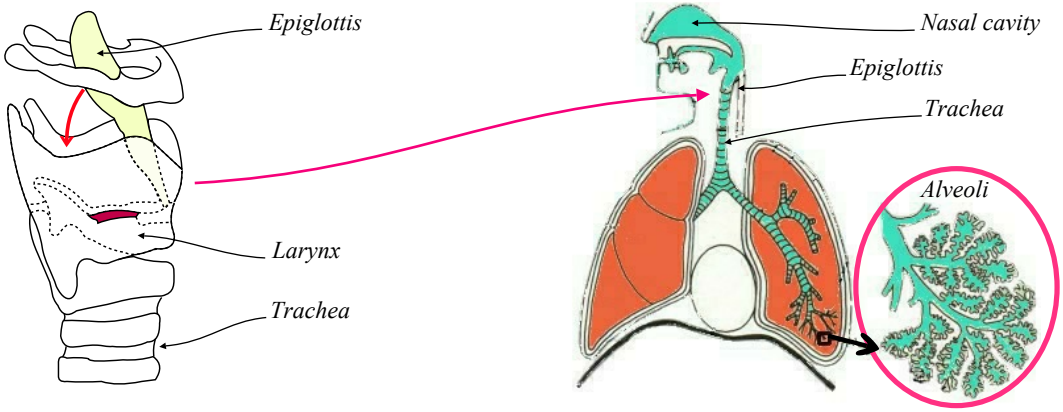
Drowning is defined as death due to asphyxia by fluid.
When patients lose consciousness due to immersion and aspiration, but subsequently recover, the term "near drowning" is used. Near drowning can lead to serious secondary complications or death, possibly up to 72 hours after the event, in this case, the term "secondary drowning" is used.

There are a lot of causes for drowning:

- Personnel falling into the sea without a floating device and not being able to swim.
- Helmet invaded by water due to neck dam failure or other maintenance problems.
- Helmet / full face mask ripped off due to external events like a blast, too strong current, or other external events.
- Loss of conscience of the diver with the head down following, hypothermia, narcosis, hypercapnia, injury, or other bad events.
- Illogical actions like removing the helmet due to narcosis, hypercapnia, panic, etc.

Elements to consider

The degree of panic of the casualty is variable and may be reduced by factors such as personality, training, resistance to narcosis, and hypothermia.
If the casualty is totally deprived of gas for some reason, he initially holds breath until the "breakpoint" is reached, and then he takes an involuntary breath. The inhalation of water usually provokes coughing and closure of the epiglottis (valve situated on larynx preventing any liquid or solid intrusion in the airways), producing involuntary breath-holding followed by unconsciousness. It is unusual that large amounts of water enter the lungs after the victim loses consciousness as the tongue and loose tissues in the throat tend to close the airway. Instead, there is often swallowing of seawater (in the stomach), making the victim susceptible to vomiting.



The temperature of the water is also important: As an example, victims with the face submerged in waters at a temperature less than 21°C can have a reflex of bradycardia *(heart rate slowing down)* with peripheral vasoconstriction *(blood flow restriction in the limbs)* and the blood shifting to the thoracic cavity to avoid the collapse of the lungs. This is triggered by the "autonomic" nervous system and can happen to conscious or unconscious persons. This reflex allows the casualty to stay underwater for extended periods. Waters warmer than 21 °C does not cause the reflex.

Fresh or saltwater entering the alveoli *(where the gas exchange normally takes place)* wash out or damage the surfactant lining them, causing alveoli to collapse and become unavailable for gas exchange.
Damage to the alveoli walls also causes the capillaries to leak blood and protein into the lungs. That interacts with air and water, producing a foam the victim may cough up in copious amounts. This state is called pulmonary oedema. Water in the lung will be the main cause of "the secondary drowning" if not detected.
In addition to what has been indicated above, there is a difference between drowning in seawater and freshwater: The main water difference between saltwater and freshwater drowning involves osmosis occurring between the lung's surface and the bloodstream:

- When saltwater enters the lung, the high salt content prevents it from crossing the capillaries' membranes. Instead, it causes blood and water from the bloodstream to cross over the capillary walls' membranes and into the lungs. This prevents oxygen from entering the bloodstream and causes the victim to drown in their own fluids.

- When freshwater enters the lungs, the low salt content allows the water to cross the membrane of the capillary walls, and it will be absorbed into the bloodstream. This causes damage to the blood and causes cardiac arrest very quickly.

The severity of pulmonary damages also depends on the aspirated material's pH, the volume of the aspirate, and if particulate matter like vomit and bacterial contamination are present in the aspirate. When the pH level of an aspirate is 2.5 or less, a severe pulmonary response occurs. When the pH is below 1.5, the patient usually dies. The mortality among patients who aspirate material grossly contaminated approaches 100%.

Symptoms

1 - Drowning:

- The patient has been in the water for too long time to be successfully resuscitated.
- Note that the statement of death is to be decided by the doctor.

2 - Near drowning:

- Increased respiratory rate
- Casualty vomiting
- Pulmonary oedema
- Unconsciousness

3 - Secondary drowning :

- Irritation or pain in the throat or chest
- Coughing after taking a deep breath or persistent coughing or wheezing
- Shortness of breath or difficulty breathing and unusual fatigue
- Dizziness/altered level of consciousness
- Hypoxia, hyperoxia, poisoning
- Vomiting or diarrhea
- Epilepsy, thermal stress, loss of conscience

Treatment

1 - Near drowning:

- Remove all foreign objects from the mouth.
- Restore breathing and heart beat as soon as possible (CPR).
- Subsequent to resuscitation, keep the patient warm and rested.
- Give therapeutic mix with the maximum allowable PPO2 on BIBS (diver in saturation) or 100-percent oxygen by mask (victim at the surface).
- Call for assistance from qualified medical personnel.
- Regardless of the severity of a near-drowning case, the victim should be transferred to a hospital as quickly as possible: Pulmonary oedema (accumulation of fluids in the lungs), pneumonia, and other complications may occur many hours after the incident. Immediate transfer to the hospital is impossible when the diver is in saturation because the ascent may take several days. Therefore, proper medical observation is essential, and communication with the diving medical specialist is essential, as well as the use of the system that allows him to check the status of the victim remotely. This remote system should be ready for use at all times.
- Notes regarding resuscitation:
  - If a neck injury is suspected, the victim's neck should be supported in a neutral position as soon as possible. If the victim must be turned, the head, neck, chest, and body should be aligned. If artificial respiration is required, maximal head-tilt should not be used. Rescue breathing should be provided with the head maintained in a neutral position without head-tilt, or chin-lift without head-tilt should be used.
  - Heimlich manoeuvre is not recommended to remove fluids. The technique may have relevance in situations where airways are obstructed by solids but not fluids. Performing the manoeuvre on drowning people not only delays ventilation but may induce vomiting, which, if aspirated, will place the patient in a far worse situation. Moreover, the Heimlich manoeuvre in any choking situation involving solids or fluids has become controversial.

2 - Secondary drowning:

- Oxygen 100% O2 should be provided as soon as possible.
- Evacuate the patient as soon as possible (MEDEVAC).

Note

The drowning can be associated with omitted decompression or decompression illness, which will have to be treated if the victim has passed the excursion limits during the incident.

Prevention

- Training and selection of the divers
- Prudent divers and supervisors, checklists always performed, and proper task plans.
- Diving equipment always in perfect condition
- The rescue diver must be ready to intervene.
- The personnel in charge of the launching and recovery of the bell working near the edge of the boat (or the moonpool) should wear a life vest + harness with stop fall.

Pulmonary barotraumas

This topic is discussed in five separate points:

1 - Definition

2 - Mediastinal and subcutaneous emphysema

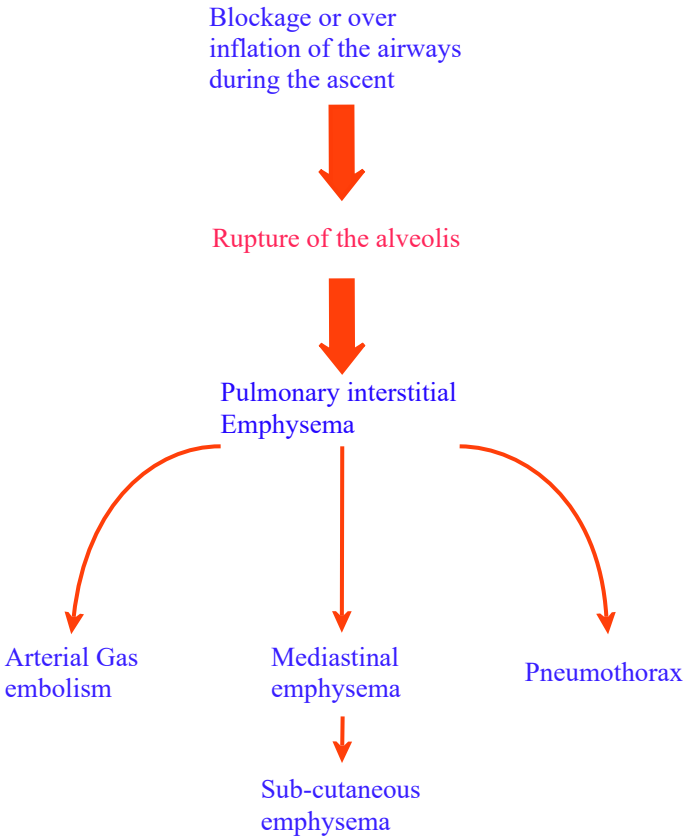
3 - Pneumothorax

4 - Arterial Gas Embolism

5 - Prevention of pulmonary barotraumas

1 - Definition

Pulmonary barotraumas are disorders that are caused by gas expanding within the lungs. If the alveolar space (in part or whole) is closed at the ascent, the volume of air they contain increases to the rib cage's elasticity limit, and the pressure increases also. It is sufficient to have the internal pressure exceeding the ambient pressure of 80 mmHg or 106 mb; thus, 1 meter of seawater, to have alveolar damage or rupture.
The disorders encountered in diving are mediastinal emphysema with possible subcutaneous emphysema, pneumothorax, and arterial gas embolism. These disorders are described in the next pages.



Pulmonary barotraumas are mostly noticed with surface supplied diving operations. That explains why most treatment procedures provided in medical manuals are based on actions at the surface. Nevertheless, pulmonary barotraumas may happen during saturation, and in such cases, the treatment is to be implemented in the chamber. Note that such an accident may be associated with other problems, such as decompression illness or wounds.

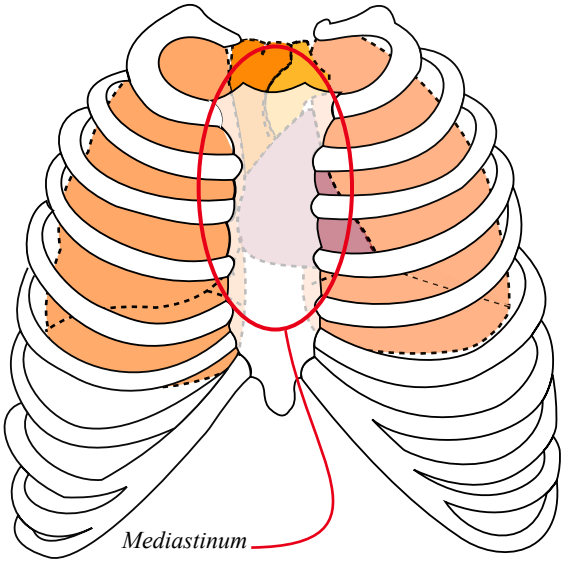
Note:

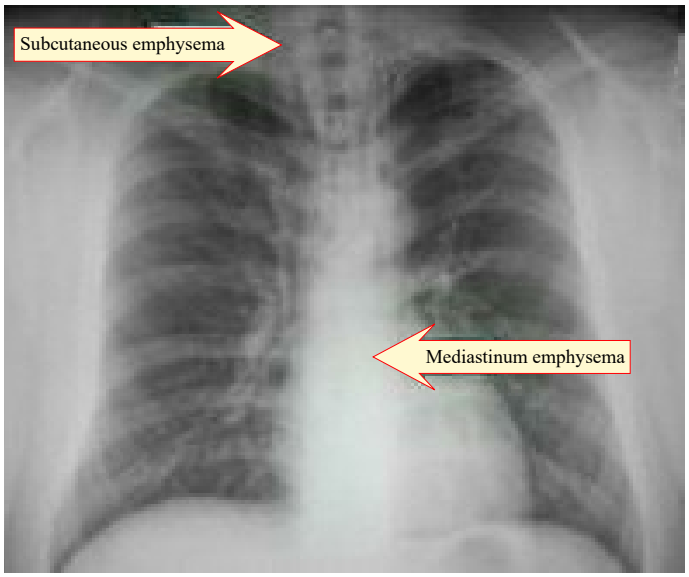
- Mediastinal emphysema causes no symptoms unless further leaking occurs.
- Pneumothorax occurs if gas accumulates between the lung and chest wall, and if accumulation continues without venting, then tension pneumothorax may result.
- If gas enters the blood circulation, potentially fatal Arterial Gas Embolism may occur.

2 - Mediastinal and subcutaneous emphysema

Description

Mediastinal emphysema is caused by gas expanding in the tissues behind the breast bone. Subcutaneous emphysema results from the movement of the gas from the mediastinal to the region under the skin of the neck and lower face. The gas can also migrate to the abdominal cavity.





Symptoms

Mediastinal emphysema:

- Often unnoticed by the casualty in mild cases
- Mild to moderate pain under the breast bone
- Dull ache
- Feeling of tightness
- Deep inspiration painful.
- Coughing and swallowing painful
- Pain radiating to the shoulders and the back

Subcutaneous emphysema :

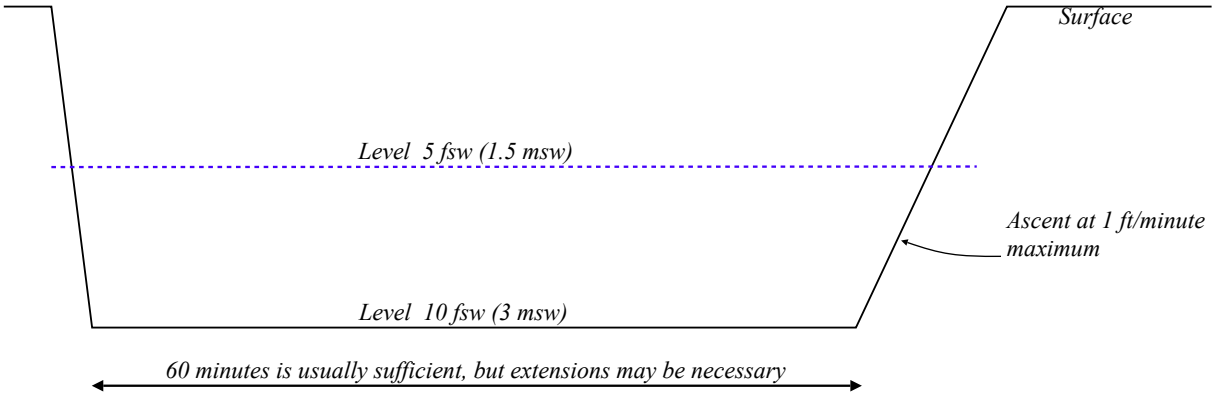
- A feeling of fullness around the neck
- Difficulties in swallowing
- Hoarse voice
- Swelling or apparent inflation of the neck
- The skin near the collar bone and the windpipe producing a cracking sound

Treatment

The treatment must be organized under the direction of the diving medical specialist:

- Breathing 100% oxygen at the surface.
- Close observation for possible pneumothorax and/or Arterial Gas Embolism to be carried out.
- Recompression is generally not required.
- If the symptoms are severe, shallow recompression to 5 or 10 feet may be beneficial. Recompression should only be carried out according to the guidelines from the hyperbaric doctor who is ruling out the occurrence. The Recompression is performed with the victim breathing 100-percent oxygen and using the shallowest depth of

relief (usually between five or ten feet). An hour of breathing oxygen should be sufficient for resolution, but longer stays may be necessary. The ascent rate must not exceed 1 foot / minute.



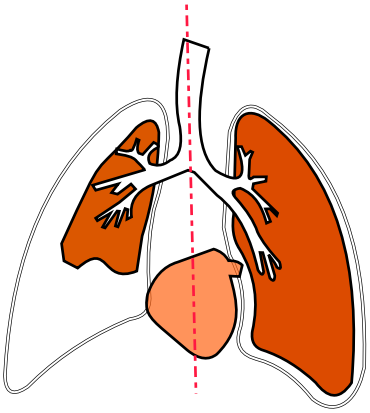
- MEDEVAC to specialized hospital to be organized when possible.

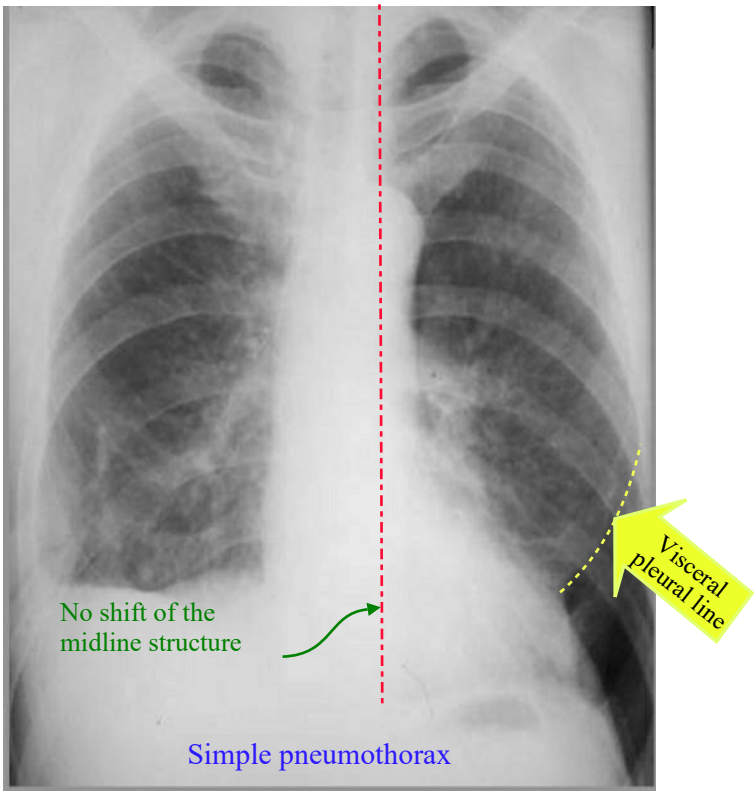
3 - Pneumothorax

Description

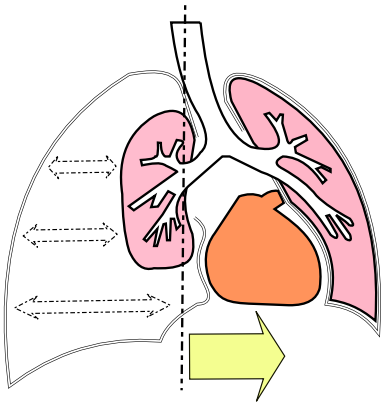
Pneumothorax is the penetration of air into the pleural cavity, which separates the lung from the chest wall and interferes with normal breathing. It is mostly accompanied by bleeding (haemothorax).
Under normal conditions, the lungs adhere to the back of the ribs by suction as there is no air in the pleural space. The escaped gas will break this suction, causing a partial or complete collapse of the lungs.
There are 2 types of pneumothorax: Simple and tension pneumothorax.

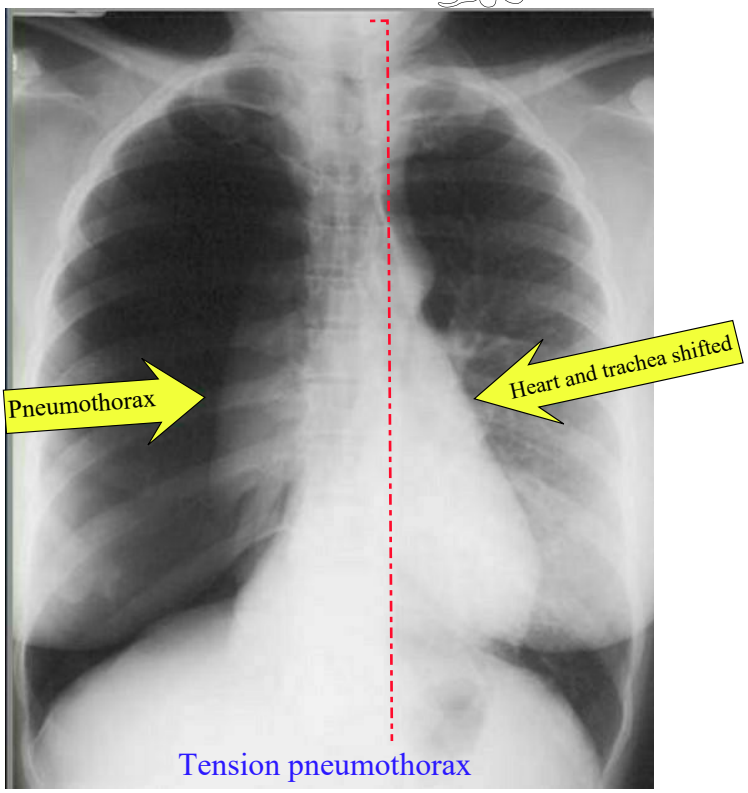
In a simple pneumothorax, air or gas comes in and out of the pleural space. The respiratory distress usually does not get worse after the initial gas leaks out of the lung.
There is no shifting of the midline structures.





A tension pneumothorax is when the damaged lung tissue acts as a one-way valve, allowing gas to enter the chest cavity but not to leave. Under these circumstances, the size of the pneumothorax increases with each breath. That can cause a shift of the heart and the mediastinal structures (esophagus, trachea…). As a result, the opposite lung is compressed with the respiratory compromised. The casualty will be quickly shocked and dead if the trapped gas/air is not removed.





Symptoms

The symptoms depend on the severity of the pneumothorax:

- Signs may not be apparent if the pneumothorax is relatively small. In this case, the pneumothorax can be identified by X-rays.

- Signs should be visible in case of severe pneumothorax:
  - Sudden, sharp chest pain.
  - Victim with tendency to bend the chest toward the involved side.
  - Cough associated with bloody sputum.
  - Inaudible breath on the affected side.
  - Percussion of the chest may be perceived as hyper-resonant (like a booming drum).
  - Short and painful breath.
  - Increased heart rate and weak pulse.
  - Cyanosis.
  - Loss of conscious and death can quickly happen.

Treatment

Medical support (Diving medical specialist) is essential.

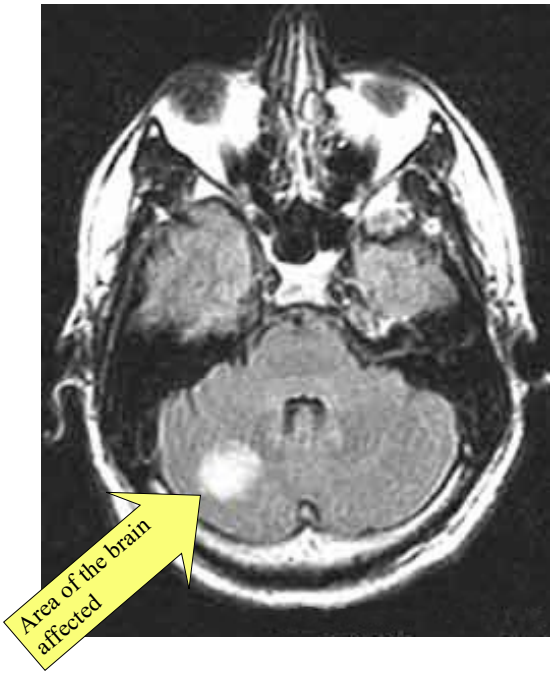
- The casualty must breathe 100% oxygen
- Cases of pneumothorax compromising the cardiac and respiratory functions may require the insertion of a chest tube or other device (like a large intravenous catheter) designed to remove the intra-thoracic gas. (To be done by a trained diver medic or nurse only)
- Close monitoring for symptoms of Arterial Gas Embolism or Decompression illness should be undertaken.
- If present in combination with Arterial Gas Embolism or decompression sickness, Pneumothorax should not prevent immediate recompression therapy. However, a pneumothorax may be vented as described above before ascending from the treatment depth.
- Decompression to be organized after stabilization according to the recommendations from the Diving Medical Specialist.
- MEDEVAC by specialized services/companies to be organized according to the doctor's recommendations.

4 - Arterial Gas Embolism

Description

Arterial Gas Embolism is caused by the entry of gas bubbles into the arterial circulation. These bubbles then obstruct one or several blood vessels and create embolism due to the pressure forcing the gas/air through the alveolar walls into the surrounding tissues and the bloodstream. If the gas enters the arterial circulation, it will be dispersed to all organs of the body. The organs that are especially susceptible to arterial gas embolism and responsible for the life-threatening symptoms are the central nervous system (CNS) and heart. In all cases of Arterial Gas Embolism, associated pneumothorax is possible and should not be overlooked.

The following elements are to be considered:

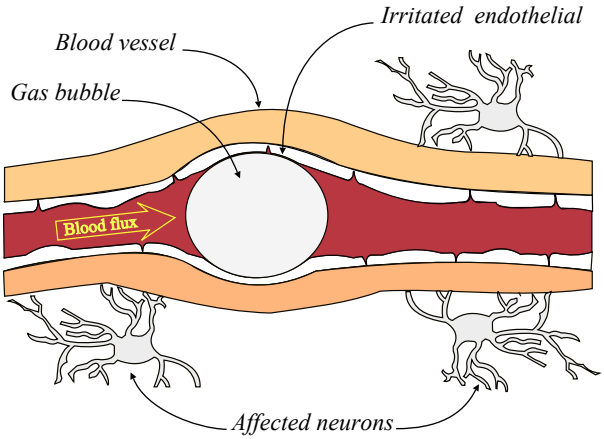


Fat has an affinity with nitrogen - Remember that the central nervous system is mainly composed of fat.

Small bubbles redistribute, and hypertension and vasodilatation promote redistribution.

Large bubbles occupying several branching arteries may trap small bubbles.

Hypotension promotes trapping.



Trapped bubbles cause a restriction of blood supply to the tissues (ischemia): That can be critical if the problem is not quickly resolved.

The bubbles damage the thin layer of cells that line the interior surface of blood vessels and lymphatic vessels (endothelium).

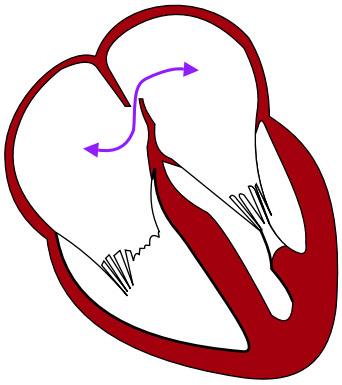
Patent foramen ovale:

Arterial Gas Embolism is mainly caused by damages to the vascular filter and associated with the mediastinal and/or pneumothorax. It can also result from a shunt in the heart due to the "foramen oval".

The "foramen ovale" is a hole between the right and the left atrium of the heart.

It normally exists while babies are in the womb but should close soon after birth. If it does not close, the condition is called “patent foramen ovale”.

This problem occurs with 25% of people. In this case, the bubbles can pass directly from one side to the other without being stopped by the pulmonary filter. An accident can affect people suffering this malformation without any noticeable events. The problems arising from shunts are discussed in detail in the chapter “Decompression sickness”.



Symptoms

The symptoms are very similar to decompression illness type 2. The main difference is the onset of AGE is usually sudden and dramatic, often occurring within seconds after arrival on the surface. For the wide majority of cases the symptoms are visible in less than 10 minutes after surfacing. The symptoms of decompression accident are generally visible after a longer time.

- Numbness or tingling
- Disturbances of movement including paralysis or/and weakness
- Sensation of fatigue
- The victim is anxious and changes of personality are visible.
- Vision disturbed
- Abnormal hearing
- Problems of elocution
- Abnormal balance or/and coordination
- Disturbances of intellectual function
- Vertigo and dizziness
- Tremors
- Heart attack including chest pain, shortness of breath and palpitations.
- Loss of conscious

Treatment

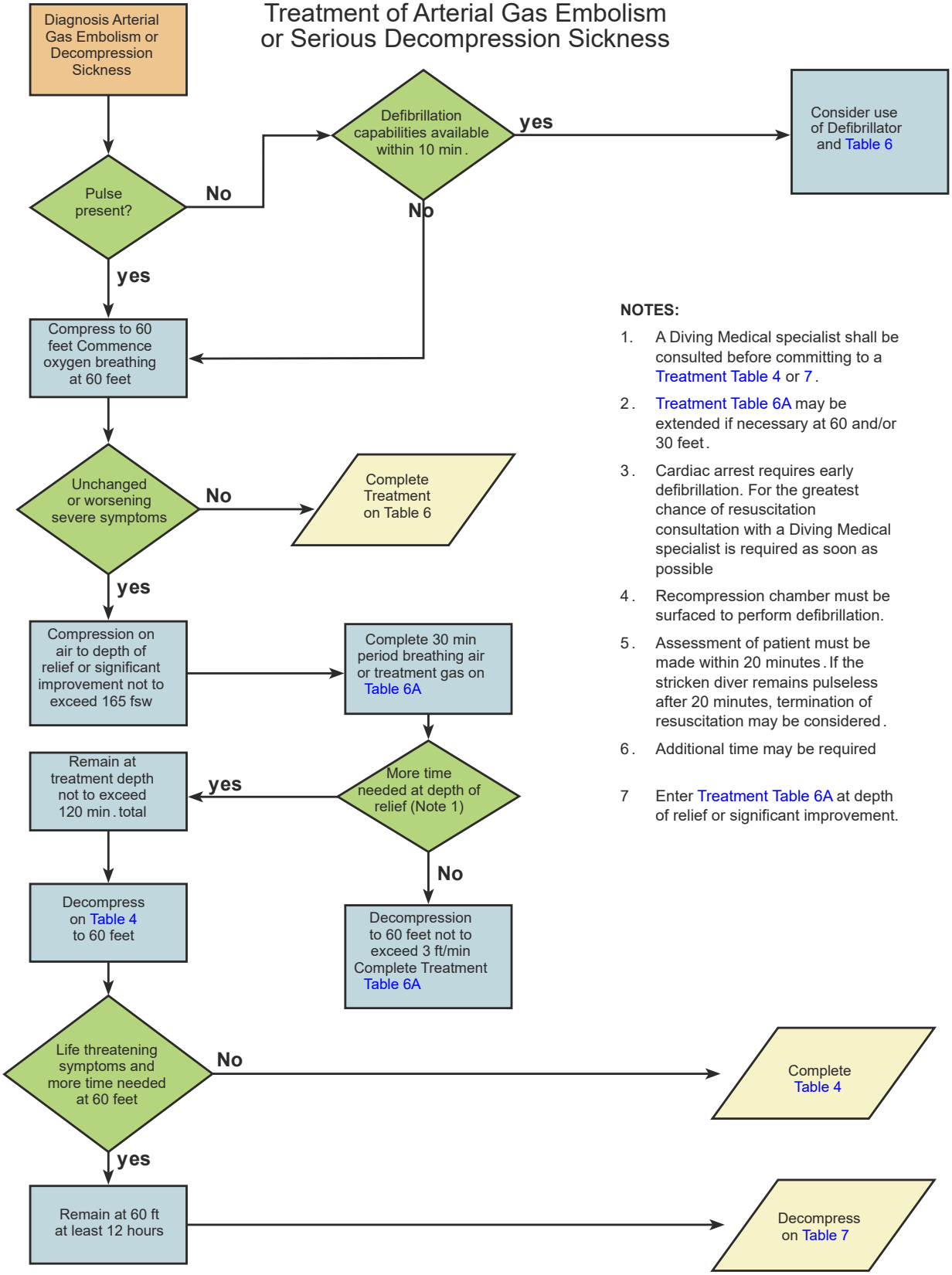
As for any emergency, the medical support must be involved as soon as possible. The Diving Medical Specialist is the person in charge of the treatment, and his recommendations are to be implemented.

- 100% O_2 is to be provided as soon as possible.
- For the majority of the cases: Immediate recompression. (*Chart US Navy is inserted next page)*
- For cases with absence of pulse and respiration, Advanced Cardiac Life Support (ACLS) is necessary. ACLS is a difficult procedure which requires immediate availability of equipment and trained specialists. ACLS procedures include diagnosis of abnormal heart rhythms and correction with the administration of drugs and defibrillation. The defibrillation must be conducted at atmospheric pressure if the equipment is not designed to be used under pressure and in a chamber (Note that it is recommended to design the chamber for this purpose). The absence of a defibrillator designed to operate under pressure may oblige the Diving Medical specialist (doctor) to decide whether he delays recompression and perform ACLS outside the chamber or begins recompression to a depth of 60 fsw, performing Basic Cardiac Life Support.

US Navy treatment tables to be used:

- The tables to be used will be table 6 *(compression at 60 ft)* at first followed by table 6A in case of severe symptoms *(165 ft)*. The patient will have to be compressed to the depth of significant improvement, but never passing below 165 ft.The patient should begin oxygen breathing periods immediately upon arrival at the 60-foot stop . If the severity of the patient's condition warrants, the Diving medical specialist (Doctor) may decide conversion to Treatment Table 4.
- Treatment Table 4 is used when it is determined that the patient would receive additional benefit at depth of significant relief, not to exceed 165 fsw. The time at depth shall be between 30 to 120 minutes, based on the patient's response.
- The breathing periods are 25 minutes on oxygen, interrupted by 5 minutes of air.
- In case of non response , the doctor can decide to apply table 7. Treatment Table 7, is an extension at 60 feet of Treatment Table 6, 6A, or 4 (or any other nonstandard treatment table). Treatment Table 7 is considered an extreme measure for treating non-responding severe gas embolism and is not designed to treat all residual symptoms that do not improve at 60 feet. It should never be used to treat residual pain. Treatment Table 7 should be used only when loss of life may result if the currently prescribed decompression from 60 feet is undertaken.
- In case of associated pneumothorax, a chest tube or other device (like a large intravenous catheter) designed to remove the intra thoracic gas must be inserted. (To be done by a trained diver medic or nurse only). The ascent rate will be decided by the doctor.
- Important point:The shifts from a treatment table to another must be decided by the hyperbaric doctor in charge of the treatment. Medical tables are considered medical treatment and the decisions regarding medical treatments to be applied have to be made by a qualified hyperbaric doctor.
- MEDEVAC by specialized services/companies to be organized in accordance to the guidelines of the doctor in charge.

Treatment of Arterial Gas Embolism or Serious Decompression Sickness



NOTES:

1. A Diving Medical specialist shall be consulted before committing to a Treatment Table 4 or 7.
2. Treatment Table 6A may be extended if necessary at 60 and/or 30 feet.
3. Cardiac arrest requires early defibrillation. For the greatest chance of resuscitation consultation with a Diving Medical specialist is required as soon as possible
4. Recompression chamber must be surfaced to perform defibrillation.
5. Assessment of patient must be made within 20 minutes. If the stricken diver remains pulseless after 20 minutes, termination of resuscitation may be considered.
6. Additional time may be required
7. Enter Treatment Table 6A at depth of relief or significant improvement.

Note:

- The medical decompression tables are explained and available point “C Medical tables US Navy 6.1”.
- The forms to follow up the treatment and the decompression are available point D “Dive reports forms”

Decompression accidents

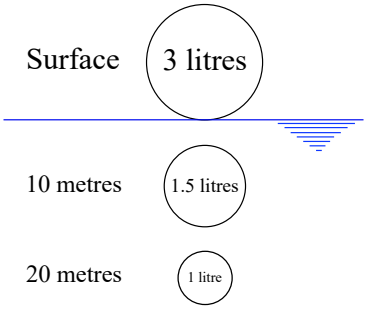
Description of decompression sickness

Decompression sickness (DCS) is a disease resulting from the liberation of gas bubbles in solution into the tissue or the blood. The effects can be relatively minor with full recovery or permanent impairment or worse, death.
Various conditions may cause the diver to absorb an excessive amount of inert gas or inhibit the elimination of the dissolved gas during normal controlled decompression. In certain individuals, decompression sickness may occur even though decompression procedures are followed meticulously. Decompression sickness may also occur with exposure to a lower pressure than sea level after the arrival at the surface. It is the case of dives performed in altitude where a correction must be applied to avoid the problem or a sudden loss of cabin pressure in an aircraft.

The mechanism is linked to 3 physical basic laws:

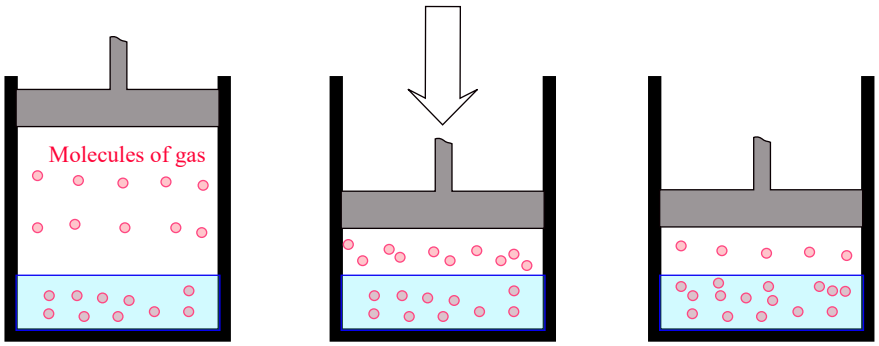
1 - Boyle - Mariotte law:

The absolute pressure exerted by a given mass of an ideal gas is inversely proportional to the volume it occupies if the temperature and amount of gas remain unchanged within a closed system.
As result a ball filled with gas will be compressed and have half the initial volume at 10 m and only 1/3 at 20 m



2 - Henry law:

At a constant temperature, the amount of a given gas that dissolves in a given type and volume of liquid is directly proportional to the partial pressure of that gas in equilibrium with that liquid.



Initial pressure — Increasing pressure — Final status

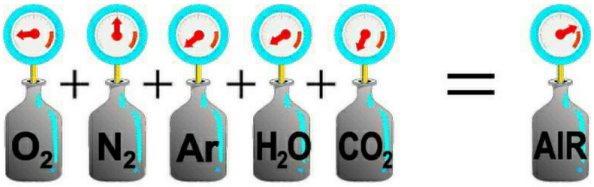
- The solubility of a gas in a liquid is the property the gas has to dissolve in the liquid to form a homogeneous solution.
- The point at which no more gas can be absorbed or assimilated is called “saturation”.
- Note that the solubility of a gas in lipids is also linked to Meyer-Overton hypothesis.

Comparison of various gas solubilities

Gas	*Molecular Mass*	*Voluminal Mass*	*Solubility in water*	*Solubility in oil*
Nitrogen	*28*	*1.25*	*13*	*67*
Oxygen	*32*	*1.43*	*29 (@ 25° C)*	*120 (@ 40° C)*
Argon	*40*	*1.78*	*26*	*140*
Helium	*4*	*0.18*	*8.6*	*15*

3 - Dalton law:

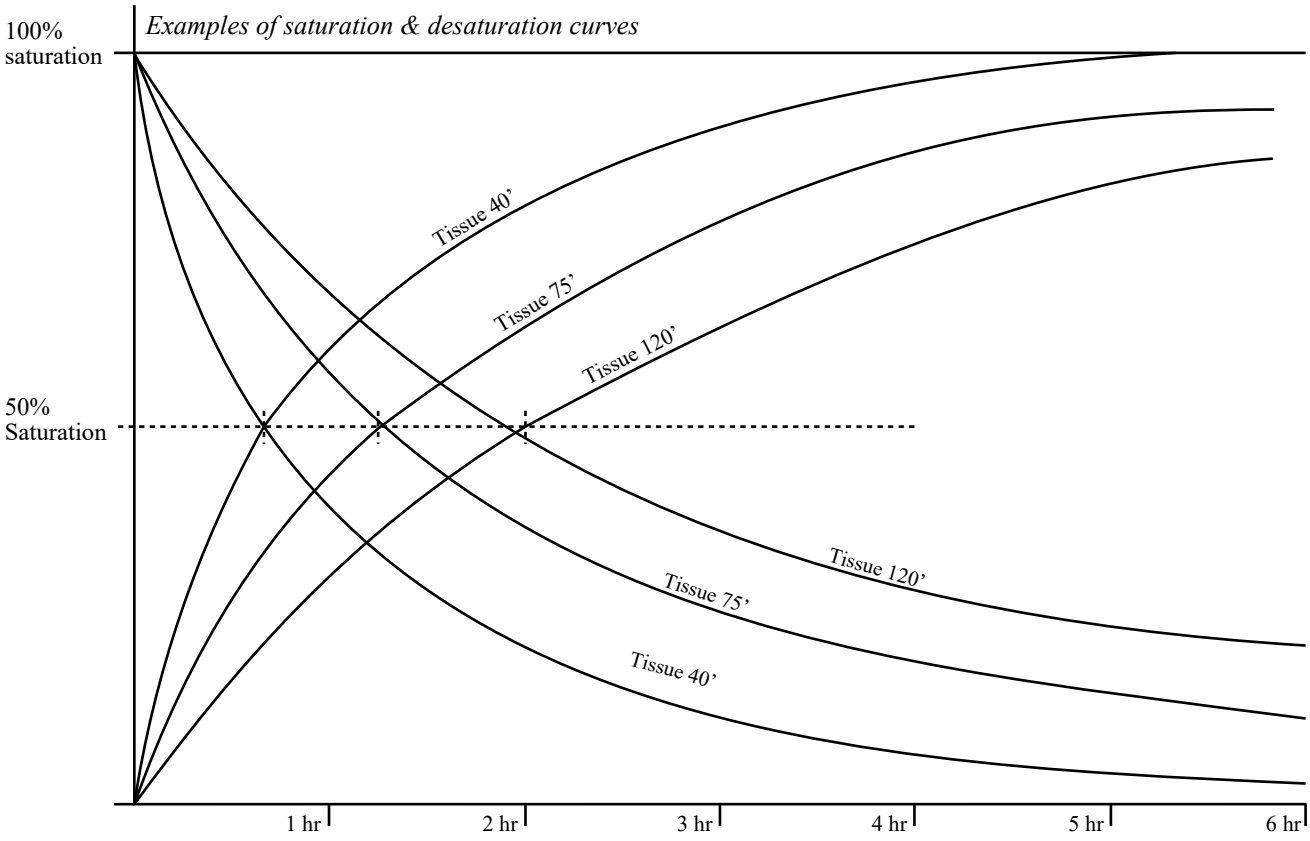
The total pressure exerted by the mixture of non-reactive gases is equal to the sum of individual gases' partial pressures. In other words, total pressure = P1 + P2 + P3 ….



Depending on the mix, nitrogen or helium (He) is gradually absorbed by the organism during the phases under pressure while it is considered that oxygen is consumed. When a diver breathes a mix at depth, the N2 or He is breathed at increased partial pressure, and the gas diffuses from areas of high concentration (high partial pressure) to areas of lower concentration. The N2 or He is taken up from the lungs by the blood and transported around the body and into the tissues. The greater the depth, the greater the partial pressure of N2/he, and therefore the amount of N2 or He absorbed.

The speed the N2 (or He) is distributed into the tissues also depends on the blood flow. The tissues with high metabolism like the brain, heart, kidneys, and liver are very vascularized. Thus, they will have a rapid N2 or He uptake. Such tissues are called "fast tissues" because of their fast N2 or He uptake. Because the blood passing through the lungs equilibrates immediately with any change in the inspired N2 or He partial pressure, the blood is the fastest tissue of all. Other issues like the ligaments, tendons, and fat, with a relatively small blood flow, have a relatively slow N2 or He uptake. These tissues are called "slow tissues". Between the two are tissues of intermediate blood flow, such as muscle. Some organs, such as the spinal cord, have both fast and slow tissue components. The rate of uptake of the N2 or He in a specific tissue is exponential; it varies depending on the amount of gas already taken up by the tissue.

The N2 or He is eliminated in a reverse process: As the diver ascends, there is a reduction in the partial pressure of N2 or He in the air he breathes, allowing blood to release N2 (or He) into the lungs. The decrease in the blood level of N2 or He causes N2 or He to diffuse into the blood from the tissues. Fast tissues naturally unload N2 or He quicker than slow tissues. Note that the speed of saturation and de-saturation are not the same, with de-saturation taking more time.



During the ascent, the pressure surrounding the diver (environmental pressure) is reducing. In this case, the pressure of N2 or He dissolved in the tissues is becoming greater than the environmental pressure. The tissue is then said to be "supersaturated". The tissues can tolerate a certain degree of gas supersaturation. Still, if the pressure of N2 or He in the tissues exceeds the environmental pressure by a critical amount, bubble formation is likely. The bubbles can form in any tissue in the body, including blood. The pressure in each bubble will be the same as the environmental pressure, and Boyle's law will govern the bubble size. Once a bubble has formed, if nothing is done, it will continue to grow until the N2 or He excess has been eliminated. Once this has occurred, the bubble will begin to decrease in size, but it may take hours, days, or weeks to disappear. In the meantime, the bubble will damage the surrounding tissues. This is called “decompression sickness”.

Bubble formation:

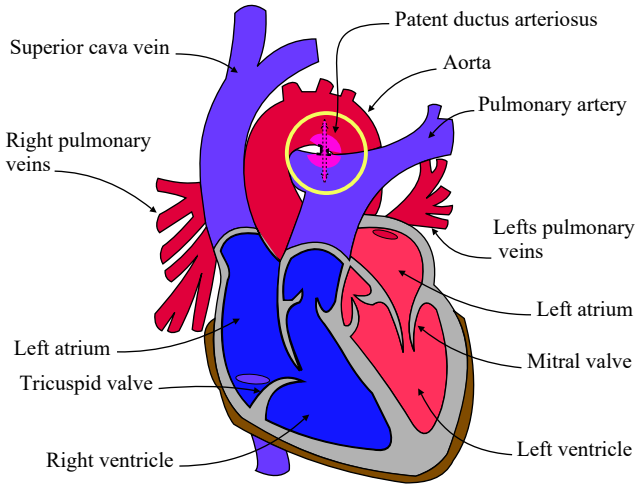
- Gas nuclei & Vascular Gas Embolism (VGE):

The bubble formation during the decompression is not only the consequence of inert gas supersaturation. Numerous experiments indicate that bubbles may originate from pre-existing gas nuclei, which are bubbles of less than 1 Micro. Vascular Gas Embolism (VGE) is the entrainment of gas into the venous or arterial vasculature, producing systemic effects. Vascular Gas Embolism involves gas entering the venous circulation with embolism into the pulmonary circulation causing right heart failure and cardiovascular compromise. In the document *"Preconditioning to Reduce Decompression Stress in Scuba Divers"*, doctors Peter Germonpré and Costantino Balestra say that the precise mechanism of micronuclei formation is still debated, with possible sites in facilitating endothelial surface regions having surfactants, hydrophobic surfaces, or crevices. They also say that studies have pointed out a significant intersubject variability to VGE for the same diving exposure. There is also a large intra-individual variation, indicating that diving time and nitrogen pressure are not the only determinants of VGE formation.
In an article called *"Static Metabolic Bubbles as Precursors of Vascular Gas Emboli During Divers' Decompression: A Hypothesis Explaining Bubbling Variability"*, Jean-Pierre Imbert, Salih Murat Egi, Peter Germonpré, and Costantino Balestra say that the first step deals with cavitation in physical systems and that Brian Hills showed that cavitation occurs at the liquid/liquid interface after decompression when one of the liquids is hydrophobic (Hills, 1967). They also say that Arieli established that nanobubbles form on flat hydrophobic surface of silicon wafers from dissolved gas (Arieli & Marmur, 2011) and that these nanobubbles expand and detach to form free-floating bubbles after decompression (Arieli & Marmur, 2013a; Papadopoulou et al., 2015). To conclude with this step, they also say that this establishes a link between stationary nanobubbles on the blood vessel wall and blood-borne bubbles, which prepares the scenario for decompression VGE. In addition, they say that the second step introduces cavitation in biological systems and that Hills studied various endothelial surfaces from sheep and humans for their hydrophobicity, using a method based on the angle of contact, and found distinct hydrophobic areas (Hills, 1992), and concluded that the oligolamellar surfactant lining and lamellar bodies were potentially essential factors in influencing bubble formation on vessel walls. Similarly, Arieli showed that the production of bubbles after decompression of ovine blood vessels is associated with active hydrophobic spots (AHS) that stain for lipids (Arieli and Marmur, 2013b, 2014) and confirmed that these AHS consist of a deposit of hydrophobic lipids similar to or even originating from lung surfactant (Arieli, 2015).
The lungs normally filter the gas nuclei except when the lungs' capacity of filtration is exceeded. In this case, the nuclei can pass through the pulmonary filter and return to the blood circulation. These nuclei can aggregate and create some pathogen bubbles.
In the absence of gas nuclei, bubbles may be generated directly by tribonucleation in supersaturated tissue.
Venous bubbles are detectable post-diving, by Doppler and precordial echocardiography. After typical near-decompression dives, they are observed some minutes after ascent and persist for some hours. For short deep dives, they occur early and are not long-lasting, but they take longer to develop for longer shallower dives and may continue to be produced for many hours. Some bubbles are pathogen, but some others that are detectable in most dives, including the well-managed dives, do not produce clinical features of DCS.
These mechanisms are still debated by scientists with also the role of body substances such as Nitric Oxide (NO), an omnipresent intercellular messenger, modulating blood flow and neural activity, which is thus responsible for vasodilatation. That opens to studies on chemical reactions and drugs that may be used to interfere in these phenomenons and be used to control the production of Vascular Gas Embolisms (VGE).

- Arterial bubbles:

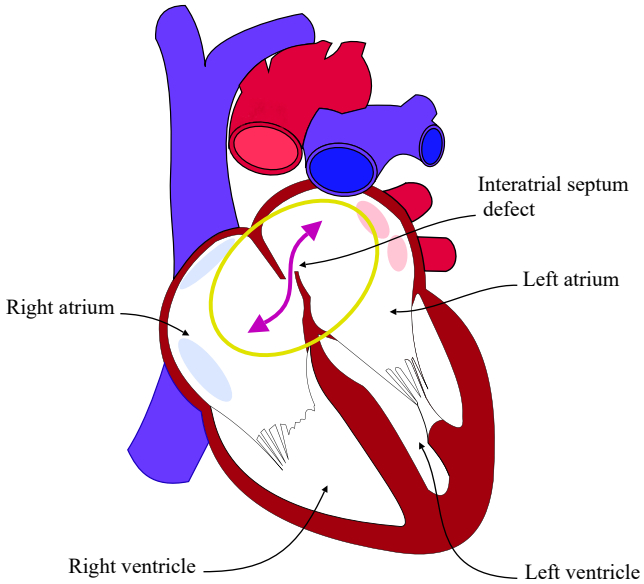
Whilst it is possible for bubbles to form from the beginning in the arterial system, this is thought to be an unlikely mechanism because the combined effects of the arterial blood pressure and the equilibration of the arterial blood with alveolar inert gas tensions will reduce the arterial pressures of these and avoid bubble formation.
Also, as indicated above venous bubbles may pass the lung filter and become arterialized if they are too many, resulting in the passage of gas nuclei and venous emboli through the left heart chambers and consequently into the arterial circulation. The venous bubbles may also gain access to the arterial circulation via congenital or pathological shunts such as those described below that are present in ¼ of people.

- A "patent ductus arteriosus" is a malformation that is a persistent communication between the descending thoracic aorta and the pulmonary artery resulting from abnormal physiologic closure of the foetal ductus of the more common congenital heart defects.

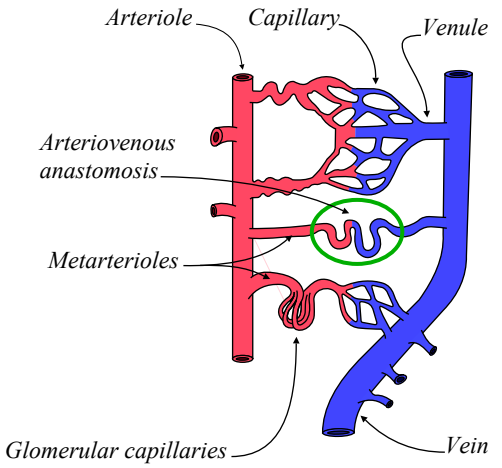


- A septal defect is one of the more commonly recognized congenital cardiac anomalies presenting in adulthood. An atrial septal defect is characterized by a defect in the interatrial septum allowing pulmonary venous return from the left atrium to pass directly to the right atrium. Depending on the size of the defect, size of the shunt, and associated anomalies, this can result in a spectrum of diseases from no significant cardiac sequelae to right-sided volume overload, pulmonary arterial hypertension, and even atrial arrhythmias.

  The importance of a right-to-left shunt in the heart, through a patent foramen oval, allowing venous gas emboli (VGE) to become Arterial Gas Embolism, has been previously observed in autopsies, and the expected incidence of this disorder among severe DCS cases has been demonstrated.



- A “Patent arteriovenous anastomosis” is a connection between two blood vessels, such as between arteries, between veins, or between an artery and a vein. "Anastomosis” normally occurs in the body in the circulatory system, serving as backup routes for blood to flow if one link is blocked or otherwise compromised, but may also occur pathologically.
  The arteriovenous anastomosis can happen, especially when the pulmonary artery pressures increase with emboli blockage of the pulmonary circuit, producing a right-to-left intra-cardiac shunt (paradoxical emboli).
  Similar effects could be induced by straining, coughing, “valsalva maneuver”, etc.…
  The greater the number of pulmonary emboli, the greater the pulmonary artery pressure, and the greater the likelihood of paradoxical gas embolism and arterial gas embolism (AGE).



Effects:

- Cerebral DCS:

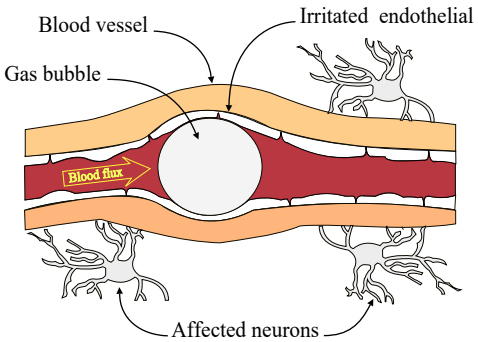
Arterial bubbles are distributed according to buoyancy relative to blood in large vessels and blood flow in small vessels. This explains the preponderance of cerebral symptoms in DCS because the bubbles will distribute preferentially to the cerebral circulation via the cerebral vessels, and multi-vessel pathology is more common.
Arterial gas bubbles may do one of the three things below.

1 - Obstruct the vessel permanently.

2 - Temporarily obstruct the blood vessel and then eventually redistribute through venous circulation.

3 - Pass through the vessel directly into venous circulation.

During their passage, the arterial emboli damage the vascular endothelium, setting in place a neutrophil-mediated inflammatory cascade that opens up the blood-brain barrier, resulting in localized decreased cerebral blood flow and “haemo-concentration” *(increased concentration of blood cells)*. This inflammatory reaction is thought responsible for cerebral DCS symptoms, such as generalized malaise, headache, lethargy, fever, and vague aches and pains.

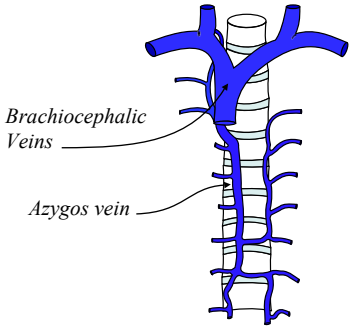


Cerebral DCS is thought largely to occur as a result of this embolic form of bubble injury; however, other pathological possibilities for cerebral DCS include myelin sheath damage and “autochthonous bubbles,” also called “tissue bubbles”, which are gas bubbles evolved from the nitrogen dissolved in white matter. These are also multi-focal and predominantly affect the white matter.
Pathology may also be induced by other emboli (lipid, platelet, etc.), while aggravated damage may result from raised intracranial pressure, and coagulopathies,

- Spinal cord DCS:

Spinal cord DCS is not thought to be principally embolic, and possible mechanisms for this condition include “venous infarction” of the cord, “autochthonous bubble” formation, and “embolic disease”.
The spinal cord changes occur predominantly in the white matter. They are most often observed in the mid-thoracic, upper lumbar, and lower cervical areas, with the lateral, posterior, and anterior columns suffering in that order. Often, there is sparing of some long sensory tracts.

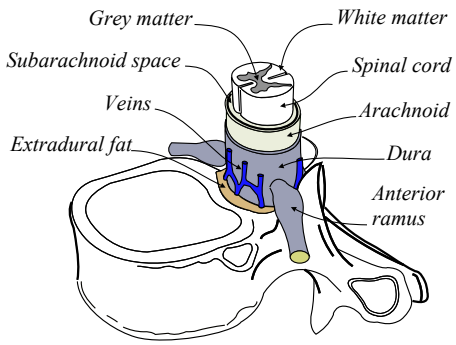
Venous infarction of the cord:
This theory proposes that when the Venous Gas Embolism blocks the pulmonary arterioles, there is a rise in intrathoracic pressure (pulmonary hypertension), which interferes with the venous drainage from the spinal cord through back pressure in the anastomoses *(connections between blood vessels)* of the spinovertebral-azygos system *(vein running up the right side of the thoracic vertebral column.)*. This interference with venous drainage causes engorgement and thrombosis of the spinal cord with infarction in the comparatively poorly vascularized areas. This theory explains why spinal DCS is more common than cerebral DCS, despite the much greater mass and blood supply (and thus emboli) to the brain.



Autochthonous Bubbles:
Autochthonous or tissue bubbles, which are the gas bubbles evolved from the nitrogen or helium dissolved in the white matter of the cord, may arise because the white matter has a high lipid content, which absorbs more gas than the grey matter, which is relatively spared.
Some scientists suggest that autochthonous bubbles may result in spinal cord dysfunction by:

- Destruction of the axons around the growing bubble;
- Stretching and compression of axons around the growing bubble; A biochemical interaction between blood and bubble *(activation of the inflammatory cascade)*.
- Hemorrhages into the tissue secondary to damage to the micro-vasculature, persisting after the resolution of the bubble.

The spinal cord is a “soft tissue”, but with limited compliance because of its confinement by the vertebrae and Durra. If the canal volume is increased by 10 percent or more, the slack is taken up, and the pressure within the cord escalates rapidly. This can develop by either the engorgement of blood or the production of gas. Once the slack has been taken up, the escalating pressure could compress the venous system. This mechanism explains how spinal DCS cases can respond to recompression *(reducing bubble size and cord pressure)* but deteriorate with ascent *(expanding bubble size and cord pressure)*.
The autochthonous bubble theory, therefore, can account for rapid-onset spinal DCS, but the autochthonous bubble theory for spinal cord disease also has its critics:

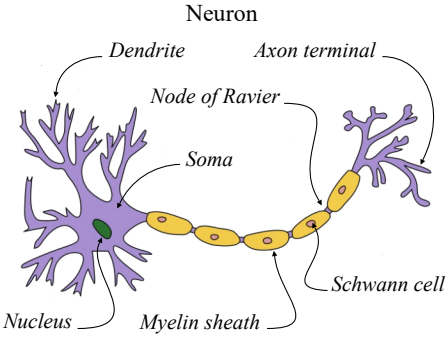


- Tissue bubbles have rarely been demonstrated pathological.
- This theory does not account for spinal cord disease developing hours after surfacing from the dive.
- For bubbles to form, there must be a degree of supersaturation. The time frame for spinal cord bubbles to form will be limited to minutes rather than hours; thus, for these bubbles to remain silent for some hours before symptom onset suggests that another mechanism such as activation of the inflammatory cascade may be involved.

Gas Emboli:
Embolic diseases are considered less frequent causes of spinal cord DCS since AGE resulting in ischemia *(restriction in blood supply to tissues)* is more likely to affect the more vascular grey matter of the cord rather than the white.
Also, the spinal cord is relatively poorly perfused compared to the brain, and the redistribution of Venous Gas Embolism (VGE) to the spinal cord will take a finite time. Because of this delay, scientists suggest that VGE cannot account for the rapidly progressive spinal cord disease observed in some divers soon after surfacing from a dive. A single mechanism cannot completely explain the varying forms of spinal cord DCS. Most likely, a combination of mechanisms coexists to explain the observed clinical condition.

- Musculoskeletal DCS:

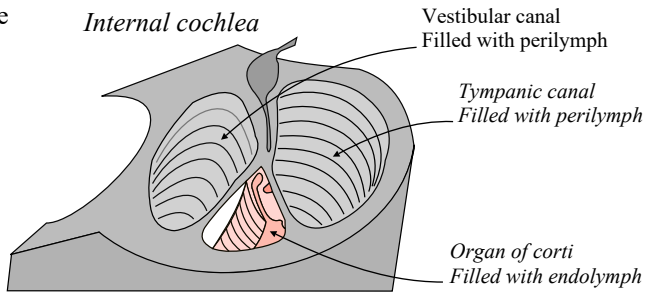
Musculoskeletal symptoms are common presentations of DCS, but the pathology is still not well understood.
Specialists consider that radiological evidence of gas in joint spaces, periarticular areas, facial planes, and tendon sheaths is occasionally seen, but this is not necessarily the causative lesion because some gas in joint space is not usually painful.
Extravascular bubbles in the sub-periosteal area, tendons, ligaments, joint capsule, fascia, and muscles are thought to cause pain (see joint drawing in "skeletal system"). These tissues are tight, and a bubble's development is likely to distort and stretch the tissue and/or its nerve supply. Bubbles in the



articular vascular supply have been proposed but are unlikely as recurrences tend to be in the same site.
Bubbles in the myelin sheaths (see drawing) of peripheral nerves and referred neurological pain have also been incriminated and verified in some cases. Some reports also suggest that bubble formation in the muscle might provoke pain via associated sensory fibres.

- Inner-ear DCS:

The precise mechanism by which inert gas bubbles injure the inner ear is uncertain.
The mechanism may involve the formation of bubbles in the perilymph *(extra-cellular fluid located within the cochlea)*, endolymph *(fluid contained in the membranous labyrinth)*, or associated blood vessels.
Also, scientists reported the observation of fractures in the semicircular canal walls following severe decompression stress in animals, suggesting that bubbles forming in restricted spaces may generate immense destructive forces.



It is notable that “pure” inner ear DCS is uncommon following air diving but became well recognized with deep diving using oxygen-helium mixtures.
This has been attributed to the expansion of silent vestibulocochlear helium bubbles by inward diffusion of nitrogen following gas switching from oxygen-helium mixes to air during decompression: The inner ear can receive helium from its own blood supply and from gas that passes across the round and oval windows bordering the middle-ear space. Thus, the perilymph can become saturated with this gas and rapidly reach a steady-state of gas content. The bubbling that develops during decompression may then disrupt the delicate inner-ear structures.

- Peripheral nerve DCS:

Autochthonous bubble formation in the myelin of peripheral nerves will result in patchy sensory damage or motor impairment, predominantly involving the limbs. Usual symptoms are tingling, tickling, prickling, pricking, numbness, and weakness. Pain may be related to a major plexus and maybe long-lasting. The differentiation between peripheral nerve and an incomplete spinal lesion is important because the prognosis is less worrisome if the clinical symptoms are due only to peripheral nerve involvement.

- Cardiopulmonary DCS:

The first micro-vessels encountered by venous bubbles are the pulmonary capillaries. It has been demonstrated that bubbles generated by decompression or directly infused into the venous circulation become trapped there. The time course for subsequent bubble resolution by diffusion into the alveoli is inversely proportional to embolic gas volume but can be accelerated by oxygen breathing. It is possible to establish a state in which the rate of venous gas infusion is equalled by its clearance by the lungs.
The lungs can trap and excrete venous bubbles. However, the pulmonary bubble filter may be overwhelmed by excessive venous bubbling.
Factors other than the degree of bubbling have also been identified as promoting, or being associated with, bubble redistribution through the pulmonary capillary bed. These factors include:

- Elevation of pulmonary artery pressure
- Decrease in mean systemic arterial pressure
- Recompression
- Administration of “aminophylline” *(used in the treatment of asthma or chronic obstructive pulmonary decease)*
- Pulmonary oxygen toxicity

The obstruction of pulmonary vessels by bubbles may be accompanied by:

- Damage to the endothelium.
- Accumulation of leukocytes.
- Release of “thromboxanes” *(synthesized by platelets and is an inducer of platelet aggregation and platelet release functions and is a vasoconstrictor)*
- Release of “leukotrienes” *(molecules produced in response to allergen exposure, and contributing to allergy symptoms)*.
- Damage to the blood-lung barrier
- Release of vaso-active substances.

There may be hypoxemia *(deficient oxygenation of the blood)* due to either a ventilation-perfusion mismatch or pulmonary oedema generated by elevated transcapillary pressure and plasma leakage through damaged or inflamed endothelium. Ultimately, there may be cardiac decompensation, respiratory arrest, and death.

To complete this point, some doctors thought that AGE might be triggered when bubbles enter the coronary arteries, especially if the diver is horizontal. But, venous bubbles may also cross an interatrial shunt, such as a patent foramen ovale (PFO). In asymptomatic persons, flow across a PFO, if any, is usually from left to right.
There is evidence that such shunting may be important in human DCS:
In recent studies, hyperbaric doctors found shunts in 52.0% of 100 divers with neurological DCS, compared with 12.2% of 123 diver controls without DCS. In a subgroup of 38 divers with spinal DCS, 26 (68.4%) had medium to large PFO

shunts. The same authors also found a strong association between cutaneous DCS and Patent Foramen Ovale (PFO). They showed that 47 of 61 divers with cutaneous DCS had a PFO, compared with 34 of 123 divers who had never suffered DCS. Using magnetic resonance imaging, another team detected multiple asymptomatic brain lesions only in those divers. With a large PFO. thus data suggest a relationship between serious DCS and a large PFO .

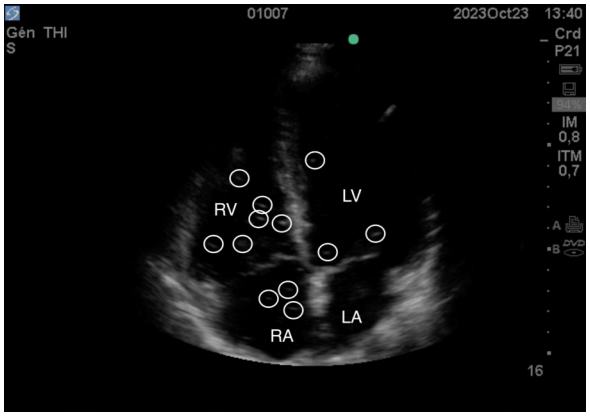
Bubble detection and interpretation:

- Bubble detection:

From the early days of diving table studies until the last quarter of the 20th century, the only way to evaluate a decompression model was by the visible symptoms of decompression sickness. As a result, animal testing was widely used to verify decompression models prior to final human testing in dedicated facilities and at sea.
However, advances in the electronics industry have resulted in new medical equipment, mostly based on ultrasonic detection techniques, which, since the mid-1970's, have provided scientists with better means of assessing and predicting bubbling.
Ultrasonic systems have been, and still are, the most widely used. These include the well-known Doppler devices and imaging systems such as echocardiography. However, although ultrasonic technologies are the most widely used for detecting bubbles, it is also important to consider other technologies, such as image processing and capacitive detection. In addition, technologies that do not detect bubbles but rather areas of stress and the health condition of divers during and after the dive, such as "bioimpedance" and "urine-specific gravity", should not be overlooked.
Manufacturers of modern ultrasonic systems today provide affordable and reliable machines, so diving companies should be encouraged to monitor their decompression profiles, create databases, interpret the results, and improve the decompression models by collaborating with table designers.
A list of manufacturers is provided in the sub-section ”Medical Equipment” of the main section “Logistics” of our website (https://diving-rov-specialists.com/medical-equip.htm).

- Interpretation:

For a long time, decompression accidents were expected to be only the result of massive off-gassing and the presence of bubbles in the arterial circulation. However, as mentioned in the previous section, “Bubble formation”, the formation of pathogen bubbles is more complex, and even though decompression accidents are confirmed to be the result of the production of bubbles and are linked to off-gassing, ultrasonic detection has also shown that a large production of bubbles does not always result in detectable decompression sickness.
For instance, in a document titled "Saturation Diving: Physiology and Pathophysiology," Alf O. Brubakk, John A.S. Ross, and Stephen R. Thom state that the U.S. Navy's "Unlimited Excursion" tables have been tested for excursions from 300 to 250 meters of seawater (msw). The results indicated that all the divers had significant amounts of vascular gas bubbles in the carotid artery, the blood vessel that supplies the brain. However, none of the divers exhibited acute clinical symptoms. Such a phenomenon has been confirmed with upward excursions from 300 to 250 msw during an experimental saturation dive, where Doctor Brubakk et al. found arterial bubbles in the carotid arteries of all six participants, not resulting in clinical cases of neurological decompression sickness. However, post-dive examinations indicated a minor cerebral lesion in the diver with the largest number of carotid bubbles.
In another document called *"Ultrasound detection of vascular decompression bubbles: the influence of new technology and considerations on bubble load"*, written by Doctors S Lesley Blogg, Mikael Gennser, Andreas Møllerløkken, and Alf O Brubakk, it is said that historically, the observation or awareness of arterial gas bubbles has always created apprehension, as they may potentially lodge, sludge, and then grow in the arterial blood supply to organs and tissues, particularly the brain and spinal cord. For example, in a paper on Doppler ultrasound for monitoring hemodynamic changes and bubbles, it was noted that a large number of bubbles were found in the aorta and carotid artery of the human subjects, but no signs of serious DCS accompanied them.
These findings are confirmed by the echocardiograph picture below, which shows bubbles in a diver's arterial circulation not presenting symptoms of decompression sickness.



Apical four chamber view taken after a dive:

Note: The apex of the heart is at the top of the picture.

RV = Right ventricle

LV = Left ventricle

RA = Right Atrium

LA = Left Atrium

The bubbles are highlighted by circles

Note that the bubbles in the left ventricle result from a shunt (Abnormal connection of the venous system with the arterial system)

(Courtesy of Doctor Emmanuel Gouin)

Therefore, we can consider the presence of bubbles as an indicator of possible decompression sickness, so their production should be minimized. However, their presence does not automatically result in decompression sickness. These examples also show that the interpretation of results should be done by specialists.

Factors influencing the likelihood of DCS:

Some physiological and environmental factors influencing the blood supply to tissues and, therefore, the speed of gas uptake or release are known to increase the likelihood or severity of DCS:

- Physical activity (exercise):

The physical activities performed at depth are likely to increase the blood supply to the muscular tissues and increase the rate of inert gas absorption. Some specialists indicate that the decompression requirements can be increased by 3, depending on the physical efforts. This is perhaps due to increased cavitation from "tribonucleation" (creation of bubble nuclei through surface friction) or to "turbulence" (flow regime characterized by chaotic and unpredictable property changes). Intense physical activities during or after decompression results in an increase in bubble development speed and the number of bubbles. Thus, it is likely to trigger decompression sickness (DCS)
A mild exercise during decompression, if the diver is in the state of "supersaturation" (state of a solution which contains more dissolved material than it could be dissolved under normal circumstances) and the bubble growth have not incurred, is thought by specialists as increasing the rate of gas elimination, perhaps by increasing tissue perfusion.
Thus, during decompression, periodic walking in the compression chamber is to be encouraged.

- Physical fitness:

Due to its relationship to more efficient muscular use and blood flow, physical fitness seems to be of some protective value to divers.

- Obesity:

Inert gas (N2 or H2) have an affinity to fat. Thus, obesity increases the tissue mass available to absorb more inert gas and may increase DCS in longer-duration dives. Nitrogen is 5.4 times more soluble in fat than in water and non-fatty tissue.

- Environmental temperature:

According to Doctor Edmonds, the temperature may influence DCS by its influence on perfusion (increased temperature producing increased blood flow) and solubility (lowered temperature producing increased gas solubility). The divers exposed to cold at maximum depth may have less tissue perfusion and DCS possibility than divers in hot-water suits.
If the diver becomes mildly hypothermic, eliminating the inert gas is decreased, and DCS is more likely. In some studies, the perfusion rate in muscle was halved, thus doubling the required decompression duration.
The divers who become cold during decompression have a lowered perfusion of tissues during ascent, less gas uptake from the tissues, and more DCS possibility. The opposite occurs in warmer conditions. It is better to be warm during decompression because the nitrogen or He elimination is increased.
After the dive, exposure to sudden excessive heat (such as with a hot shower) produces increased superficial blood flow and lowered solubility of gas, resulting in a bolus of nitrogen or He being mobilized, with gas-phase separation and delivery to the lungs. Both skin and generalized DCS manifestations could develop.

- Carbon dioxide retention:

Increased carbon dioxide pressures from the effects of pressure, exercise, or breathing resistance with equipment may cause increased perfusion during the dive, with increased nitrogen loading. It is also a factor with inadequate ventilation in chambers and helmets.

- Dehydration:

While diving, dehydration caused by the water loss from respiration, physical activity, external temperature, and the non-replacement of fluid reduce the perfusion of tissues and thus the elimination of inert gas. This is, for example, proved by doctors Andreas Fahlman and David M. Dromsky in a paper called *"Dehydration Effects on the Risk of Severe Decompression Sickness in a Swine Model"*.

- Age:

Increasing age increases DCS incidence, possibly due to impaired perfusion or already damaged vessels being more susceptible to other flow interferences. Abnormalities and degeneration within joint surfaces also increase the likelihood of tribonucleation in the aged.

- Alcohol and drugs:

Alcohol and drugs are strictly forbidden on worksites and influences dehydration, vasodilatation, heat loss. Thus aggravating DCS. Methods for managing alcohol and drug abuse are discussed in the "Diving Study CCO Ltd #3".

- Physical injury:

Physical injury, such as a sprained joint or a previous decompression sickness (DCS), may predispose to DCS due to scarring and alterations in local tissue perfusion. Thus, some clinicians are concerned that spinal operations may be associated with spinal DCS risk.

- Position when decompressing:

Decompression staging in a horizontal position in the chamber results in an increased gas elimination rate compared with seated positions. Small activity is also preferable to a static position without any movement.

- Dive profiles:

Square dives, which involve sending the divers to a single depth and recovering them to the surface at the end of the planned bottom time, are the most common. However, the diver often has to perform multilevel operations. In this case, the following guidelines should be considered:

In the procedures of use of the MT92 tables, it is stated the following: *"In the case where work requires the diver to operate at different levels, it will be necessary to organize the dive so that work commences at the deepest level and repeated rises are avoided"*.

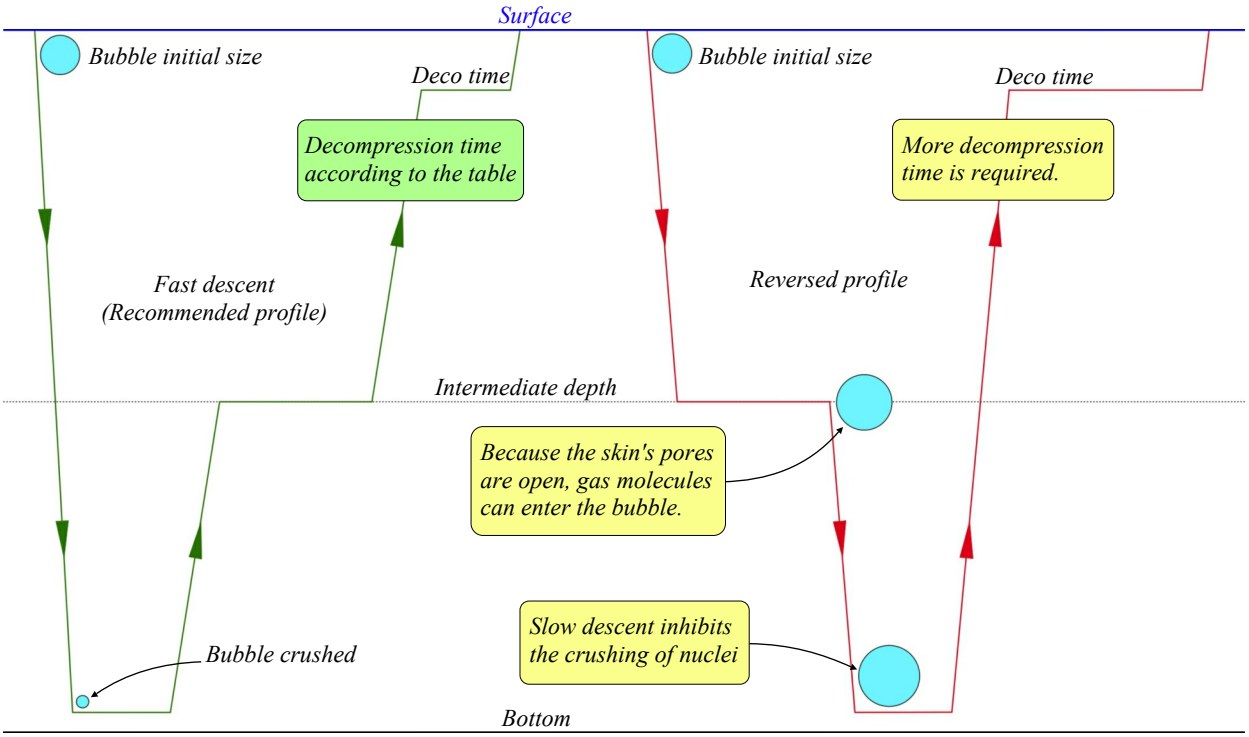
There are several reasons for starting the work at the deepest depth, the most obvious being that starting from the deepest depth allows decompression to begin during the ascent to the next level, where the gas absorption process will be more reduced than at depth. All multilevel procedures are based on this profile. For example, in a presentation titled *"Short and Repetitive Decompression in Air Diving Procedure: The Commercial Diving Experience"*, JP. Imbert, X. Fructus, and S. Montbarbon explain that they introduced a method for determining decompression after a split level based on the equation: P1 . t1 + P2 . t2 <= Pe . (t1+ t2)

Where:

- "P1" is the pressure at the 1st level,
- "t1" is the time at the 1st level,
- "P2" is the pressure at the 2nd level,
- "t2" is the time at the 2nd level,
- "Pe" is the equivalent depth

They also say that using an exponential tissue model, this holds true when the first level is the deeper one and allows selection of a decompression for a two-level dive using an equivalent depth (Pe), computed from the above equation. It must be noted that the DCIEM manual shows the same principle of calculation.

In addition to the elements above, a theory based on the Varying Permeability Model (VPM) from Doctors Yount et al., on which the decompression software "V-Planner" is built, suggests that a gas bubble is surrounded by a "skin" that comprises pores through which the gas molecules can pass. This skin becomes impermeable if the bubble is compressed by external pressure to a size where its pores are closed. The pressure at which this phenomenon occurs varies according to the bubble characteristics. During the dive, as long as the bubble is not at the size where its skin pores are closed, gas molecules continue to enter. In other words, and to simplify, given that the bubble has an internal pressure, the dissolved gases in each tissue compartment will move across the skin into the bubble and increase its size as long as the tissue tension of the compartment exceeds its internal pressure. As a result, if the diver starts working at an intermediate depth and then finishes the dive at the planned maximum depth, more gas is absorbed by the existing bubbles and nucleus than if the diver goes first to the maximum planned depth, which is a pressure closer to those where the mentioned bubbles and nucleus are expected to become impermeable. In a document called *"Bubble Formation in Supersaturated Gelatin: A Further Investigation of Gas Cavitation Nuclei"*. Doctors Yount and Yeung concluded that a slow descent inhibits the crushing of nuclei and thus leads to enhanced bubble growth compared to rapid compressions. Therefore, the faster the diver descends, the less time the tissue tension in the faster compartments adjusts to ambient pressure. In another document called *"Implications of the Varying Permeability Model for Reverse Dive Profiles"*, Doctors D. Yount and E. Baker stated that the crushing of the bubble should be maximized to the greatest extent possible for any dive, whether a single dive or a repetitive exposure. They also concluded that a reverse dive profile, such as a shallow dive followed by a deeper one, can likely result in a substantial volume of released gas and many bubbles.



However, while keeping in mind what is said above, we must take into account the particular diving practices in force in fish farms and other particular industries, where yoyo dive profiles are commonly practiced and have been considered safe by renowned scientists if undertaken under certain conditions.
In a document entitled *"Tasmania's Aquaculture Industry: A Ten-Year Review of Improved Diving Safety"*, which outlines the improvement in diving procedures in Tasmanian fish farms since the inception of this industry in 1986, when no safety practices were in place, resulting in numerous diving accidents, Drs. David Smart, Sean Rubidge, Peter McCartney, & Corry Van Den Broek explain that empirical procedures based on the documents *"Analysis of the risk of decompression illness to yoyo diving using the USN probabilistic decompression model"* by Doctors Parker et al. (US Navy) and *"Decompression sickness arising from diving at fish farms. Final report"* by Doctor Shield et al. were implemented to avoid the numerous cases of decompression sickness from yoyo diving recorded in the early days of the industry. These procedures limited the number of ascents during bounce dives, resulting in dive profiles and practices similar to those shown in the table and explanations below.

Examples	***Depth in metres***	***Bottom time in minutes***	***Maximum bounces***
Example 1	*10*		*8*
	>10		*6*
Example 2	*6*	*150*	*8*
	12	*120*	*8*
	15	*60*	*4*
	18	*40*	*2*
	21	*30*	*1*
	30	*15*	*1*
Example 3	*9 - 12*	*150*	*8*
	12 - 15	*75*	*6*
	15 - 18	*50*	*4*
Example 4	*0 - 6*	*240*	*10*
	7 - 9	*180*	*8*
	10 - 12	*110*	*8*
	13 - 15	*75*	*4*
	16 - 18	*50*	*4*
	19 - 21	*35*	*2*

The practices enforced were the following:

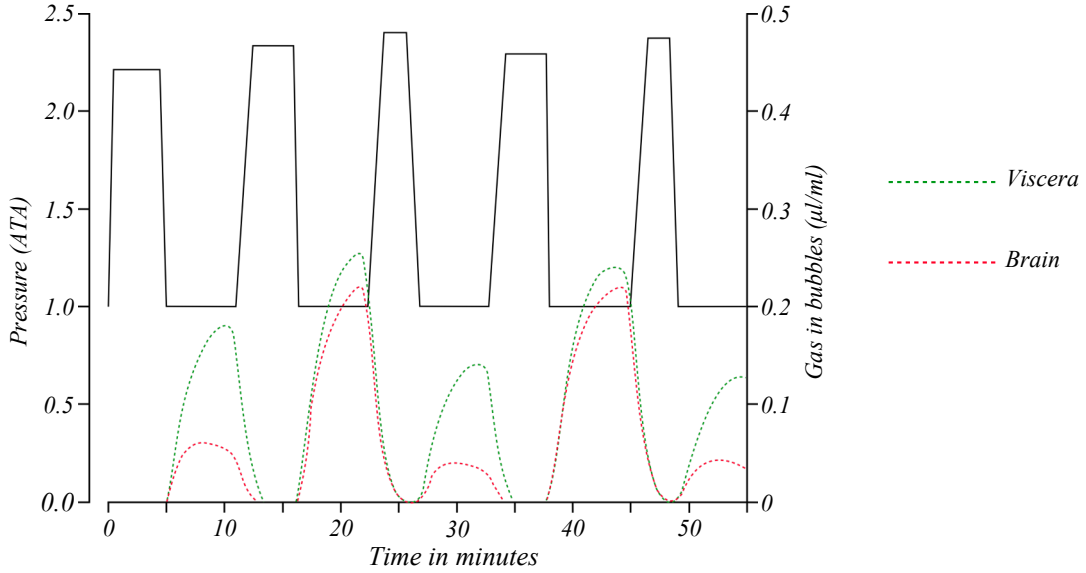
- The limitation of the number of ascents to 10.
- The deepest dive to be performed first.
- The addition of 10 minutes to the bottom time for every bounce.
- The implementation of slow ascents with, in addition, hand over hand for the last 3 meters.
- The addition of a safety stop at 3 meters for 5 minutes after diving deeper than 10 meters or bounce diving.
- The reduction of dive times in case of hard work.
- The recommendation to use DCIEM tables.

These procedures resulted in a significant reduction in decompression accidents, as shown in the table below.

Periods	***Number of divers***	***Number of dives per year***	***Cases of DCI during the period***	***DCI rate per 100 divers/year***	***DCI rate per 10 000 dives***
1988 - 1990	50	2100	11	11	26.19
1992 - 1994	86	5600	4	2.32	3.57
1996 - 1998	81	8768	1	0.62	0.57

We can, therefore, assume that these empirical procedures have been beneficial to a population of divers initially accustomed to practicing multiple ascents and descents without any rules during their dives.

In 2004, Dr. Valerie Flook published the results of a series of studies undertaken for the UK HSE, titled *"Yo-yo Diving and the Risk of Decompression Illness"*, classified as “HSE Safety Report #214”.
Using Doppler and a mathematical model that simulated inert gas uptake and bubble formation in the various tissues of the body, she investigated several series of no-decompression dive profiles as practiced by fish farm divers (similar to the one in the drawing below), taking into account gas uptake into three categories of tissues: the viscera, brain, and muscles, but focusing more on the viscera and the brain that appeared to be the more affected by this type of dive profile.



Doctor Valerie Flook did not publish specific guidelines for the dive profiles considered. However, she emitted the following conclusions:

- The dives considered in the report are relatively shallow depths, and it may be incorrect to extrapolate these conclusions for diving at greater depths.
- No-decompression (No-D) diving puts the brain at particular risk. The rates of ascent, though generally slow enough to protect the faster tissues, are not slow enough to protect the brain. In general, the accepted No-D tables do not protect the brain.
- Tables drawn up for repeat diving do not take into account the way in which the presence of bubbles slows down the removal of gas from the body.
- Yo-yo diving of the type traditionally practised in fish farm diving can be very safe and that dividing the total bottom time into several shorter dives alternating with a surface interval is less of a risk than diving the envelope.

Another document on Tasmanian fish farm decompression procedures, which can be seen as a continuation of the one published in 1999, has been published in 2014 under the title *"Field Validation of Tasmania's Aquaculture Industry Bounce-Diving Schedules Using Doppler Analysis of Decompression Stress"* by Drs. David R. Smart, Corry Van den Broek, Ron Nishi, P. David Cooper, and David Eastman. It is worth noting that Drs. David R. Smart and Corry Van den Broek were among the authors of the previous report published in 1999, and Dr. Ron Nishi is well known for his work on the use of Doppler technology in decompression studies and his contribution to the creation of the DCIEM tables.
This study was conducted over six years using Doppler ultrasound to assess subclinical decompression stress. Monitoring procedures, such as comparing bubble grades (See the note * below) with variables like the number of bounces or the percentage of DCIEM time limits, and correlation procedures were used to scientifically evaluate the results. Analyses were also carried out to assess the factors identified in the pre-dive questionnaires and the dive-related factors affecting bubble grades in this population of divers.
*Note: Kisman-Masurel bubble grades range from 0 to 4, where 0 = no bubbles detected, 1 = occasional bubbles, 2/low-stress = frequent bubbles, 3 = numerous bubbles, and 4 = continuous bubbles.
100 bounce-dive series and a minimum of 20-25 bounce-dive series for each depth range were logged, allowing data collection consistent with DCIEM methods over the four most commonly dived profiles: 10-12 msw, 13-15 msw, 16-18 msw, and 19-21 msw. However, it was not logistically possible to assess every depth range. Also, it was considered important that the dives evaluated produced sufficient decompression stress to provide valid guidance for the development of the tables.
Based on these assessments, it was determined that ninety-seven percent of the dive profiles evaluated were below or equal to grade 2 (low stress). While this provided evidence of acceptable risk, it also led to difficulties with any multivariate analysis of the causes of decompression stress, as very few dives were sufficiently stressful as defined by Kisman-Masurel bubble grades (> grade 2). The only factors associated with higher decompression stress were time spent in the water as a percentage of the DCIEM table limit and ascent rates.
As a result of the evaluation of the procedures in place, the authors considered that there was sufficient off-gassing during the surface intervals so that the "effective" bottom time was less than or equal to the DCIEM limit, even though the sum of the actual times spent on the bottom may have exceeded the DCIEM limit. Therefore, the off-gassing between each

bounce dive reduced the nitrogen load in the body. In addition, the criterion of restricting in-water time to less than the DCIEM limit added conservatism because this time includes all ascent times plus the 3-minute decompression stop on the last bounce. With seven bounces, the time spent at depth was 10 minutes less than the DCIEM limit. With 12 bounces, the reduction was 15 minutes. Because bottom times included surface intervals with many bounces, the DCIEM limit for bottom time could be exceeded, but the in-water time could be less than the DCIEM limit. Hence, it was considered that less depth-time exposure may have offset the multiple decompressions. Also, the decompression stop may have independently reduced the risk of decompression stress, although this was not assessed in the study.

To conclude on the above, the authors of this study say that these factors explain the low decompression stress observed in their data. Bounce diving has reduced the 'area under the curve' compared with a square dive profile. Hence, there would be less nitrogen uptake during the ascent, surface interval, and descent phases of each surface bounce compared to staying at depth. As an example, a diver conducting bounce diving with five returns to the surface for a bottom time of 48 minutes. Compared to the square dive profile, the bounce diver has less depth-time exposure by the equivalent of 20 min at 18 msw (i.e., the diver had 42% less depth-time exposure). An additional factor may have been that there was insufficient time for bubbles to grow until after the last ascent because divers were under pressure again quickly following their brief surface interval. The study also state that these data are consistent with the mathematical modelling of yo-yo diving conducted by Valerie Flook (see before).

This study resulted in the publication of the "Tasmanian Bounce Diving Tables" below:

1. Initial bounce dive series

Depth (metres)	*Number of allowable bounces in dive series*	*In-water † dive time limit (min)*
≤ 9	10*	300‡
10 – 12	10*	150‡
13 – 15	10	75
16 – 18	6	50
19 – 21	4	35
> 21	Use DCIEM repetitive dive tables	

- Ascent rates shall be ≤ 18 metres per minute;
- Surface intervals between bounces shall be < 15 minutes;
- 3-minute decompression stop at 3 metres shall be performed during the last ascent;
- A second bounce dive series is possible after a 2- hour surface interval, provided specific criteria are obeyed (see below).
- Notes: * Bounce numbers based on validated safety of 13 to 15-metre bounce-dive series;
  † In-water time limit defined as: the total time the diver spends in the water, minus the time spent at the surface during surface intervals. It does include the time of the last ascent and the decompression stop.
  ‡ It is recommended bounce-series dive times are less than DCIEM table limits until fully validated.

2. Criteria for two consecutive series of bounce dives
   - Divers are required to be DCIEM Repetitive Factor 1.0 (see aside) at the commencement of the first bounce-dive series.
   - The maximum depth for the first bounce-dive series is no more than 18 metres.
   - The in-water time for the first bounce-dive series is calculated as the time from commencing first descent to the time of exiting the water, minus the sum of all time spent on surface intervals.
   - The in-water time includes time spent in the water for the decompression stop.
   - The repetitive group for DCIEM tables is calculated from the first bounce-dive series in-water time, after surfacing (see the method for calculation in Book #3).

A. REPETITIVE FACTORS/SURFACE INTERVALS TABLE

Repet. Group (RG)	Repetitive Factors (RF) for Surface Intervals (SI) in hr:min										
	0:15 → 0:29	0:30 → 0:59	1:00 → 1:29	1:30 → 1:59	2:00 → 2:59	3:00 → 3:59	4:00 → 5:59	6:00 → 8:59	9:00 →11:59	12:00 →14:59	15:00 →18:00
A	1.4	1.2	1.1	1.1	1.1	1.1	1.1	1.1	1.0	1.0	1.0
B	1.5	1.3	1.2	1.2	1.2	1.1	1.1	1.1	1.1	1.0	1.0
C	1.6	1.4	1.3	1.2	1.2	1.2	1.1	1.1	1.1	1.0	1.0
D	1.8	1.5	1.4	1.3	1.3	1.2	1.2	1.1	1.1	1.0	1.0
E	1.9	1.6	1.5	1.4	1.3	1.3	1.2	1.2	1.1	1.1	1.0
F	2.0	1.7	1.6	1.5	1.4	1.3	1.3	1.2	1.1	1.1	1.0
G	-	1.9	1.7	1.6	1.5	1.4	1.3	1.2	1.1	1.1	1.0
H	-	-	1.9	1.7	1.6	1.5	1.4	1.3	1.1	1.1	1.1
I	-	-	2.0	1.8	1.7	1.5	1.4	1.3	1.1	1.1	1.1
J	-	-	-	1.9	1.8	1.6	1.5	1.3	1.2	1.1	1.1
K	-	-	-	2.0	1.9	1.7	1.5	1.3	1.2	1.1	1.1
L	-	-	-	-	2.0	1.7	1.6	1.4	1.2	1.1	1.1
M	-	-	-	-	-	1.8	1.6	1.4	1.2	1.1	1.1
N	-	-	-	-	-	1.9	1.7	1.4	1.2	1.1	1.1
O	-	-	-	-	-	2.0	1.7	1.4	1.2	1.1	1.1

   - A minimum surface interval of 2 hours must occur between the first and second bounce-dive series.
   - The repetitive group is then used to calculate the allowable bottom time for the second bounce-dive series.
   - The maximum depth of the second bounce-dive series shall be no deeper than the maximum depth of the first bounce dive series.
   - The number of allowable bounces in the second bounce dive series shall be restricted to half the number of the first bounce-dive series (maximum of 5 bounces), and with maximum bottom time as defined by the DCIEM repetitive group allowable bottom time.

Note that the Tasmanian Bounce dive table has been adopted by the government of New Zealand in November 2018.

However, although these procedures have been considered safe, the following elements should be taken into account by diving teams involved in activities other than those for which these tables were designed:

- Multiple ascents and descents result in a loss of working time. It is, therefore, more advantageous for teams involved in construction projects to organize a square profile with level changes during the final ascent.
- This procedure is considered valid in Australia, New Zealand, and perhaps Canada because some authors are Canadian, but nothing has been published in other countries. Therefore, it will be judicious to consult the authorities of the country where the diving operations are planned to ensure that it can be implemented.
- This procedure has been validated for the DCIEM table only. Therefore, it should be limited to DCIEM tables only as long as similar validation processes are not implemented for the other tables.
- In a document titled *"Yo-Yo Diving and Risk of Decompression Sickness in Trainee Military Divers"*, doctors Emmanuel Gempp and Christophe Peny indicate that the use of these types of profiles to train military divers has resulted in a significantly higher number of incidents than in the commercial diving industry. Therefore, we must keep in mind that, as mentioned previously in this chapter, the mechanisms that make a bubble pathogenic are not yet fully clarified and seem not only linked to the gas charge.

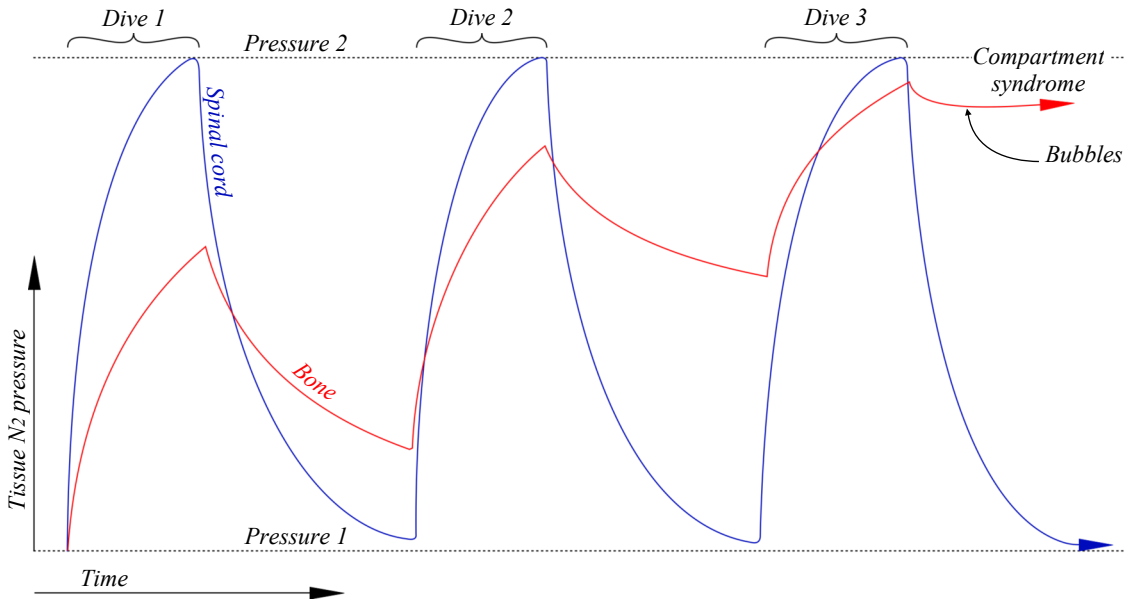
- Repetitive Diving

Repetitive diving involves planning a dive within a period during which the diver has not yet entirely eliminated the residual gas from the previous dive. Specifically, this refers to dives conducted within 18 hours, according to the DCIEM table, and within 12 hours, according to the MT 92 table.

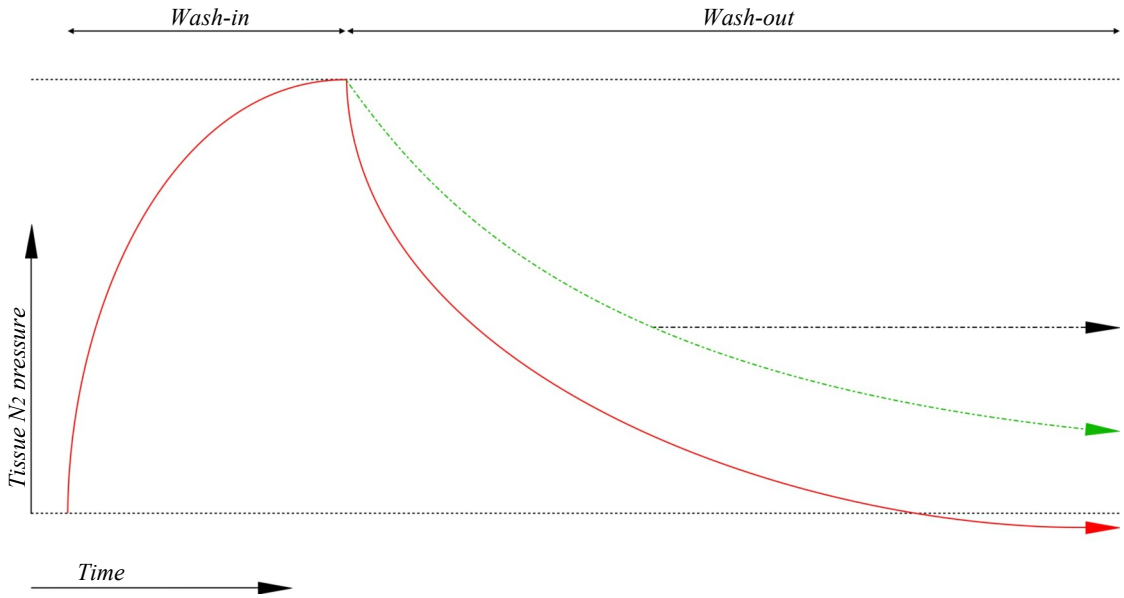
Repetitive diving has long been discussed by divers, with some believing that several repetitive dives can be organized and others believing that such operations should be undertaken with more caution. For example, during the Repetitive Diving Workshop organized by Duke University in March 1991, Max H. Hahn presented a document entitled *"1991 recreational multi-day diving operation survey"*, which indicated that multi-day repetitive dives were common among recreational divers, with more than three dives per day and sometimes five to six dives per day. This document states that the survey found no particular evidence of unusual risk from multi-day repetitive diving. Although the method of investigation made it difficult to have a high level of confidence in its accuracy, this document can be considered an example of practices in force in the recreational diving industry.

During the same workshop, in a presentation entitled "*Dive profiles and adaptation: Pressure profiles target specific tissues for decompression injury"*, Dr. Charles E. Lehner explained that repetitive diving could influence decompression injury based on the fact that nitrogen loading of tissues can be cumulative with repetitive diving, particularly in slow tissues such as fat marrow and cortical bone, resulting in tissues not fully completing their N_2 washout during short surface intervals.

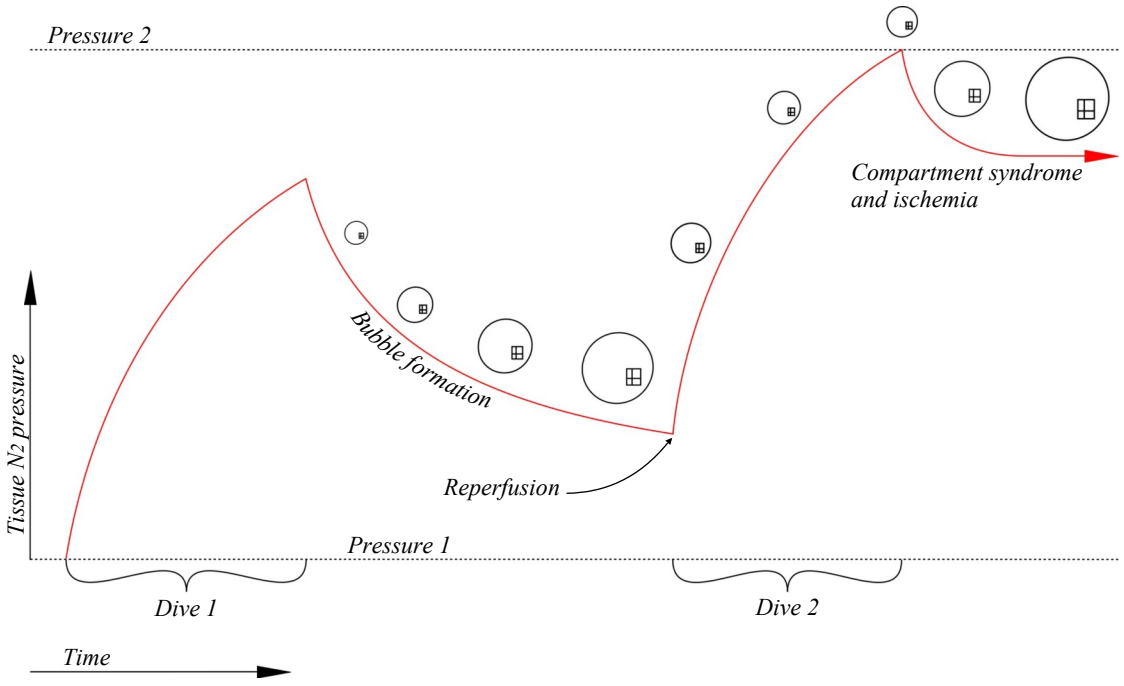
Developing this explanation further, Doctor Lehner said that due to vascular architecture, tissue inert gas washout may be considerably slower than expected from a Haldanian exponential washout model, with a countercurrent exchange of inert gas between parallel but opposing arterial inflow and venous outflow in blood vessels that can extend tissue gas washout times, especially in such anatomically complex tissues as the long bones and spinal cord, reducing the venous outflow in many tissues. Therefore, latent silent bubbles formed after a single hyperbaric exposure may become significant with successive repetitive dives, promoting ischemia and increasing tissue pressure, causing compartment syndrome in tissues susceptible to decompression injury. In addition, Doctor Lehner suggested that repetitive diving may also affect fast tissues, such as the spinal cord, based on the reports of Doctor Paulev (1965), who noted decompression sickness after a series of closely spaced, repetitive breath-hold dives to 15–20 m at intervals of 1–2 minutes in a submarine escape training tank, which is similar to the potentially fatal "taravana" disease of pearl divers. Doctor Lehner believed that, depending on the interval between dives, one tissue may be favoured over another in N_2 loading, which can be illustrated by the scheme below, based on Haldanian wash-out and wash-in relationships in bone (a slow tissue) and spinal cord (a fast tissue). Bone is eventually loaded with enough N_2 that bubbles form on the third decompression, inducing a compartment syndrome that stops bone N_2 washout. Spinal cord N_2 is unaffected.



To continue this explanation, Doctor Lehner said that Haldanian inert gas washin and washout assumptions are especially useful as a starting point. Still, tissue washouts appear overly brisk in the Haldane model. Homer, Weathersby, and Novotny (1990) have demonstrated the importance of countercurrent exchange in slowing tissue washouts. In addition to countercurrent exchange, bubbles may also form and slow tissue washouts. Bubble formation and growth may cause a compartment syndrome that ceases blood flow to the tissue, and the tissue's inert gas washout essentially ceases, as explained in the scheme below.



In complement to the above, Doctor Lehner stated that prompt recompression treatment can reduce the size of the bubbles and initiate the resumption of the inert gas washout process. Initially silent, asymptomatic bubbles may be significant in the development of decompression injury in repetitive scuba diving.
In the schematic description below, gas bubbles, mostly N2, form in tissue upon decompression. Tissue perfusion decreases as tissue bubbles grow upon decompression and increase tissue pressure, so that tissue pressure approaches arteriolar pressure in the tissue. Tissue perfusion then increases as the second dive reaches maximum depth and the tissue's bubbles decrease in size. Additional dissolved N2 washes into the tissue. Upon the second decompression, bubbles already present, which survived recompression in the second dive, again enlarge to cause a compartment syndrome, with high tissue pressure and no tissue blood flow.
The relatively high percentage of spinal cord decompression sickness cases after repetitive diving reported by the “Divers Alert Network (DAN)” may reflect the importance of latent bubbles slowing tissue washouts and increasing the risk of decompression injury to the spinal cord.



Doctor Lehner's conclusions seem confirmed by the presentation *"Short and Repetitive Decompression in Air Diving: The Commercial Diving Experience"* by J.P. Imbert, X. Fructus, and S. Montbarbon. This document explains that during the design of the 1974 COMEX tables, Doctor Fructus attempted to design tables for a second repetitive dive. However, the project was abandoned after a series of severe cases of decompression sickness. As a result, the COMEX 1974 tables only allowed one repetitive dive.

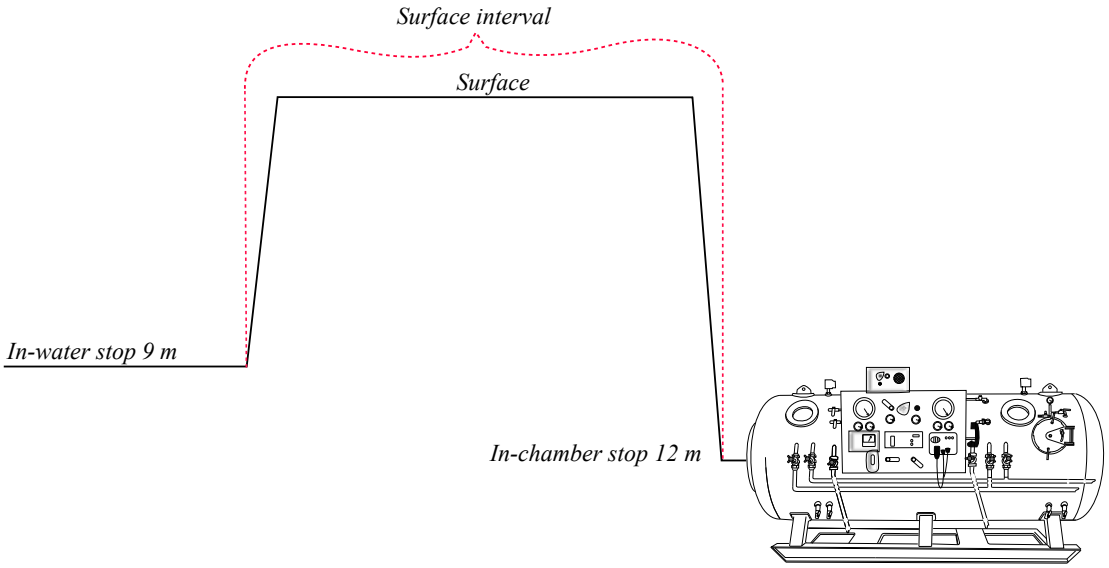
This document also states that the COMEX 74 tables were calculated using the classic "Workman model", where repetitive tables were assumed to be the worst case for calculating the second decompression. Therefore, at the end of the first dive, the tissues' residual nitrogen content should equal their M-values (see the document "Understanding M-values"). Of course, this is never the case, but the assumption allows a repetitive table to be calculated without considering the characteristics of the previous dive. The advantage was that the 1974 COMEX repetitive tables were printed for each surface interval and ready to use without any calculation.
The principle of only one repetitive dive and the residual nitrogen contents supposed to be equal to their M-values at the end of the first dive has been kept for COMEX 86 and MT 92 tables.
In addition to the above, J.P. Imbert, X. Fructus, and S. Montbarbon presented the following data on repetitive dives performed from 1977 to 1986:

Interval *(hours)*	*6:00*	*4:00*	*3:00*	*2:00*	*1:30*	*1:00*	*0:30*	*0*
Number of dives	*2688*	*1371*	*321*	*254*	*110*	*287*	*343*	*140*
Pain-only decompression sickness *(Type 1)*	*4 (0.15%)*	*5 (0.36%)*	*2 (0.62%)*	*1 (0.39%)*	*1 (0.90%)*	*4 (1.39%)*	*1 (0.29%)*	*0 (0%)*
serious decompression sickness *(Type 2)*	*0 (0%)*	*0 (0%)*	*0 (0%)*	*0 (0%)*	*0 (0%)*	*0 (0%)*	*0 (0%)*	*0 (0%)*

They stated that, according to the arterial bubble model, problems in repetitive diving are expected to occur with short surface intervals. However, the data collected in this area is insufficient to draw any definitive conclusions, as only longer surface intervals, particularly the 6-hour surface interval, can support a statistical analysis.
These repetitive tables have only produced pain-only (Type I) decompression sickness, and their rate of incidence does not significantly differ from that of single dives. As a result, it is concluded that this data confirms the importance of the time factor, and it appears that the number of available bubbles capable of passing the lung filter significantly decreases as time passes. It seems that, after 6 hours, the recompression following the beginning of the second dive no longer produces arterial bubbles.
It must be noted that the DCIEM table's calculation method is more classical and allows for several repetitive dives. However, this calculation method results in significant bottom-time restrictions.
It is also important to keep in mind that in the document mentioned, J.P. Imbert, X. Fructus, and S. Montbarbon explained that repetitive diving represented only a small fraction of COMEX diving activities, as the supervisors preferred to organize the job with one long dive per day rather than two short ones. This allowed for the rotation of various functions within the team (diver, tender, stand-by diver), as it must be acknowledged that the repetitive dives were, and still are, considered difficult to fit within a 12-hour shift. It should be noted that IMCA had this rule in its guidelines for a long time before withdrawing it recently, although it was written incorrectly, which resulted in numerous misinterpretations. We can, therefore, consider that the balanced procedure should be to consider the above practices initially implemented by COMEX and also consider that a short dive may have to be performed at the beginning of the shift and that implementing a repeat dive is not to be considered unsafe, particularly passed 6 hours interval.

- Type of dive implemented:

When decompression stops are to be performed, the classical procedures consist of decompressing in the water, wet bell, or closed bell. However, another method, called “surface decompression,” consists of performing an in-water stop at 9 m/30 ft, then recovering the diver to the surface in 1 minute, transferring him to the chamber as soon as possible, and starting oxygen decompression at 12 m (MT 92 & DCIEM) or 15 m (US Navy).



Using only surface decompression procedures instead of in-water decompression, or vice versa, is a debate that has never ended. Proponents of surface decompression argue that the diver is in a safe and fully controlled environment, as he is not confronted with the elements resulting from environmental conditions (cold, current, waves, etc.). Many of them strictly adhere to the US Navy's assertion: "Decompression in the water column is time-consuming, uncomfortable, and inhibits the ability of the support vessel to get underway". On the other hand, some consider that this procedure exposes the diver to decompression stress. For example, in the DCIEM manual, it is said that this procedure intentionally violates the decompression process. As a result, the diver is exposed to a higher level of decompression stress and, therefore, may experience signs or symptoms of decompression sickness during the phase of transfer. Thus, surface decompression procedures result in longer decompression times to compensate for the effects of the decompression process violation. Additionally, many divers highlight that some have had problems clearing their ears (the "Vasalva" manoeuvre) during the subsequent compression into the chamber, increasing decompression sickness risks due to the surface transfer phase. To consider this debate from a scientific point of view, it is essential to take into consideration that in the document *"The safety analysis of French 1974 air decompression tables"*, published in 1986 by J.P. Imbert and M. Bontoux (COMEX), it is said that in-water decompressions tend to produce pain-only (type I) accidents, and surface decompression tends to produce a large proportion of serious decompression sickness (type II) accidents (see in the table below).

	Surface decompression	***In water Air Oxy tables***
Number of dives	*14691*	*10063*
Pain only** (Type 1) **DCS	*39*	*67*
Serious** (Type 2) **DCS	*34*	*1*
Total DCS	*73*	*68*
% DCS	*0.0049*	*0.0068*
% serious** (type 2) / **Total DCS	*0.46585*	*0.0147*

In another article entitled " Decompression safety", published in 1993, J.P. Imbert (COMEX) said:
The comparison of the type I DCS occurrences does not allow differentiating between the two techniques of decompression. However, the comparison of the type II DCS occurrences shows that their incidence becomes significantly much higher with the surface decompression than with in-water decompression (see below)

Exposures	***Prt ≤ 25** (Dr T. Shields) **Moderate***		***Prt < 25 ≤ 35** (Dr. Shields) **Standard***		***Prt > 35** (Dr T. Shields) **Severe***	
Method	***In-water deco.***	***Surface O2 deco.***	***In-water deco.***	***Surface O2 deco.***	***In-water deco.***	***Surface O2 deco.***
Number of dives	*37551*	*10674*	*22643*	*54230*	*8349*	*9323*
***Number of Pain only** (Type 1) **DCS** & %*	*30* *0.08%*	*4* *0.04%*	*78* *0.34%*	*118* *0.22%*	*77* *0.92%*	*87* *0.93%*
***Number of serious** (Type 2) **DCS** & %*	*5* *0.01%*	*1* *0.01%*	*3* *0.01%*	*74* *0.14%*	*12* *0.14%*	*35* *0.38%*

Based on the above, we can conclude that in-water decompression should be preferred over surface decompression, at least for the tables considered. However, "preferred" does not imply that surface decompression must not be practiced. It should be interpreted that if the environmental conditions are favourable, in-water decompression should be the first choice for the abovementioned reasons. Additionally, note that when implementing "continuous dive" procedures to minimize the time lost in decompression, multiplying the number of baskets allows for performing in-water decompression, as mentioned in the CCO Ltd. diving study #1 (https://diving-rov-specialists.com/index_htm_files/cco-study-1-continuous-diving.pdf). Furthermore, it should be considered that the sentence from the US Navy mentioned above reflects a military point of view, where a static vessel is regarded as a perfect target. This perspective does not apply to individuals engaged in peaceful activities. However, even though conditions are favourable for in-water decompression, surface decompression procedures should be ready for use in case of an emergency that may result in the impossibility of carrying on in-water decompression. Therefore, the selection of the decompression procedure should be risk-assessed and based on scientific considerations rather than on texts interpreted in a biased manner or assumptions based on undocumented beliefs.

- Change of mix:

Breathing different gas mixtures, after air diving, decompressing on air after diving on heliox, or breathing a slow diffusing gas while in a fast diffusing gas environment, may produce local or general pressure gradients which cause bubbles to develop. Also, COMEX medical book says that heliox diving accidents should never be treated with air or nitrox mixtures. This phenomenon, called Isobaric inert gas counterdiffusion, is more detailed in the next point.

- Travelling by flight or through mountains:

Exposure to altitude after diving may provoke DCS by producing or expanding existing bubbles, as the decompression tables are calculated to return the diver to the surface and not to altitude, where the ambient pressure is less than at sea level. For this reason, procedures for transfer to altitude are provided. Regarding this point, both MT 92 and DCIEM tables offer these procedures. However, it is preferable to select the DMAC one, which is more stringent.

DMAC 7 - Table 1: Minimum times before flying at cabin altitude *Diving without decompression illness problems or any symptoms*	***2000 feet (600 m)***	***All other flights***
1.1 - No stop dives. Total time under pressure less than 60 minutes within the last 12 hours	2 hours	18 hours (24 hours)*
1.2 - All other air and nitrox diving, heliox and mixed gas bounce diving *(less than 4 hours under pressure)*	12 hours	24 hours
1.3 - Heliox saturation *(more than 4 hours under pressure)*		
1.4 - Air, nitrox or trimix saturation *(more than 4 hours under pressure)*	24 hours	48 hours

** 18 hour time applies to short flights (less than 3 hours). For longer flights the time is extended to 24 hours*

- Adaptation:

One factor reducing the likelihood of developing DCS is adaptation or acclimatization to the repetitive exposure to increased pressure: It appears that a slight degree of resistance to DCS can develop. Several records have highlighted that decompression sickness is more probable the first week of diving operations. The explanation for this adaptation is the removal of naturally occurring gas nuclei, which are thought to be the support on which the bubble develops. This is the reason why divers and caisson workers are advised to work up to their maximum exposures gradually.

- Pre-dive activity:

Experiments made by doctors Gennser et al. concluded that five weeks of bed-rest significantly increased bubble grades after decompression. The reasons given to explain these results are the following:

- Bedrest conditions are associated to minimal activity and therefore to a minimal metabolism. The consequence is that the initial Static Metabolic Bubbles (SMB) volume in the divers prior to the dive was maximal.
- The lack of exercise reduces vibrations and it is likely that most of the available Active Hydrophobic Spots (AHS) were populated by SMB.
- After a bedrest, the divers started the dive with a high density of SMB with a maximal volume that favored higher grades of detected VGE.

- Many other possible endogenous (serum complement, lipids, smooth muscle activating factors, etc.) and exogenous (smoking, migraine) aggravating factors can be taken into account.

Symptoms and treatments

- Decompression sickness symptoms usually occur shortly following the dive or other pressure exposure. If the controlled decompression during ascent has been shortened or omitted, the diver could be suffering from decompression sickness before reaching the surface. In analyzing several thousand air dives in a database set up by the U.S. Navy for developing decompression models, the time of onset of symptoms after surfacing was 42 percent occurred within 1 hour, 60 percent occurred within 3 hours, 83 percent occurred within 8 hours, 98 percent occurred within 24 hours.
- The usual strategy for treating decompression illness involves reducing bubble size by compression to reduce its aggressive effects and providing oxygen to eliminate the dissolved inert gas. The symptoms are generally divided into two categories to allow the person in charge to decide on the most appropriate treatment procedure
    - Type I decompression sickness (also called pain-only decompression sickness) includes skin symptoms, lymph node swelling, and joint and/or muscle pain and is not life threatening.
    - Type II decompression sickness (also called serious decompression sickness) includes symptoms involving the Central Nervous System (CNS), respiratory system, or circulatory system. Type II decompression sickness may become life threatening.

  Note that there is a wide range of symptoms accompanying the initial episode of decompression sickness. Some of the symptoms or signs will be so pronounced that there will be little doubt as to the cause. Others may be subtle, and some of the more important signs could be overlooked in a cursory examination. Thus, type 1 and type II symptoms may or may not be present simultaneously, and depending on the evolution, the treatment may move from type 1 to type 2.

Symptoms and treatment Type 1 decompression sickness:

- Pain:

- The characteristic pain of Type I decompression sickness usually begins gradually, is slight when first noticed, and may be difficult to localize.
- It may be located in a joint or muscle, increasing in intensity, and usually described as a deep, dull ache.
- The pain may or may not be increased by movement of the affected joint and the limb may be held preferentially in certain positions to reduce the pain intensity (so-called guarding).
- The hallmark of Type I pain is its dull, aching quality and confinement to particular areas. It is always present at rest; it may or may not be made worse with movement.
- The pain may lessen if local pressure is applied manually or with a blood pressure cuff.
- Pain in the abdominal and thoracic areas may be localized to joints between the ribs and spinal column, joints between the ribs and sternum, present a shooting-type pain that radiates from the back around the body (radial or girdle pain), or appear as a vague, aching (visceral) pain. Because it is difficult for non-medical personnel to differentiate between the Type I joint pain and Type 11 radial or visceral pain in the abdominal and thoracic areas, any pain occurring in these regions should be considered by non-medical personnel as arising from spinal cord involvement. In this case, the treatment will be Type II decompression sickness.

- Cutaneous (skin):

- The most common skin manifestation of diving is itching. Itching by itself is generally transient and does not require recompression.
- Faint skin rashes may be present in conjunction with itching. These rashes also are transient and do not require recompression.
- Mottling or marbling of the skin, is a symptom of decompression sickness and should be treated by recompression. This condition starts as intense itching, progresses to redness, and then gives way to a patchy, dark bluish discoloration of the skin. The skin may feel thickened. In some cases, the rash may be raised.

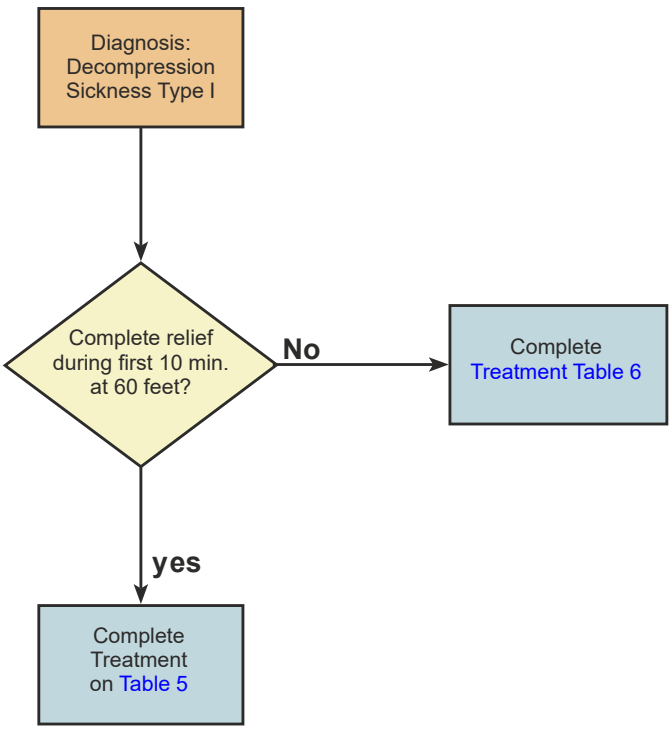
- Lymphatic Symptoms:

- Lymphatic obstruction may occur, creating localized pain in involved lymph nodes and swelling of the tissues drained by these nodes. Recompression will usually provide prompt relief from pain. The swelling, however, may take longer to resolve completely, and may still be present at the completion of treatment

Treatment using US Navy tables:

- Type I decompression sickness is treated using table 5 in accordance with the charts below and on the next page. If a full neurological exam is not completed before initial recompression, the casualty will have to be treated as a Type II symptom.
- Symptoms of musculo-skeletal pain that have shown absolutely no change after the second oxygen breathing period at 60 feet may be due to orthopedic injury rather than decompression sickness. If, after reviewing the patient's history, the Hyperbaric Doctor feels that the pain can be related to specific orthopedic trauma or injury, a Treatment Table 5 may be completed. If a Doctor is not reachable, treatment Table 6 should be used.

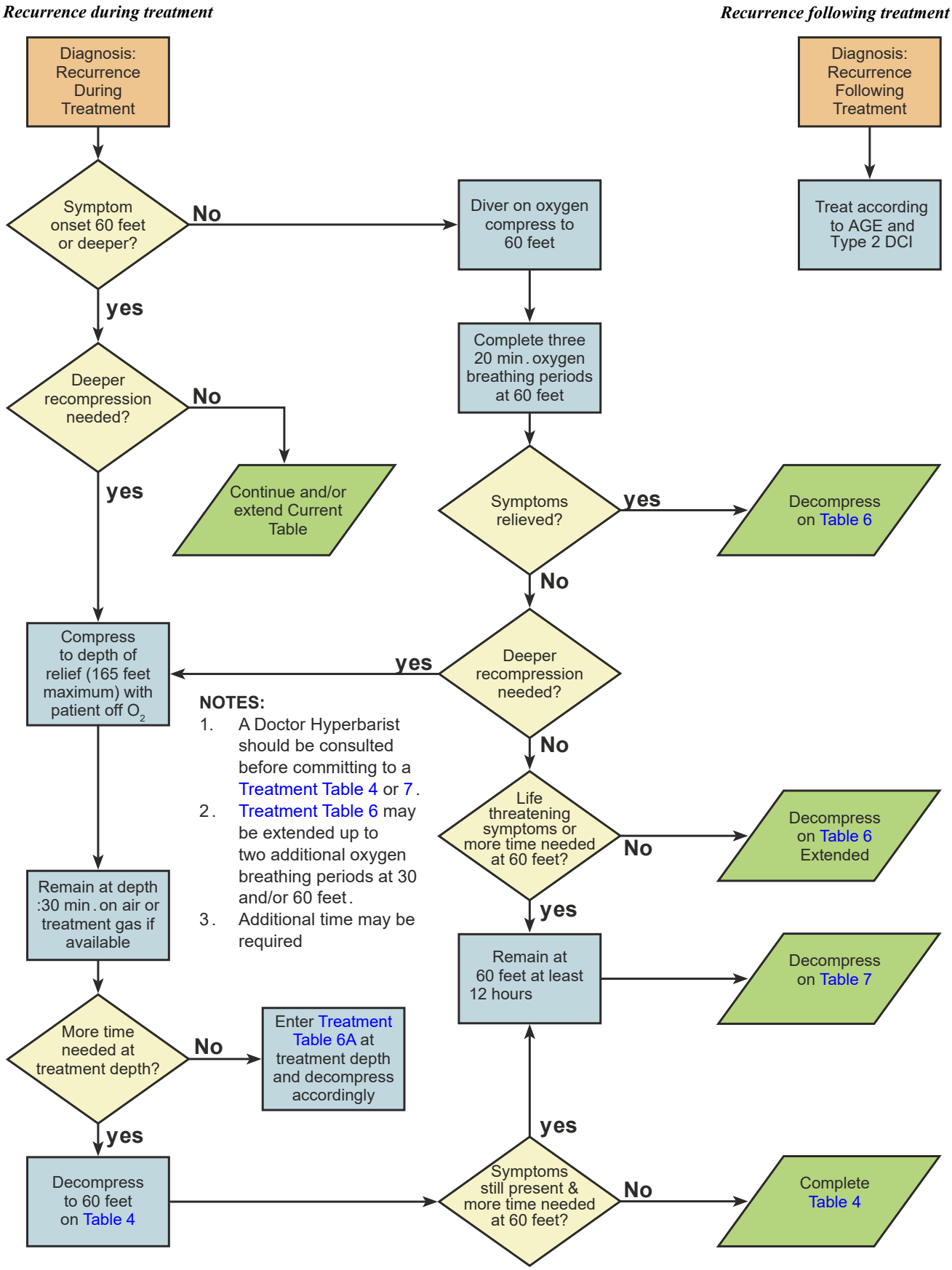
Treatment of type 1 Decompression Sickness



NOTES:

1. If a complete neurological exam was not completed before recompression, treat as a Type II symptom.
2. Treatment Table 6 may be extended up to four additional oxygen-breathing periods, two at 30 feet and/or two at 60 feet.
3. Diving Supervisor may elect to treat on Treatment Table 6.
4. Treatment Table 5 may be extended two oxygen-breathing periods at 30 fsw.

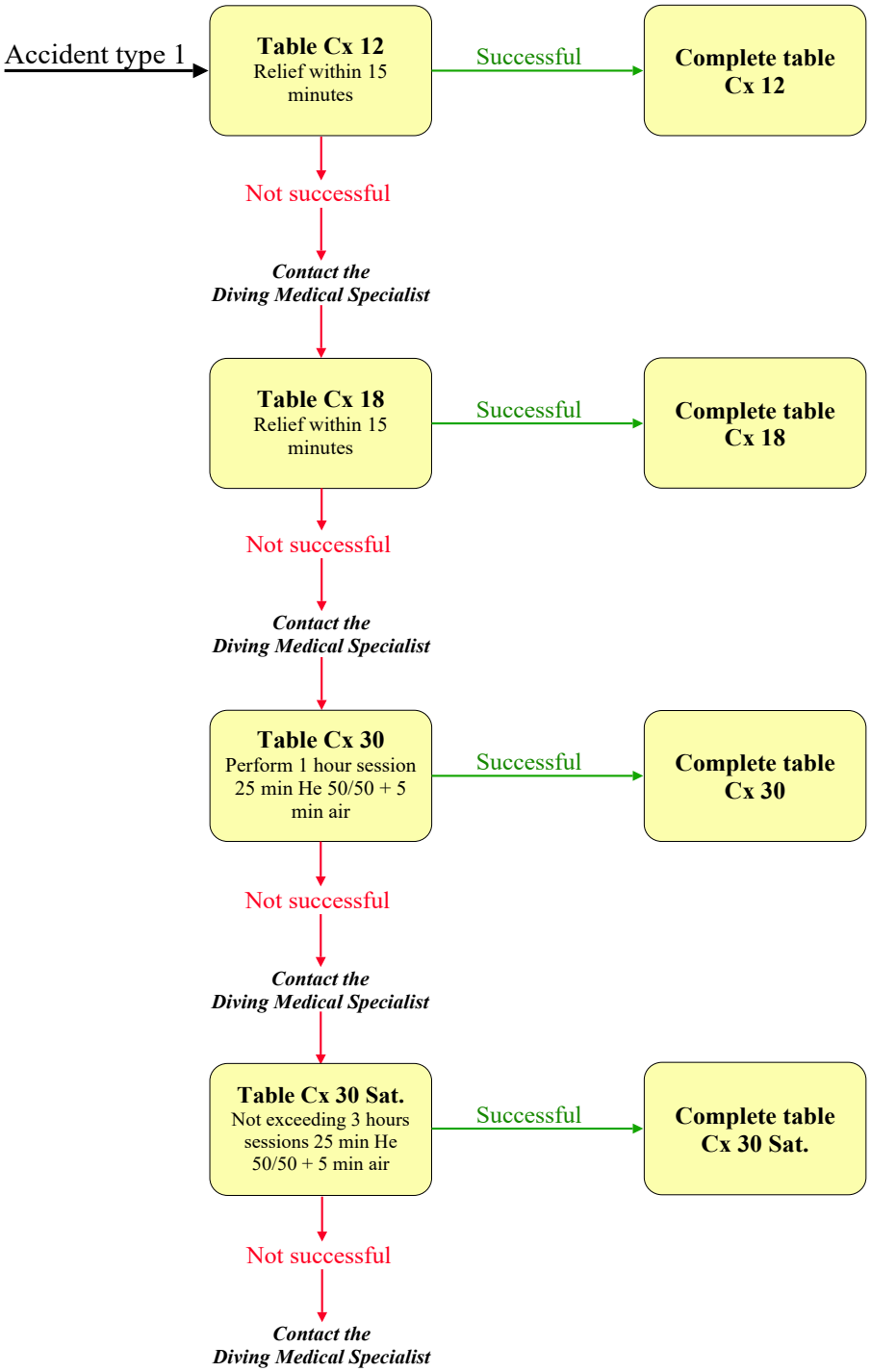
Treatment of Symptom Recurrence



Notes:

- The medical decompression tables are explained and available point “C Medical tables US Navy 6.1”.
- The forms to follow up the treatment and the decompression are available point “D Dive reports forms medical tables US navy rev 6”

Treatment using COMEX tables:



- No relief, recurrence, or worsening of symptoms when using Cx12:
  - No relief within 15 min: Apply Table Cx18
  - Recurrence between 12 m to surface: Apply a second Cx12
  - Worsening of symptoms during treatment: Apply Table Cx30
- No relief, recurrence, or worsening of symptoms when using Cx18:
  - Contact the diving medical specialist
  - If bottom time permits, change the attendant using air or heliox decompression table.
  - No relief within 15 min: Apply Table Cx30
  - Recurrence between 18 m and 12 m: Apply a second Cx18
  - Recurrence between 12 m to surface: Apply Table Cx12
  - Worsening of symptoms during treatment: Apply Table Cx 30
- No relief, recurrence, or worsening of symptoms when using Cx30:
  - Contact the diving medical specialist
  - If bottom time permits, change the attendant using air or heliox decompression table.
  - Recompress to 30 m and apply Cx 30 saturation.

Symptoms and treatment Type 2 decompression sickness:

- In the early stages, symptoms of Type 2 decompression sickness may not be obvious and the stricken diver may consider them inconsequential.
- Many of the symptoms of Type II decompression sickness are the same as those of arterial gas embolism, although the time course is generally different (AGE usually occurs within 10 minutes of surfacing.). Since the initial treatment of these two conditions is the same and since subsequent treatment conditions are based on the response of the patient to treatment, treatment should not be delayed unnecessarily in order to make the diagnosis in severely ill patients (see initial evaluation).
- Type II, or serious symptoms, are divided into neurological and cardio respiratory symptoms. Type I symptoms may or may not be present at the same time.

-1) - Neurological symptoms

- Numbness,
- Tingling
- Decreased sensation to touch
- Tingling, "pins and needles", or "electric sensations"
- Muscle weakness or paralysis
- Mental status or motor performance alterations
- Vertigo, dizziness,
- Ringing in the ears, and hearing loss can also occur. (These symptoms may be difficult to distinguish from a round or oval window rupture.)
- Disturbances of higher brain function may result in personality changes,
- Amnesia, bizarre behavior, lightheadedness, incoordination, and tremors.
- Lower spinal cord involvement can cause disruption of urinary function.
- Some of these signs may be subtle and can be overlooked or dismissed by the stricken diver as being of no

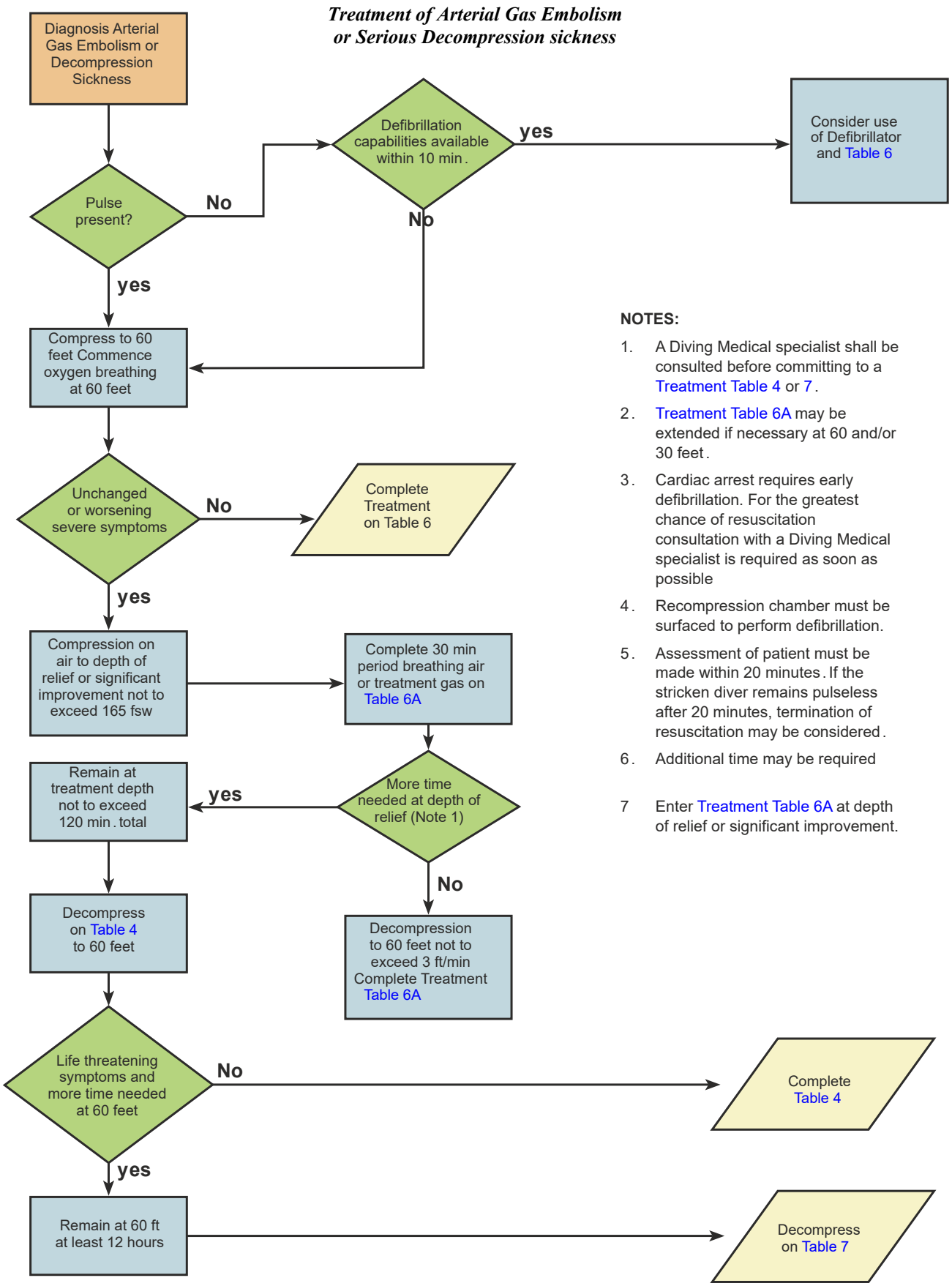
- 2) -Pulmonary symptoms

If profuse intra vascular bubbling (chokes) occurs, symptoms may develop due to congestion of the lung circulation.

- Chest pain aggravated by inspiration, and/or as an irritating cough.
- Increased breathing rate is usually observed.
- Increasing lung congestion may progress to complete circulatory collapse,
- Loss of consciousness, and death if recompression is not instituted immediately.

Treatment using US Navy tables

- Type II decompression sickness is treated with initial compression to 60 fsw using table 6 in accordance with the chart displayed on the next page.
  - If the symptoms improve within the first oxygen breathing period, then treatment is continued on a treatment table 6.
  - If severe symptoms (e.g. paralysis, major weakness, memory loss) are unchanged or worsen within the first 20 minutes at 60 fsw, assess the patient during descent and compress to depth of relief (or significant improvement), not to exceed to 165 fsw. Treat on Treatment Table 6A. To limit recurrence, severe Type II symptoms warrant full extensions at 60 fsw even if symptoms resolve during the first oxygen breathing period.
  - As for the Arterial Gas Embolism, other tables may be used, but the decision to use the tables 4 and 7 have to be taken only by the hyperbaric doctor in charge of the treatment.



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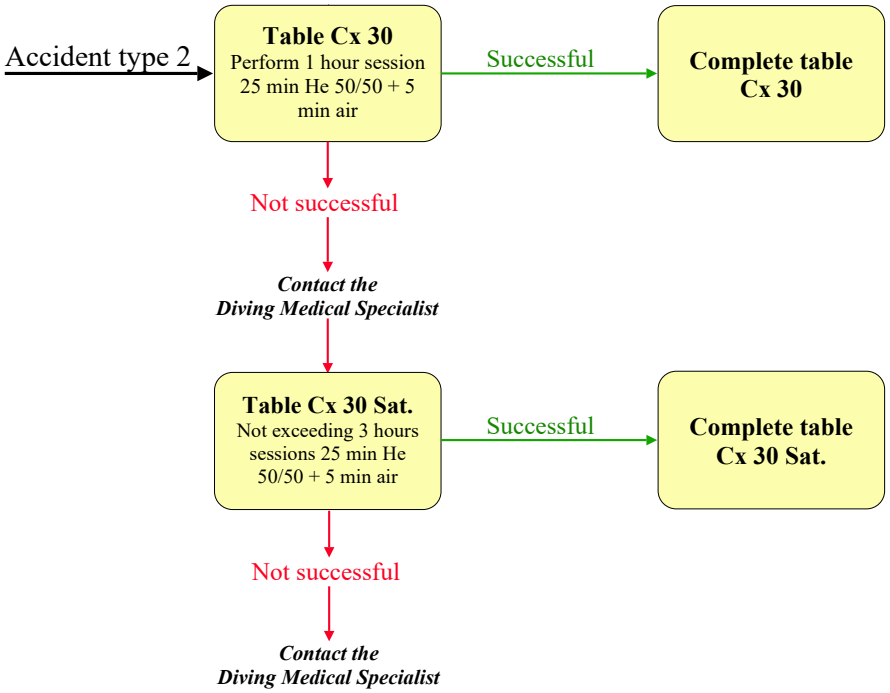
Treatment using COMEX tables



- No relief, recurrence , or worsening of symptoms when using Cx30:
  - Contact the diving medical specialist
  - If bottom time permits, change the attendant using air or heliox decompression table.
  - Recompress to 30 m (if above 30 m), and apply Cx 30 saturation.

Symptomatic omitted decompression *(based on US Navy procedures)*

In case of uncontrolled ascent and visible symptoms, the diver must be compressed immediately in a recompression chamber to 60 fsw/18 m. A rapid assessment of the patient and appropriate treatment has to be organized.

- Treatment Table 5 is not an appropriate treatment in case of symptomatic omitted decompression.

- If the diver surfaced from 50 fsw or shallower, compress to 60 fsw and begin Treatment Table 6 .

- If the diver surfaced from a greater depth compress to 60 fsw or the depth where the symptoms are significantly improved, not to exceed 165 fsw, and begin Treatment Table 6A. Consultation with a Diving Medical specialist should be obtained as soon as possible. For uncontrolled ascent deeper than 165 feet, the diving supervisor may elect to use Treatment Table 8 at the depth of relief, not to exceed 225 fsw.

Precautions when evaluating the case and applying a treatment

The rule is:

- Treat the uncertain case
- Treat the suspicious case
- If the diver seems to have Decompression sickness, he should be treated.

Inadequate treatment

If he is treated promptly, the diver will often be cured even on the wrong table. Where treatment proves inadequate, it may be due to inaccurate evaluation of the diver, lack of aggressive follow-up, or simply a bad break.

Inaccurate evaluation

The usual error is that a diver's report of pain is taken at face value, assumed to be a pain-only bend, and the diver receives an automatic table 5. This is an error for two reasons:

- The diver may actually have pain that is typical of a spinal bit and not know the difference.
- The diver may truly have joint pain but also have symptoms of spinal cord DCS which hasn't been noticed but could be detected with a neuro exam.

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- The diver may actually have pain that is typical of a spinal bit and not know the difference.
- The diver may truly have joint pain but also have symptoms of spinal cord DCS which hasn't been noticed but could be detected with a neuro exam.
- If a diver reports pain, it must be determined whether it is joint pain or spinal cord pain. If it is joint pain, a careful neuro exam must be done to be sure serious symptoms are not present. If a diver reports a neurological symptom, this can quickly be verified, if necessary, then he should be immediately recompressed.

Inadequate follow-up

A treatment table is a one-shot dose of medicine (oxygen under pressure) and, like any medicine, sometimes has to be repeated-good treatment is not necessarily enough treatment. Although long experience has shown that standard treatment tables offer a high probability of cure when used properly, there is never any guarantee of success. Principles of follow-up after apparently successful treatment are:

- A diver should not be considered cured until he bas been cured for 12 hours.
- Following treatment, he should be kept in the chamber area for one hour. During this time, breathing oxygen at 1 ATA for 30-45 minutes will help ensure the success of the treatment. After this hour, he should be instructed to report any recurrence of symptoms or any new symptoms immediately.
- During the 12-hour follow-up, neuro exams should be done upon exiting the chamber and after 1, 6, and 12 hours. Divers with pain-only bends can usually return to diving in 36 hours.
- Divers who were treated for serious symptoms should have neuro exams upon exiting the chamber, in one hour, then every 6 hours as long as they are at the dive site. They should be kept on the dive site for 12 hours observation, perhaps even 24 hours, so that they do not find themselves far from a chamber if there is a recurrence.
- After treatment for serious symptoms, the diver may not return to diving until be is examined and cleared by a qualified doctor.
- The transfer and demobilization of a diver under or after treatment must be decided by a qualified doctor (Reference is DMAC 7). Decompression to altitude after diving always carries a risk for decompression illness, in particular if the diver has any symptoms.

Inappropriate use of US Navy Tables 5 and 6

Many diving contractors are using Table 6 as the minimum treatment for pain-only bends. This has led to misunderstandings about Table 5 and potential problems from using Table 6 in this way.

Table 5:

Proper use of this table requires the following:

- The diver has pain in or around a joint that is not typical of spinal cord pain.
- A careful neuro exam must be normal (no weakness or sensory abnormalities) and there must be no other serious symptoms.
- After recompression, the joint pain must be relieved completely within 10 minutes; otherwise, treat on Table 6. When used properly, US Navy statistics show Table 5 has a success rate of 96 %.

Table 6:

The standard use of this table is for the various serious symptoms listed above. Using proper precautions, it can be extended virtually indefinitely at 60 and/or 30 feet. If it is used as treatment for pain-only bends, beware of the following pitfalls:

The pain-only case must still be diagnosed as outlined above. Using a serious-symptom table does not remove this duty.

- If the pain-only case also has neurological symptoms, these must be found in advance so the table can be extended if they are slow to clear. If the neurological symptoms are missed, the diver treated only for pain may be prematurely returned to diving and be at risk of further DCS.
- Unnecessary use of Table 6 may increase the risk of oxygen toxicity, obligate the chamber for extended periods of time, and interfere with diving operations.

While deliberately giving excess treatment can often be medically and operationally justified, treatment tables should always be used as precisely and rigorously as possible.

The use of heliox in treating decompression illness

DMAC 23 revision 1 says:

There are many ways of treating decompression illness (DCI) at increased pressure. In the past 20 years, much has been published on the use of oxygen and helium/oxygen mixtures at different depths. There is, however, a paucity of carefully designed scientific studies. Most information is available from mathematical models, animal experiments and case reports.

During a therapeutic compression, the use of a different inert gas from that breathed during the dive may facilitate bubble resolution. Gas diffusivity and solubility in blood and tissue is expected to play a complex role in bubble growth and shrinkage. Mathematical models, supported by some animal studies, suggest that breathing a heliox gas mixture during recompression could be beneficial for nitrogen elimination after air dives. In humans, diving to 50 msw, with air or nitrox, almost all cases of DCI can be adequately treated at 2.8 bar (18 msw), where 100% oxygen is both safe and effective. Serious neurological and vestibular DCI with only partial improvements during initial compression at 18 msw on oxygen may benefit from further recompression to 30 msw with heliox 50:50 (Comex therapeutic table 30 – CX30). There have been cases successfully treated on 50:50 heliox (CX30), on the US Navy recompression tables with 80:20 and 60:40 heliox (USN treatment table 6A) instead of air and in heliox saturation. The rationale for deep treatments is strongest when there is a high inert gas supersaturation (e.g. blow-up). Animal studies on cardiopulmonary decompression sickness have failed to demonstrate the advantageous effects of heliox in comparison to air or oxygen, but this kind of DCI does not occur frequently.

When decompression illness occurs during decompression from saturation, divers should be treated with increased partial pressure of oxygen and/or recompression. No industrial standard has been established for such treatment, but repeated cycles of 20-30 min of breathing treatment gas (1.5-2.5 bar pO 2) with recompression 0-30 msw depending on symptom severity is commonly recommended. DCI symptoms appearing after finished saturation decompression can normally be treated using USN treatment table 6 (but the chamber gas may be heliox).

Heliox has been used as a therapeutic breathing gas during recompression for many years predating the introduction of short oxygen tables. The use of helium-oxygen during therapeutic recompression might be theoretically advantageous, however experience with the use of deeper treatment tables with either helium or nitrogen as inert gas in a treatment mixture with oxygen, has not consistently demonstrated an advantage of helium. However, there is growing evidence that helium is biochemically not inert and has biological effects on organs and tissues. In experimental research helium reduces ischemia-reperfusion damage in the brain, which is one of the mechanisms in DCI and therefore heliox mixtures in treatment of DCI could have advantages and enhance treatment results.

At the present time there is insufficient knowledge to recommend the routine use of heliox in the treatment of DCI occurring after air dives or after finished heliox saturation dives. Inert gas shift is not recommended if symptoms of DCI occur during heliox saturation dives and in such cases, the inert gas component of the treatment gas should be helium. DMAC advises that, based on their expertise and practical experience in combination with the type of diving, company medical advisers prepare the treatment plans for DCI. Recompression options may include shallow oxygen or heliox treatment tables. In addition, DMAC recognises the need for further scientific work to validate the treatment algorithms for DCI.

Quick history of heliox medical treatments *(source: Professor Philip B James)*

The dates indicated in this quick history are extracted from the document from professor Philip B James " Milestones in recompression treatment of DCI - Justification for the use of heliox"

1939 - Behnke AR. The use of helium and oxygen mixtures in the salvage of the USS Squalus in 1939: the first use of helium oxygen mixtures in treatment.

1959 - The US Navy Manual for this year states that:-
- a) "Helium-oxygen mixtures (ratio about 80:20) can be used in place of air in all types of treatment and at any depth."
- b) "Use of helium-oxygen is especially desirable in any patient who
  - 1) Has serious symptoms that fail to clear within a short time at 165 feet.
  - 2) Has recurrence or otherwise becomes worse at any stage of treatment.
  - 3) Has any difficulty breathing."

1966 - Royal Navy reports the recompression treatment of a diver who had been decompressed breathing only helium and oxygen mixture from a dive to 185 msw. He was initially recompressed on air and deteriorated dramatically. There was immediate relief of all symptoms when the diver was given helium and oxygen to breathe. Barnard EEP, Elliott DH. Decompression sickness: paradoxical response to recompression therapy. Br Med 1966;2:809-810.

1970 - The first introduction of Tables 5 and 6 in the U.S. Navy Diving Manual was accompanied by a warning of worsening symptoms when breathing oxygen at 2.8 ATA. The advice given which remains the same today is to revert to further compression using air on USN Tables 4. No explanation has ever been offered for this effect but it germane that a period of 20 minute oxygen breathing at 2.8 ATA was chosen because an extension of the time to just 30 minutes is accompanied by a significant risk of a convulsion. Nevertheless, convulsions have been recorded using Table 6.

1974 - The only edition of the U.S. Navy Diving Operations Handbook to be released *(Navships 0994-009-6010 -1974)* endorses the use of helium and oxygen treatment in "Rules for Recompression Treatment" on page 99. "Always consider the use of 80% helium- 20% oxygen in cases of serious symptoms, recurrence of symptoms, or when patient has difficulty breathing." Also in the section "Saturation diving recompression treatment for decompression sickness" it is stated that: "Air may be used for recompression only in extreme emergency."

1974 to 1977 - Experiments to study the feasibility of dives to extreme depths at Duke and Pennsylvania *(Predictive Studies V to 5000 ft)* examined breathing resistance using chamber 'dives'. Divers given mixtures of nitrogen and oxygen surrounded by mixtures of helium and oxygen (in both laboratories) developed gross cutaneous lesions. The same effects were seen with divers breathing neon/oxygen mixtures in a helium/oxygen environment at 1200 fsw at the University of Pennsylvania. Many animal experiments using rabbits and pigs confirmed the effect. Subcutaneous gas leads to fatal venous gas embolism and the effect is due to the fact that helium passes through skin, whereas nitrogen and neon do not.

1978 - The above gas switching experiments led to unfounded fears in the UK that the recompression treatment of air divers with helium oxygen mixtures may be dangerous. A nitrogen/oxygen saturation method was published in the Lancet but has not been adopted.

1979 - The first medical book published by Comex includes Comex 30 (based on a French Navy table) recompressing to 30 msw, (4 ATA) with advice to use either 50/50 nitrox, or 50/50 heliox,

1986 - Comex issued a second Medical Book after Dr. Xavier Fructus accepted that the preference using Comex 30 is to use a 50/50 helium/oxygen mixture. There was no subsequent case in the company records of using Comex 30 in which deterioration occurred on 50/50 heliox. No oxygen convulsion was recorded *(statistics from J-P Imbert – Comex)*. It should be noted that Comex 30 heliox is included in the U.S. Navy Student Manual for Diving Physicians.

1986 to 2004 - An extensive series demonstrating the value of heliox in the elimination of bubbles was published by Drs. Madsen and Hyldegaard. Their experiments covered their observation of bubbles in the peritoneum and spinal cord of rats following compressed-air exposures. The flux of the gases was as predicted by the product of their solubility and diffusion coefficients and clearly in favour of using helium and oxygen. Two small animal studies during this period using an extreme model of decompression shock did not, however, indicate benefit from heliox.

1976 to-date - commercial experience - Helium/oxygen mixtures, 80/20 have been used successfully in place of air for recompression therapy in commercial diving on US Navy tables since 1976 by Oceaneering (Aberdeen), and Ocean Technical Services, (Gt Yarmouth). A 50/50 helium/oxygen mixture on Comex was in use by Comex, (Aberdeen and Marseille) from 1986. The Grampian Health Board hyperbaric unit also uses Comex 30 heliox.

1992 - The Comex table 50/50 heliox has been officially considered as a valid therapeutic table by the French government in the "decree of 15th May 1992". This decree is defining procedures to be used in a hyperbaric environment.

Procedure for applying heliox therapeutic tables

If the project team plan to use heliox treatment tables, in place of the O_2 and air therapeutic tables for a project, the diving medical specialist must be consulted. The tables to apply (US Navy with heliox or COMEX) should be selected to his advice and the procedures indicated in the emergency response plan.

During a treatment started with air and pure O_2, the decision to apply heliox therapeutic procedures is from the diving medical specialist (DMS).

COMEX tables

COMEX has edited a set of tables to be used for air and heliox diving. These tables that are explained in the "chapter D" of this document are designed for heliox or can be used with heliox (Cx12 & Cx18)

- ***Cx 12*** (oxygen) can be used for treatment of "type 1" accident following an air, nitrox, or heliox dive.
- ***Cx 18*** (oxygen) can be used for treatment of "type 1" accident after no relief or worsening of symptoms in a Table Cx 12.
- ***Cx 30*** (heliox + oxygen) can be used for treatment of "type 2" accident following an air, nitrox or heliox dive.
- ***Cx 30 saturation*** (heliox + oxygen) can be used for treatment of "type 2" accident following an air, nitrox or heliox dive, or after no relief or worsening of symptoms in a Table Cx 30
- ***Cx B*** (heliox + oxygen) can be used for treatment of "type 1" accident during normal decompression or after a blow-up from deeper than 9 m in an heliox bounce dive or air heliox saturation
- ***Cx SB*** (heliox + Oxygen) can be used in case of failure of a table "Cx B" during normal decompression or after a blow-up from deeper than 9 m in an heliox dive or air/heliox saturation.
- ***Cx N*** (heliox + oxygen) can be used for treatment of "type 2" accident during normal decompression or after a blow-up from deeper than 9 m in an heliox dive or an air/heliox saturation.

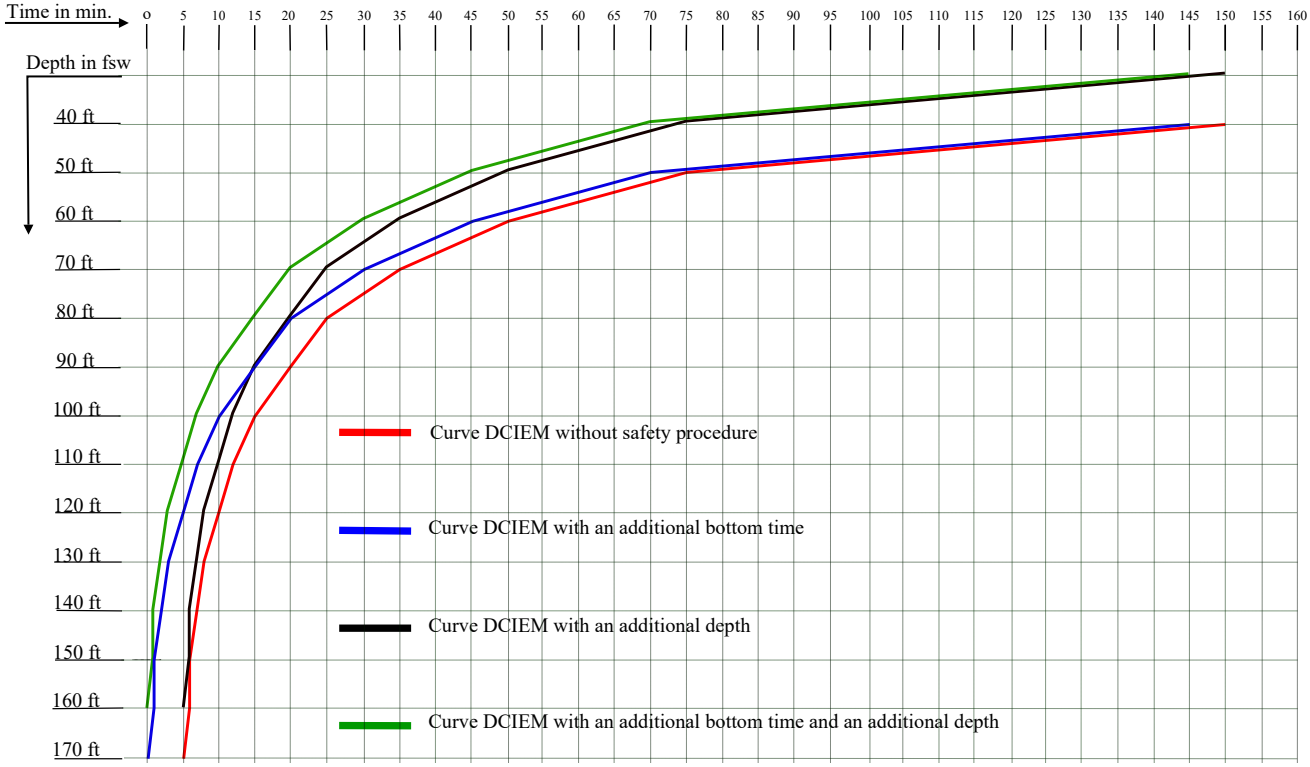
Prevention

1 - Reinforcement of the original decompression procedures

Current diving tables control the risks linked to Decompression Sickness (DCS) by managing factors such as the dive duration, the depth, the ascent rate, and the duration of the stops. However, in the paper *"Preconditioning Methods and Mechanisms for Preventing the Risk of Decompression Sickness in Scuba Divers: A Review"*, doctors Emmanuel Gemp & Jean-Eric Blatteau say that clinical data supporting the importance and the role of each factor on Decompression Sickness (DCS) development are lacking due in part to the great inter/intra-variability between individuals regarding susceptibility to DCS. They also say that based on their clinical experience and Divers Alert Network (DAN) statistics, most injured divers presenting neurological DCS (75%–90%) followed their dive profile and did not performed inadequate decompression schedules, which puts forward the notion that conservative dive profiles are no guarantor of protection against DCS and that novel means are required for DCS prevention.
For these reasons, it is common to apply reinforcement procedures to the initial decompression models selected by the diving companies according to the dive profile, the weather conditions, the hardness of the work, etc.

1.1 - Decompression safety procedure (also called Jesus factor)

The "Decompression safety procedure", previously called "Jesus factor", is an old concept based on the fact that a table is developed for a determined population of divers, which does not always correspond to the divers operating on the job site. This procedure was initially implemented because most tables used during the 60s and 70s were initially designed for military divers which resulted in numerous decompression accidents. This "Jesus factor" is described in detail in the document "The incidence of decompression sickness arising from commercial offshore air-diving operations in the UK sector of the North sea during 1982/83" issued in December 1997 by doctors Shields and Lee.
Even though the tables currently used, such as MT 92 or DCIEM, have provided considerable improvements, this procedure that consists in adding bottom time or switching to the next deeper depth continues to be applied by numerous supervisors and is mandatory with some companies because databases have demonstrated that the tables need to be sometimes reinforced according to the tasks performed, the environmental conditions, and the age of the diver.
In addition to preserving the divers' health, many companies ensure that no decompression sickness happens because such an undesirable event results in incident reports that may damage their reputation. This point is not the most glorious, but it must be taken into account.
This concept has also been adopted by manufacturers of diving computers designed for scuba diving that provide the possibility to reinforce the basic decompression profile. These reinforcements usually consist of shifting the deco curve and do not modify the mathematic model.



Safety procedures are officially introduced in some tables. It is the case of the COMEX /MT 92, where it indicated the following: "When diving or working conditions are difficult, the risk of a decompression accident is higher. It is an established fact that poor physical condition, nervous tension, poor visibility, cold and accumulated fatigue after weeks of intensive diving, predispose a diver to decompression sickness. Similarly, a current, uncertain depth control and poor sea conditions make decompression procedures difficult to follow and thus increase the risk of a decompression accident. All these factors must be taken into consideration when a decompression table is chosen. In the case where diving conditions are such that they may adversely affect decompression safety, the next longest time on the bottom in the table

should be used in order to give the divers an additional margin of safety".
It is also the case of the Norwegian tables where it is said in chapter "prevention for decompression illness": "If there are circumstances increasing the risk for decompression illness, the decompression should be more conservative than prescribed by the tables. Especially this is true if multiple risk increasing factors are present and for dives with bottom times bordering the maximum allowed bottom time. In such cases the standard air decompression tables should be used more conservatively by decompressing according to a table time one or two steps longer than otherwise".
This handbook recommends applying at minimum an additional bottom time or depth, except for dives with perfect sea and underwater conditions for performing light works and using short bottom times.

1.2 - Predive conditioning procedures

Among the solutions investigated to improve decompression, the authors of this document insist on the benefits of "pre-dive conditioning", which refers to experimental studies made to demonstrate that exercises, oxygen, or substances uptake before the immersion have beneficial effects on decompression. These beneficial effects are assumed to result from eliminating nuclei by physical processes or/and chemical reactions. Scientists have successfully tested the pre-dive conditioning solutions listed below on humans.

- Endurance exercise:
  This process consists of exercises requiring 70 to 90% of maximum heart rate performed before the dive. Note that the maximum heart rate is often calculated with the formula "220 minus the age of the person tested".
- Hydration:
  This concept is based on the fact that it has long been suggested that dehydration may increase the risk of Decompression Sickness (DCS) and that experiments have been made on animals that correlate it.
- Heat exposure:
  This concept leans on papers that demonstrated that moderate dehydration resulting in stroke volume reduction induced by a predive exercise could decrease venous circulating bubbles in divers.
- Oxygenation:
  These procedures are based on the assumption that oxygen breathing before diving eliminates pre-existing gas micronuclei before they can grow into bubbles. The proposed mechanism is based on the ability of oxygen to replace nitrogen in the nucleus by diffusion. The reduction of oxygen pressure after switching from oxygen to air could enhance the consumption of oxygen from the nucleus, thus eliminating it completely.
- Vibration:
  This procedure consists of submitting the diver to sessions on vibrating mattresses sold to all public. The effects expected are similar to those obtained with predive exercise except that more efficiency is looked for.
- Jumping:
  This technique aims to provoke blood displacement and muscular contractions to dislodge VGE nuclei. The method selected to obtain the expected result consists of jumping on a mini trampoline.
- Specific substances uptake:
  This terminology refers to drugs or food that can be used to control chemical reactions linked to decompression, such as nitric oxide (NO) production.

The processes of these experiments are described in papers available on the "Diving and ROV Specialists.com" website and through recognized scientific article publishers.
We, nevertheless, need to take into account the fact that these reinforcement processes are experimental and that, despite the positive results obtained, they may not apply to commercial diving operations due to implementation issues and the fact that the procedures described have been tested with military and sportive SCUBA divers, so initially thought in the function of the concept to be tested and according to methods practiced for this type of diving instead of the intensive operations we commonly organize in commercial diving. For these reasons, it is reasonable to be conservative regarding these new procedures, so only to apply what has been tested and not go outside these limits, even though we may feel that some variations of the solutions described may work.

- Regarding endurance exercise and hydration, doctors Gempp & Blatteau conclude their article *"Preconditioning Methods and Mechanisms for Preventing the Risk of Decompression Sickness in Scuba Divers: A Review"* by saying *"Evidence suggests that, for a population of trained and military divers, endurance exercise (even in a warm environment) associated with oral hydration prior to the dive is beneficial in vascular bubble reduction"*.
- Normobaric pre-dive oxygen breathing is a procedure that is easy to implement with standard air diving, and is described in a paper called *"Pre-dive normobaric oxygen reduces bubble formation in scuba divers"*, published by doctors Olivier Castagna, Emmanuel Gempp, and Jean-Eric Blatteau. Because no tests have been made with nitrox and oxygen decompression stops, we must abstain from merging this concept with these procedures, even though there is no apparent conflict, and we feel that the two concepts used together may give excellent results. This is, of course, based on the idea of the conservative approach discussed previously
- Whole body vibration results better than normobaric oxygen breathing and endurance exercise, and this concept can also be implemented for standard air diving.
  However, in an article called "Pre-dive Whole-Body Vibration Better Reduces Decompression-Induced Vascular Gas Emboli than Oxygenation or a Combination of Both", doctors Costantino Balestra, Sigrid Theunissen, Virginie Papadopoulou, Cedric Le Mener, Peter Germonpré, François Guerrero, & Pierre Lafère say that pre-dive conditioning with only whole body vibration was more efficient during experiments than predive-conditioning with normobaric oxygen and body vibration performed together. They say that this

absence of synergy could be explained by the fact that the two modes of preconditioning, mechanical or diffusion, could act on the same nuclei and thus be in direct competition. That demonstrates that procedures that have not been tested must not be implemented, so the idea of the conservative approach must always prevail.

Note that the diving management study CCO Ltd #11 *"About pre-dive conditioning and commercial diving"* makes an analysis of the predive conditioning procedures mentioned above and their possible implementation. As the other documents, this study is available for downloading on the website "Diving and ROV Specialists.com (https://diving-rov-specialists.com/)"

Operational limits UK-HSE and IOGP

The safe operational limits of most tables are based on the repeat groups. The UK-HSE limitations from the report *"The incidence of decompression sickness arising from commercial offshore air-diving operations in the UK sector of the North sea during 1982/83"* issued in December 1997 by doctors Shields and Lee to solve or at least limit the numerous decompression accidents arising in UK waters in the early time of the offshore industry. They are more stringent than those from most tables, and their implementation is mandatory in UK waters and by some organizations. This report is available in the databases of the "Diving and ROV specialists" and "UK HSE" websites.

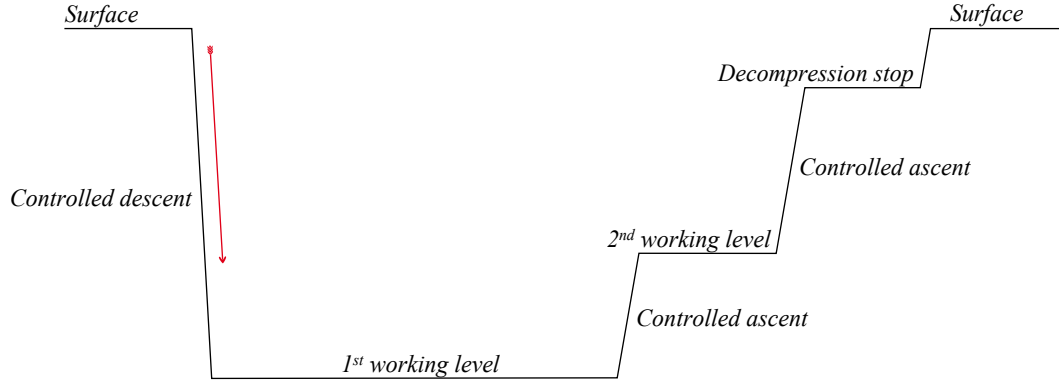
Depth		***Bottom times limits SD & In water***
Metres	***Feet***	
0 - 12	*0 - 40*	*240*
15	*50*	*180*
18	*60*	*120*
21	*70*	*90*
24	*80*	*70*
27	*90*	*60*
30	*100*	*50*
33	*110*	*40*
36	*120*	*35*
39	*130*	*30*
42	*140*	*30*
45	*150*	*25*
48	*160*	*25*
50	*164*	*20*

2 - Management of dive profiles

2.1 - Implementation of square dive profiles

In "Factors influencing the likelihood of DCS", it is explained that the dive profile is among the main elements that influence the generation of DCS and that some profiles must be avoided or forbidden.

A "square dive profile" that consists of starting the dive with the most profound depth planned and finishing the operations by the shallowest depth is to be considered a rule.



- Ascent rates should be scrupulously respected
- For the reasons already explained in "Factors influencing the likelihood of DCS", inverse profiles that start shallow and finish at the most profound level should be avoided. If applied, a reinforcement of the decompression procedure should be implemented.

- "Yoyo profiles", also explained in "Factors influencing the likelihood of DCS" are forbidden and should be considered an omitted decompression if applied.

2.2 - Management of repetitive dives

As explained in "Factors influencing the likelihood of DCS", each repetitive dive begins with an N_2 load from the previous dive. This N_2 load will be supplemented by the nitrogen taken up during the subsequent dive, making favourable conditions for decompression illness. Also, there may be residual physiological effects of the previous dive, increasing the likelihood of decompression sickness. These physiological effects may include a lower body temperature and dehydration from immersion and recent work. For these reasons, some diving organizations recommend not using repetitive dives. It is, for example, the case of IMCA that in the diving guideline D 022 says in chapter 10, *"The divers and standby diver must all be medically fit to dive and clear of any decompression penalties"*.

2.3 - Management of post dive procedures

A distinction between different types of diving is reasonable:

1. After surface-orientated dives requiring decompression stops the divers should remain in the vicinity (within 20 minutes) of a suitable chamber for 4 hours. They should then remain within two hours travelling time of a two-compartment chamber until 12 hours post-surfacing.
2. Shallower than 10 m and for one or two dives within accepted no-stop limits, the divers should remain in the vicinity of a suitable chamber (within 20 minutes) for one hour. The diving contractor's diving rules should make provision for any subsequent emergency procedures after these intervals.

It should be emphasised to all divers that:

- Any symptom should be reported before departure from a dive location.
- Treatment begun soon after the onset of symptoms is often relatively straightforward but treatment which has been delayed for a while after the onset of symptoms may be difficult because the condition has become less responsive.

During the two hours following decompression, it is recommended that divers limit their activities to tasks which do not involve sustained physical effort, and in particular, it is recommended that they avoid running, climbing stairs or participating in intense sports exercises.

Transfer by plane should be organized according to the last revision of DMAC 7, published in November 2017, or the procedures of the decompression table used if they are more stringent.

3 - On site logistic support

Most organizations apply the following procedures:

- A two compartment chamber must be available all the time.
- A full emergency medical kit conforming to the list DMAC 15, or a similar standard must be available in the chamber room. (The list for the kit DMAC 15 is in attachment chapter E " Medical check list and records" of this module). Note that if the therapeutic procedures selected for a project are based on heliox mixes, there should be sufficient quantities of gas to complete any treatment.
- There must be a direct communication with the diving medical specialists in the chamber control.
- Not mandatory, but recommended, a medical monitoring system allowing to record, display, and transmits patient physiological data in real time from within the chamber to the diving medical specialist onshore is recommended.

Post treatment procedures

Chamber proximity & post-Treatment Observation Period:

This point discusses post-treatment performed at the arrival of the saturation dive and not of treatment performed after eventual recompression.

Patient observation time near the recompression facility and:
A patient should remain at less than 15 minutes of a recompression facility for 24 hours and be accompanied throughout that period. He should not be released unless the Diving Medical Specialist authorizes him.

Evacuation to a hospital immediately after surfacing:

Patients treated are likely to be evacuated to a medical facility for a hospitalisation period immediately after surfacing from the treatment. Such evacuation is done only on the recommendation of the Diving Medical Specialist (doctor) in charge.

This medical evacuation (MEDEVAC) should be organized with medical personnel trained to transfer patients suffering DCS or AGE. In the eventuality that this personnel is missing, diver medics may be used with the approval of the Diving

Medical Specialist. During this transfer, the patient should breathe 100 percent oxygen.

Air transfer may be necessary for medical or logistical reasons. However, the Diving Medical Specialist should decide whether this procedure is suitable.
As the aircraft cabin is usually not or insufficiently pressurized, it must fly as low as safely possible and never expose the casualty to an altitude above 300 m (1,000 feet). To compensate for the depression in the aircraft cabin, and if available, an emergency evacuation hyperbaric stretcher that maintains the patient at the sea level pressure (1.013 bar) can be used if the Diving Medical Specialist allows for it.

As an example of a hyperbaric stretcher, the model in the photo is the SOS Hyperlite hyperbaric stretcher JFD.

Residual Symptoms.

After completion of the initial recompression treatment and after a surface interval sufficient to allow complete medical evaluation, additional recompression treatments may be organized by the Diving Medical Specialist. These treatments are more often performed in the hyperbaric hospital, but it may happen that the diving medical Specialist request some sessions on board for several reasons.

In the case that some complementary treatments are undertaken, the team in charge must be aware that it is common that some residual symptoms may remain unchanged during the first one or two treatments. The delay time between completion of initial treatment and the beginning of follow-up hyperbaric treatments is known to decrease the probability of benefit from additional treatments, but this is not a valid reason to preclude follow-up treatments.

The follow up of the patient must be the same as for the initial recompression. Such treatments should be continued until the Diving Medical Specialist judges that there is no further benefit noted.

Post-Treatment Transfer:

The decision to transfer the patient to his home after successful treatment or to a unit for further recompressions is only from the Diving Medical Specialist. Without any clear guidance from the Diving Medical Specialist, the patient must stay on the job site.

The patient may have a handicap resulting from the decompression illness. Regarding this problem, the US Navy says:
"If ambulatory patients are sent home, they should always be accompanied by someone familiar with their condition who can return them to the recompression facility should the need arise."

Most of the time, the patient will return to his home by flight. In this case, the recommendations from DMAC 7 (see below) are to be applied as a minimum:

Table 2: ***Following therapy for DCI, advice must be sought from a diving medical specialist***	***Minimum time from completion of therapy (completion of recompression treatment)***	
	2000 feet (600 m)	***All other flights***
2.1 - Immediate and complete resolution of symptoms on first recompression	24 hours	72 hours
2.2 - Cases without immediate response or with residual symptoms must be decided on an individual basis by a diving medical specialist. Generally wait as long as practical	Consult a diving medical specialist	

Residual risks will be reduced by giving 100% oxygen during the flight. Following landing the diver should be assessed by a diving medical specialist.

DCI in Flight:
In most circumstances, the medical decisions concerning an air passenger who develops symptoms of decompression illness during flight will be the responsibility of the air crew and airline. The following guidance may be helpful.

- *Where the diver's symptoms consist only of pain in a limb, the diver should be treated with analgesics, oral*

and oxygen if available. Advice should be sought from a diving medical specialist. It may be possible for the plane to continue to its destination without diversion or adjustment of altitude, but the risk of development of more serious symptoms, the duration of flight and route need to be considered.

- *When the diver has any other symptoms, immediate advice should be sought from a diving medical specialist. The diver should be given 100% oxygen and oral fluids. Reduction in cabin altitude and diversion to an airport where further treatment can be given may be necessary.*

Assessing Fitness to Return to Diving (DMAC 13):

The DMAC (Diving Medical Advisory Committee) suggests the following minimum intervals before returning to diving after decompression illness. Please consider that the Diving Medical Specialist is the only person who can make the final decision. In some cases, he may decide on longer intervals to complete the investigations required.

The period begins after the completion of successful treatment (there are no residual manifestations).

A - Limb pain, or non-specific manifestations only:

1) With uncomplicated recovery: 7 days
2) Where there has been a recurrence or relapse requiring further recompression: 14 days

B - Neurological or pulmonary manifestations (*Return to diving only after a review by a Diving Medical Specialist):*

1) Sensory disturbance ONLY *(paraesthesia or loss of sensation)*: 28 days.
2) All other neurological or pulmonary symptoms: 3 months

C - Cutaneous and lymphatic manifestations without neurological involvement, i.e. skin rash with severe itching or marbling (Cutis Marmorata) or swelling of tissues: 28 days *(the diver should be assessed by a Diving Medical Specialist)*.

D - Pulmonary barotrauma resulting in a pneumothorax or mediastinal/subcutaneous emphysema. Following appropriate investigation, including High-resolution computed tomography (HRCT) of chest, a diver may be considered fit to return to diving, but no earlier than 3 months after complete recovery.

E - In cases where there are significant residual neurological manifestations, even after repeated treatment, the diver should normally be considered unfit to dive. Return to diving should only be permitted if sanctioned by a diving medicine specialist.

Investigations regarding the use of chemical substances to cure decompression sickness

We have seen that chemical substances have been tested to prevent decompression sickness though pre-dive conditioning procedures. Such substances are also actively tested to resume cases of decompression accidents. Even though the results obtained are still experimental, these studies allow to whish for more efficient treatments than those currently used

Use of antibiotics

In an article published the 8/21/19, it is said that US Naval Medical Research Center (NMRC) scientists explained that an additional therapy compatible with a classical recompression process could reduce decompression illness associated diseases and death in a document called *"A meta-analysis of doxycycline as an adjunctive therapy to prevent decompression sickness (DCS)"* (this document is unfortunately not present on our website).

This meta-analysis was based on three sets of similar hyperbaric data evaluating doxycycline's effect on DCS associated diseases and mortality:

- The primary evaluating factor was mortality.
- The secondary evaluating factor includes the occurrence of cardiovascular and neurological DCS.
- The third estimating factor included time to death based on cardiovascular and neurological DCS.

The team said that doxycycline reduces mortality, although this effect was not statistically significant.
Also, they said that future studies are underway incorporating doxycycline prophylaxis in a simulated scenario that includes decompression from saturation. If worthwhile, the results from these studies will be communicated to the US NAVSEA. In conclusion, such treatments are for the moment only experimental.

Note that doxycycline is an antibiotic sold under numerous brand names such as Acticlate, Adoxa CK, Adoxa Pak, Adoxa TT, Alodox, Avidoxy, Doryx, Mondoxyne NL, Monodox, Morgidox, Okebo, Oracea, Oraxyl, Periostat Targadox, Vibramycin calcium, Vibramycin Hyclate, Vibramycin monohydrate, Vibra-Tabs, and others.
This medication is usually employed to treat bacterial infections, such as acne, urinary tract infections, intestinal infections, respiratory infections, eye infections, gonorrhea, chlamydia, syphilis, periodontitis (gum disease), and others. Some doxycycline forms are used to prevent malaria, treat anthrax, or treat infections caused by mites, ticks, or lice.
It must be taken into account that this antibiotic may have side effects, and that specialized agencies say that people taking it should be in good physical condition. Also, it can affect the tooth and bone development of the fetus of pregnant women. As a conclusion, side effects of medications should be considered before implementing them in the future and may oblige to take into account the individual aspect of divers.

Use of perfluorocarbon emulsion

Perfluorochemicals are synthetic hydrophobic hydrocarbons first developed for the industry (notably for refrigeration devices) and later made miscible with water through combination with organic emulsification agents and thereby adapted for biological use. Their most crucial relevance to biology is their high capacity for dissolving respiratory gases.

In a paper called “Effect of oxygen breathing and perfluorocarbon emulsion treatment on air bubbles in adipose tissue during decompression sickness”, doctors T. Randsoe and O. Hyldegaard say that in experiments undertaken with rats, Decompression Sickness (DCS) after air diving has been treated with success using combined normobaric oxygen breathing and intravascular perfluorocarbon (PFC) emulsions causing increased survival rate and faster bubble clearance from the intravascular compartment. The beneficial PFC effect has been explained by the increased transport capacity of oxygen and inert gases in the blood.

Their assumption was based on the fact that in previous reports, Hyldegaard et al. shown that decompression-induced nitrogen bubbles in adipose tissue or micro-air bubbles injected into tendons and the white substance of the spinal cord will initially grow, then shrink and disappear during oxygen breathing at sea level. This undesirable initial bubble growth during the initial oxygen breathing can be explained by the fact that at equal partial pressure, the blood will carry more dissolved oxygen to the tissue than it can concomitantly remove inert gas so that there is a higher carrying capacity of oxygen than nitrogen. Based on this fact, they considered it conceivable that combined oxygen breathing and perfluorocarbon (PFC) infusion could promote the growth of extravascular bubbles on decompression due to the increased oxygen supply and that, based on PFC’s high capacity for dissolving nitrogen, it is possible that the initial bubble growth seen during oxygen breathing could be either reduced or even eliminated because of the greater solubility of nitrogen in PFC and thereby a greater transport capacity of this gas in the blood, increasing its elimination.

They conclude their paper by saying that according to the experiments undertaken, although transient bubble growth caused by the increased oxygen tension is not eliminated, the combined effect of oxygen breathing and PFC infusion at sea level will result in faster bubble disappearance explained by a combined effect of the increased oxygen window and the greater N2 transport capacity in the blood increasing the rate of nitrogen desaturation.

Isobaric inert gas counter-diffusion

Description

"Isobaric" means equal ambient pressure, and "counterdiffusion" means two (or more) gases diffusing in opposite directions. Thus, what is called "Isobaric Counterdiffusion" (ICD) is a phenomenon that consists of two inert gases of different solubilities and diffusion coefficients moving in opposite directions under equal ambient pressure in the tissues and the blood. For commercial diving activities, these two gases are nitrogen and helium.

Lighter gases diffuse faster than heavier gases. In the case of helium and nitrogen:

- Surrounding nitrogen loaded tissue and blood with helium will result in greater total gas loading because helium will diffuse into tissue and blood faster than nitrogen diffuses out, resulting in higher total inert gas tensions.
- Surrounding helium loaded tissues and blood with nitrogen will produce the opposite effect, helium will outgas faster than nitrogen ingas and total inert gas tensions will be lower.

In his book *"Hyperbaric physics with bubble mechanics and decompression theory in depth"*, Doctor Bruce Wienke (1940 - 2020) said that, counter-diffusion can induce high tissue gas supersaturation levels and greater susceptibility for bubble formation and decompression illness.

- These problems can be avoided by employing light-to-heavy gas mixture switches and by using more slowly diffusing gases than the breathing mixture inside dry suits or chambers. Such a procedure promotes "isobaric desaturation". Note that this principle is used to reduce the stop times of surface orientated heliox tables. For instance, a dive to 130 fsw for 120 min on 80/20 heliox with a switch to 80/20 nitrox at 60 fsw requires 45 min of decompression time, while 210 min is required without the switch.
- The opposite, switching from heavy-to-light gas mixtures and using more rapidly diffusing gases than the breathing mixture inside dry suits or the chamber, promotes "isobaric saturation" and enhanced susceptibility to bubble formation. The former procedure reduces gas loading, while the latter increases gas loading. The effects of gas switching can be dramatic, as it is well known that skin lesions and vestibular dysfunctionality have developed in divers breathing nitrogen while immersed in helium (test chambers and exposure suits).

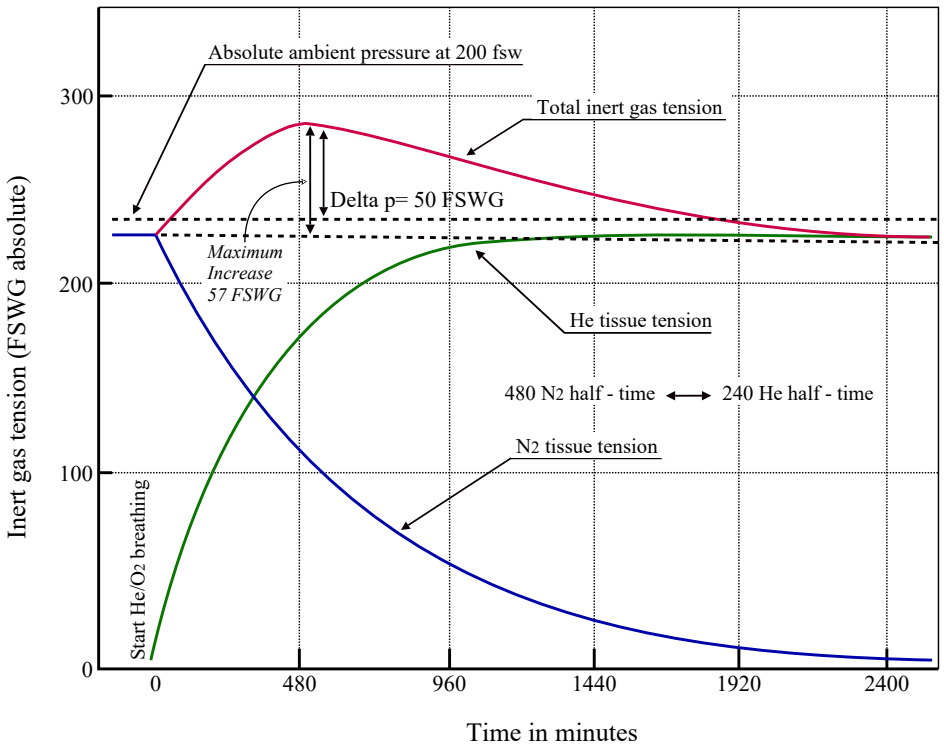


Diagram of "deep tissue" Isobaric Supersaturation *caused by breathing helium after prolonged exposure to N2-02 mixture. Total inert gas pressure rises, and a composite supersaturation is maintained for many hours in "slow-perfusing" tissues. The opposite affect, an equivalent degree and time course of useful subsaturation, is subsaturation, is expected with breathing N2-02 after prolonged exposure to helium (from doctor Lambertsen report)*

Classification & treatment

Christian James Lambertsen (1917 - 2011) a well known diving medicine specialist, wrote several articles regarding counter-diffusion and said that two forms of isobaric counterdiffusion supersaturation can produce gas lesion or venous gas emboli.

- "Superficial" Isobaric counterdiffusion occurs through the body surface when air or Nitrox is breathed and the external environment is helium.
- "Deep tissue- Isobaric counterdiffusion occurs when any different inert gases are breathed in sequence.

As hyperbaric hyperbaric-counter diffusion supersaturation promotes decompression illness, Lambertsen said that Hyper-oxygenation therapy is rational for isobaric counter-diffusion gas lesions, as it is for gas lesions of decompression sickness.

Precautions

The following procedures were recommended by Dr. Lambertsen to avoid counter-diffusion supersaturation:

- Avoidance of mask breathing of air or nitrox while body surface or the ear canals are exposed to helium.
- Avoidance of an abrupt change from air or nitrox breathing to helium-oxygen at a constant or decreasing pressure.
- The use of increased partial pressure of oxygen in inert gas - oxygen mixtures remains the most effective means to reduce tissue total inert gas pressure in the prevention and therapy of each form of gas lesion disease.

The prevention also consists of gas management procedures to avoid the introduction of nitrogen in breathing mixes:

- Nitrox based blends, should never be stored with heliox mixes and must be clearly identified.
- All onboard gasses must be analyzed. The analysis of heliox breathing mixes must identify precisely the proportion of oxygen and helium. If a gap between the two values is detected, the mix must not be used.
- Filtration systems such as “Helipure” or similar are designed to remove the nitrogen from reclaimed gasses to be reused in a system. Unfortunately, such systems, which should be used at all times when recycling heliox, are commonly used for saturation operations but not for bounce diving.

Beneficial use

The phenomenon of counter-diffusion can be used beneficially if well managed:

- For severe decompression sickness, compression on heliox is recommended despite the potential hazard of deep-tissue counter-diffusion supersaturation. Such change to heliox should be accompanied by prompt compression and appropriate oxygen pressure to counter the tendency for bubble growth or development. It is the principle of the table COMEX Cx 30.
- Switching from prolonged heliox breathing to air or nitrox breathing to achieve deep counter-diffusion sub-saturation is a desirable aid to preventing decompression sickness in helium diving.
  In a chamber, it should occur by a change in the ambient atmosphere rather than by mask breathing to avoid superficial counter-diffusion. The transition should take place gradually, rather than abruptly, to avoid exaggerated sensations of narcosis, which may be confused with vestibular effect.

Compression arthralgia

Description

Arthralgia refers to joint pain *(arth = joint; algia = pain)*. Compression arthralgia is linked to fast increased external pressure surrounding the body, which results in pains affecting the joints such as knees, shoulders, fingers, back, hips, neck, ribs. Occasionally, this phenomenon triggers severe lower back pain.

The mechanism of compression pain is not precisely known. It is thought to result from the sudden increase in tissue gas tension surrounding the joints causing fluid shifts and interfering with joint lubrication.

Symptoms

Pains resulting from compression arthralgia are described as deep aching pains, similar to those of Type I decompression sickness. These pains may be relatively sudden and intense. The pains may be accompanied by “popping” of joints or a dry “gritty” feeling within the joint.

- The symptoms are dependent on depth, rate of compression, and individual susceptibility.
- The symptoms may occur around 100 fsw at rapid compression rates and increases with depth and exercise.
- In helium saturation dives with slow compression rates, the symptoms are generally not present above 90 m (300 fsw) and commonly not before 180 m (600 fsw). These pains may be severe enough to limit diving activities. Some improvements are generally noted with the time spent at depth, but these pains may continue during the decompression phase of the dive.
- The symptoms of compression arthralgia can be distinguished from those of decompression sickness because they are present before the decompression start and do not increase in intensity with decreasing depth.

Treatment

The symptoms usually disappear in reverse order during the ascent.

- Unless damaged during the activities at depth, the joint pain disappears, requiring no treatment.
- In case of damaged articulation, the pains might be difficult to differentiate from the pains from decompression illness. One means of differentiation is the fact that recompression to treat a decompression accident should relieve the pains, and in contrast, should increase the pains in case of compression arthralgia...

Prevention

The only method to avoid compression arthralgia is to reduce the compression speeds. For that, the maximum compression speeds indicated in the diving tables must be strictly respected.

- The DCIEM air and surface gas tables descent rate is 60 ft/min.
- The medical tables USN compression rate is 20 ft/min.
- The MT92/2019 compression rate is 30 m/min
- US Navy air tables compression rate is 75 ft/min
- The NORMAM 15 saturation procedures promote the following compression rates to which stabilization periods are added:
    - Surface to 180 m: 1 m / minute
    - Surface to 300 m: 1 m / 2 minutes up to 100 m + 1 m / 4 minutes from 100 to 200 m + 1 m / 6 minutes from 200 m to 300 m
    - Surface to 300 m: 1 m / 2 minutes up to 100 m + 1 m / 4 minutes from 100 to 200 m + 1 m / 6 minutes from 200 m to 300 m + 1 m / 8 minutes from 300 m to 350 m

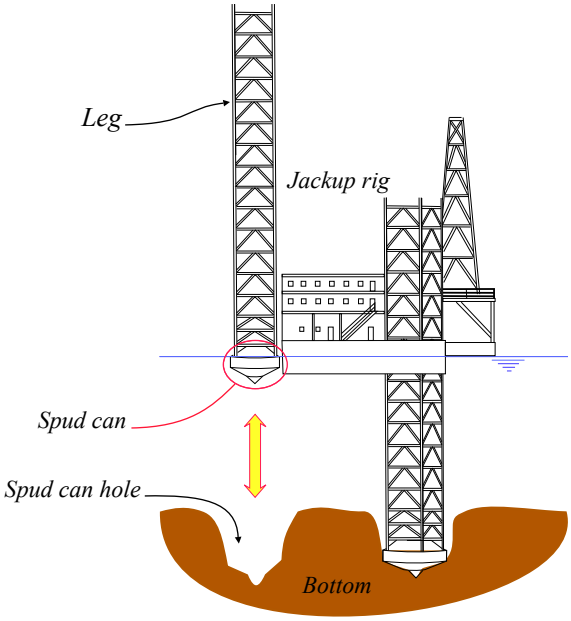
Hydrocarbons

Description

Hydrocarbons are organic compounds containing only carbon and hydrogen, such as alkanes, alkenes, alkynes, terpenes, and arenes. The majority of hydrocarbons are found in crude oil and natural gas, where decomposed organic matter provides an abundance of carbon and hydrogen. Of course, the hydrocarbons will be found in all product derivatives from crude oil like lubricants, plastics, rubbers, solvents, paints, and other industrial chemicals.

Hydrocarbons that pose the greatest risk of harm have a low viscosity, high volatility, and high surface tension or adhesion of molecules along a surface. These characteristics combine to allow hydrocarbons to enter the pulmonary tree, causing aspiration pneumonitis and the potential for systemic effects such as Central Nervous System depression with respiratory and cardiac failure. Some other potential damage can be done to the liver, kidneys, or bone marrow. The onset of these effects is usually rapid. Note that hydrocarbons can also burn and penetrate the skin.

During diving operations, hydrocarbons may be present around platforms and underwater installations. It is also common to find drilling muds saturated in hydrocarbons dumped into spud cans holes by jack-up rigs at the end of their campaigns. These muds may have chemical reactions that produce sudden and massive bubbling that can quickly pollute the atmosphere of the wet bell if it is positioned above. They can also contaminate the chamber atmosphere if surface decompression is performed and the diver's clothes and body are polluted by such substances. In addition, the diver can be contaminated through the skin if he is not protected by a relevant suit.



Hydrocarbons can also be present on deck in the vicinity of air compressors' intakes, affecting air divers in such a case. Also, because today we are surrounded by plastics, the combustion of plastic items in a chamber will quickly pollute the atmosphere in case of fire. Besides, hydrocarbons may be present in the products used to maintain equipment and diving compressors' lubrication.

Symptoms

The common visible symptoms and consequences of hydrocarbon toxicity include the following:

- Burning of the skin exposed to polluted muds during the dive
- Burning sensation on swallowing
- Nausea and vomiting
- Abdominal cramps
- Weakness
- loss of sensation and awareness
- Hallucinations
- Changes in colour perception
- Blindness
- Loss of conscious
- Coma
- Death if nothing is done

Treatment

Emergency care for hydrocarbon intoxication is supportive and includes:

- Removal of the patient from the poisoned environment
- Decontamination (Note that in cases of only gas coming from external, flushing the bell can be efficient, but in cases of contaminated materials in the bell the flushing will not be efficient due to the ongoing evaporation)
- Contact with Doctor .
- Breathing and circulatory support.
- Vital sign monitoring.
- Fluid therapy.
- MEDEVAC as soon as possible.

Emergency actions in bells before being intoxicated:

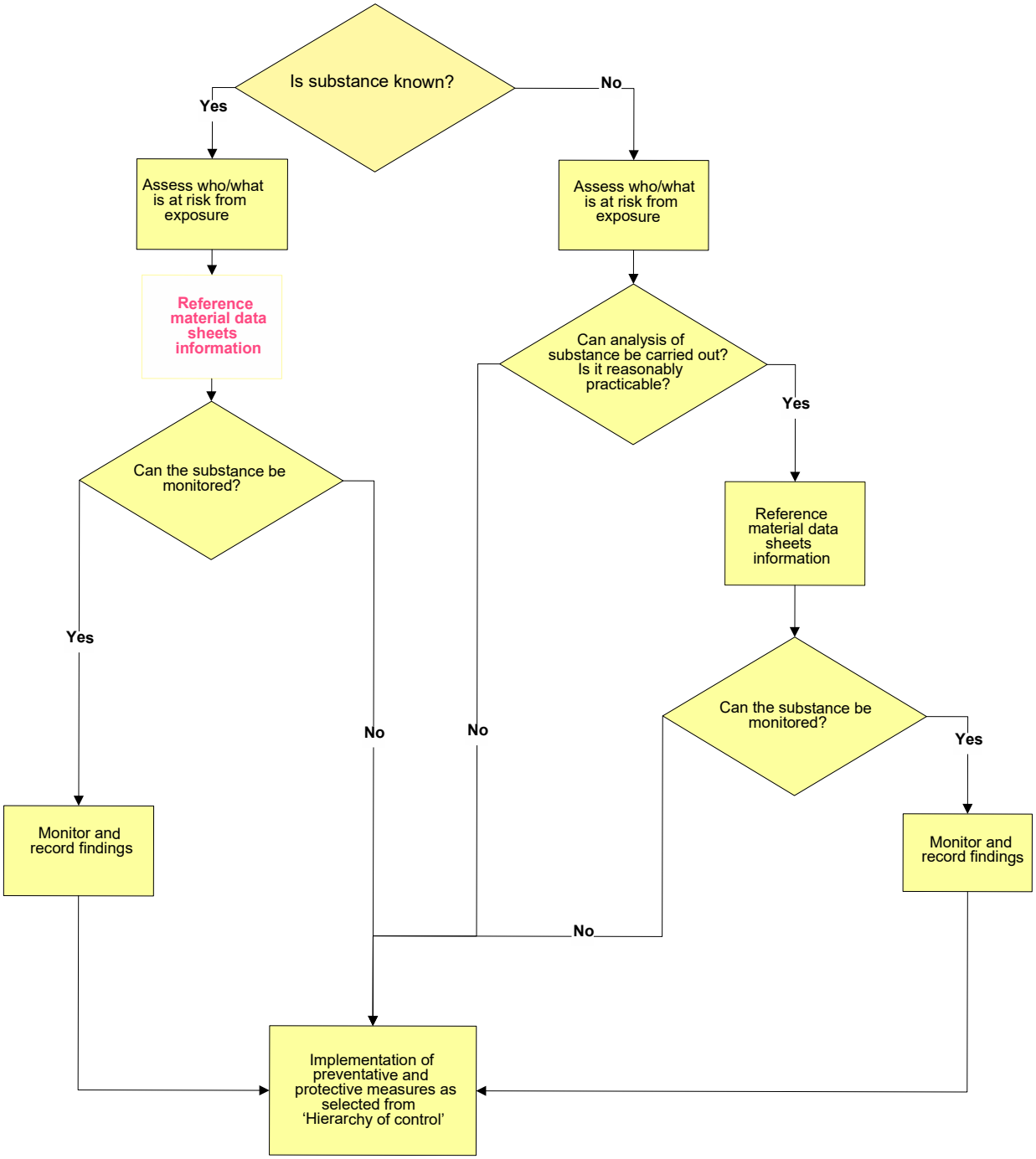
- In case of pollution of the wet bell, flushing may be sufficient to clean the atmosphere only in case of gas bubbles coming inside.
- Flushing the wet bell will not be efficient if the pollution is due to contaminated objects and suits. In this case, the contaminated thing is to be removed from the bell, or the divers must not remove the helmets. If there are some O2 stops planned in the bell, they must be canceled. In this case, the supervisor can organize a surface decompression, but the divers will have to be decontaminated before transferring into the Deck Decompression Chamber.
- Decontamination prior to entering the chamber includes the removal of all clothes and washing the diver if his body has been in contact with the pollutant. If the chamber atmosphere is polluted, continue flushing and ensure the diver is on BIBS.

Prevention

- For chambers, the hazard of atmospheric contamination can be reduced by ensuring that only gases that meet the appropriate specifications are used and that appropriate gas transfer procedures are used. All gas storage and chamber piping should be cleaned using approved cleaning procedures to remove substances that may become chamber contaminants. Once cleaned, care shall be taken to prevent introduction of contaminants back into these systems during maintenance by marking and bagging openings into the piping system. Inadvertent chamber contamination can be prevented by limiting the items that may be taken inside. Only approved paints, lubricants, solvents, glues, equipment, and other materials known not to off-gas potential toxic contaminants are allowed in the chamber. Strict control of all substances entering the chamber is an essential element in preventing chamber contamination.
- The US Navy manual indicates the following maximum concentration in chamber atmosphere:

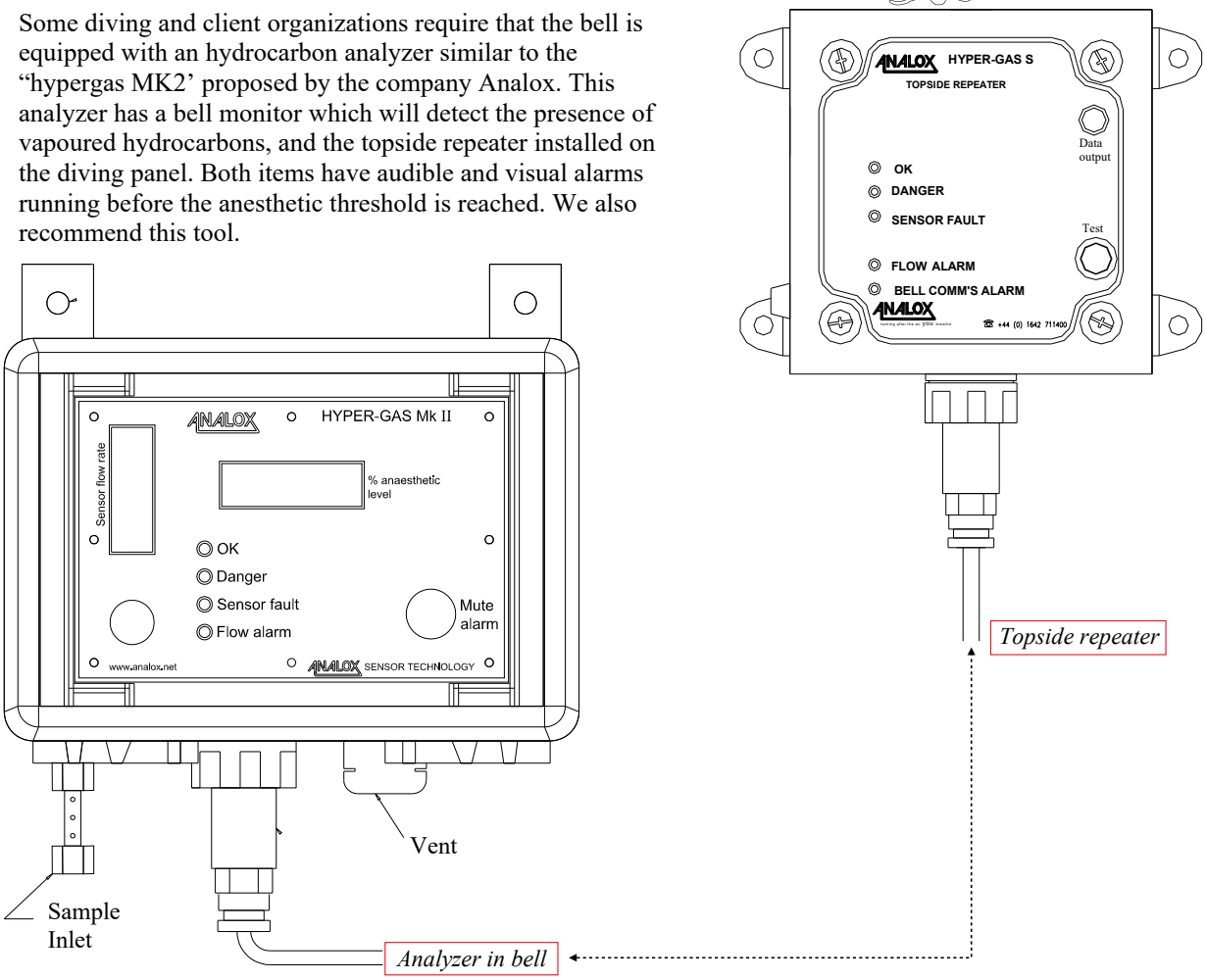
Hydrocarbons	Maximum concentration	Description
Acetone	200 ppm	Acetone is produced directly or indirectly from propylene, also known as propene or methyl ethylene, and is the second simplest member of the alkenes class of hydrocarbons.
Benzene	1 ppm	Benzene is a natural constituent of crude oil, and is one of the most elementary petrochemicals. Benzene is an aromatic hydrocarbon. It is a colourless and highly flammable liquid with a sweet smell. It is an important component of gasoline.
Trimethyl Benzenes	3 ppm	The Trimethyl benzene constitute a group of substances of aromatic hydrocarbons, which structure consists of a benzene ring with three methyl groups ($–CH_3$) as a subsistent.
Toluene	20 ppm	Toluene is an aromatic hydrocarbon that is widely used as an industrial feedstock and as a solvent. Like other solvents, toluene is sometimes also used as an inhalant drug for its intoxicating properties; however, inhaling toluene has potential to cause severe neurological harm.
Xylenes	50 ppm	A Xylenes is an aromatic hydrocarbon consisting of a benzene ring with two methyl substituents. The mixture is a slightly greasy, colourless liquid commonly encountered as a solvent

- For air coming from compressors the European norm BS/EN 12021 indicates that the lubricant content shall not exceed 0.5 mg/m3 and there must be no odors.
- OSHA regulation (USA) is also indicating 5mg/m3 of hydrocarbons by weight for air purity.
- A sampling of the mud and analysis is to be carried out before intervention on the bottom near facilities. The sampling is to be done using ROV.
- IMCA D 021 " Diving in contaminated waters" indicates that a risk assessment has to be carried out to decide wether the operation is feasible or not . The following chart should be used:



- IMCA D 006 "Diving in vicinity of pipe lines" and IMCA D 044 "guidelines for isolation and intervention" must be also considered.
- If necessary, barrier creams, washing chemicals, protective suits, gloves, etc. shall be used.
- In the case of strong pollution and possible problems for the health of the divers, ROV Shall be used.
- A clear dive plan will take into account all the possible problems which can be encountered due to the presence of hydrocarbons must be written. This dive plan will integrate the necessary time to clean or remove the clothes , boots, and umbilical in case of use of wet bell.
- The teams should request a clear plan of the bottom near the facility, indicating the possible spud cans holes, and in particular the ones which have been filled with drilling mud. The location of the flanges and other elements susceptible to have leaks must be also indicated by the client.

- Some diving and client organizations require that the bell is equipped with an hydrocarbon analyzer similar to the "hypergas MK2' proposed by the company Analox. This analyzer has a bell monitor which will detect the presence of vapoured hydrocarbons, and the topside repeater installed on the diving panel. Both items have audible and visual alarms running before the anesthetic threshold is reached. We also recommend this tool.



- In the case that a suitable analyzer is not available in the wet bell, chemical tubes similar to those proposed by the company DRÄGER must be used at regular intervals.
- Note that the market offers today a full range of portable electronic analyzers which could be used in place of model above and the chemical tubes.
- The maintenance of the diving system should be performed using only the products recommended by the manufacturers. In case of no particular recommendation from the manufacturer, products without hydrocarbons and harmful components shall be used inside the bell and for the breathing gas supply systems.
- Hydrocarbons must be stored far from the dive station. Having an analyzer with visual and audible alarm near the air compressor inlet is a good practice.

Precautions during the diving operations

Protection of the divers:

- The supervisor must not store the wet bell directly above the work site and any element which could have leaks (valves, flanges…)
- The wet bell must not be stored above spud can holes which are areas from where unexpected bubbling can happen if filled by dumped drilling mud, and above other suspicious areas.
- If bubbling or hydrocarbon leaks are detected, the dive must be aborted. Then an investigation and a risk assessment shall be done to decide whether the operation using divers is safe.
- The umbilicals, diving suits, and boots shall be cleaned before the recovering in the wet bell.
- If using over suits, they must be removed outside the bell if possible.
- The bellman and the supervisor should ensure that the analyzer is active. If using chemical tubes, samplings of the bell atmosphere should be done to make sure of the absence of hydrocarbons at regular intervals.
- The surface team should ensure that no hydrocarbons are dumped or stored near the air compressors intakes.
- In case of hydrocarbons detected in the wet bell, the dive must be aborted, and the emergency precautions implemented.

Protection of the personnel operating and maintaining the diving system:

The areas where the diving system is installed must be clear from hydrocarbons for the reasons indicated previously.

However, the personnel in charge of maintaining the system and operating it may need to maintain equipment such as generators situated in the vessel's machinery space and close to engines or other equipment continuously running. Motor oils and solvents used for the boat machinery may expose them to hydrocarbons emissions while performing their duties.

To control such exposures, the limits for the control of hazardous substances at work provided by national safety organizations can be used. They are indicated under various names, depending on the organization. However the principle of calculation is similar. As an example the UK Health and Safety Executive (HSE) calls them “Workplace Exposure Limits (WELs)”. These limits are concentrations of hazardous substances in the air, averaged over a specific period referred to as a time-weighted average (TWA). Two duration of exposures are usually used:

- Short-Term Exposure Limits (STEL) are set to prevent acute effects such as eye irritation, which may occur following exposure for a few minutes. They are usually limited to 15 minutes.
- Long term exposure limits (LTEL) are set to prevent effect that may not be noticeable during the intervention but may appear as a result of long and repeated exposures. They are normally limited to 8 hours.
  Note that the "8-hour reference period" refers to occupational exposures within a 24-hour period. It can be a single uniform exposure for 8 hours (the 8-hour time-weighted average (TWA) exposure) or an addition of several short exposures.
  The calculation proposed is: T*ime in decimals x concentration in mg.m³ / 8*
  Note that time is decimal consists in dividing the minutes by 60 and multiply them by 100. Example: 7:20 hours = 7.33.
- The list of contaminants and their maximum exposure limits to can be found in the UK HSE document EH40 *“Workplace exposure limits”* that can be downloaded free of charge.
  Note that a lot of substances have several names, and are usually listed under one of them only: That obliges to find the name used through the list of synonyms of the document or through the Internet.

Workplace exposure limits (surface) document UK HSE EH 40

	Long-term exposure limit (8-hr TWA reference period)		***Short-term exposure limit (15 minutes reference period)***	
Substances	***ppm***	***mg.m³***	***ppm***	***mg.m³***
Acetone	500	1210	1500	3620
Toluene	50	191	100	384
Benzene	1	3.25	—	—
Trimethylbenzenes	25	125	—	—
Xylenes	50	220	100	441

Hydrogen sulfide (H2S)

Description

Hydrogen sulfide is the chemical compound with the formula H2S that results from the bacterial breakdown of organic matter in the absence of oxygen. This process is commonly known as anaerobic digestion.

Hydrogen sulfide occurs in crude petroleum, but natural gas can contain up to 90%, making the oil and gas industry one of the largest sources of H2S. The locations linked to H2S occurrence include, but are not limited to:

- Drilling Operations
- Well Stimulation Operations
- Well Service Operations
- Field Production Facilities
- Plant Production Facilities

That means that for diving teams working in the petroleum industry, H_2S can be found near the following:

- The bottom muds near production facilities
- The wellheads
- The piping systems and pipe lines
- Pressure vessels and storage tanks

Other potential sources of H2S are:

- Volcanic springs, where probably arises from sulfide minerals.
- Well water, due to the action of sulfate-reducing bacteria.
- Swamps and sewers (where it is a danger for the workers).

Dissolved in water, hydrogen sulfide is known as hydro sulfuric acid or sulfhydric acid and recorded as a weak acid.
Properties of hydrogen sulfide are:

Physical state	*Generally encountered as gas.*
Colour	*Colourless: No visible sign to warn of its presence.*
Odour	*Smells like rotten eggs at low concentration, not noticeable at high concentration.*
Vapour density	*Slightly heavier than air, can be present in gas mixtures even lighter than air.* *"Heavier than air" means the gas may collect in low areas or be trapped by buildings or other confined spaces".*
Flammability	*Highly flammable. It ignites at 260 C°, burns with a blue flame, and gives off sulfur dioxide (SO_2) gas, which is harmful to the eyes and the respiratory system.* *H2S is also explosive when mixed with air.*
Solubility	*Dissolves in the water, oil, and other liquids and emulsions.* *H2S is released when the liquids are agitated, heated or depressurized.*

Symptoms

Hydrogen sulfide is considered a broad-spectrum poison, meaning that it can poison several different systems in the body, although the nervous system is most affected. The toxicity of H2S is comparable with that of hydrogen cyanide or carbon monoxide.

At lower levels of exposure, H_2S irritates the eyes and throat. Higher exposure levels impair the sense of smell. At still higher levels, it has a major effect on the nervous and respiratory systems. Individual sensitivity to H2S exposure may vary from person to person.

Visible symptoms linked to partial pressures exposure:

Less than 1 ppm	Rotten egg smell noticeable, no effect on the body
10 ppm	No known adverse health effects for most people Notice that respiratory protection is required beyond this level
20 to 200 ppm	Eye and respiratory tract irritation Loss of smell Headache and nausea Prolonged exposure (for several hours or days) to concentrations as low as 50-100 ppm can cause a runny nose, cough, hoarseness, and shortness of breath Prolonged exposure to higher concentrations than 100 ppm can produce bronchitis, pneumonia and a potentially fatal build-up of fluid in the lungs (pulmonary oedema)
200 to 500 ppm	Above effects, but sooner and more severe Loss of breathing and death within hours
500 to 700 ppm	Affects the central nervous system Loss of reasoning Loss of balance Unconsciousness and breathing to stop within minutes
700 to 1000 ppm	Immediate loss of consciousness Permanent brain damage and death if not rescued immediately

Note that workers who survive a serious short-term H2S exposure may recover completely, or may experience long-term effects:

- Permanent or persistent nervous system effects including fatigue, anxiety, irritability, intellectual decline, reduced attention span, impaired learning and memory, altered sense of smell, and motor deficits have been reported. It is indicated that some of the nervous system effects may be due to a lack of oxygen reaching the brain cells during a severe H_2S exposure and not the H_2S itself.
- Respiratory effects to people exposed to high concentrations can lead to hypersensitivity of the airways and permanent lung damage with significant reductions of the residual volume of the lungs.

Treatment

Hydrogen sulphide is extremely flammable and very toxic.

If detected in the wet bell during the dive:

- Remove the H_2S by immediate flushing.
- Stop the dive and recover the divers.

If detected on deck:

- The dive must be stopped immediately.
- Stop compressors, thermal engines, and devices that may emit sparks.
- The personnel recovering the divers must wear appropriate breathing apparatus.
- The unnecessary personnel must evacuate the workplace and go to the dedicated safe place on the vessel.
- DO NOT transfer the divers to the chamber, or it will be contaminated during their transfer, and the divers will have to remove their hat and be exposed to the pollution during this phase. Instead, decompress the divers in the water. If it is necessary to recover them on deck and do not remove the helmets as long as they are on deck.
- The vessel must be removed from the contamination source at a slow speed (Move the vessel away). Note that the diver can be kept in the bell/basket during this phase and recovered only when the vessel is outside the polluted area.

In case of a casualty in the wet bell *(the wet bell should be designed with oxygen breathing system)*:

- Flush the bell. And recover it to the stop level
- Administer oxygen when possible, particularly in case of breathing difficulties.
- If breathing has stopped, begin artificial respiration and cardiopulmonary resuscitation (CPR) if the heart has stopped.

- Contact the diving medical specialist (DMS) and organize the MEDEVAC as soon as possible.
- In case of casualty on deck:
- Move the victim to fresh air and a secured area.
- If breathing is difficult, administer emergency oxygen.

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- If breathing is difficult, administer emergency oxygen.
- If breathing has stopped, start artificial respiration and cardiopulmonary resuscitation (CPR) if the heart has stopped. Note that automated defibrillation system can be used for cardio-pulmonary resuscitation.

Prevention

The prevention consists of protecting the divers but also the personnel operating the diving system.

Provision of breathing systems and refuges:

Systems allowing to breathe in a polluted atmosphere should be provided and available at all times on deck, in the dive control, and of course, in the bell and chambers:

- Sufficient emergency breathing apparatus with communications allowing the supervisor to manage the divers' recovery safely must be available in the dive control.
- Sufficient emergency breathing apparatus with communications should also be available for the people in charge of the recovery of the divers.
- A refuge must be organized on the vessel for the people working on deck. Generally, this refuge is in the highest parts of the vessel and is easy to seal.
- Sufficient numbers of breathing apparatus, allowing the unnecessary personnel to escape from the dive station to the protected refuge, must be available on the dive station.

Provision of analyzers and gas detectors:

The places where hydrogen sulfide is likely are usually known. Thus, except in a very new location, the team should be warned on time, and specific procedures and means of detection should be organized:

- If H2S is suspected in the workplace, it must be clearly indicated to the diving team, and a risk assessment must be done to ensure that the intervention is safe. As usual, if the conditions are considered too dangerous, an ROV can be used in place of divers as it can be launched far from the source. If it is decided to launch the operations, the emergency procedures must be integrated into the emergency response plan. These emergency procedures consist of the recovery of the divers and the site's abandonment in safe conditions.
- H2S analyzers should be available in the wet bell. Note that recent electronic models can be used. In the eventuality that a suitable electronic analyzer is not available, chemical tubes similar to those proposed by the company DRÄGER must be present.
- If the surface standby divers are supplied by air compressors, each compressor must be equipped with an analyzer that shuts it down if Hydrogen sulfide is detected. This analyzer must be equipped with visual and audible alarms. Note that it is more suitable to supply the stand by diver and the divers with compressed air.
- Detectors should be installed on the launching station. As the weight of H_2S is higher than air, these analyzers should be situated in the floor's proximity. Multi-gas analyzers exist that can be used for this purpose.
- A room gas detector should also be present in the dive control. The dive control may also be protected by systems against gas intrusion. Please remember that Room detectors are specific for analyzing the room's atmosphere. As for deck analyzers, multi-gas models are currently available.

Avoid and control exposures

Except for operations exceptionally performed from fixed locations, diving operations are usually performed from vessels that can escape sudden gas emissions. Thus exposure to hydrogen sulfide can be minimized and is mostly accidental when diving from a vessel. Nevertheless, these accidental exposures should be controlled, and the “exposure limits to H2S” published by national safety organizations can be used for this purpose.

Safety organizations set up limits for controlling hazardous substances at work that are indicated under various names, depending on the organization. These limits are concentrations of hazardous substances in the air, averaged over a specific period, and their principle of calculation is similar. For example, the UK Health and Safety Executive calls them “Workplace Exposure Limits (WELs)”, and the duration of exposure is called Time-Weighted Average (TWA). Such exposure limits can be employed for people working on deck during emergencies.

- Two duration of exposures are usually used by the UK HSE, that are also used by other organizations:
    - Short-Term Exposure Limits (STEL) are set to prevent acute effects such as eye irritation, which may occur following exposure for a few minutes. They are usually limited to 15 minutes.

- Long term exposure limits (LTEL) are set to prevent effects that may not be noticeable during the intervention but may appear as a result of long and repeated exposures. They are normally limited to 8 hours.
  Note that the "8-hour reference period" refers to occupational exposures within a 24-hour period for people working 5 days per week. It can be a single uniform exposure for 8 hours (the 8-hour time-weighted average (TWA) exposure) or an addition of several short exposures.
  The calculation proposed is: Time in decimals x concentration in $mg.m^3$ / 8
  Note that time in decimal consists in dividing the minutes by 60 and multiply them by 100. Example: 7:20 hours = 7.33.

The UK HSE exposure limits for hydrogen sulfide below can be found in the document EH40. Note that these values are also those of a lot of national standards. Note that exposure limits for 12 hours shift are not published.

- Long term exposures: 5 ppm or 7 $mg.m^3$
- Short term exposure: 10 ppm or 14 $mg.m^3$

In case of the presence of H_2S on the deck, the best procedure is to escape from the source of emission as soon as possible and not operate any tool or chamber's transfer lock as long as the vessel is within the polluted area.

Avoid wet bell contamination:

Wet bell's contamination can be avoided by applying the following procedure, but not limited to. Note that these procedures are similar to those of hydrocarbons:

- The bell should not be stored directly above areas from where hydrogen sulfide bubbling is likely.
- If H_2S bubbling is detected, the dive must be aborted. An investigation and a risk assessment should be performed following the dive to decide whether the operation can be safely performed, and elaborate adequate management of change to write a new dive plan.
- The umbilicals, diving suits, and boots should always be cleaned before recovering the diver into the bell.
- If using over suits, they must be removed outside the bell and stored in the box of the clump weight or the standoff frame.
- The bellman and the supervisor should make regular analyses of the bell atmosphere. If traces of hydrogen sulphide are detected in the bell, it must be flushed and the divers recalled. The dive should be aborted, and the process indicated above undertaken.

Carbon monoxide (CO)

Description

Carbon monoxide is a toxic gas that is colourless, odourless, tasteless, and initially non-irritating.
It is a product of incomplete combustion of organic matter due to insufficient oxygen supply to enable complete oxidation to carbon dioxide (CO2). It is often produced on vessels by equipment powered by thermal engines.

Carbon monoxide has a higher diffusion coefficient than oxygen. The affinity between hemoglobin and carbon monoxide is more than 200 times greater than the affinity between hemoglobin and oxygen, so hemoglobin binds to carbon monoxide in preference to oxygen. Carbon monoxide associated with hemoglobin is called “carboxyhemoglobin”. Exposures at 100 ppm or greater are dangerous to human health.

CO poisoning initially was thought to be exclusively due to the cellular hypoxia imposed by replacing oxyhemoglobin with carboxyhemoglobin and producing relative anaemia. More recent studies have proved that CO poisoning is much more complex than initially presumed and has mechanisms of toxicity beyond the formation of carboxyhemoglobin.
The current understanding of CO poisoning relates its clinical effects to a combination of “hypoxia-ischemia” *(Tissue hypoxia resulting from a slower circulation through the tissues)* due to carboxyhemoglobin formation and direct CO toxicity at the cellular level due to its binding to many proteins other than hemoglobin:

- The binding to “cytochromes” *(iron-containing proteins important in cell respiration as catalysts of oxidation-reduction reactions)* generates oxygen free radicals that impair the electron transport from oxygen radicals and impair cellular respiration due to the inactivation of mitochondrial enzymes. The cellular energy metabolism is inhibited even after the normalization of the carboxyhemoglobin level.
- The binding to “myoglobin” *(an iron-containing protein that is the primary oxygen-carrying pigment of muscle tissues)* may reduce oxygen availability in the heart, lead to arrhythmias and cardiac dysfunction, and contribute to direct skeletal muscle toxicity.
- CO stimulates “guanylyl cyclase “*(enzyme allowing the entry of calcium into the cell)*, resulting in cerebral vaso-dilation, which has been associated with loss of consciousness in CO poisoning.

Also, there is speculation that loss of consciousness during exposure to Carbon Monoxide may be related to produced Oxygen free radicals like Nitric Oxide (NO) acting as a peripheral vasodilator with hypotension as a result. In addition, Nitric Oxide seems to play a pivotal role in a cascade of events culminating in oxidative damage to the brain, which may be responsible for delayed neurological sequels.

Symptoms

The effects produced by carbon monoxide are in relation to ambient concentration in part per million

Concentration	Symptoms
35 ppm (0.0035%)	Headache and dizziness within six to eight hours of constant exposure
100 ppm (0.01%)	Slight headache in two to three hours
200 ppm (0.02%)	Slight headache within two to three hours; loss of judgment
400 ppm (0.04%)	Frontal headache within one to two hours
800 ppm (0.08%)	Dizziness, nausea, and convulsions within 45 min; insensible within 2 hours
1600 ppm (0.16%)	Headache, tachycardia, dizziness, and nausea within 20 min; death in less than 2 hours
3200 ppm (0.32%)	Headache, dizziness and nausea in five to ten minutes. Death within 30 minutes
6400 ppm (0.64%)	Headache and dizziness in one to two minutes. Convulsions, respiratory arrest, and death in less than 20 min.
12800 ppm (1.28%)	Unconsciousness after 2–3 breaths. Death in less than three minutes.

One of the major concerns following acute carbon monoxide poisoning is the severe delayed neurological manifestations that may occur and may include difficulties with higher intellectual functions:

- Short term memory loss,
- Dementia,
- Amnesia
- Psychoses
- Irritability
- Strange gait
- Speech disturbances,
- Parkinson's disease-like syndromes,
- Cortical blindness,
- Depression may even occur to those who did not have pre-existing depression.

It is difficult to predict who will develop delayed sequels; however, advanced age, loss of consciousness while poisoned, and initial neurological abnormalities may increase the chance of developing delayed symptoms.

In the case that divers are affected, note that carbon monoxide poisoning is particularly treacherous because conspicuous symptoms may be delayed until the diver begins to ascend:

- While at depth, the greater partial pressure of oxygen in the breathing supply forces more oxygen into the blood plasma. Some of this additional oxygen reaches the cells and helps to offset the hypoxia. In addition, the increased partial pressure of oxygen forcibly displaces some carbon monoxide from the hemoglobin.
- However, during the ascent, the full effect of carbon monoxide poisoning is felt as the partial pressure of oxygen diminishes.

Treatment

First action:

The first action consists of removing the people affected by the source of contamination as soon as possible:

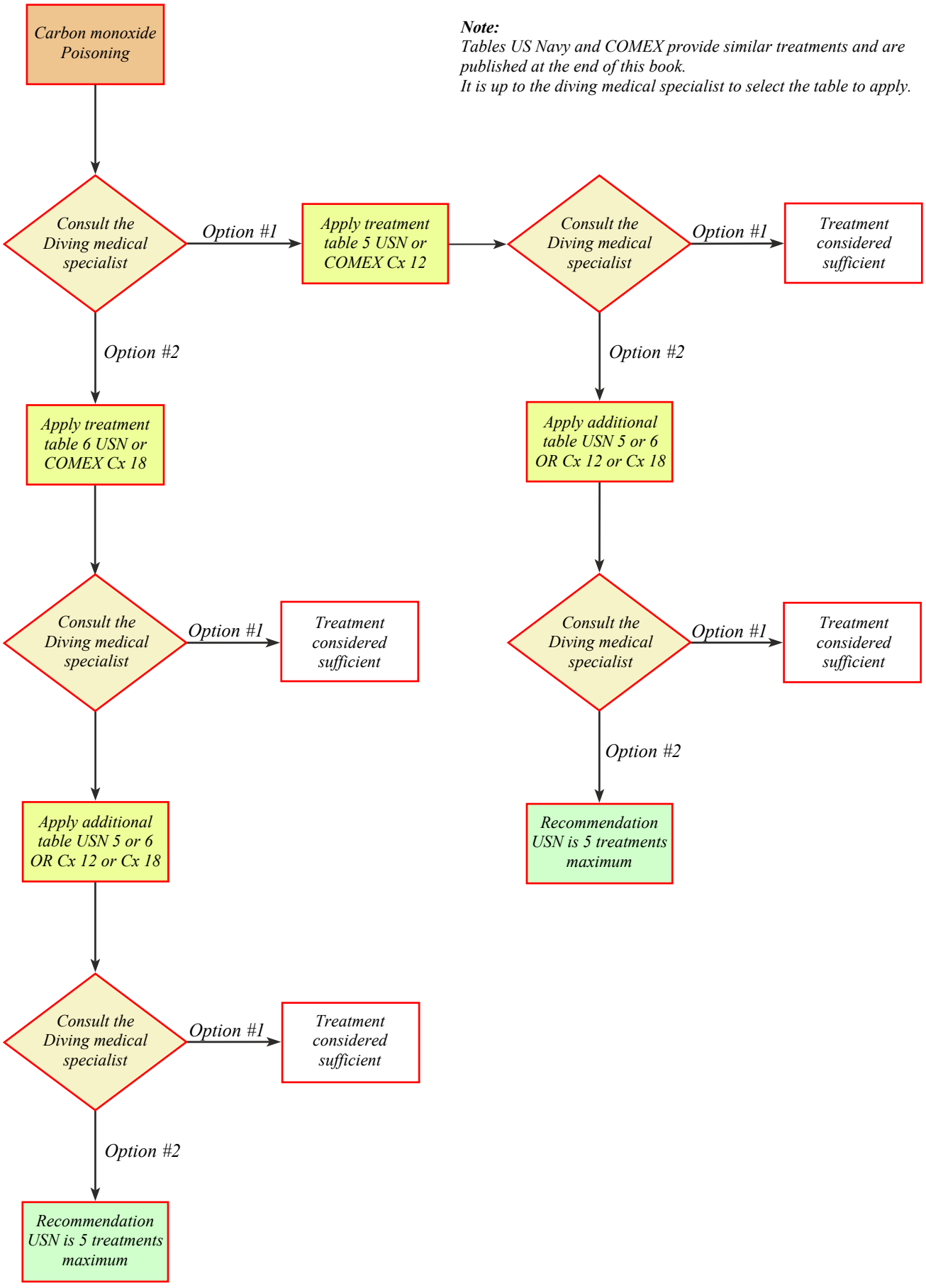
- When the presence of carbon monoxide is discovered on the dive station and in the dive and saturation controls:
  - The not essential personnel must be warned, and evacuated until the environment is again safe. The personnel assigned to the recovery of the bell should wear breathing apparatus.
  - If the dive controls is not protected against gas intrusion, the diving supervisors should wear breathing apparatus, and the rooms must be ventilated with fresh air.
  - The source of pollution is to be identified and switched off as soon as possible.
- If carbon monoxide is detected online during the dive:
  - The supervisor must immediately switch to the backup supply, and the dive is to be aborted.
  - The affected diver must flush his hat with clean gas.
  - If used, the wet bell must be flushed with clean gas if it is contaminated by the polluted source.
  - The suspect breathing gas source should be isolated and gas samples forwarded for analysis as soon as possible.

In case of poisoning:

The Diving Medical Specialist (doctor) must be warned, and the treatment of the CO-poisoned patient begins with supportive care, including:

- Airways management, blood pressure support, and stabilization of cardiovascular status
- High-flow oxygen therapy should be administered immediately to treat the hypoxia resulting from the CO poisoning and accelerate the elimination of carbon monoxide from the body. Supplemental O2 inhalation is essential in treating CO poisoning as the “carboxyhemoglobin” dissociation is hastened by an elevation in the PPO2 of inspired gas.
- Hyperbaric oxygen treatment must be started as soon as possible: Hyperbaric oxygen hastens dissociation beyond a rate achievable by breathing pure O2 at sea-level pressure. Therefore, it has been used to treat severe CO poisoning for several decades.
- Depending on the recommendation of the doctor in charge, US Navy table 5 or 6 or COMEX Cx 12 or Cx 18 can be used in hyperbaric oxygen therapy. Note that the US Navy says that the treatment of CO poisoning can be performed a maximum of 5 times consecutively *(See the chart on the next page)*.

Procedure for oxygen poisoning hyperbaric treatment



Note:
Tables US Navy and COMEX provide similar treatments and are published at the end of this book.
It is up to the diving medical specialist to select the table to apply.

Prevention

Breathing gas management

Breathing gases should be accompanied by an analysis certificate. These certificates should not be accepted as correct until a competent member of the dive team has analyzed them for O_2 and CO content, as a minimum.

- The gas purity standard EN 1221 limits the presence of carbon monoxide to:
  - Air: ≤ 5 ml m³
  - Nitrox made with enriched natural air: ≤ 5 ml m³
  - Nitrox made with synthetic air: ≤ 3 ml m³
  - Heliox < 0.2 ml m³
- Gasses should be analyzed before being transferred and connected to the breathing systems.
- According to NORSOK, the maximum Hyperbaric Exposure Limit should be as below:

NORSOK Hyperbaric exposure limits for CO

	Duration of exposure	***Exposure limits***
Breathing gas at work or at rest in bell, chamber, welding habitat etc	*Continuous*	*0,5 Pa (5 µbar)*
	<12 h	*2,0 Pa (20 µbar)*
	<15 min	*5,0 Pa (50 µbar)*
Ambient gas when diver is using breathing apparatus		*10 Pa (100 µbar)*
Breathing gas in emergency situations	*No exposure planned, but system to be active for a minimum of 24 h*	*5,0 Pa (50 µbar)*

- Calculation using the UK HSE exposure limits EH40 and UK HSE study EH75/2 *"Occupational exposure limits for hyperbaric conditions",* already described in the topics "Hydrogen sulfide" and "hydrocarbons" can be done to ensure of another control of the limit of exposure per 24 hours.
  Using the formula *Surface 8-hour TWA in mg.m³ / 5* , we obtain 23 mg.m³ / 5 = 4.6 mg.m³.
- Divex "gas pure" system is designed to remove co from the reclaimed heliox and this system should be always activated and maintained.
- Air compressors' intakes must be away from engine exhausts. Also, the direction of the wind on the worksite should be taken into account. All compressors must also be fitted with carbon monoxide alarms designed to shut them down if such gas is detected.

Protection of people on deck

People should never be exposed to carbon monoxide, even at tiny levels. Thus, the maximum exposure limits should be used only to manage an emergency situation.
Also, note that offshore shifts durations are 12 hours instead of 8 hours. Using UK HSE exposure limits EH40, 8 hours exposure is limited to 20 ppm & 23 mg.m³, and 15 minutes exposure is limited to 100 ppm & 117 mg.m³.
Breathing apparatus must be provided to the essential crew.

Thermal engines' maintenance is crucial. For this reason, all of them should be provided with adequate certificates, and inspections should be carried out at the mobilization and regular intervals. Note that, incomplete combustion levels can be easily detected on engines:

- Appropriate electronic tools can be used for this purpose.
- An engine emitting white or black fumes is obviously not working correctly and has a high potential to emit CO.

Treatment of various diseases using hyperbaric oxygen

Description

The beneficial properties of hyperbaric oxygen are used for purposes other than reducing decompression times and solving diving-related accidents or gasses intoxications, and many scientists highlight the beneficial results of such treatments. It is, for example, the case of a paper called *"Hyperbaric Oxygen Therapy: An Overview"*, written by doctors Gordon Slater, Martin O'Malley, Tayla Slater, and Tandose Sambo. In another article called *"Chronicles of hyperbaric oxygen treatment"*, doctor Tahreem Fatima suggests that such therapies enhance the anti-microbial effects of the immune system and have an additive or synergistic effect with anti-microbial agents.
In their paper entitled *"Hyperbaric Oxygen Therapy: Descriptive review of the technology and current application in chronic wounds",* doctors Babak Hajhosseini, Britta A. Kuehlmann, Clark A. Bonham, Kathryn J. Kamperman, & Geoffrey C. Gurtner, make an assessment of the hyperbaric oxygen treatments approved by the Undersea and Hyperbaric Medical Society (UHMS). The acknowledged applications of these procedures can be listed as follows.
Note that the procedures for resolving decompression sickness, arterial gas embolisms, and CO poisoning intoxications are not in this list as they are already described in the previous section of this book.

- Gas gangrene:
  Gas gangrene is a life-threatening condition. It is most commonly caused by bacteria called "Clostridium perfringens" that gather in an injury or surgical wound and produce toxins that release gas and cause tissue necrosis.
- Acute traumatic peripheral ischemia:
  A traumatic ischemia is a condition of inadequate supply of blood to organs and body tissues as a result of a severe physical injury.
- Crush injuries and suturing of severed limbs
- Acute peripheral arterial insufficiency:
  Acute arterial insufficiency is a condition in which the tissues do not receive enough oxygen and nutrients (ischemia) due to a poor arterial blood supply.
- Progressive necrotizing infections:
  Necrotising infections or "necrotising fasciitis" are life-threatening conditions that can be caused by many bacteria and most commonly by streptococcus type A. Similarly to the diagnosis of gaseous gangrena, the diagnosis of fasciitis at its earlier stage can be difficult although the potentially lethal process is already ongoing and must be treated immediately.
- Preparation and preservation of compromised skin grafts:
  A skin graft is a surgical procedure in which a piece of skin is transplanted from one area to another.
- Chronic refractory osteomyelitis:
  Osteomyelitis is an infection of the bone or marrow caused by bacteria or mycobacteria that can be life-threatening. Refractory osteomyelitis is chronic osteomyelitis that does not respond to treatments.
- Radionecrosis:
  Radionecrosis is a complication of cancer radiation therapy resulting in the death of tissues.
- Cyanide poisoning:
  Known as one of the most powerful and rapidly lethal poisons, cyanide targets the mitochondrial respiratory enzymatic process. Through the ages, criminals have been using cyanide to execute their victims.
- Actinomycosis:
  Actinomycosis is a potentially severe sub-acute to chronic infection caused by the bacteria Actinomyces Israeli. Its diagnosis is often difficult.
- Diabetic wounds of the lower extremities:
  Diabetic wounds form on the feet, ankles, and lower legs due to diabetes damage to blood vessels, which reduces blood circulation and thus deprives these parts of the body of oxygen and other essential nutrients.
- Failure of standard wound therapy
- Osteoporosis:
  "Osteoporosis" means "porous bone". It is a disease that weakens bones, making them fragile and more likely to break. It develops slowly and is often only diagnosed when a fall or sudden impact causes a fracture.
  In a paper called *"Application of hyperbaric oxygen therapy in osteoporosis"*, Doctors Hodor Haidar Hassan, Joelle Azzi, Gian Marco Oppo, Hadi Raef Rida, Roberto Vecchioni, Mohamad Ali Chahrour, Mehdi Raef Rida, Fadel Nahle, Hadi Farhat, Edwin Parra Prada, and Ahmad Najib Ballout, report that twenty osteoporosis patients treated at 1.5 ATA and 100% oxygen for a total of 15 sessions had their average bone mineral density increased by 18 to 19%.

Implementation of the recommended treatments

The treatments indicated on the previous page are usually performed in hospitals under the close surveillance of the doctors who have ordered them. However, the chamber operators of the diving team may be asked to apply such treatment, which of course, should be under the full responsibility of the doctor ordering it and, thus, should not be started without his instruction to proceed.
Note that the US Navy diving manual provides the following guidelines extracted from the Undersea and Hyperbaric Medical Society's Hyperbaric Oxygen Therapy Committee Report-2014 that can be used, depending on the decision of the doctor in charge.

Indication	*Treatment Table*	*Minimum # Treatments*	*Maximum # Treatments*
Gas Gangrene (Clostridial Myonecrosis)	Treatment Table 5	3 times in 24 hours 2 times per day for the next 2-5 days	10
Crush Injury, Compartment Syndrome, and other Acute Traumatic Ischemia	Treatment Table 9	2 times per day for 2-7 days	14
Central Retinal Artery Occlusion	Treatment Table 6	2 times daily to clinical plateau (typically < 1 week) plus 3 days	3 days after clinical plateau
Diabetic Foot Ulcer	Treatment Table 9	Daily for 3-4 weeks, based on healing response	30
Healing of Other Problem Wounds	Treatment Table 9	Daily for 3-4 weeks, based on healing response	60
Severe Anemia	Treatment Table 5 or Table 9 as recommended by the doctor in charge	3-4 times per day until blood replacement by transfusion or regrowth	variable, guided by clinical response
Intracranial Abscess	Treatment Table 9	1-2 times daily for up to 3 weeks	20
Necrotizing Soft Tissue Infection	Treatment Table 9	2 times daily until stabilization	30
Refractory Osteomyelitis	Treatment Table 5 or Table 9 as recommended by the doctor in charge	20-40 treatments	40
Delayed Radiation Injury, Soft Tissue Necrosis, Bony Necrosis	Treatment Table 9	For radiation injury: 30-60 treatments For prophylaxis: 20 treat-ments before surgery in radiated field; 10 sessions after surgery	60
Compromised Grafts and Flaps	Treatment Table 9	2 times daily up to 30 treatments	30
Acute Thermal Burn Injury	Treatment Table 9	2 times daily up to 30 treatments	30
Idiopathic Sudden Sensori-neural Hearing Loss	Treatment Table 9	10-20 treatments	20

Note: The US Navy treatment tables are available at the end of this book

Another relevant document that is highly recommended for Hyperbaric Oxygen Therapy is called *"Tenth European Consensus Conference on Hyperbaric Medicine: Recommendations for accepted and non-accepted clinical indications and practice of hyperbaric oxygen treatment"*. It was written by doctors Daniel Mathieu, Alessandro Marroni, and Jacek Kot. This paper, available on our website, was published in 2017.

Contraindications to hyperbaric oxygen treatments and adverse effects

Contraindications to hyperbaric oxygen treatments are those that are in force for diving-related illnesses and indicated in the *"Handbook on hyperbaric medicine"*, edited by Daniel Mathieu, which is available on our website (https://diving-rov-specialists.com/index_htm_files/scient-c_32-handbook-hyperbaric-medicine-daniel-mathieu_2006.pdf). Thus, untreated pneumothorax (risk of becoming a tension pneumothorax), restrictive airway disorders (air becomes trapped with decompression and can lead to an alveolar rupture with gas expansion), and simultaneous chemotherapy (that may lead to associated morbidity), inability to equalize the middle ear. Claustrophobia is also mentioned by numerous authors.

As suggested above, the document issued after the Tenth European Consensus Conference on Hyperbaric Medicine: *"Recommendations for accepted and non-accepted clinical indications and practice of hyperbaric oxygen treatment"*, provides a relevant extended list of all contraindications.

Doctors Babak Hajhosseini, Britta A. Kuehlmann, Clark A. Bonham, Kathryn J. Kamperman, & Geoffrey C. Gurtner also say that although hyperbaric oxygen therapy remains relatively safe, several adverse side effects have been observed. Reversible myopia has occurred as a direct result of oxygen's impact on the eye's lens, whereas others have experienced barotrauma in the ears and sinuses and, in rare cases, the teeth and lungs. Middle ear barotrauma is among the most common side effects of hyperbaric treatments, affecting up to 2% of the patients undergoing therapy. That can be prevented and managed by auto-inflation techniques and inserting tympanostomy tubes, respectively. Other side effects include chest tightness, coughing, fatigue, headaches, vomiting, and a burning sensation in the chest.
Although undesirable, these effects are reversible and nonfatal, leaving hyperbaric oxygen therapy as a safe adjunctive treatment method for approved morbidities.
Note that decompression sickness may occur in patients breathing compressed air that contains nitrogen. Also, oxygen toxicity is among the more severe complications associated with hyperbaric oxygen treatment. These problems are explained in this book.

Cleaning fluids and other pollutants

Description

A wide range of chemical products is used to perform the maintenance of a diving system. Most of them are sold in the form of solids or liquids able to produce gases classified as “Volatile Organic Compounds”.

- Volatile Organic Compounds include various chemicals, some of which may have short and long-term adverse health effects.
- Paints and cleaning agents all contain organic solvents, as do many disinfecting and degreasing products. These products can release organic compounds while in use, and to some degree, when they are stored.
- Gaseous contaminants can be introduced into the chamber through a contaminated gas supply such as chamber piping or gas storage containers or by the divers and the personnel responsible for the maintenance.
- Also, accidents caused by cleaning fluids left in the system and carried into the diver’s lungs are described by safety organizations such as IMCA and ADCI. These incidents usually result from inadequate procedures or equipment not provided with draining valves in the circuit's lowest points.

Symptoms and effect to health

The ability of organic chemicals to cause health problems varies greatly from those that are highly toxic to those with no known health effect.
As with other pollutants, the extent and nature of the health effect will depend on many factors, including the level of exposure and length of time exposed: Eye and respiratory tract irritation, headaches, dizziness, visual disorders, and memory impairment are among the immediate symptoms that some people have experienced soon after exposure to some organics.
In cases of prolonged exposure, the effects can lead to:

- Nausea, vomiting and abdominal cramps
- Weakness
- Anesthesia
- Blindness
- Coma

Not much is known currently about long-term effects on health arising from low levels of exposure to the organics usually found in workshops and used to maintain the diving systems. Still, many organic compounds are known to cause cancer.

Contaminants that can be found in the gas supply systems and chambers are listed in the US Navy manual revision 7. For more clarification, their description and effect on health have been added. Note that some of them are hydrocarbons. Also, more contaminants than listed here may be present in the diving system as the list focuses on the main ones.

Contaminant	***Description***	***Effects to health***
Acetone	Acetone is produced directly or indirectly from propylene, also known as propene or methyl ethylene, and is the second simplest member of the hydrocarbons' alkene class.	At very high vapor concentrations, acetone is irritating, and, like many other solvents, may depress the central nervous system. It is also a severe irritant on contact with eyes, and a potential pulmonary aspiration risk. In one documented case, ingestion of a substantial amount of acetone led to systemic toxicity, although the patient eventually recovered fully.
Toluene	Toluene is an aromatic hydrocarbon that is widely used as a solvent.	Inhalation of toluene in low to moderate levels can cause tiredness, confusion, weakness, drunken-type actions, memory loss, nausea, loss of appetite, and hearing and color vision loss. These symptoms usually disappear when exposure is stopped. Inhaling high levels of toluene in a short time may cause light-headedness, nausea, or sleepiness. It can also cause unconsciousness and even death

Contaminants which can be found in a gas/air supply and in chambers (continuation)

Contaminant	***Description***	***Effects to health***
Benzene	Benzene is a natural constituent of crude oil. It is a colourless and highly flammable hydrocarbon with a sweet smell. Because it has a high octane value, it is an important component of gasoline.	Human exposure to benzene is a global health problem: - Benzene targets the liver, kidney, lung, heart, and brain and can cause DNA strand breaks, chromosomal damage, etc. - Women who inhaled high levels of benzene for many months had irregular menstrual periods, and their ovaries' size decreased. - Men exposed to high levels of benzene are more likely to have an abnormal amount of chromosomes in their sperm, impacting fertility, and foetal development. - Benzene causes cancer: Substantial quantities of epidemiologic, clinical, and laboratory data, link benzene to aplastic anemia, acute leukemia, and bone marrow abnormalities.
Trimethyl-benzenes	The Trimethyl-benzenes constitute a group of aromatics hydrocarbons whose structure consists of a benzene ring with three methyl groups as a substituent	Trimethyl-benzenes are irritating to the skin, and breathing the vapour is irritating to the respiratory tract causing pneumonitis. Breathing high concentrations of chemical vapour causes headache, fatigue, and drowsiness.
Xylenes	Xylenes are aromatic hydrocarbons consisting of a benzene ring with two methyl substituents These mixtures are slightly greasy, colourless liquids commonly encountered as solvents.	Exposure may irritate the nose, throat, lungs, and respiratory tract. Central nervous system (brain) effects may include headache, dizziness, loss of balance and coordination, unconsciousness, coma, respiratory failure, and death. Effects on the blood (including decreased platelet and white blood cell counts), cardiovascular system, nervous system, retina, lungs, gastrointestinal system, spleen, and kidneys have been reported from large, acute (short), and repeated or prolonged exposures.
Ethanol	Ethanol, also called ethyl alcohol, pure alcohol, grain alcohol, or drinking alcohol, is a volatile, flammable, colourless liquid with the structural formula CH_3CH_2OH, often abbreviated C_2H_5OH or C_2H_6O. It is a psychoactive drug and one of the oldest recreational drugs known. It produces a state known as alcohol intoxication when consumed as a beverage. It is also used in thermometers, as a solvent, and as a fuel. In common usage, it is often referred to simply as alcohol or spirits.	Ethanol is a central nervous system depressant and has significant psychoactive effects in sub-lethal doses. It is considered a psychoactive drug. Death from ethanol consumption is possible when the blood alcohol level reaches 0.4%. A blood level of 0.5% or more is commonly fatal. Levels of even less than 0.1% can cause intoxication, with unconsciousness often occurring at 0.3 to 0.4%.
Chloroform	It is a clear, colourless, heavy, sweet-smelling liquid, used in refrigerants, propellants, and resins, as a solvent, and sometimes as an anesthetic. Once widely used in human and veterinary surgery, chloroform has generally been replaced by less toxic, more easily controlled agents.	Chloroform was a widely used anaesthetic. However, its vapours depress the patient's central nervous system. It was quickly abandoned due to its tendency to cause fatal cardiac arrhythmia analogous to what is now termed "sudden sniffer's death".
Methanol	Methanol, also known as methyl alcohol, wood alcohol, wood naphtha, or wood spirits, is a chemical with the formula CH_3OH. Methanol acquired the name "wood alcohol" because it was a byproduct of wood's distillation. Modern methanol is produced directly from carbon monoxide, carbon dioxide, and hydrogen in a catalytic industrial process. It is a light, volatile, colourless, flammable liquid with a distinctive odour very similar to, but slightly sweeter than, that of ethanol (drinking alcohol). It is used as an antifreeze, solvent, fuel, and as a denaturant for ethanol.	Workers repeatedly exposed to methanol have experienced several adverse effects. Effects range from headaches to sleep disorders and gastrointestinal problems to optic nerve damage.

Contaminants which can be found in a gas/air supply and in chambers (continuation)

Contaminant	***Description***	***Effects to health***
Freon	The name "Freon" is a trademark registered by E.I. du Pont de Nemours & Company. They are fluorinated aliphatic organic compounds that have a low boiling point, low surface tension, and low viscosity. They are colourless, odourless, non-flammable, non-corrosive gases or liquids of low toxicity introduced as refrigerants. They are also used as propellants for aerosols and in numerous technical applications. Because they are stable, inert compounds, they are also used as cleaning agents. Freons often contain hydrogen, chlorine, or bromine, in addition to fluorine and carbon.	Overexposure to Freon may cause dizziness, loss of concentration, central nervous system depression, and/or cardiac arrhythmia. Vapours displace air and can cause asphyxiation in confined spaces. Although non-flammable, their combustion products include hydrofluoric acid and related elements. *Freon is a hydro-chlorofluorocarbon, which new production and import were banned in 2020.*
Methyl Chloroform	Trichloroethane, also known as methyl chloroform, is a chloroalkane. It is colourless, sweet-smelling liquid used as a solvent. It is classified as an ozone-depleting substance.	Methyl chloroform is corrosive to the eyes, skin, and mucous membranes of the respiratory tracts. Eye and skin contact with the liquid can result in severe burns. Accidental ingestion causes burns of the gastrointestinal tracts.
Methyl Ethyl Ketone	Methyl ethyl ketone, also known as Butanone or MEK, is an organic compound with the formula $CH_3COCH_2CH_3$ or C_4H_8O. It is a colourless liquid with a sharp, sweet odour reminiscent of butterscotch and acetone. It is produced industrially on a large scale, and also occurs in trace amounts in nature. It is soluble in water and is commonly used as an industrial solvent.	Methyl Ethyl Ketone can irritate the nose and throat, and can harm the nervous system. Symptoms may include headache, nausea, dizziness, drowsiness and confusion. A severe exposure can cause unconsciousness. It may cause mild skin irritation and can be absorbed through the skin, but harmful effects are not expected. It causes moderate to severe irritation to the eyes. The symptoms include sore, red eyes, and tearing.
Methyl Isobutyl Ketone	Methyl isobutyl ketone (MIBK) is an organic compound manufactured from acetone. It is used as a solvent for nitrocellulose, lacquers, and certain polymers and resins.	Acute exposure to methyl isobutyl ketone can irritate the eyes and respiratory tracts and cause narcosis at high air concentrations.
Methylene Chloride	Methylene chloride or Dichloromethane (DCM) is an organic compound with the formula CH_2Cl_2, is widely used as a paint stripper, degreaser, aerosol spray propellant, and blowing agent for polyurethane foams. It is a colourless volatile liquid with a moderately sweet aroma Although it is not miscible with water, it is miscible with many organic solvents.	Methylene chloride is a potential occupational carcinogen. Short-term exposures to high concentrations may cause mental confusion, headache, lightheadedness, nausea, and vomiting. Continued exposure may also cause eye and respiratory tract irritation. Skin exposure to its liquid form may cause irritation or burns.
Isopropyl Alcohol	Isopropyl alcohol is a common name for a chemical compound with the molecular formula C_3H_8O or C_3H_7OH. It is a colourless, flammable chemical compound with a strong odour. Isopropyl alcohol is commonly used as a disinfectant, antifreeze, and solvent and typically comprises 70 percent of "rubbing alcohol".	Isopropyl alcohol is a sedative-hypnotic agent. If ingested, isopropyl alcohol functions primarily as a central nervous system (CNS) inebriant and depressant, and its toxicity and treatment resemble that of ethanol. The precise mechanism of action in the central nervous system remains uncertain. Changes in membrane fluidity and/or function, and interactions with neurotransmitter receptors, are believed to account for the CNS effects of alcohols and other simple hydrocarbons.

Notes:

The US Navy diving manual revision 7 lists refrigerants such as "Freon 11 (Trichlorofluoromethane)", "Freon 12 (Dichlorodifluoromethane)", and "Freon 114 (1,2-Dichlorotetrafluoroethane)" that are classified among the ozone-depleting agents, and no more produced since 1996. Considering that their production has been halted for more than 20 years, the probability of finding them is extremely low, and they have not been referenced in the list above.
However, "Freon" is listed here, although it is a hydro-chlorofluorocarbon that has been banned in 2020 for the same reasons, because there is the possibility that this compound will be found during the next 10 years.

Treatment

The treatment includes the removal of the patients from the source of contamination and the support of their vital functions.

Removal from source of contamination:

- Identify the source of pollution, Stop the gas supply affected and switch to backup.
- Evacuation of the polluted area if the problem is from another source than breathing gas.
- In the case that the source of contamination cannot be eliminated, the diving operations must be halted. Divers decompressing in a polluted chamber should be provided with clean breathing medium through the Built-In Breathing System (BIBS).
- Decontamination of the element or the area polluted should be undertaken. However, in the case of contaminated elements that are part of the breathing system, the wet bell, or the chamber (example: paint), that may not be possible offshore.

Support the contaminated people:

- Contact the Diving Medical Specialist.
- Breathing (Therapeutic mix) and circulatory support.
- Vital sign monitoring.
- Fluid therapy.
- Sampling of the contaminated unit to identify the cause of the contamination.
- Protection of the divers not affected (Breathing masks), and regular reports.
- MEDEVAC as soon as possible (to be decided and organized by the diving medical specialist).

Evaluation of the contamination:

When the source and the nature of the contamination is identified, the team can evaluate the problem posed by the exposure to the contaminant. Two methods can be used for this purpose:

1 - The US Navy manual revision 7 provides maximum exposure values of the compounds described previously. Note that US Navy says that these exposure calculations are based on those of the following administrations:

- The US National Research Council Committee on Toxicology Emergency and Continuous Exposure Limits for Selected Airborne Contaminants.
- The US National Aeronautics and Space Administration, and the US Office of Space Transportation Systems.
- The U.S. Naval Sea Systems Command Nuclear Powered Submarine Atmosphere Control Manual, NAVSEA S9510-AB-ATM-010 (U), Vol. 1, Revision 2, 30 July 1992.

Substance	*ppm max*	*Exposure limit*	*Substance*	*ppm max*	*Exposure limit*
Acetone	200 ppm	90 days	Methanol	10 ppm	90 days
Toluene	20 ppm	90 days	Methyl Chloroform	30 ppm	90 days
Benzene	1 ppm	7 days	Chloroform	1 ppm	90 days
Trimethyl-benzenes	3 ppm	7 days	Methyl Ethyl Ketone	2.5 ppm 10 ppm	90 days 1 day
Xylenes	50 ppm	90 days	Methyl Isobutyl Ketone	20 ppm	7 days
Ethanol	100 ppm	7 days	Methylene Chloride	25 ppm	90 days
Freon	100 ppm	90 days	Isopropyl Alcohol	1 ppm	90 days

2 - Another method of evaluation is to use the limits for the control of hazardous substances at work set up by national safety organizations that are already discussed in the topics “hydrocarbons”, “Hydrogen sulfide”, and “carbon monoxide”.
As a reminder, these limits are concentrations of hazardous substances in the air, averaged over a specific period referred to as a time-weighted average (TWA) in the UK HSE document EH40. This publication provides a list of contaminant and two types of maximum exposure.

- Short-Term Exposure Limits (STEL) are set to prevent acute effects such as eye irritation, which may occur

- Long term exposure limits (LTEL) are set to prevent effect that may not be noticeable during the intervention but may appear as a result of long and repeated exposures. They are normally limited to 8 hours.
  Note that the "8-hour reference period" refers to occupational exposures within a 24-hour period. It can be a single uniform exposure for 8 hours (the 8-hour time-weighted average (TWA) exposure) or an addition of several short exposures. The calculation proposed is: *Time in decimals x concentration in mg.m³ / 8*
  Note that time in decimal consists in dividing the minutes by 60 and multiply them by 100. Example: 7:20 hours = 7.33.

Note that the document EH40 is available free of charge on the UK HSE and the Diving and ROV specialists websites.
Note that a lot of substances have several names, and are usually listed under only one of them: That obliges to find the name used through the list of synonyms of the document or through the Internet.

Workplace exposure limits (surface) document UK HSE EH 40

	Long-term exposure limit (8-hr TWA reference period)		***Short-term exposure limit (15 minutes reference period)***	
Substances	***ppm***	***mg.m³***	***ppm***	***mg.m³***
Acetone	500	1210	1500	3620
Toluene	50	191	100	384
Benzene	1	3.25	—	—
Trimethylbenzenes	25	125	—	—
Xylenes	50	220	100	441
Ethanol	1000	1920	—	—
Freon *(Chlorodifluoromethane)*	1000	3590	—	—
Methanol	200	266	250	333
Methyl Chloroform *(1,1,1-Trichloroethane)*	100	555	200	1110
Chloroform	2	9.9	—	—
Methyl Ethyl Ketone	—	—	0.2	15
Methyl Isobutyl Ketone *(4-Methyl-pentan-2-one)*	50	208	100	416
Methylene Chloride *(Dichloromethane)*	100	353	200	706
Isopropyl Alcohol *(Propan-2-ol)*	400	999	500	1250

Important rule:
We must consider that the procedures above should not be applied to solve a known situation linked to a problem of maintenance before launching the dive, but only to control an emergency.
As indicated in the chapters regarding hydrocarbons or H2S exposures, the only acceptable level of pollutants in the breathing systems and the chambers should be zero.

Prevention

The best prevention consists of hygiene, method, and seriousness of people:

- The diving system's cleanness must be checked following maintenance. That should be done using various complementary procedures:

  - Technicians should look for traces of pollutants on the components of the system using appropriate complementary processes. For example, Hydrocarbons fluoresce under the UV light, so they can be detected using a long-wave UV light (3600-3900 Angstrom). However, most synthetic oils and greases do not fluoresce, and other compounds are not detected using this method. That is why this system of detection cannot be used alone but associated with others such as “direct visual inspection”, “wipe test” , “water break/ink test”, “odour test”, “Chromatography”, “spectrometry”, and other detection methods.
  - Gas analysis should always be performed: Chemical tubes similar to those proposed by the company Dräger can detect contaminants in chambers and the gas distribution system. Also, manufacturers are developing new ranges of electronic analysers able to detect tiny levels of harmful pollutants.
- Strict cleaning procedures should be established and followed. As a reminder, they are indicated through this manual. For example, Procedures for cleaning the gas distribution systems are indicated in point 1.2.6.10 “Maintenance” of the chapter “Gas storage and distribution” In book #2; the pre-dive preparation of the chambers is indicated in point 3.1 of Book #2; bell maintenance as indicated in point 1.1.2.9 of Book #3; etc. Cleaning procedures are also published by equipment and cleaning agents manufacturers.
- Chamber and bell contamination can be prevented by the strict control of the substances used for maintenance and the items that may be taken inside. Only approved paints, lubricants, solvents, glues, equipment, and other materials are known not to give off potentially toxic contaminants are allowed in chambers and for gas systems maintenance.
- It is essential to ensure that residues and contaminants have been entirely removed before returning the diving system to operations after performing its maintenance. That includes ensuring that even tiny traces of cleaning substances and other harmful compounds have been removed. It must be kept in mind that breathing systems and chambers may be contaminated by the addition of various agents whose concentrations are not harmful if considered separately.
- Another method to prevent chamber contamination is the drastic control of the objects introduced in the system by the divers. As an example, in addition to their flammability, most plastics contain Volatile Organic Compounds (VOCs), which are carbon-containing chemicals that are volatile enough to evaporate at room temperature, which may contribute to polluting the chamber atmosphere.

Contamination by chemicals and radiation

Description

Contaminated water is defined as water that contains any chemical, biological, or radioactive substance that poses a chronic or acute health risk to exposed personnel. Apart from hydrocarbons (discussed in a specific point of this book), contaminations may be due to the presence of chemicals, heavy metals, radioactivity, and biological materials. These can occur naturally or from a variety of sources like leaking vessels, industrial discharges, and/or sewer effluent.

The effects on personnel may become evident immediately (acute) or delayed for many years (chronic), depending on the substance, the routes of entry into the body, and the time and frequency of exposures.

Chemical contaminants (Other than hydrocarbons)

Chemical contaminants can be very corrosive and harmful and cause people illness, injury, disability, or death. Some chemical contaminants easily blend with water and enter the body by ingestion, inhalation, or contact with the skin.

The primary sources of industrial chemical contamination are industrial spills, urban and industrial sewage, commercial ships, hazardous waste sites, and agricultural runoff.

More than 5,000,000 chemicals are referenced, but little to no information is available on either the acute or chronic toxicity of these chemicals or their environmental fate.

Heavy metals

Heavy metal contamination is normally encountered as a result of past industrial activity. Examples of such substances that may be encountered are mercury, cadmium, and arsenic, although other substances may also be found.

Heavy metals do not normally have acute (sudden) effects but tend toward chronic (long term) health effects. Skin contact will normally have to be repeated and prolonged to become a serious hazard. Toxic vapours that may contaminate the surrounding are usually limited. The main risk with heavy metals is if they are ingested, which means that the risk of contamination of divers at work is normally relatively small.

Radioactive substances

In most countries, massive radioactive contamination could occur only due to the wreckage of a vessel transporting such materials, as nuclear facilities are found in countries with the resources to implement these technologies, representing 41 states of the 197 recognized by the United Nations. However, that does not exclude pollution following an accident such as Mayak, Chernobyl, or Fukushima, resulting in massive pollution by radioactive substances. More linked to everyday activities, contamination to divers and other personnel can happen to those manipulating NDT equipment employed to visualize the internal structures of solid and hard materials using X and gamma radiation. Also, small quantities of solid, naturally occurring radioactive materials can be generated by petroleum production facilities and associated pipes, flow lines, and other equipment. Exposure to ionising radiation can cause serious injuries and chronic illness.

Symptoms

Contamination by chemicals:

Acute and chronic symptoms will depend on elements like the pollutant's nature, the concentration in the waters, and the duration and frequencies of exposure. Visible acute symptoms can be among those described below:

Upper respiratory flu or cold-like symptoms

- Coughing
- Vomiting
- Diarrhea
- Pneumonia
- Headaches
- Dizziness
- Skin lesions
- Headache
- Visual disturbances
- Paralysis,
- Convulsions

- Loss of conscious

In addition to the above, some of the symptoms described below can happen later on:

- Fatigue
- Trouble with fertility
- Cancer
- Loss of memory
- Central Nervous System impairments

Note that similar acute and chronic symptoms are noticeable after infection by toxins. Exposure to heavy metals can also trigger similar chronic symptoms.

Exposure to radiation sources

The sickness usually begins when a person has been exposed to a very high dose of radiation that is able to penetrate his body, thus reaching the internal organs, in a short span of time (usually within minutes). The damages to cells caused by ionizing radiation are the alteration of the genetic code (DNA). Moreover, high radiation doses (particularly over a short period of time) tend to kill cells. In fact, high doses can sometimes kill so many cells that tissues and organs are damaged immediately.

The magnitude of the dose is the single most determining factor for the health impact. The following exposure levels are indicative of the seriousness of radiation exposure:

- A high dose (500 rad and above) of radiation to the whole body in a short amount of time means that death is the most likely outcome within a few days to a few weeks.
- A moderate dose (< 500 rad) of radiation can cause the symptoms to appear within a few hours or days following exposure. The symptoms will develop predictably, and there is a good chance of survival, especially with prompt medical care. Such exposure is likely to increase the likelihood of cancer later in life.
- A low dose (< 5 rad) of radiation means that there will be no radiation sickness following and probably no increased likelihood of observable health effects later in life. However, there may be an elevated risk of cancer when compared to the average population.

Nausea and vomiting are typically the earliest symptoms of acute radiation sickness after exposure to a high radiation dose. The higher the dose of radiation, the sooner and the worse these symptoms are. They can occur between 2 and 12 hours after exposure. Sometimes people with radiation sickness feel bad at first and then start to feel better. Generally, the symptoms listed below appear within hours, days, or even a few weeks:

- Diarrhea
- Skin burns (skin reddening)
- Weakness
- Lethargy and fatigue
- Loss of appetite (anorexia)
- Fainting
- Dehydration
- Inflammation of tissues (swelling, redness, or tenderness)
- Hemorrhages under the skin
- Bleeding from the nose, gums, or mouth
- Anemia
- Hair loss (usually from just the scalp)
- Decrease in platelets
- Depending on the dose absorbed, death can happen quickly…

As with other diseases, some people can manage massive amounts of radiation with no ill effects.
The severity of symptoms and illness (acute radiation sickness) depends not only on the type and amount of radiation, but also on which part of the body was exposed.

Symptoms of chronic effects, like certain forms of cancer, can happen several years after the exposure. This is likely to happen for those exposed to a smaller amount of radiation repeatedly, like those in direct contact with an accumulation of NORM (Naturally Occurring Radioactive Material) when maintaining petroleum facilities.
Various cancers have been associated with exposure to ionizing radiation, including leukaemia, cancers of the lung, stomach, oesophagus, bone, thyroid, and the brain and nervous system. It is important to understand that the potential health effects are strongly dose-related. Also, based on extensive scientific studies over many decades, radiation exposure is not associated with all forms of cancer.

Treatment

Contamination by chemicals or toxins

The onsite treatment is similar as for contamination by hydrocarbons:

- Remove the patient from the poisoned environment.
- Decontamination
- Contact the Diving Medical Specialist
- Breathing and circulatory support.
- Vital sign monitoring.
- Fluid therapy
- Medical Evacuation as soon as possible.
- A sample of the water and the mud from the bottom can be sent to a specialized laboratory to identify the contamination.

A precise description and history of the job site and the weather conditions before and during the operations can help identify the chemical contaminant or toxin. It is a good practice to introduce these elements in the incident / medical report.

Personnel contaminated by radiation (acute)

- Contact with the diving medical specialist
- The patient's decontamination includes removing the clothes, washing, and segregation of the clothes that could be infected. (The contaminated elements to be segregated in a specific container)
- Support of the vital functions
- Medical evacuation to a specialized medical unit.
- If the diver is in the saturation system and cannot be evacuated immediately, depending on the contamination and the decision of the diving medical specialist, a complementary treatment may include:
  - Treatment for damaged bone marrow by using proteins that promote white blood cells' growth, and may counter the effect of radiation sickness of bone marrow and transfusions of red blood cells or blood platelets.
  - Treatment for internal contamination, which reduces damage to internal organs caused by radioactive particles and speeds up the elimination of these radioactive particles.
  - Supportive treatment to treat infections, Headache, fever , diarrhea , vomiting, burns, etc.
  - Psychological support
  - End of life care for those who have absorbed too large a dose of radiation (500 rad and over).

Prevention

Diving procedures should not be used in the presence of agents that can contaminate the divers. The precautions referring to contamination indicated in this chapter and the previous chapters are the procedures to implement in accidental contamination only.
In case of exposure of life to danger, the ROV becomes the preferred means of intervention.

Offshore

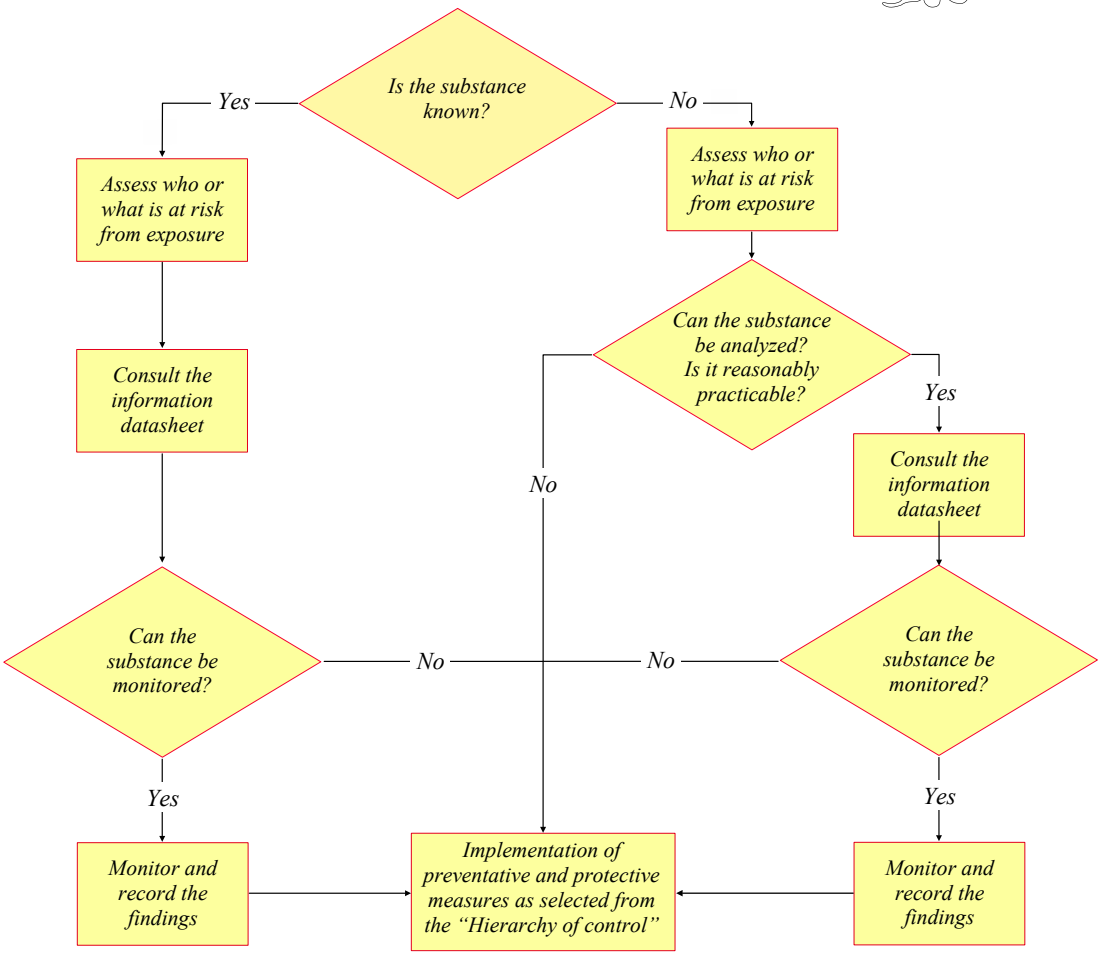
A sample of the bottom must be analyzed to detect harmful products prior to interventions near facilities where pollution is suspected.

In rivers and lakes

Remember that diving operations can be organized in lakes, deep rivers, estuaries, etc. It is essential to analyze water and mud samples to be aware of the pollutants that may be present.

- The observation of the river banks and/or of the quays of the port or/and the beaches can give some information concerning the possible dumping of industrial chemicals.
- Public services can transmit some information regarding the chemical agents found in rivers and then dumped to the sea. Still, they have to be considered with caution as contamination levels can change from the time the investigation has been performed.
- Note that the fact some local people are sometimes drinking the water from the river they are living along is not a reliable indicator that there are no chemicals in the water

A risk assessment must be carried out to decide whether the intervention using divers is safe or not on sites where chemicals, pathogens, and any other agents which could harm the health of the divers is suspected. The chart displayed on the next page, which is also used for hydrocarbons should be used for this purpose.



Complementary guidelines regarding exposure of personnel to radiations

Radiation sources like some Non-Destructive Technics (NDT) equipment, the maximum time of exposure is normally indicated by the manufacturer. Still, the team must limit the exposure and organize to restrict access to areas where the inspection is carried out and where these materials are stored.
For information, the occupational limit of exposure per year for an adult (the limit for a worker using radiation) is "as low as reasonably achievable"; however, it is not to exceed 5,000 millirems (5 rad) above the 300 millirems coming from natural sources of radiation and medical radiation.
Radioactive materials used by Non-Destructive Technics (NDT) devices should normally not affect the divers during the dives. However, they can affect them and their supporting team if they are inappropriately stored. For this reason, national agencies require strict storage procedures:

- Radioactive materials must not be transferred from one container to another except by procedures approved by the statutory authority.
- The store must be constructed of materials of sufficient durability and strength to resist fire and unauthorised entry. It must be under the control of a competent person and be kept locked except when removing or replacing sources. The dose rate outside the store must be as low as reasonably achievable and such that no member of the public receives a dose exceeding the maximum limit of exposure per year.
- The store must not be located in proximity to explosives, combustible, corrosive materials, or any element that is part of the saturation system. It must have a conspicuous notice bearing the word 'CAUTION' and a radiation hazard warning symbol and lettering that makes clear that it is a store for radioactive substances. Apart from any emergency exits normally kept closed outside, a store should have only one door for access so that it may not be used as a thoroughfare.

NORM (Naturally Occurring Radioactive Material) radiation is not considered harmful for divers under normal conditions due to their small values and the limited exposure time underwater. For example, the document IMCA SEL 024 "Guidance on Handling Naturally Occurring Radioactive Materials" says: *"Large radiation doses are very unusual and are unlikely to occur even under accident conditions. There is no possibility of anyone working with NORM receiving doses that could cause any health effects in the short term. Working with NORM will not cause sickness, nor can it cause coughs, rashes or burns. The risks involved in working with NORM are very low when compared with other every day risks."*
However, other documents say that the level of NORM accumulation can vary substantially from one facility to another depending on geological formation, operational and other factors.
For this reason NORM survey, sampling and analysis should be conducted with an analysis of the risk of contamination of the equipment and the possible spreading of contamination to the diving bell and then into the chamber.

Pathogen attacks

Pathogen diseases transmitted to the divers by contact with the skin during a dive are already described in the previous point. For this reason, the following description takes more categories of pathogens into account, as apart from skin contact, pathogens can be transmitted by ingestion, contact of contaminated hands or objects with the mouth, pricks of infected transporters such as mosquitos, pricks with contaminated objects, or by inhaling infected droplets dispersed by sneezing or coughing.

Classification of pathogens

Viruses

Viruses are the smallest of all biological agents. They are incapable of independent life since they are only genetic material and some encapsulating proteins. They can be very dangerous when they enter a human cell and corrupt its contents to reproduce themselves because they are difficult to detect and treat.
Some virus protection can be gained by prior immunization, but there are no vaccines for most viruses.
Examples of viruses are displayed in the table below.

Name	*Intoxication process*	*Symptoms*
Adenovirus	This virus are spread by having direct contact with an infected person, by using a contaminated object, or by inhaling infected droplets dispersed by sneezing or coughing.	It causes respiratory illness, but may also cause gastroenteritis, conjunctivitis, cystitis (bladder infection) and rashes.
Coronaviruses	These virus are spread by having direct contact with an infected person, by using a contaminated object, or by inhaling infected droplets dispersed by sneezing or coughing.	Cause respiratory tract infections that can range from mild to lethal. Mild illnesses in humans include some cases of the common cold, while more lethal varieties can cause Evere Acute Respiratory Syndromes (SARS), Middle East respiratory syndrome (MERS), and COVID-19. SARS produces flu-like symptoms and may include fever, muscle pain, lethargy, cough, sore throat, and other nonspecific symptoms. MERS produces similar symptoms to SARS with shortness of breath. COVID-19 Symptoms include fever, cough, fatigue, breathing difficulties, and loss of smell and taste that start between one to fourteen days after exposure to the virus. Around one in five infected individuals do not develop any symptoms. While most people have mild symptoms, some people develop acute respiratory distress syndrome, multi-organ failure, septic shock, and blood clots.
Dengue	This virus, spread through the bite of an infected Aedes aegypti mosquito	Symptoms include high fever, rashes, severe headache, pain behind the eyes, and muscle and joint pain, which can be very severe.
Hepatitis A	Ingestion of contaminated food or fresh water	Symptoms are only acute (no chronic stage to the virus) and include fatigue, fever, abdominal pain, nausea, diarrhea, weight loss, itching, jaundice and depression.
Hepatitis E	Ingestion of contaminated food or fresh water	Symptoms include fatigue, fever, abdominal pain, nausea, diarrhea, Dark urine, clear feces, itching, jaundice, skin rash or itching, Joint pain.
Herpes	There are eight types of herpes viruses, out of over 100, that routinely affect humans: Common infection is through contact with the saliva, cold sores or mouth surface of an infected person.	This virus commonly causes cold sores or ulcers in or around the mouth.

Name	*Intoxication process*	*Symptoms*
Human immunodeficiency virus (HIV)	HIV is transmitted by sexual contact, blood transfusion, contaminated needles and during delivery of a newborn.	It attacks our immune cells (CD4+) and lowers its count progressively, resulting in the patient becoming immunocompromised, or too weak to fight off normal infections.
Influenza *(Commonly called "flu")*	These virus are spread by having direct contact with an infected person, by using a contaminated object, or by inhaling infected droplets dispersed by sneezing or coughing.	Symptoms can be mild to severe and typically begin two days after exposure. The most common symptoms include: High fever, runny nose, sore throat, muscle and joint pain, headache, coughing, and feeling tired. With mild cases, these symptoms last within 15 days. Complications may include viral pneumonia, secondary bacterial pneumonia, sinus infections, and worsening of previous health problems such as asthma or heart failure.[
Measles	Transmitted through contact with droplets from an infected person when they cough or sneeze.	Fever, cough, runny nose and inflamed eyes. A red, flat rash usually starts on the face, then spreads to the rest of the body.
Mumps	Transmitted through contact with droplets from an infected person when they cough or sneeze.	Swollen and tender salivary or parotid glands, difficulty chewing, fever, headache, muscle aches and loss of appetite.
Noroviruses	Ingestion of infected food water or contact with infected objects.	Diarrhea; Vomiting; Nausea; Stomach pain. Develops 12 to 48 hours after being exposed, and ends within 1 to 3 days.
Poliomyelitis	Ingestion of water contaminated by the faeces of contaminated individuals	90-95% of patients show no symptoms, 4-8% have minor symptoms (comparatively) with delirium, headache, fever, and occasional seizures, and spastic paralysis, 1% have symptoms of non-paralytic aseptic meningitis. The rest have serious symptoms resulting in paralysis or death.
Polyomavirus infection	Very widespread, can be found in fresh water. Transmission by ingestion	The virus produces a mild respiratory infection and can infect the kidneys and the respiratory system. Can also cause damages in the brain (which is fatal).
Rotaviruses	Mainly transmitted through faeces. Transmission by ingestion of contaminated food.	Symptoms usually start about 2 days after exposure with vomiting, watery diarrhea, loss of appetite, dehydration, and can last 3 to 8 days.
Sapovirus	Transmitted through oral-faecal contact, most commonly with children and infants	Symptoms commonly include diarrhea and vomiting

Bacteria and fungi

Bacteria *(singular: bacterium)* are microscopic, single-celled organisms that live in diverse environments. Unlike viruses, they are capable of reproduction outside living cells. If they enter the body, the microorganism will multiply and incapacitate the host. Bacteria can be found in almost any environment and sometimes concentrate in a thin layer on the water surface or a thin layer on the top of the sediment. Bacteria reproduce by binary fission. In this process the bacterium divides into two identical daughter cells.

Fungi *(singular: fungus)* are multi-cellular organisms classified into a separate group from both plants and animals. However, some scientists say that they are often more closely related to animals than plants. They cannot produce their own food. Thus, they acquire nutrients from the animals, plants, or decaying matter on which they live.
They are metabolically versatile and can grow on diverse organic substrates. As a result, they interact with other organisms by either forming beneficial or mutualistic associations or causing infections.
Document DMAC 26 says that some fungi are normally present on human skin, and these are more likely to cause superficial infections in saturation conditions
Fungi reproduce by forming and releasing vast quantities of spores produced either by asexual or sexual methods.
Asexual reproduction is the process used by bacteria that consists of the fragmentation of an existing cell to produce a new cell.
Sexual reproduction is the process in which new organisms are created by combining the genetic information from two individuals of different sexes.

The table on the next page lists some common infections from bacteria and fungi:

Disease	*Agent classification*	*Intoxication process*	*Symptoms*
Bacillus cereus	Bacterium	Commonly found in the soil as well as a variety of foods.	Nausea, vomiting and abdominal cramping occur within 1–5 hours of ingestion, with recovery usually within 6–24 hours
Campylobacter jejuni	Bacterium	Ingestion of undercooked meat, including seafood, and drinking untreated water.	Abdominal pain, fever, headache, nausea, and/or vomiting. The symptoms typically last 3 to 6 days. Complications such as bacteraemia (presence of bacteria in the blood), hepatitis, pancreatitis (infections of liver and pancreas, respectively), and miscarriage have been reported
Clostridium perfringens	Bacterium	Meet, gravies, and dried or pre-cooked foods are common sources. The infection often occurs when food is kept warm for a long time before serving.	Diarrhea and abdominal cramps within 6 to 24 hours. The illness usually begins suddenly and lasts for less than 24 hours. People infected usually do not have fever or vomiting.
Shiga toxin-producing E. Coli	Bacterium	Lives in the intestines of healthy cattle. Infection occurs by eating contaminated food, particularly raw or undercooked meat, or contaminated produces, including lettuce, alfalfa sprouts, salami, and unpasteurized milk, juice, or cider.	Often severe diarrhea, stomach cramps,vomiting, bloody diarrhea, fever. These symptoms typically appear 3-4 days after contamination, and can range from 1-10 days.
Enterotoxigenic E. Coli	Bacterium	food, drinks water or ice contaminated with human or animal wastes	Watery diarrhea, abdominal cramping, fever, nausea with or without vomiting, chills, and appetite loss. Headache, muscle aches, and bloating can also occur.
Nontyphoidal Salmonella	Bacterium	Direct and indirect contact with infected animals and the foodstuffs derived from them, and water are common sources of contamination	Usually starts 12 to 48 hours after ingestion with nausea, cramping abdominal pain followed by diarrhea, fever, and sometimes vomiting. The disease is usually mild, lasting 1 to 4 days.
Salmonella Typhi	Bacterium	Infect the intestinal tract and the blood. Transmitted by food or drink contaminated with faeces.	Usually begin 6 to 30 days after exposure with gradual onset of a high fever, accompanied by weakness, abdominal pain, constipation, headaches, and mild vomiting over several days. Skin rash with rose-coloured spots may be apparent.
Shigella species	Bacterium	It is only naturally found in humans and gorillas. infection is typically by ingestion. During infection,	Typically causes dysentery. Common symptoms are diarrhea, fever, nausea, vomiting, stomach cramps, and flatulence, and large and painful bowel movements.
Staphylococcus aureus	Bacterium	These bacteria are spread by having direct contact with an infected person, by using a contaminated object, or by inhaling infected droplets dispersed by sneezing or coughing. Frequently found in the upper respiratory tract and on the skin. It can grow without the need for oxygen.	Can cause minor skin infections, such as pimples, impetigo, boils, cellulitis, folliculitis, carbuncles, scalded skin syndrome, and abscesses. It can also cause life-threatening diseases such as pneumonia, meningitis, osteomyelitis, endocarditis, toxic shock syndrome, bacteremia, and sepsis. It is often the cause of wound infections following surgery.
Streptococcus pyogenes	Bacterium	Transmission by inhalation of respiratory droplets, skin contact, contact with objects, surface, or dust that is contaminated, or through food.	It colonizes the throat, genital mucosa, rectum, and skin. It causes streptococcal pharyngitis, rheumatic fever, rheumatic heart disease, and scarlet fever.
Botulism	Bacterium	Can enter in an open wound from contaminated water sources or by ingestion. It is present in the marine and freshwater sediments of wetlands, rivers, and lakes.	Dry mouth, blurred and/or double vision, difficulty swallowing, muscle weakness, difficulty breathing, slurred speech, vomiting, and sometimes diarrhea. Death is usually caused by respiratory failure.

Disease	*Agent classification*	*Intoxication process*	*Symptoms*
Yersinia enterocolitica	Bacterium	Ingestion of undercooked pork meat contaminated with the bacteria.	Fever, abdominal pain, bloody diarrhea, and pain on the right side of the abdomen. May be confused with appendicitis.
Tetanus	Bacterium	Emit spores that are found everywhere, particularly in soil, ash, faeces of animals and humans, and on the surfaces of skin and rusty tools like nails, needles, barbed wire, etc. They are resistant to heat and most antiseptics, and can survive for years.	Jaw cramping or the inability to open the mouth; Muscle spasms often in the back, abdomen, and extremities; Sudden painful muscle spasms often triggered by sudden noises; Trouble swallowing; Seizures; Headache; Fever and sweating; Changes in blood pressure or fast heart rate. Death can occur
Campylobacteriosi	Bacterium	Water contaminated with faeces (ingestion)	Diarrhea with blood, accompanied by extreme abdominal pain, rectal pain along with a high fever. Usually lasts 2–10 days.
Cholera - Vibrio cholera	Bacterium	Ingestion of contaminated food & fresh water	In severe forms, it is known to be one of the most rapidly fatal illnesses known. Symptoms include very watery diarrhea, nausea, cramps, nosebleed, rapid pulse, vomiting, and hypothermic shock (in severe cases), at which point death can occur in 12–18 hours.
Coliform / E-coli	Bacterium	Ingestion of contaminated food or fresh water	Mostly diarrhea. Can cause death due to dehydration from prolonged illness.
Dysentery	Bacterium	Ingestion of contaminated fresh water	Faeces with blood and/or mucus and in some cases vomiting of blood. Cramps that affect the muscles surrounding the entire upper intestine that may be severe enough to cause the lining of the intestine to separate from the wall, leading to systemic infection. Weight loss and muscle aches
Legionellosis *(Discussed in the previous topic)*	Bacterium	The organism thrives in warm aquatic environments only. Often found in water supply canalization exposed to heat. Transmission by inhalation of contaminated aerosols produced in conjunction with water sprays, jets or mists. Infection can also occur by aspiration of contaminated water	Pontiac fever produces milder symptoms resembling acute influenza without pneumonia. Legionnaires' disease has severe symptoms such as fever, chills, pneumonia (with a cough that sometimes produces sputum), ataxia anorexia, muscle aches, malaise, and occasionally diarrhea and vomiting.
Salmonellosis	Bacterium	Ingestion of contaminated fresh water or food	Diarrhea, fever, vomiting, and abdominal cramps
Typhoid fever	Bacterium	Ingestion of contaminated freshwater	Fever up to 40°C (104°F), profuse sweating, diarrhea may occur. Symptoms progress to delirium. In this case, it can last up to four weeks and cause death. Some people with typhoid fever develop a rash called "rose spots," which are small red spots on the abdomen and chest.
Otitis Externa	Bacterium	Ear contact with the water contaminated by the pathogens when swimming or diving. Divers wearing helmets have not their ears exposed to water, but those wearing full-face masks (Standby divers) are exposed. Also, various Otitis forms are common in saturation due to moisture and the high partial pressure of oxygen.	Ear canal swells causing pain and tenderness to the touch
Vibrio Illness *(Discussed in the previous topic)*	Bacterium	Can enter wounds from contaminated water. Also got by drinking contaminated water or eating undercooked oysters	Abdominal tenderness,agitation, bloody stools, chills, confusion, attention deficit, delirium, fluctuation of mood, hallucination, nosebleeds, severe fatigue, slowness, sluggishness, lethargy, weakness.

Disease	*Agent classification*	*Intoxication process*	*Symptoms*
Pseudomonas	Bacterium	Pseudomonas bacteria tend to live and breed in water, soil, and damp areas. The warmer and wetter it is, the better the conditions are for the bacteria to multiply. an open wound can increase the risk of infection.	Ears pain, itching, and liquid discharge Skin rashes, which may consist of pus-filled pimples. Pain and redness of the eyes Pneumonia, coughing, and congestion. Discharge of pus. Joint pain and stiffness, fever, chills, fatigue, headache, diarrhea, or urinary tract infection
Ringworm	Fungus	Contact with someone who has the infection. Shared clothing, towels, bedding.	Itchy skin; Ring-shaped rash; Cracked skin; Hair loss
Fungal ear infection (Also called Otomycosis)	Fungus	Fungal ear infection is an infection of the outer ear which medical term for it is otomycosis. Appears when the cerumen is washed away by water, or is reduced by overuse of cotton buds. The fungus prefers moisture and warmth to grow.	Itching and discharge a thick fluid. Redness of the outer parts of the ear canal with pain and swelling. Hearing problem or feeling of fullness of the ear;

Protozoans, worms, and algae

Protozoans are microscopic unicellular eukaryotes (organisms whose cells have a nucleus enclosed within a nuclear envelope.) that have a relatively complex internal structure and carry out complex metabolic activities. Some Protozoans have structures for propulsion or other types of movement. Their propulsion can be made through moving flagella extensions, small cilia that undulate in waves, or tiny undulating waves in the cell membrane.
Infections from Protozoans range from asymptomatic to life-threatening, depending on the species and strain of the parasite and the resistance of the host.

Worms are invertebrate animals that have soft, slender, elongated bodies. Pathogen worms often live in the bowel of the people or the animal they parasite. Also, some of them can migrate to other parts of the body where they nest. They have various shapes:

- Flatworms have a flat body because they lack a fluid-filled body cavity and an incomplete digestive system with a single opening.
- Tapeworms are flatworms with long bodies up to 20 m long that look like ribbons. Their bodies are made up of segments of the size of a grain of rice.
- Roundworms have a round body because they have a partial fluid-filled body cavity and a complete digestive system allowing them to eat, digest food, and eliminate wastes all at the same time.

Algae are like plants organisms green, red, or brown colours that grow mostly in water, ranging in size from single cells to large spreading seaweeds. They produce their food through photosynthesis and release large amounts of oxygen into the atmosphere. They are not normally considered common pathogens. However, some of them excrete toxins that are pathogen to humans through hosts such as fishes and shells.

The table below lists some infections from these organisms:

Disease	*Agent classification*	*Intoxication process*	*Symptoms*
Cryptosporidium species	Protozoan	This parasite lives in the intestine of infected humans or animals and is transmitted through the food and water. It is protected by an outer shell that allows it to survive outside the body for long periods of time and makes it very tolerant to chlorine disinfection.	Watery diarrhea; Stomach cramps or pain; Dehydration; Nausea & vomiting; Fever; Weight loss.
Entamoeba histolytica	Protozoan	Ingestion of mature cysts in faecal contaminated food, water, or touching the mouth with dirty hands or objects.	Associated with intestinal and extra-intestinal infections with gradual onset over 1 to 3 weeks. Most patients have abdominal pain, tenderness, watery or bloody diarrhea, while only one third are febrile.
Malaria (Plasmodium parasite)	Protozoan parasite	Transmitted through the bite of an infected Anopheles mosquito that lives in tropical and equatorial areas. It can be contracted in ports, rivers, lakes, Note that mosquitos can nest in the boat while in port.	Symptoms include: Moderate to severe shaking chills High fever with profuse sweating Headache, nausea, vomiting Abdominal pain, diarrhea with bloody faeces anemia, muscle pain, convulsions, Coma

Disease	*Agent classification*	*Intoxication process*	*Symptoms*
Giardia intestinalis	Protozoan	Parasite found in contaminated food or water. Contamination is usually through faeces.	Diarrhea; Gas; Greasy faeces that tend to float; Stomach or abdominal cramps; Weight loss; failure to eat fat, lactose, vitamins A and B12. Nausea/vomiting; Dehydration (loss of fluids) Less common symptoms include itchy skin, hives, and swelling of the eyes and joints. Sometimes, the symptoms might seem to resolve,and come back again after several days or weeks.
Schistosomiasis	Flatworm	Ingestion of contaminated fresh water	Blood in the urine (depending on the type of infection), rash, or itchy skin. Fever, chills, cough, and muscle aches.
Echinococcosis	Tapeworm	Ingestion of water contaminated with faeces containing eggs.	Liver enlargement, itchy rash, throat swelling, and "hypotension".
Hymenolepiasis	Tapeworm	Ingestion of contaminated fresh water or food	Abdominal pain, severe weight loss, itching around the anus, nervous manifestation.
Taeniasis	Tapeworm	Ingestion of contaminated fresh water or food	Intestinal disturbances, neurologic manifestations, loss of weight.
Taeniasis-cysticercosis	Tapeworm	Three parasite species cause taeniasis in humans, Taenia solium, Taenia saginata and Taenia asiatica. Only Taenia solium causes major health problems. Transmission via ingestion of raw or undercooked, infected pork..	The larvae (cysticerci) may develop in the muscles, skin, eyes and the central nervous system. When cysts develop in the brain, symptoms include severe headache, blindness, convulsions, and epileptic seizures, and can be fatal.
Ascariasis	Roundworm	Ingestion of water contaminated with faeces containing eggs.	Infection of the small intestine. Fever, diarrhea, nausea, vomiting, difficulty breathing.
Dracunculiasis	Roundworm	Ingestion of contaminated stagnant water	Allergic reaction, urticaria rash, nausea, vomiting, diarrhea, asthmatic attack.

Comments

The lists above are intentionally limited as thousands of pathogens can attack the diving team and vessel crew. Some of them can lead to epidemic conditions and have the potential to kill. An epidemic can put the entire ship out of order and compromise the safety and the diving operation planned. Such events have already happened in the past and may happen in the future.

Most pathogens listed in this document are linked to the presence of faeces in the water and the food. Such conditions are likely to increase with the earth's population that multiplies exponentially, and the fact that a lot of national authorities do not have enough resources to protect the environment from human waste pollution efficiently. The huge areas where plastic spoilage groups in the middle of oceans and the fact that some detritus have been found in the Marianne trench are alerting indicators, in addition to the fact that it is today frequent to smell sewers odours when working at the proximity of the territorial waters limits.

It must also be noted that many pathogens have similar symptoms, so an error of diagnosing is easy. It is the reason the diving medical specialist must be contacted the soonest when an abnormal condition is detected in the chamber or at the surface. Then his/her guidelines will have to be strictly followed.

Note that the document DMAC 26 says that gram-negative bacteria, principally the pseudomonas and the coliform groups, are predominant among the many microbes present in a saturation diving system.
The pseudomonas group is a natural inhabitant of fresh and seawater and can thus readily enter a saturation system.
DMAC 26 also confirms that superficial infections from gram-negative bacteria are more common in the hyperbaric environment than in normobaric conditions.

Treatment

Unless the diving medical specialist confirms that the infection is not contagious, precautions should be implemented to prevent it from spreading to others. ***Diving operations should also be halted if a contagious disease is detected.***

Control measures to be in place:

The scenario below considers support in case of a suspected contagious disease.

- The diving medical specialist must be contacted. A full report of the condition of the casualty must be sent. Note that the diving medical specialist may require a blood sample.
- Protective gloves, clothes and masks must be worn by the diver medics.
- Decontamination of the patient to be carried out. That includes the removal of the clothes which could be infected and the washing of the casualty.
- Strict isolation from the rest of the personnel: The purpose of isolation is to separate ill persons who possibly have a contagious disease from those who are healthy. Also, only the necessary people (nurse & diver medics) should be around the casualty.
- Close contact with the Doctor should be maintained, and his guide lines strictly followed.
- Breathing and circulatory support should be provided.
- Vital sign monitoring
- Fluid therapy
- Psychological support
- Medical evacuation is to be organized as soon as possible (If no restriction from the medical authorities)
- Like contamination by chemicals and toxins, a sample of the water and the mud of the bottom can be sent to the specialized laboratory in addition to a precise description of the job site for investigation.
- With particular reference to blood spillage, but applying also to vomit, diarrhoea, etc., the principles are to clean up thoroughly using disposable gloves and paper cloths, soiled materials are to be placed into a plastic bag. Treat the wiped surface by washing, followed by disinfection.
- A medical check-up to be organized for all people who have been in contact with the casualty to be sure that they are not contaminated.
- Disinfection of all the parts of the diving system which have been in contact with the water and the casualty.
- Disinfection of the items that have been in contact with the casualty. That includes the nurse and diver medics' clothes, tools, etc.
- The patient treated is likely to be evacuated to a medical facility for a hospitalisation period immediately after a dive, and not wait for the normal standby period before flying. Such evacuation is done only on the recommendation of the Diving Medical Specialist (doctor) in charge.
    - This medical evacuation (MEDEVAC) should be organized with medical personnel trained to transfer patients suffering DCS or AGE. In the eventuality that this personnel is missing, diver medics may be used with the approval of the Diving Medical Specialist. During this transfer, the patient should breathe 100 percent oxygen.
    - Air transfer may be necessary for medical or logistical reasons. However, the Diving Medical Specialist should decide whether this procedure is suitable.
    - As the aircraft cabin is usually not or insufficiently pressurized, it must fly as low as safely possible and never expose the casualty to an altitude above 300 m (1,000 feet). Note that some national regulations limit the altitude to 250 m. To compensate for the depression in the aircraft cabin, and if available, an emergency evacuation hyperbaric stretcher that maintains the patient at the sea level pressure (1.013 bar) can be used if the Diving Medical Specialist allows for it.

The diving medical specialist can decide to lighten the precautions in place to isolate the casualty. However, he is the only person who can decide it. The recovery of the casualty to the surface may have to be organized. In this case he must be accompanied by a diver medic.

Note that national medical authorities can decide a quarantine of the vessel: The quarantine's purpose is to separate and restrict the movement of persons who may have been exposed to a contagious disease to ensure that they do not transmit it. This is a classical preventive measure applicable worldwide.

Diagnostic during pandemic periods

A pandemic condition may result in panic and obsessions in various populations that may interfere with evaluation processes and diagnostics. As already said, symptoms of diseases are very similar, and a mistake is easy. For this reason, symptoms must be collected methodically, not taking into account news from public media. The doctor in charge is the only person who can decide the nature of the pathogens involved in the disease, and his recommendations must always be followed. Due to this process's complexity, the doctor may need more investigations to identify the pathogen responsible.

Protocol for assessing fitness to return divers to diving after a pathogen attack

The Diving Medical specialist is the only person who can decide whether a diver is “fit to dive”.

Following the COVID-19 pandemic, numerous documents have been published by organizations.
Protocols linked to such diseases cannot be static as these pathogens evolute, and the process of control have to be modified at the same time the virus evolute. For this reason, the best procedure is to follow the recommendations of the Diving Medical Specialist, who is usually in close contact with official organizations such as the DMAC and other medical institutions.
Regarding return to diving, the Diving Medical Advisory Committee has published the document DMAC 33, *“return to diving after COVID-19”*, which is regularly updated (https://www.dmac-diving.org/guidance/DMAC33.pdf). However, the Diving Medical Specialist may decide on a more stringent process.
The DMAC has also published the document DMAC 34, *“Guidance for medical examiners of divers conducting face-to-face medicals during the COVID-19 pandemic*” (https://www.dmac-diving.org/guidance/DMAC34.pdf) that explains the precautions to be in place during the medical evaluation to avoid transmission of the disease.

Kindly note that there are many other infections than COVID-19 that need to have such a protocol, and it is the responsibility of the doctor in charge to organize them.

Prevention

New arrivals observation period and World Health Organization (WHO) recommendations to travelers

Recent pandemic events also prove that divers and the surface support staff should not be transferred to the vessel on arrival. Periods of observation have been recently set up to 14 days for the COVID-19. However, this procedure should be kept in place even during non-pandemic periods. The reason is that we ignore when pathogen attack starts and the extend of this attack. As discussed previously, any contagious disease on board the vessel has the power to contaminate all staff, which results that the operation must be stopped.
The contractor should ensure that the personnel is not in contact with contaminated people during these periods of isolation. Also, transfers should be preferably organized in countries not (or less) affected by a pandemic if this condition is observed. Note that the hygiene of the facilities where the personnel is quarantined should be closely monitored.
It is clear that such precautions have an impact on the way operations are organized and may increase costs.

Note that the World Health Organization (WHO) has emitted the following guidelines to travellers that should be remembered to people travelling to their mobilization point even during non-pandemic periods:

- Keeping a distance of at least one metre from persons showing symptoms is particularly important.
- Hand hygiene must be performed frequently, particularly after contact with respiratory secretions. Hand hygiene includes either cleaning hands with soap and water or with an alcohol-based hand rub. Alcohol-based hand rubs are preferred if hands are not visibly soiled; wash hands with soap and water when they are visibly soiled;
- Nose and mouth must be covered with a flexed elbow or paper tissue when coughing or sneezing and the tissue disposed of immediately in addition to performing hand hygiene;
- Refrain from touching mouth and nose;
- A medical mask is not required if exhibiting no symptoms, as there is no evidence that wearing a mask – of any type – protects non-sick persons. However, in some cultures, masks may be commonly worn. If masks are to be worn, it is critical to follow best practices on how to wear, remove and dispose of them and on hand hygiene after removal:
  - place mask carefully to cover mouth and nose and tie securely to minimise any gaps between the face and the mask;
  - while in use, avoid touching the mask;
  - remove the mask by using appropriate technique (i.e. do not touch the front but remove the lace from behind);
  - after removal or whenever you inadvertently touch a used mask, clean hands by using an alcohol-based hand rub or soap and water if visibly soiled
  - replace masks with a new clean, dry mask as soon as they become damp/humid;
  - do not re-use single-use masks;
  - discard single-use masks after each use and dispose of them immediately upon removal.

The World Health Organization also says that travellers should also be advised to follow proper food hygiene practices. However, this point is difficult to follow as travellers usually have no means to check how the food served in airports and planes is made.

Food hygiene

The lists displayed previously shows that the majority of pathogen attacks are through food and water. Thus these points are to be closely monitored:

Food and beverages should be checked on arrival onboard:

- Fruits and vegetables should be cleaned with fresh water to eliminate parasites they may host prior to being stored in the cargo.

- Freeze and cold chains of meats must not be broken during the travel: Minimum temperature of a freezer is -18 Cº and the temperature of a fridge should be below 5 Cº. Note that these temperatures must be kept while transferring food from delivery containers.
- There should be a tractability of the fruits and the meat transferred onboard with adequate certificates of conformity.
- Expiration dates should be respected, and the food that has passed the limit should be returned to shore.
- Fridges and freezers should be regularly cleaned and disinfected.

The quality of the water is of utmost importance. Contaminated water often results from water transferred from dirty water tankers or canalizations.

- Potable water should conform with the "guidelines for drinking water quality" from the World Health Organization (WHO).
- A lot of ships are able to product their water. Reverse osmosis systems are today the most used. However, evaporators are also often employed. They should be adequately maintained.
- Canalizations must be regularly disinfected.
- The water must be regularly analyzed to ensure that it conforms with the requirements indicated above.

Kitchen hygiene should be monitored.

- Kitchens and restaurants must be fully cleaned and disinfected daily.
- Swabs should be taken from the walls, floors, tables, and cooking tools twice a week.
- The temperatures and internal condition of the fridges (< 5 C°) and freezers (< -18 C°) should be regularly checked.
- Hygiene and physical condition of the personnel working in the kitchen should be monitored, and they should wear hygienic barriers that are renewed every shift. Also, the staff should be reminded to follow the recommendations from the World Health Organization (WHO).
  - Wash hands before handling food and often during food preparation, and after going to the toilet.
  - Wash and sanitize all surfaces and equipment used for food preparation.
  - Protect kitchen areas and food from insects, pests and, and people who are not part of the kitchen staff.
  - Separate raw meat, poultry and seafood from other foods
  - Use separate equipment and utensils such as knives and cutting boards for handling raw foods
  - Store food in containers to avoid contact between raw and prepared
  - Cook food thoroughly, especially meat, poultry, eggs and seafood
  - Bring foods like soups and stews to boiling to make sure that they have reached 70°C. For meat and poultry, make sure that juices are clear, not pink. Ideally, use a thermometer.
  - Reheat cooked food thoroughly.
  - Do not leave cooked food at room temperature for more than 2 hours
  - Refrigerate promptly all cooked and perishable food (preferably below 5°C)
  - Keep cooked food piping hot (more than 60°C) prior to serving
  - Do not store food too long even in the refrigerator
  - Do not thaw frozen food at room temperature
  - Use safe water or treat it to make it safe
  - Select fresh and wholesome foods
  - Choose foods processed for safety, such as pasteurised milk
  - Wash fruits and vegetables, especially if eaten raw
  - Do not use food beyond its expiry date.

Personnel hygiene

The procedures indicated above should be in force for the entire diving and vessel crew.

- Coveralls and other clothes should be cleaned daily.
- Bedding should be regularly renewed, and the beds disinfected.
- Cabins should be cleaned daily.
- People should wash regularly, and wash their hands any time is is necessary (When they are dirty, before meals and after going to toilets, etc.).
- People should be warned not touching their mouth and nose with dirty hands, and wear gloves.
- A regular medical check up should be organized. In addition, DMAC 26 says that regular visits to the dentist, at least ones a year are to be encouraged to ensure continuing dental health and hygiene.
- Note that infections by identified pathogens can be prevented by vaccination.

Chamber cleansing

DMAC 26 also provides guidelines regarding chamber cleansing and environmental control and says:

- Chamber cleansing is designed to limit microbial growth (particularly the predominant Gram-negative bacteria) and, therefore, to protect against infection.
- Cleansing (with liquid anti-microbial - specific agents considered below) is started at the top of the chamber and is continued downwards, with excess cleanser ultimately drained from the bilges. Relays of fresh cloths / sponges should be used on each occasion and discarded after limited use.
- Before diving operations, the entire chamber (including e.g. service locks, "rims" of toilets, bunk brackets) is most thoroughly cleansed (with the deck plates lifted), and allowed to dry. The parts of the chamber which will be in direct or indirect contact with the skin (e.g. toilettes, BIBS masks) and other personal equipment e.g. headsets, should be disinfected using chamber cleanser, left for a minimum of 10 minutes, then rinsed and dried thoroughly. Shower-heads should be removed, cleansed, rinsed after 10 minutes, and dried. The chamber should be ventilated and clean bedding provided.
- Bilges or floor areas beneath deck plates should be drained of cleanser, but should not be actively cleansed or otherwise disturbed.
- Several cleaning agents are in use or recommended. These include "Panacide M", "Tego 103G", “Tego 2000”, and “Trigene” . Various other products may also be suitable.
- The prime requirements of the disinfectant agents is that they should be very effective against the microbes known to flourish in the chamber environment and be non-toxic to man. Additionally, the disinfectants should be odourless, non-volatile, and be free from irritant and sensitising properties.
- "Panacide M" is now less used than formerly because of its undesirable properties of strong odour and skin irritation. The Tego products are increasingly being used, and combine good anti-microbial properties with relatively few disadvantages, e.g. they are odourless and less likely to be an irritant to the skin.
- All chamber disinfectants should be used at the appropriate dilution, skin contact should be minimised by the use of personal protective equipment, and they should be applied by cloth or sponge to avoid the formation of an airborne aerosol.

Helmet cleaning

Helmets are parts of the diving system that are the most susceptible to be contamination vectors, as pathogens accumulate here during the dive. An additional problem linked to common practices is that helmets are usually shared between divers, increasing the risk of contamination. For this reason, they must be cleaned after each dive.
IMCA suggests using a quick procedure between two dives and a full cleaning procedure at the end of the day.

The proposed quick sanitizing procedure implies the following steps:

1. Wet or immerse all components to be sanitized with an appropriate disinfectant solution for at least 10 minutes and lightly scrub over the components with a nylon brush or a clean dishrag to remove saliva mucus build up.
2. After 10 minutes, thoroughly rinse components using running potable water.
3. Allow to dry or pat dry with clean towel.

This cleaning procedure requires time to be performed adequately. Also, there is a risk that some parts of the oro-nasal remain contaminated, particularly the exhaust regulator. Thus, considering these problems and the fact that each diver uses the same helmet during the bell run, the best option can be replacing the helmet used with a clean one that has been fully cleaned and reviewed by the technicians after the bell run. Changing a helmet takes 10 minutes, which is the time given to allow the disinfectant to operate.

The full cleaning procedure presented by several safety organizations implies the following steps:

1. Secure and bleed the gas supplies. Disconnect the gas connections, disconnect the communication wires, and secure the open ends with a dedicated cap or tape them to ensure that no water can enter them.
2. Transfer the helmet outside the bell.
3. The demand regulator clamp is opened, and the components such as cover, diaphragm, assembly oral-nasal mask, and nose cleaning pad are dismantled and stored adequately not to lose them.
    - The demand regulator must be rinsed with mild detergent and fresh water and then rinsed thoroughly.
    - Depending on the recommendations of the manufacturer, the parts are soaked for at least 5 minutes. Note that IMCA suggests the sanitary solution stays in contact for 10 minutes.
    - Then the elements are scrubbed using a small nylon brush. The pieces that have been in contact with the detergent must be rinsed.
4. The pieces that have been in contact with the detergent are then soaked in fresh water to ensure that the detergent is fully rinsed off.
5. The head cushion assembly must be removed. If it is wet with perspiration or water, it must be cleaned and dried.
6. Then, the technician should inspect the spares for damages.
7. The helmet liner should be washed with soap and water, rinsed in freshwater, dripped, and dried.
8. The earphone covers and the microphone should be removed from the oral-nasal mask, washed with a mild detergent solution, rinsed with fresh water, and dried.

9. The components should be laid out to allow for drying before storing
10. The neck-dam ring assembly is then cleaned with a mild detergent solution and thoroughly rinsed with fresh water.
11. When the components are fully dried, they can be reinstalled.
12. The helmet should be then tested.
13. When the tests are satisfactory, The helmet is protected from contamination in a sealed bag and then be transferred into the system when required.

Note that excessive contact time with the sanitized might damage equipment, while insufficient dilution reduces effectiveness. The product recommended by the helmet manufacturer should be used. However, it should be approved by the relevant public health authority. In the absence of such approval, it should conform with EN 16777 or EN 14476. Note that IMCA suggest “RELY ON VIRKON” and Chemgene HLD4H.

Essential practice:

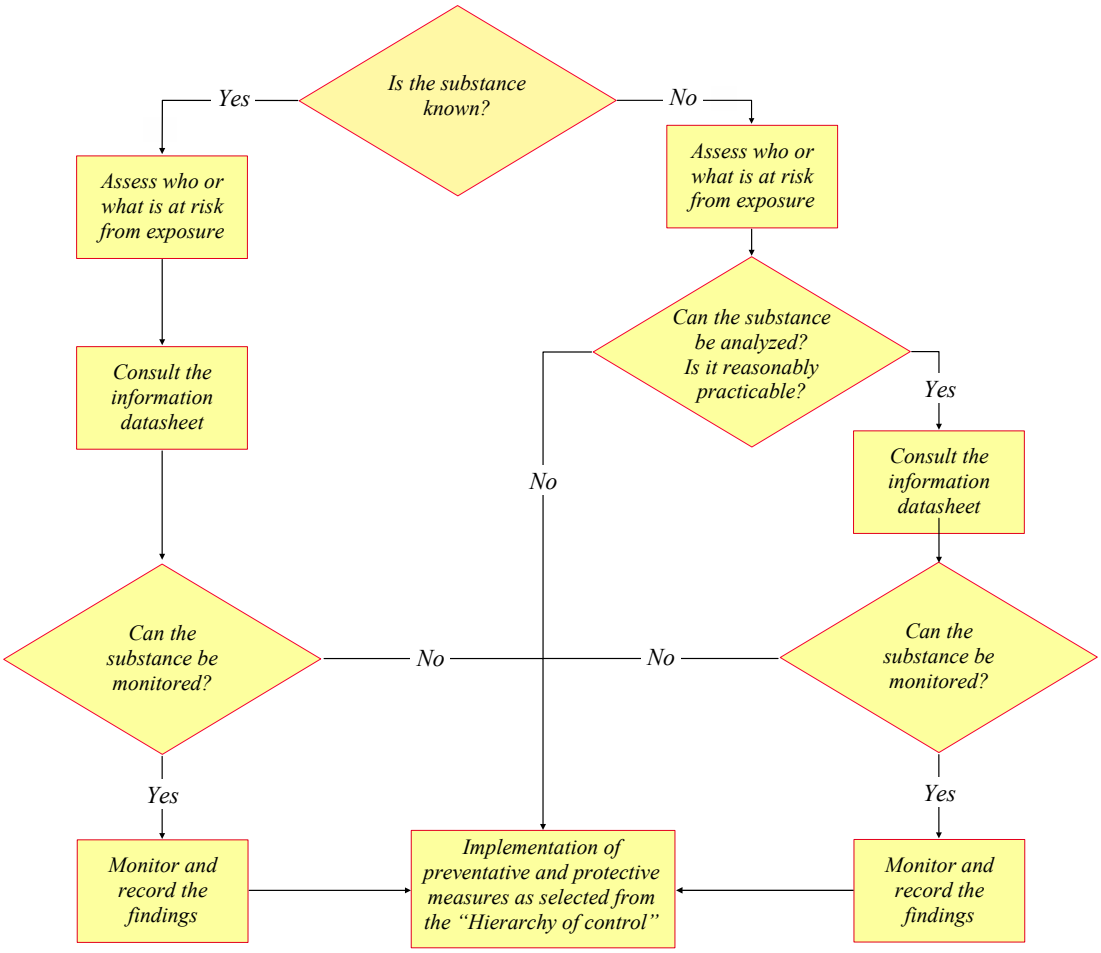
Pre-dive function tests have to be performed by the diver using the helmet.

Investigation of the area of intervention prior to launching the operations

Investigations regarding the quality of the water and possible dumping of pollutants to the sea should be undertaken:

- Samplings of the bottom and of the water should be organized to detect pathogens and faeces prior to starting the interventions in the selected area
- The observation of the river banks and/or of the quays of the port or/and the beaches is another means of information concerning the possible dumping of faeces and other human and animal wastes.
- As for chemical agents, public services can transmit some information regarding the pathogen agents found in rivers and then dumped to the sea. Still, they have to be considered with caution as contamination levels can change from the time the investigation has been performed.
- Note that the fact some local people are sometimes drinking the water from the river they are living along is not a reliable indicator that there are no pathogens in the water

A risk assessment must be carried out to decide whether the intervention using divers is safe or not on sites where chemicals, pathogens, and any other agents which could harm the health of the divers is suspected. The chart displayed below, with is also used for hydrocarbons, chemicals, and radioactive materials can be used as a guide,
As with hydrocarbons, chemicals, and radioactive substances, diving operations should not be launched in the presence of large quantities pathogens agents that can contaminate the divers if they are not adequately protected.
In case of exposure to dangerous conditions, the ROV becomes the preferred means of intervention.



Specific diving systems for intervention in unhealthy surrounding

In the eventuality that diving operations have to be organized in unhealthy waters, they must never be carried on using classical materials. Thus, appropriate equipment must be supplied to the divers:


DSL-B-2CNF

- Fully closed suits that are easy to clean and disinfect, such as vulcanized rubber dry suits, should be used for all diving in contaminated water. Such dry suits should have a watertight seal with the helmet to avoid skin contact with the contaminated water. A rigid neck ring is bonded to the suit for this purpose. Reinforced rubber gloves should be sealed using rigid rings at the wrist level. To avoid nips (see in helmet squeeze and nips), care should be taken to ensure divers and dry suits are matched appropriately by size and can be inflated.
- Of course, the helmet must be perfectly waterproof to be sure that contaminated water will never enter. Continuous flux helmets like the AH4 & AH5 proposed by the company "Divex", or the DSL-B-2CNF, designed by "Composite Beat Engel", are appreciated and certified for use in the nuclear industry and polluted waters. The diver needs to be trained to use such helmets, as continuous flux helmets can inflate the suit and trigger a blow-up if the exhaust valve is not adjusted correctly and no neck dam is installed to separate the suit from the helmet.
- Vulcanized rubber suit manufacturers usually publish the data for resistance of their models to a host of chemicals. This information should be referenced and easily accessible. The durability of other components of the diving ensemble, such as gloves, umbilical, and harnesses, must be taken into account. Common signs that contaminants have degraded the integrity of a suit include swelling of the material, colour changes, tackiness, and stiffness.

- Another type of helmet used in polluted surroundings is a demand valve regulator helmet called "Dirty Harry", also designed by "Divex". This helmet is attached directly to the vulcanized rubber dry suit using a neck ring similar to those used for the continuous flow models. Its breathing system is a closed circuit system whereby the divers exhausted gas is returned to the surface and released into the atmosphere rather than into the water. The reclaim system consists of the Divex Ultraflow 601 demand regulator fitted to a Superlite 17C Kirby Morgan, also used in saturation diving, and a control module of the closed circuit in the dive control. The advantage of the demand regulator is that the diver does not need to readjust the inlet and exhaust valve according to the depth, which is more comfortable. Its main inconvenience is that the exhaust system is complex compared to a continuous flow system and, of course, more expensive.

Operating in pathogenic and chemically polluted environments

Strict hygiene must be applied by the diving team when operating in environment polluted by pathogens:

- When the diver is back on the deck, he must not be immediately undressed, as his helmet and suit are contaminated and must be immediately fully disinfected, using appropriate products. Thus, the helmet is removed only when the team is 100% sure that no pathogen is remaining on the suit.
- Then, the diver is fully undressed, and the cleaning of the umbilical, bailout and any tool used is completed. Of course, the helmet is thoroughly disinfected, as explained previously. At the end of the cleaning phase, the suit, helmet, and complementary equipment must be as new and be exempt from foul odours. Note that bad odours (for example, sewer odours) are indicators that pathogens are still active.
- During the operation, the tenders should use disposable plastic over-suits and gloves. The skin should never be exposed to pathogens, so only thoroughly cleaned equipment can be handled without waterproof gloves.
- Surface O2 decompression must be avoided not to take the risk of contaminating the chamber. It must be considered that undressing the diver as recommended above the chamber may take a too long time.

In addition, the precautions for intervening using wet bells are widely more complex than those used when diving in areas polluted by hydrocarbons situated in delimited areas on the bottom because the contamination can be from the water itself. Thus, open or closed bells should not be used in such waters.

Acute otitis externa

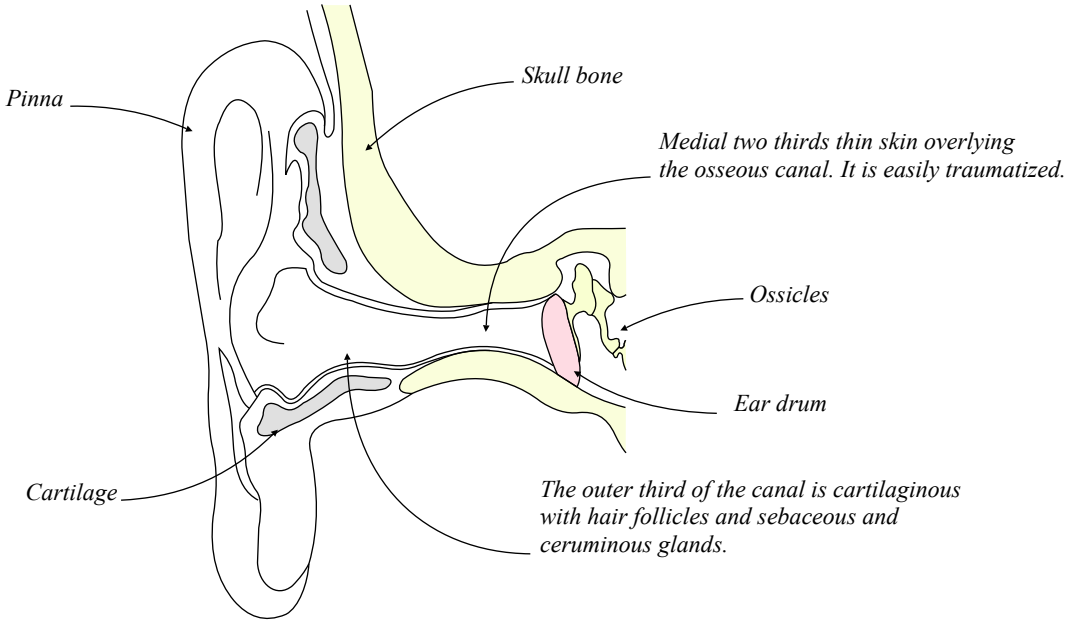
Acute otitis externa is a common pathogen infection of the ear canal previously indicated in the previous point “Pathogen attacks”, and is more discussed in this point.

Description:

Otitis externa is an inflammation of the external ear canal that may extend to the pinna and the tympanic membrane. On rare occasions, the infection invades the surrounding soft tissue and bone. It is primarily caused by a bacterial infection, with “pseudomonas aeruginosa” and “staphylococcus aureus” the most common pathogens.
Note that chronic otitis externa is often the result of allergies, chronic dermatologic conditions, or inadequately treated acute otitis externa.

Mechanism:

The external auditory canal of the ear is a skin-lined tube with the end closed by the eardrum. This skin is fragile, and the lateral third overlies cartilage, while the rest has a base of bone. The auditory canal curve also impedes the exit of debris, secretions, and foreign bodies at the junction of the cartilage and bone. Besides, this canal is warm, dark, and prone to becoming moist, making it an excellent bacterial and fungal growth environment.



The external auditory canal defenses consist of secretions of cerumen that creates an acidic coat containing lysozymes and other substances that inhibit bacterial and fungal growth. The lipid-rich cerumen is also hydrophobic and prevents water from penetrating the skin and causing maceration. Additionally, the canal is defended by an epithelial migration from the tympanic membrane outward.
Despite its hydrophobic capabilities, the cerumen can be removed during a dive if the ears contact the water for an extended period. This is normally not the case of surface-supplied divers using closed helmets, but that can happen to those diving with face masks. Also, excessive or too viscous cerumen can lead to obstruction and retention of water and debris. In addition, the cerumen's efficiency can be altered when the diver is continuously exposed to a moist atmosphere, which can be the case in tropical and equatorial areas. As a result, bacterias such as “pseudomonas aeruginosa” and “staphylococcus aureus”, which live in the soil, water, and plants, and appreciate moist, dark, and hot surrounding, can nest and quickly proliferate.
These bacteria can be contracted by the insertion of foreign objects such as cotton swabs, fingernails, hearing aids, and earplugs, and contact with contaminated water.
Acute otitis externa symptoms of canal inflammation can range from mild discomfort, itching, minimal edema to severe pain, complete canal obstruction, and the pinna and surrounding skin's involvement. Fever may be present, which may suggest extension beyond the auditory canal.

Treatment:

The treatment of each infected patient must be performed under the direction of the Diving Medical Specialist.
The external auditory canal should be cleaned as much as possible, and a wick inserted if swelling is severe.
Acidification with 2 percent acetic acid is usually performed. However, the doctor may select another treatment.
Specialized websites say that antibiotics are rarely needed but may be used when otitis externa is persistent, or if the infection spreads, or the patient has a fever.

Precautions:

This infection is common with divers in saturation and less encountered with divers involved in surface-supplied diving operations for the reasons discussed previously.

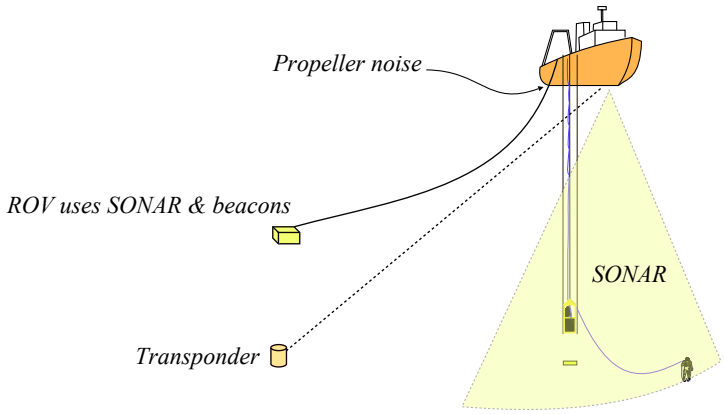
Prophylactic ear drops containing acetic acid and aluminium acetate designed to minimize the chance of infection by maintaining the external ear canal acidic are provided to saturation divers. However, this preventive treatment is not necessary for surface-supplied divers.

Preventive measures for surface-supplied divers should include not touching ears with dirty fingers and objects and keeping them as dry as possible.
Another preventive measure consists of promoting closed helmets instead of face masks and not sharing helmet liners that must be dedicated to each diver and systematically cleaned after each dive.

Harmful noises

Purpose

Work tools like pile & Pal planks driving hammers, drillers, hydraulic and pneumatic jackhammers, hydraulic cutters, Hp water jets, and others are very noisy and frequently used on underwater work sites.
In addition, some important parts of the vessels and numerous tools used on worksites to measure the depth of the ocean floor, locate objects and animals underwater, give precise pictures of immerged structures, guide divers and ROV, check wall thickness and perform other tasks are systems based on the emission and the reception of sounds.



Note: SONAR means “Sound Navigation and Ranging”

Sounds

Sounds are the result of vibrations.
A vibrating object sets up a motion of the molecules around it so that when the object moves in the direction of an observer, it compresses the medium (gas, liquid or solid). When it moves away, it produces a rarefaction. This sequence of compressions and rarefactions is transmitted in a straight line at a characteristic speed. Unlike light, sound waves cannot travel through a vacuum but require some medium, gaseous, liquid, or solid medium with a different speed of conduction.

The frequency of a signal is usually expressed in a unit called "hertz" (Hz). One Hz equals one cycle per second. One megahertz (MHz) equals one million cycles per second.
The unit used to measure the intensity of a sound is called decibel (abbreviated dB), which is the logarithmic measure of the effective sound pressure measured in micro-Pascal (µPa).
The arbitrary references are 20 µPa in air and 1 µPa in water. On the decibel scale, the smallest audible sound (near total silence) is 0 dB. A sound 10 times more powerful is 10 dB. A sound 100 times more powerful than near total silence is 20 dB. A sound 1,000 times more powerful than near total silence is 30 dB.
It is common to link emitted decibels to common sound intensities. The following levels that are taken while standing near the sound emission can be taken as references:

- Near total silence - 0 dB
- A whisper - 15 dB
- Normal conversation - 60 dB
- A small thermal engine - 90 dB
- A horn - 110 dB
- A jet engine - 120 dB
- A gunshot - 140 dB

It must be remembered that the distance affects the intensity of a sound. As a result, the intensities indicated above will be greatly diminished far away.

The speed of sound is the distance sound waves travel in a given amount of time. Sound waves travel most quickly through solids, followed by liquids, and then by gases. Particles of matter are closest together in solids and farthest apart in gases. When particles are closer together, they can more quickly pass the energy of vibrations to nearby particles.

The speed of sound also depends on the temperature of the medium. For a given medium, the sound has a slower speed at lower temperatures. The table below provide values in metres per second (m/s).

Medium	*0 C°*	*10 C°*	*20 C°*	*30 C°*	*40 C°*
Dry air	331	337	343	349	354
Helium	892	908	924	939	955
Fresh water	1403	1447	1481	1507	1526
Sea water	1449	1490	1522	1546	1563

The speed of sound in water increases with increasing water temperature, but also with increasing salinity and increasing pressure. The approximate change in the speed of sound with a change in each property is:

- Temperature - 1°C = 4.0 m/s
- Salinity - 1 Practical Salinity Unit (PSU) = 1.4 m/s (average salinity is 35 g/kg= 35 PSU)
- Depth - 1 m = 0.017 m/s

The human ear

The auditory apparatus with which we detect sounds can be divided into three parts:

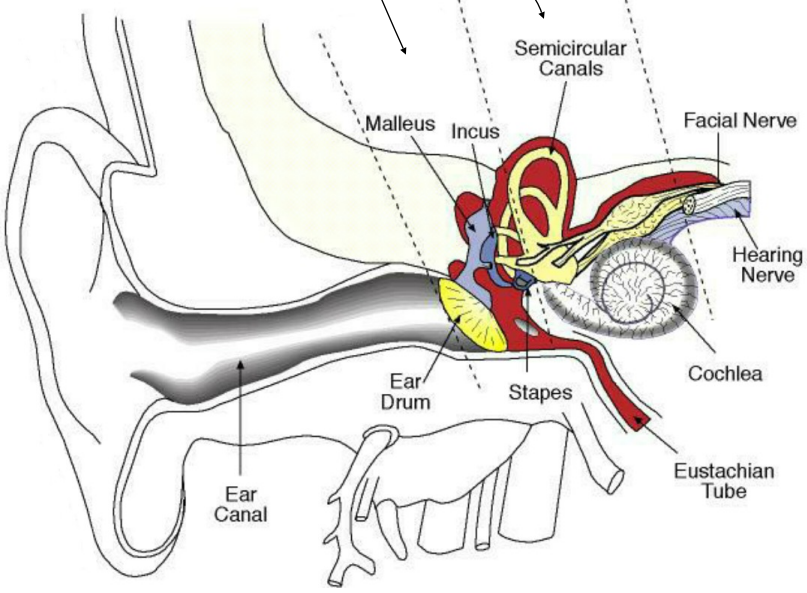
1. The external ear is made of the pinna and the ear canal. The pinna is the visible part of the ear, and it helps us focus and localize sounds. Sound is transmitted to the middle ear through the auditory canal.
2. The middle ear consists of the tympanic membrane or eardrum, the malleus, the incus, and the stapes. The tympanic membrane covers the end of the auditory canal and is attached to the malleus. The incus connects the malleus to the stapes, which is connected to the inner ear. When the pressure of air in the external ear changes, the tympanic membrane moves in or out. This makes the stapes move in such a way as to convey sound vibrations to the inner ear.

The middle ear *is a rigid air-filled space, surrounded by bone (skull). The eardrum forms an outer wall, and the airspace is connected to the rear of the throat by the Eustachian tube.*

The middle ear comprises a series of bones, which transmit vibrations from the eardrum to the inner ear.

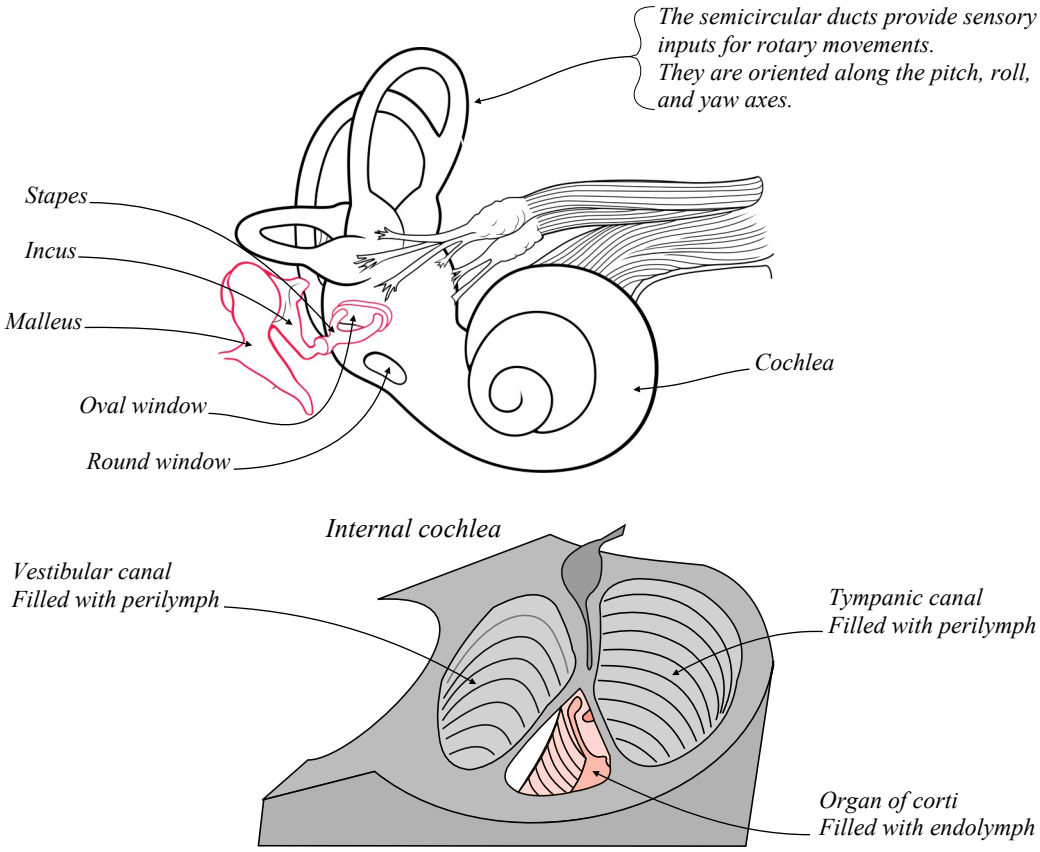
The inner ear *is filled with fluid and is embedded in the skull. Vibrations picked up at the eardrum and transmitted to the inner ear are converted into nerve impulses sent to the brain and perceived as sounds.*

The semicircular canals are considered part of the inner ear but play no part in the hearing. They are of major importance for a sense of balance and position, and their function can be upset by certain conditions affecting the middle ear.

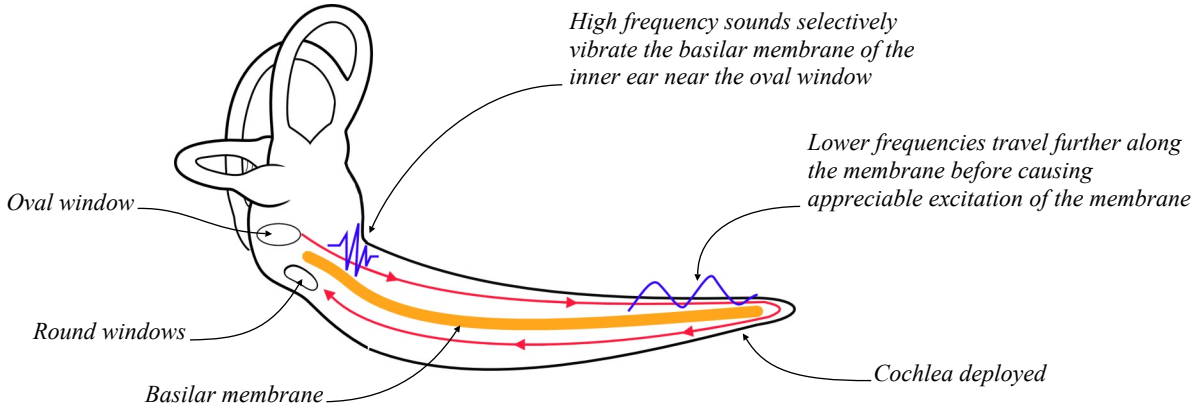


The external ear *consists of the pinna and the ear canal, which is closed off at its inner end by the eardrum.*
The outer ear is open to the air, and its purpose is to collect sound waves and direct them to the eardrum.

3. The cochlea is part of the inner ear and is shaped like a snail's shell. It is filled with a fluid through which sound waves pass.
   Sound vibrations are transmitted into the cochlea when the ossicles move the oval window. Just below, the round window is covered by a flexible membrane, which allows the fluid in the cochlea to move back and forth in response to sound vibrations.
   Because the oval window is much smaller than the tympanic membrane, small vibrations of the tympanic membrane at some frequencies result in relatively large vibrations of the oval window; in other words, the middle ear amplifies certain tones.



Inside the cochlea is a structure called the basilar membrane, which runs nearly the cochlea's entire length. Attached to the basilar membrane are hair cells, which move when the fluid of the cochlea moves; the hair cells convert sound into signals sent to the brain.
Different parts of the basilar membrane flex in response to different kinds of sound. Hair cells at the basilar membrane area, which is flexed, are activated and send messages through the cochlear nerve to the brain. At the top of each hair cell, many cilia are connected to each other by extremely fine fibers of a protein called tip links. Hair cells are activated by the tension placed on the tip links when the cilia are moved. Ordinarily, there is a little bit of tension on the tip links and, consequently, the hair cells are very slightly activated. The movement of fluid over the hair cells causes them to send impulses to the brain.
The ear sends impulses to the brain through the auditory nerve to the brainstem. Most of these signals are projected to a structure called the superior olivary complex, which is involved in determining the location of sounds. This area sends messages to another structure called the inferior colliculus, which is in the midbrain. This information passes through the thalamus and is sent to the temporal lobe of the cerebral cortex, which is involved in decoding the information sent from the ears and processing the more complex aspects of sounds.



Higher frequencies are processed by a system called "place coding." As already mentioned, specific portions of the

basilar membrane move in response to certain frequencies of sound.
The brain recognizes sounds of moderate to high frequency by the portion of the basilar membrane that is moving. However, specific regions of the basilar membrane do not move in response to sounds with a low pitch. These sounds are identified by "rate coding." Certain cells at the end of the basilar membrane move along with the liquid in the cochlea. It is thought that the brain perceives sounds with lower frequencies using the frequency of oscillation of these cells.

Possible damages to ear

The hair cells of the Basilar membrane can be broken or damaged if a person is exposed to too much noise. Hearing impairment can occur when the high-frequency or low-frequency areas of the cochlea are harmed by loud sounds. If the cochlea is damaged, the cilia can change direction. Because the cilia are connected to the brain by way of the auditory nerve, the result is that impulses can be sent to the brain via the auditory nerve even when there isn't any sound.
Also, because linked, the vestibular system can be damaged, with loss of balance and orientation.

- In their book “Diving medicine” doctors Bove & Davis say:
  “Noise trauma is, by far, the most common cause of neuro-sensory hearing loss in divers. Transient auditory threshold shifts of 20 to 30 dB lasting up to 24 hours are not uncommon after noisy dives…... Comparison of pre- and post-dive audio-grams reveals the extent of the loss”... “Repetitive noise trauma leads to permanent neuro-sensory losses, and most divers show such changes”.
- In the document DMAC 06, is discussed points 3 and 4:
  “Divers exposed to high levels of underwater sound can suffer from dizziness, hearing damage or other injuries to other sensitive organs, depending on the frequency and intensity of the sound.”
  “The single most important criterion related to diver safety resulting from low frequency sonar is that of disorientation due to vestibular stimulation. Whilst exposure to sonar transmissions below a level necessary to cause disorientation can give rise to temporary hearing threshold shifts, these are considered operationally acceptable for diving operations over limited periods”

A healthy human ear can hear frequencies that range from 20 hertz (Hz) to 20,000 Hz. The sounds below 20 Hz are classified as “infra-sound”, and the sounds above 20 kHz are classified as “ultra-sounds”.
If their hearing is not impaired, most people can hear the rustling of leaves, which is at a level of about 0 dB.
According to official publications, any sound above 85 dB can cause hearing loss, and the loss is related both to the power of the sound and the length of exposure. Eight hours of 90-dB sound can cause damage ears; any exposure to 140-dB sound causes immediate damage (and causes actual pain).
The maximum time exposure above 85 DB in air without permanent hearing loss can be listed below:

Continuous dB	*Max exposure time*		*Continuous dB*	*Max exposure time*
85	8 hrs		109	< 2 min
88	4 hrs		112	< 1 min
91	2 hr		115	< 30 sec.
94	1 hr		118	< 15 sec.
97	30 min		121	< 7 sec.
100	15 min		124	< 3 sec
103	7.30 min		127	< 1 sec
106	< 4 min		140	No exposure

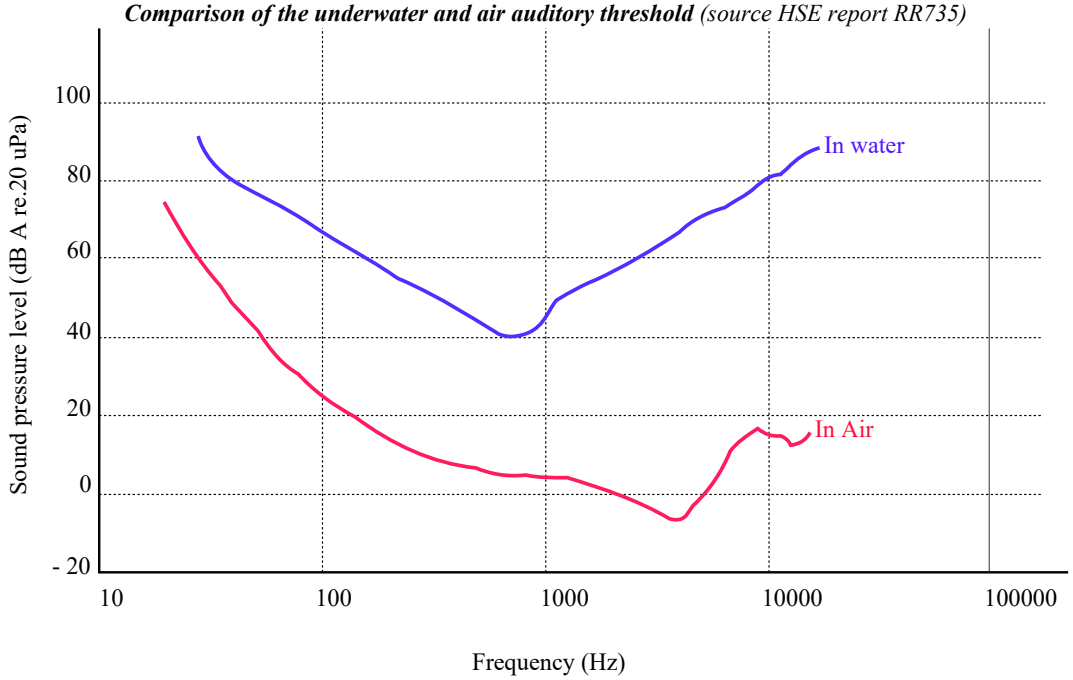
Hearing underwater

Hearing underwater differs from hearing in the air as the acoustic properties of water and air are different. Also, the sound in water can propagate relatively freely through the human body.
According to the last researches, it is likely that both bone sound conduction and tympanic sound conduction combine underwater. Bone conduction would appear to play a much greater role in hearing underwater than it does in the air.
Evidence that tympanic conduction is also involved in hearing underwater has come from studies investigating the ability of some divers to locate sound:
In the air, sound localization occurs by detecting the delay between sounds arriving at each ear, which involves tympanic

conduction of sound.
With bone conduction only, the sound would arrive simultaneously at each ear, and the localization would not be possible. Some studies suggest that tympanic conduction appears to be predominantly at low frequencies because sound localization seems more acute at these frequencies.
At high frequencies, bone conduction seems to be the dominant factor.

Underwater, a “wet ear” is less sensitive than an ear in a dry surrounding; thus, the underwater noises are believed to produce less hearing damage than in air. The decreased sensitivity may be due to the movement of the tympanic membrane being damped by the mass of water. Due to this reduced hearing sensitivity of the ear when immersed in water, the noise exposure of a diver with “wet ears” has to be adjusted using an “underwater weighing scale”.
On the opposite, because the ears of a diver wearing a diving helmet are “dry”, the noise exposure is considered the same as before starting the dive. In addition, it has been proved that hearing sensitivity in hyperbaric environments is similar to that in the air at atmospheric pressure, regardless of the breathing gases. In this case, determining the noise hazard is achieved using the same method for occupational noise hazards above the surface.

Comparison of the underwater and air auditory threshold *(source HSE report RR735)*



Standard references must always be used to compare sound levels given in dB in air and water.
To calculate the “sound Pressure Level”, which is the logarithmic measure expressed in decibels, scientists have arbitrarily agreed to use the intensity of a sound wave at a pressure of 1 micro-Pascal (µPa) as the reference intensity for underwater sound.
For the calculations in air, scientists have selected a reference of 20 micro-Pascal, which is higher pressure. 20 micro Pascal corresponds to the minimum threshold of young human adults in their range of best hearing (1000 -3000 Hz).
Therefore, sound levels given in dB in water are not the same as sound levels given in dB in air.
Underwater sounds are expressed using the form “dB re. 1 µPa”, and the sounds in the air “dB re. 20 µPa”.
A weighted scale, denoted by dB(A), is used and indicates how the noise in air is related to the human perception of sound.
Thus, the presentation for noise in the air, related to human hearing, is: “dB (A) re. 20 µPa”.
A quick method, generally accepted in ocean acoustics to convert decibels in the air to decibels in water, is to add 62 dB to the initial value in the air as explained below:

- The 1st step is to add a 26 dB difference related to the arbitrary reference level from which the sound intensity is measured. So (considering only this first correction factor), a sound measured at 126 dB in water will be as loud as a sound that measures 100 dB in the air.
- For the 2nd step, because water is much denser than air, water has a higher impedance, so it is considered that sounds of equal measured pressure will be measured at 36 dB higher in water. So, a sound at 90 dB in the air would be measured as 126 dB in the water.
- The 3rd step is the addition of these 2 values to the value given in air. So, a sound reported at 85 decibels in the air should be around 147 decibels in water.

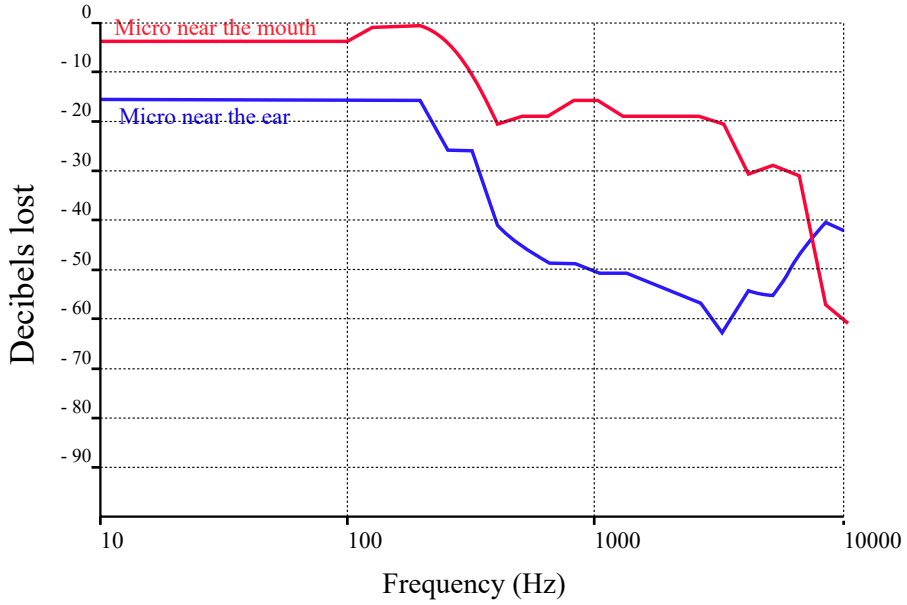
The method can be used, of course, to perform a quick conversion of sounds in the water to sounds in air.
This method is based on abstractions and sometimes gives overestimated values. Still, it has the advantage of giving an instantaneous idea of the sound pressure level in the water or the air.

The measurements of noise radiation from the sources are often undertaken from different distances.
As the noise level varies with the distance and the surrounding, it is important to have normalized measurements to allow direct comparison of levels. The convention is to specify the apparent noise at 1 m from the source. This is indicated as the “Source Level”. Thus, a noise measured at several kilometres range may be expressed as an estimated Source Level at 1 m in the form: “dB re. 1 µPa @ 1 m”.

Other elements to be considered in the evaluation of sound pressure:

- A neoprene hood is agreed as an efficient protection against underwater noises. DMAC 06 /point 6 says:
  "The neoprene hoods normally worn by hooded divers do afford some attenuation. A 3 mm neoprene wet-suit hood provides some hearing protection (10 dB) from sound frequencies between 400 and 500 Hz at shallow depth (<10 msw)".
  However, except for the standby diver, band masks are not considered suitable for offshore diving projects.
- A modern rigid helmet made of composite resins isolates the human ear from the surrounding water medium. It is considered that sound waves penetrate the helmet and the layer of air but reach the eardrum partly absorbed and scattered. DMAC 06 / point 6 says:
  "*Helmeted divers are considered to have sufficient attenuation from the helmet itself to allow them to be safe from any known sonar in commercial use with frequencies above 500 Hz. In lower frequencies (<500 Hz) the minimal audible threshold is approximately 40 dB lower in humans in air than in water, and the estimated level of sound attenuation of divers helmets is between 25-30 dB over the same frequency. Therefore the divers helmets offers no advantage for hearing protection over the wet-suit hood for low frequencies".*

Several studies regarding sound attenuation by diving helmets have been performed by doctor Parvin from the UK Defence Research Agency using a KMB 17. Two microphones were installed in the helmet: 1 at ear level, 1 at mouth level, and 1 hydro-phone in the water adjacent to the helmet. The curves resulting from these experiences are below:



Notes:

- The curves resulting from the experiences confirm what is indicated by the DMAC.
- The attenuation was better at ear level. The reasons for this are reported "not clear" doctor Parvin said: "it may be due to the additional attenuation of pressure at the ear by the helmet liner, or possibly by preferential transmission through the helmet visor\demand valve body to the oral-nasal".

The tools used on the worksites are generally noisy and must be considered as the main source of harm to the ear. It must also be highlighted that these tools emit audible and inaudible sounds, which can vary depending on the surrounding.

In chambers

Several tests performed in chambers have given the following results that can be considered as average values. However, it must be noted that the noise in the chamber also depends on the operator.

Step	***Noise level: dB @ re. 20 µPa***
Compression from surface	108 to 145.3
Ventilation (removal of CO_2 in excess)	118
Ascent	108

- Some frequencies between 300 and 4800 have been reported during these phases. Note that these values correspond to short periods.
- Noise levels were increased from 93.4 to 96.7 dB during the helmet flushing
- Communication requires a noise level at about 15 dB above the background to be audible. The levels of noise during communication was between 100.2 and 106.9 dB.

Diving helmets

Several studies have been done to analyse the noise produced by regulators and exhaust of several helmet models during a dive. In the comparison on the next page 3 different types of helmets are considered:

I. KMB 17 B is a helmet manufactured by Kirby Morgan that uses a demand valve regulator and a double exhaust valve system. The breathed gas is exhausted using whiskers ending underneath the ear level. It corresponds to the majority of the models used for air/gas surface orientated diving.

II. "Dirty Harry" is a helmet designed to dive into unhealthy surroundings produced by Divex. It is a demand valve helmet with a closed-circuit breathing system whereby the diver's breathed gas is returned to the surface and exhausted to the atmosphere rather than exhausting into the water. This is done using a "helinaut" type valve. Similar models are used in saturation.

III. AH3 is another helmet commonly used to dive into unhealthy surroundings produced by Divex. It is today replaced by the AH5 that has a similar design. They are continuous flux helmets with the adjustable exhaust valve situated near the back of the ear on the right side of the unit.

	KMB 17B 	Dirty harry 	AH3 
Noise levels at normal work - dB (A) re. 20 µPa	91.1	78.7	90 to 100
Noise during ventilation - dB (A) re. 20 µPa	96.7	101.6	104.1
Noise during communication - dB (A) re. 20 µPa	101.6	105.8	108 to 110

Tools commonly used by divers

Noise levels generated by underwater tools have been measured in numerous studies that indicate a high potential for auditory damage. The chart below shows the results for some of them.

Source of noise	***Noise level in water dB re. 1 µPa @ 1 m***	***Noise level in helmet dB (A) re. 20 µPa***
Chainsaw (Stanley CS11)	162	101.5
Disk grinder (Stanley GR24)	158	111
Rock breaker (Stanley B67)	180	112.6
Rock chipper (Stanley CH18)	163	111.5
Hand drill (Stanley DL08)	159	109.5
Impact wrench (Stanley IW16)	167	107.7
Oxy arc	148	100.7

Sounds emitted on diving surface supports

Noise levels recorded on deck are generally high, particularly during installation or repair jobs:

- Diesel generator: 105.6 dB
- Compressor diesel: 94 to 100 dB
- Diving compressor: 82 dB
- Diesel engine room : 120 dB
- Welding machine: 111 db

Sound emitted by simultaneous activities

The records displayed in the table on the next page are average values that should only be considered indicators.
It must be taken into account that the emitted noises are dependant on the size of the material used.
Also, noise levels generated by activities like drilling or seismic surveys are very high and can be heard far from emission sources. For example, the Diving Medical Advisory Committee (DMAC) document DMAC 12 says that recent incidents have demonstrated that divers may experience significant adverse effects at distances of up to 27 km (16.8 miles) from the seismic source, which is a considerably greater distance than was previously recognized. Regarding this point, the safe distance recommended has widely evolved, taking into account new studies and incident records. As a result, the

latest revision of DMAC 12 says that a risk assessment must be conducted between the clients and operators involved and the seismic and diving contractors in advance of any simultaneous operations when diving and seismic activity are planned to occur within a distance of 30 km (18.6 miles). In the initial DMAC 12 guideline, published in 1979, the safe distance was 1500 metres.

Source of noise	*Noise level in water dB re. 1 µPa @ 1 m*	*Noise level in helmet dB (A) re. 20 µPa @ 1000 m*
Seismic survey using air guns	240	154
Dredging	185	99
Oil rig drilling	159 - 167	81
Pilling using hydraulic impact hammer	215	Not available

SONAR and other acoustic tools

Three categories of SONAR (Sound Navigation and Ranging) are usually considered:

- Low-frequency SONARs emit sounds below 1000 Hz and can have very long ranges (up to 200 miles). Militaries and scientists use them for deepwater surveys and mapping.
- Medium frequency active SONARs emit sounds at frequencies between (1000 and 10,000 Hz). These systems are designed to provide tens of miles detection. They are also used by militaries and for mapping.
- High-frequency active SONARs operate between approximately 30,000 and 500,000 Hz (30 kHz and 500 kHz); these systems allow greater resolution as the frequency increases. However, they are only effective over short distances due to the rapid attenuation of high-frequency sounds in seawater. These systems are commonly used for pipeline inspection, object detection, acoustic imaging, guiding, mapping, etc. The majority of the acoustics used with divers are high frequencies.

Sonar and acoustic tools are using audible frequencies and also infra sound and ultrasounds:

- Absorption of Infra-sounds (< 20 Hz) by fluids and solids is low, which allows for the use of these frequencies for long distances. For example, in air, an infrasonic wave at a frequency of 10 Hz decreases only by 0.1 dB per kilometre, when an audible frequency sound at 1 kHz decreases by 10 dB per kilometre. Because of this low absorption, the protection against these emissions is quite impossible. This fact is confirmed by the results regarding the attenuation of noises in a helmet done by S. J. Parvin. As for all noises, the physiologic effects of the infra-sounds are proportional to the level absorbed. A recent study recommends no more than 102 dB for 8 hrs of exposure at frequencies between 2 and 50 Hz with a limit of 145 Hz during concise periods.
- Ultrasounds (20 kHz and above) easily reflect the objects encountered because their mitigation in the solids and liquids are more important than the audible sounds and, of course, the infrasounds. Due to this characteristic, the isolation from ultrasound is easier than from infra-sounds. Nevertheless, some studies have demonstrated that exposure to ultrasound above 155 dB may produce heating effects harmful to the human body. It has been calculated that exposures above 180 dB may lead to death. There is no evidence that exposure below 120 dB is dangerous for hearing. But many reports confirm the permanent loss of hearing to workers exposed to emissions above this level.

There is no report indicating health problems from inspection tools, positioning tools, and other short range tools commonly used by the diving teams.

Symptoms

Post dive visible signs and symptoms of hearing loss may include:

- Tinnitus
- Difficulty hearing in noisy areas.
- Sensation of muffling
- Difficulties understanding words, especially when there is background noise.
- The need to ask others to speak more slowly, clearly, and loudly.
- Memory disturbances
- Intellectual impairment
- Depression

Post dive signs of severe cases may include:

- Permanent sensorineural hearing loss
- Dizziness
- Nausea

Exposure to high levels of underwater sounds may include:

- Blurred vision
- Lightheadedness
- Vibratory sensations in hands, arms, and legs
- Tremors in upper extremities
- Pain cochleo-vestibular
- Change in the rate of respiration
- Skin tension

Most existing reports focus on acute injuries rather than chronic disability. Nevertheless, several medics consider that repeated hyperbaric exposure to noisy surroundings can be responsible for hearing loss.
For information, the patterns of hearing loss observed by specialists include:

- A major loss across all frequencies from 250 to 8000 Hz
- A linear decrease in auditory acuity as frequency increases
- Relative preservation of auditory acuity at the lower frequencies with a precipitous fall off at higher frequencies

Treatment

On the work site, except determining the nature of the hearing loss, there is nothing the team can do. In their book "Diving medicine" doctors Bove and Davis said:
"The first step is to determine whether a true hearing loss exists when a patient complains of hearing loss, stuffiness of the middle ear, or tinnitus. … In the field, tuning forks, watches, and the whispered and spoken voice must be used. If a hearing loss is discovered, it is imperative to determine whether this loss is conductive or neurosensory because neurosensory losses may require more urgent therapeutic intervention".

As explained before, the ear is a fragile and complex organ, and only a competent doctor can determine the extent of hearing loss. For this reason, the patient must be transferred to a specialized unit where audiometry and other examinations shall be used to determine the damages.

It has been proved that impairments to the external and middle ear can be reversible. However, if this trauma is repeated over lengthy periods, the changes can become irreversible.

Damages to the inner ear may come from damages of the acoustic nerve, which carries information from the inner ear to the brain, or from damage to the cochlea (hair cells or other elements), from where sound waves are translated into nerve impulses to the brain. The cells composing these organs are unable to repair themselves, and the resulting hearing loss can be permanent because there are no known treatments to restore auditory cells or acoustic nerves.

Prevention

Noise exposure must be integrated into the risk assessment to decide whether the operation can be done safely, or must be reorganized, or cancelled. The report HSE RR735 "Review of diver noise exposure" identifies three fundamental approaches to reducing noise exposure:

- Elimination or reduction of noise at source
- Reduction of environmental noise at the ear
- Wearing hearing protection.

Limitation of exposure durations

The UK HSE diving information sheet #14, "noise exposure of working divers", recommends calculating an individual's noise dose taking into account the level of noise and the duration of exposure as explained in the guidance "Control of Noise at Work Regulations 2005" (CoNWR05). A "noise exposure calculator" (see next page) has been developed in 2007 by the UK HSE for this purpose.

Base on researchs the UK HSE has retained the following values:

- Lower exposure action values:
  - Daily or weekly exposure of 80 dB

- Peak sound pressure of 135 dB
- Upper exposure action values:
  - Daily or weekly exposure of 85 dB
  - Peak sound pressure of 137 dB
- Exposure limit values:
  - Daily or weekly exposure of 87 dB
  - Peak sound pressure of 140 dB

UK HSE Noise exposure calculator

Sound pressure level, L_{Aeq} (dB)	*Duration of exposure (hours)*								*Total exposure points*	*Noise exposure $L_{EP,d}$ (dB)*
	¼	*½*	*1*	*2*	*4*	*8*	*10*	*12*		
105	320	625	1250							
104	250	500	1000							
103	200	400	800							
102	160	320	630	1250						
101	125	250	500	1000						
100	100	200	400	800					3200	100
99	80	160	320	630	1250				2500	99
98	65	125	250	500	1000				2000	98
97	50	100	200	400	800				1600	97
96	40	80	160	320	630	1250			1250	96
95	32	65	125	250	500	1000			1000	95
94	25	50	100	200	400	800			800	94
93	20	40	80	160	320	630			630	93
92	16	32	65	125	250	500	625		500	92
91	12	25	50	100	200	400	500	600	400	91
90	10	20	40	80	160	320	400	470	320	90
89	8	16	32	65	130	250	310	380	250	89
88	6	12	25	50	100	200	250	300	200	88
87	5	10	20	40	80	160	200	240	160	87
86	4	8	16	32	65	130	160	190	130	86
85		6	12	25	50	100	125	150	100	85
84		5	10	20	40	80	100	120	80	84
83		4	8	16	32	65	80	95	65	83
82			6	12	25	50	65	75	50	82
81			5	10	20	40	50	60	40	81
80			4	8	16	32	40	48	32	80
79				6	13	25	32	38	25	79
78				5	10	20	25	30	20	78
77					8	16	20	24	16	77
76					6	13	16	20		
75					5	10	13	15		

Instructions:

- For each task or period of noise exposure in the working day look up in the table on the left the exposure points corresponding to the sound pressure level and duration (e.g. exposure to 93 dB for 1 hour gives 80 exposure points).
- Add up the points for each task or period to give total exposure points for the day.
- Look up in the table on the right the total exposure points to find the corresponding daily noise exposure (e.g. a total exposure points for the day of 280 points gives a daily noise exposure of between 89 and 90 dB).
- Another daily noise exposure calculator is available at the following link: *http://www.hse.gov.uk/noise/calculator.htm*

Limitation of sound pressure

The chambers should be isolated from external noise. NORSOK standards U-100 suggest 60 dB maximum in a sleeping chamber and 65 dB in a living chamber.

Specific mufflers must be installed in chambers to reduce the noise during the compression and decompression.

Noisy equipment such as those using thermal engines should be far from the dive station.

Safe distances must be considered in case of use of powerful tools like seismic, pile driving hammers, drill rigs and similar tools. DMAC 12 provides the following guidelines:

1. *Where possible, plans should be made to avoid overlapping seismic and diving activities. Where this is not possible, the activities should be prioritised and a simultaneous operations (SIMOPS) plan developed.*
2. *Where diving and seismic activity are scheduled to occur within a distance of 45 km (28 miles), it would be good practice for all parties to be made aware of the planned activity where practicable. This should include clients/operators, diving and seismic contractors.*
3. *Where diving and seismic activity will occur within a distance of 30 km (18.6 miles) a joint risk assessment should be conducted, between the clients/operators involved and the seismic and diving contractors in advance of any simultaneous operations. The risk assessment should consider ramp-up trials as well as other risk control measures e.g. reduction in source sizes, changes to firing intervals, timeshare/prioritisation etc. Seismic operators should consider whether a source output modelling study should be undertaken to predict sound pressure levels at diving locations. If so, these sound pressure levels should be considered together with other relevant factors in the risk assessment.*
4. *The maintenance of effective communication and co-operation between the seismic vessel and the diving vessel is essential. If the risk assessment generates a requirement for a ramp up trial, it should define the start point or location at which the trial commences taking into account the planned movement of the vessel and an appropriate predetermined communication plan between seismic party manager and diving supervisor.*
5. *The minimum safe distance, as determined from the risk assessment or testing outlined above, should not be compromised by either party.*
6. *There should be regular effective communication between the seismic vessel and diving vessel so that those in control of seismic and diving operations are aware of each other's work programmes. A communications check should be conducted between vessels at a pre-defined regular frequency in order to reduce the chance of an unknown communications failure.*
7. *Should any member of the diving team in the water suddenly experience discomfort, the seismic source should be turned off immediately or the bell run terminated if a request is made to do so. The SIMOPS plan should include contingency arrangements for this situation.*
8. *Following the risk assessment and any ramp-up trials local factors may change. This combined with individual diver susceptibility may produce the need for further risk assessment and a management of change process.*
9. *The health impact of exposure to noise in the underwater environment is difficult to assess. A diver's exposure should be terminated if the noise level:*
   - *interferes with diver communications;*
   - *is considered to exceed acceptable noise exposure levels;*
   - *induces discomfort; or*
   - *places the diver at risk in any other way.*

   Diving operations may continue if none of these criteria for terminating diving operations are present, including diving within 30 km (18.6 miles) of seismic surveying operations.
10. *Diver reports suggest that communications problems may often provide the earliest and most reliable/objective indication that the underwater noise from a seismic source has reached an unacceptable level. It is therefore strongly emphasised that the seismic source must be turned off immediately or the bell run terminated if the noise level compromises communications between the diver(s) and diving supervisor. In order to conduct diving operations safely there must always be good communications between the divers in the water and the supervisor in dive control.*
11. *When simultaneous operations are conducted, the diving contractor should generate and submit a short online Report of Simultaneous Seismic and Diving Operations at www.dmac-diving.org/data. DMAC will periodically review the data gathered from such reports.*
12. *Organisations which provide consent for seismic operations may wish to take into account the potential impact of seismic activity on divers and consider whether a requirement for monitoring the area for new diving activity is appropriate.*
13. *Diving contractors and clients/operators should seek to ensure they are aware of planned or consented seismic operations using all reasonable means. For example, in some jurisdictions survey consent details are made publicly available e.g. the UK Kingfisher Bulletin; the Norway NPD Seismic Survey Notification System etc.*

Tools used for construction jobs like grinders, drillers, impact wrenches, and others, should be built according to official norms (BS, CE, etc.). It is a good practice to have information about the noises they emit. Notice that the pneumatic tools are emitting more decibels than hydraulic ones (average 7 dB more, according to a study from the "Institut für Arbeitsschutz der Deutschen" (Germany).

Specific ear defenders must be worn on the dive station and in the chamber if this one is too noisy. For those who need to communicate with the dive control or the chamber operator, a set must be integrated into this material. It must be understood that the communications must be clear all the time, even in noisy surroundings.

The quality of helmet components like earphones, microphones, and head cushions have to be considered.

SONAR concerns

Low-frequency SONARs are not normally used on diving support vessels. But, some mapping operations could be in progress in the vicinity of the job site. These simultaneous operations have to be avoided, and the supervisor has to ensure a safe distance between the sonar and the diver. Regarding this point, the US Navy manual rev. 6 says:
"If possible, you should avoid diving in the vicinity of low-frequency sonar (LFS). LFS generates a dense, high-energy pulse of sound that can be harmful at higher power levels. Because a variety of sensations may result from exposure to LFS, it is necessary to inform divers when exposure is likely and to brief them regarding possible effects…"

DMAC 06 says the following for the calculation of distances:
"It is not possible to set general safe distances with accuracy and appropriate figures must be calculated for each type of sonar or noise exposure."

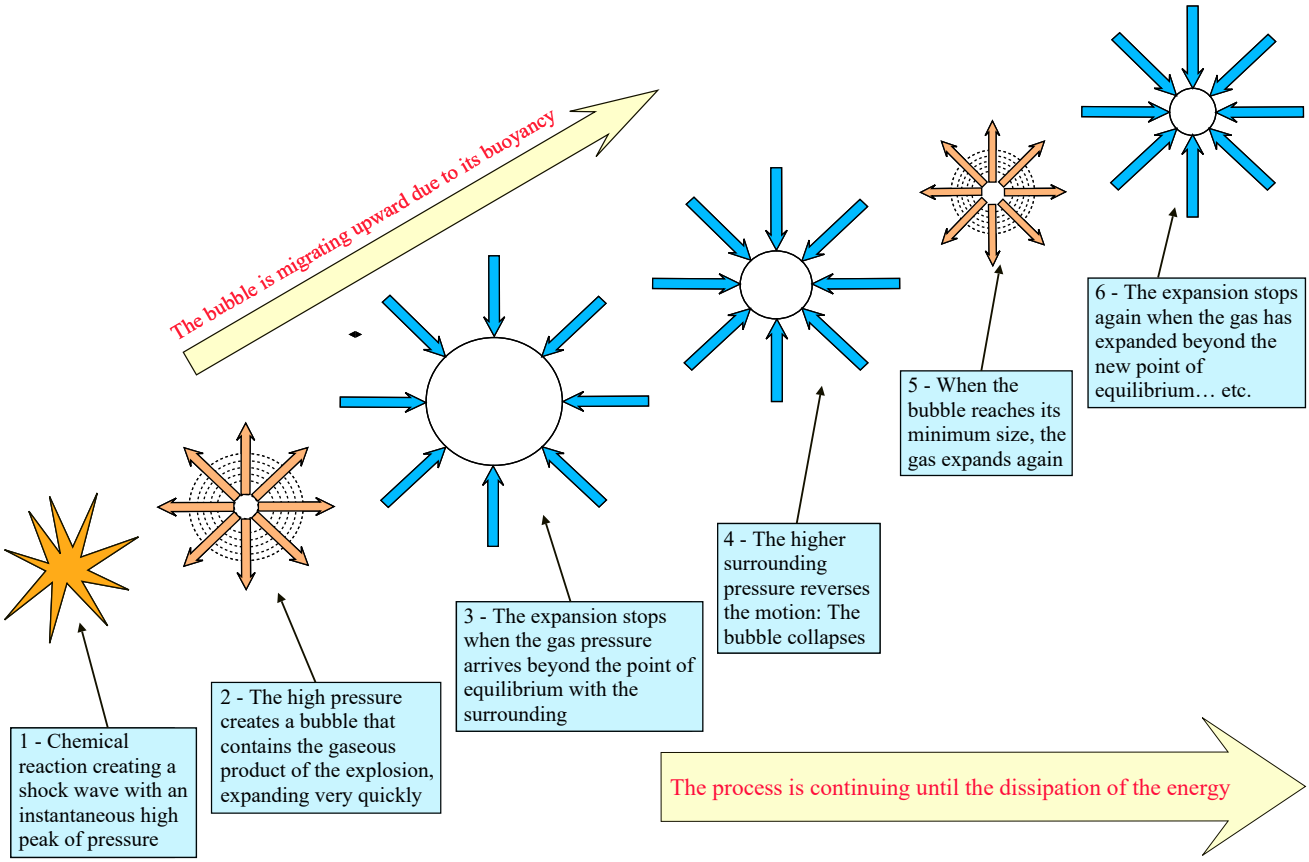
Diving Support Vessels are designed with SONARs that are normally not harmful to divers. Nevertheless, a good practice is not using these systems during the time the divers are in the water and making sure they will not be activated by mistake. Regarding this point, DMAC 06 states:
"The frequencies used in ultrasonic SONARs (> 250 KHz) are above the human hearing threshold. Because the power of ultrasonic sonar rapidly falls off with distance, a safe operating distance is 10 metres or greater. Diving may be conducted around this type of sonar provided the diver does not stay within the sonar focus beam.
None of the above avoids the need for positive safety measures to be adopted when divers are working on or very close to sonar sources which are inactivated. The possibility of accidental activation must be precluded."

Underwater explosion

Description

Explosions are physical, chemical, or nuclear reactions that involve the rapid release of considerable amounts of energy.

In the water, the detonation of a chemical mix leads to the formation of a gas bubble. This bubble is spherical in shape and may have an initial temperature of 30,000 C and a pressure equivalent to 50,000 atmospheres. This peak pressure is reached within a very short period of time, on the order of milliseconds after detonation.
This bubble tends to expand, compressing the water in its immediate vicinity. The bubble continues to expand beyond the point of equilibrium due to the inertia of the water. Then the effect of the surrounding water pressure applies to the bubble, which compresses quickly. Following this collapse, another pulse is generated with the re-expansion of the bubble as a result.
The process of expansion and collapse continues until the energy is dissipated. The result is an oscillating system.



The initial high-intensity shock wave is the most dangerous. It loses its intensity as it travels outward from the explosion source.

During an explosion in the air, the atmosphere is compressing, and it is absorbing some of the explosive energy. In an underwater explosion, the surrounding water doesn't absorb the pressure like air does but moves with it. An underwater explosion transmits pressure with greater intensity and for a longer duration. Nevertheless, the effects of the explosion will decrease with the distance.

The composition of the seabed and its topography can modify the pressure of the waves. A soft bottom tends to absorb the energy, while a hard bottom tends to reflect and amplify it. The rocks and other obstacles can create interferences to the waves: In some cases, colonies of fishes are destroyed after a massive explosion, while a few metres further, other colonies closer to the explosion remain intact. Also, at great depth, the shock and pressure waves are drawn out by the greater water volume and are thus reduced in intensity. An explosion near the surface is not weakened to the same degree.

During the explosion, the blast wave passes through the body as it is of a similar consistency to water. Molecules are displaced very little except in gas spaces, which are instantaneously compressed by the pressure wave. Due to this effect, the anatomic structures containing air, like lungs, enteric tract, nasal, sinuses, and middle ear, are very vulnerable to blast injury. (Note that for the lungs, the transmission of the wave is directly through the thoracic wall).

Symptoms:

The severity of the initial symptoms and signs of immersion-blast casualties varies as a function of the distance of the subjects from the explosion and their positions in the water. The damage can occur anywhere from the lower part of the esophagus to the rectum. The survivors and rescuers involved in an underwater blast have reported:

- Sudden acute abdominal pain is likened to a kick in the stomach.
- Transient paralysis of the lower limbs.
- Nausea, vomiting (with or without blood)
- The sensation of an electric current passing through the body
- Testicular pain
- Chest discomfort followed by expectoration of blood or sputum (hemoptysis) and hiccups.
- Tachycardia
- Cyanosis
- Mild to severe shock
- Disorientation
- Loss of hearing
- Delirium
- Unconsciousness
- Amnesia
- Rectal bleeding may be apparent.
- Death can occur quickly.

Numerous reports indicate that the gravity of the injuries are usually apparent within 6 to 12 hours after exposure.

Treatment

The management of the patient is similar to that for one who is suspected of having total body trauma. Hence the patient must be transferred to a specialized unit, even with no visible symptoms.

- Close contact with the doctor must be maintained.
- The patient should not have any oral intake and be maintained on intravenous fluids with gastric suction until the full extent of the damages is visible.
- Allow the patient to adopt the most comfortable position.
- Treat for shock, plus oxygen treatment
- Hyperbaric oxygen therapy is considered beneficial.
- Antibiotics and tranquillizers by intravenous, depending on the decision of the doctor.
- Medical evacuation to be organized

Prevention

Respect for procedures and good practices is the best way to avoid explosions during the job.

Diving in the vicinity of seismic or any site where explosives are likely to be used is forbidden.

DMAC 12 “Safe diving distance from seismic surveying operations” says:
“Where diving and seismic activity are scheduled to occur within a distance of 45 km (28 miles), it would be good practice for all parties to be made aware of the planned activity where practicable. This should include clients / operators, diving and seismic contractors.

Precautions must be implemented when diving in close vicinity of live installations, or when working on them.

- IMCA D 006 “diving operations in the vicinity of pipelines” says point 5.3:
  “Wherever possible the work should be carried out during planned shutdowns with the pipeline depressurised to ambient. All work should be detailed in written procedures derived from a risk analysis. Changes to approved procedures should be appropriately controlled.”
- Also, IMCA D 044 “Diver access to subsea systems” says:
  “… when working on any sub sea system containing liquid or gas under positive or negative pressure, there should be no pressure differential, relevant to the seabed ambient, trapped within a space or void“.
  “In many cases, diving operations cannot commence until the topside installation has firstly applied primary

isolation(s) to the main energy source(s), following which, manual and tangible final isolations will then need to be applied at the sub sea work site location. All isolations need to be proven, to demonstrate to diving personnel that protection from all potential energy sources has been established".

- IMCA D 019 "diving operations in support of intervention on wellhead and subsea facilities" indicates:
  "Adequate and appropriate isolations must be in place. Where practicable such isolations should be tested prior to the operation taking place. General advice on good isolation practices is contained in the Oil Industry Advisory Committee publication "The Safe Isolation of Plant & Equipment"

Underwater cutting using exothermic lances or similar systems are usual operations that can trigger explosions: Some alternative methods must always be studied to not expose the diver to potential risks.

- IMCA D 003 oxy arc cutting operations underwater indicates in point 5:
  "Oxy-arc cutting involves the use of large quantities of oxygen and generates hydrogen during the process. When the proportion of hydrogen to oxygen reaches a certain level,an explosive mixture is formed which will ignite when the arc, or a spark, reaches it. The following factors are known to cause, or contribute to the possibility of, an explosion during oxy-arc cutting.
- Some client and organizations promote alternative cutting methods. For example, IOGP says the following 471 says the following:
  " The decision to use burning should always be considered against other methods and the risks identified, assessed and controlled. Many alternative cutting methods are safer and, in some cases, faster and more cost effective than oxy-arc burning. The use of unmanned submersibles or ROVs with power-operated saws can be considered and the choice to use a diver in an oxy-arc burning scenario should be balanced with alternative methods.

In the case that burning is the only possible option:

- The preparation of a burning operation must be very meticulous. All the elements which could lead to explosion have to be removed, and the material to be cut must be well known - IOGP 471 "Oxy-arc underwater cutting recommended Practice" recommends point 9:
  "No burning operation shall be executed unless planned and managed in accordance with the requirements of this RP. The metal in the cut area should be thoroughly cleaned before burning begins…. Paint or other petroleum based coatings can produce a flammable gas when not completely burned; mixed with oxygen this can create an explosive environment.
  "Proper cleaning prior to cutting will reduce the amount of oxygen consumed while making a cut."
  "An accurate assessment of the burning requirements is always required…"
- IOGP 471 recommends in point 12:
  "The diver must ensure that there are no hydrocarbons present that can ignite during the burning process".
- IMCA D 003 indicates:
  "Blow backs are spontaneous explosions of varying intensity which appear to be generated at the cutting point"
- IMCA D 003 recommends in point 5.3
  "Depending on the substance involved,various gases or fumes can be released during cutting which can contribute to the mechanism of blow back..."

Burning requests also to make sure that there will not be any trapped gas able to lead to an explosion.

- IOGP 471 "Oxy-arc underwater cutting recommended Practice" indicates in point 9.1:
  "It is a mandatory requirement that a suitable gas path for the elimination of volatile gases from adjacent to and above the cut is achieved prior to commencing the oxy-arc cutting operation".
- IOGP 471 recommends in point 12:
  "Underwater burning produces a combination of pure oxygen and hydrogen gases as a by-product of electrolysis and heat generated over 2000°F. When trapped in a confined, or unvented, area this gas mixture will produce a serious explosion when ignited"
- IMCA D 003 recommends in point 6.7:
  "if the presence of a flammable gas is suspected before the cutting operation starts, the cutting of the vent hole should be carried out using cold cutting techniques".

In the document *"International Consensus Standards For Commercial Diving And Underwater Operations"*, the association of diving contractors (ADCI) indicates similar procedures similar to those indicated above, plus precautions if explosives are to be used.

Many areas have been the theatre of battles with all the associated consequences from such scenarios. For this reason, war explosives must be considered in some areas because old ammunitions remain active and are likely to explode at any moment. Note that:

- Munitions dumped in the water by troops retreating or plane/boats in difficulty are not localised.
- Unexploded underwater war mines, shells, and torpedoes are still able to cause a lot of accidents. Also, a lot of war wrecks full of unstable explosives are still undiscovered.

Handling of old ammunition requires specific knowledge. For this reason, old ammunitions discovered by the divers must not be removed by them. The Area must be secured, and the discovery clearly indicated to be found easily by the specialists (usually Navy divers) who will proceed with the neutralization. If the specialists consider that an evacuation is necessary, an ROV may be used for this purpose.

High Pressure water jet injuries

High pressure water jet injuries are not directly linked to diving or the surrounding of the diver, but to a particularly powerful and dangerous tool. This chapter has been incorporated with the diving accidents because this tool is in use on the majority of the work sites, and the wounds it can create can lead to infections which are likely to become life threatening.

Description

High pressure water jets are devices designed to provide pressures up to 1000 bar, depending on the model used and the usage planned. These pressures are obtained through specific piston pumps and a nozzle that concentrate the jet to accelerate the water flow (based on the Venturi effect). To avoid that the pressure delivered pushes back the diver, a counter jet is provided that delivers the same pressure and is isolated from the diver in a tube commonly called "diffuser". These tools can be used for various works such as:

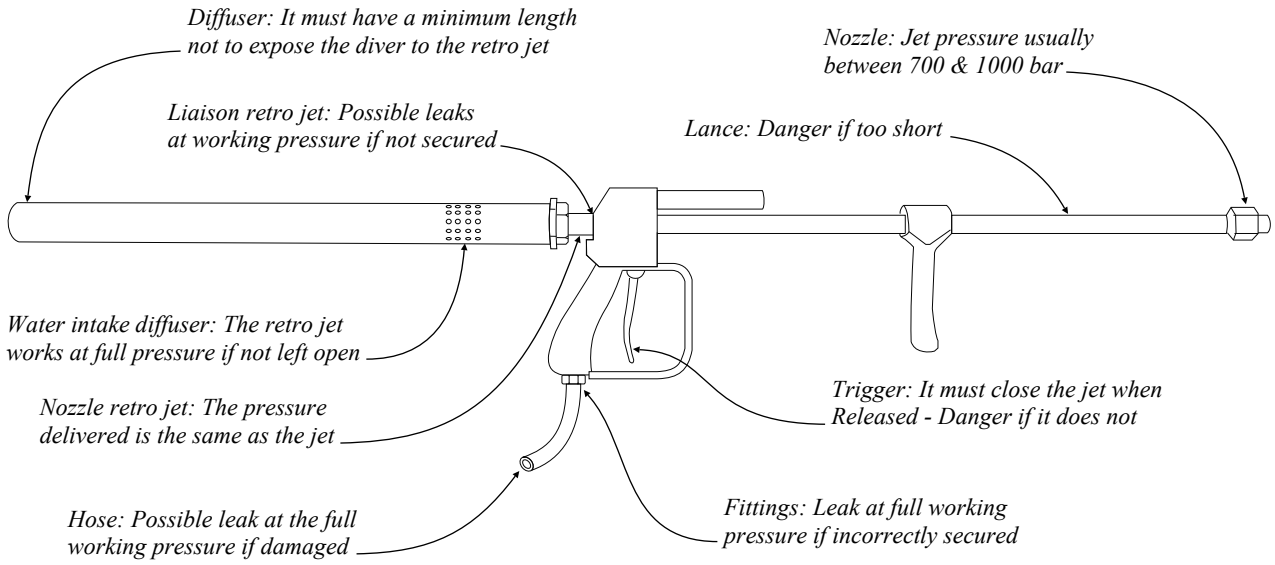
- Marine growth removal;
- Paint and coating removal;
- Seabed material removal by breaking down clays and stiff soils for air lifting;
- Cutting several materials such as concrete and steel;

Note that ultra high pressure (UHP) water jets that operate above 1,700 bar pressure are being increasingly used by the offshore industry for the removal of surface coatings, cleaning, and cutting.

HP water jet systems used by divers are typically composed of the following main elements:

- Diesel or electric drive motor;
- Pressurising pump;
- HP hose;
- HP hose reel;
- HP jetting gun

The risk of accidents can come from any of these parts, as the machine deals with high or very high pressure. Regarding the diver at work, the risk of accident mostly comes from the jetting gun and the water supply hose parts at his direct proximity.



During the operations, the diver can be exposed to accidents due to mistakes, bad engineering, or bad maintenance of the pressure hose and the gun. Any direct exposure to the jet, the retro jet (if insufficiently isolated), or a leak on the gun, the hose, or a connector is likely to create wounds.
Unlike many other injuries, the wounds caused by a high-pressure water jet are frequently more extensive than they look because a large amount of water can enter the body through a relatively small hole within a short time and cause major damage:

- The skin can be penetrated by water jets even at a relatively low pressure of approximately 40 bar.
- The jet can be deflected internally or intercepted by harder tissue and cause internal injuries.

- A large amount of fluid can be injected within a short time.
- The first impression of the wound may be that it is only superficial; however, it is possible for major internal damage to occur.
- Some foreign substances can be injected with the water and contaminate the tissues.
- Because of the power of the jets, the limbs or any body part exposed can be easily cut.

Symptoms

It must be understood that injuries that look trivial at first can quickly become life-threatening.

- Bruises may appear, and the area becomes increasingly painful.
- Crush injury of the skin and destruction of deeper-seated anatomical structures.
- Lacerated tissues.
- Incised tissue, seriously damaged, even at some distance from the wound due to the impact pressure produced.
- Damaged muscles, which can swell, compressed nerves and blood vessels.
- Injected particles can spread in the tissues along with muscles, nerves, tendons, or blood vessels and create infections.
- Severed bone and amputated extremities or parts of extremities as a result.
- The development of subsequent infection is significant in abdominal and joint injuries.
- The development of fever and a rising pulse rate suggest that the injury is serious.

Treatment

The procedure consists of protecting the victim, establish medical support, and organize the evacuation as soon as possible. Note that a diver in saturation cannot reach the surface immediately and that depending on the depth of storage, the decompression can take more than 12 days if the diver is stored below 300 m.

- Examination of the injury - Take photos that should be sent to the doctor.
- Control any bleeding by applying pressure over the bleeding site and elevation of the injured limb where possible.
- Check for a local swelling.
- General examination
- Observe the patient closely and check for infection:
  The medical evacuation of a patient in saturation is not immediately possible. For this reason, he must be closely observed for the development of infection until his final evacuation. Usually, the doctor orders preventive antibiotic therapy. If, due to exceptional conditions, the doctor cannot be contacted, such treatment may have to be decided on board. A broad-spectrum antibiotic is normally provided in the DMAC 15 medical kit. However, the treatment started must be indicated to the doctor as soon as the communication is re-established.
- In case of a severed limb:
  - Control the bleeding (compression + elevation). Note that an amputated limb may not bleed profusely early in the incident as blood vessels retract into the body as a self-defense mechanism. However, severe bleeding may follow this 1st step.
  - Recover the limb cut, and preserve it:
    - Washing with sterile water or a saline solution if possible (no scrubbing and immersion).
    - Wrapping in a moist, clean material such as a sterile gauze moistened with sterile saline solution or sterile water.
    - Protection in an adequate container and cooling.
    - The decompression of the limb is to be organized with the patient as a too fast decompression will damage the tissues.
  - Medical guidelines will have to be strictly followed.
- Organize the decompression of the injured diver and his medical evacuation. Also, prepare the following essential information that must be transmitted to the doctor in charge, and also accompany the victim in a document with visible warnings regarding the nature of the injury:
  - Time of the injury.
  - The pressure of the water jet.
  - The nature of the water supply (filtered salt water, fresh water, or other…).
  - Added chemicals, if any (with data sheet if available).

- Detail of grit or other abrasive added if any.
- Detail of possible residues from water jetting operations (marine growth, corroded metal, paint, concrete coating).
- Note that the “Water Jetting Association” (*https://waterjetting.org.uk/*) provides generic plastic information cards designed for these types of accidents.

Prevention

A risk assessment must be carried out before starting the intervention to decide whether these tools are appropriate for the planned task. Also, the use of less dangerous procedures must be envisaged. Note that “Cavi Blaster” systems and similar tools, that are based on cavitation instead of very high pressures, are suitable for the removal of marine growths and are less dangerous.

IMCA D 049 recommends a material in perfect condition and comply with the last safety requirements. This guideline also recommends life support equipment, personal protective equipment, good training, in addition to safe operational procedures. Safe operational procedures include the following safe practices:

- The entire machine must be cold when the tool is deployed, and the diver relocate.
- An emergency shut down command must be in the direct proximity of the diving supervisor.
- The sensitive parts of the gun must be checked before its deployment.
- The trigger must returns automatically to the closed position when released and must not be kept in the open position by a wedged or other means;
- These tools must not be used when the visibility underwater is reduced.

Protective devices must be worn. The problem often encountered is that most these protections are not originally designed for divers and may reduce their ability to swim. Thus, the problem is not creating a risk of accident while trying to protect the divers from another one. However, new kevlar protections exist that are light, allow for free movements, and can be adapted to divers.

Electrical shocks

Quick description of electricity

Electricity is energy in the form of tiny, charged particles (electrons) that flow through a closed conducting path (a circuit) like water flowing through a pipe. Electrical current is the number of electrons flowing past a given time and is measured in AMPERES. One ampere is defined as the flow of 6,280,000,000,000,000,000 electrons (negatively charged particles) through a certain cross-sectional area of a conductor per second. The symbol of electric current is the letter I with an arrow indicating its direction.

A pressure, electromotive force, causes current flow. The pressure is measured in VOLTS.

As the current flows through a circuit, it is being opposed. This opposition to current is called resistance, and is measured in OHMS.

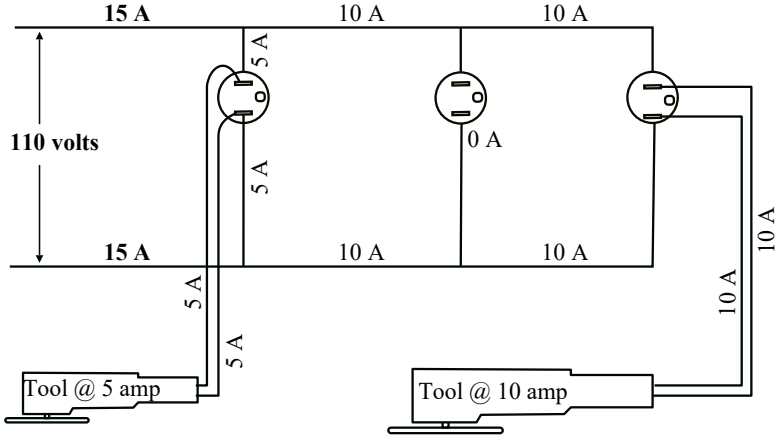
The amount of current (amperes) flowing through a circuit is directly proportional to the electromotive forces (volts) and inversely proportional to the resistance (ohms) of the circuit.
This relationship is called OHM'S LAW and is expressed by the equation I = E/R
where:

I = current in amperes
E = electromotive force in volts
R = resistance in ohms

In other words, the amount of electrical current is determined by dividing the force of the flow by the conductor's resistance. The result is current in amperes.

An energized conductor in an electrical circuit is often called “hot wire”. Electrical current flows to the loads such as portable tools and lights in a circuit through “hot” and “neutral grounded” wires. An additional conductor provides grounding to prevent user contact with the current if the insulation fails. Electrical accidents and injuries can occur when a “short circuit” (the accidental connection between two points in an electrical circuit) or “accidental ground” (unintentional connection of a current-carrying conductor to the ground) provides an alternate path through which the current can flow.

Current will not flow through an open circuit, but some current will flow through every complete conducting path provided. Current divides itself among all the loads connected to a circuit according to their resistance. Total circuit current depends on the total load.
For example:
In a circuit where a tool #1 draws 10 amperes of current and a tool # 2 draws 5 amperes, the total current is 15 amperes. If a tool # 3 is added to the circuit current and draws an additional 7 amperes of current, the total circuit current will increase to 22 amperes. In other words, added loads cause the total circuit to increase.

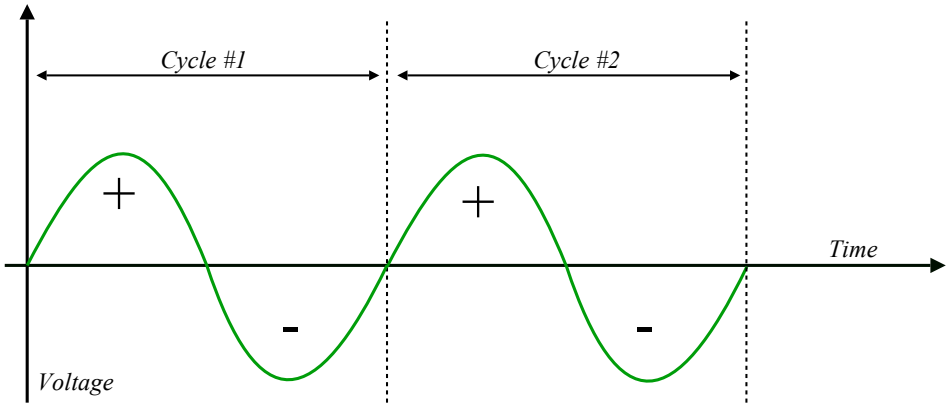


If somebody touches a hot conductor and any grounded conductor at the same time, his body becomes an added load which also increases the total circuit current. The amount of current increase will be determined by the electrical resistance of the body at that particular time. The magnitude of current flowing through the body will determine the severity of shock and extent of injury.

Direct current is a unidirectional flow of current; the electron flows in one direction only. (It is noted DC or dc)

Alternating current has the flow of electric charge which periodically reverses direction. (Noted AC or ac). One positive plus one negative alternation is called a cycle. The frequency is the number of cycles produced every second. The frequency is measured in Hertz (Hz).
The frequencies in use are between 50 and 60 Hz depending on the country:

- It is 50 Hz in Europe, China, India and in the ASEAN, except Philippines.
- It is 60 Hz in USA and Canada and the majority of the south American countries.



Electrical circuits are referred to by their voltages:

- Depending on the country, the circuits for ordinary lighting and small fractional horsepower motors are usually from 110 to 240 volts. For large motors and plant machinery, 380 to 460 volts circuits are common.
- The International Electro-Technical Commission defines 'high voltages' those exceeding 1000 volts AC and 1500 volts DC. 'Low voltages' are those below these values. The Commission has classified 50 v AC and 120 volts DC as "low-risk voltages". This classification does not mean low hazard because a shock or burn can be received from any energized circuit, no matter what the voltage is.
- "Safety low voltages" are the voltages lower than 24 volts, not because they eliminate shock hazards but because they reduce the severity of injury when shock occurs. Typical applications include hand lamps and portable electric tools powered by 6 to 12 volts. Thus, safe, low voltage devices are recommended for work in the water.

Insulators are materials that completely block the passage of electric current (Plastic, rubber, paper, air, etc.).

Conductors are materials that easily allow the flow of electric current (Copper, steel, and all metals, etc.).

"Conductivity" is a measure of the ability of fluids and solids to pass an electrical current.

- Note that the ability to carry current is better at warm than at cold temperatures.
- Distilled water does not contain dissolved salts, and it does not conduct electricity. Thus, water conductivity depends on the presence of inorganic dissolved solids such as chloride, nitrate, sulfate, and phosphate anions *(ions that carry a negative charge)* or sodium magnesium, calcium, iron, and aluminium cations *(ions that carry a positive charge)*.
- Because the sea has about 3.5% of dissolved salt and other particles, it has a low electrical resistance. In contrast, depending on the particles in suspension, freshwater has a relatively high electrical resistance, so a low conductivity.
- Referring to what is indicated, a leak of electricity in salt water will be transmitted to the ground, and a leak of electricity in freshwater will not pass easily to the ground.

Description	***Electrical resistance Ω***	***Comments***
Plastic PVC	100 000 000 000 000 000	Plastic PVC is a good isolator commonly used by electricians. At the opposite end, copper is one of the best conductors used for electrical cabling. These 2 values can be used as references of isolator and conductor in this chart. The chart clearly shows the evolution of the skin's resistance from dry to fully wet, with a resistance of 666.66 less than dry when in the water. This fact has to be taken into account during the diving operations, and of course, on the deck.
Dry wood	200 000 to 200 000 000	
Human skin "Dry"	100 000 to 500 000	
Human skin "Wet"	1000	
Human skin in the water	150	
Copper	0.02	

Electrical shock occurs when the body becomes part of an electrical circuit:

- Contact with both the hot and neutral conductors of a circuit: In this case, the body of the victim is then just like a light bulb filament or the windings in a motor. It becomes a load between the conductor through which the current will flow.
- Short circuits: These occur when the hot conductor contacts metallic parts (handles or frames of a tool, for example), causing them to become energized. The victim can receive a shock if he/she is in contact with these energized parts.

- Contacting a current-carrying conductor while it is in contact with the ground. This is unintentional grounding.
- An electric shock can happen to people or animals that find themselves immersed in an electric field that has been established in the water.

It must be noted that working boats and offshore or onshore facilities are potential sources of unintentional leakage of electrical energy into the water:

- Some leakages like those from underwater power cables or transformers can be massive.
- Several reports are indicating that electrical elements like transformers may explode.
- Electricity is used underwater for burning, welding, and other activities.
- Electricity is permanently present on the diver (light, communications).
- Electricity is the base of systems like" impressed current systems", which are intentional electric fields designed to protect from corrosion.

All of these elements can impact the safety of the diver.

Underwater transformer

Effects on the human body

The extent of injury received from an electric shock is determined by the amount of current that passes through the body, the path it takes, and the length of time the victim is in contact with the current.

- Voltage never killed anyone. It is the current that does the damage.
- The safe current/time limit for a victim to survive at 500 mA is 0.2 seconds and is 2 seconds at 50 mA.

One effect that may be encountered and indicated in the chart below is where an electrical source applies sufficient power to cause some of the human body's muscles to contract involuntarily. It is this effect that results in the phenomenon commonly called the 'let go' level. It should more correctly be called the 'can't let go' level as its commonest manifestation on land is where a person catches hold of an item that is electrically live and is unable to let go of it because the muscles in their hand and forearm have been involuntarily contracted

Effects of electric current on the human body. Note that the current values are given in milliamperes (thousandths of an ampere), so 200 milliamperes equal to 0.2 of an ampere.

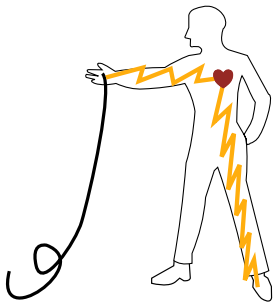
Current *(milliamperes)*	***Effects***	***Classification***
1 or less	No sensation	Trivial
1 to 8	Tingling. Individual can let go at will because muscular control is not lost.	
8 to 15	Painful shock. Muscular control is not lost.	Serious but not lethal
15 to 20	Painful shock. Muscular contractions with loss of control. Cannot let it go.	
20 to 50	Severe painful and uncontrolled muscular contractions. Cannot let it go.	Lethal
50 to 200	Possible ventricular fibrillation *(A heart condition that results in death)*. Uncontrolled muscular contraction. Nerve damage.	
> 200	Severe burns and severe muscular contractions with the heart clamped and stopped by the contraction of the chest muscles.	

Note that an ordinary 220-volt lighting circuit may have as much as 10 to 20 amperes current flowing through it. Most fatal electrical shocks occur because the current flow is through or near the heart, which can cause ventricular fibrillation of the heart, a condition where the heart flutters uselessly, and circulation stops. It usually results in death if immediate Cardio Pulmonary Recovery is not undertaken.

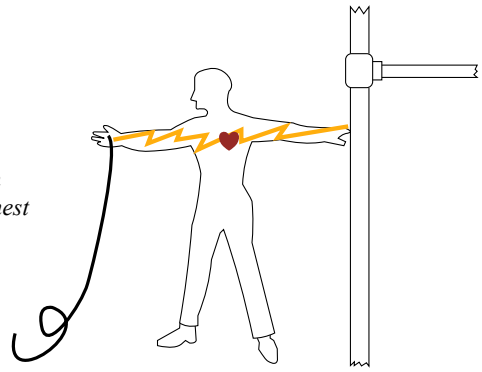
Alternating current is considered more dangerous than direct current. That is why the "low-risk voltage" is 50 volts with Alternating Current (AC) and 120 with Direct Current (DC). But, the fact is that both are potentially lethal:

- DC makes a single continuous contraction of the muscles compared to AC, which will make a series of contractions depending on the frequency it is supplied at. For the let-go threshold in Alternative Current, a current of 3 to 22 mA is required against 15 to 88 of Direct Current.
- Either Alternating Current or Direct Currents can cause fibrillation of the heart at high enough levels. This typically occurs at 30 mA of AC @ 60 Hz or 300 – 500 mA of DC.

- Contact with any AC sources has been documented to produce respiratory arrest and death from tetany of respiration muscles.
- Low-frequency AC is more dangerous than high-frequency AC.



If the path is through both arms or an arm and a leg, the current passes across the chest and either through or around the heart.



Awareness/Feeling:
An electrical fault underwater results in the creation of an electrical field surrounding the fault. This field is a sphere centered on the fault, with the intensity decreasing as it radiates outwards.
IMCA D 049 explains that experiments were carried out in 1985 with divers wearing normal equipment to determine the electrical fields' effects at sea. These tests have proven that as a diver enters this field, he will become aware of a sensation surrounding him, and as he gets closer to the source, the 'feeling' will get stronger and progressively more uncomfortable. It will normally reach a level alerting the diver to the presence of the electrical field and cause him to stop his approach well before it becomes in any way hazardous to his health.
IMCA D 049 considers it highly unlikely that a diver will ever stray into an electrical field underwater that is strong enough to offer any potential to be a possible hazard to him.
However, one possible hazard is a fault developing underwater when the diver is already in the vicinity. The likelihood of this situation arising needs to be one of the things considered during any risk assessment.

Effect of the underwater pressure:
It has been demonstrated that the human body reacts to electricity in a hyperbaric situation in the same way as it reacts in atmospheric pressure.

Symptoms

External burn injuries:

Three basic types of external burn injuries may occur in the air as a result of contact with an electric current.

- Direct contact burns occur when an electric current directly penetrates the resistance of the skin and underlying tissues.
- Arc injuries occur when a person is close enough to a high-voltage source that the current overcomes the resistance in the air.
- Flame and flash burn injuries can occur when an electric current's heat ignites a nearby combustible source.
- The specialists consider that these effects are not relevant underwater because the diver is surrounded by water. However, because diving operations are not performed in the water only and on the deck, these symptoms are explained in the appendix attached at the end of this chapter.

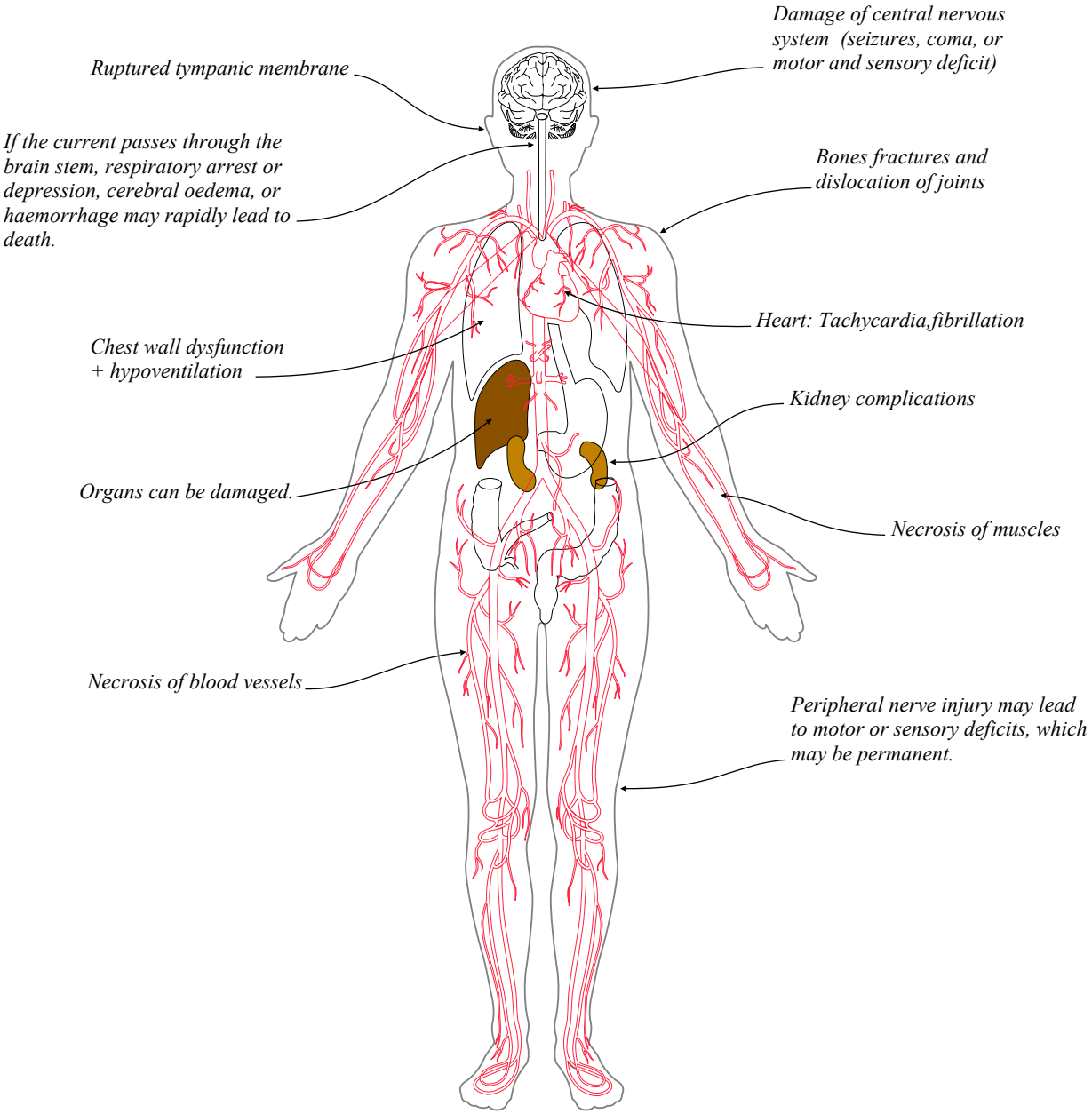
Underwater electrocution:

Injuries following underwater electrocution are mostly internal and unpredictable:

- Hypertension and tachycardia associated with a large release of catecholamines (adrenaline, noradrenaline, dopamine)
- Electrical current also may cause significant dysrhythmias, including ventricular fibrillation and asystole, and damage to the myocardium as it passes through the body. This can lead to cardiac arrest.
- Ventilation may be impaired as electrical current may produce tetany of the muscles of respiration and chest wall dysfunction. The resulting hypoventilation can lead to patient death.
- Severe muscle spasms can produce bony fractures and dislocations, even of major joints.
- Nerve tissue is an excellent conductor of electrical current and may therefore be commonly affected in electrical injuries. Central nervous system damage may result in seizures or coma with or without focal neurological findings; peripheral nerve injury may lead to motor or sensory deficits, which may be permanent. If the current passes through the brain stem, respiratory arrest or depression, cerebral oedema, or haemorrhage may rapidly lead to death.
- Ruptured tympanic membranes are reported in some electrical injuries. Hearing loss also may appear as late as 1

year after the event.

- An electrical injury can cause extensive necrosis of blood vessels and muscles. These injuries can cause immediate or delayed internal haemorrhage or arterial or venous thrombosis and embolism with subsequent complications.
- Acute renal failure is a serious complication that affects about 10% of significant direct-contact electrical injuries.



Rescuing procedure

Important note:

Before rescuing the victim, the team must be 100% sure that the rescuer's intervention is safe. Also, some important elements must be recorded for further treatment by the specialist.

- The electrical source must be identified and neutralized.
- The voltage and the amperage of the electrical source must be recorded with the duration of exposure.
- The level of consciousness of the victim during the accident, during the rescue, and later must also be recorded.

Treatment:

- If the patient is not breathing and/or no pulse, Cardio-Pulmonary Resuscitation is to be undertaken.
- Medical support to be obtained As Soon As Possible.

- Immobilization, particularly the cervical spine.
- Breathing support using O2.
- Close examination.
- Monitoring of the cardiac pulse and respiration.
- Frequent assessments of the victim (injuries may be internal and not visible).
- Medical evacuation shall be organized As Soon As Possible.

Prevention

Electrical accident prevention is based on avoiding contact with energized circuits. For that, a job site analysis and a risk assessment integrating the risk of electrical shock must be undertaken before starting any underwater operation.
This risk assessment should be based on the elements indicated above and appropriate guidelines. Among the guidelines that can be used, note those listed below, but not limited to:

- IMCA D 003 - Guidelines for oxy-arc cutting.
- IMCA D 044 - Guidelines for Isolation and Intervention: Diver Access to Subsea System.
- IMCA D 045 - The Safe Use of Electricity Under Water.
- IOGP 471 - Oxy-arc underwater cutting recommended practice.
- IMCA D 018 - Code of practice for the initial and periodic examination, testing and certification of diving plant and equipment.
- IMCA D 024 - DESIGN for saturation (bell) diving systems.
- Precautions for diving with ROV are indicated in chapter #10 of Book #2 of this manual.
- Electrical hazards are also discussed in the document *"International Consensus Standards For Commercial Diving And Underwater Operations"* published by the Association of Diving Contractors International (ADCI)

Regarding the intervention on subsea controls and umbilical systems, most guidelines suggest three ways in which the safety of the diver can be assured about possible electrical hazards:

1. ***Safe voltage:*** The voltage of any item that the diver may come in contact with must be less than a 'safe' level
2. ***Safe distance:*** If the voltage involved is above the 'safe' limit, it may still be possible to carry out the work if the diver remains a safe distance away from the energized system or equipment.
3. ***Isolation/disconnection from the power source:*** If the voltage involved is above the 'safe' level and the diver cannot be guaranteed to stay at least the minimum safe distance away from the energized components, then the only way to carry out the dive safely will be to isolate and completely de-energize the power source and the components involved.

Typical scenarios and good practices to avoid electrical accidents are provided in the guidelines indicated above. These scenarios discuss the following topics, but not limited to.

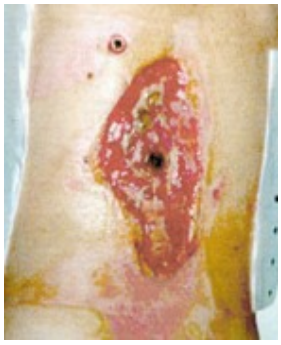
- Diving on subsea equipment or cables (the generic scenario).
- Impressed current anode systems.
- Diver carried or operated equipment.
- Wet welding, cutting, and burning.
- Inside a hyperbaric chamber.
- Inside a welding (or other) habitat.
- Divers working with ROVs.
- Welding power source.
- Safety switch or circuit breaker of welding devices.
- Burning leads or burning umbilical.
- Ground leads, wire splices, connectors & terminations.

Appendix: Electrical burns in air

Direct contact burns occur when an electric current directly penetrates the resistance of the skin and underlying tissues. The hand and wrist are common entrance sites, and the foot is a common exit. Although the skin may initially resist current flow, continued contact with the source lessens resistance and permits increased current flow.
The greatest tissue damage occurs directly under and adjacent to the contact points and may include fat, fascia, muscle, and bone. Tissue destruction may be massive at the entrance and exit sites; however, it is the area between these wounds that poses the greatest threat to the patient's life.

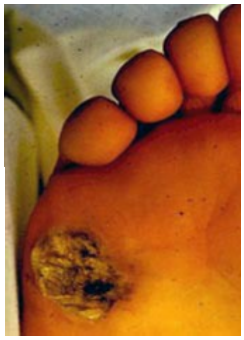
Entrance Wound.

The entrance site is often a characteristic "bull-eye" wound and may appear dry, leathery, charred, or depressed.

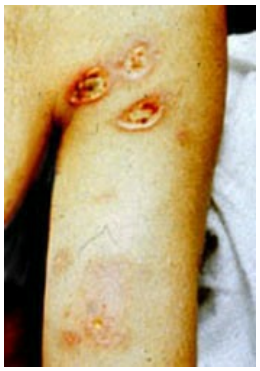


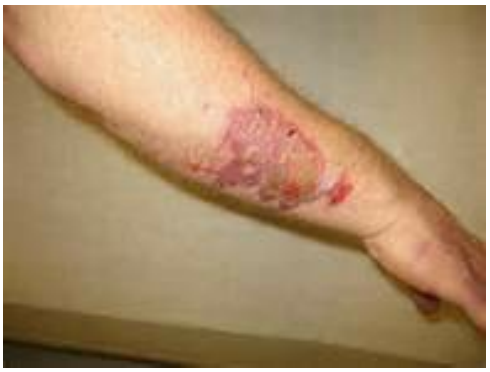
Exit Wound

The exit wound may be ulcerated and may have an "exploded" appearance where tissue areas are missing.

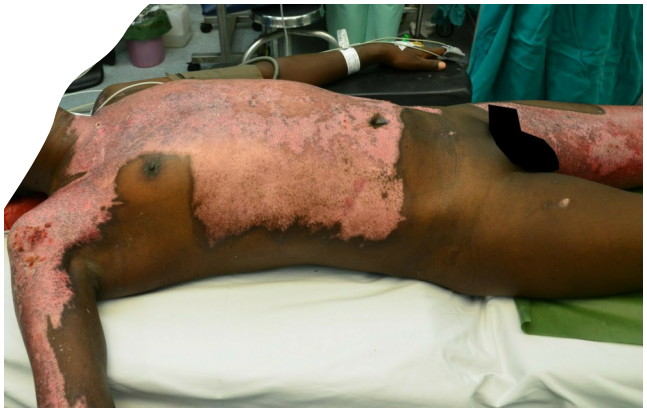


Arc injuries occur in the air when a person is close enough to a high-voltage source that the current between two contact points near the skin overcomes the resistance in the air, passing the current flow through the air to the bystander. Temperatures generated by these sources can be as high as 2000° C to 4000° C (3632° F to 7232° F), and the arc may jump as far as 3 metres (10 feet).





Flame and flash burn injuries can occur when an electric current's heat ignites a nearby combustible source. Common injury sites include the face and eyes (Welder's flash). Flash burns also may ignite a person's clothing or cause a fire in the surrounding environment. No electrical current passes through the body in this type of bum.



Internal injuries are unpredictable, often vary. They are similar to those that can happen underwater:

- Hypertension and tachycardia associated with a large release of catecholamines (adrenaline, noradrenaline, dopamine)
- Electrical current also may cause significant dysrhythmias (including ventricular fibrillation and asystole) and damage to the myocardium as it passes through the body. If the patient has suffered cardiac arrest and early rescue and resuscitation can be initiated by the paramedic, success rates are high.
- Ventilation may be impaired when electrical burns produce central nervous system injury or chest wall dysfunction. If the respiratory centre is disrupted, hypo-ventilation can lead to immediate patient death. Contacts with AC sources have also been documented to produce respiratory arrest and death from tetany of respiration muscles.

- Nerve tissue is an excellent conductor of electrical current and may therefore be commonly affected in electrical injuries. Central nervous system damage may result in seizures or coma with or without focal neurological findings; peripheral nerve injury may lead to motor or sensory deficits, which may be permanent. If the current passes through the brain stem, respiratory arrest or depression, cerebral oedema, or haemorrhage may rapidly lead to death.
- Conjunctival and corneal bums and ruptured tympanic membranes are common.. Cataracts and hearing loss also may appear as late as 1 year after the event.
- An electrical injury can cause extensive necrosis of blood vessels. These injuries can cause immediate or delayed internal haemorrhage or arterial or venous thrombosis and embolism with subsequent complications.
- After an electrical burn, damage within the extremities is similar to crush injury with severe muscle necrosis, which releases myoglobin and hemoglobin. (Some patients may require amputation of the affected extremity due to decreased circulation and compartment syndrome).
- Severe muscle spasms can produce bone fractures and dislocations in the electrocuted patient. A patient may also fall after the electrical shock and sustain significant skeletal trauma, including damage to the cervical spine.
- Acute renal failure is a serious complication that affects about 10% of significant direct-contact electrical injuries.

Eye: Conjunctiva, cornea burn

Damage of the nervous system (seizure, coma, sensory impairments)

Bones fractures and dislocation of joints

Ruptured tympanic membrane

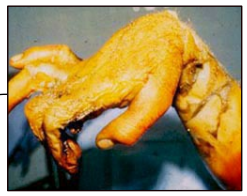
Heart: Tachycardia; fibrillation

chest wall dysfunction, hypoventilation

kidney complications

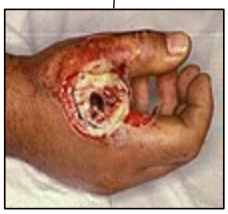
Necrosis of muscles

Internal organs damaged

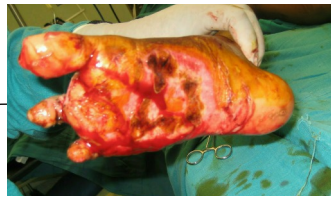


Damage to extremities with severe necrosis to muscle and tendon

Necrosis of blood vessels



The entrance site is often a characteristic "bull-eye" wound and may appear dry, leathery, charred, or depressed.



The exit wound may be ulcerated and have exploded appearance with missing areas.

Venomous and aggressive animals

Purpose

Diving companies intervene on various sites in estuaries, rivers, and sea, where underwater life exists for millions of years. Underwater animals are, for most of them, not dangerous for the divers. However, some of them use poison to catch their prey or defend themselves from predators, and thus, have the potential to cause injuries or kill. In addition, carnivores on the top of the food chain are often massive animals measuring several metres that are sufficiently strong to kill a diver or at least amputate a limb. Deliberate attacks from these massive animals against divers are uncommon, but that can happen in exceptional case scenarios. More often, accidents linked to marine life are from accidental contact with small poisonous animals.
This chapter discusses some marine life species potentially dangerous for divers that can be encountered at sea, estuaries, and rivers. Nevertheless, some may be missing.
The purpose of this chapter is not to make a masterly study of underwater life but to give some useful information that can be exploited to avoid or solve the problems arising from these animals.

List of animals described

1 - Coral

Description

Coral is the hard calcareous outer skeleton (exoskeleton) secreted by many types of marine polyps. Exoskeletons can be very sharp and colourful.

Symptoms

- Because coral formations are rigid and sharp, injury can occur after accidental contact, leaving a small amount of animal protein and calcareous material in the wound.
- The small, harmless-appearing cut may quickly develop into an infected wound. Some corals have cells that can inject toxins, which can produce a more significant injury.
- Occasionally, a cut or abrasion from the coral will expose the open skin to other pathogens floating in the water like vibrio bacteria, which are found more often in the marine environment than on land. Such an infection can rapidly cause an infection and, eventually, death. The symptoms include a wound that heals poorly or continues to drain pus or cloudy fluid, swelling around the cut, swollen lymph glands, fever, chills, and fatigue.

Treatment

- Scrub the cut vigorously with soap and water, and then flush the wound with large amounts of water.
- Flush the wound with a half-strength solution of hydrogen peroxide in water. Rinse again with water.
- Apply antiseptic ointment, and cover the wound with a dry, sterile, and non-adherent dressing.
- If the wound shows any sign of infection (extreme redness, pus, swollen lymph glands), the doctor of the company must be contacted for clear guidance. Normally an antibiotic treatment should be started, taking into consideration the possibility of a vibrio infection, and medical evacuation to a specialized hospital must be organized as soon as possible.

2 - Fire coral

Description

Fire corals are colonial marine organisms that look rather like real coral. Technically they are not corals, since they are more closely related to jellyfish and other stinging anemones. Fire corals have a bright yellow-green and brown skeletal covering and are widely distributed in tropical and subtropical waters. They appear in small brush-like growths on rocks and coral.

Fire corals' most often visible structures are the short, thin, hollow, potent stinging tentacles looking like fine transparent hairs, which functionally is to provide defense and capture food. These cells are used to inject toxins that are toxic to humans.

Symptoms

- Accidents with Fire coral contact is common, and intense pain can be felt that can last from two days to two weeks.
- The effects seen are possible nausea and vomiting for two to three hours afterward.
- The sting caused by these animals results from the injection of a water-soluble proteinaceous toxin, which is affected by heat. The discharged nematocysts (a sting found in the jellyfish family) cause small welts on the skin with red lesions around the raised areas.
- Swelling, blisters, and pus-filled incrustations may occur soon after being stung.
- Itching or welts remaining on the skin for up to several weeks after being stung.
- Other complications that may arise are likely related to cuts or scratches that may accompany the sting.

Treatment

- Seek medical advice.
- Breakdown of the protein by soaking the affected area in hot water. The venom must be quickly inactivated to avoid any hypersensitive reaction.
- The rash can generally be treated with antihistamines like cortisone or Benadryl.
- The welts can be swabbed with vinegar.
- Some anaesthetics may be applied to ease the burning sensation.
- The cuts and scratches shall be treated the same way as for wounds from coral.
- Medical evacuation to be prepared in case of need…

3 - Anemones

Description

Sea anemones are a group of water-dwelling, predatory animals of the order “Actiniaria”. Sea anemones are related to corals, jellyfish, and Hydra.

A sea anemone is a polyp attached to the surface beneath it by an adhesive foot, called a basal disc, with a column-shaped body ending in an oral disc. Most are from 1.8 to 3 centimetres in diameter, but anemones as small as 4 millimetres or as large as nearly 2 metres are known. They can have anywhere from a few tens to a few hundred tentacles.

A few species are pelagic and are not attached to the bottom; instead, they have a gas chamber within the pedal disc, allowing them to float upside down in the water.

The mouth, also the anus of the sea anemone, is in the middle of the oral disc surrounded by tentacles armed with many “Cnidocytes”, which are stinging cells that function as defense and a means to capture prey. Each stinging cell contains a small vesicle filled with toxins, an inner filament, and an external sensory hair. A touch to the hair mechanically triggers a cell explosion, which launches a harpoon-like structure that attaches to organisms and injects a dose of venom to the aggressor or the prey. The venom is a mix of toxins, including neurotoxins, that paralyses the prey so the anemone can move it to the mouth for digestion inside the gastro-vascular cavity. The sea anemone eats small fish and shrimp.

Most sea anemones do not present a serious risk to humans, but a few highly toxic species have caused severe injuries and are potentially lethal.

Symptoms

- The lesions initially appear as small linear eruptions that develop rapidly at times surrounded by a raised inflammation.

- Pain is immediate and may be severe; itching is common.
- The intensity can increase over 10 minutes or so, and the red, inflamed area may develop blisters or even necrotic ulcers in severe cases. The pain may spread centrally, with swollen lymph nodes, and can be associated with abdominal pain and chest pain.
- Systemic manifestations may include:
    - Weakness
    - Nausea
    - Headache
    - Muscle pain and spasms
    - Abundant tears and nasal discharge
    - Increased sweating
    - Changes in pulse rate
    - Chest pain

Treatment

- Prevent drowning.
- Seek medical support (doctor appointed by the company)
- Apply copious quantities of vinegar or mild denaturing agents (or carbonated beverages if vinegar is not available) to reduce the likelihood of discharge of the nematocysts.
- The tentacles (if any) should be removed as rapidly and gently as possible.
- Cardiopulmonary resuscitation may be needed and may need to be repeated on several occasions.
- Apply local anesthetic ointment and local steroid later.
- Analgesics which include morphine or similar may be administered intravenously (depending on the doctor)
- Intravenous steroids may be administered every 2 hours if needed.
- Local steroid preparations are valuable for treating local manifestations such as swelling, itching, and urticaria.
- Oxygen assistance, when needed.
- Tranquilizers or other sedatives may be of value after the immediate resuscitation because they calm the patient without causing significant respiratory depression.
- Monitoring (pulse rate, blood pressure, central venous or pulmonary pressure, respiratory rate).

4 - Sponges

Description

Sponges are multi-cellular organisms with bodies full of pores and channels, allowing water to circulate through them, consisting of jelly (mesohyl) sandwiched between two thin layers of cells. Sponges have unspecialized cells that can transform into other types, which often migrate between the main cell layers and the mesohyl in the process. Sponges do not have nervous, digestive, and circulatory systems. Instead, most rely on maintaining a constant water flow through their bodies to obtain food, oxygen and remove wastes.

These sedentary animals require some defense from mobile predators. They have a form of toxin which is not well understood. About a dozen sponges are toxic from the 5000 or so species, mainly in the temperate or tropic zones. Skin lesions have developed from sponges that have been deep-frozen or dried for many years.

Symptoms

- The symptoms relate to the skin areas which have been in contact with the sponge:
    - After a variable time, between 5 minutes and 2 hours, dermal irritation is felt. It may be precipitated by wetting or rubbing the area and may progress over the next day or so and feel as if the ground glass has been abraded into the skin.
    - An abnormal increase in sensitivity to stimuli of the sense and sensation of tingling, tickling, prickling, pricking, or burning may be noted.
    - The symptoms can persist for a week or more with inflammatory and painful reactions around the area. The degree of severity is not related to the clinical signs, and the symptoms may incapacitate some patients without any objective manifestations.
    - The dermal reaction may appear as a redness of the skin due to increased blood flows of the capillaries, with or without papule and vesicle development.

- There is sometimes skin peeling in the second or third week, but in other cases the skin lesions have recurred over many months.

Treatment

- The use of alcohol, lotions, or hot water will usually aggravate the condition.
- First, the skin should be gently dried, and then attempts should be made to remove any of the "spicules" from the skin using adhesive tape, rubber cement, or facial peel product (spicules are tiny spike-like structures of diverse origin and function found in many organisms. In sponges, spicules perform a structural function).
- Diluted acetic acid (vinegar) should be soaked over the affected area for 10 to 30 minutes (up to 4 times a day).
- Local application of cooling lotion may be of some value.

5 - Sea Urchin

Description

- Sea urchins are members of the phylum Echinodermata order, which includes sea stars, sea cucumbers, brittle stars, and crinoids.
- Like other echinoderms, they have fivefold symmetry and move through hundreds of tiny, transparent, adhesive "tube feet". The symmetry is not obvious in the living animal but is easily visible in the dried test.
- Their shell, or "test", is round and spiny, typically from 3 to 10 cm across. Common colours include black and dull shades of green, olive, brown, purple, blue, and red.
- They move slowly, feeding mostly on algae. sea otters, wolf eels, triggerfish, and other predators feed on them. The spines, long and sharp in some species, protect the urchin from predators

Symptoms

- Sea urchin thorns inflict a painful wound when they penetrate human skin but are not dangerous if fully removed promptly. If left, there may be further problems.
- It is not clear if the spines are venomous, but the “pedicellariae”, which are small claw-shaped structures between the spines, are considered venomous.

Treatment

- Remove the spines by hand or with some tweezers.
- In some cases, a surgical extraction may be the best option (seek medical advice for that).
- Remove the “pedicellaria” by applying some shaving cream or soap and gently scraping them off.
- The affected area is to be soaked with vinegar diluted in hot water.
- Protect the wounds from infection. Normally a treatment using antibiotics should be started.
- If necrosis, eczema-like skin rash, or secondary bacterial infection sets in, seek medical advice.

6 - Crown of thorns starfish

Description

- Starfish are marine invertebrates that typically have a central disc and five arms, though some species have more than this. The aboral or upper surface may be smooth, granular or spiny, and is covered with overlapping plates.
- Many species are brightly coloured in various red or orange shades, while others are blue, grey, or brown. Starfish have tube feet operated by a hydraulic system and a mouth at the centre of the oral or lower surface. They are opportunistic feeders and are mostly predators on benthic invertebrates. Several species having specialized feeding behaviours, including reversion of their stomachs and suspension feeding. The starfishes are normally not dangerous to humans.
- “Crown of Thorns Starfish” (Acanthaster planci) is the only venomous starfish. It can grow up to a metre in diameter and can have as many as 21 arms. It is covered with sharp spines all over its body except on its underside. The spines can grow up to 6 cm in length and easily penetrate a wetsuit *(See the photo on the next page)*.

- Crown of thorns starfish's general body surface is membranous and soft. When the starfish is removed from the water, the body collapses and flattens. The thorns bend over and flatten as well. They recover their shape when re-immersed if the creature is still alive.
- Adult crown-of-thorns starfish normally range in size from 25 to 35 cm. They are usually of subdued colours, pale brown to grey-green, but they may be garish with bright warning colours in some parts of their wide range.

Symptoms

- Upon contact, spines may break off and remain embedded.
- Thorns release various toxins, which, although not fatal, are painful and cause redness and local bleeding and swelling.
- The symptoms are usually limited, lasting from 30 minutes to 3 hours and then resolving.
- More severe reactions can include numbness, tingling, weakness, nausea, vomiting, joint aches, headaches, cough, and in rare cases, paralysis.

Treatment

- The affected area is to be soaked with vinegar diluted in hot water.
- Remove the spines by hand or with some tweezers.
- Seek medical advice if surgical extraction appears necessary.
- Protect the wounds from infection. Normally a treatment using antibiotics should be started.
- If necrosis, eczema-like skin rash, or secondary bacterial infection sets in, seek medical advice.
- Medical support and evacuation to be organized in case of complication.

7 - Shells (Cone shell)

Description

- Conus is a large genus of small to large predatory sea snails, marine gastropod molluscs, with the common names of “cone snails”, “cone shells”, or “cones”. These species have shells that are shaped more or less like geometric cones. Many species have colourful patterning on the shell surface. Cone snails are mostly tropical in distribution.
- Cone snails are venomous and capable of "stinging" humans. The species most dangerous to humans are the larger ones, that prey on small bottom-dwelling fish; the smaller species hunt and eat marine worms.
- Cone snails use a hypodermic-like modified radula tooth and a venom gland to attack and paralyse their prey before engulfing them. The tooth is sometimes likened to a dart or a harpoon. It is barbed and can be extended some distance out from the snail's mouth at the end of the proboscis *(Radula is a chitinous band in the mouth of most molluscs, set with numerous, minute, horny teeth and drawn backward and forward over the floor of the mouth in the process of breaking up food).*
- The harpoon can be fired numerous times if needed, and with such force, it can pierce wetsuits and clothing.

Symptoms

- The initial puncture effects may vary from painless to excruciating agony and can be aggravated by saltwater.
- The puncture region may become inflamed and swollen, sometimes white and ischaemic (insufficient blood supply), with a cyanotic area surrounding it, and it may be numb to the touch
- Numbness and tingling may ascend from the bite to involve the whole body, especially the mouth and lips; this may take about 10 minutes to develop.
- Skeletal, muscular paralysis may spread from the site of injury and result in anything from mild weariness to complete flaccid paralysis.
- Difficulty with swallowing and speech may occur before total paralysis.
- Visual disturbances may include double and blurred vision (paralysis of voluntary muscles and pupillary reactions).
- Respiratory paralysis may dominate the clinical picture, resulting in shallow rapid breathing and a cyanotic appearance, proceeding to apnea, unconsciousness, and death.
- Other cases are said to result in cardiac failure, although this is probably secondary to respiratory paralysis.
- The extent of neuro-toxic damage is variable; if the patient survives, he/she is active and mobile within 24 hours. However, neurological sequels and the local reaction may last many weeks.

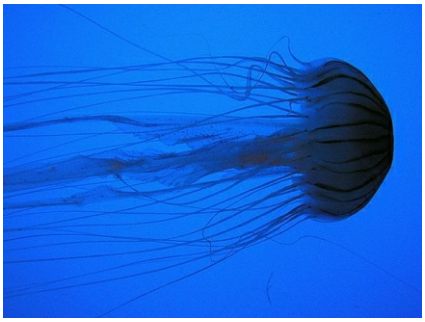
Treatment

- Medical assistance is necessary.
- Without paralysis:
  - The limb must be immobilized.
  - A pressure bandage should be applied to slow down the progression of the venom.
- With paralysis:
  - Breathing assistance (Pure O2 + inflator) may be needed.
  - CPR may be needed if the patient has neither pulse nor respiration. (If available and not in the chamber, defibrillation is to be used).
  - Reassure.
  - Anaesthetic can be injected into the wound.
  - Endotracheal intubation prevents aspiration of vomit.
  - Monitoring of the unconscious patient.
  - Medical evacuation to be organized as soon as possible.
- Note: Respiratory depressants, respiratory stimulants, and drugs used against neuromuscular blockade are not to be used.

8 - Jellyfish

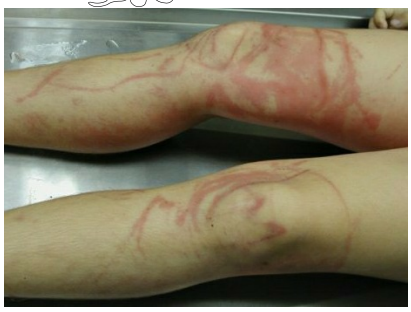
Description

- Jellyfishes are very simple animals with no bones or cartilage, no blood, no heart, no brain, and are made of 95% water. They move in the water by opening and closing their bodies, or bell, by muscular contractions. Most species have tentacles attached to the bell's edges, armed with several billion stinging cells that can inoculate venomous substances. The jellyfish uses these tentacles to catch their prey. Depending on the species, these preys vary from plankton to fish. The sting of most of the species does not affect humans. For some others, it causes mild skin irritation to excruciating pain.
- Only a few species are considered extremely dangerous and able to be lethal. The majority of these jellyfishes are from the “Box Jellyfish” family.
- “Chironex Fleckeri” is considered by some specialists as the most deadly animal of the sea. It is found in shallow water at the edge of beaches from Northern Australia to southeast Asia, notably Thailand and Malaysia. This box jellyfish can see through four eyes, but the way it proceeds without a brain is still not understood. When an adult, the weight of the Chironex Flekeri is around 2 kg, and its 10 to 15 tentacles can extend up to 5 m. These tentacles are armed with 5 billion stinging cells triggered when in contact with fish or potential prey. The venom of this box jellyfish has cardiotoxic, neurotoxic, and highly dermatonecrotic components. Only 3 m of one tentacle can kill a human adult.



Symptoms

- Excruciating pain occurs immediately on contact and increases in intensity, often coming in waves.
- Multiple interlacing whiplash lines red, purple, or brown, 0.5 cm wide develop within seconds.
- The patient may become confused, act irrationally
- The patient may develop cardiac shock, with a disturbance of consciousness.
- Hypotension, tachycardia, and a raised venous pressure may also occur
- Impairment of the conscious state may proceed to coma and death
- It is also possible that the clinical state will oscillate within minutes from episodes of hypertension, tachycardia, rapid respirations, and normal venous pressure to those of hypotension, bradycardia, apnea, and elevated venous pressure. The oscillation may give a false impression of improvement just prior to the patient’s death.
- Respiratory distress, pulmonary congestion, oedema, and cyanosis may be due to the cardiac effects or a direct midbrain depression.
- If death occurs, it usually does so within the first 10 minutes; survival is likely after the first hour.
- Amnesia occurs for most of the incident following the sting.
- Paralysis and abdominal pains may occur.
- Pain diminishes in 4 to 12 hours.



- Malaise and restlessness may persist, with physical convalescence lasting up to a week.
- The red, swollen skin may develop large wheals, and, after 7 to 10 days, necrosis and ulceration develop over the area of contact. Itching may also be troublesome and recurrent. The skin lesions may take many months to heal if deep ulceration occurs.
- Pigmentation and scarring at the site of these lesions may be permanent.
- Irritability and difficulty with psychological adjustment may take weeks or months to disappear.

Treatment

- Prevent drowning.
- Contact the doctor for medical support.
- Box jellyfish anti-venom has been developed by the Australian Commonwealth Serum Laboratories. If this anti-venom is on board proceed to treatment (*address manufacturer: CSL Limited 45 Poplar Road Parkville Victoria 3052 Australia).*
- Apply copious quantities of vinegar or mild denaturing agents (wine or carbonated beverages if vinegar is not available) to reduce the likelihood of discharge of the nematocysts.
- Remove the tentacles as rapidly and gently as possible.
- Cardiopulmonary resuscitation may be needed and may need to be repeated on a number of occasions.
- Breathing assistance (pure O2 + inflator).
- Local applications also may include a local anaesthetic ointment and local steroid.
- Analgesics administered intravenously (confirm the doses with the medic)
- Intravenous steroids may be administered every 2 hours if needed (confirm with the doctor).
- Local steroid preparations are valuable for treating local manifestations such as swelling, pain, itching, and urticaria. (Confirm with the doctor).
- Monitor the patient.
- Organise medical evacuation.

9 - Sea snakes

Description

- Sea snakes are widely distributed throughout the shallow waters of the Indian and Pacific oceans.
  They can be identified by their flattened tail, which allows them to propel effectively in the water, and are very well adapted to the marine environment. However, like all snakes, they have lungs and must return to the surface to breathe. Nevertheless, most species can also satisfy about 25% of their O2 by breathing through their skin and can remain submerged for several hours, depending on their degree of activity and the water temperature. Their size, depending on the species, vary from 50 cm to around 3 m long. Sea snakes do not like extremely hot or cold temperatures; that is why they are attracted by the heat of pipelines in service at some depths.
  By night they also seem attracted by the working boats' lights, and it is not unusual to have the surface of the sea covered by hundreds of them during the night time operations in some places. They are reported “inquisitive”, and sometimes aggressive to some divers and the fact is that some accidents have already happened, though some consider them placid, if not disturbed.
- Sea snake venom is 2 to 10 times more toxic than that of the cobra. It is a heat-stable protein that appears to block the neuromuscular transmission. Most species have a small mouth, and the apparatus to deliver the venom is not sufficiently developed to pierce a diving suit. Unfortunately, some species, such as the “olive-brown” and the “Stoke’s” sea snakes, have well-developed fangs that could easily pierce some wet suits.

Symptoms

Sea snake bites are usually felt, but not all the time. The number of fang and teeth marks varies from 1 to 20, but usually, there are 4, and the brittle teeth may remain in the wound. There is little or no local pain or swelling. The Symptoms may be visible after a latent period, which may vary from 10 minutes to several hours, and then progress as indicated below.

- Euphoria, anxiety, or restlessness.
- Thirst, dry throat, nausea, and vomiting occasionally develop.
- Generalized stiffness and aching.

- Muscle weakness.
- Paralysis may happen, usually with the legs involved prior to the trunk, then the arms and neck, or paralysis which extends centrally from the bite area.
- Muscular twitching, writhing, and spasms may occur.
- The patient may experience difficulty speaking and swallowing as the paralysis extends.
- Facial and ocular paralysis may develop.
- Respiratory distress due to involvement of the diaphragm may result in shortness of breath, cyanosis, and finally, death.
- Cardiac failure, convulsions, and coma may develop terminally.
- Necrosis of skeletal muscles and tubular damage in the kidneys may develop in several hours.
- When recovery occurs, it is usually rapid and complete.

Treatment:

- Recover the diver.
- Identify the snake if possible.
- Seek medical advice and support.
- The amount of venom injected cannot be predicted, and therefore, any suspected bite by a sea snake should be considered potentially life-threatening.
- Use the pressure immobilization: The limb should be wrapped with a broad pressure bandage, starting at the wound site and extending as high up the extremity as possible. The bandage should be wrapped to venous occlusive pressure (approximately 0.9 bar) like wrapping a sprained ankle. However, if more than 30 minutes have passed after the bite, the pressure immobilization technique will not be helpful.
- The affected extremity should also be immobilized to prevent it from bending at the joints.
- The victim is to be kept calm, warm, and as comfortable as possible.
- Note that there is no benefit to suctioning or cutting the bite area to "suck the venom out".
- Breathing support (High PPO_2 therapeutic mix)
- Endotracheal intubation May be needed.
- Anti-venom treatment should be started *(address manufacturer: CSL Limited 45 Poplar Road Parkville Victoria 3052 Australia)*.
- Medical evacuation should be organized.

10 - Small venomous fishes

10.1 - Scorpion fish family

Description

“Scorpaenidae” (Scorpion-fish) family is composed of hundreds of species with some of the most venomous fish living in tropical and temperate seas. They are widespread mostly in the Indo-Pacific, and some have been found at more than 2000 m depth. “Scorpaenidae” generally have 12-13 dorsal thorns, 2 pelvic thorns, and 3 anal ones. Each thorn is associated with a pair of venom glands. A loose integumentary sheath covers each thorn. The sheath is pushed down the thorn during the envenomation, causing compression of the venom glands located at the thorns' base. The venom then travels from the glands through anterolateral depressions in the thorns and into the wound. The venom toxicity is due to antigenic, heat-labile proteins of high molecular weight. Scorpaenidae are mostly using their dorsal thorns as a means of defense against predators.

Two dangerous representatives of the family are discussed in this point:

10.1.1 - “Stonefish” (Synanceia)

- These fish grow to about 30 cm in length. They lie dormant in shallow waters, buried in mud, coral, or rocks, and are practically indistinguishable from the surroundings because they look like a stone covered with sponges. They catch small passing fish by sucking them into their gaping mouth. The 13 dorsal thorns, capable of piercing wet suit boots, sneakers, and skin, become erect when the fish is disturbed. Each thorn has 5 to 10 mg of venom. Occasionally, a stonefish prick is associated with no venom. It is thought that it is because the venom is regenerated very slowly. This fish can live for hours out of the water.
- Stonefish’s poison is an unstable protein that produces intense vasoconstriction and therefore tends to localize itself. The toxin acts on skeletal, involuntary, and cardiac muscles, blocking conduction in these tissues. It provokes muscular paralysis, respiratory depression, peripheral vasodilation, shock, and cardiac arrest. The toxin can also produce cardiac arrhythmias.

10.1.2 - "Lionfish"

- These fish are characterized by their attractive coloration with red, white, creamy, and black bands. They have developed pectoral and dorsal spiky fin rays. They are hunters known to use their specialized bilateral bladders to control and adjust their depth very quickly and fall to their prey by this means. Similar to "Stonefish", "Lionfish" feed on small prey. The venom from "Lionfish" is similar to the one of "Stonefish" and the majority of the "Scorpaenidae". The mechanisms of defense against predators are similar to those of "Stonefish".

Symptoms

- Immediate excruciating pain that increases in severity over the next 10 min and sometimes comes in waves. It may be sufficient in some cases to cause unconsciousness and thus drowning.
- Ischemia of the area is followed by cyanosis.
- The area becomes swollen and edematous, often hot, with numbness in the centre and extreme tenderness around the periphery.
- Paralysis of the adjacent muscles is said to immobilize the limb and be painful.
- The pain is likely to spread proximally to the regional lymph glands in the axilla or groin. Both the pain and the other signs of inflammation may last for many days.
- Signs of cardiac failures like bradycardia or cardiac arrhythmias may be visible.
- Pallor, sweating, hypotension are also visible, and syncope may happen
- Cardiac arrest is also possible.
- Respiratory failure may be due to pulmonary oedema, depression of the respiratory centre, and the respiratory musculature's paralysis. All these can be associated with cardiac arrest.
- Delayed healing, necrosis, and ulceration may persist for many months.

Treatment

- Recover the diver.
- Seek medical advice and support.
- Removal of the visible thorns.
- Some specialists recommend heat treatment (40 - 54°C) by soaking the affected body part for about 30 min.
- Injection of stonefish anti-venom ((address manufacturer: CSL Limited 45 Poplar Road Parkville Victoria 3052 Australia)
- Appropriate resuscitation techniques using external cardiac massage and defibrillation if necessary.
- Respiratory assistance (O2 apparatus).

Some specialists also recommend-Endo-tracheal intubation.

- Monitoring (pulse, respiration, blood pressure).
- Clinical complications of bulbar paralysis should be treated as they arise. (contact the doctor).
- Medical evacuation to be organized.

Note: A prick is said to confer some degree of immunity for future episodes.

10.2 - Other small fish

Description

Many fish have thorns and venom apparatus, usually for protection and occasionally for incapacitating prey. Other fish have thorns that do not produce envenomation. Injuries associated with fish fin spines can cause high morbidity because such injuries are often underestimated. In many cases, the spines may contribute to leaving residual fragments of foreign matter in the soft tissues, leading to infections as much as possible venom. The clinical symptoms depend on pathogen organisms and vary from local infection to systemic or fatal multi-system disease. Because identifying the fish species responsible for the wound is not always possible, and some pathogen organisms like Vibrio bacteria are present in the marine environment, any wound must be considered potentially dangerous.

Symptoms

- Usually, immediate local pain increases in intensity over the following few minutes. It may become excruciating, but the pain from an average sting usually lessens after a few hours.
- Anaesthetized puncture wound with the hypersensitive surrounding area is common.
- Pain and tenderness in the regional lymph glands may extend even more centrally.
- Locally, the appearance is of one or more puncture wounds, with an inflamed and sometimes cyanotic zone.

- Surrounding the cyanotic zone is an area that is pale and swollen, with pitting oedema.
- The patient may be distressed by the degree of pain that can develop into a delirious state.
- Malaise, nausea, vomiting, and sweating may be associated with temperature elevation and leukocytosis *(white blood cell count above the normal range in the blood)*.
- Cardiovascular shock state may supervene and lead to death.
- Respiratory distress may develop in severe cases.
- Due to marine infections, a foreign body reaction, or venom effects, chronic localized inflammation, oedema, necrosis, and severe disability may continue for many months.

Treatment

- Lay the casualty down and reassure him
- The affected area should be rested in an elevated position
- Rinse the wound with soap and water.
- Soak the wound with tolerably hot (40-45°C) water to relieve pain for 30 to 90 minutes, or longer if the pain continues.
- Remove any visible spines
- Keep the wound clean and apply a local antibiotic
- Tetanus prophylaxis may be indicated if there is necrotic tissue or if the wound has been contaminated
- Injection of local anaesthetic without adrenaline through the puncture wound is considered beneficial
- Treatment may be needed for generalized symptoms of cardiogenic shock *(decreased cardiac output and evidence of tissue hypoxia in the presence of adequate intravascular volume)* or respiratory depression.
- Medical advice
- Medical evacuation is organized in obvious infection cases, cardiogenic shock, respiratory depression, or other complications.

11 - Octopus

Description

- There are around 300 species of known octopuses measuring from a few centimetres to 9 m arm span for 75 kg. Octopuses have no internal or external skeleton which allows them to squeeze through tight places. They are among the most intelligent and behaviorally flexible of all invertebrates. Octopuses can build their shelter and barricade their refuge entrance using stones and debris. They elaborate complex strategies to catch their prey. They have two eyes, 3 hearts, and eight arms, which can be renewed if cut off by a predator. Like other cephalopods, they are bilaterally symmetric. The octopuses have a hard beak at the center point of the arms. They have numerous strategies for defending themselves against predators, including the expulsion of ink, the use of camouflage, and the ability to jet quickly through the water.
- The octopuses use toxins and a specific protein named “cephalotoxin” to kill and pre-digest their prey. All octopuses are venomous, but only one group, the blue-ringed octopus, is known to be deadly to humans. These animals are shy and not aggressive to humans, but some accidental bites may happen if provoked.

11.1 - Blue-ringed octopus

- Blue-ringed octopuses are found on coral reefs (but also on onshore and offshore facilities) of the Pacific and Indian Oceans, from Japan to Australia. They are recognized as some of the world's most venomous marine animals. Despite their small size and relatively docile nature, they can prove a danger to humans. They can be recognized by their characteristic blue and black rings and yellowish skin. When the octopus is agitated, the brown patches darken dramatically, and iridescent blue rings or clumps of rings appear and pulsate within the maculae. Typically 50-60 blue rings cover the dorsal and lateral surfaces of the mantle. They hunt small crabs, hermit crabs, and shrimp. They may bite attackers, including humans.
- There are two blue-ringed octopus species: the “Hapalochlaena lunulata”, which is the largest and grows up to 20 cm across its stretched tentacles. The other, the “Hapalochlaena maculosa”, more common, is a golf ball's size.

Symptoms

- Most bites cause minimal pain for the first 5-10 minutes, then begin to throb and may get numb and involve the rest of

the extremity. There may be some bleeding.
- Nausea, vomiting, and difficulty swallowing.
- Double vision, blurred vision, fixed dilated pupil.
- Numbness or loss of feeling around the lips and mouth.
- After approximately 10 minutes, the victim may have a general weakness, difficulties breathing, and become paralyzed.
- Respiratory failure may occur, which may lead to unconsciousness, cardiac arrest, and death.
- The paralysis duration is between 4 to 12 hours, but the weakness and in-coordination may persist for another day.

Treatment

- Immediately recover the diver *(send the rescue diver)*.
- Seek medical advice and support *(doctor appointed by the company in priority or another if not reachable)*.
- Medical evacuation to be organized urgently.
- Use the pressure immobilization: The limb is to be wrapped with a broad pressure bandage, starting at the wound site and extending as high up the extremity as possible. The bandage should be wrapped to venous occlusive pressure (approximately 0.9 bar) like wrapping a sprained ankle.
- The patient to be rested in a "recovery position".
- Breathing assistance *(Therapeutic mix)* with periods on air, particularly when the paralysis is starting.
- Endotracheal intubation for the aspiration of vomit and clean the tracheobronchial.
- Close observation and management of the casualty.
- Some doctors recommend the use of "Neostigmine" *(Prostigmin Bromide)* and "edrophonium" *(Enlon, Tensilon)* during the period of paralysis (to be confirmed by the doctor in charge).

12 - Moray eels

Description

- "Moray eels" are from the family of "Muriaenidae", which includes about 200 species that settle in tropical, subtropical, and temperate waters. A few species can live in freshwater. They look like snakes with the dorsal fin extending behind the head along the back and joining seamlessly with the caudal and anal fins. The body is generally patterned, and various colours like brown, yellow, black, and white and others are observed. Their eyes are small, and they have a very developed sense of smell. Morays are ambush hunters and feed on small fish, cephalopods, sea snakes, molluscs, and crustaceans. In addition to their normal jaws with rear hooked teeth, these animals have a 2nd set of jaws called "pharyngeal jaws" situated at the back of the throat to grasp their prey and transport them into the digestive system. Morays secrete mucus, which contains toxins on their skin. Some varieties inoculate venom when they bite.
- Moray eels are generally shy and hide from divers in their refuges, but they become aggressive if they feel hunted. Accidents may happen if a diver waves his hand in the proximity of the eel, provokes it, or if the animal feels trapped, which can happen in wrecks or some underwater installations like pipelines manifolds and others. These animals can amputate fingers or make deep wounds that may become infected.

Symptoms

- It is indicated that in some cases, the moray cannot separate from the victim due to its hooked teeth.
- The wounds are usually ragged with massive bleeding.
- Secondary infection in the area is common.
- Sweaty (cold and clammy) appearance, rapid pulse, hypotension, may be noticeable.
- Syncope may happen.

Treatment

- Recover the diver, and reassure him.
- If the eel has not released its hooked teeth, kill the animal.
- Contact the doctor.
- The wound to be cleaned with soap and water (alcohol is not recommended)
- The wound is to be treated for bleeding.
- Antibiotic treatment should be started.
- Organize the medical evacuation

13 - Stingrays

Description

- Stingrays belong to the superorder “Batoidea”, which includes all the rays, guitarfish, sawfish, and other species... Like sharks, they have skeletons made of cartilage. Most “batoids” have a flat body, except for the guitar-fishes and sawfishes. Many species have developed pectoral fins similar to wings. Their eyes and spiracles are located on top of the head. The gill slits are located on their abdominal (ventral) surface, which means that when the fish is resting on the seafloor, the gill slits are obstructed. Therefore, they have slits located behind their eyes that enable them to breathe in these situations. Their mouth is located ventrally, and they can considerably protrude their upper jaw far enough away to capture their prey, which are mainly small fish, crustaceans, and shells. These animals can be seen in sandy areas, coral reefs, and open water.
- Some “batoids” are equipped with venomous spines on their tail, which serve to deliver a painful sting if they are provoked. The biggest member of the family is the manta ray, which can weigh 2 tons. The manta ray feeds on plankton, and its tail does not have a spine.

- Stingrays can be found in all oceans, and, as the majority of the batoids, they feed on mollusks and crustaceans. Most of them have venomous spines at the base of their tail used for protection against their predators. These spikes, which often remain inside the victim's body, can cause acute pain and infections and may cause profuse bleeding with death due to the blood loss, depending on where the wound is situated. Their greatest predators are the sharks (not considering the human activities). Stingrays do not attack humans, but they can react aggressively if they feel threatened.

Symptoms

- Pain is usually immediate and increasing over 1 to 2 hours and easing after 6 to 10 hours.
- The area is swollen and pale, with a bluish rim several centimetres in width, and spreads around the wound after one or two hours.
- The pain may be constant, pulsating, or stabbing.
- Possibility of massive bleeding (death can follow if it is not stopped) and a mucoid (like mucus) secretion.
- Integument from the spine may be visible in the wound.
- Anorexia, nausea, vomiting, diarrhea, salivation with frequent urination.
- Muscular cramps, tremors, tonic paralysis with fever, and sweating.
- Difficulty in breathing with cough and pain when inhaling.
- Fainting, palpitation, hypotension, cardiac irregularities (conduction abnormalities, blocks), and ischemia are possible.
- Confusion, or delirium.
- Recurrence of symptoms with aggravation of the pain and secondary infection with necrosis and ulceration within days or weeks implies retention of a foreign body (integument or spine).
- The symptoms may persist for weeks or months after the injury, even though the wound may have closed over.
- Fatalities are possible, especially if the stingray’s spine perforates the pericardial, peritoneal, or pleural cavities.

Treatment

- Recover the diver immediately. (send the rescue diver)
- Stop the bleeding, if any. (compression)
- Seek medical advice and support
- Clean the wound and remove foreign objects if possible. But if the spine is found deeply embedded (impaled) in the body, no attempt to remove it should be made because the internal portion may be occluding a blood vessel that will haemorrhage without this "plug.
- Soak the wound with tolerably hot (40-45°C) water to relieve pain for 30 to 90 minutes, or longer if the pain continues.
- Relieve the pain via infiltration of local anaesthetics without adrenaline (epinephrine) into and around the wound.
- Monitor temperature, pulse, respiration, blood pressure.
- Broad-spectrum antibiotics to be used at an early stage.
- Organize medical evacuation.

14 - Big biting fish

Description

Big biting fish have sharp teeth associated with a huge force. Most of them can make unpredictable and brutal attacks with big trauma or death as a result. The fish concerned are mainly barracudas and sharks. About 40 species of teethed sharks can bite, but the attacks reported involve only a few of them, which does not mean that the other sharks will not attack. The big fish listed below are among the most aggressive seen.

- Barracuda

Barracuda are fast swimmers (speed around 45 km/hrs). They are known to travel in schools, but large ones are often alone. Their length can be around 2 m. They are known as territorial fish eating small fish, living around reefs (thus platforms), and sometimes muddy places. Some attacks on swimmers and divers have been reported.

- Reef black-tip shark

Reef black-tip sharks are found around reefs and are sometimes seen near platforms and other facilities. They are territorial fish. Their maximum length is about 1.5 m. they eat cephalopods, crustaceans, and small fish. Some attacks are due to stimulation by food.

- Common black-tip shark

Found offshore and onshore. The size is about 2.5 m. It eats cephalopods, crustaceans, and small fish. Indicated potentially dangerous if stimulated by food.

- Silver tip shark

Found onshore and offshore. The size is about 2.75 m. Eats cephalopods, crustaceans, and small fish. Potentially dangerous when provoked.

- Grey reef shark

Found near coral reefs and lagoon passage. It can grow up to 2.55 m. Eats fish and cephalopods. Can become aggressive and considered potentially dangerous.

- Blue shark

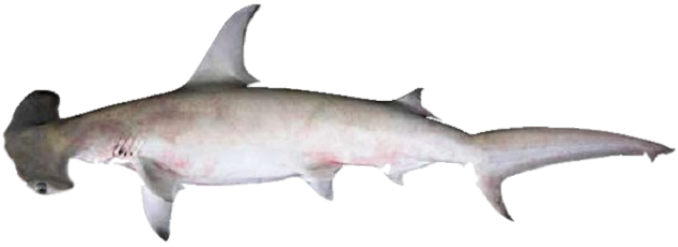
Pelagic sharks encountered from the surface to 600 m. They live in all oceans. Their length can reach 3.8 m. They eat small pelagic fish and cephalopods. Considered potentially dangerous but not particularly aggressive.

- Scalloped Hammerhead shark

Often seen near the shore and adjacent deep waters, from the surface to 300 m. Can reach 3.5 m long. Eats fish and cephalopods. It has a characteristic flat extended head with the eyes at the extremities of the extensions. Classified potentially dangerous

- Great Hammerhead shark

Seen near the shore and adjacent deep waters, from the surface to 100 m. Can reach 4.5 m long. Eats cephalopods, crustaceans, and fish. Its head is similar to the scalloped hammerhead. Classified potentially dangerous.



- Indian sand tiger shark

It has very prominent teeth. It is indicated from surface to 1200 m depth, mostly near the coastlines in the western Pacific, Indian ocean, and the Mediterranean sea. The length can reach around 3 m. Feeds on small, middle size fish and cephalopods. Potentially dangerous, particularly at night.

- Whitetip shark

They are seen in deep and open waters, with temperatures greater than 18 °C from the surface to 150 m. They feed on pelagic fish and cephalopods. Their size can reach 4 m. They are aggressive sharks with numerous deadly attacks to wreck survivors. They have a reputation for attacking in groups.

- Shortfin Mako shark

Visible everywhere, even in cold seas from the surface to 700 m, and sometimes seen very close to the shore. It is the fastest shark and one of the most aggressive. It feeds on small and big fish, dolphins, and other big prey. It can reach about 4 m. It is known to attack and considered dangerous. The Shortfin Mako is from the same family as the great white shark.

- Tiger shark

It lives close to the shore and the outer continental shelf, from the surface to around 150 m.
This shark has a reputation for eating everything and does not hesitate to attack big prey. Considered as one of the most dangerous sharks, its length is about 6 m. It has a characteristic square snout and some stripes on its back.

- Bull shark

This shark can be found far at sea, near the coast, in estuaries, in rivers, as it can adapt to freshwater and from the surface to around 150 m. It is comfortable in clear water but also in muddy waters without any visibility. Its size can reach 4 m long. It attacks everything, including dolphins and crocodiles. It is responsible for a lot of deadly attacks, and it is considered extremely aggressive. Some of its attacks are in less than 1 m water depth.

- Great white shark

The great white shark (Carcharodon carcharias) can be found in the coastal waters and the middle of all the major oceans. Mature individuals can reach 6.5 m in length. However, reports have been published of great white sharks measuring over 8 m and 3,324 kg in weight.

This shark reaches its maturity around 15 years of age, and its lifespan is estimated to approximately 70 years.

The great white shark can accelerate to over 60 km/h and has no natural predators other than the killer whale. It is the primary predator of marine mammals and is also known to eat various other marine animals, including seabirds. It is responsible for many deadly attacks.

Attacks

- Barracuda attack

Barracudas are territorial fish that are inquisitive about any intruders. It is common to have these animals observing divers. They generally remain a few meters away and do not dare to move closer. Nevertheless, they may bite reflexively any fast-moving bright object. Luminous or fluorescent colours and abnormal movements seem to attract them and may trigger an attack. Experts consider that the risk of attack may be increased at night due to portable lights that may cause these fish to panic, resulting in erratic behaviours and reflex biting possible injury to any diver in their path. These attacks are generally fast with direct biting.

- Shark attack

Sharks have good vision and are especially sensitive to motion and contrast. Their sense of smell and taste is very developed, with two-thirds of their brains involved in processing this information. Sharks also have specialized organs called ampullae of Lorenzini that detect tiny electrical currents, such as those put out by active muscle contractions and the motion of injured fish. They can detect diluted bleeding and abnormal splashing at the surface. They also detect bright and contrasting colours, and shiny objects attract them the same as the barracuda.
The reasons for shark attacks are still not fully understood and are probably linked to each species' characteristics. It is considered that it is a territorial and/or a feeding response. It has been reported in some scenarios that the sharks were circulating in a frenzy and savagely biting at anything, including each other.
One of the most deadly attacks by sharks was reported by the US Navy following the sinking of the cruiser "Indianapolis", which was torpedoed by a Japanese submarine when sailing from Guam to Leyte on the 30th of July 1945. Of the 900 survivors from the wreck, only 317 had been rescued five days later. Nearly 600 men lost their lives to what is believed to be white tip sharks. The survivors said that the sharks were always by the hundreds, swimming just below their dangling feet, picking off the men one by one. Similar situations have been reported with other wrecks in tropical waters.

Experts indicate 3 types of attacks:

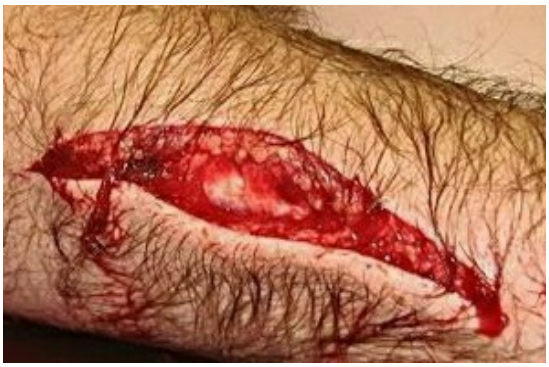
- The shark takes a single bite and does not return. The experts think that this attack is maybe because the shark mistakes a human for its normal prey.
- The shark bumps the victim prior to returning for more bites.
- The shark bites without warning and then follow up with further attacks.

The last two types of attacks are the source of the most severe shark bite injuries.

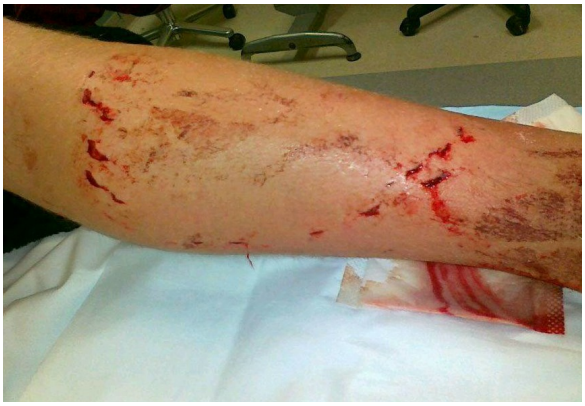
Symptoms

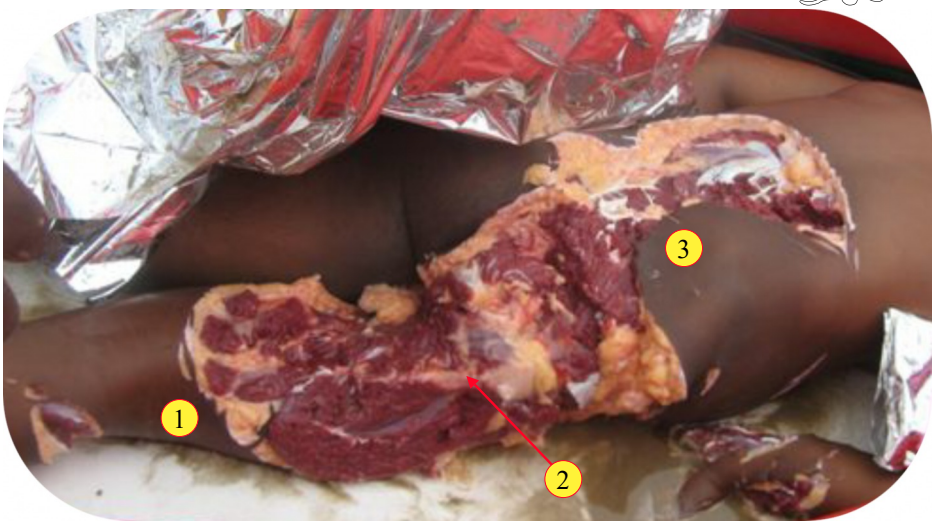
Injuries range from a single bite with not too deep cuts to tissue loss, limbs ripped off, and death due to massive bleeding and traumas.

- Barracuda bites are associated with a shearing type injury



- Classic shark bites are crescent shaped. Another common wound pattern is a series of parallel cuts caused by the shark raking its teeth on the person.





Wounds resulting from a big shark attack:

1 - There is a clear cut at the bottom of the thigh with the disappearance of all the rear muscles.

2 - The femur seems apparent.

3 - The missing gluteal and back muscles seem to have been torn off by the same bite.

- There may be the following symptoms associated with wounds.
  - Emotional shock: Caused by the fright, stupor, restlessness.
  - Bruising and rubbings (shark skin is like sandpaper)
  - Massive haemorrhages
  - Foreign objects in the wounds (sharks teeth)
  - Fractures may be present
  - Internal organs may be injured (due to crushing) or coming out due to large openings
  - Internal haemorrhages
  - Hypovolemic shock due to the massive loss of blood (several phases of tachycardia followed by a sudden fall in pulse rate)
  - Cardiogenic shock due to inadequate cardiac function (sudden acute breathlessness, fatigue with some relief in sitting position)
  - Vasodilatation shock due to bacteria infection (probably not visible on-site if transferred to the hospital quickly)
  - Septic shock due to the bacterial infection (probably not visible on-site if transferred to the hospital quickly)
  - Death may occur quickly depending on the haemorrhages and traumas.

Treatment

The treatment depends on the injury and whether it is life-threatening or not.

- In all cases:

- Immediately recover the diver.
- Seek medical advice and support.
- Organize the medical evacuation.

- Small wounds:

- Stop the bleeding, if any.
- Clean the wound and remove foreign objects if possible.
- Examine and report to the doctor in charge.
- Look for other injuries depending on the type of wound (broken limb, internal bleeding…)
- Relieve the pain via administration of local anaesthetics without adrenaline (epinephrine) into and around the wound.
- Monitor temperature, pulse, respiration, blood pressure.
- Broad-spectrum antibiotics to be used at an early stage.
- Treat for an emotional shock: Patient to be laid flat with raised legs + support.

- Big wounds with massive bleeding

- The bleeding to be stopped by direct pressure (application of cold packs or ice packs over the compress, not under it may hasten the process by initiating spasm and closure of disrupted blood vessels).
- Clean the wound and remove the foreign objects if possible.
- Treat for a hypovolemic shock: lay the patient with the legs raised + intravenous infusion normal saline + colloid (to maintain the osmotic pressure exerted by proteins in a blood vessel's plasma).

- Treat the cardiogenic shock: Breathing support using O2, Patient sat upright.
- In case of heart failure: resuscitation (CPR or defibrillation if available + breathing support O2).
- A tourniquet is to be used only in a life-threatening situation and is best applied by an experienced person. Only in the case of torrential bleeding is a tourniquet more advantageous than continuous pressure. The decision to apply a tourniquet is one in which a limb is sacrificed to save a life.
- If the victim has suffered a large wound through which internal organs (such as loops of bowel) or bones are protruding, no attempt should be made to push these back inside the body or under the skin unless they slide back in without assistance. The extruded internal organs or bones must be covered with continually moistened bandages (pads of gauze or cloth) and held in place without excess pressure. Seek immediate medical attention.
- In case of severe cuts on the victim's neck, the diver medic must carefully take care not to disturb the wound because such disturbance might remove a blood clot that controls the bleeding from a large blood vessel. A firm pressure dressing is to be applied, and close observation is to be done with continual airway assessment because an expanding blood clot within the neck can compress the throat and windpipe. Evacuation is urgent.
- If some teeth are found deeply embedded in the body, there must not be any attempt to remove them because they may occlude some blood vessels that will haemorrhage without these.
- When the external bleeding is stopped, the injury is to be immobilized. A subsequent examination and a report to the doctor in charge must be undertaken (photos are welcome in complement).
- Look for other injuries depending on the type of the wound. (Broken limb, internal bleeding…)
- Relieve pain via infiltration of local anaesthetics without adrenaline (epinephrine) into and around the wound.
- Monitor temperature, pulse, respiration, blood pressure.
- If internal bleeding from a broken bone or injured organ is suspected, the only treatment is surgery. So, such intervention cannot be done on-site by a nurse or a diver medic.

15 - Whales

Whales and sea mammals often live in structured groups. They are generally not aggressive to humans if they are not attacked. Nevertheless, two species may have aggressive behaviour, particularly if the group considers that young individuals may be threatened.

- Orcas

"Orcas" are toothed whales, also called "killer whales", that are part of the oceanic dolphin family, of which they are the largest members. They are found in all oceans, from the Arctic and Antarctic regions to tropical seas.
Adult males are larger overall than their female counterparts. They are also provided with larger pectoral flippers and dorsal fins than the females, which allow to differentiate them easily.

The largest recorded male was 9.8 m in length and weighed 10,000 kg.

Orcas populations are composed of family groups. They have sophisticated hunting techniques and vocal behaviours, which are often specific to a particular group and passed across generations. They are powerful predators capable of killing prey much larger than humans, such as leopard seals, great white sharks, and whales.
Wild Orcas are not considered a real threat to humans. Nevertheless, there are few documented cases of wild Orcas attacking people or small boats. Also, there have been several non-fatal and fatal attacks on humans by orcas in captivity. Experts are divided as to whether the injuries and deaths were accidental or deliberate attempts to cause harm.

- Sperm whales

Sperm whales (Physeter macrocephalus), also called cachalot, are the largest toothed whales and the largest toothed predators. They are called “sperm whale” because an oil named “spermaceti” is found in their head. During the 18th and 19th centuries, this substance was credited with medicinal properties and was used for candle oil.
Mature males may reach approximately 20 metres, with the head representing up to one-third of the animal's length. They feed primarily on squid, and adults are expected to reach more than 2500 metres in depth.
Sperm whales’ clicking vocalizations, which are as loud as 230 decibels, is the form of echolocation and communication they use underwater.
Sperm whales can live for more than 60 years. They are normally not aggressive to humans. Nevertheless, aggressive behaviours have been reported from groups protecting young individuals.

Note that the famous novel Moby-Dick by Herman Melville is based on the whaleboat Essex's sinking by an enraged sperm whale 2,000 miles from the western coast of South America in 1820.

16 - Crocodiles and other crocodilians

- Crocodiles

Crocodiles (subfamily Crocodylinae) or true crocodiles are large aquatic reptiles that live throughout the tropics in Africa, Asia, the Americas, and Australia. The term is sometimes used even more loosely to include all extant members of the order Crocodilia, including alligators, caimans (family Alligatoridae), and gharials (family Gavialidae). According to statistics, crocodiles kill 2500 people/year, and sharks kill between 60 and 70 people/year.

Two species of crocodile are present in Southeast Asia:

- Siamese crocodile

The Siamese crocodile lives exclusively inland. Its maximum length is 4 m. This species can be found in Cambodia, Indonesia (Borneo and possibly Java), Laos, Malaysia, Thailand, Vietnam, Brunei, and Burma. It is in regression, but a significant population is indicated in Kalimantan.

- Sea crocodile

The “Sea crocodile” (Crocodylus porosus) is a semi-aquatic reptile, living mainly in rivers, mangroves, and estuaries. Its geographical range extends over 10,000 km² of the Southeast Pacific, from East India to Fiji and from Southern China to North Australia. This crocodile spends most of its life in salt-water or melted waters. Its maximum length is nearly 7 m. It is known to travel at sea for long distances. Its jaws can apply a pressure of 250 bar. It is a very patient animal, able to wait for hours without any movements. It can stay around 30 minutes submerged, with only the top of its snout apparent at the surface of the water when it takes its breath. These huge animals can run more quickly than a man. The sea crocodiles are sufficiently strong to attack aggressive sharks like bull sharks.

One species is present in Africa:

- Nile crocodile

The “Nile crocodile” (Crocodylus niloticus) is an African crocodile that can be considered the second largest crocodile after the saltwater crocodile. It is quite widespread throughout Sub-Saharan Africa, occurring mostly in the central, eastern, and southern regions of the continent and lives in different aquatic environments such as lakes, rivers, and marshlands. This species is rarely found in saltwater but occasionally inhabits deltas and brackish lakes. On average, the adult Nile crocodile can range between 2.8 and 5 m in length and weigh around 70 to 700 kg. However, specimens exceeding 6 m in length and weighing more than 900 kg have been recorded.

The “Nile crocodile” is an opportunistic apex predator and a very aggressive crocodile species capable of taking almost any animal within its range. It is involved in many attacks and is considered very dangerous.

One species is present in America:

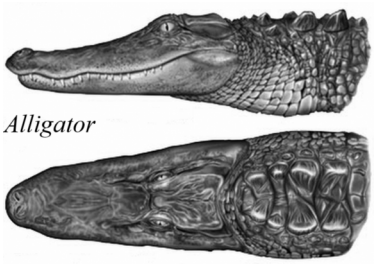
- American crocodile

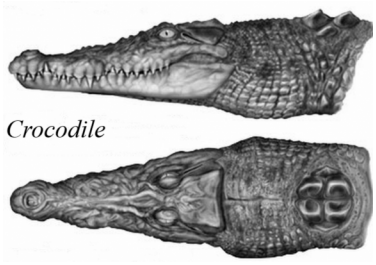
The American crocodile (Crocodylus acutus) is a crocodilian species found in the Neotropics from the Atlantic and Pacific coasts of southern Mexico to South America as far as Peru and Venezuela. It also lives on many Caribbean islands such as Cuba, Jamaica, Hispaniola, Grand Cayman, Greater Antilles, the West Indies, and the southern half of Florida.
Males can reach lengths of 6 m, weighing up to 900 kg. On average, mature males are more in the range of 4.5 m in length weighing about 400 kg. Females are smaller, rarely exceeding 3.8 m in length.
The American crocodile is found in coastal areas and river systems. It tends to prefer some salinity level and is often found in brackish lakes, mangrove swamps, lagoons, and small islands without any freshwater source.
Fish, reptiles, birds, and small mammals make up the majority of their diet. On occasion, large mammals such as deer and cattle are taken. Their dietary habits in coastal regions are not well studied. Like any other large crocodilian, the American crocodile is potentially dangerous to humans, but it is not a very aggressive species, and attacks are rare.

- Other crocodilians that can be encountered in costal areas

- Alligators

here are two species of alligators: The American alligators inhabit the Southeast parts of the USA, and the Chinese alligator found only in the Yangtze River valley.
The average size for an adult female is approximately 2.6 m, and the average size for a male is 3.4 m. Regarding physical differences, the easiest way to tell the difference between the two is that a crocodile has a very long, narrow, V-shaped snout while the alligator's snout is wider and U-shaped. Because of the alligator's wide snout, it packs more crushing power to eat prey like turtles that constitute part of its diet. Although still very powerful, the narrow crocodile snout is not suited for prey like turtles but is very versatile for fish and mammals.





Alligators are generally timid towards humans and tend to walk or swim away. Nevertheless, approaching alligators and their nests may provoke the animals to attack.

- Caimans

Caimans inhabit Central and South America. They are relatively mid-small-sized crocodilians, with the smallest being Cuvier's dwarf caiman (Paleosuchus palpebrosus), which grows to 1 m long, the largest being the black caiman (Melanosuchus niger), which can grow to 5 m or more.
Alligators and caimans belong to the same family Alligatoridae. Caimans' teeth are longer and more narrow than alligators. Also, caimans are known to be more agile animals.

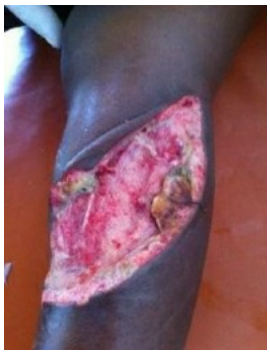
- Crocodilian attacks

- Experts indicate similar reasons as the big biting fish to explain the attacks from crocodiles:
  - Territorial defence (nest or hunt territory)
  - Self defense
  - Feeding reflexes
- Most of the time, nothing is visible at the surface of the water before the attack.
- The attacks are described as very violent and sudden, with no chance for the prey to escape. As an example, a sea crocodile is known to be able to leap its full body out of the water in a vertical position.
- Following the initial attack, the crocodile violently shakes its victim to tear it to pieces. That explains why the wounds from crocodiles bites are very extended, with often limbs ripped off for those who had the chance to survive.

- Symptoms

Injuries arising from crocodile attacks are from deep cuts, to tissue loss, limbs ripped off, and possible death due to the massive bleeding and traumas received.
- Similarly to injuries from big fish, there may be the following symptoms associated with the wounds:
  - An emotional shock caused by the fright resulting in stupor and restlessness.

- Massive haemorrhages.
- Fractures may be present.
- Internal organs may be injured with internal haemorrhages (due to crushing by the jaws and the violent shaking) or coming out due to big openings of the abdomen or the thorax.
- Hypovolemic shock due to the massive loss of blood (several phases of tachycardia followed by a sudden fall in pulse rate).
- Cardiogenic shock due to inadequate cardiac function (sudden acute breathlessness, fatigue with some relief in sitting position).
- Vasodilatation shock due to bacteria infection (probably not visible on-site if transferred to the hospital quickly).
- Septic shock due to the bacterial infection (probably not visible on-site if transferred to the hospital quickly).
- Death may occur quickly depending on the haemorrhages and traumas.



- Treatment

The treatment to implement is similar to what is done for the fish bites, depending on the injury and whether it is life-threatening or not.

- In all cases:

- Immediately recover the diver.
- Organize the medical evacuation.

- Small wounds:

- Stop the bleeding, if any.
- Clean the wound and remove foreign objects if possible and if there are any.
- Examine and report to the doctor in charge.
- Look for other injuries depending on the type of the wound (Broken limb, internal bleeding).
- Relieve the pain via infiltration of local anaesthetics without adrenaline (epinephrine) into and around the wound.
- Monitor temperature, pulse, respiration, blood pressure.
- Broad-spectrum antibiotics to be used at an early stage.
- Treat for an emotional shock: Patient laid flat with raised legs + support.

- Big wounds with massive bleeding

- The bleeding must be stopped by direct pressure *(application of cold packs or ice packs over the compress, not under it, may hasten the process by initiating spasm and closure of disrupted blood vessels)*.
- Clean the wound and remove foreign objects if possible and if there are any.
- Treat for the hypovolemic shock: Lay the patient with the legs raised + intravenous infusion of normal saline + colloid *(to maintain the osmotic pressure exerted by proteins in a blood vessel's plasma)*.
- Treat the cardiogenic shock: Breathing support using O_2, the patient sat upright.
- In case of heart failure: resuscitation *(CPR or defibrillation if available + breathing support O_2)*
- A tourniquet is to be used only in a life-threatening situation and is best applied by an experienced person. Only in the case of torrential bleeding is a tourniquet more advantageous than continuous pressure. The decision to apply a tourniquet is one in which a limb is sacrificed to save a life.
- If the victim has suffered a large wound through which internal organs (such as loops of bowel) or bones are protruding, no attempt should be made to push these back inside the body or under the skin unless they slide back in without assistance. The extruded internal organs or bones must be covered with continually moistened bandages (pads of gauze or cloth) held in place without excess pressure. Seek immediate medical attention.
- If the victim has a severe cut on his neck, the diver medic should be sufficiently careful not to disturb the wound because such disturbance might remove a blood clot that controls the bleeding from a large blood vessel. A firm pressure dressing is to be applied, and close observation is to be done with continual airway assessment because an expanding blood clot within the neck can compress the throat and windpipe. Evacuation is urgent
- When the external bleeding is stopped, the injury is to be immobilized. A complimentary examination and a

report to the doctor in charge must be undertaken (photos are welcome in complement)

- Look for other injuries depending on the wound's type (broken limb, internal bleeding…).
- If internal bleeding, such as from a broken bone or injured organ, the only treatment is surgery and cannot be done on-site.
- Relieve the pain via administration of local anaesthetics without adrenaline (epinephrine) into and around the wound.
- Monitor temperature, pulse, respiration, blood pressure.
- Broad-spectrum antibiotics are to be used at an early stage.

17 - Precautions against venomous and aggressive animals

Basic precaution against dangerous underwater life can be based on three points:

→ Protection

→ Behavioral

→ Prevention

- Protection consists mostly of the use of full Personal Protective Equipment:

- The diver must wear a diving wet suit or a dry suit. If the diver wears a coverall, it must be sufficiently thick to protect him from contact with urchins, coral, or all sorts of venomous species. There must not be any openings that could let some small poisonous animals like Jellyfish larva or small octopus be trapped inside the suit (so, closing by buttons is prohibited).
- The diver must wear gloves and boots and/or booties at all times, and there must not be any unprotected skin at the wrist, ankle, or neck levels.
- Protections against big animals like nets can be organized on some work sites, but not on the vast majority. It is also clear that electric fields repel the sharks. This has been seen by ROV pilots and 3.4 u inspectors when doing CP reading. Note that an Australian company (named “Shark shield”) has developed an electronic shark deterrent to protect the surfers and divers. Unfortunately, a report from the government of South Australia (SARDI publication N°2012/000123-1) concludes that it works some of the time but not all the time. Notice that the products based on “copper sulfate” and “copper acetate” do not work.
- The behavioural consists of the diving team's basic common sense and the people living on the facilities.
- Avoid touching things directly by hand is a basic precaution (sticks, knives, tools to be used instead), particularly if visibility is bad or a lot of marine growth (think of scorpionfish and other venomous animals).
- The diver must not put hands into cavities because these cavities are often used as shelter by animals like snakes, moray eels, and octopuses.
- Avoid provoking fish by flashing objects, and avoid any aggressive behaviour.
- Stop the dive when big predators are becoming too inquisitive.
- Forbid the dumping of rubbish in the sea, particularly fish and meat remains because that attracts and causes aggressive behaviour. The interdiction of food dumping is already in force for some companies, but others continue this practice despite the risk of bites to divers and people falling into the sea.
- Forbid fishing on the worksites: Fish caught online are emitting vibrations that will be detected by the big predators. (Barracuda, sharks, and crocodiles)
- The diver must be humble and consider that animals can become extremely aggressive to defend their eggs or territory, even those with no teeth and are not indicated as aggressive.
- The big biting fish indicated in this chapter are those classified as aggressive. For example, the reef blacktip shark, which is 1.5 m long, has been indicted for numerous non-fatal aggressions to swimmers and divers and is common everywhere in tropical areas. The great white shark, which is the most famous and powerful shark, is responsible for many deadly attacks. Also, note that some big predators could wander outside their normal surroundings. It has already happened in the past and probably happens every day… To be safe, the diver must consider that every fish can become aggressive and create a huge problem, even those with no teeth.

- Prevention is associated with the organization of the job.

- Young divers must be briefed on the dangers of underwater life. Normally they will mostly focus on the big fish. Still, safety records clearly indicate that the most deadly creatures they are likely to encounter first are small poisonous animals like the box jellyfish or the blue-ringed octopus.
- The team must be prudent when diving in water with high turbidity, or worse, brown colour, usually found in deltas and rivers. This kind of water is the favourite hunting domain of crocodiles, bull sharks, stingrays, and other fish, which could cause accidents. If any danger from the fauna is suspected, the dive must be aborted. It must also be considered that crocodiles will target the diver and perhaps the people working just over the side.
- Regarding the poisonous animals, “CSL Corporate” based in Australia has developed some efficient anti-venom to treat poisoning from: Box jellyfish; Sea snake; Stone fish.

Unfortunately, there is at the moment NO anti-venom to solve the poisoning from the blue-ringed octopus.
The head office address of this company is:
CSL Corporate *Head Office 45 Poplar Rd, Parkville Victoria 3052 Australia Phone: +61 3 9389 1911 Fax: +61 3 9389 1434 - Website:http://www.csl.com.au/contact-csl*
These anti-venoms should be included in the medical kit to prevent any poisoning arising from these 3 animals.

- In addition, acetic acid or vinegar must also be included in the medical kit as it is the basic complementary treatment for stings from urchin, anemones, jellyfishes, hydroids. It is also recommended to include additional compresses in case of extended traumatic wounds with massive bleeding.

Seasickness

Seasickness affects many people to varying degrees, even sailors with years of experience.

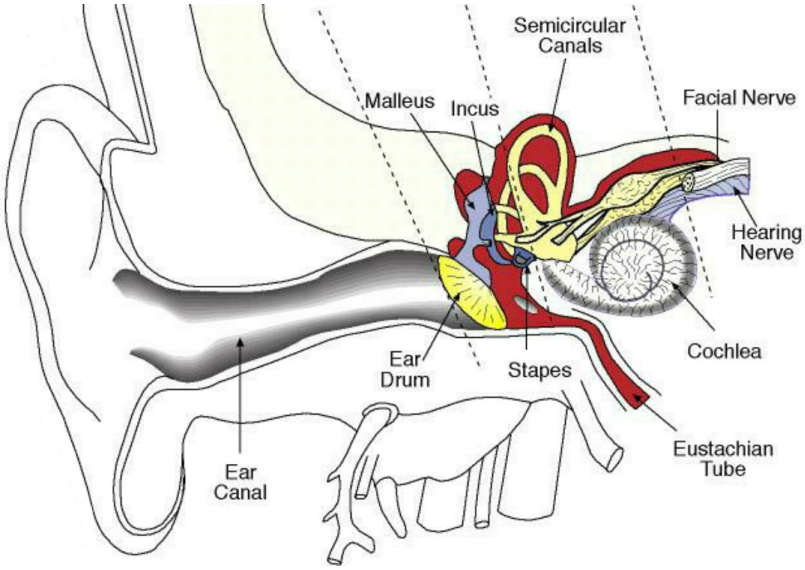
Symptoms:

Seasickness usually results in the following symptoms:

- Cold sweats
- Dizziness
- Fatigue
- Headache
- Irritability
- Inability to concentrate
- Increased salivation
- Pale skin
- Rapid breathing
- Nausea and vomiting

Mechanism:

The movements of the body are registered by the brain through the inner ear, notably the vestibule and the semicircular canals that sense balance and posture to assist in equilibrium.

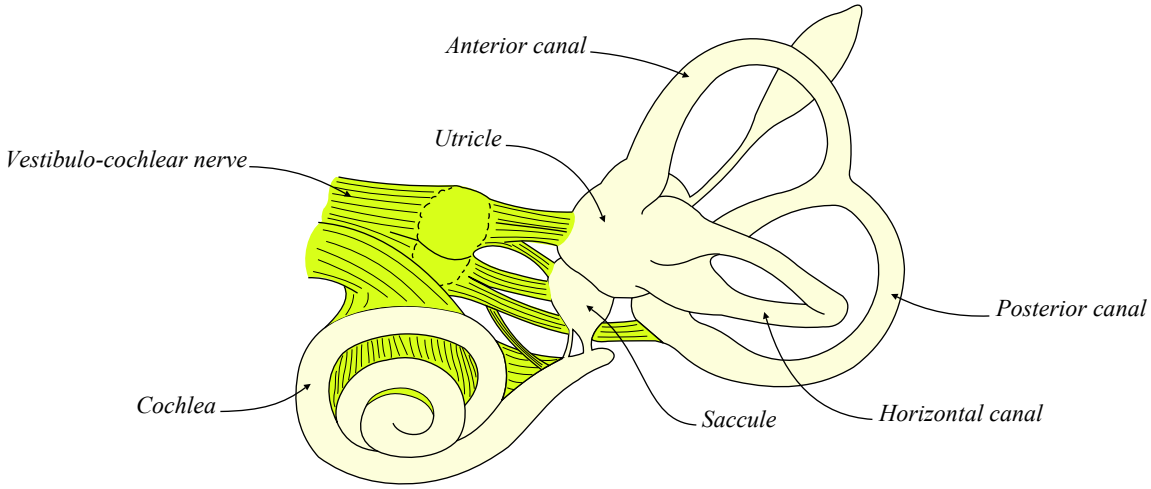


The semicircular canals are motion sensors lined with cilia (microscopic hairs) and filled with a liquid substance, known as endolymph. They are composed of three parts:

- The anterior canal detects forward and back head movements.
- The posterior canal detects head tilt like tipping the head toward the shoulders.
- The horizontal canal detects horizontal movement of the head, such as swivelling the head from side to side.

The endolymph moves the cilia when the head or the entire body moves, and the cilia movements are communicated to the cerebellum.
The semicircular canals are linked to the cerebellum by the vestibule that comprises the “utricle” and the “saccule”, that lie against the inner ear walls between the semicircular ducts and the cochlea. These receptors are patches of hair cells topped by small, calcium carbonate crystals called otoconia. The saccule and utricle lie at 90 degrees to each other. Thus, when the head moves, the gravity bends the cilia of one patch of hair cells due to the weight of the otoconia to which a gelatinous layer attaches them. This bending of the cilia produces afferent activity going through the brainstem.

The utricle is most sensitive to tilt when the head is upright. The saccule is most sensitive to tilt when the head is horizontal. Note that the saccule is also linked to the cochlea.
The balance organs of the right ear are paired with those of the left ear. Besides, sensors in the skin, joints, and muscles provide information to the cerebellum on the position of parts of the body in relation to each other and the body's position in relation to the environment. Information on the environment is also provided by the eyes that confirm the indications from the ear and body sensors and allow to adjust the posture of the body and calculate distances used for the next moves.



Seasickness usually affects people when they are inside the vessel, and their ear and body sensors detect changes in the vessel's motion and accelerations, resulting in the condition of the sea, where their eyes register a stable scene. As a response to such incongruity, their brains produce stress-related hormones that lead to nausea, vomiting, and vertigo.

Control:

It is well known that the body adapts after time. Also, seasickness tablets and patches allow controlling the problem partially. These tablets should be selected by the diving medical specialist and be part of the medical kit of the Self Propelled hyperbaric lifeboat and provided to the divers in the chambers and supporting teams. Numerous brands exist that can be bought over the counter. Among them, note the following:

- Hyoscine, also known as scopolamine, is the medication recommended in IMCA D 052 to treat motion sickness. Its effects begin after about 20 minutes and last for up to 8 hours. It is provided in the form of tablets, patches, or by injection.
- Dimenhydrinate is sold under the brands Dramamine, Driminate, &Triptone. Its effects begin after 30 minutes to 1 hour, and it should be renewed every 4 to 6 hours.
- Meclizine is sold under the brands Agyrax, Antivert, Bonine, D-Vert, Dramamine Less Drowsy, Driminate II, Meclicot, Medivert, Ru-Vert-M, Meni-D. Note that drowsiness and headache may result as a side effect of taking this medicine. Its effects begin after 1 hour and last for up to 24 hours.

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Part D - Recompression tables US Navy revision 7

1 - Introduction

The “decompression tables” are designed to avoid decompression sickness (DCS), and as indicated in in the “presentation of the DCIEM tables” in the manual “Air diving procedures”, they are mainly based on mathematical models and statistics. Their procedure of calculation of the necessary decompression time is according to the depth and the bottom time at this depth.

The “recompression treatment tables” are designed to solve the problems arising in cases of decompression sickness or Arterial Gas Embolism. The selection of the tables to apply is mainly based on symptoms (AGE or DCS type 1 or 2…), and the dive profile is used only to refine the treatment. In fact the recompression treatment tables are medical procedures, and must be applied under the supervision of a competent hyperbaric physician. The mainstay of treatment for DCS is recompression followed by a slow decompression back to atmospheric pressure with the hope the patient will be cured. Many competent bodies editing decompression tables are also promoting their own recompression treatment tables. But instead of focusing on only one set of tables, a lot of physicians are using tables from different origins to solve the problems they can be confronted with. For example in the book “diving sub aquatic medicine 4th edition” the doctor Robyn Walker indicates the US navy recompression tables, but also the “RAN” tables and “COMEX” tables… In the well-known book “diving medicine 4th edition Bove & Davis”, the doctor Richard E Moon uses the US navy tables, but also “Comex”, and other tables. It is the same for the professor Fructus, who despite the fact he was employed by Comex, indicated the possibility of using the USN tables in his book “plongee Sante Securite” (diving, Health, safety). Thus, there is the possibility that for a treatment, the hyperbaric physician in charge decides to put in service a particular table preferably to the one associated with the decompression tables in use by the contractor… But, on company side, to make sure of clear procedures and avoid any confusion and loss of time, it is preferable to use only one set of well-known recompression tables. The set of tables to be used is selected according to the recommendations of the hyperbaric doctor appointed by the company. This is the reason why the US Navy recompression tables have been selected. The procedures indicated below are from those indicated by the US Navy.

2 - Description

- Aim of the recompression treatment tables

Treat diving disorders

This is the original purpose of these tables. In this case, the aim of recompression treatment is:

- Produce an immediate reduction in bubble size, which will:
  - Cause the surface tension pressure acting on the bubble to increase, which may lead to resolution
  - Increase the surface area of the bubble relative to its volume, enhancing gas diffusion out of the bubble
  - Reduce the length of the gas column that is trapped intravascularly and hence may allow perfusion pressure to push it into the venous circulation
  - Reduce the compression of adjacent tissues
  - Reduce the bubble-tissue and bubble-blood interface and the secondary inflammatory reactions.
- Increase the diffusion gradient of gas out of the bubble
- Increase the blood oxygen content and thus oxygen delivery to injured tissues
- Relieve ischemia *(restriction of blood supply to tissues)* and hypoxia *(restriction of Oxygen supply to tissues)*
- Restore normal tissue function.

Treat some non diving disorders:

- In addition to individuals suffering from diving disorders, U.S. Navy recompression chambers are also being used to conduct emergent hyperbaric oxygen (HBO_2) therapy to treat individuals suffering from cyanide poisoning, carbon monoxide poisoning, gas gangrene, smoke inhalation, necrotizing soft-tissue infections, or arterial gas embolism arising from surgery, diagnostic procedures, or thoracic trauma.
- Notice that any treatment of a non-diving related medical condition shall be done under the cognizance of the Diving Medical Specialist (doctor).

- The recompression tables US Navy

Two sets of tables are designed:

1 - The recompression tables using oxygen are the tables 5, 6A, 4, 7, 8 and 9

- The descent rate for all these tables is 20 feet per minute.
- For treatment depths deeper than 60 fsw, other treatment gas than air is to be used if available.

2 - The Air recompression treatment tables are the tables 1A, 2A, and 3, and are provided for use only as a last resort when oxygen is not available.

- "Air Treatment Table 1A" is to be used if the pain is relieved at a depth less than 66 feet.
- If the pain is relieved at a depth greater than 66 feet, the "Air Treatment Table 2A" is to be used
- "Treatment Table 3" is to be used for treatment of serious symptoms where oxygen cannot be used, and if the symptoms are relieved within 30 minutes at 165 feet. If the symptoms are not relieved in less than 30 minutes at 165 feet, "Treatment Table 4' shall be used

Note: Particular case for table 4":
The Air treatment tables shall only be used after oxygen system failure or intolerable patient oxygen toxicity problems with the hyperbaric medical specialist (doctor) recommendation. "Treatment Table 4" can be used with or without oxygen but should always be used with oxygen if it is available.

Oxygen tables versus air tables

The standard oxygen therapeutic tables have the following advantages over the standard air tables:

- Economy of time
- Increased speed of bubble resolution (increased nitrogen gradient)
- Increased oxygenation of tissues
- Flexibility of combining with mixture and air tables
- Better results than air tables

Disadvantages include:

- Less immediate reduction in bubble size
- Intolerance of patient to oxygen or mask
- Increased risk of fire
- Chronic oxygen toxicity

Note: As indicated before, the oxygen tables are the normal procedure, and the air tables must not be used except in cases of lack of oxygen. It must be remembered that IMCA D 050 recommends 90 m^3 O_2 be available for emergency treatments purpose, and the dive cannot be launched without this gas reserve.

High oxygen mixes & treatment of heliox dives :

- High oxygen $_{N2O2/HeO2}$ mixtures may be used to treat patients when recompression deeper than 60 fsw is required. These mixtures offer significant therapeutic advantages over air. Select a treatment gas that will produce a ppO_2 between 1.5 and 3.0 ata at the treatment depth. The standardized gas mixtures shown in the table below are suitable over the depth range of 61-225 fsw.
- Decompression sickness following helium dives can be treated with either nitrogen or helium mixtures. For recompression deeper than 165 fsw, helium mixtures are preferred to avoid narcosis. The situation is less clear for treatment of decompression sickness following air or nitrogen-oxygen dives. Experimental studies have shown both benefit and harm with helium treatment. Until more experience is obtained, high oxygen mixtures with nitrogen as the diluent gas are preferred if available. High oxygen mixtures may also be substituted for 100% oxygen at 60 fsw and shallower on Treatment Tables 4, 7, and 8 if the patient is unable to tolerate 100% oxygen.

Depth (fsw)	*Mix (heO_2 or $N2O_2$)*	*PPO_2*
0 - 60	*100 %*	*1.00 to 2.82*
61 - 165	*50 %*	*1.42 to 3.00*
166 - 225	*36% O2*	*2.17 to 2.81*

- Regarding the use of Heliox , DMAC 23 states:
"There are many ways of treating decompression illness at raised environmental pressure. DMAC endorses the use by company medical advisers of helium/oxygen mixtures as an option in the treatment of decompression illness arising from air or Trimix diving. Cases have been successfully treated on 50/50 Heliox (Comex 30) and on the US Navy recompression tables with 80/20 Heliox substituted for air. Helium and oxygen saturation therapy may also be employed."

Remembering the procedure for selecting the tables

Treatments of Arterial Gas Embolism (AGE)

- The tables to be used will be table 6 *(compression at 60 ft)* at first followed by table 6A in cases of severe symptoms *(165 ft)*. The patient will have to be compressed to the depth of significant improvement, but never passing below 165 ft. The patient should begin oxygen breathing periods immediately upon arrival at the 60-foot stop . If the severity of the patient's condition warrants, the hyperbaric medical specialist (Doctor) may decide conversion to a Treatment Table 4.
- Treatment Table 4 is used when it is determined that the patient would receive additional benefit at depth of significant relief, not to exceed 165 fsw. The time at depth shall be between 30 and 120 minutes, based on the patient's response.
- In case of non response , the doctor can decide to apply table 7.

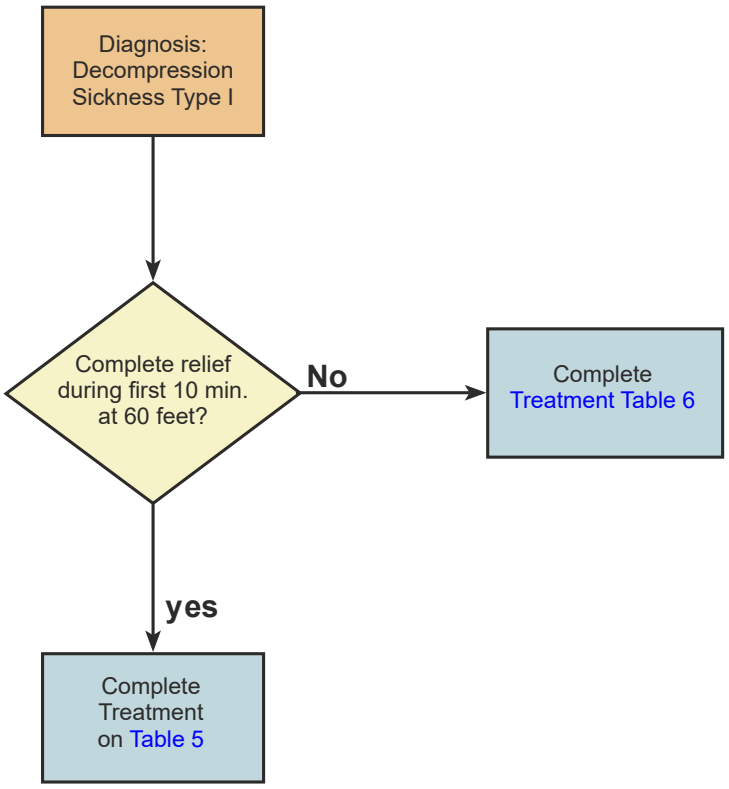
Treatment of decompression sickness (DCS):

- Type I Decompression Sickness is treated using table 5 in accordance with the dedicated chart. If a full neurological exam is not completed before initial recompression, the casualty will have to be treated as a Type II symptom.
- Symptoms of musculo-skeletal pain that have shown absolutely no change after the second oxygen breathing period at 60 feet may be due to orthopedic injury rather than decompression sickness. If, after reviewing the patient's history, the Hyperbaric Doctor feels that the pain can be related to specific orthopedic trauma or injury, Treatment Table 5 may be completed. If a Doctor is not reachable, treatment Table 6 shall be used.
- Type II Decompression Sickness is treated with initial compression to 60 fsw using table 6 in accordance with the chart displayed next page.
- If the symptoms improve within the first oxygen breathing period, then treatment is continued on a treatment table 6.
- If severe symptoms are unchanged or worsen within the first 20 minutes at 60 fsw, assess the patient during descent and compress to depth of relief , not to exceed 165 fsw. Treat with Treatment Table 6A. To limit recurrence, severe Type II symptoms warrant full extensions at 60 fsw even if symptoms resolve during the first oxygen breathing period.
- As for the Arterial Gas Embolism, other tables may be used, but the decision to use tables 4 and 7 have to be taken only by the hyperbaric doctor in charge of the treatment.

Treatment charts:

- Some treatment charts are provided with the US Navy recompression tables. The aim of these charts is to help the team to visualize the procedure to follow according to the response of the casualty to the treatment.
- Notice that these charts have to be strictly followed

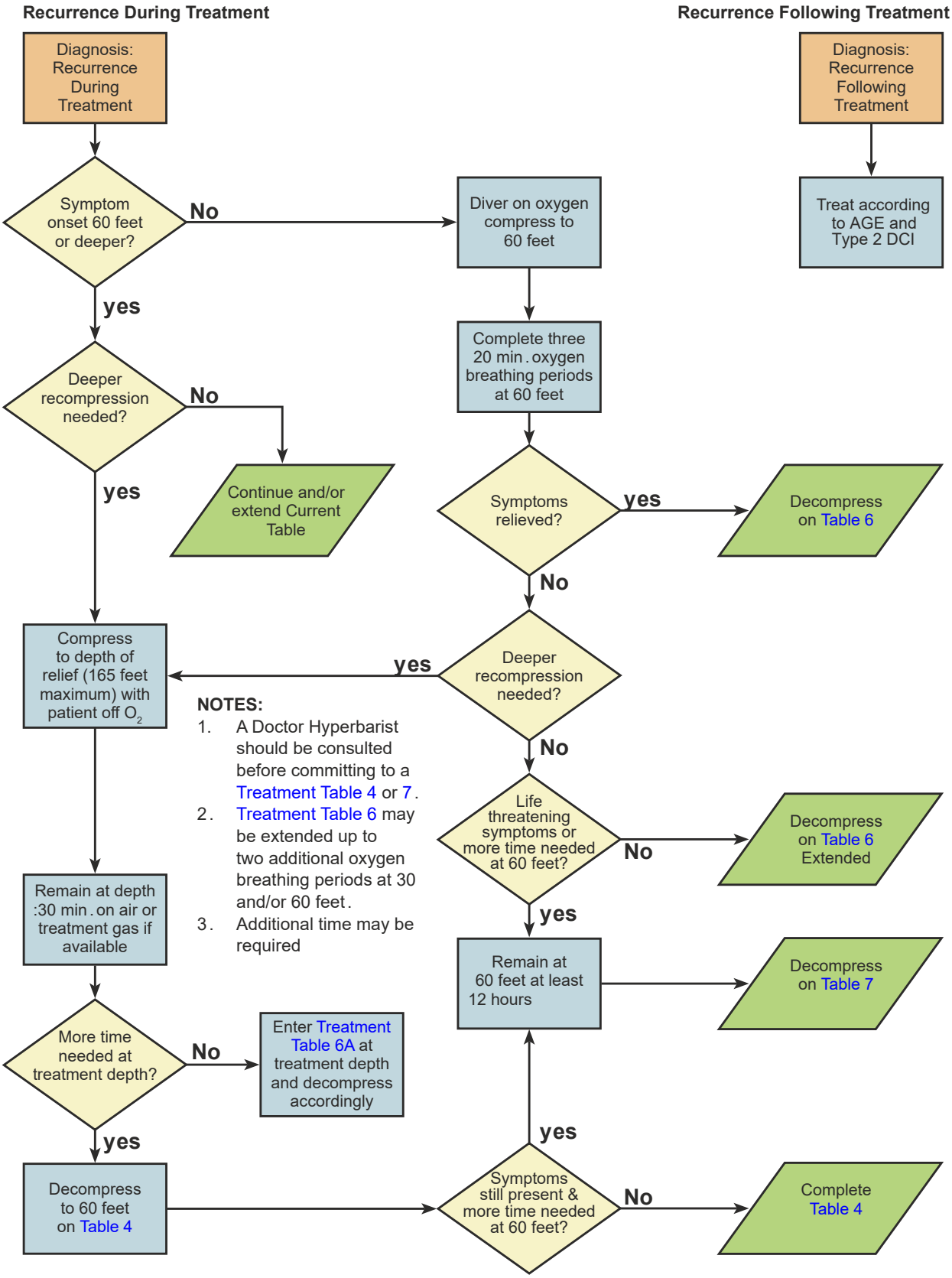
Treatment of Type I Decompression Sickness



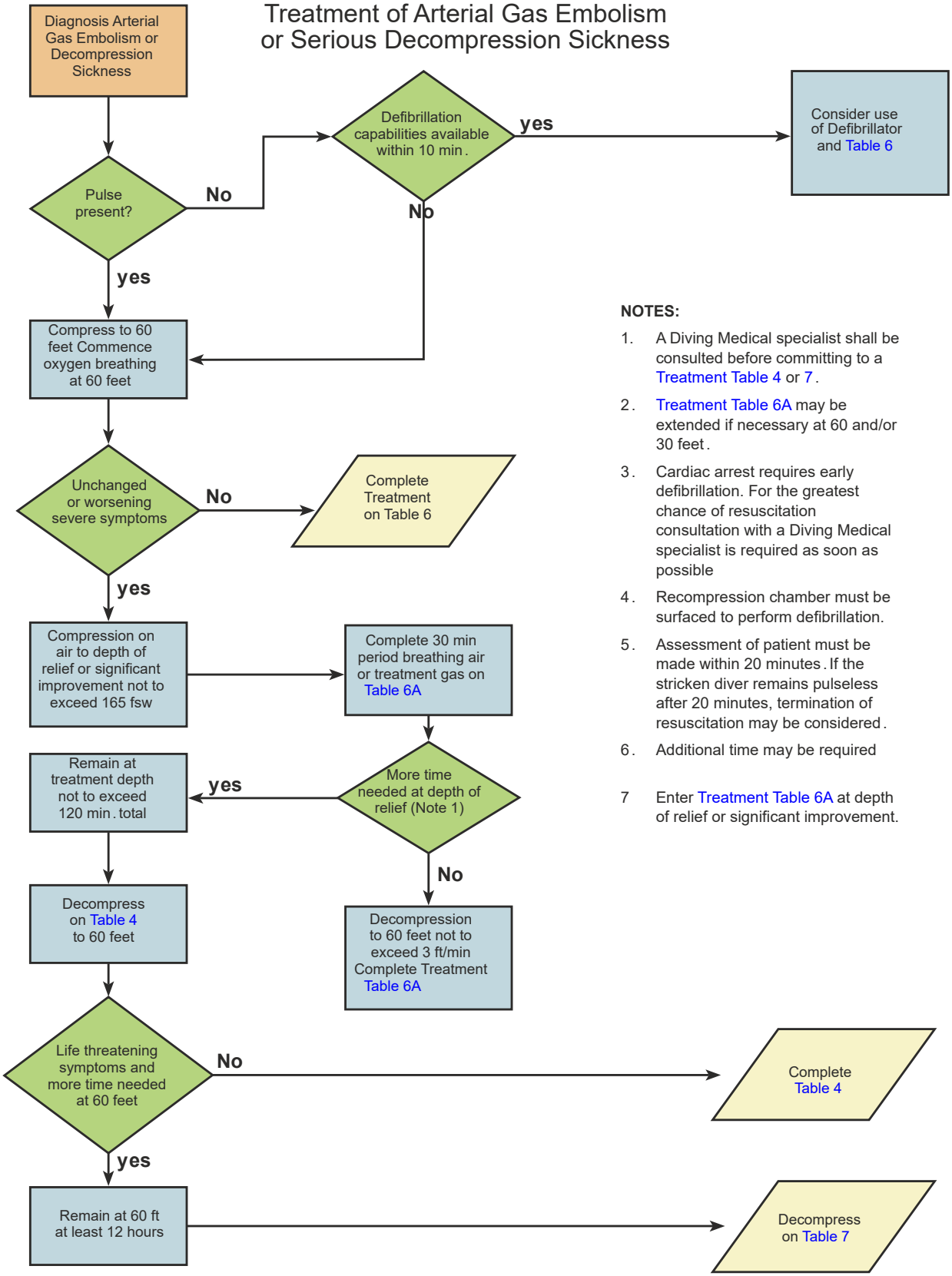
NOTES:

1. If a complete neurological exam was not completed before recompression, treat as a Type II symptom.
2. Treatment Table 6 may be extended up to four additional oxygen-breathing periods, two at 30 feet and/or two at 60 feet.
3. Diving Supervisor may elect to treat on Treatment Table 6.
4. Treatment Table 5 may be extended two oxygen-breathing periods at 30 fsw.

Treatment of Symptom Recurrence



Treatment of Arterial Gas Embolism or Serious Decompression Sickness



NOTES:

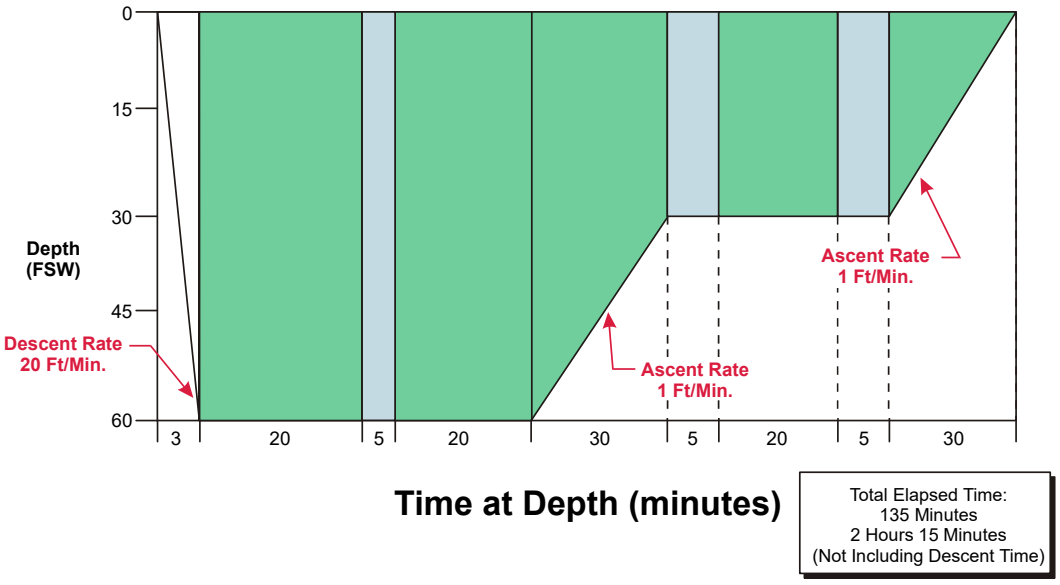
1. A Diving Medical specialist shall be consulted before committing to a Treatment Table 4 or 7.
2. Treatment Table 6A may be extended if necessary at 60 and/or 30 feet.
3. Cardiac arrest requires early defibrillation. For the greatest chance of resuscitation consultation with a Diving Medical specialist is required as soon as possible
4. Recompression chamber must be surfaced to perform defibrillation.
5. Assessment of patient must be made within 20 minutes. If the stricken diver remains pulseless after 20 minutes, termination of resuscitation may be considered.
6. Additional time may be required

7 Enter Treatment Table 6A at depth of relief or significant improvement.

Treatment Table 5

1. Descent rate - 20 ft/min.
2. Ascent rate - Not to exceed 1 ft/min. Do not compensate for slower ascent rates. Compensate for faster rates by halting the ascent.
3. Time on oxygen begins on arrival at 60 feet.
4. If oxygen breathing must be interrupted because of CNS Oxygen Toxicity, allow 15 minutes after the reaction has entirely subsided and resume schedule at point of interruption
5. Treatment Table may be extended two oxygen-breathing periods at the 30-foot stop. No air break required between oxygen-breathing periods or prior to ascent.
6. Tender breathes 100 percent O_2 during ascent from the 30-foot stop to the surface. If the tender had a previous hyperbaric exposure in the previous 18 hours, an additional 20 minutes of oxygen breathing is required prior to ascent.

Treatment Table 5 Depth/Time Profile



Description

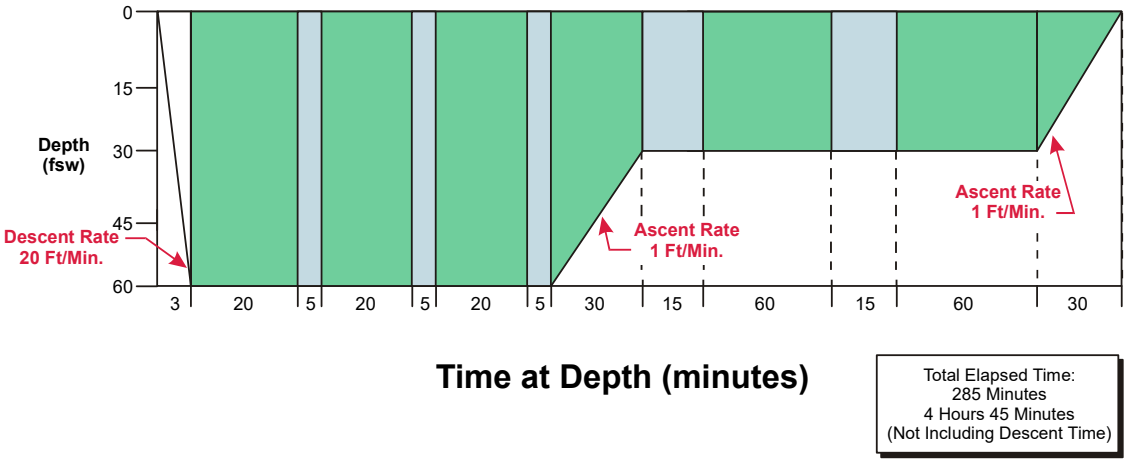
Treatment Table 5 may be used for the following:

- Type I DCS
- Asymptomatic omitted decompression
- Treatment of resolved symptoms following in-water recompression
- Follow-up treatments for residual symptoms
- Carbon monoxide poisoning
- Gas gangrene

Treatment Table 6

1. Descent rate - 20 ft/min.
2. Ascent rate - Not to exceed 1 ft/min. Do not compensate for slower ascent rates. Compensate for faster rates by halting the ascent.
3. Time on oxygen begins on arrival at 60 feet.
4. If oxygen breathing must be interrupted because of CNS Oxygen Toxicity, allow 15 minutes after the reaction has entirely subsided and resume schedule at point of interruption (see paragraph 20-7.11.1.1).
5. Table 6 can be lengthened up to 2 additional 25-minute periods at 60 feet (20 minutes on oxygen and 5 minutes on air), or up to 2 additional 75-minute periods at 30 feet (15 minutes on air and 60 minutes on oxygen), or both.
6. Tender breathes 100 percent O_2 during the last 30 min. at 30 fsw and during ascent to the surface for an unmodified table or where there has been only a single extension at 30 or 60 feet. If there has been more than one extension, the O_2 breathing at 30 feet is increased to 60 minutes. If the tender had a hyperbaric exposure within the past 18 hours an additional 60-minute O_2 period is taken at 30 feet.

Treatment Table 6 Depth/Time Profile



Description

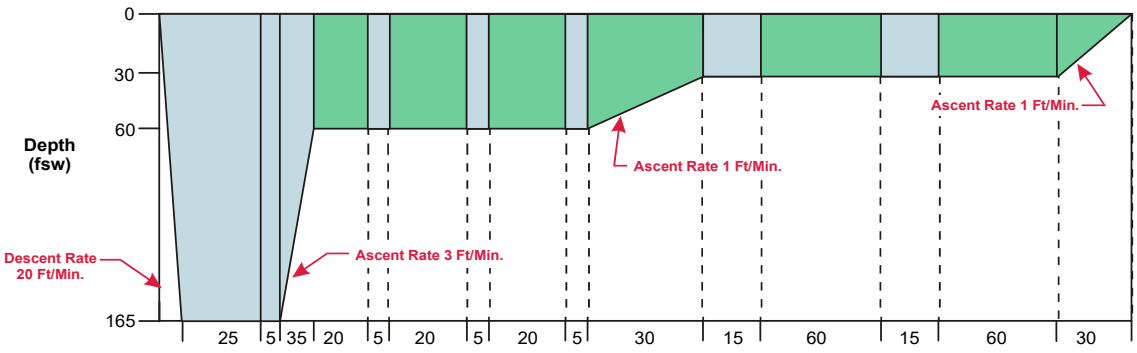
Treatment Table 6 is used for the following:

- Arterial gas embolism
- Type II DCS symptoms
- Type I DCS symptoms where relief is not complete within 10 minutes at 60 feet or where pain is severe and immediate recompression must be instituted before a neurological examination can be performed
- Cutis marmorata
- Severe carbon monoxide poisoning, cyanide poisoning, or smoke inhalation
- Asymptomatic omitted decompression
- Symptomatic uncontrolled ascent
- Recurrence of symptoms shallower than 60 fsw

Treatment Table 6A

1. Descent rate - 20 ft/min.
2. Ascent rate - 165 fsw to 60 fsw not to exceed 3 ft/min, 60 fsw and shallower, not to exceed 1 ft/min. Do not compensate for slower ascent rates. Compensate for faster rates by halting the ascent.
3. Time at treatment depth does not include compression time.
4. Table begins with initial compression to depth of 60 fsw. If initial treatment was at 60 feet, up to 20 minutes may be spent at 60 feet before compression to 165 fsw. Contact a Diving Medical Officer.
5. If a chamber is equipped with a high-O_2 treatment gas, it may be administered at 165 fsw and shallower, not to exceed 3.0 ata O_2
   Treatment gas is administered for 25 minutes interrupted by 5 minutes of air. Treatment gas is breathed during ascent from the treatment depth to 60 fsw.
6. Deeper than 60 feet, if treatment gas must be interrupted because of CNS oxygen toxicity, allow 15 minutes after the reaction has entirely subsided before resuming treatment gas. The time off treatment gas is counted as part of the time at treatment depth. If at 60 feet or shallower and oxygen breathing must be interrupted because of CNS oxygen toxicity, allow 15 minutes after the reaction has entirely subsided and resume schedule at point of interruption
7. Table 6A can be lengthened up to 2 additional 25-minute periods at 60 feet (20 minutes on oxygen and 5 minutes on air), or up to 2 additional 75-minute periods at 30 feet (60 minutes on oxygen and 15 minutes on air), or both.
8. Tender breathes 100 percent O_2 during the last 60 minutes at 30 fsw and during ascent to the surface for an unmodified table or where there has been only a single extension at 30 or 60 fsw. If there has been more than one extension, the O_2 breathing at 30 fsw is increased to 90 minutes. If the tender had a hyperbaric exposure within the past 18 hours, an additional 60 minute O_2 breathing period is taken at 30 fsw.
9. If significant improvement is not obtained within 30 minutes at 165 feet, consult with a Diving Medical Officer before switching to Treatment Table 4.

Treatment Table 6A Depth/Time Profile



Time at Depth (minutes)

Total Elapsed Time:
350 Minutes
5 Hours 50 Minutes
(Not Including Descent Time)

Description

Treatment Table 6A,is used to treat:

- Arterial gas embolism
- Decompression symptoms when severe symptoms remain unchanged or worsen within the first 20 minutes at 60 fsw.

1 - The patient is compressed to depth of relief (or significant improvement), not to exceed 165 fsw.

2 - Once at the depth of relief, begin treatment gas (N_2O_2, HeO_2) if available. Consult with a Diving Medical Specialist (doctor) at the earliest opportunity.

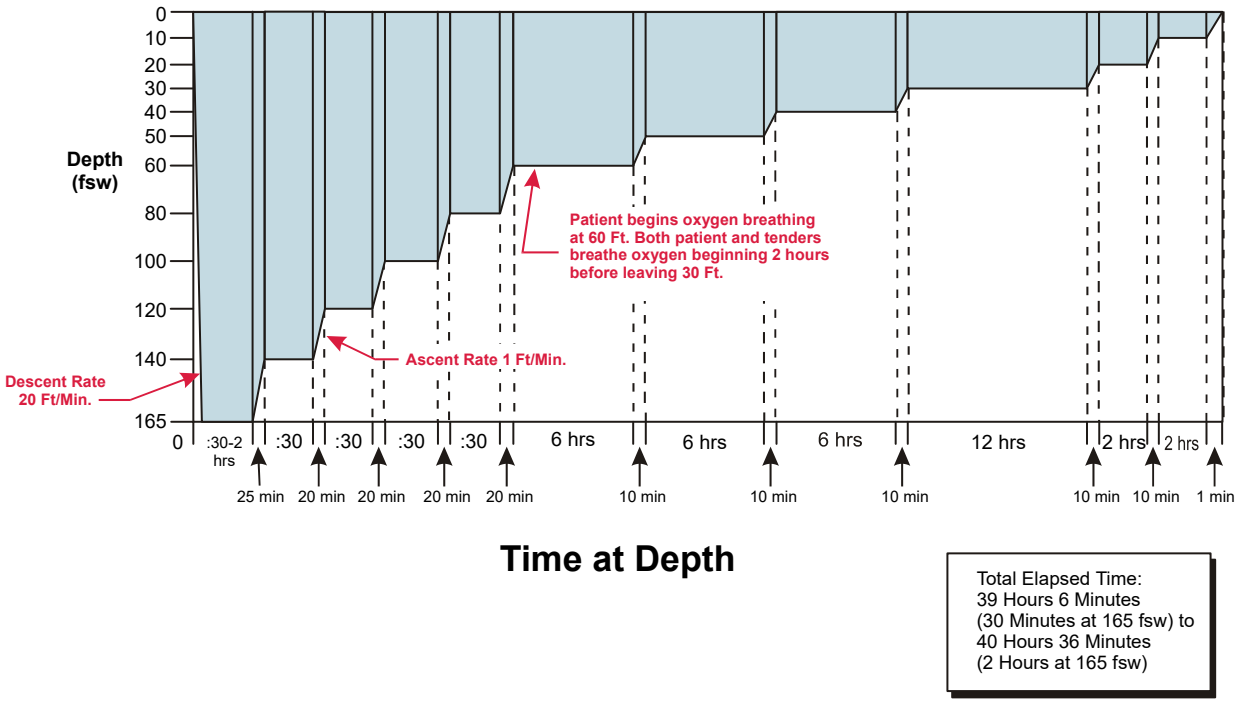
3 - If the severity of the patient's condition warrants, the Diving Medical Officer may recommend conversion to a Treatment Table 4.

Note - If deterioration or recurrence of symptoms is noted during ascent to 60 feet, treat as a recurrence of symptoms

Treatment Table 4

1. Descent rate - 20 ft/min.
2. Ascent rate - 1 ft/min.
3. Time at 165 feet includes compression.
4. If only air is available, decompress on air. If oxygen is available, patient begins oxygen breathing upon arrival at 60 feet with appropriate air breaks. Both tender and patient breathe oxygen beginning 2 hours before leaving 30 feet.
5. Ensure life-support considerations can be met before committing to a Table 4.
6. Internal chamber temperature should be below 85° F.
6. If oxygen breathing is interrupted, no compensatory lengthening of the table is required.
7. If switching from Treatment Table 6A or 3 at 165 feet, stay a maximum of 2 hours at 165 feet before decompressing.
8. If the chamber is equipped with a high-O_2 treatment gas, it may be administered at 165 fsw, not to exceed 3.0 ata O_2. Treatment gas is administered for 25 minutes interrupted by 5 minutes of air.

Treatment Table 4 Depth/Time Profile



Total Elapsed Time:
39 Hours 6 Minutes
(30 Minutes at 165 fsw) to
40 Hours 36 Minutes
(2 Hours at 165 fsw)

Description

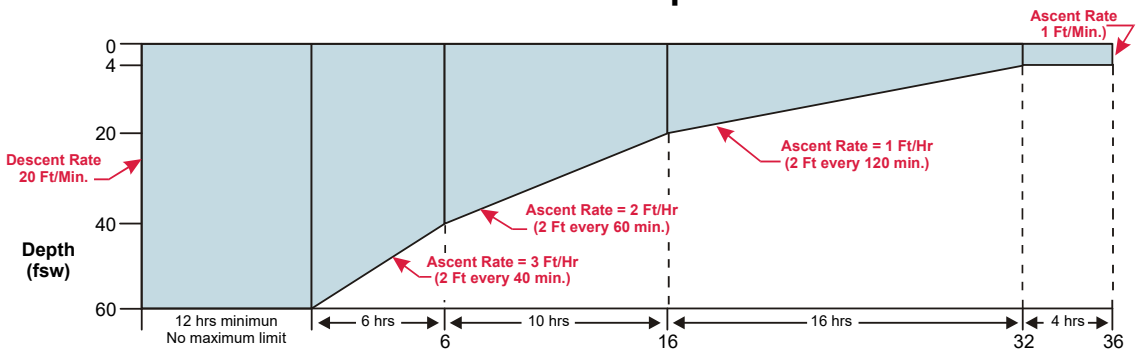
Treatment Table 4 is used when it is determined that the patient would receive additional benefit at depth of significant relief, not to exceed 165 fsw.

- The time at depth shall be between 30 to 120 minutes, based on the patient's response.
- If a shift from Treatment Table 6A to Treatment Table 4 is contemplated, the Diving Medical Specialist should be consulted before the shift is made.
- If oxygen is available, the patient should begin oxygen breathing periods immediately upon arrival at the 60-foot stop. Breathing periods of 25 minutes on oxygen, interrupted by 5 minutes of air, are recommended because each cycle lasts 30 minutes. This simplifies timekeeping.
- Immediately upon arrival at 60 feet, a minimum of four oxygen breathing periods (for a total time of 2 hours) should be administered.
- After that, oxygen breathing should be administered to suit the patient's individual needs and operational conditions.
- Both the patient and tender must breathe oxygen for at least 4 hours (eight 25-minute oxygen, 5-minute air periods), beginning no later than 2 hours before ascent from 30 feet is begun.
- These oxygen-breathing periods may be divided up as convenient, but at least 2 hours worth of oxygen breathing periods should be completed at 30 feet.
- Note - If deterioration or recurrence of symptoms is noted during ascent to 60 feet, treat as a recurrence of symptoms

Treatment Table 7

1. Table begins upon arrival at 60 feet. Arrival at 60 feet is accomplished by initial treatment on Table 6, 6A or 4. If initial treatment has progressed to a depth shallower than 60 feet, compress to 60 feet at 20 ft/min to begin Table 7.
2. Maximum duration at 60 feet is unlimited. Remain at 60 feet a minimum of 12 hours unless overriding circumstances dictate earlier decompression.
3. Patient begins oxygen breathing periods at 60 feet. Tender need breathe only chamber atmosphere throughout. If oxygen breathing is interrupted, no lengthening of the table is required.
4. Minimum chamber O_2 concentration is 19 percent. Maximum CO_2 concentration is 5 millibar. Maximum chamber internal temperature is 85°F (2 9 °C)
5. Decompression starts with a 2-foot upward excursion from 60 to 58 feet. Decompress with stops every 2 feet for times shown in profile below. Ascent time between stops is approximately 30 seconds. Stop time begins with ascent from deeper to next shallower step. Stop at 4 feet for 4 hours and then ascend to the surface at 1 ft/min.
6. Ensure chamber life-support requirements can be met before committing to a Treatment Table 7.
7. A Diving Medical Officer should be consulted before committing to this treatment table.

Treatment Table 7 Depth/Time Profile



Time at Depth (hours)

Description

Treatment Table 7 is an extension at 60 feet of Treatment Table 6, 6A, or 4 (or any other nonstandard treatment table). This means that considerable treatment has already been administered.

- Treatment Table 7 is considered a heroic measure for treating non-responding severe gas embolism or life-threatening decompression sickness and is not designed to treat all residual symptoms that do not improve at 60 feet and should never be used to treat residual pain.
- Treatment Table 7 should be used only when loss of life may result if the currently prescribed decompression from 60 feet is undertaken.
- Committing a patient to a Treatment Table 7 involves isolating the patient and having to minister to his medical needs in the recompression chamber for 48 hours or longer. Experienced diving medical personnel shall be on scene.

- A Diving Medical Specialist (doctor) should be consulted before shifting to a Treatment Table 7 and careful consideration shall be given to life support capability of the recompression facility.
- When using Treatment Table 7, a minimum of 12 hours should be spent at 60 feet, including time spent at 60 feet from Treatment Table 4, 6, or 6A.
- Severe Type II decompression sickness and/or arterial gas embolism cases may continue to deteriorate significantly over the first several hours. This should not be cause for premature changes in depth.
- Do not begin decompression from 60 feet for at least 12 hours.
- At completion of the 12-hour stay, the decision must be made whether to decompress or spend additional time at 60 feet. If no improvement was noted during the first 12 hours, benefit from additional time at 60 feet is unlikely and decompression should be started. If the patient is improving but significant residual symptoms remain (e.g., limb paralysis, abnormal or absent respiration), additional time at 60 feet may be warranted.
- While the actual time that can be spent at 60 feet is unlimited, the actual additional amount of time beyond 12 hours that should be spent can only be determined by a Diving Medical Officer (in consultation with on-site supervisory personnel), based on the patient's response to therapy and operational factors.

- When the patient has progressed to the point of consciousness, can breathe independently, and can move all extremities, decompression can be started and maintained as long as improvement continues.
- Solid evidence of continued benefit should be established for stays longer than 18 hours at 60 feet. Regardless of the duration at the recompression deeper than 60 feet, at least 12 hours must be spent at 60 feet and then Treatment Table 7 followed to the surface.
- Additional recompression below 60 feet in these cases should not be undertaken unless adequate life support capability is available.

- Decompression:

Decompression on Treatment Table 7 is begun with an upward excursion at time zero from 60 to 58 feet. Subsequent 2-foot upward excursions are made at time intervals listed as appropriate to the rate of decompression:

Depth	Ascent rate	Time interval
58 - 40 feet	3 ft/hr	40 min
40 - 20 feet	2 ft/hr	60 min
20 - 4 feet	1 ft/hr	120 min

The travel time between stops is considered as part of the time interval for the next shallower stop. The time intervals shown above begin when ascent to the next shallower stop has begun.

- Tenders:

When using Treatment Table 7, tenders breathe chamber atmosphere throughout treatment and decompression.

- Preventing Inadvertent Early Surfacing:

Upon arrival at 4 feet, decompression should be stopped for 4 hours. At the end of 4 hours, decompress to the surface at 1 foot per minute. This procedure prevents inadvertent early surfacing.

- Oxygen Breathing:

On a Treatment Table 7, patients should begin oxygen breathing periods as soon as possible at 60 feet.

- Oxygen breathing periods of 25 minutes on 100 percent oxygen, followed by 5 minutes breathing chamber atmosphere, should be used.
- Normally, four oxygen breathing periods are alternated with 2 hours of continuous air breathing.
- In conscious patients, this cycle should be continued until a minimum of eight oxygen breathing periods have been administered (previous 100 percent oxygen breathing periods may be counted against these eight periods). Beyond that, oxygen breathing periods should be continued as recommended by the Diving Medical Specialist (doctor), as long as improvement is noted and the oxygen is tolerated by the patient.
- If oxygen breathing causes significant pain on inspiration, it should be discontinued unless it is felt that significant benefit from oxygen breathing is being obtained.
- In unconscious patients, oxygen breathing should be stopped after a maximum of 24 oxygen breathing periods have been administered. The actual number and length of oxygen breathing periods should be adjusted by the Diving Medical Specialist to suit the individual patient's clinical condition and response to pulmonary oxygen toxicity.

- Sleeping, Resting, and Eating:

At least two tenders should be available when using Treatment Table 7, and three may be necessary for severely ill patients.

- Not all tenders are required to be in the chamber, and they may be locked in and out as required following appropriate decompression tables.
- The patient may sleep anytime except when breathing oxygen deeper than 30 feet. While asleep, the patient's pulse, respiration, and blood pressure should be monitored and recorded at intervals appropriate to the patient's condition. Food may be taken at any time and fluid intake should be maintained.

- Ancillary Care:

Patients on Treatment Table 7 requiring intravenous and/or drug. Therapy should have these administered in accordance with the recommendation of the Diving Medical Specialist

- Life Support:

Before committing to a Treatment Table 7, the life-support considerations must be addressed.
Do not commit to a Treatment Table 7 if the internal chamber temperature cannot be maintained at 85°F (29°C) or less.

Treatment Table 8

1. Enter the table at the depth which is exactly equal to or next greater than the deepest depth attained in the recompression. The descent rate is as fast as tolerable.
2. The maximum time that can be spent at the deepest depth is shown in the second column. The maximum time for 225 fsw is 30 minutes; for 165 fsw, 3 hours. For an asymptomatic diver, the maximum time at depth is 30 minutes for depths exceeding 165 fsw and 2 hours for depths equal to or shallower than 165 fsw.
3. Decompression is begun with a 2-fsw reduction in pressure if the depth is an even number. Decompression is begun with a 3-fsw reduction in pressure if the depth is an odd number. Subsequent stops are carried out every 2 fsw. Stop times are given in column three. The stop time begins when leaving the previous depth. Ascend to the next stop in approximately 30 seconds.
4. Stop times apply to all stops within the band up to the next quoted depth. For example, for ascent from 165 fsw, stops for 12 minutes are made at 162 fsw and at every two-foot interval to 140 fsw. At 140 fsw, the stop time becomes 15 minutes. When traveling from 225 fsw, the 166-foot stop is 5 minutes; the 164-foot stop is 12 minutes. Once begun, decompression is continuous. For example, when decompressing from 225 feet, ascent is not halted at 165 fsw for 3 hours. However, ascent may be halted at 60 fsw and shallower for any desired period of time.
5. While deeper than 165 fsw, a helium-oxygen mixture with 16-36 percent oxygen may be breathed by mask to reduce narcosis. A 64/36 helium-oxygen mixture is the preferred treatment gas. At 165 fsw and shallower, a HeO_2 or N_2O_2 mix with a ppO_2 not to exceed 3.0 ata may be given to the diver as a treatment gas. At 60 fsw and shallower, pure oxygen may be given to the divers as a treatment gas. For all treatment gases (HeO_2, N_2O_2, and O_2), a schedule of 25 minutes on gas and 5 minutes on chamber air should be followed for a total of four cycles. Additional oxygen may be given at 60 fsw after a 2-hour interval of chamber air. See Treatment Table 7 for guidance. If high O_2 breathing is interrupted, no lengthening of the table is required.
6. To avoid loss of the chamber seal, ascent may be halted at 4 fsw and the total remaining stop time of 240 minutes taken at this depth. Ascend directly to the surface upon completion of the required time.
7. Total ascent time from 225 fsw is 56 hours, 29 minutes. For a 165-fsw recompression, total ascent time is 53 hours, 52 minutes, and for a 60-fsw recompression, 36 hours, 0 minutes.

Depth (fsw)	Max Time at Initial Treatment Depth (hours)	2-fsw Stop Times (minutes)
225	0.5	5
165	3	12
140	5	15
120	8	20
100	11	25
80	15	30
60	Unlimited	40
40	Unlimited	60
20	Unlimited	120

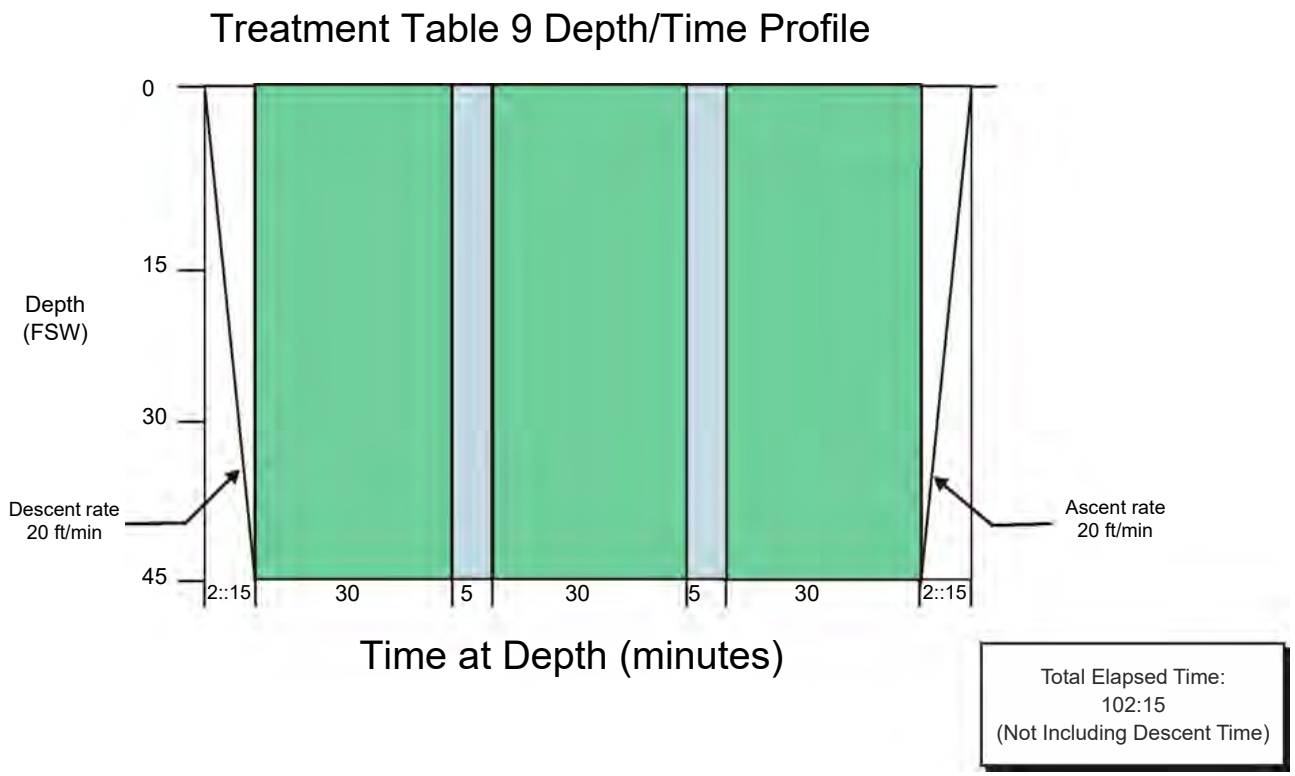
Description

Treatment Table 8 is an adaptation of Royal Navy Treatment Table 65 mainly for treating deep uncontrolled ascents when more than 60 minutes of decompression have been missed.

- Compress symptomatic patient to depth of relief not to exceed 225 fsw.
- Initiate Treatment Table 8 from depth of relief.
- The schedule for Treatment Table 8 from 60 fsw is the same as Treatment Table 7.
- The guidelines for sleeping and eating are the same as Treatment Table 7.

Treatment Table 9

1. Descent rate - 20 ft/min.
2. Ascent rate - 20 ft/min. Rate may be slowed to 1 ft/min depending upon the patient's medical condition.
3. Time at 45 feet begins on arrival at 45 feet.
4. If oxygen breathing must be interrupted because of CNS Oxygen Toxicity, oxygen breathing may be restarted 15 minutes after all symptoms have subsided. Resume schedule at point of interruption.
5. Tender breathes 100 percent O_2 during last 15 minutes at 45 feet and during ascent to the surface regardless of ascent rate used.
6. Patient may breathe air or oxygen during ascent.
7. If patient cannot tolerate oxygen at 45 feet, this table can be modified to allow a treatment depth of 30 feet. The oxygen breathing time can be extended to a maximum of 3 to 4 hours.

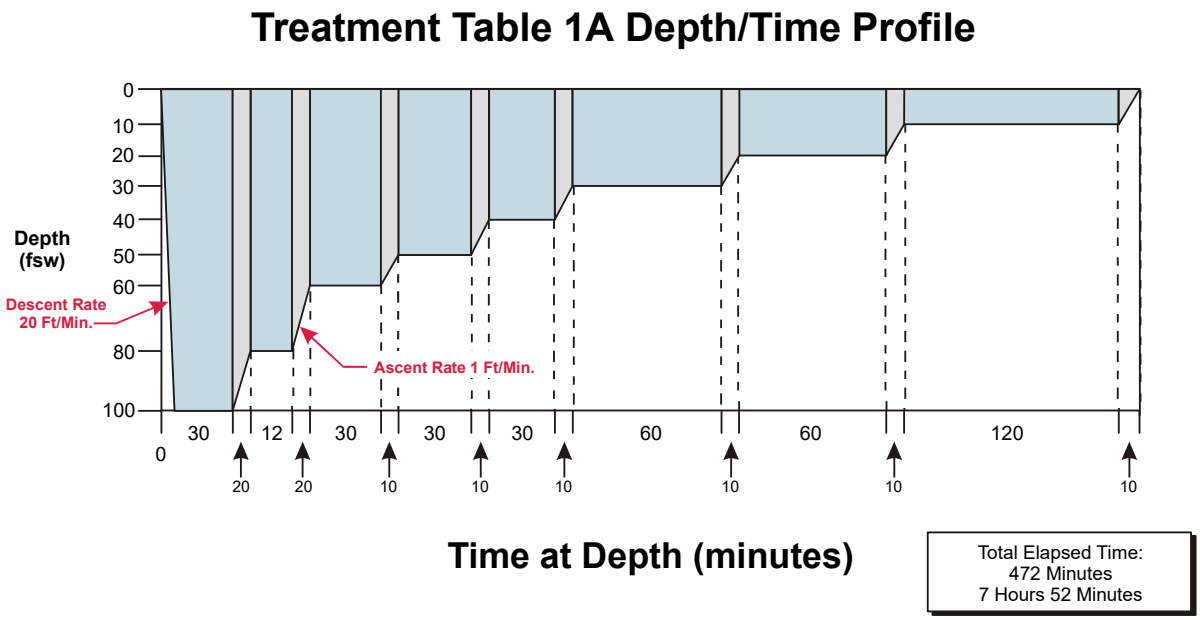


Description

- Treatment Table 9 is a hyperbaric oxygen treatment table providing 90 minutes of oxygen breathing at 45 feet.
- This table is used only on the recommendation of a Diving Medical Specialist cognizant of the patient's medical condition. - Treatment Table 9 is used for the following:
  - Residual symptoms remaining after initial treatment of AGE/DCS
  - Selected cases of carbon monoxide or cyanide poisoning
  - Smoke inhalation
- This table may also be recommended by the cognizant Diving Medical Specialist when initially treating a severely injured patient whose medical condition precludes long absences from definitive medical care.

Air Treatment Table 1A

1. Descent rate - 20 ft/min.
2. Ascent rate - 1 ft/min.
3. Time at 100 feet includes time from the surface.



Description

Air treatment Table 1A is used for treatment of pain only when oxygen cannot be used and the pain is relieved at a depth less than 66 ft

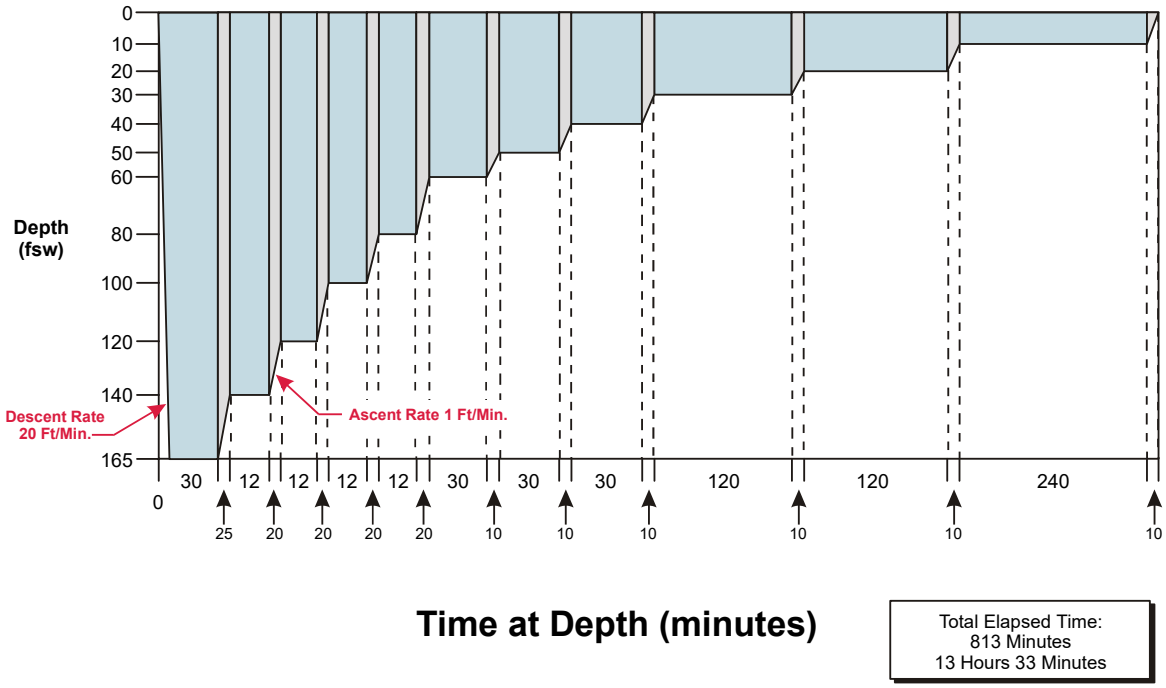
Switching to an Air Treatment Table is to be done only if O_2 breathing cannot be restored in 2 hours.

- The rate of ascent must not exceed 1 fpm between stops.
- If the symptoms worsen and an increase in treatment depth deeper than 60 feet is needed, use Treatment Table 4.

Air Treatment Table 2A

1. Descent rate - 20 ft/min.
2. Ascent rate - 1 ft/min.
3. Time at 165 feet includes time from the surface.

Treatment Table 2A Depth/Time Profile



Time at Depth (minutes)

Total Elapsed Time:
813 Minutes
13 Hours 33 Minutes

Description

Air treatment Table 2A is used for treatment of pain only when oxygen cannot be used and the pain is relieved at a depth greater than 66 ft

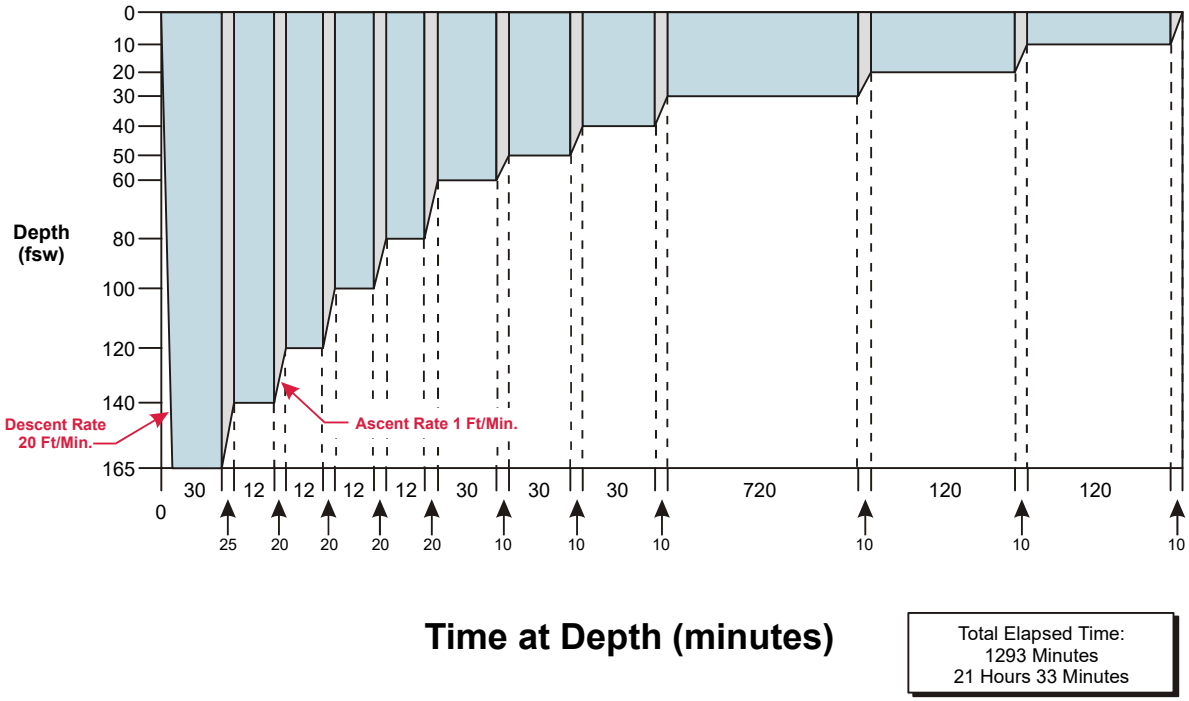
Switching to an Air Treatment Table is to be done only if O_2 breathing cannot be restored in 2 hours.

- The rate of ascent must not exceed 1 fpm between stops.
- If the symptoms worsen and an increase in treatment depth deeper than 60 feet is needed, use Treatment Table 4.

Air Treatment Table 3

1. Descent rate - 20 ft/min.
2. Ascent rate - 1 ft/min.
3. Time at 165 feet-includes time from the surface.

Treatment Table 3 Depth/Time Profile



Time at Depth (minutes)

Total Elapsed Time:
1293 Minutes
21 Hours 33 Minutes

Description

Air treatment Table 3 is used for treatment of serious symptoms when oxygen cannot be used and the symptoms are relieved within 30 minutes at 165 ft

Switching to an Air Treatment Table is to be done only if O_2 breathing cannot be restored in 2 hours.

- The rate of ascent must not exceed 1 fpm between stops.
- If the symptoms worsen and an increase in treatment depth deeper than 60 feet is needed, use Treatment Table 4.

3 - Life support personnel

1 - Key people to be contacted As Soon As Possible

Diving Medical Specialist appointed by the company:

- The Diving Medical Specialist (Doctor) appointed by the company shall be consulted as early as possible in all recompression treatments. He will confirm the treatment started or give guidances to modify it. Also, his advice is necessary before committing the patient to a Treatment Table 4, 7, or 8.
- The Diving Medical Specialist may be on site or not. In case the Diving Medical Specialist is not present on site, reports of the statement of the casualty and his response to the treatments shall be sent to him regularly. Also, a record of the dive shall be attached to this report to give him a full picture of what happened on site.
- In all cases the 'Diving Medical Specialist' shall be consulted prior to releasing the patient from treatment.
- The list of the hyperbaric hospitals and doctors appointed by the company is indicated in the "reporting chart" in the chapter E "Medical check list and record"
- The official reporting document DMAC 1 is attached to this document chapter E "Medical check list and records" with the "dive report forms"

Other essential key people:

- The Operation manager, the HSE department of the company , and the client representative shall be informed of the incident as soon as possible. Notice that it is the duty of the 'Operation Manager' to inform the director of the company. This point is very important as these people will help the 'Offshore Construction Manager' or the Diving superintendent/ Diving supervisor in charge of the project to organise the logistical support and the evacuation of the casualty when it will be authorized by the doctor in charge.
- The names and means of contact of the Operation manager and the HSE manager or the person of the department in charge is also indicated in the "reporting chart" in the chapter E "Medical check list and record" with also the coordinates of the director of the company.

2 - Manning requirement:

As indicated in the part B / chapter "Decompression Sickness", the treatment should never be delayed, thus the team must be always ready to recompress the casualty quickly.

Minimum personnel:

- The diving supervisor is in charge of the operations.
- One diver medic must be in chamber to support the casualty. A minimum air diving team conforming to IMCA / OGP guidances is 4 divers + 1 supervisor. Two divers must be diver medics with a valid certificate. (which means 4 diver medics minimum for 24 hrs operations) One diver medic must be free of dive penalty and ready to go in the chamber if requested.
- Because the diving supervisor has to rely on the shore and the client, a minimum of one qualified chamber operator is necessary

Additional personnel:

- If the patient has symptoms of serious decompression sickness or arterial gas embolism, the team will require additional personnel. In the case that this personnel is not on board, the transfer of additional personnel from the shore or another job site must be considered.
- The size of the chamber and its capacity to accept the people must always be considered before starting long tables like tables 4, 7 or 8. In the eventuality that the chamber is sufficiently wide to accept 2 diver medics in addition to the casualty, it can be better to let them in the chamber and act rotationally. If the chamber cannot accept more than 2 people, a rotation of diver medics must be organized taking into account that a diver medic who has been committed in the chamber cannot return in it with diving penalties. Also, it must be considered that if the dive is too long, the diver medic may have to be on a recompression treatment table.

The tending frequency between 2 medical tables indicated by the US Navy is as indicated below:

Table	1A	2A	3	4	5	6	6A	7	8
Surface interval minimum (hrs)	18	18	18	48	18	18	18	48	48

Notice 1: In extreme emergency, the tenders may repeat treatment Tables 5, 6, or 6A within the 18-hour surface interval. The requirements of oxygen breathing are outlined on the table "tender oxygen breathing requirements " in the next point "Chamber control" .

Notice 2: The text in the table 5, 6 and 6A USN requires additive stops if less than 18 hrs of "Hyperbaric Exposure". Thus the minimum interval for the tender must be more than 18 hrs after a normal in water exposure.

4 - Chamber control

Equalizing during descent.

- It is vital to attain treatment depth in a timely manner, especially for a suspected arterial gas embolism patient, but the descent rates may have to be decreased as necessary to allow the patient to equalize.
- Notice: If the patient is unconscious, the eustachian are released and the equilibration is normally automatic.

Oxygen control

- The minimum percentage of O2 in chamber during the treatment is 19%. The maximum percentage of O2 in the chamber during treatment is 23% (IMCA D 022 chapter 11/ point 2.11). The percentage of O2 in the chamber must be indicated by a calibrated oxygen analyser .
- Oxygen leaks on masks are common in the chamber with the result that the % of O2 rises. To recover the correct value of O2 inside the chamber, the tender may be obliged to ventilate the chamber. It can be done automatically though pre-calibrated specific valves on some modern chambers, but for the majority of the chambers in service it has to be done manually by opening the inlet and the outlet valves simultaneously. This exercise requires some practice to be sure that the chamber will stay at the initial depth. That is why only well trained chamber operators must be appointed to the chamber.
- IMCA D 050 Indicates clearly that *"90 m3 (3200 cu ft) of breathing oxygen must be available for emergency treatments procedures".* Which means that normally there should be sufficient O2 for any recompression treatment table. But, unplanned events can happen:

In case of break down of the O2 supply system:

- If the repair can be completed within 15 minutes:
  - Maintain depth until repair is completed.
  - After O_2 is restored, resume treatment at point of interruption.
- If the repair can be completed after 15 minutes but before 2 hours:
  - Maintain depth until repair is completed.
  - After O_2 is restored: If original table was Table 5, 6, or 6A, complete treatment with maximum number of O_2 extensions.
- If O_2 breathing cannot be restored in 2 hours:
  - Switch to the comparable air treatment table at current depth for decompression if 60 fsw or shallower.
  - Rate of ascent must not exceed 1 fpm between stops.
  - If symptoms worsen and an increase in treatment depth deeper than 60 feet is needed, use Treatment Table 4.

In case of acute O2 toxicity *(The full explanations are in the chapter "O2 toxicity" Part B of the manual*):

- For Treatment Tables 5, 6, and 6A:
  1) Remove the mask.
  2) After all symptoms have completely subsided, decompress 10 feet at a rate of 1 fsw/min. For convulsions, begin travel when the patient is fully relaxed and breathing normally.
  3) Resume oxygen breathing at the shallower depth at the point of interruption.
  4) If another oxygen symptom occurs after ascending 10 fsw, contact the hyperbaric physician appointed by the company to have appropriate modifications to the treatment schedule. In the eventuality that the physician cannot be contacted and O2 breathing cannot be restored within 2 hours, the procedure will be to switch to comparable air table at current depth for decompression if 60 fsw or shallower (table 7 to be considered). The rate of ascent must not exceed 1 fpm between stops. If the symptoms worsen and an increase in treatment depth deeper than 60 feet is needed, treatment Table 4 shall be used.
- For Treatment Tables 4, 7,and 8:
  1) Remove the mask.
  2) Consult with the hyperbaric physician appointed by the company before administering further oxygen breathing. No compensatory lengthening of the table is required for interruption in oxygen breathing. The same procedure as above has to be applied in the eventuality the hyperbaric doctor cannot be contacted.

Notes:

Breathing air or chamber atmosphere every 20 minutes (Indicated period) will usually prevent problems of acute O2 poisoning.

Tables 5, 6, 6A have not the potential to reach a Unit Pulmonary Toxicity Dose (UPTD) of 1400. The other tables are closer or slightly above the values, but should be authorized by the diving medical specialist.

It must be remembered also that 10 - 20% of lung damage is considered tolerable by the specialists…
The "air breaks" commonly seen in both decompression and re-compression treatment tables are based on the fact that if the oxygen exposure is periodically interrupted by a short period of time at low oxygen partial pressure, the total exposure time needed to produce a given level of toxicity can be increased significantly. In more simple words, the Air breaks are to be strictly applied…

Carbon dioxide control:

- Chamber carbon dioxide should be monitored with a carbon dioxide analyser. The maximum partial pressure is 5 mb.
- When using air , the carbon dioxide can be eliminated by flushing (same method as for excess of O2) or using the scrubber. In this case, depending on the machine, the cartridges shall be changed when the values of CO2 will start to raise above 5 mb.

  If the chamber is filled on Heliox, due to the cost of the gas, the flushing shall be reduced to a minimum . In this case the scrubber, or better, the reclaim shall be the means of control.
- Displaying of the partial pressure by the analysers:
  Some analysers can display the reading in the unit desired by the chamber operator. Some others are displaying only one unit and it could be in % CO2 and mostly in "Part Per Million" (PPM)
  - To convert the depth in feet to absolute pressure in atmosphere: Depth in ft / 33 + 1
  - To convert the millibar in atmosphere: 1 millibar = 0.001 bar = 0.000987 atmosphere
  - To find the Partial Pressure (PP): PP = abs pressure x %
  - To find the percentage: % = PP x 100 / Abs pressure
  - To find the part per million: PPM = PP x 1000000 / Abs pressure
  - To convert Percentage to Part Per Million (PPM): PPM = % x 10000

The table below gives the values in percentage and PPM of 5 mb at several depth indicated in the treatment tables.

Depth (Ft)	Abs press (Ata)	%	PPM		Depth (ft)	Abs press (ata)	%	PPM
10	1.3	0.379	3787.03		50	2.52	0.196	1961.96
15	1.45	0.339	3392.55		60	2.82	0.175	1750.99
20	1.61	0.307	3072.5		80	3.42	0.144	1441.08
25	1.76	0.281	2807.63		100	4.03	0.122	1224.38
30	1.91	0.258	2584.8		120	4.64	0.106	1064.33
35	2.06	0.239	2394.74		140	5.24	0.094	941.29
40	2.21	0.223	2230.72		165	6	0.082	822.44
45	2.36	0.209	2087.72		225	7.82	0.063	631.17

Temperature control:

The ideal temperature is 24°C and a chamber temperature below 29°C is always desirable, no matter which treatment table is used. When treating victims of AGE or severe neurological DCS, hot environments that elevate body temperature above normal should be avoided, whenever possible. As in DCS, patient temperature should be a routinely monitored vital sign.
Because all the chambers in service for the company are in refrigerated containers, there is generally an ideal temperature inside the chamber. But several events could compromise the ideal temperature of the chamber:

- A too rapid compression, in addition to the discomfort to the occupant, the possible barotrauma it could cause to those who have difficulties to equilibrate their ears, and the narcosis of some occupants if the compression is below 60 ft, will significantly elevate the temperature inside the chamber. In this case a corrective cooling can be accomplished by ventilation . This kind of situation must be avoided by the strict respect of the descent speed.
- In case of break down of the air conditioning system, the temperature inside the container will become quickly unbearable. If the situation happens before or during the dive, the dive must be stopped until the full repair of the air conditioning system , as the chamber must be always in perfect condition. If the situation happens during the treatment (which is the worse case), the team shall use all the means (ventilation, water cooling…) to adjust the temperatures in accordance to the table below:

Temperature inside the chamber	Maximum tolerance time	Permissible treatment table
Over 40°C (104°F)	Intolerable	No treatment
34 to 40°C (95 to 104°F)	2 hours	Tables 5 & 9
29 to 34°C (85 to 94°F)	6 hours	Tables 5, 6, 6A, 1A, 9
Under 29°C (85°F)	Unlimited	All treatments

Additional oxygen breathing requirements for tables 5, 6, 6A:

The tables 5, 6, and 6A propose possible extensions which can be applied to reinforce the treatments . The application of these extensions can significantly change the dive profile of the tender. To protect him from DCS, it is important to make sure of the Oxygen breathing he will need. The following table summarizes these requirements by taking into account the extension performed and also whether the tender had more than 18 hrs interval from his previous dive or not.

Note 1: All tender O_2 breathing times in table are conducted at 30 fsw . In addition, tenders will breathe O_2 on ascent from 30 fsw to the surface.

Note 2: The minimum interval without penalty is more than 18 hrs after an "hyperbaric exposure".

Note 3: In some instances, tender's oxygen breathing obligation exceeds the table stay time at 30 fsw . Extend the time at 30 fsw to meet these obligations if patient's condition permits . Otherwise, administer O_2 to the tender to the limit allowed by the treatment table and observe the tender on the surface for 1 hour for symptoms of DCS.

Note 4: The tender oxygen breathing requirements are also indicated on the tables.

Table	Extension applied	O2 breathing time if more than 18 hrs interval (No penalty)	Additional O2 breathing time to add if less than 18 hrs interval
Table 5	No extension	Nothing	20 min
Table 5	Extension @ 30 ft	Nothing	20 min
Table 6	No extension	30 min	60 min
Table 6	One extension at 30 or 60 ft	30 min	60 min
Table 6	More than one extension	60 min	60 min
Table 6A	No extension	60 min	60 min
Table 6A	one extension @ 30 or 60 ft	60 min	60 min
Table 6A	More than one extension	90 min	60 min

Additional treatments in chamber:

Warning: Drug therapy shall be administered by a qualified Diver Medic Technician only after consultation with a Diving Medical Specialist (doctor).

- Fluids:

Fluid administration may help by replenishing intravascular volume and reversing hemo-concentration. Thus, blood pressure may be maintained and microcirculatory flow augmented which may accelerate the washout of excess inert gas. Therefore, fluid administration may offer an advantage even in divers who are not dehydrated.
Any non-alcoholic fluid may be given by mouth (avoid large amounts of caffeine)
For unconscious victims or those unable to swallow, Ringer's lactate or normal saline should be used intravenously. A good starting point with IV fluids is 200-300 cc run in rapidly, then about 100 cc. per hour, adjusted downward when urine output becomes adequate. Whether by mouth or vein, intake should be sufficient to produce a urine output above 3000 cc. per hour (> 3 litres or 5.3 pint).

- Drugs:

No medications are currently recommenced for routine use in diving accidents, but the medic may be ordered to give medications in specific situations.
The following points should be remembered:

- The medic should only administer drugs on the order of a physician, either verbal or written. Verbal orders should be repeated and decimal points clearly stated.
- The medic should be familiar with the major properties of the drugs he has on hand. To guard against errors, he should be able to check the common doses used for average-sized males
- While drugs are usually helpful, they are always potentially harmful The exact benefit of drugs in diving accidents is frequently unclear and occasionally controversial.
- The use of drugs is usually delayed or in complicated cases. The need for drugs can largely be eliminated by prompt recompression, aggressive use of oxygen, generous fluid intake and sufficient time.

- The medic should always remain conscious of the following fundamental information:

- Oxygen is a drug or medication; administered under pressure, it is the drug used to treat diving accidents.
- A treatment table is a one-shot treatment, like one injection of an antibiotic. The severity of the case is estimated and the appropriate table is chosen based on this estimate.
- Since severity and treatment are estimated in advance, it follows that the victim must be observed for the desired response and treatment lengthened if necessary.
- Old cases usually respond to treatment slower than fresh cases. Even the worst symptoms usually disappear quickly when the victim is treated soon after onset.

Abort procedures:

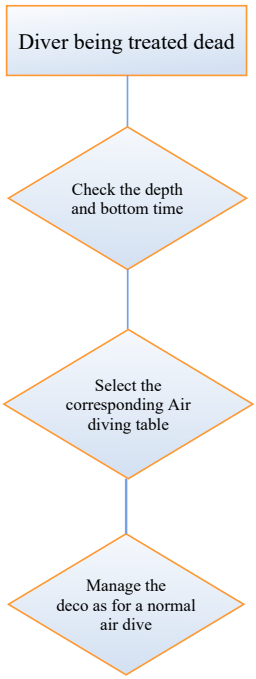
1 - Death During Treatment.

If it appears that the diver being treated has died, the Diving Medical Specialist (Doctor) shall be consulted before the treatment is aborted. Once the decision to abort is made, there are a number of options for decompressing the tenders depending on the depth at which the death occurred and the preceding treatment profile.

- If death occurs following initial recompression to 60, 165, or 225 ft on Treatment Tables 6, 6A, 4 or 8,

  Decompress the tenders on the Air/Oxygen schedule in the Air Decompression Table having a depth exactly equal to or deeper than the maximum depth attained during the treatment and a bottom time equal to or longer than the total elapsed time since treatment began.

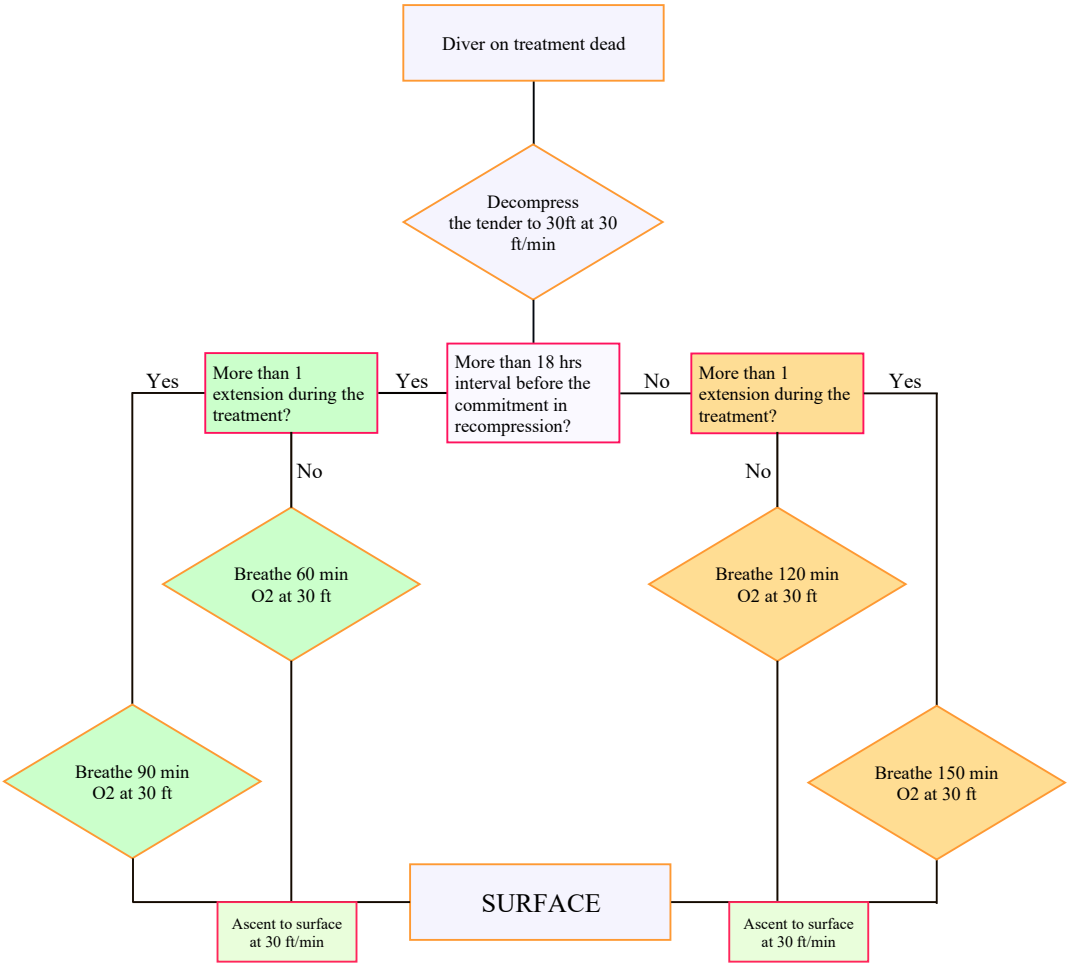
  The Air/Oxygen schedule can be used even if gases other than air (nitrogen-oxygen or helium-oxygen mixtures) were breathed at depth.



- If death occurs after leaving the initial treatment depth on Treatment Tables 6:

  Decompress the tenders at 30 fsw/min to 30 fsw and have them breathe oxygen at 30 fsw for the times indicated for the “tender additive oxygen requirements” previous page, and as explained visually in the chart below.

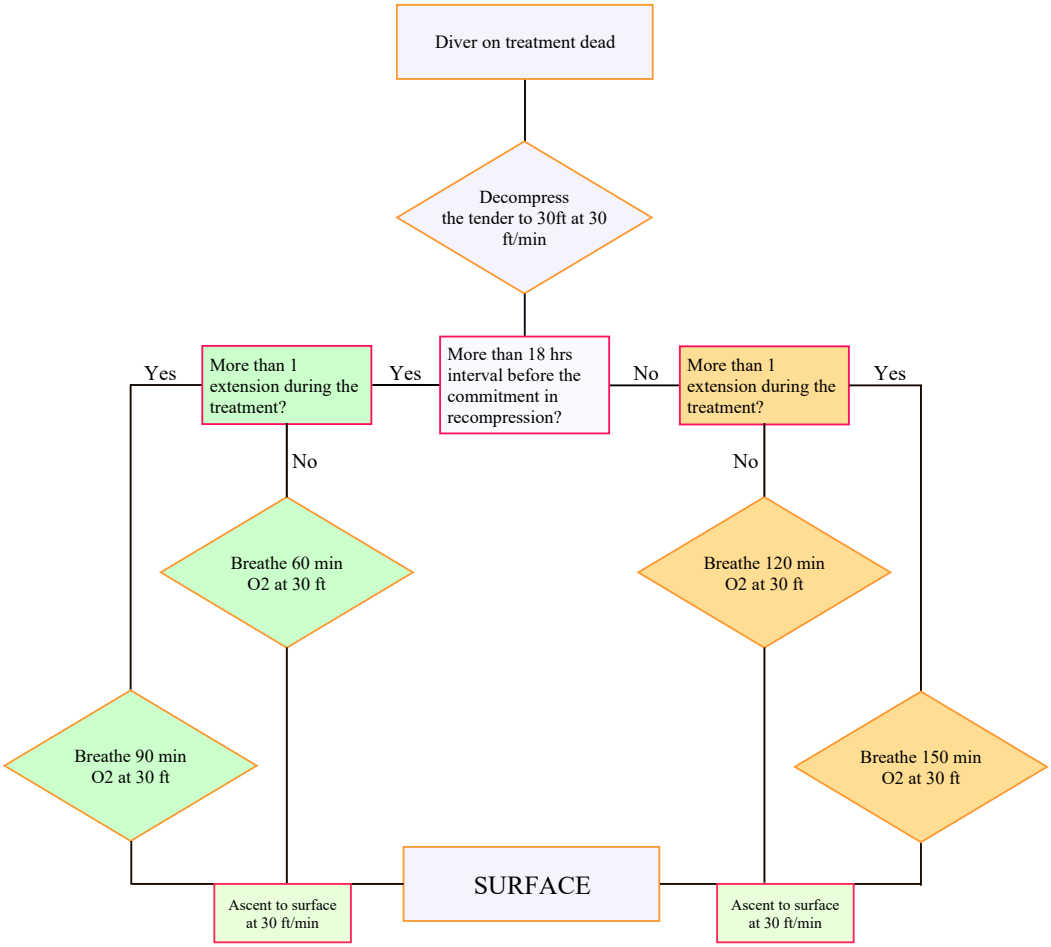
  Following completion of the oxygen breathing time at 30 fsw, decompress the tenders on oxygen from 30 fsw to the surface at 1 fsw/min.



- If death occurs after leaving the initial treatment depth on Treatment Tables 6A

Decompress the tenders at 30 fsw/min to 30 fsw and have them breathe oxygen at 30 fsw for the times indicated for the "tender additive Oxygen requirements" (See in "chamber control") and as explained visually in the chart below.

Following completion of the oxygen breathing time at 30 fsw, decompress the tenders on oxygen from 30 fsw to the surface at 1 fsw/min.



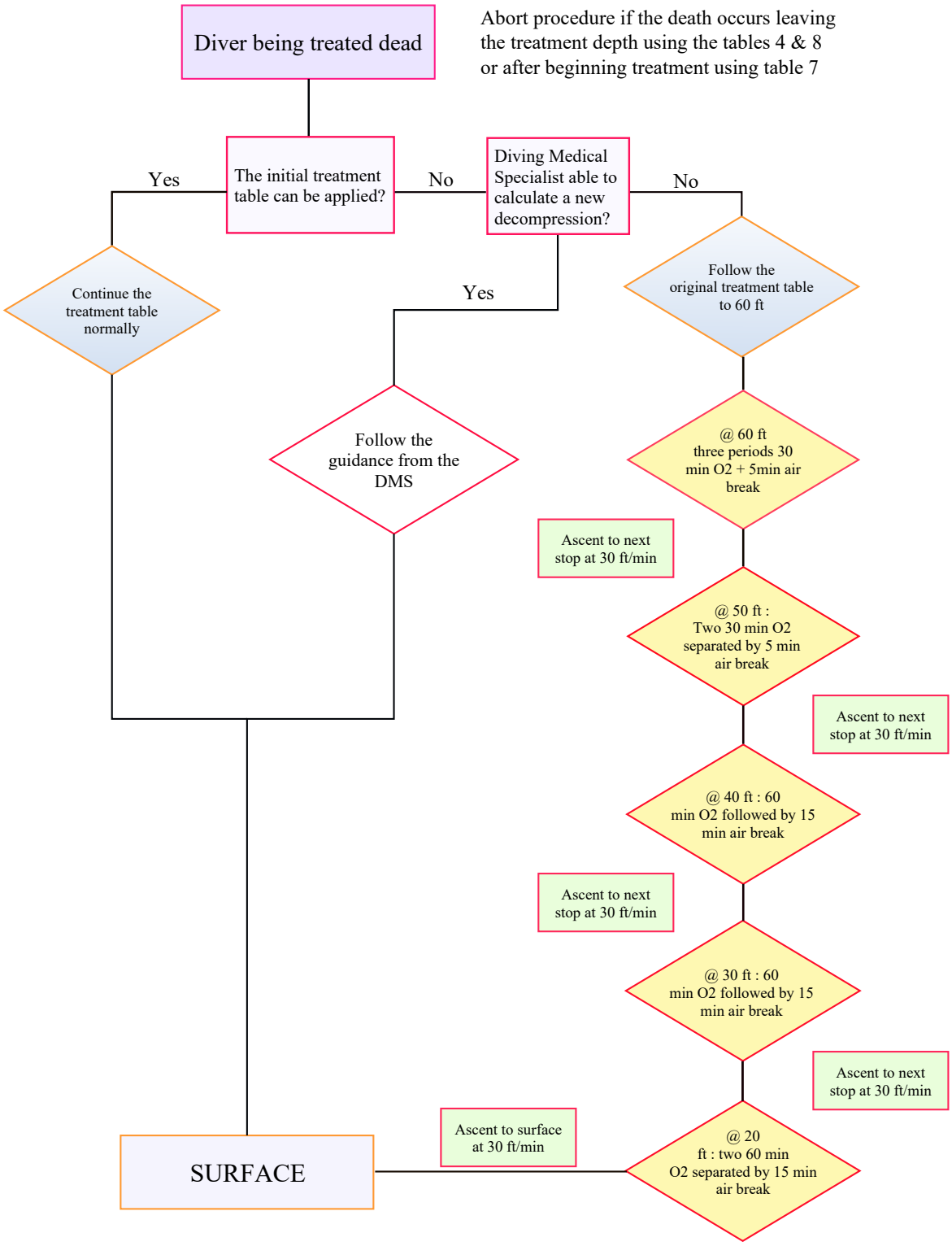
- If death occurs after leaving the initial treatment depth on Treatment Tables 4 or 8, or after beginning treatment on Treatment Table 7 at 60 fsw:

Have the tenders decompress by continuing on the treatment table as written, or consult the Diving Medical Specialist for a decompression schedule customized for the situation at hand.

If neither option is possible:

- Follow the original treatment table to 60 fsw.
- At 60 fsw, have the tenders breathe oxygen for 90 min in three 30-min periods separated by a 5-min air break.
- Ascend to the next stop at 30 fsw/ min.
- Continue decompression at 50 fsw by breathing oxygen for 60 min , breathe oxygen in two 30-min periods separated by a 5-min air break.
- Ascend to the next stop at 30 fsw/ min.
- Continue decompression at 40 fsw by breathing oxygen for 60 min. Breathe oxygen for the full 60-min period followed by a 15-min air break
- Ascend to the next stop at 30 fsw/ min.
- Continue decompression at 30 fsw by breathing oxygen for 60 min. Breathe oxygen for the full 60-min period followed by a 15-min air break.
- Ascend to 20 fsw at 30 fsw/min.
- At 20 fsw breathe oxygen for 120 min. Divide the oxygen time at 20 fsw into two 60-min periods separated by a 15 min air break. When oxygen breathing time is complete at 20 fsw, ascend to the surface at 30 fsw/min.
- Upon surfacing, observe the tenders carefully for the occurrence of decompression sickness.

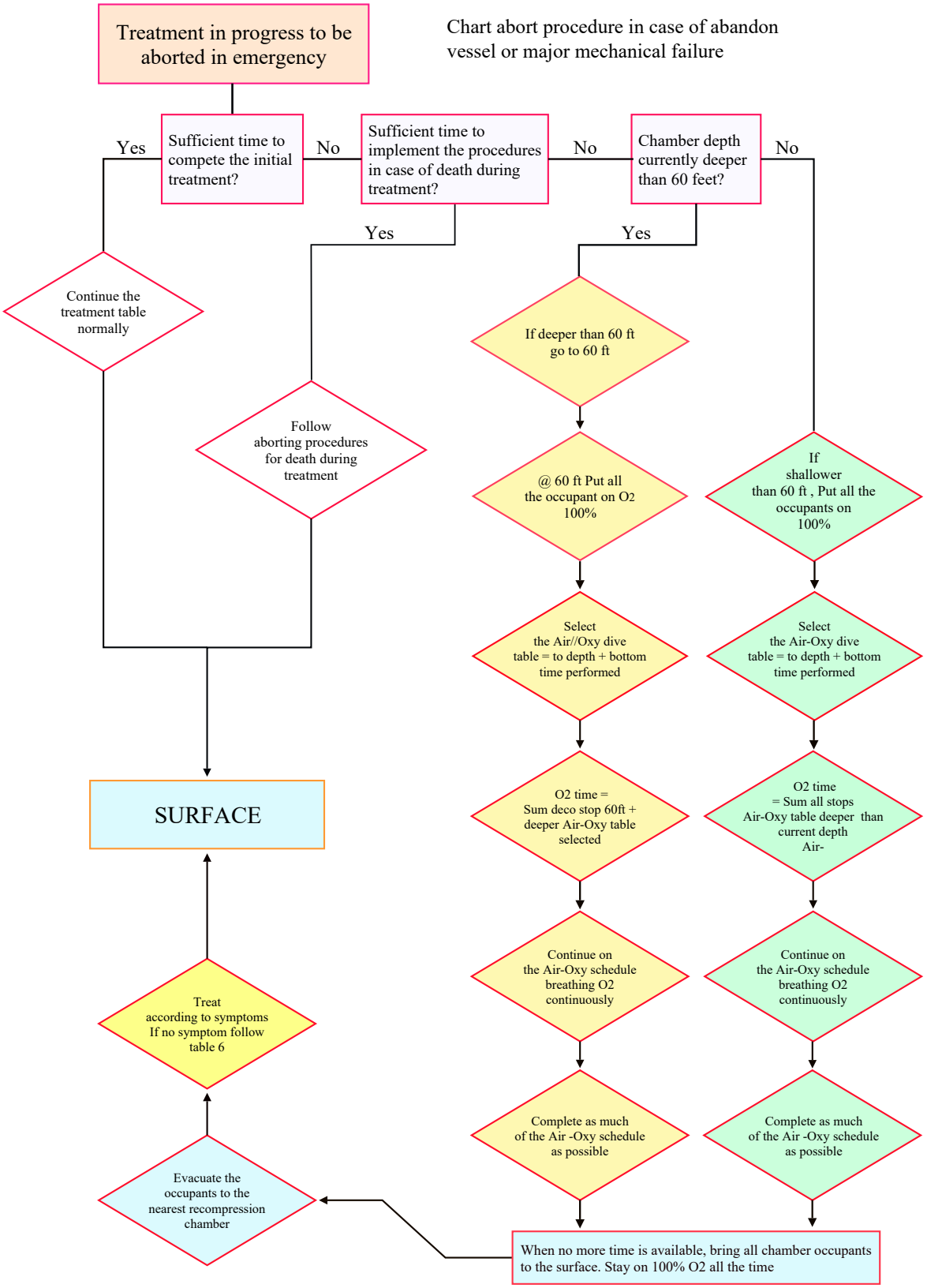
See the chat on the next page.



2 - Disasters or mechanical failures:

- Disasters or mechanical failures may force the treatment to be aborted. For instance, the ship where the chamber is located may be in imminent danger of sinking, or a fire or explosion may have severely damaged the chamber system to such an extent that completing the treatment is impossible.
- In these cases, the abort procedure described in case of death could be used for all chamber occupants (including the stricken diver) if time is available. If time is not available, the following may be done:
  - If deeper than 60 feet, go immediately to 60 feet.
  - Once the chamber is 60 feet or shallower, put all chamber occupants on continuous 100 percent oxygen.
  - Select the Air/Oxygen schedule in the Air Decompression Table corresponding to the maximum depth attained during treatment and the total elapsed time since treatment began.
  - If at 60 fsw, breathe oxygen for a period of time equal to the sum of all the decompression stops 60 fsw and deeper in the Air/Oxygen schedule, then continue decompression on the Air/Oxygen schedule, breathing oxygen continuously.

- If shallower than 60 fsw, breathe oxygen for a period of time equal to the sum of all the decompression stops deeper than the divers current depth, then continue decompression on the Air/Oxygen schedule, breathing oxygen continuously.
- Complete as much of the Air/Oxygen schedule as possible.
- When no more time is available, bring all chamber occupants to the surface (try not to exceed 10 feet per minute) and keep them on 100 percent oxygen during evacuation, if possible.
- Immediately evacuate all chamber occupants to the nearest recompression facility and treat according to symptoms. If no symptoms occurred after the treatment was aborted, follow Treatment Table 6.



5 - Post treatment procedures

Chamber proximity & post-Treatment Observation Period:

- Table 5:
  The patients treated on a Treatment Table 5 should remain at the recompression chamber facility for 2 hours.
- Table 6:
  The patients who have been treated for Type II decompression sickness or who required a Treatment Table 6 for Type I symptoms and have had complete relief should remain at the recompression chamber facility for 6 hours.
- Tables 6, 6A, 4, 7, 8 or 9:
  The patients treated on Treatment Tables 6, 6A, 4, 7, 8 or 9 are likely to require a period of hospitalisation, and the Diving Medical Specialist (doctor) appointed by the company will need to determine a post-treatment observation period and location appropriate to their response to recompression treatment.
  These times may be shortened upon the recommendation of a Diving Medical Specialist, provided the patient will be with personnel who are experienced at recognizing recurrence of symptoms and can return to the recompression facility within 15 minutes.
- Patients observation time near recompression facility:
  All patients should remain within 15 minutes travel time of a recompression facility for 24 hours and should be accompanied throughout that period. No patient shall be released until authorized by the Diving Medical Specialist.
- Tenders observation time:
  Treatment table profiles place the inside tender(s) at risk for decompression sickness. After completing treatments, inside tenders should remain in the vicinity of the recompression chamber for 1 hour. If they were tending for Treatment Table 4, 7, or 8, inside tenders should also remain within 15 minutes travel time of a recompression facility for 24 hours.

Emergency Evacuation:

- Some patients will require air evacuation to another treatment or medical facility immediately after surfacing from a treatment. They will not meet surface interval requirements as described above. Such evacuation is done only on the recommendation of a Diving Medical Specialist (doctor) in charge. In this case the MEDEVAC is to be organized with medical personnel trained for medical transfer of patients suffering DCS or AGE. In the eventuality that this personnel is missing, diver medics may be used with the approval of the Diving Medical Specialist.
- During this transfer, the patient should breaths 100 percent oxygen. If available, an Emergency Evacuation Hyperbaric Stretcher to maintain the patient at 1 ata may be used.
- If the transfer is done by air, an aircraft pressurized to one ata should be used if possible. If the aircraft is unpressurized, it must fly as low as safely possible and no more than 300 m (1,000 feet).

Assessing Fitness to Return to Diving (DMAC 13)

The following minimum periods are recommended By DMAC before returning to diving after decompression illness.

The period begins after completion of successful treatment (there are no residual manifestations). It is stressed that these recommendations represent minima and longer lay-offs may be necessary in individual cases.

- A Limb pain, cutaneous (*skin rash with severe itching*), lymphatic (*swelling of tissues)* or non-specific (*persistent headache, excessive fatigue, loss of appetite, nausea*) manifestations only
  - With uncomplicated recovery: 24 hour lay-off
  - Where there has been a recurrence or relapse requiring further recompression: A 7 day layoff
- B Neurological or pulmonary manifestations:
  - Altered sensation involving the limbs only: A 7 day layoff
    Return to diving only after review by a diving medical specialist.
  - Other neurological (*including audio-vestibular*) or pulmonary manifestations: A 28 day lay-off *Return to diving only a after review by a diving medical specialist.*
- C After an incident of pulmonary barotrauma resulting in a pneumothorax or mediastinal/subcutaneous emphysema, the diver should be assessed by a diving medicine specialist. Return to diving may be permitted, but not normally until at least 28 days following complete recovery.

In cases where there are significant residual neurological manifestations, even after repeated treatment, the diver should normally be considered unfit to dive. Return to diving should only be permitted if sanctioned by a diving medical specialist.

Residual Symptoms:

- After completion of the initial recompression treatment and after a surface interval sufficient to allow complete medical evaluation, additional recompression treatments may be instituted by the Diving Medical Specialist. These treatment are more often performed in the hyperbaric hospital, but for several reasons, it may happen that the diving medical Specialist request some sessions on board.

- In the case that some complementary treatments are undertaken, the team in charge must be aware that it is common that some residual symptoms may remain unchanged during the first one or two treatments. The delay time between completion of initial treatment and the beginning of follow-up hyperbaric treatments is known to decrease the probability of benefit from additional treatments, but this is not a valid reason to preclude follow-up treatments.
- The follow-up of the victim must be the same as for the initial recompression. Such treatments should be continued until the Diving Medical Specialist judges that there is no further benefit noted.

Post-Treatment Transfer:

- The decision to transfer the patient to the shore after a successful treatment or a treatment requesting further recompression, is only from the Diving Medical Specialist. Without any clear guidance from the Diving Medical Specialist, the patient must stay on the job site.
- US Navy states:
  "If ambulatory patients are sent home, they should always be accompanied by someone familiar with their condition who can return them to the recompression facility should the need arise."
- In the case the patient has to transfer by flight, DMAC 7 indicates:
  "Flying in the presence of even minor symptoms or residual of decompression illness carries a considerable risk of provoking serious neurological illness."

2 - Following therapy for DCS, advice should be sought from a diving medical specialist. The times given below are minimum times		Minimum time from completion of therapy	
		2000' (600 m)	All other flights
2.1	Immediate and complete resolution of symptoms on first recompression	24 hours	48 hours
2.2	Cases without immediate response or with residual symptoms must be decided on an individual basis by a diving medical specialist. Generally wait as long as possible.	Consult a diving medical specialist	

- The patients with residual symptoms should be MEDEVAC, also the following recommendations from DMAC 7 must be considered:
  "Consideration should be given for 100% oxygen during flight. Following landing, the diver should be assessed by a competent diving doctor."
  Decompression sickness during the flight:
  "Where the diver's symptoms consist only of pain in a limb, he should be treated with analgesics, oral fluids, oxygen if available, and the plane can continue to its destination without diversion or adjustment in altitude."
  "When the diver has any other symptoms, immediate advice should be sought from a diving medical specialist. It may be necessary to reduce the cabin altitude or divert to the nearest airport. In the meantime, the patient should be given oxygen and oral fluids if available."

Return the chamber ready for use:

When the treatment has been completed , the chamber must be cleaned and the reserve of gas restored before any return to normal diving operations

The reserve of gas should be verified and restored if necessary. They must at least conform with the guidelines of IMCA D 050 reinforced with the recommendations CCO Ltd diving study #7, "History and evaluation of IMCA D 050 rev. 1 - Minimum quantities of gas required offshore", and discussed point 3.3 "maintaining gas reserves" of book #3.

Gas purpose	*Classification IMCA D 050*	*Minimum requirement IMCA*	*Comments / Additional precautions CCO Ltd*
Operational in water gas + in-water decompression gas	Consumable	Sufficient gas should be provided for the bottom time and decompression, based on a breathing rate of 35 l/min at work & 25 I/min at rest.	Operational in-water gas and in-water decompression are grouped. There should be sufficient gas for two dives instead of one (working time + decompression).
Diver personal gas reserve (Bailout)	Reserve	10 m/min of umbilical deployed from the surface (basket) or the wet bell at emergency breathing rate.	Breathing rates from UK HSE report RR 1073 (50 to 75 l/min) should be promoted to the detriment of the IMCA rate of 40 l/min
Diver rescue air or nitrox	Reserve	2 dives of 30 min bottom time to the maximum intended diving depth at emergency breathing rate.	Emergency breathing rate of 62.5 litres /min instead of 40 litres (see above)
Wet bell / basket gas reserve	Reserve	It must be sufficient to recover the divers safely from the longest and deepest planned dive at emergency breathing rate.	Emergency breathing rate of 62.5 litres /min instead of 40 litres (see above)

Gas purpose	*Classification IMCA D 050*	*Minimum requirement IMCA*	*Comments / Additional precautions CCO Ltd*
Surface decompression gasses	Consumable	Sufficient gasses to compress both chamber's locks to the max. surface deco depth + three (3) surface decompression cycles per chamber	The surface decompression cycles include the full compression and decompression of the chamber + the gas used for flushing. Note that 20 - 25 l/min is the breathing rate.
Chamber scrubber		Not indicated in the guidance IMCA. Consumption: 0.25 kg/diver/hour	Soda lime and Purafil for 3 surface deco. dives + the longer therapeutic treatment planned + the same quantity as a reserve.
Therapeutic treatment gasses	Reserve	Sufficient gas to pressurize both locks of each DDC to the maximum possible treatment depth + 90 m³ Oxygen	The quantity of gas to pressurize the locks should be doubled Also, plan for sufficient gas for 3 decompression of medics and 3 compressions of the entry lock. If a heliox table such as COMEX 30 is used: add 90 m³ heliox 50/50 and 90 m³ heliox 20/80.
Calibration gasses (Analysers)	Consumable	No calibration gas required	Sufficient quantities for the calibration processes recommended by the manufacturer for the entire duration of the project + the same quantity as reserve

The chamber must also be restituted to its original condition before being back to service:

- The chamber must be washed and disinfected using products designed for this purpose and not containing agents able to contaminate the breathing atmosphere.
- If used the cartridge of the scrubber must be changed, even if not used for long time. Also, there should be at least 1 spare unit in a sealed should stored in the chamber.
- If the O2 analysers use fuel cells, they must be replaced after a long treatment. After a short treatment, the analyzer must be re-calibrated, and the duration the fuel cell was in service must be noted and compared with the maximum use time indicated by the manufacturer. Note that the fuel cell installed must be able to work for lengthy treatment. Also, remember that fuel cells must be changed every six months even though the chamber has not been activated. There must always be spare fuel cells in the chamber room in case the replacement has to be done before the expected time. Having a replacement analyzer is also a suitable precaution.
- The checklist, the recompression tables, procedures of control, dive record sheet, and reporting forms must be in the chamber control ready for use. Note that the checklist must be designed for the model of the chamber in service, with all valves and electrical devices indicated and located.
- The medical kit (DMAC 15.3 or similar) must be checked and ready for use. Note that the medications used, must be renewed: The chamber cannot be declared ready for used as long as the medical kit is not ready.
- The medical reports DMAC 1, and company forms must be in the chamber control.
- The emergency chart with the list of the diving Medical Specialist (doctors) appointed by the company must be in the dive control and the chamber control if separated.
- There must be a satellite phone in the chamber control (direct link to the diving medical specialist). Regarding this point, many boats are equipped with an internet connection allowing the team to connect through wifi. For this reason, other systems of contact than satellite phones, such as messaging systems using cell phones, can be used in complement. When the operations are performed in an area where cell phones allow connection to the phone number of the diving medical specialist without being cut, it is admitted that these means of contact can replace satellite phones (this case notably applies to operations in harbours and inland).
- The chamber's checklist and function tests taking into account the elements described above, must be performed. At the completion of the checklist, the chamber is declared ready for use, and the diving operations can resume again if only one chamber is available.

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Part E - Therapeutic tables COMEX

1 - Presentation

The therapeutic procedures COMEX are efficient alternatives to US Navy tables. They are designed for air, and heliox surface supplied diving and air and heliox saturation diving. Some tables are initially designed for the therapeutic use of heliox, and the set proposed is composed as follows:

- ***Cx 12*** can be used for treatment of "type 1" accident after an air, nitrox or heliox dive.
- ***Cx 18*** can be used for treatment of "type 1" accident after no relief or worsening of symptoms in a table Cx 12.
- ***Cx 30*** can be used for treatment of "type 2" accident after an air, nitrox or heliox dive.
- ***Cx 30 saturation*** can be used for treatment of "type 2" accident after an air, nitrox or heliox dive. after no relief or worsening of symptoms in a Table Cx 30.
- ***Cx B*** can be used for treatment of "type 1" accident during normal decompression or after a blow-up from deeper than 9 m in a heliox bounce dive or air/heliox saturation.
- ***Cx SB*** can be used in case of failure of a table "Cx B" during normal decompression or after a blow-up from deeper than 9 m in a heliox dive or air/heliox saturation.
- ***Cx N*** can be used for treatment of "type 2" accident during normal decompression or after a blow-up from deeper than 9 m in a heliox dive or a air/heliox saturation.

The particularity of the CX30 and CX30 saturation is that these tables use heliox 50/50 as a treatment mix at 30 m. For this reason, Heliox 50/50 must be provided in addition to oxygen as indicated in point 4 of chapter C, "Medical tables US Navy rev. 7"
It is usual and prudent to plan for more quantities of therapeutic gas than the minimum calculated not to be obliged to stop the operations if a medical table is to be implemented. For this reason, most companies calculate a set of therapeutic gasses usable for all operations they usually carry on. These therapeutic sets must remain untouched as long as there is no need for therapeutic treatment. They will usually be reused in the following operations. However, note that these mixes can be recycled when they are no more needed. The table below from the medical manual COMEX gives an example of the minimum quantities of oxygen and heliox that should be kept on-site according to the depth.

Maximum depth	***Oxygen***	***50/50 heliox***	***20/80 heliox***
40 metres	90 m³	90 m³	90 m³
110 metres	90 m³	90 m³	220 m³

Table Cx 12

This table can be used for treatment of "type 1" accident

Procedure:

- The chamber should be pressurized to 12 metres on Heliox 20/80.
- Put patient on Oxygen on BIBS ASAP. The patient breathes 25 min. oxygen and 5 min. Air as per table.
- Note the time and depth of relief (if relevant).

No relief or worsening of symptoms:

- No relief within 15 minutes: Apply Cx 18
- Recurrence between 12 m and surface: Apply a second Cx 12
- Worsening of symptoms: Apply table Cx 30

Depth	Duration	Breathing mix		Elapsed time
		Patient	Attendant	
12 metres	120 minutes	Oxygen 4 BIBS sessions 25 min on + 5 min off	Ambient	02:00
12 - 0 metres	30 minutes	Oxygen 30 min on BIBS	Oxygen 30 min on BIBS	02:30

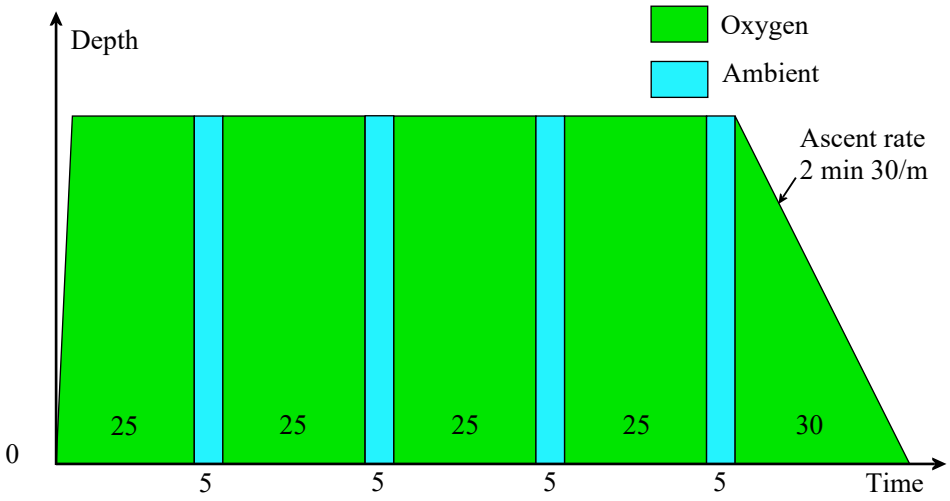


Table Cx 18

This table can be used for treatment of "type 1" accident after no relief or worsening of symptoms in Table Cx 12

Procedure:

- The chamber should be pressurized to 18 metres on Heliox 20/80.
- Put patient on Oxygen on BIBS ASAP. The patient breathes 25 minutes oxygen and 5 minutes air as per table.
- Note the time and depth of relief (if relevant).

No relief or worsening of symptoms:

- No relief within 15 minutes: Apply Cx 30
- Recurrence between 18 m and 12 m: Apply a second Cx 18
- Recurrence between 12 m and surface: Apply table Cx 12
- Worsening of symptoms: Apply table Cx 30

Depth	Duration	Breathing mix		Elapsed time
		Patient	Attendant	
18 metres	90 minutes	Oxygen 3 BIBS sessions 25 min on + 5 min off	Ambient	01:30
18 to 12 metres	30 minutes	Oxygen 1 BIBS session 25 min on + 5 min off	Ambient	02:00
12 metres	150 minutes	Oxygen 3 BIBS sessions 25 min on + 5 min off	90 min ambient. Then, oxygen 2 BIBS sessions 25 min on + 5 min off	04:30
12 m to surface	30 min	Oxygen 30 min on BIBS	Oxygen 30 min on BIBS	05:00

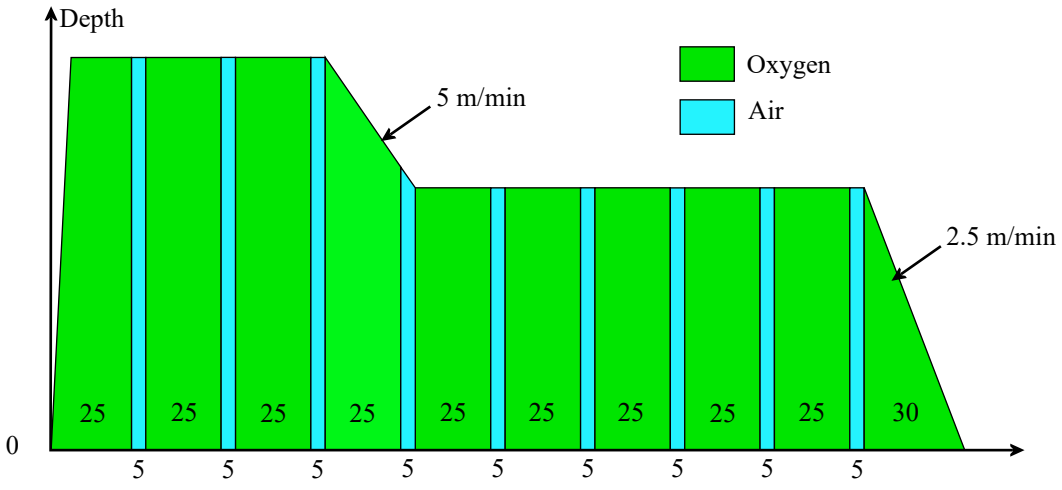


Table Cx 30

This table can be used for treatment of "type 2" accident.

Procedure:

- The chamber should be pressurized to 30 metres on Heliox 20/80.
- Put patient on BIBS ASAP. The patient breathes heliox and oxygen as per table.
- Note the time and depth of relief (if relevant).

No relief or worsening of symptoms:

- No relief or worsening of symptoms: Keep the patient on bibs on heliox for 25 min/ 5 min session during a period not exceeding 3 hours and wait for instructions from the Diving Medical Specialist (DMS). "Cx 30 saturation" should be applied for more than 60 minutes at treatment depth..
- Recurrence: Recompress to 30 m and apply the procedure above

Depth	Duration	Breathing mix		Elapsed time
		Patient	Attendant	
30 metres	60 minutes	Heliox 50/50 60 minutes on BIBS	Ambient	01:00
30 to 24 metres	30 minutes	Heliox 50/50 1 BIBS session 25 min on + 5 min off	Ambient	01:30
24 metres	30 minutes	Heliox 50/50 1 BIBS session 25 min on + 5 min off	Ambient	02:00
24 to 18 metres	30 minutes	Heliox 50/50 1 BIBS session 25 min on + 5 min off	Ambient	02:30
18 metres	60 minutes	Heliox 50/50 2 BIBS sessions 25 min on + 5 min off	Ambient	03:30
18 to 12 metres	30 minutes	Oxygen 1 BIBS session 25 min on + 5 min off	Ambient	04:00
12 metres	180 minutes	Oxygen 6 BIBS sessions 25 min on + 5 min off	Oxygen 6 BIBS sessions 25 min on + 5 min off	07:00
12 m to surface	Min	Oxygen 30 min on BIBS	Oxygen 30 min on BIBS	07:30

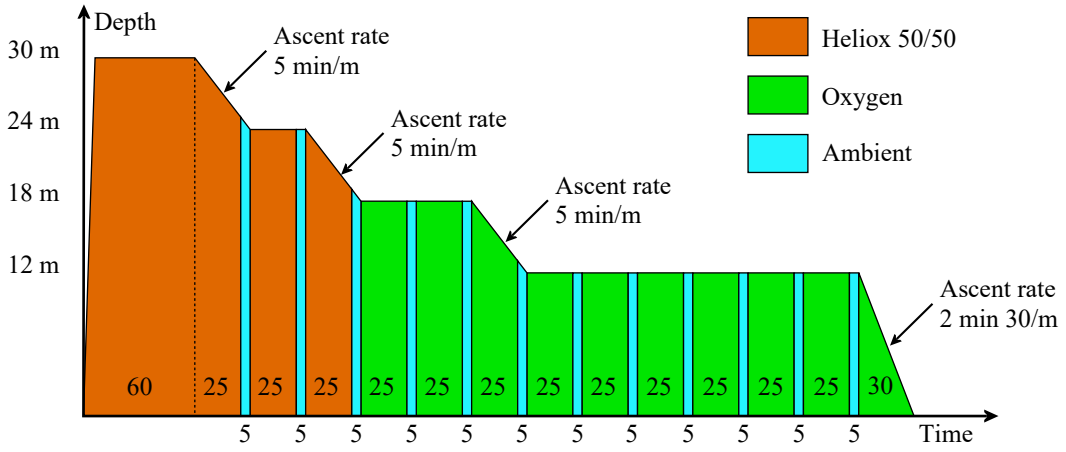


Table Cx 30 saturation

This table can be used for treatment of "type 2" accident after no relief or worsening of symptoms using Table Cx 30.

In the case where a Type II accident does not respond to a standard Cx 30, the diving medical specialist may decide to switch to heliox saturation at 30 metres.

Procedure:

- Saturation treatments are long and the chamber selected should be sufficiently wide to welcome comfortably the patient and at least one diver medic. More possibilities such: additional bunks, toilet, and shower are welcome. The evacuation of rubbish, vomit, fluids and faeces should be organized prior to starting the diving operations.

- The chamber should be pressurized with 20/80 heliox:
  - If the chamber was pressurised on 20/80 heliox, no action is required.
  - If the chamber was initially pressurised on air, change the chamber atmosphere with heliox 20/80.

Treatment at 30 metres:

- Oxygen percentage in the chamber should not be less 20%, and not above 23%.
- The patient should be on BIBS on 50/50 heliox for 25 minutes/5 minutes sessions for a period not exceeding 3 hours.
- At the end of this period, the patient breathes the chamber atmosphere
- The patient should be kept at depth until the symptoms have resolved, and instruction from the Diving Medical Specialist
- Note the time and depth of relief (if relevant).
- After successful treatment, the patient should be held at treatment depth for 6 hours. The decompression can start after green light from the Diving Medical Specialist.

Ascent to surface:

- From 30 to 15 metres, the ascent speed is 60 minutes/metre.
- From 15 metres to surface, the ascent speed is 120 minutes/metre.
- The oxygen percentage must be kept between 20% to 23%.

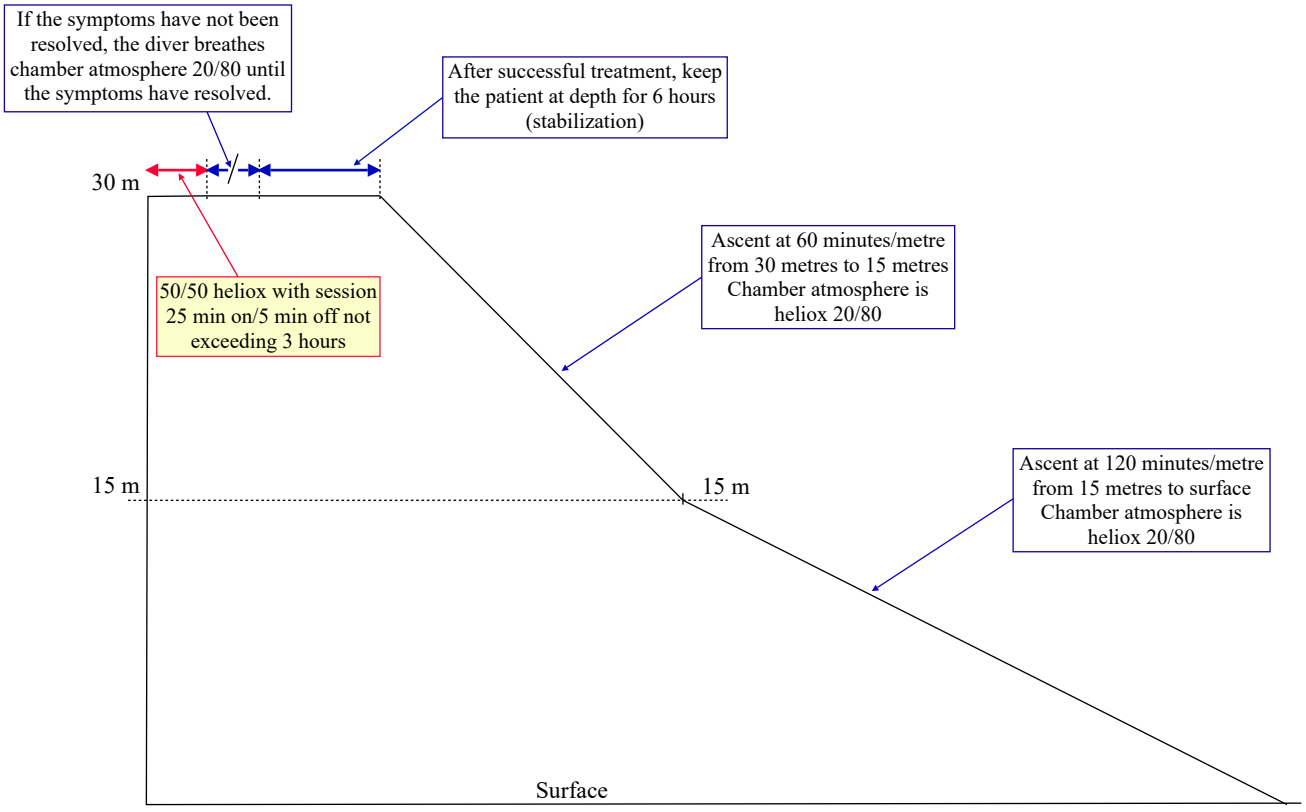


Table Cx B

This table can be used for treatment of "type 1" accident during normal decompression or after a blow-up from deeper than 9 m in an heliox bounce dive or air/heliox saturation.

Procedure:

- Stop the decompression
- Put patient on BIBS. The patient breathes heliox for 60 minutes without interruption as per table.
- Note the time of relief.

No relief or worsening symptoms:

- No relief after 15 minutes: Apply table Cx SB
- No relief or worsening of symptoms: Apply table Cx N
- Recurrence: Apply a second table CX B

STOP decompression

Put divers on BIBS for 60 minutes without interruption

Depth of treatment	Mix to be used
0 - 18 metres	Oxygen
19 - 40 metres	Heliox 50/50
41 - 110 metres	Heliox 20/80
111 - 210 metres	Heliox 10/90
211 - 360 metres	Heliox 5/95
361 - 450 metres	Heliox 3/97

After successful treatment, resume decompression

Heliox bounce dive

Use the same table (same depth, same bottom time) and add 10 minutes to each stop of the initial decompression protocol

Heliox saturation

Apply standard heliox saturation decompression

Table Cx SB

This table can be used in case of failure of Table "Cx B" during normal decompression or after a blow-up from deeper than 9 m in an heliox dive or air/heliox saturation.

Procedure:

- Recompress the patient with the depth increment indicated according to the depth at which the accident occurred.
- Pressurize the chamber with heliox mix
  - Bottom mix for surface supplied diving
  - Pressurization mix for saturation diving
- Note the depth and time of relief.

No relief or worsening of symptoms:

- No relief or worsening of symptoms: Apply table Cx N
- Recurrence: Apply table CX B

Recompress the diver

Put diver on BIBS (25 minutes on + 5 minutes off)

Depth of accident	Depth of treatment	BIBS breathing duration	Mix to be used
0 - 18 metres	Plus 6 metres	120 minutes	Oxygen
13 - 31 metres	Plus 9 metres	120 minutes	Heliox 50/50
32 - 98 metres	Plus 12 metres	120 minutes	Heliox 20/80
99 - 195 metres	Plus 15 metres	120 minutes	Heliox 10/90
196 - 345 metres	Plus 15 metres	120 minutes	Heliox 5/95
346 - 450 metres	Plus 15 metres	120 minutes	Heliox 3/97

After successful treatment, resume decompression

Heliox bounce dive

Apply standard heliox saturation decompression depending on chamber atmosphere.

Heliox saturation

Apply standard heliox saturation decompression

A decompression procedure has been prepared in the case of short intervention of a paramedic inside the chamber. This procedure is available at the end of this chapter.

Table Cx N

This table can be used for treatment of "type 2" accident during normal decompression or after a blow-up from deeper than 9 m in an heliox dive or an air/heliox saturation.

Procedure:

- Recompress the patient 20 metres deeper than the depth at which the accident occurred.
- Pressurize the chamber with heliox mix
  - Bottom mix for surface supplied diving
  - Pressurization mix for saturation diving
- Put the patient on BIBS as per table below (the patient breathes 25 minutes on + 5 minutes off in chamber atmosphere for each 30 minute BIBS period indicated)
- Note the depth and time of relief.

No relief or worsening of symptoms:

- No relief or worsening of symptoms: Keep the patient on bibs on heliox for 25 min/ 5 min session during a period not exceeding 3 hours and wait for instructions from the Diving Medical Specialist (DMS).
- Recurrence: Apply table Cx N

Recompress the diver 20 metres deeper than the depth of accident

Put diver on BIBS (25 minutes on + 5 minutes off)

Depth of treatment after recompression	BIBS breathing duration	Mix to be used
20 - 40 metres	120 minutes	Heliox 50/50
41 - 110 metres	180 minutes	Heliox 20/80
111 - 210 metres	240 minutes	Heliox 10/90
211 - 360 metres	300 minutes	Heliox 5/95
361 - 450 metres	360 minutes	Heliox 3/97
346 - 450 metres	120 minutes	Heliox 3/97

After successful treatment, hold at treatment depth for 6 hours, then resume decompression

Heliox bounce dive

Apply heliox saturation decompression:
- 60 minutes/metre from treatment depth to 15 m. (PPO2 = 600 to 630 Mb)
- 120 minutes/metre from 15 m to surface (O2 percentage < 23%)

Heliox saturation

Apply heliox saturation decompression:
- 60 minutes/metre from 200 to 15 m. (PPO2 = 600 to 630 Mb)
- 120 minutes/metre from 15 m to surface (O2 percentage < 23%)

A decompression procedure has been prepared in the case of short intervention of a paramedic inside the chamber. This procedure is available at the end of this chapter.

Intervention of paramedics inside the chamber

The decompression procedure below has been prepared for short interventions of paramedics inside the chamber.

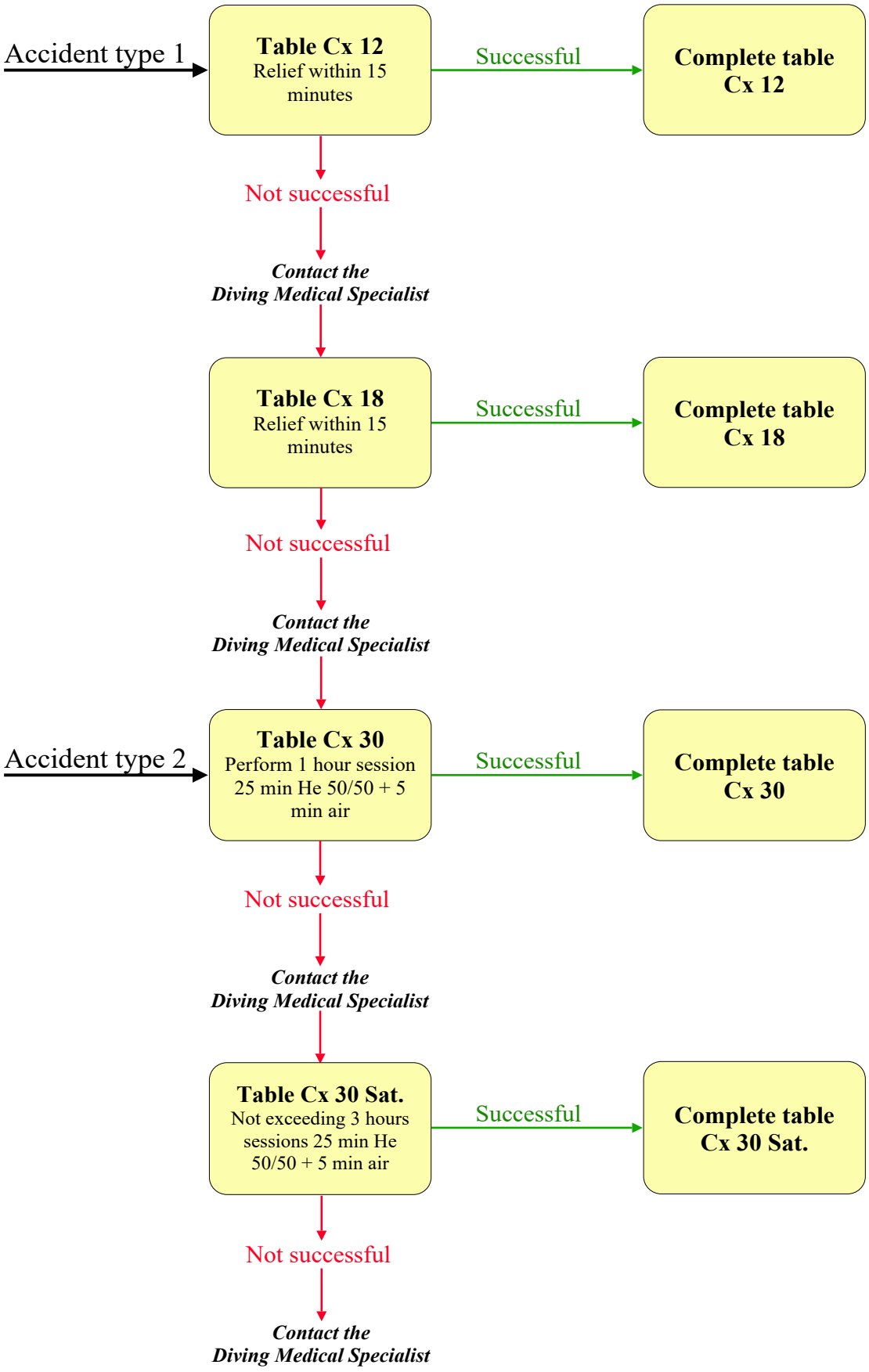
- Intervention of personnel (400 to 600 millibar PO2 in the chamber).
- Decompress personnel in chamber lock
- Personnel is put on BIBS from the beginning to the end of his decompression. He first breathes Heliox 20/80 and then Oxygen from 12m. to surface.
- If Oxygen stops are not mentioned at 3, 6 or 9 m., the personnel will slowly ascend from his last stop to surface with Oxygen on BIBS in 2 minutes

Chamber depth	Bottom time	Time to first stop	Heliox 20/80	Oxygen	Total decompression (h: min)
			30m - 27m - 24m - 21m - 18m - 15m	12m - 9m - 6m - 3m	
12 metres	90 min	1 min			0:01
20 metres	30 min	1 min		10 - - -	0:13
	60 min	1 min		20 - - -	0:23
	90 min	1 min		30 - - -	0:33
30 metres	30 min	2 min		15 - - -	0:19
	60 min	2 min		30 - 10 - -	0:44
	90 min	1 min	- - - - - 5	30 - 40 - -	1:18
40 metres	30 min	2 min	- - - 3 - 3 - 3	30 - 5 - -	0:48
	60 min	2 min	- - - 5 - 10 - 15	30 - 40 - 30 -	2:14
	90 min	2 min	- - - 5 - 15 - 15	30 - 40 - 50 -	2:39
50 metres	30 min	2 min	- 3 - 3 - 3 - 5 - 10	30 - 40 - -	1:38
	60 min	2 min	3 - 5 - 5 - 10 - 15 - 10	30 - 40 - 50 - 40	3:37
	90 min	2 min	5 - 5 - 15 - 15 - 20 - 30	30 - 40 - 50 - 80	4:54

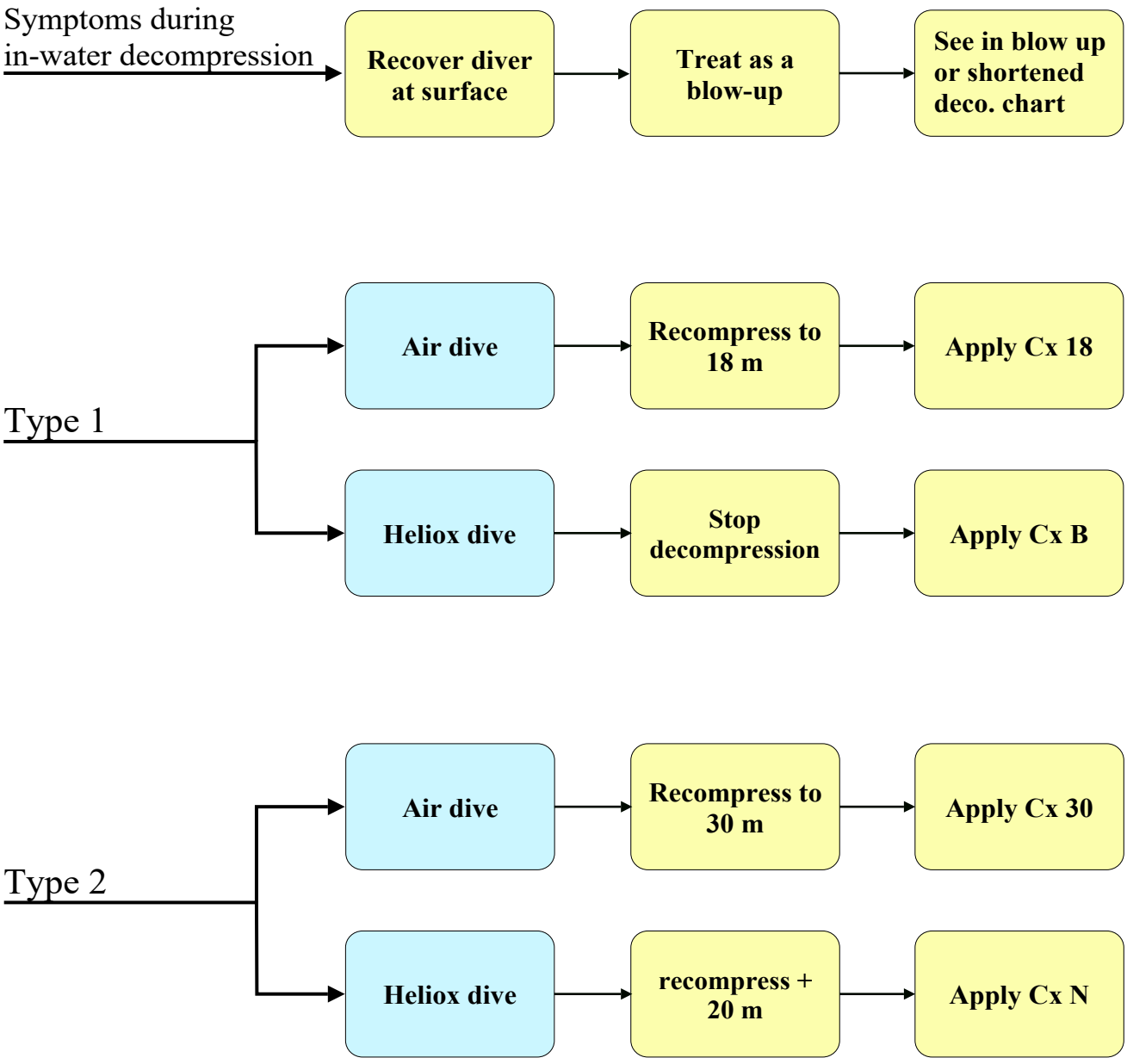
Obviously, where the intervention is too deep or too long, that personnel will have to stay in the chamber and be decompressed in saturation with the divers.

2 - Charts

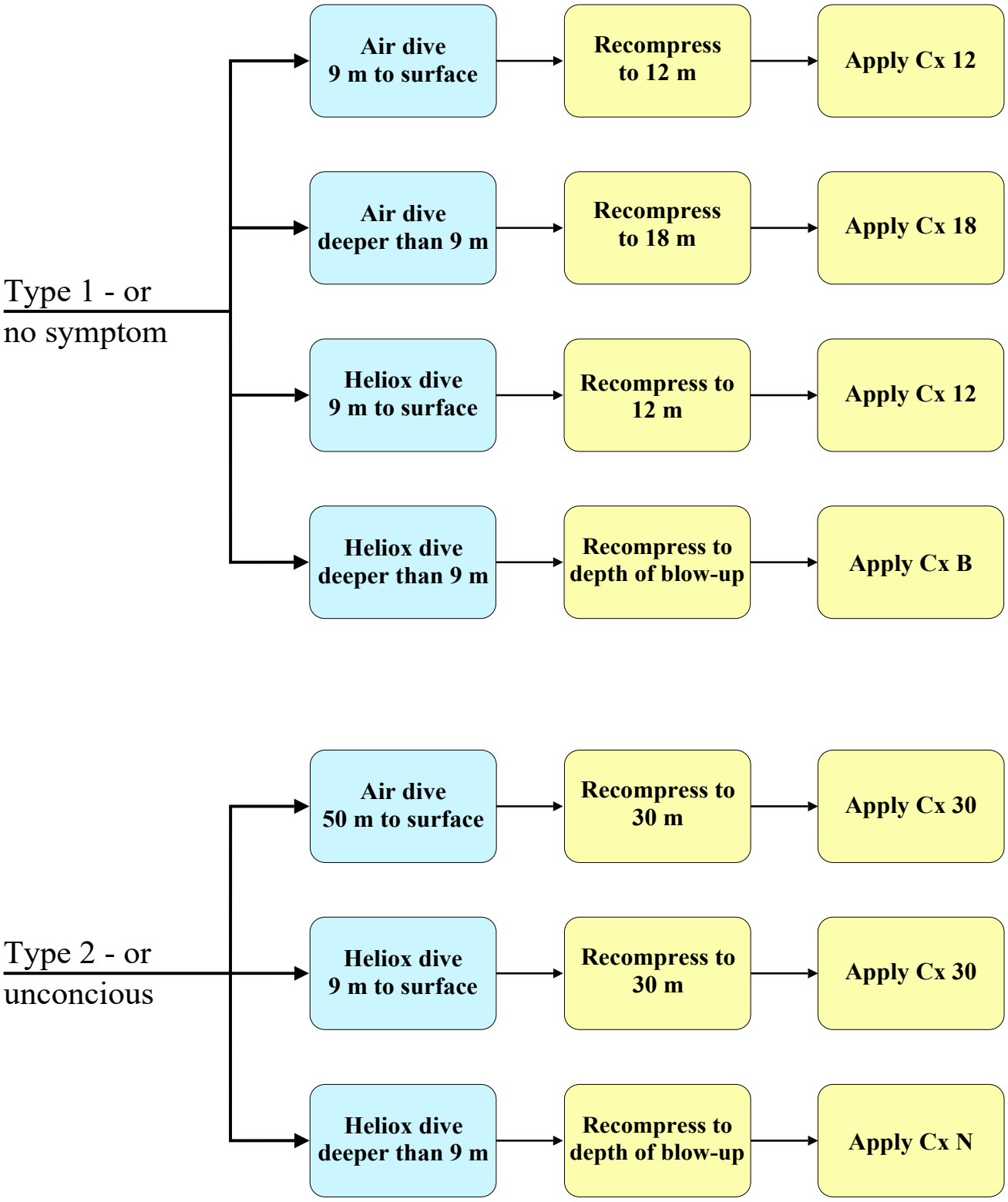
Decompression accident after normal decompression following air or heliox dive



Decompression accident during decompression of an air or heliox dive



Blow-up or shortened decompression



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Part F - Medical equipment checklist, recording data forms, and UPTD tables

DMAC 15 Rev. 5 - December 2021

Medical Equipment to be Held at the Site of an Offshore Diving Operation

Commercial diving operations include both surface supplied and saturation diving operations and cover a wide range of work activities. The appropriate medical equipment to be held at any particular site is best determined by an occupational health service with special knowledge of commercial diving operations. This document is designed to provide guidance on equipment and medical supplies to be held at the site where such advice is not available. It is recognised that in certain circumstances similar or greater facilities may be available from other sources which are sufficiently close and reliable. Geographical distances to both equipment, pharmaceutical agents and other competent personnel/specialists should be considered and evaluated before the commencement of Diving operations (i.e., SAR helicopter capabilities). This document will use the term Vessel Medic for the personnel with the responsibility for medical examination and treatment of ill or injured divers. We recognize that this function in some areas will be handled by a vessel medical officer, a dedicated vessel nurse or a vessel physician, or even a DMT in surface supplied operations.

The document covers equipment and drugs suitable for the treatment of diving related disorders on the surface or in a recompression chamber and for other potential problems (e.g. trauma) which may occur during diving operations. The document takes into account situations where the diving operation may be remote from a vessel or installation sickbay and medical services. It includes equipment for use in an immediate first aid situation, equipment and drugs which may be used by personnel with advanced first aid training, as well as equipment which would almost certainly only be used by medical staff. Medical staff who attend a casualty at a dive site may not necessarily be able to bring the appropriate equipment.

Each diving contractor's Diving Medical Advisor (DMA) is responsible for a review of the equipment and drugs, as some of the drugs mentioned in this document may not be available in some geographical areas and in such cases alternative drugs to those identified and 'suggested' should be considered. Compliance with the local laws relating to pharmaceutical products will also have to be considered, as these vary in different countries. It is anticipated that except in emergency situations, equipment other than that in the bell or chamber immediate care medical kits would be for use by or on the direction of medical staff. DMAC advises that the medical emergency preparedness is regularly reviewed, and that the robustness for the defined possible scenarios is clearly defined.

Medical equipment and drugs must be sourced and purchased from reputable suppliers capable of assuring the quality and safety of the purchases made. There should be an appropriate system for the control and maintenance of this Medical Kit and responsibility for this Kit should be vested in the Diving Superintendent or vessel Medic. The respective responsibilities and authority of the parties involved need to be clearly documented and understood.
This Kit should be accompanied by a list of contents. This list provides an opportunity for stock control, shelf-life control, and the recording of the use of the contents. These measures should be regarded as standard procedure. A specific logbook should be maintained with the equipment and supplies, in which these checks and all use of equipment and drugs is recorded.

The Diving Supervisor needs to have access to this Medical Kit at all times. Scheduled or Controlled drugs should be held in a dedicated and secure double locked container (with the vessel's medical supplies or in the installation sickbay).

The Medical Kit should be appropriately labelled and then inspected regularly (during each mobilisation and at least every six months) to ensure that all items are in working order (e.g. batteries) and to exchange drugs and other equipment which are nearing the end of their shelf life. These regular inspections should be recorded in the logbook.

Consideration should be given to the need for pressure testing mechanical or electrical equipment. Local conditions may require a more frequent inspection regime.

People involved in the provision of care in diving situations should be part of a system of continuous training to help prevent skill fade.

At all times, including during transit to the diving worksite or diving support vessel, this Medical Kit must be kept dry and clean, locked in an appropriately labelled and dedicated light-resistant container. Care must be also taken to ensure that this equipment is stored within a temperature range of +4° to +25° Celsius.

The kit should be packaged and labelled in a logical manner that allows easy and rapid access to the contents, specifically those contents that are often used at the same time.

This is a guidance document based on current information. This Medical Kit will not cover all the aspects of medical care in a diving system and should be used in conjunction with the installation or vessel's medical kit. DMAC 15 should also be read in conjunction with DMAC 28 "The provision of emergency medical care for divers in Saturation".

There should thus always be good communication between the diving team and the Vessel Medic.

Audits of any DMAC 15 kits should involve a Vessel Medic with experience in the field, who understands the limitations of this guidance note, understands the diving project, and understands the potential difficulties of the procurement of certain drugs depending on the country jurisdiction where the diving is taking place. A copy of DMAC 15 should accompany each kit with the final page (Appendix 1) completed as appropriate.

Equipment to be Held in a Diving Bell (*to be stored in a watertight bag or container*)

The same equipment should be available for rapid use in each living chamber of a saturation system and in air diving chambers

In 'living' chambers a foot or gas powered suction pump may be preferred. The watertight bag is not required within 'dry' living chambers and it is acceptable that this equipment is stored outside the system ready to be passed in immediately, as required.

If more of specific items are required these should be found in the normal ships' medical stores.

Description	Check
1 Emergency limb tourniquet (e.g. CAT or MAT)	
3 Polythene bags – these can be used to cover burns or as waste bags	
1 Resuscitation face mask (preferably with a silicone filled face seal and a non-return valve) or shield, for mouth-to-mouth ventilation (e.g. Laerdal pocket mask or face shield)	
2 Oropharyngeal airway size 3 and 4 (e.g. Guedel type)	
1 or 2 correctly sized Supraglottic airways (e.g. IGel sizes 3, 4 or 5) with catheter mounts and filters. This will require the assessment of the divers using the bell or habitat at any given time.	
1 Tuf cut scissors	
2 Triangular bandages	
1 Role of 1 inch adhesive tape	
1 Hand operated suction pump	
1 Wide bore suction catheter (for example: Yankeur)	
1 Watertight bag	
1 Adult adjustable cervical spine collar	
2 Pairs of non-sterile gloves (non-latex if possible, appropriately sized)	
2 x small "pods/sachets" of sterile water or NaCl for eye wash	
1 Medium dressing	
1 Large dressing	

Equipment to be Held at the Dive Site

Diagnostic equipment

Description	Check
1 Pencil torch	
1 Thermometer (electronic) to cover the range 26 0 C to 43 0 C - This could be one or 2 thermometers - Sheaths to allow multiple use.	
1 Stethoscope	
1 Aneroid sphygmomanometer	
1 Reflex hammer	
1 Tape measure	
2 Tuning forks (128Hz and 256Hz)	
Pins for testing sensation (e.g. Neurotips)	
Blood sugar testing strips	
Tongue depressors	
Urine testing strips	
1 Otoscope (with spare bulb and batteries and disposable ear pieces)	

Thoracocentesis / Chest decompression

Description	Check
2 Intercostal drain and drainage kits – preferably those without sharp metal 'trocar' introducers (e.g. Portex flexible introducer type)	
4 Large bore intravenous cannulae (approx. 14g)	
2 Heimlich valves	
Suitable strapping/fixing to secure the system appropriately	

Urinary Catheterisation

Description	Check
2 Urinary catheters sizes 16 and 18 (e.g. Foley type)	
2 Urine collection bags	
2 Catheter spigots (optional)	
2 x 20ml sterile water	

Urinary Catheterisation (continuation)

Description	Check
2 Urethral anaesthetic gel	
Suitable strapping/fixing to secure the system appropriately	

Dressings

Description	Check
10 Packets gauze squares 10 x 10 cm	
5 Packets cotton wool balls	
4 Triangular bandages	
4 Trauma care bandage	
12 Safety pins	
2 Adhesive bandages 75mm x 3m	
2 Adhesive bandages 25mm x 3m	
2 Crepe bandages 6 inch	
2 Crepe bandages 3 inch	
2 Large dressings	
2 Medium dressings	
40 Adhesive plasters	
2 Dressing bowls	
4 Eye pads	
1 Eye wash kit	
5 Soft silicone primary wound dressing (8cm x 10cm) (e.g. Mepitel)	

Note: Equipment to immobilise fractured/sprained limbs (e.g. a 36 inch SAM splint, which can be cut to size as required)

Sterile Supplies – General

There are many options of disposable sterile supplies which can be considered but should contain at least the following.

Description	Check
4 Universal containers	
2 Drapes 60 x 90 cm	
10 Alcohol swabs or sachets of skin disinfectant (e.g. Cetrimide solution)	
10 pairs of sterile gloves (selection of sizes, preferably non-latex)	

Sterile Supplies – General *(Continuation)*

Description	Check
6 Sutures non-resorbable (e.g. nylon) (2/0 and 3/0), preferably with cutting needles attached	
1 Medium sized sharps bin	
5 x 20 ml syringes	
5 x 10 ml syringes	
5 x 2 ml syringes	
10 x 18 g needles	
10 x 21 g needles	
4 packets of Steri-strips (paper stitches)	
One bottle of antimicrobial surgical skin cleanser (e.g. Hibiscrub, similar chlorhexidine based cleansers)	

Sterile Supplies – Specific

Description	Check
1 Kidney Dish	
1 x 60-100 ml bowl (Gallipot)	
10 sachets of skin disinfectant (e.g. Cetrimide solution)	
10 x 7.5 cm 4 ply Non Woven Cotton Swabs	
4 x Dressing Forceps	
2 x Tissue Backed Drapes 60 x 90 cm	
1 x Yellow Bag (for disposal of used items)	

Sterile Instruments

Description	Check
2 Spencer Wells forceps 5 inch	
1 Mosquito forceps	
1 Dressing forceps	
2 Disposable scalpels	
1 Forceps – fine toothed	
1 Dressing scissors	

Sterile Instruments *(Continuation)*

Description	Check
1 Scissors – fine pointed	
1 Stainless steel ring cutter (for removal of rings and other piercings) – this does not need to be sterile	

Intravenous Access

Description	Check
1 Tourniquet to aid venous access	
3 IV/IO Giving sets	
4 IV cannulae 16 g	
4 IV cannulae 18 g	
4 Butterfly infusion sets 19 g (optional)	
2 Magnetic hooks	
4 x 3-way IV taps	
10 x IV cannula dressings	
A pressure bag for rapid infusion of fluids	
1 Intraosseous (IO) infusion system. A minimum of 2 sets of devices are required. If used, the DMTs must be appropriately trained in and up to date with the system chosen and used by each diving company. The use of battery operated placement systems in a hyperbaric chamber environment is contraindicate	

Resuscitation

Description	Check
Self-inflating bag-valve-mask / Resuscitator to include reservoir and connection for BIBS gas (e.g. Laerdal type)*	
3 Resuscitation masks with silicone face seals (varied sizes	
1 Pocket resuscitator with one-way valve (e.g. Laerdal pocket mask with a silicone face seal and non-return valve) or face shield for mouth-to-mouth ventilation	
3 Supraglottic airways sized for adult males (e.g. IGel sizes 3, 4 and 5, or Combitube) with catheter mounts and filters – if female divers on board ensure there are correctly sized airways for them	
(optional) Endotracheal tubes (ET tubes) (e.g. sizes 7, 8 and 9) with catheter mounts and filters**	
(optional) Laryngoscope and batteries and spare bulbs. The use of Laryngoscopes with fibre optic disposable blades is encouraged. This is required if ET tubes are stored	
2 Oropharyngeal airways sizes 3 and 4 (e.g. Guedel type)	

Resuscitation *(continuation)*

Description	Check
1 Foot operated or gas powered suction device	
2 Endotracheal suction catheters	
2 Naso gastric tubes size 16	
2 Wide bore suckers	
(optional) 2 Nasopharyngeal airways e.g size 6 and 7 with flange	
1 Magill Forceps	
A nebuliser mask and associated tubing	

1 Automated external defibrillator: If this is to be used inside the chamber or hard wired into the chamber it should be appropriately tested for such use.

* *Resuscitators may require modification to gas inlet to ensure adequate filling at pressure*

** *Endotracheal tubes should be provided for use by doctors only*

For Surface Supplied diving there should be oxygen delivery systems available to enable normobaric oxygen therapy for each diver for the time required to reach any therapeutic facilities. As an absolute minimum there should be capacity for 4 hrs treatment with F I O 2 ≈1,0.
Rebreather systems such as the "Wenoll emergency oxygen rebreather" system should be considered to extend the life of any oxygen cylinder.

Drugs

These drugs should be present on the vessel (DSV) or installation and stored within the sick bay or medical facility. There is no requirement to have a specific set of DMAC 15 drugs completely separate from the vessel's or installation's normal inventory. However, the below drugs should be auditable as present prior to each diving operation in sufficient quantities to support the diving operations. There should be an appropriate system in place to replenish used items in a timely fashion.

A risk assessment must take place prior to any diving operation to consider the risks involved, the location and the time it would take to get any sick or injured diver to a place of definitive medical care. Thus, for example, for Saturation diving the type of work being performed (E.g. risk of trauma), the depth of the diving and the time for decompression are amongst the issues that need to be considered. This risk assessment may guide the choice of drugs required.

The drugs mentioned below are suggestions and the Diving Medical Advisor can always make appropriate changes and document these in Appendix 1 as needed. There are some drugs (IM or IV) that now come in prefilled syringes which may be a more suitable alternative to glass filled vials.

Anatomical Therapeutic Chemical (ATC) codes are provided in brackets for guidance.

Anaesthesia

Description	Check
Lidocaine injection without adrenaline (N01B B02) *Suggested: Lidocaine 10mg/ml or 20 mg/ml ampoules 5 x 10ml* *Indication: Lidocaine is a useful local anaesthetic in concentrations up to 20%*	

Analgesia

Description	Check
Soluble aspirin tablets (N02B A01) *Suggested: Soluble aspirin, 20 x 300mg or 20 x 500mg tablets* *Indication: Mild to moderate pain, pyrexia, chest pain of suspected cardiac origin 1 to 2 tablets every 4 to 6 hours*	

Description	Check
Paracetamol tablets (N02B E01) *Suggested: Paracetamol, 25 x 500mg tablets* *Indication: Mild to moderate pain, pyrexia (fever) 1 to 2 tablets every 4 to 6 hours to a maximum of 8 tablets in 24 hour period.*	
Non-Steroidal Anti-Inflammatory Drug (NSAID) (M01A XXX) *Suggested: Ibuprofen 30 x 400 mg tablets* *Indication: Mild to moderate pain and musculoskeletal inflammatory disorders. 1 tablet every 6 hours.*	
Codeine or dihydrocodeine tablets (N02A A08 or R05D A04) *Suggested: Codeine or dihydrocodeine, 20 x 25-30mg tablets* *Indication: Moderate to severe pain 1 x 25 or 30mg tablet every 4 to 6 hours when necessary*	
Morphine injection (N02A A01) *Suggested: Morphine, 5 x 10mg ampoules* *Indication: Severe and acute pain Patients should be closely monitored for pain relief as well as for side-effects especially respiratory depression.* *Patients should be closely monitored for pain relief as well as for side-effects especially respiratory depression.* *It may be appropriate to consider the use of an antiemetic after administration of Morphine.* *Other Opioids or Ketamine could be considered following a risk assessment by the companies Diving Medical Advisor*	
Naloxone injection (V03A B15) *Suggested: Naloxone, 0.4mg/ml ampoules – 2 x 1ml* *Indication: Opioid (morphine) overdose, respiratory depression due to administration of opioid (morphine) analgesia.* *Respiratory depression is a major concern with opioid analgesics and it may be treated by artificial ventilation or be reversed by Naloxone. Naloxone will immediately reverse opioid-induced respiratory depression but the dose may have to be repeated because of the short duration of action of naloxone; however, naloxone will also antagonise the analgesic effect*	
NSAID injection (M01A B05) *Suggested: Diclofenac 4 x 75mg Ampoules* *Indication: Severe and acute pain Can be given IM or IV (as a continuous infusion according to pharmaceutical protocols)*	

Resuscitation Drugs (refer to the appropriate resuscitation guidelines)

Description	Check
Adrenaline/Epinephrine injection (C01C A24) *Suggested: Adrenaline, 10 x 10ml ampoules. 100µg/ml ampoules (1 in 10,000)* *Indication: Emergency treatment for cardiopulmonary resuscitation* *Important: Intravenous route or intraosseous route to be used in resuscitation, during CPR only*	
Amiodarone injection (C01B D01) *Suggested: Amiodarone, 6 x 150mg ampoules* *Indication: Amiodarone is used for the treatment of arrhythmias particularly during CPR, particularly when other drugs are ineffective or contraindicated* *In some countries Amiodarone is sold as a powder – ensure the correct amount of the appropriate solute is included if this is the case* *Important: Intravenous route or IO route to be used in resuscitation during CPR only*	
Furosemide injection (C03C A01) *Suggested: Furosemide, 5 x 40mg ampoules* *Indication: Oedema, pulmonary oedema, resistant hypertension*	

Nausea, Vomiting, and Diarrhoea

Description	Check
Fentiazin or prochlorperazine injection (preferred) or oral (optional) (N05A B) *Suggested: Prochlorperazine, 5 x 25mg ampoules or Prochlorperazine, 20 x 5 mg tablets* *Indication: Severe nausea, vomiting, vertigo, labyrinthine disorders (not for use in motion sickness)* *Where available, prochlorperzine in 3mg buccal tablets (dissolves sublingually) is a good choice as opposed to the tablets as such as there is no need to swallow a tablet; 10 x 3mg tablets*	

Nausea, Vomiting, and Diarrhoea *(continuation)*

Description	Check
See notes on equipment to be held in diving bell above. These are additional: Hyoscine hydrobromide (Scopolamine) tablets/dermal patches (A04A D01) *Suggested: Hyoscine, 40 x 300µg tablets (e.g. Kwells)* *Indication: Short acting drug for sea sickness and hyperbaric evacuation. These tablets are chewable* *Suggested: Scopolamine, 20 x dermal patches (e.g. Scopoderm plasters)* *Indication: Long acting slow release drug for sea sickness and for hyperbaric evacuation. One patch to be placed behind one ear*	
Loperamide (A07D A03) *Suggested: Imodium 100x30 mg tablets* *Indication: Symptomatic treatment of diarrhoea. 1 tablet after each defecation. A minimum of 2 h should pass before next tablet.*	

Allergic Reactions

Description	Check
Antihistamine for injection (R06A B) *Suggested: Chlorpheniramine, 2 x 10mg ampoules or dekschlorpheniramine, 2 x 5 mg ampoules Indication: Symptomatic relief of allergy, urticaria, emergency treatment of anaphylactic reaction*	
Oral antihistamine (R06A E) *Suggested: Cetirizine, 20 x 10mg tablets* *Indication: Symptomatic relief of allergies – non-sedating* *There are different types of non-sedating oral antihistamines that can be substituted as needs be*	
Corticosteroid for injection (H02A B) *Suggested: Hydrocortisone 5 x 100mg ampoules* *Indication: Hypersensitivity reaction e.g. anaphylaxis, angiodema, asthma*	
Adrenaline/epinephrine autoinjector (C01C A24) *Suggested: Epinephrine autoinjector (EpiPen autoinjector) 0.3mg of 1 in 1000 (1mg/ml) adrenaline (giving 300mcg) 2ml auto-injector* *Indication: Emergency treatment of acute anaphylaxis*	

Drugs Various

Description	Check
Atropine injection (A03B A01) *Suggested: Atropine, 4 x 1ml ampoules (600µg/ml)* *Although atropine is no longer recommended in the treatment of asystole or pulseless electrical activity in the European resuscitation guidelines, it may be useful in the treatment of bradycardia*	
Glucose injection (B05B A03) *Suggested: Glucose 2 x 500mg/ml 50ml* *Indication: Hypoglycaemia*	
Glyceryl trinitrate sublingual tablets (C01D A02) *Suggested: Glyceryl trinitrate sublingual tablets x 10 tablets Indication: Cardiac chest pain*	
Intravenous fluids (B05B B01) *Suggested: Crystalloid Infusion – 6 litres* *Sodium Chloride Infusion 0.9% and/or Hartman's or Ringers lactate. Company doctor to advise on exact make up*	
Antipsychotic drug for injection (N05A A) *Suggested: Chlorpromazine, 2 x 50mg ampoules or levomepromazine, 2 x 25 mg ampoules* *Indication: For relief of acute symptoms, schizophrenia and other psychoses, mania short-term adjunctive management of severe anxiety, psychomotor agitation, excitement, and violent or dangerously impulsive behaviour*	
Anxiolytics for injection (N05B A) *Suggested: Diazepam, 5 x 10 mg ampoules* *Indication: Short-term use in anxiety or insomnia, status epilepticus and for muscle relaxant effect where indicated*	

Drugs Various

Description	Check
Anxiolytics for oral use (N05B A) *Suggested: Diazepam, 10 x 5 mg tablets* *Indication: Short-term use in anxiety or insomnia, status epilepticus*	
Anticonvulsant for nasal or buccal use (N05C D08) *Suggested: Midazolam 10mg in 2ml with the administration device (for example nasal atomiser or oro-mucosal solution)* *Indication: Short-term use in status epilepticus*	

Treatment of burns

Description	Check
Sulfonamides for topical use (skin) (D06B A01) *Suggested: Silver sulphadiazine cream, 1 tube of 1%* *Indication: for example prophylaxis and treatment of infection in burn wounds, as an adjunct to short-term treatment of extensive abrasions; for conservative management of finger-tip injuries*	

Description	Check
Broad spectrum antibiotic for parenteral administration (J01D D04) *Suggested: Ceftriaxone 3 x 2g vials (1-2g once daily Intravenously or by deep intramuscular injection.)* *Indication: For treatment of sepsis or other serious infections in patients unable to receive oral treatment.*	
Broad spectrum antibiotics for oral use (J01C A, J01C F or J01C R02) *1 - Suggested: Co-amoxiclav, 21 x 625mg tablets (1 tablet 3 times per day for 7 days) or dicloxacilline 30 x 500 mg tablets.* *Indication: A broad spectrum antibiotic useful in the treatment of Gram-negative bacterial infections.* *If this drug is not available the company doctor may choose to recommend a similar penicillinase resistant antibiotic* *2 - A Quinolone, for example:* • *Ciprofloxacin, 20 x 500mg tablets (1 tablet 2 times per day for 10 days) OR* • *Levofloxacin, 20 x 500mg tablets (1 tablet 2 times per day for 10 days)* *Indication: A Quinolone antibiotic is useful in the treatment of Pseudomonas infections*	
Macrolide antibiotic for oral use (J01F A) *Suggested: Clarithromycin 14 x 250mg tablets (1 tablet twice daily for 7 days) or erythromycin 30 x 250 mg tablets (2 tablets twice daily for 7 days)* *Indication: Susceptible infections in patients with penicillin hypersensitivity*	
Antibiotic ear drops with (S03C A) or without (S02A A) corticosteroids *Suggested: Sofradex (framycetin sulphate/ dexamethasone/gramicidin) ear drops or hydrocortisone/ polymyxine B ear drops one bottle 2–3 drops 3-4 times daily. May be substituted by other ear drops containing combinations of antibiotics (e.g. Polymyxine B, Neomycine, a.o.) and corticosteroid (hydrocortisone, dexamethasone a.o.)* *Sofradex drops can also be used for eye infections*	
Recommended additional antibiotic ear drops for saturation diving operations *Suggested: Ciprofloxacin ear drops (S02A A15)*	

There should be a sensible supply of therapeutic ear drops as ear infections are common.

Description	Check
Antibiotic eye drops/ointment (S01A A01) *Suggested: Chloramphenicol eye drops or ointment* *Indications: Eye infections*	
An antifungal drug *Suggested: Clotrimazole cream (D01C A01)* *Indications: For fungal skin infections*	
An Antibacterial ointment *Suggested: Bactroban / Bacitracin (D06A X05)* *Indications: For skin infections*	

Appendix 1

Company		Vessel	
Project (if relevant)		Dive site (if relevant)	
Vessel Medic's name		Vessel Medic's signature	

The following table is for the company Diving Medical Advisor to note down any variations from the DMAC 15 document, with the reason. This allows for the detailing of any geographical difficulties with drug or equipment supply. Any additional equipment or drugs required by the medical adviser or details of equipment issues should be noted here with the required remedial actions.

Name of DMAC 15 drug	***Replacement drug or "omitted"***	***Reason for this action***	***Doctor's Initials***
Equipment issues			***Doctor's Initials***

Name of Company Diving Medical Advisor *Signature:* *Date:*

DMAC 01 - Rev. 1 - July 2015

Aide mémoire for recording and transmission of medical data to shore

Purpose

DMAC 1 is a form created by the Diving Medical Advisory Committee (DMAC) to transmit the symptoms of an injured or ill diver to the diving medical specialist onshore. It is the complementary document of the electronic data transmission system that allows the doctor to remotely and permanently check the condition of a casualty in the saturation system. This document can be used to discuss with the diver medics in the chamber and provide them guidelines. Note that modern voice communication systems such as those discussed in point 1.2.8.8, "Communications and transmission of data" of Book #2, allow for unscrambled discussion of the doctors with the divers in the chamber.

This document can be downloaded at this address: https://www.dmac-diving.org/guidance/DMAC01.pdf

DMAC guidelines for filling the document

This form has been designed in three parts to make it easier to use.

Part 1 is an aide mémoire to obtain the initial essential information for transmission ashore in event of a medical emergency. This information will enable the onshore doctor to advise on immediate management of the casualty.

Part 2 collects more detailed information to provide a permanent record of the incident and to assist in accident analysis. Obviously, in urgent cases there must be no delay in contacting medical assistance with the information in Part 1. Part 2 should and can be completed later.

The onshore doctor will frequently ask for some further examination(s) to be carried out.

Part 3 provides a form for recording this information. This part will need to be used initially for the first examination and may need to be used repetitively at the request of the onshore doctor.

This part will be a record of your findings at any given time point. There is space to record which time point this is and to be clear regarding date and time and before or after which treatment. This is important in order to be able to get a decent time line on the treatment and progression of each case.

In all parts of this form there are sections which will not be relevant for the type of diving and situation you are in. It is recognised that it will not be necessary to complete the form fully in most cases. You are not necessarily expected to fill in every page for every patient.

Where a question (or section) is not applicable, 'N/A' should be entered.

If you are uncertain of the meaning of a question, do not attempt to answer it, but ring the question number, and annotate accordingly.

It is particularly useful to attempt to get photos by whatever means possible, for any unusual occurrence and to forward these on to the Doctors involved in supporting you.

For consistency, please use local time throughout.

Note that such a document is confidential

Initial Essential Information for Transmission Ashore In Event of an Emergency

Part 1 – Section A: General Information

1.	**Patient Family name:**		**First name:**	
	Age:		**Date of birth** (dd/mm/yyyy)**:**	
2.	**Company:**			
3.	**Worksite/vessel:**			
4.	**Country and/or location:**			
5.	**Date of onset of incident:**		**Local time** (HH:MM)**:**	
6.	**Type of incident:** A) Surface supplied ☐ i) Potentially DCI related ☐ ii) Trauma related ☐ iii) Other (e.g. possible myocardial infarction) ☐ B) Saturation ☐			
7.	**Reason for contacting shore doctor:** Assistance required urgently (life threatening) ☐ Assistance required as soon as possible ☐ Assistance required when practicable ☐ Assistance required when patient gets ashore ☐ For information only ☐			
8.	**State of consciousness:** Fully alert and orientated ☐ Drowsy (tends to fall asleep) ☐ Confused ☐ Unconscious but responds to pain ☐ Unconscious and unresponsive to pain ☐			
9.	**Has there been any disease and/or treatment since the last medical certificate for fitness to dive was issued:** Yes ☐ No ☐			
10.	**Specifically detail any significant past or recent medical history. Please identify any medication taken recently and ask for allergies:**			

Note: Cumulative hyperbaric exposure is a relevant parameter for assessing the level of individual susceptibility to DCI. Copies of the diver's logbook will show numbers of surface supplied dives – number of days spent in saturation – number of variations in storage depths – number of saturation excursions. A copy of the last medical certificate for fitness to dive and a copy of the last pre-saturation medical examination should be provided.

Part I – Section B: **Information about the Dive related to the Incident**

(If the illness is not related to diving, skip to Section E)

Patient Family name:		**First name:**	
Age:		**Date of birth** (dd/mm/yyyy)**:**	

1. Method:

SCUBA – open circuit	☐	Wet bell	☐
SCUBA – semi-closed circuit	☐	Bell bounce	☐
SCUBA – closed circuit	☐	Saturation	☐
Surface supplied	☐		

2. Breathing gas:

Air mixture	☐	Nitrox	☐
Heliox	☐	Trimix	☐

3. Job:

Diver	☐	
Bellman	☐	
Other (specify):	☐	_____

4. Working depth: ____ metres ☐/feet ☐

5. Bell depth (where relevant)**:** ____ metres ☐/feet ☐

6. Storage depth (where relevant)**:** ____ metres ☐/feet ☐

7. Time spent at working depth: ____ minutes

8. Decompression table and method selected: _____

In-water	☐	Nitrox	☐

If surface decompression, indicate the duration of the surface interval ____ minutes

Depth selected: ____ metres ☐/feet ☐

Bottom time selected: ____ minutes

Surface interval selected (repetitive dives): ____ hours ____ minutes

9. Type of work performed during the last working dive:

_____

_____

_____

Workload intensity during the dive (to be assessed by diver and supervisor)**:**

Low ☐ Fair ☐ High ☐ Very high ☐

10. Adverse conditions, if any (e.g. sea state, tidal stream, temperature, fouling, disorderly ascent, hard work, etc.)**:**

_____

_____

_____

11. Did the incident begin:

In the water	☐	In the deck chamber	☐
In the bell	☐		
Other (specify):	☐	_____	

12. At the onset of symptoms, was the patient:

Descending (ambient pressure increasing)	☐	Ascending (ambient pressure decreasing)	☐
On the bottom	☐	On the surface	☐
Undergoing no pressure change	☐	In the DDC	☐

Part I – Section C: **Compression/Decompression Incident**

(If the illness is not related to diving, skip to Section E)

Patient Family name:		**First name:**	
Age:		**Date of birth** (dd/mm/yyyy)**:**	

1.	**The incident occurred during or immediately following compression:**	Yes ☐	No ☐
2.	**The incident occurred during normal decompression:**	Yes ☐	No ☐
3.	**The incident occurred after surfacing following normal decompression:**	Yes ☐	No ☐
	Time of end of decompression at:	_ _ _ _ hours	_ _ _ _ minutes
4.	**The incident occurred following excursion from saturation:**	Yes ☐	No ☐
	Was this an upward or downwards excursion:	Up ☐	Down ☐
	Time of onset after return to storage depth:	_ _ _ _ hours	_ _ _ _ minutes
5.	**The incident occurred following blow-up/drop in pressure:**	Yes ☐	No ☐
	From: Depth: _ _ _ _ metres ☐/feet ☐ Local time (HH:MM):		
	To: Depth: _ _ _ _ metres ☐/feet ☐ Local time (HH:MM):		
6.	**The incident occurred in other circumstances:**	Yes ☐	No ☐
	Specify: _		
7.	**How many divers are in the chamber with the affected diver:** _ _ _ _ _ _ _ _ _		
8.	**How many of these divers have the DMT qualification:** _ _ _ _ _ _ _ _ _		
9.	**Onset of first symptom at:**		
	Depth: _ _ _ _ metres ☐/feet ☐ Local time (HH:MM):		
10.	**Niggles** (minor aches or itchings, often transient)**:**	Yes ☐	No ☐
11.	**Pain in joints:**	Yes ☐	No ☐
	State location: _		
12.	**Pain in muscles:**	Yes ☐	No ☐
	State location: _		
13.	**Pins and needles** (paraesthesia, tingling)**:**	Yes ☐	No ☐
	State location: _		
14.	**Patches of numbness, or altered sensation:**	Yes ☐	No ☐
	State location: _		
15.	**Muscle weakness or total loss of power** (paralysis)**:**	Yes ☐	No ☐
	State location: _		
16.	**Difficulty in urinating:**	Yes ☐	No ☐
17.	**Pain in the lumbar region, around waist, or in the abdomen:**	Yes ☐	No ☐

Part I – Section C: **Compression/Decompression Incident** (continued)

(If the illness is not related to diving, skip to Section E)

Patient Family name:		**First name:**	
Age:		**Date of birth** (dd/mm/yyyy)**:**	

18.	**Standing upright is difficult or impossible:**			Yes ☐	No ☐
19.	**Nausea:**			Yes ☐	No ☐
20.	**Vomiting:**			Yes ☐	No ☐
21.	**Vertigo, loss of balance** (dizziness, often with a sense of rotation of either themselves or their surroundings)**:**			Yes ☐	No ☐
22.	**Affected hearing:**			Yes ☐	No ☐
	If yes which side	Both ☐	Right ☐	Left ☐	
23.	**Speech problems:**			Yes ☐	No ☐
24.	**Visual problems** (visual acuity, blurred vision, affected field of vision)**:**			Yes ☐	No ☐
	If yes which side	Both ☐	Right ☐	Left ☐	
25.	**Drowsiness:**			Yes ☐	No ☐
	Specify: _____				
26.	**Agitation:**			Yes ☐	No ☐
	Specify: _____				
27.	**Breathlessness, painful breathing:**			Yes ☐	No ☐
	Specify: _____				
28.	**Respiratory distress worsening with decompression:**			Yes ☐	No ☐
29.	**Blood-stained froth in or coming from the airways:**			Yes ☐	No ☐
30.	**Skin: Pruritus** (itching) **with or without a marbling or erythematous rash:**			Yes ☐	No ☐
31.	**Sensation of swollen and/or painful skin in any area with or without rash:**			Yes ☐	No ☐
32.	**Others** (please specify any symptoms and their time of appearance and development below)**:**			Yes ☐	No ☐
	_____				
	_____				
	_____				
	_____				
	_____				
	_____				
	_____				
	_____				

It is particularly useful to attempt to get photos by whatever means possible, for any unusual occurrence and to forward these on to the doctors involved in supporting you.

Part I – Section D: **Previous Dive**

(If ended less than 24 hours before the accident)

Patient Family name:		**First name:**	
Age:		**Date of birth** (dd/mm/yyyy)**:**	

1.	**Method:**				
	SCUBA – open circuit	☐	Wet bell		☐
	SCUBA – semi-closed circuit	☐	Bell bounce		☐
	SCUBA – closed circuit	☐	Saturation		☐
	Surface supplied	☐			
2.	**Breathing gas:**				
	Air mixture	☐	Nitrox		☐
	Heliox	☐	Trimix		☐
3.	**Depth:**			_____ metres ☐/feet ☐	
4.	**Bottom time** (where relevant)**:**			_____ minutes	
5.	**Decompression table and method selected:**				
	In-water	☐	Nitrox		☐
	If surface decompression, indicate the duration of the surface interval			_____ minutes	
	Depth selected:			_____ metres ☐/feet ☐	
	Bottom time selected:			_____ minutes	
	Surface interval selected (repetitive dives)**:**			_____ hours	_____ minutes
6.	**Normal decompression:**			Yes ☐	No ☐
7.	**End of decompression:**				
	Date (dd/mm/yyyy)		Local time (HH:MM)**:**		
8.	**If saturation, when back to storage depth from last working dive:**				
	Date (dd/mm/yyyy)		Local time (HH:MM)**:**		
9.	**Type of work performed during last working dive:**				
10.	**Workload intensity during the dive** (to be assessed by diver and supervisor)**:**				
	Low ☐	Fair ☐	High ☐	Very high ☐	
11.	**What type of activity does he/she do between dives:**				

Part I – Section E: **Accident or Illness not related to Decompression**

Patient Family name:		**First name:**	
Age:		**Date of birth** (dd/mm/yyyy)**:**	

1.	**Nature of accident or illness:**		
2.	**Does he/she have difficulty or pain with breathing:**	Yes ☐	No ☐
3.	**Is the diver obviously injured:**	Yes ☐	No ☐
	Describe:		
4.	**Is he/she bleeding:**	Yes ☐	No ☐
5.	**If yes, is bleeding controlled:**	Yes ☐	No ☐
6.	**State of consciousness:**		
	Fully alert and orientated ☐		
	Drowsy (tends to fall asleep) ☐		
	Confused ☐		
	Unconscious but responds to pain ☐		
	Unconscious and unresponsive to pain ☐		
7.	**Details of symptoms:**		
8.	**Treatment given:**		

Part 2

Additional Information for Record Purposes

*NB. Do **not** delay transmission of Part 1 in order to complete this part of the form*

Part 2 – Section A: General Information

(Some of this may be repetitive from Part 1 Section A, but can be completed in greater detail)

1.	**Patient Family name:**		**First name:**	
2.	**Age:**		**Date of birth** (dd/mm/yyyy)**:**	
	Height (metres)**:**		**Weight** (kilos)**:**	
	Smoker:		Yes ☐	No ☐
3.	**Date of last medical examination** (dd/mm/yyyy)**:**			
4.	**Where are the medical records held:**			
5.	**Details of main medical carer, including address** (be it a GP or other)**:** E-mail address: Phone number:			
6.	**Details of any previous or possible decompression sickness:**			
7.	**Has there been any disease and/or treatment since the last medical certificate for fitness to dive was issued:**		Yes ☐	No ☐
8.	**Specifically detail any significant past or recent medical history. Please identify any medication taken recently and ask for allergies:**			
9.	**Have there been any specific activities or travel during the previous two months:**		Yes ☐	No ☐
10.	**Name of diving supervisor:** E-mail address: Phone number:			
11.	**Name of medical attendant:** E-mail address: Phone number:			
12.	**Local time** (HH:MM) **of transmission of Part 1:**		**Date** (dd/mm/yyyy)**:**	
13.	**Addressee:**			
14.	**Copied to:**			

Part 2 – Section B: **Brief Statement of the Problem**

Patient Family name:		**First name:**	
Age:		**Date of birth** (dd/mm/yyyy)**:**	

Part 2 – Section C: **Summary of Advice/Instructions Received from Ashore**

Part 2 – Section D: **Details of Treatment Given**

(Including therapeutic tables by number as well as depth, duration and gases,and all supplementary therapy). State also times of implementation

Patient Family name:		**First name:**	
Age:		**Date of birth** (dd/mm/yyyy)**:**	

Part 2 – Section E: **Record of Progress;Summary of History of the Condition, Times of Significant Changes**

(Please use this alongside the use of Part 3 as needed)

Part 2 – Section F: **Summary of Facilities and Equipment Available**

Patient Family name:		**First name:**	
Age:		**Date of birth** (dd/mm/yyyy)**:**	

Record of Medical Examination

(This part may be used repeatedly)

All or part of this examination may be carried out at the request of the onshore doctor. Results should be recorded in the appropriate section and the questions which are not relevant to the particular incident should be left blank.

Part 3 – Section A: Examination/General

Patient Family name:		**First name:**	
Age:		**Date of birth** (dd/mm/yyyy)**:**	
Country and/or location:			

Time point (please complete date (dd/mm/yyyy) and local time (HH:MM))**:**		
Initial examination:	Date	Time
Pre- or post-therapy:	Pre ☐	Post ☐
This examination:	Date	Time
Therapy number:		
Examination number:		
Name of person examining:		

1. **Is the patient in pain:** Yes ☐ No ☐

If 'yes', specify site, intensity and any factors which worsen or relieve it:

Pain score. Visual analogue scale (0 being no pain, 10 being the worst pain imaginable)

0 ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐ 7 ☐ 8 ☐ 9 ☐ 10 ☐

2. **Does he/she have any major injury:** Yes ☐ No ☐

If 'yes', name the site and describe briefly. If there is bleeding give an estimate of blood loss (if yes, please attempt to take a photo of the affected area and send to the doctor):

3. **What is his/her temperature:** ____ °C

How and where was this temperature taken: ______

If he/she has just come out of the water, what is the water temperature: ____ °C

What is the ambient temperature: ____ °C

4. **Does he/she have any skin rashes** (undress the patient to his/her underwear)**:** Yes ☐ No ☐

If 'yes', describe appearance and site (if yes, please attempt to take a photo of the affected area and send to the doctor):

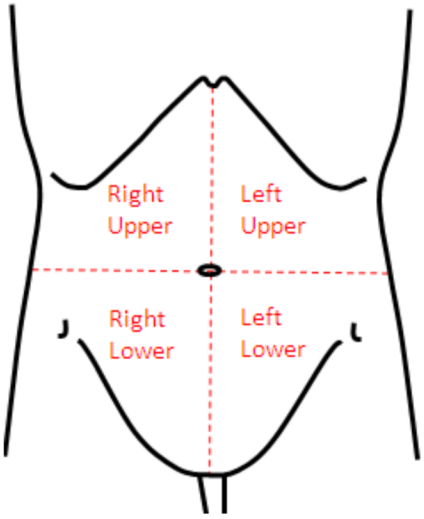
Part 3 – Section B: **Cardiorespiratory Systems**

Family name:		**First name:**		**Date of birth:**	
Exam number:		**Time:**		**Date:**	
Country and/or location:					

1.	**Is his/her colour** (look at lips and nail beds):	Normal ☐	Pale ☐	Cyanosed (blue) ☐
2.	**Is he/she visibly sweating:**		Yes ☐	No ☐
3.	**What is his/her:** i) Pulse (count for 30 seconds, then x 2)			_____ per min
	Is the pulse regular:		Yes ☐	No ☐
	ii) Blood pressure		_____ Syst	_____ Diast
	iii) Respiratory rate (count for 30 seconds, then x 2)			_____ per min
	iv) Pulse oximetry			_____ %
	v) Measure the capillary refill time (use your thumb to press down the nail on the patient's index finger for 5-10 seconds. Release the pressure – how long does it take to get normal nail bed colour back?)			_____ seconds
4.	**Does he/she have pain or difficulty breathing:**		Yes ☐	No ☐
	If 'yes', describe: _____			
5.	**Does he/she have a cough:**		Yes ☐	No ☐
	If 'yes', has he/she coughed blood:		Yes ☐	No ☐
6.	**Is he/she short of breath:**		Yes ☐	No ☐
	If 'yes', has this been affected by:			
	i) increase of pressure		Yes ☐	No ☐
	ii) decrease of pressure		Yes ☐	No ☐
7.	**Is the trachea** (windpipe) **central** (i.e. normal):		Yes ☐	No ☐
8.	**Are breath soundsaudible equally on both sidesof the chest** (listen at three different levels – preferably over the back):		Yes ☐	No ☐
9.	**Is there any subcutaneousemphysema** (crackling sensation in the tissues around the thorax) (carefully feel in each triangular area between the shoulder and the neck)		Yes ☐	No ☐

Part 3 – Section C: **Abdomen**

Family name:		**First name:**		**Date of birth:**	
Exam number:		**Time:**		**Date:**	
Country and/or location:					

1.	**Does the patient have abdominal pain:**	Yes ☐	No ☐
	If 'yes', specify site by writing on the chart, and make a note of the character:		
			
	When examining points 2-4, gently touch (palpate) **the abdomen in each of the four quadrants**		
2.	**Does it feel soft:**	Yes ☐	No ☐
	If 'no', specify the site (by writing 2 on the chart above, over the appropriate area)		
3.	**Are there any swellings:**	Yes ☐	No ☐
	If 'yes', specify site (by writing 3 on the chart above, over the appropriate area), size and consistency:		
4.	**Is the abdomen tender:**	Yes ☐	No ☐
	If 'yes', specify the site (by writing 4 on the chart above, over the appropriate area; assess tenderness by slowly depressing and then abruptly removing pressure in each quadrant)		
5.	**Can you hear bowel sounds with a stethoscope used for one minute:**	Yes ☐	No ☐
6.	**Does the patient have diarrhoea:**	Yes ☐	No ☐
	If 'yes', specify: a) When the patient last opened his/her bowels b) Specify the frequency in the last 24 hours		
7.	**Has the patient vomited:**	Yes ☐	No ☐
	If 'yes', specify: a) When the patient last vomited b) If he/she is still vomiting, specify frequency and character:		

Part 3 – Section C: **Abdomen** (continued)

Family name:		**First name:**		**Date of birth:**	
Exam number:		**Time:**		**Date:**	
Country and/or location:					

8.	**Has he/she vomited blood:**	Yes ☐	No ☐
9.	**Can the patient passurine without difficulty:**	Yes ☐	No ☐
	When did the patient last pass urine		
10.	**Is urinating painful:**	Yes ☐	No ☐
11.	**Is the urine**		
	Clear ☐	Blood-stained (red) ☐	
	Other (explain) ☐		
12.	**Urine colour** (tick applicable reference colour):		
	☐ ☐ ☐ ☐ ☐ ☐	Unable to provide ☐	
13.	Urine dip stick results (when reading results from the side of a bottle check order of results – they are usually, but not always, in order given below):		
	Leukocytes	Positive ☐	Negative ☐
	Nitrite	Positive ☐	Negative ☐
	Urobilinogen	Positive ☐	Negative ☐
	Protein	Positive ☐	Negative ☐
	pH	Positive ☐	Negative ☐
	Haemoglobin	Positive ☐	Negative ☐
	Specific gravity	Positive ☐	Negative ☐
	Ketone	Positive ☐	Negative ☐
	Bilirubin	Positive ☐	Negative ☐
	Glucose	Positive ☐	Negative ☐
	Record in more detail any positive results (semi-quantitative values are usually reported as: trace, 1+, 2+, 3+ and 4+):		
14.	**Additional comments**		

Part 3 – Section D: **Nervous System**

(This may be the most frequently repeated of the examinations, and this section may need to be used and transmitted several times)

Patient Family name:		**First name:**	
Age:		**Date of birth** (dd/mm/yyyy)**:**	
Country and/or location:			

Time point (please complete date (dd/mm/yyyy) and local time (HH:MM))**:**		
Initial examination:	Date	Time
Pre- or post-therapy:	Pre ☐	Post ☐
This examination:	Date	Time
Therapy number:		
Examination number:		
Name of person examining:		

1.	**Has he/she had any visual disturbance:** Yes ☐ No ☐ If 'yes', specify: _____	
2.	**Has he/she had a headache:** Yes ☐ No ☐ If 'yes', specify the location and timing of this headache: _____	
	Headache pain intensity score. Visual analogue scale (0 being no pain, 10 being the worst pain imaginable) 0 ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐ 7 ☐ 8 ☐ 9 ☐ 10 ☐	
3.	**State of consciousness:** Fully alert and orientated ☐ Drowsy (tends to fall asleep) ☐ Confused ☐ Unconscious but responds to pain ☐ Unconscious and unresponsive to pain ☐	
4.	**Are his/her pupils equal and reactive to light:** Yes ☐ No ☐ Quickly point a flashlight beam into the right pupil. Does it contract? Repeat the test. Does the opposite pupil contract similarly? Repeat examination on the left eye in a similar fashion. If 'no', specify: _____	
5.	**Does the patient have vertigo** (dizziness, often with a sense of rotation of either themselves or their surroundings)**:** Yes ☐ No ☐	
6.	**Does the patient have normal vision:** Yes ☐ No ☐ Put your index finger approx. arm's length in front of the patient's nose. Ask the patient to focus on it. Can he/she see it sharply: Yes ☐ No ☐ Does he/she have double vision: Yes ☐ No ☐	

Part 3 – Section D: **Nervous System** (continued)

Family name:		**First name:**		**Date of birth:**	
Exam number:		**Time:**		**Date:**	
Country and/or location:					

7. **Does the patient have nystagmus** (eye flickering): Yes ☐ No ☐

Put your index finger approx. arm's length in front of the patient's nose. Ask the patient to focus on it.
Slowly move your finger in an 'H' pattern. Is there any nystagmus (uncontrolled oscillatory or jerky eye movements)? Particularly look for this when the patient is gazing almost at the extreme of eye movement.
You may be able to get a video of this movement and send it to the onshore doctor.

8. **Is hearing equal and normal in both ears:** Yes ☐ No ☐

Stand behind the patient or ask him to close his/her eyes. Slide/rub your fingers against each other outside his/her right ear.
Ask him to tell you when he/she can hear it. Repeat the examination on the left ear. Is there any difference?
Alternatively: Put the patient 2-3 m in front of you with his/her back against you. Ask him to cover his/her right ear.
Whisper two-digit numbers (21-99) and ask him to repeat it. Redo with left ear.
Is there any difference between the two sides?
In a normal diving environment this is likely to be difficult – we are only looking for obvious problems.

9. Are the remainder of the cranial nerves normal:

Are both eye movements equal and normal:	Yes ☐	No ☐
Is facial sensation normal (test on three levels on each side):	Yes ☐	No ☐
Are facial movements normal on both sides (smile, close and open both eyes shrug his/her upper face and blow out cheeks):	Yes ☐	No ☐
Can he/she swallow easily (look for movement of the larynx):	Yes ☐	No ☐
Is the soft palate symmetrically positioned in the throat without deviation of the uvula:	Yes ☐	No ☐
Can he/she shrug his/her shoulders equally (with and without resistance):	Yes ☐	No ☐
Are his/her tongue movements normal (does the tongue deviate when stretched straight forwards and can it move normally?):	Yes ☐	No ☐

10. **Can the patient voluntarily move his:**

R Shoulder	Yes ☐	No ☐	L Shoulder	Yes ☐	No ☐
R Elbow	Yes ☐	No ☐	L Elbow	Yes ☐	No ☐
R Wrist	Yes ☐	No ☐	L Wrist	Yes ☐	No ☐
R Fingers	Yes ☐	No ☐	L Fingers	Yes ☐	No ☐
R Hip	Yes ☐	No ☐	L Hip	Yes ☐	No ☐
R Knee	Yes ☐	No ☐	L Knee	Yes ☐	No ☐
R Angle	Yes ☐	No ☐	L Angle	Yes ☐	No ☐
R Toes	Yes ☐	No ☐	L Toes	Yes ☐	No ☐

11. **Has he/she any weakness:** Yes ☐ No ☐

Examine the force that is achievable in each joint listed above.
Wherever possible ask him to contract muscles on both sides against your resistance.
Carefully consider: Is the force equal on the two sides?
Is the force considered normal by yourself and the patient?
Most joints (e.g. wrist, elbow, knee and ankle) have only two directions of movement (one plane) but the hip and shoulder should be assessed for movements in two planes. It is common to be slightly stronger on the dominant (usually right) side.

If 'yes', specify:

Part 3 – Section D: **Nervous System** (continued)

Family name:		**First name:**		**Date of birth:**	
Exam number:		**Time:**		**Date:**	
Country and/or location:					

12. Are reflexes (tendon jerks)**:**

		Normal	Increased	Absent	Not clear
Triceps	R	☐	☐	☐	☐
	L	☐	☐	☐	☐
Biceps	R	☐	☐	☐	☐
	L	☐	☐	☐	☐
Knee	R	☐	☐	☐	☐
	L	☐	☐	☐	☐
Ankle	R	☐	☐	☐	☐
	L	☐	☐	☐	☐

The reflex examination is only to be completed by trained persons and should not delay other examinations. When assessing reflexes note carefully any difference between the two sides (this is more important than the strength of the reflexes).

13. Is the plantar response:

	↑R ☐	↑L ☐
OR	↓R ☐	↓L ☐
or not clear	R ☐	L ☐

The plantar response is tested by drawing a moderately sharp, but non-injuring object (e.g. the tip of a pen) on outermost part of the sole of the foot from the heel to the fifth toe. Carefully observe the first direction of movement of the first (great) toe.

14. Does he/she feel any altered feelings in the skin (numbness, pins and needles)**:** Yes ☐ No ☐

If 'yes', specify and identify on the diagram on page 19 _____

15a. Is there a normal sensory response to soft touch: Yes ☐ No ☐

If 'no', specify and identify on the diagram on page 19 _____

Use a cotton wool or textile to lightly touch the back of his/her right big toe. Repeat on the left side.
Is there normal sensation?
Is this equal on the two sides?
Repeat successively on representative parts of the rest of the body ensuring you touch both outer and inner parts of the legs and arms.
It is not necessary to examine skin sensation on the back if you compare right and left side on 5-6 levels on abdomen and thorax

15b. Is there a normal sensory response to pinprick: Yes ☐ No ☐

If 'no', specify and identify on the diagram on page 19

Use a toothpick or a similar sharp (but not wounding) object to test for sensation of sharpness. Otherwise do the test as per 15

16. Can you detect a level of sensory change: Yes ☐ No ☐

17. Does he/she have distal vibration sensation (using 128Hz tuning fork)**:** Yes ☐ No ☐

If 'no', specify:

_____

Ask him to close his/her eyes. Put the 128Hz tuning fork on the top of the joint of the big toe. It should explained to the patient that it is the sensation of vibration, not cold or touch, which is being tested. The test may be made more objective and sensitive by giving a careful explanation and demonstration of the vibratory sensation. Place the tuning fork on the joint of the big toe, sometimes vibrating and sometimes not. If he/she has absent vibration sensation on the big toe, perform this test again on the lateral malleolus (ankle bone).

Part 3 – Section D: **Nervous System** (continued)

Family name:		**First name:**		**Date of birth:**	
Exam number:		**Time:**		**Date:**	
Country and/or location:					

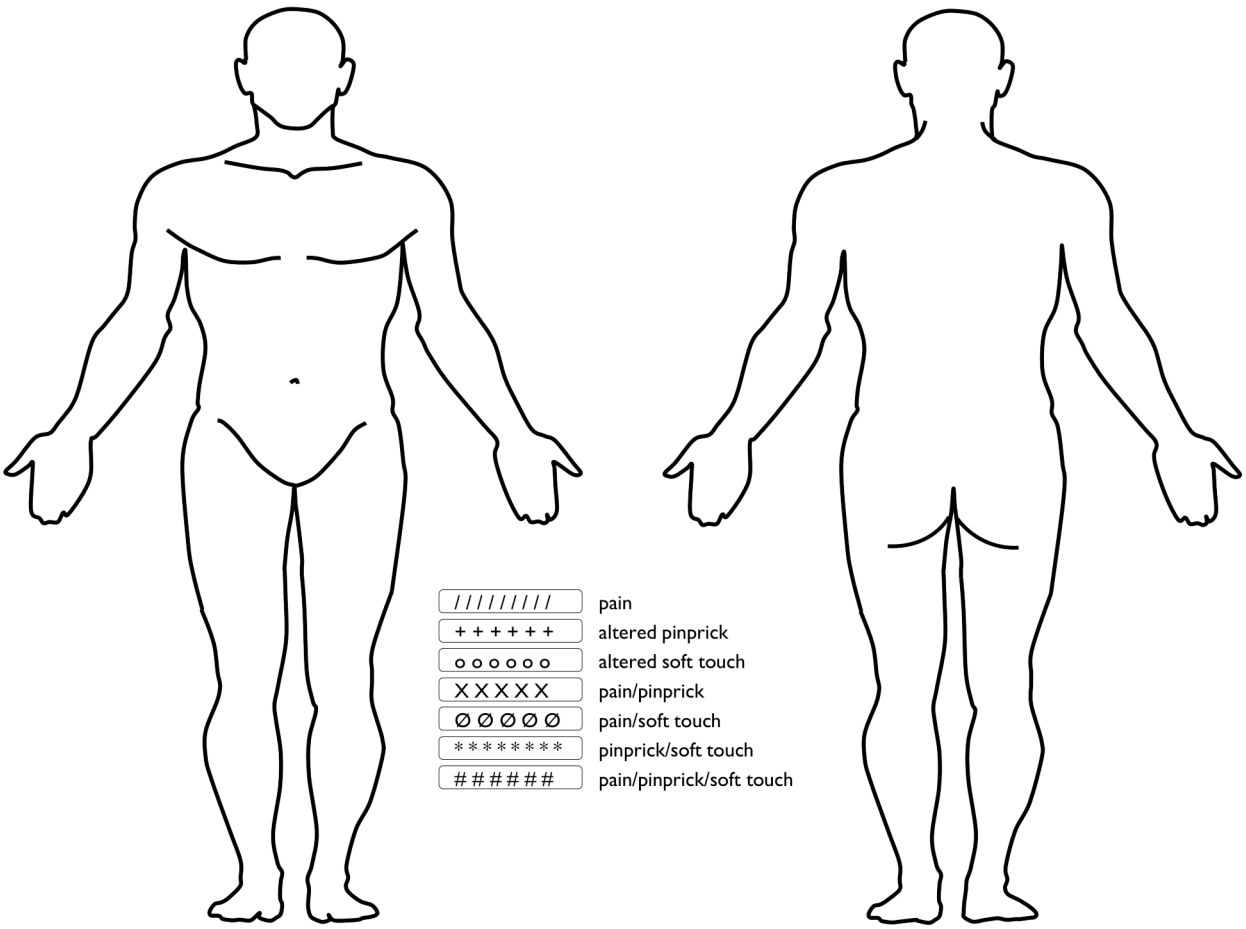
18.	**Does he/she have equal and seemingly normal proprioceptive** (joint position) **sense:** Yes ☐ No ☐ If 'no', specify: Grasp his/her right big toe with one finger on each side (not on sole/back). Ask him to close his/her eyes and keep them closed for this test. Put the toe in the maximum upper position and explain 'This is up'. Then move the toe to the maximum downward position and explain 'This is down'. Then position the toe in the mid/neutral position and ask him to notify the direction of the movement when you slowly move the toe upwards. Repeat the movement in different directions (a few times) to learn his/her threshold for joint movement. Repeat the examination on the left side. Ensure you move the toe more than 10 degrees in arc, with larger movements first, then smaller ones.

Please document your findings on the diagrams below.

Mark areas of altered sensation as shown below. Please note these may change over time with treatment.

Also use this diagram to show any other findings of interest.

If there are visible signs – please remember that photos are very useful.



Part 3 – Section D: **Nervous System** (continued)

Family name:		**First name:**		**Date of birth:**	
Exam number:		**Time:**		**Date:**	
Country and/or location:					

19.	**Does he/she have a normal finger–nose test:** Yes ☐ No ☐ This is a useful test of co-ordination in the arms. Stand in front of him and ask him to touch the tip of your index finger held in front of his/her nose, with the index finger of his/her outstretched right arm and then to touch the tip of his/her nose. He should be able to do this repeatedly, both with his/her eyes open and closed. You needed to assess smoothness of movement and accuracy. Repeat the test using his/her left arm. The sensitivity of this test can be increased by the examiner moving his/her finger to a different place whilst the patient's finger is en route to it with his/her eyes open.
20.	**Does he/she have a normal heel–shin test:** Yes ☐ No ☐ This is a useful test of co-ordination in the legs. When he/she is lying flat ask him to place the heel of his/her right foot on the opposite knee and then slide it down accurately, on the shin, to the ankle and back. He should be able to do this with his/her eyes open and closed. You needed to assess smoothness of movement and accuracy. Repeat the test using his/her left foot.
21.	**Can he/she perform a normal tandem gait walk** (heel–toe walking)**:** Yes ☐ No ☐ Ask him to walk heel to toe for about 10 steps both forwards and backwards with his/her eyes open first and then with his/her eyes closed. Be prepared to catch him in case he/she falls.
22.	**Is the Romberg test positive or negative** Positive ☐ Negative ☐ Ask him to remove his/her shoes and stand with his/her two feet together. his/her arms should be held by his/her sides. You should ask him to first stand quietly with eyes open, and then with his/her eyes closed. he/she should try to maintain his/her balance. Be prepared to catch him in case he/she falls. This test is scored by counting the seconds he/she is able to stand with eyes closed. Record seconds he/she is able to stand with eyes closed. Romberg's test is positive if he/she sways significantly or falls while his/her eyes are closed. This test may not be possible to use on a vessel in motion – it should only be performed on a stable and solid 'platform'.
23.	**Is the Sharpened Romberg test positive or negative** Positive ☐ Negative ☐ Test results in seconds: 1st _____ 2nd _____ 3rd _____ 4th _____ Ask him to remove his/her shoes and stand with his/her feet in a tandem gait (heel–toe) position. his/her arms should be crossed over the chest. You should ask him to first stand quietly with eyes open, and then with his/her eyes closed. he/she should try to maintain his/her balance. Be prepared to catch him in case he/she falls. This test is scored by counting the seconds he/she is able to stand with eyes closed. The test should be conducted up to four times for up to a minute. This Sharpened Romberg's test is positive if he/she sways or falls while his/her eyes are closed. This test may not be possible to use on a vessel in motion – it should only be performed on a stable and solid 'platform'.

Part 3 – Section E: **Any Other Relevant Findingsnot listed Above**

Patient Family name:		**First name:**	
Age:		**Date of birth** (dd/mm/yyyy)**:**	
Country and/or location:			

Time point (please complete date (dd/mm/yyyy) and local time (HH:MM))**:**		
Initial examination:	Date	Time
Pre- or post-therapy:	Pre ☐	Post ☐
This examination:	Date	Time
Therapy number:		
Examination number:		
Name of person examining:		

Dive record form

The form below is an example of a record transmitted to the doctor, the company management and be kept onboard.

Date:	***Location:***	***Contract:***	
Incident report #:	***Recompression table:***		***Page nb:***
Patient :	***DMS:***	***Superintendent:***	***Supervisor:***
LST # 1:	***LST # 2:***	***Tender #1:***	***Tender #2:***

Time	***Depth***	***Duration of the step***	***Patient therapeutic mix***	***Tender breating mix***	***Chamber atmosphere + O_2 & CO_2***	***Total Time elapsed***	***Events***

Emergency contacts list

This document lists the main contacts that should be indicated in the emergency response plan and displayed in the dive control.

Project management

Company director:

Mob:
Tel:
Fax:
E Mail:

Operation manager:

Mob:
Tel:
Fax:
E Mail:

HSE manager:

Mob:
Tel:
Fax:
E Mail:

Project manager:

Mob:
Tel:
Fax:
E Mail:

Emergency response team:

Mob:
Tel:
Fax:
E Mail:

Medical Support

Diving Medical Specialist #1:

Mob:
Tel:
Fax:
E Mail:

Diving Medical Specialist #2:

Mob:
Tel:
Fax:
E Mail:

Hospital # 1:

Mob:
Tel:
Fax:
E Mail:

Hospital # 2:

Mob:
Tel:
Fax:
E Mail:

MEDEVAC:

Mob:
Tel:
Fax:
E Mail:

Unit Pulmonary Toxic Doses (UPTD) tables as calculated by Doctor W. Brandon Wright

Unit Pulmonary Toxic Dose - PP oxygen 0.6 ata

Time (minutes)	***1***	***2***	***3***	***4***	***5***	***6***	***7***	***8***	***9***	***10***
UPTD	*0.26*	*0.52*	*0.78*	*1.05*	*1.31*	*1.57*	*1.83*	*2.09*	*2.35*	*2.62*
Time (minutes)	***11***	***12***	***13***	***14***	***15***	***16***	***17***	***18***	***19***	***20***
UPTD	*2.88*	*3.14*	*3.4*	*3.66*	*3.92*	*4.18*	*4.45*	*4.71*	*4.97*	*5.23*
Time (minutes)	***21***	***22***	***23***	***24***	***25***	***26***	***27***	***28***	***29***	***30***
UPTD	*4.49*	*5.75*	*6.02*	*6.28*	*6.54*	*6.8*	*7.06*	*7.32*	*7.58*	*7.85*
Time (minutes)	***31***	***32***	***33***	***34***	***35***	***36***	***37***	***38***	***39***	***40***
UPTD	*8.11*	*8.37*	*8.63*	*8.89*	*9.15*	*9.42*	*9.68*	*9.94*	*10.2*	*10.46*
Time (minutes)	***41***	***42***	***43***	***44***	***45***	***46***	***47***	***48***	***49***	***50***
UPTD	*10.72*	*10.98*	*11.25*	*11.51*	*11.77*	*12.03*	*12.29*	*12.55*	*12.82*	*13.08*
Time (minutes)	***51***	***52***	***53***	***54***	***55***	***56***	***57***	***58***	***59***	***60***
UPTD	*13.34*	*13.6*	*13.86*	*14.12*	*14.38*	*14.65*	*14.91*	*15.17*	*15.43*	*15.69*

Unit Pulmonary Toxic Dose - PP oxygen 0.7 ata

Time (minutes)	***1***	***2***	***3***	***4***	***5***	***6***	***7***	***8***	***9***	***10***
UPTD	*0.47*	*0.93*	*1.4*	*1.86*	*2.33*	*2.8*	*3.26*	*3.73*	*4.19*	*4.66*
Time (minutes)	***11***	***12***	***13***	***14***	***15***	***16***	***17***	***18***	***19***	***20***
UPTD	*5.13*	*5.59*	*6.06*	*6.52*	*6.99*	*7.46*	*7.92*	*8.39*	*8.85*	*9.32*
Time (minutes)	***21***	***22***	***23***	***24***	***25***	***26***	***27***	***28***	***29***	***30***
UPTD	*9.79*	*10.25*	*10.72*	*11.18*	*11.65*	*12.12*	*12.58*	*13.05*	*13.51*	*13.98*
Time (minutes)	***31***	***32***	***33***	***34***	***35***	***36***	***37***	***38***	***39***	***40***
UPTD	*14.45*	*14.91*	*15.38*	*15.84*	*16.31*	*16.78*	*17.24*	*17.71*	*18.17*	*18.64*
Time (minutes)	***41***	***42***	***43***	***44***	***45***	***46***	***47***	***48***	***49***	***50***
UPTD	*19.11*	*19.57*	*20.04*	*20.5*	*20.97*	*21.44*	*21.9*	*22.37*	*22.83*	*23.3*
Time (minutes)	***51***	***52***	***53***	***54***	***55***	***56***	***57***	***58***	***59***	***60***
UPTD	*23.77*	*24.23*	*24.7*	*25.16*	*25.63*	*26.1*	*26.56*	*27.03*	*27.49*	*27.96*

Unit Pulmonary Toxic Dose - PP oxygen 0.8 ata

Time (minutes)	***1***	***2***	***3***	***4***	***5***	***6***	***7***	***8***	***9***	***10***
UPTD	*0.65*	*1.31*	*1.96*	*2.61*	*3.27*	*3.92*	*4.57*	*5.23*	*5.88*	*6.53*
Time (minutes)	***11***	***12***	***13***	***14***	***15***	***16***	***17***	***18***	***19***	***20***
UPTD	*7.19*	*7.84*	*8.49*	*9.15*	*9.8*	*10.45*	*11.11*	*11.76*	*12.41*	*13.07*
Time (minutes)	***21***	***22***	***23***	***24***	***25***	***26***	***27***	***28***	***29***	***30***
UPTD	*13.72*	*14.37*	*15.03*	*15.68*	*16.33*	*19.99*	*17.64*	*18.29*	*18.95*	*19.6*
Time (minutes)	***31***	***32***	***33***	***34***	***35***	***36***	***37***	***38***	***39***	***40***
UPTD	*20.25*	*20.91*	*21.56*	*22.21*	*22.87*	*23.52*	*24.17*	*24.83*	*25.48*	*26.13*
Time (minutes)	***41***	***42***	***43***	***44***	***45***	***46***	***47***	***48***	***49***	***50***
UPTD	*26.79*	*27.44*	*28.09*	*28.75*	*29.4*	*30.05*	*30.71*	*31.36*	*32.01*	*32.67*
Time (minutes)	***51***	***52***	***53***	***54***	***55***	***56***	***57***	***58***	***59***	***60***
UPTD	*33.32*	*33.97*	*34.63*	*35.28*	*35.93*	*36.59*	*37.24*	*37.89*	*38.55*	*39.2*

Unit Pulmonary Toxic Dose - PP oxygen 0.9 ata

Time (minutes)	***1***	***2***	***3***	***4***	***5***	***6***	***7***	***8***	***9***	***10***
UPTD	*0.83*	*1.66*	*2.49*	*3.32*	*4.15*	*4.98*	*5.81*	*6.64*	*7.47*	*8.3*
Time (minutes)	***11***	***12***	***13***	***14***	***15***	***16***	***17***	***18***	***19***	***20***
UPTD	*9.13*	*9.96*	*10.79*	*11.62*	*12.45*	*13.29*	*14.12*	*14.95*	*15.78*	*16.61*
Time (minutes)	***21***	***22***	***23***	***24***	***25***	***26***	***27***	***28***	***29***	***30***
UPTD	*17.44*	*18.27*	*19.1*	*19.93*	*20.76*	*21.59*	*22.42*	*23.25*	*24.08*	*24.91*
Time (minutes)	***31***	***32***	***33***	***34***	***35***	***36***	***37***	***38***	***39***	***40***
UPTD	*25.74*	*26.57*	*27.4*	*28.23*	*29.06*	*29.89*	*30.72*	*31.55*	*32.38*	*33.21*
Time (minutes)	***41***	***42***	***43***	***44***	***45***	***46***	***47***	***48***	***49***	***50***
UPTD	*34.04*	*34.87*	*35.7*	*36.53*	*37.36*	*38.19*	*39.02*	*39.86*	*40.69*	*41.52*
Time (minutes)	***51***	***52***	***53***	***54***	***55***	***56***	***57***	***58***	***59***	***60***
UPTD	*42.35*	*43.18*	*44.01*	*44.84*	*45.67*	*46.5*	*47.33*	*48.16*	*48.99*	*49.82*

Unit Pulmonary Toxic Dose - PP oxygen 1.1 ata

Time (minutes)	***1***	***2***	***3***	***4***	***5***	***6***	***7***	***8***	***9***	***10***
UPTD	*1.16*	*2.33*	*3.49*	*4.66*	*5.82*	*6.98*	*8.15*	*9.31*	*10.48*	*11.64*
Time (minutes)	***11***	***12***	***13***	***14***	***15***	***16***	***17***	***18***	***19***	***20***
UPTD	*12.8*	*13.97*	*15.13*	*16.3*	*17.46*	*18.63*	*19.79*	*20.95*	*22.12*	*23.28*
Time (minutes)	***21***	***22***	***23***	***24***	***25***	***26***	***27***	***28***	***29***	***30***
UPTD	*24.45*	*25.61*	*26.77*	*27.94*	*29.1*	*30.27*	*31.43*	*32.59*	*33.76*	*34.92*
Time (minutes)	***31***	***32***	***33***	***34***	***35***	***36***	***37***	***38***	***39***	***40***
UPTD	*36.09*	*37.25*	*38.41*	*39.58*	*40.74*	*41.91*	*43.07*	*44.24*	*45.4*	*46.56*
Time (minutes)	***41***	***42***	***43***	***44***	***45***	***46***	***47***	***48***	***49***	***50***
UPTD	*47.73*	*48.89*	*50.06*	*51.22*	*52.38*	*53.55*	*54.71*	*55.88*	*57.04*	*58.2*
Time (minutes)	***51***	***52***	***53***	***54***	***55***	***56***	***57***	***58***	***59***	***60***
UPTD	*59.37*	*60.53*	*61.7*	*62.86*	*64.02*	*65.19*	*66.35*	*67.52*	*68.68*	*69.85*

Unit Pulmonary Toxic Dose - PP oxygen 1.2 ata

Time (minutes)	***1***	***2***	***3***	***4***	***5***	***6***	***7***	***8***	***9***	***10***
UPTD	*1.32*	*2.65*	*3.97*	*5.29*	*6.62*	*7.94*	*9.27*	*10.59*	*11.91*	*13.24*
Time (minutes)	***11***	***12***	***13***	***14***	***15***	***16***	***17***	***18***	***19***	***20***
UPTD	*14.56*	*15.88*	*17.21*	*18.53*	*19.85*	*21.28*	*22.5*	*23.83*	*25.15*	*26.47*
Time (minutes)	***21***	***22***	***23***	***24***	***25***	***26***	***27***	***28***	***29***	***30***
UPTD	*27.8*	*29.12*	*30.44*	*31.77*	*33.09*	*34.41*	*35.74*	*37.06*	*38.39*	*39.71*
Time (minutes)	***31***	***32***	***33***	***34***	***35***	***36***	***37***	***38***	***39***	***40***
UPTD	*41.03*	*42.36*	*43.68*	*45*	*46.33*	*47.65*	*48.98*	*50.3*	*51.62*	*52.95*
Time (minutes)	***41***	***42***	***43***	***44***	***45***	***46***	***47***	***48***	***49***	***50***
UPTD	*54.27*	*55.59*	*56.92*	*58.24*	*59.56*	*60.89*	*62.21*	*63.54*	*64.86*	*66.18*
Time (minutes)	***51***	***52***	***53***	***54***	***55***	***56***	***57***	***58***	***59***	***60***
UPTD	*67.51*	*68.83*	*70.15*	*71.48*	*72.8*	*74.12*	*75.45*	*76.77*	*78.1*	*79.42*

Unit Pulmonary Toxic Dose - PP oxygen 1.3 ata

Time (minutes)	***1***	***2***	***3***	***4***	***5***	***6***	***7***	***8***	***9***	***10***
UPTD	*1.43*	*2.96*	*4.44*	*5.92*	*7.4*	*8.88*	*10.36*	*11.84*	*13.32*	*14.79*
Time (minutes)	***11***	***12***	***13***	***14***	***15***	***16***	***17***	***18***	***19***	***20***
UPTD	*16.27*	*17.75*	*19.23*	*20.71*	*22.19*	*23.67*	*25.15*	*26.63*	*28.11*	*29.59*
Time (minutes)	***21***	***22***	***23***	***24***	***25***	***26***	***27***	***28***	***29***	***30***
UPTD	*31.07*	*32.55*	*34.03*	*35.51*	*36.99*	*38.47*	*39.95*	*41.42*	*42.9*	*44.38*
Time (minutes)	***31***	***32***	***33***	***34***	***35***	***36***	***37***	***38***	***39***	***40***
UPTD	*45.86*	*47.34*	*48.82*	*50.3*	*51.78*	*53.26*	*54.74*	*56.22*	*57.7*	*59.18*
Time (minutes)	***41***	***42***	***43***	***44***	***45***	***46***	***47***	***48***	***49***	***50***
UPTD	*60.66*	*62.16*	*63.62*	*65.1*	*66.58*	*68.05*	*69.53*	*71.01*	*72.49*	*73.97*
Time (minutes)	***51***	***52***	***53***	***54***	***55***	***56***	***57***	***58***	***59***	***60***
UPTD	*75.45*	*76.93*	*78.41*	*79.89*	*81.37*	*82.85*	*84.33*	*85.81*	*87.29*	*88.77*

Unit Pulmonary Toxic Dose - PP oxygen 1.4 ata

Time (minutes)	***1***	***2***	***3***	***4***	***5***	***6***	***7***	***8***	***9***	***10***
UPTD	*1.63*	*3.26*	*4.9*	*6.53*	*8.16*	*9.79*	*11.42*	*13.06*	*14.69*	*16.32*
Time (minutes)	***11***	***12***	***13***	***14***	***15***	***16***	***17***	***18***	***19***	***20***
UPTD	*17.95*	*19.58*	*21.22*	*22.85*	*24.48*	*26.11*	*27.74*	*29.38*	*31.01*	*32.64*
Time (minutes)	***21***	***22***	***23***	***24***	***25***	***26***	***27***	***28***	***29***	***30***
UPTD	*34.27*	*35.9*	*37.54*	*39.17*	*40.8*	*42.43*	*44.06*	*45.7*	*47.33*	*48.96*
Time (minutes)	***31***	***32***	***33***	***34***	***35***	***36***	***37***	***38***	***39***	***40***
UPTD	*50.59*	*52.22*	*53.86*	*55.49*	*57.12*	*58.75*	*60.38*	*62.02*	*63.65*	*65.28*
Time (minutes)	***41***	***42***	***43***	***44***	***45***	***46***	***47***	***48***	***49***	***50***
UPTD	*66.41*	*68.55*	*70.18*	*71.81*	*73.44*	*75.07*	*76.71*	*78.34*	*79.97*	*81.6*
Time (minutes)	***51***	***52***	***53***	***54***	***55***	***56***	***57***	***58***	***59***	***60***
UPTD	*83.23*	*84.87*	*86.5*	*88.13*	*89.76*	*91.39*	*93.03*	*94.66*	*96.29*	*97.92*

Unit Pulmonary Toxic Dose - PP oxygen 1.5 ata

Time (minutes)	***1***	***2***	***3***	***4***	***5***	***6***	***7***	***8***	***9***	***10***
UPTD	*1.78*	*3.56*	*5.35*	*7.13*	*8.91*	*10.69*	*12.47*	*14.25*	*16.04*	*17.82*
Time (minutes)	***11***	***12***	***13***	***14***	***15***	***16***	***17***	***18***	***19***	***20***
UPTD	*19.6*	*21.38*	*23.16*	*24.95*	*26.73*	*28.51*	*30.29*	*32.07*	*33.85*	*35.64*
Time (minutes)	***21***	***22***	***23***	***24***	***25***	***26***	***27***	***28***	***29***	***30***
UPTD	*37.42*	*39.2*	*40.98*	*42.76*	*44.54*	*46.33*	*48.11*	*49.89*	*51.67*	*53.45*
Time (minutes)	***31***	***32***	***33***	***34***	***35***	***36***	***37***	***38***	***39***	***40***
UPTD	*55.24*	*57.02*	*58.8*	*60.58*	*62.36*	*64.14*	*65.93*	*67.71*	*69.49*	*71.27*
Time (minutes)	***41***	***42***	***43***	***44***	***45***	***46***	***47***	***48***	***49***	***50***
UPTD	*73.05*	*74.84*	*76.62*	*78.4*	*80.18*	*81.96*	*83.74*	*85.53*	*87.31*	*89.09*
Time (minutes)	***51***	***52***	***53***	***54***	***55***	***56***	***57***	***58***	***59***	***60***
UPTD	*90.87*	*92.65*	*94.44*	*96.22*	*98*	*99.78*	*101.56*	*103.34*	*105.13*	*106.91*

Unit Pulmonary Toxic Dose - PP oxygen 1.6 ata

Time (minutes)	***1***	***2***	***3***	***4***	***5***	***6***	***7***	***8***	***9***	***10***
UPTD	*1.93*	*3.86*	*5.79*	*7.72*	*9.65*	*11.57*	*13.5*	*15.43*	*17.36*	*19.29*
Time (minutes)	***11***	***12***	***13***	***14***	***15***	***16***	***17***	***18***	***19***	***20***
UPTD	*21.22*	*23.15*	*25.08*	*27.01*	*28.94*	*30.87*	*32.79*	*34.72*	*36.65*	*38.58*
Time (minutes)	***21***	***22***	***23***	***24***	***25***	***26***	***27***	***28***	***29***	***30***
UPTD	*40.51*	*42.44*	*44.37*	*46.3*	*48.23*	*50.16*	*52.09*	*54.01*	*55.94*	*57.87*
Time (minutes)	***31***	***32***	***33***	***34***	***35***	***36***	***37***	***38***	***39***	***40***
UPTD	*59.8*	*61.73*	*63.66*	*65.59*	*67.52*	*69.45*	*71.38*	*73.31*	*75.23*	*77.16*
Time (minutes)	***41***	***42***	***43***	***44***	***45***	***46***	***47***	***48***	***49***	***50***
UPTD	*79.09*	*81.02*	*82.95*	*84.88*	*86.81*	*88.74*	*90.67*	*92.6*	*94.53*	*96.45*
Time (minutes)	***51***	***52***	***53***	***54***	***55***	***56***	***57***	***58***	***59***	***60***
UPTD	*98.38*	*100.31*	*102.24*	*104.17*	*106.1*	*108.03*	*109.96*	*111.89*	*113.82*	*115.75*

Unit Pulmonary Toxic Dose - PP oxygen 1.7 ata

Time (minutes)	***1***	***2***	***3***	***4***	***5***	***6***	***7***	***8***	***9***	***10***
UPTD	*2.07*	*4.15*	*6.22*	*8.3*	*10.37*	*12.44*	*14.52*	*16.59*	*18.67*	*20.74*
Time (minutes)	***11***	***12***	***13***	***14***	***15***	***16***	***17***	***18***	***19***	***20***
UPTD	*22.82*	*24.89*	*26.96*	*29.04*	*31.11*	*33.19*	*35.26*	*37.33*	*39.41*	*41.48*
Time (minutes)	***21***	***22***	***23***	***24***	***25***	***26***	***27***	***28***	***29***	***30***
UPTD	*43.56*	*45.63*	*47.71*	*49.78*	*51.85*	*53.93*	*56*	*58.06*	*60.15*	*62.22*
Time (minutes)	***31***	***32***	***33***	***34***	***35***	***36***	***37***	***38***	***39***	***40***
UPTD	*64.3*	*66.37*	*68.45*	*70.52*	*72.6*	*74.67*	*76.74*	*78.82*	*80.89*	*82.97*
Time (minutes)	***41***	***42***	***43***	***44***	***45***	***46***	***47***	***48***	***49***	***50***
UPTD	*85.04*	*87.11*	*89.19*	*91.26*	*93.34*	*95.41*	*97.49*	*99.56*	*101.63*	*103.71*
Time (minutes)	***51***	***52***	***53***	***54***	***55***	***56***	***57***	***58***	***59***	***60***
UPTD	*105.78*	*107.86*	*109.93*	*112*	*114.08*	*116.15*	*118.23*	*120.3*	*122.38*	*124.45*

Unit Pulmonary Toxic Dose - PP oxygen 1.8 ata

Time (minutes)	***1***	***2***	***3***	***4***	***5***	***6***	***7***	***8***	***9***	***10***
UPTD	*2.22*	*4.43*	*6.65*	*8.87*	*11.09*	*13.3*	*15.52*	*17.74*	*19.96*	*22.17*
Time (minutes)	***11***	***12***	***13***	***14***	***15***	***16***	***17***	***18***	***19***	***20***
UPTD	*24.39*	*26.61*	*28.82*	*31.04*	*33.26*	*35.48*	*37.69*	*39.91*	*42.13*	*44.34*
Time (minutes)	***21***	***22***	***23***	***24***	***25***	***26***	***27***	***28***	***29***	***30***
UPTD	*46.56*	*48.78*	*51*	*53.21*	*55.43*	*57.65*	*59.87*	*62.08*	*64.3*	*66.52*
Time (minutes)	***31***	***32***	***33***	***34***	***35***	***36***	***37***	***38***	***39***	***40***
UPTD	*68.73*	*70.95*	*73.17*	*75.39*	*77.6*	*79.82*	*82.04*	*84.25*	*86.47*	*88.69*
Time (minutes)	***41***	***42***	***43***	***44***	***45***	***46***	***47***	***48***	***49***	***50***
UPTD	*90.91*	*93.12*	*95.34*	*97.56*	*99.78*	*101.99*	*104.21*	*106.43*	*108.64*	*110.86*
Time (minutes)	***51***	***52***	***53***	***54***	***55***	***56***	***57***	***58***	***59***	***60***
UPTD	*113.08*	*115.3*	*117.51*	*119.73*	*121.95*	*124.16*	*126.38*	*128.6*	*130.82*	*133.03*

Unit Pulmonary Toxic Dose - PP oxygen 1.9 ata

Time (minutes)	***1***	***2***	***3***	***4***	***5***	***6***	***7***	***8***	***9***	***10***
UPTD	*2.36*	*4.72*	*7.08*	*9.43*	*11.79*	*14.15*	*16.51*	*18.87*	*21*	*23.58*
Time (minutes)	***11***	***12***	***13***	***14***	***15***	***16***	***17***	***18***	***19***	***20***
UPTD	*25.94*	*28.30*	*30.66*	*33.02*	*35.38*	*37.74*	*40.09*	*42.45*	*44.81*	*47.17*
Time (minutes)	***21***	***22***	***23***	***24***	***25***	***26***	***27***	***28***	***29***	***30***
UPTD	*49.53*	*51.89*	*54.24*	*56.6*	*58.96*	*61.32*	*63.68*	*65.04*	*68.4*	*70.75*
Time (minutes)	***31***	***32***	***33***	***34***	***35***	***36***	***37***	***38***	***39***	***40***
UPTD	*73.11*	*75.47*	*77.83*	*80.19*	*82.55*	*84.91*	*87.26*	*89.62*	*91.98*	*94.34*
Time (minutes)	***41***	***42***	***43***	***44***	***45***	***46***	***47***	***48***	***49***	***50***
UPTD	*96.7*	*99.06*	*101.41*	*103.77*	*106.13*	*108.49*	*110.85*	*113.21*	*115.57*	*117.92*
Time (minutes)	***51***	***52***	***53***	***54***	***55***	***56***	***57***	***58***	***59***	***60***
UPTD	*120.28*	*122.64*	*125*	*127.36*	*129.72*	*132.07*	*134.43*	*136.79*	*139.15*	*141.51*

Unit Pulmonary Toxic Dose - PP oxygen 2.0 ata

Time (minutes)	***1***	***2***	***3***	***4***	***5***	***6***	***7***	***8***	***9***	***10***
UPTD	*2.5*	*5*	*7.49*	*9.99*	*12.49*	*14.99*	*17.49*	*19.98*	*22.48*	*24.98*
Time (minutes)	***11***	***12***	***13***	***14***	***15***	***16***	***17***	***18***	***19***	***20***
UPTD	*27.48*	*29.98*	*32.47*	*34.97*	*37.47*	*39.97*	*42.47*	*44.96*	*47.46*	*49.96*
Time (minutes)	***21***	***22***	***23***	***24***	***25***	***26***	***27***	***28***	***29***	***30***
UPTD	*52.46*	*54.96*	*57.46*	*59.96*	*62.46*	*64.96*	*67.46*	*69.96*	*72.46*	*74.94*
Time (minutes)	***31***	***32***	***33***	***34***	***35***	***36***	***37***	***38***	***39***	***40***
UPTD	*77.44*	*79.94*	*82.44*	*84.93*	*87.43*	*89.93*	*92.43*	*94.93*	*97.42*	*99.92*
Time (minutes)	***41***	***42***	***43***	***44***	***45***	***46***	***47***	***48***	***49***	***50***
UPTD	*102.42*	*104.92*	*107.42*	*109.91*	*112.41*	*114.91*	*117.41*	*119.91*	*122.4*	*124.9*
Time (minutes)	***51***	***52***	***53***	***54***	***55***	***56***	***57***	***58***	***59***	***60***
UPTD	*127.4*	*129.9*	*132.4*	*134.89*	*137.39*	*139.89*	*142.39*	*144.89*	*147.38*	*149.88*

Unit Pulmonary Toxic Dose - PP oxygen 2.1 ata

Time (minutes)	***1***	***2***	***3***	***4***	***5***	***6***	***7***	***8***	***9***	***10***
UPTD	*2.64*	*5.27*	*7.91*	*10.54*	*13.18*	*15.82*	*18.45*	*21.09*	*23.72*	*26.36*
Time (minutes)	***11***	***12***	***13***	***14***	***15***	***16***	***17***	***18***	***19***	***20***
UPTD	*29*	*31.63*	*34.27*	*36.91*	*39.54*	*42.18*	*44.81*	*47.45*	*50.09*	*52.72*
Time (minutes)	***21***	***22***	***23***	***24***	***25***	***26***	***27***	***28***	***29***	***30***
UPTD	*55.36*	*57.99*	*60.63*	*63.27*	*65.9*	*68.54*	*71.17*	*73.81*	*76.45*	*79.08*
Time (minutes)	***31***	***32***	***33***	***34***	***35***	***36***	***37***	***38***	***39***	***40***
UPTD	*81.72*	*84.35*	*86.99*	*89.63*	*92.26*	*94.9*	*97.53*	*100.17*	*102.81*	*105.44*
Time (minutes)	***41***	***42***	***43***	***44***	***45***	***46***	***47***	***48***	***49***	***50***
UPTD	*108.08*	*110.72*	*113.35*	*115.99*	*118.62*	*121.26*	*123.9*	*126.53*	*129.17*	*131.8*
Time (minutes)	***51***	***52***	***53***	***54***	***55***	***56***	***57***	***58***	***59***	***60***
UPTD	*134.44*	*137.08*	*139.71*	*142.35*	*144.98*	*147.62*	*150.26*	*152.89*	*155.53*	*158.16*

Unit Pulmonary Toxic Dose - PP oxygen 2.2 ata

Time (minutes)	***1***	***2***	***3***	***4***	***5***	***6***	***7***	***8***	***9***	***10***
UPTD	*2.77*	*5.55*	*8.32*	*11.09*	*13.86*	*16.64*	*19.41*	*22.18*	*24.95*	*27.73*
Time (minutes)	***11***	***12***	***13***	***14***	***15***	***16***	***17***	***18***	***19***	***20***
UPTD	*30.5*	*33.27*	*36.04*	*38.82*	*41.59*	*44.36*	*47.14*	*49.91*	*52.68*	*55.45*
Time (minutes)	***21***	***22***	***23***	***24***	***25***	***26***	***27***	***28***	***29***	***30***
UPTD	*58.23*	*61*	*63.77*	*66.54*	*69.32*	*72.09*	*74.86*	*77.63*	*80.41*	*83.18*
Time (minutes)	***31***	***32***	***33***	***34***	***35***	***36***	***37***	***38***	***39***	***40***
UPTD	*85.95*	*88.73*	*91.5*	*94.27*	*97.04*	*99.82*	*102.59*	*105.36*	*108.13*	*110.91*
Time (minutes)	***41***	***42***	***43***	***44***	***45***	***46***	***47***	***48***	***49***	***50***
UPTD	*113.68*	*116.45*	*119.23*	*122*	*124.77*	*127.54*	*130.32*	*133.09*	*135.86*	*138.63*
Time (minutes)	***51***	***52***	***53***	***54***	***55***	***56***	***57***	***58***	***59***	***60***
UPTD	*141.41*	*144.18*	*146.45*	*149.72*	*152.5*	*155.27*	*158.04*	*160.82*	*163.59*	*166.36*

Unit Pulmonary Toxic Dose - PP oxygen 2.3 ata

Time (minutes)	***1***	***2***	***3***	***4***	***5***	***6***	***7***	***8***	***9***	***10***
UPTD	*2.91*	*5.82*	*8.72*	*11.62*	*14.52*	*17.42*	*20.32*	*23.22*	*26.12*	*29.02*
Time (minutes)	***11***	***12***	***13***	***14***	***15***	***16***	***17***	***18***	***19***	***20***
UPTD	*31.99*	*34.9*	*37.8*	*40.71*	*43.62*	*46.53*	*49.43*	*52.34*	*55.25*	*58.16*
Time (minutes)	***21***	***22***	***23***	***24***	***25***	***26***	***27***	***28***	***29***	***30***
UPTD	*61.07*	*63.97*	*66.88*	*69.79*	*72.7*	*75.61*	*78.51*	*81.42*	*84.33*	*87.24*
Time (minutes)	***31***	***32***	***33***	***34***	***35***	***36***	***37***	***38***	***39***	***40***
UPTD	*90.15*	*93.05*	*95.96*	*98.87*	*101.78*	*104.69*	*107.59*	*110.5*	*113.41*	*116.32*
Time (minutes)	***41***	***42***	***43***	***44***	***45***	***46***	***47***	***48***	***49***	***50***
UPTD	*119.23*	*122.13*	*125.04*	*127.95*	*130.86*	*133.77*	*136.67*	*139.58*	*142.49*	*145.4*
Time (minutes)	***51***	***52***	***53***	***54***	***55***	***56***	***57***	***58***	***59***	***60***
UPTD	*148.3*	*151.21*	*154.12*	*157.03*	*159.94*	*162.84*	*165.75*	*168.66*	*171.57*	*174.48*

Unit Pulmonary Toxic Dose - PP oxygen 2.4 ata

Time (minutes)	***1***	***2***	***3***	***4***	***5***	***6***	***7***	***8***	***9***	***10***
UPTD	*3.04*	*6.08*	*9.13*	*12.17*	*15.21*	*18.25*	*21.29*	*24.34*	*27.38*	*30.42*
Time (minutes)	***11***	***12***	***13***	***14***	***15***	***16***	***17***	***18***	***19***	***20***
UPTD	*33.46*	*36.5*	*39.55*	*42.59*	*45.63*	*48.67*	*51.71*	*54.76*	*57.8*	*60.84*
Time (minutes)	***21***	***22***	***23***	***24***	***25***	***26***	***27***	***28***	***29***	***30***
UPTD	*63.88*	*66.92*	*69.96*	*73.01*	*76.05*	*79.09*	*82.13*	*85.17*	*88.22*	*91.26*
Time (minutes)	***31***	***32***	***33***	***34***	***35***	***36***	***37***	***38***	***39***	***40***
UPTD	*94.3*	*97.34*	*100.38*	*103.38*	*106.43*	*109.47*	*112.55*	*115.59*	*118.64*	*121.68*
Time (minutes)	***41***	***42***	***43***	***44***	***45***	***46***	***47***	***48***	***49***	***50***
UPTD	*124.72*	*127.76*	*130.8*	*133.85*	*136.89*	*139.93*	*142.97*	*146.01*	*149.06*	*152.1*
Time (minutes)	***51***	***52***	***53***	***54***	***55***	***56***	***57***	***58***	***59***	***60***
UPTD	*155.14*	*158.18*	*161.22*	*164.27*	*167.31*	*170.35*	*173.39*	*176.43*	*179.48*	*182.52*

Unit Pulmonary Toxic Dose - PP oxygen 2.5 ata

Time (minutes)	***1***	***2***	***3***	***4***	***5***	***6***	***7***	***8***	***9***	***10***
UPTD	*3.17*	*6.35*	*9.52*	*12.7*	*15.87*	*19.05*	*22.22*	*25.4*	*28.57*	*31.75*
Time (minutes)	***11***	***12***	***13***	***14***	***15***	***16***	***17***	***18***	***19***	***20***
UPTD	*34.92*	*38.1*	*41.27*	*44.45*	*47.62*	*50.8*	*53.97*	*57.15*	*60.32*	*63.5*
Time (minutes)	***21***	***22***	***23***	***24***	***25***	***26***	***27***	***28***	***29***	***30***
UPTD	*66.67*	*69.87*	*73.02*	*76.2*	*79.37*	*82.54*	*85.72*	*88.89*	*92.07*	*95.24*
Time (minutes)	***31***	***32***	***33***	***34***	***35***	***36***	***37***	***38***	***39***	***40***
UPTD	*98.42*	*101.59*	*104.77*	*107.94*	*111.12*	*114.29*	*117.47*	*120.64*	*123.82*	*126.99*
Time (minutes)	***41***	***42***	***43***	***44***	***45***	***46***	***47***	***48***	***49***	***50***
UPTD	*130.17*	*133.34*	*136.52*	*139.69*	*142.87*	*146.04*	*149.22*	*152.39*	*155.57*	*158.74*
Time (minutes)	***51***	***52***	***53***	***54***	***55***	***56***	***57***	***58***	***59***	***60***
UPTD	*161.91*	*165.09*	*168.26*	*171.44*	*174.61*	*177.79*	*180.96*	*184.14*	*187.31*	*190.49*

Unit Pulmonary Toxic Dose - PP oxygen 2.6 ata

Time (minutes)	***1***	***2***	***3***	***4***	***5***	***6***	***7***	***8***	***9***	***10***
UPTD	*3.31*	*6.61*	*9.92*	*13.23*	*16.53*	*19.84*	*23.15*	*26.45*	*29.76*	*33.07*
Time (minutes)	***11***	***12***	***13***	***14***	***15***	***16***	***17***	***18***	***19***	***20***
UPTD	*36.37*	*39.68*	*42.99*	*46.29*	*49.6*	*52.9*	*56.21*	*59.52*	*62.82*	*66.13*
Time (minutes)	***21***	***22***	***23***	***24***	***25***	***26***	***27***	***28***	***29***	***30***
UPTD	*69.44*	*72.74*	*76.05*	*79.36*	*82.66*	*85.97*	*89.28*	*92.58*	*95.89*	*99.2*
Time (minutes)	***31***	***32***	***33***	***34***	***35***	***36***	***37***	***38***	***39***	***40***
UPTD	*102.5*	*105.81*	*109.12*	*112.42*	*115.73*	*119.04*	*122.34*	*125.65*	*128.96*	*132.26*
Time (minutes)	***41***	***42***	***43***	***44***	***45***	***46***	***47***	***48***	***49***	***50***
UPTD	*135.57*	*138.87*	*142.18*	*145.49*	*148.79*	*152.1*	*155.41*	*158.71*	*162.02*	*165.33*
Time (minutes)	***51***	***52***	***53***	***54***	***55***	***56***	***57***	***58***	***59***	***60***
UPTD	*168.63*	*171.94*	*175.25*	*178.55*	*181.86*	*185.17*	*188.47*	*191.78*	*195.09*	*198.39*

Unit Pulmonary Toxic Dose - PP oxygen 2.7 ata

Time (minutes)	***1***	***2***	***3***	***4***	***5***	***6***	***7***	***8***	***9***	***10***
UPTD	*3.44*	*6.87*	*10.31*	*13.75*	*17.19*	*20.62*	*24.06*	*27.5*	*30.94*	*34.37*
Time (minutes)	***11***	***12***	***13***	***14***	***15***	***16***	***17***	***18***	***19***	***20***
UPTD	*37.81*	*41.25*	*44.68*	*48.12*	*51.56*	*55*	*58.43*	*61.87*	*65.31*	*68.74*
Time (minutes)	***21***	***22***	***23***	***24***	***25***	***26***	***27***	***28***	***29***	***30***
UPTD	*72.18*	*75.62*	*79.06*	*82.49*	*85.93*	*89.37*	*92.81*	*96.24*	*99.68*	*103.12*
Time (minutes)	***31***	***32***	***33***	***34***	***35***	***36***	***37***	***38***	***39***	***40***
UPTD	*106.55*	*109.99*	*113.43*	*116.87*	*120.3*	*123.74*	*127.18*	*130.62*	*134.05*	*137.49*
Time (minutes)	***41***	***42***	***43***	***44***	***45***	***46***	***47***	***48***	***49***	***50***
UPTD	*140.93*	*144.36*	*147.8*	*151.24*	*154.68*	*158.11*	*161.55*	*164.99*	*168.43*	*171.86*
Time (minutes)	***51***	***52***	***53***	***54***	***55***	***56***	***57***	***58***	***59***	***60***
UPTD	*175.3*	*178.74*	*182.17*	*185.61*	*189.05*	*192.49*	*195.92*	*199.36*	*202.8*	*206.23*

Unit Pulmonary Toxic Dose - PP oxygen 2.8 ata

Time (minutes)	***1***	***2***	***3***	***4***	***5***	***6***	***7***	***8***	***9***	***10***
UPTD	*3.57*	*7.13*	*10.7*	*14.27*	*17.83*	*21.4*	*24.97*	*28.54*	*32.1*	*35.67*
Time (minutes)	***11***	***12***	***13***	***14***	***15***	***16***	***17***	***18***	***19***	***20***
UPTD	*39.24*	*42.8*	*46.37*	*49.94*	*53.5*	*57.07*	*60.64*	*64.21*	*67.77*	*71.34*
Time (minutes)	***21***	***22***	***23***	***24***	***25***	***26***	***27***	***28***	***29***	***30***
UPTD	*74.91*	*78.47*	*82.04*	*85.61*	*89.17*	*92.74*	*96.31*	*99.87*	*103.44*	*107.01*
Time (minutes)	***31***	***32***	***33***	***34***	***35***	***36***	***37***	***38***	***39***	***40***
UPTD	*110.58*	*114.14*	*117.71*	*121.28*	*124.84*	*128.41*	*131.98*	*135.54*	*139.11*	*142.68*
Time (minutes)	***41***	***42***	***43***	***44***	***45***	***46***	***47***	***48***	***49***	***50***
UPTD	*146.25*	*149.81*	*153.38*	*156.95*	*160.51*	*164.08*	*167.65*	*171.21*	*174.78*	*178.35*
Time (minutes)	***51***	***52***	***53***	***54***	***55***	***56***	***57***	***58***	***59***	***60***
UPTD	*181.91*	*185.48*	*189.05*	*192.62*	*196.18*	*199.75*	*203.32*	*206.88*	*210.45*	*214.02*

Unit Pulmonary Toxic Dose - PP oxygen 2.9 ata

Time (minutes)	***1***	***2***	***3***	***4***	***5***	***6***	***7***	***8***	***9***	***10***
UPTD	*3.7*	*7.39*	*11.09*	*14.79*	*18.48*	*22.17*	*25.87*	*29.57*	*33.26*	*36.96*
Time (minutes)	***11***	***12***	***13***	***14***	***15***	***16***	***17***	***18***	***19***	***20***
UPTD	*40.65*	*44.35*	*48.04*	*51.74*	*55.44*	*59.13*	*62.83*	*66.52*	*70.22*	*73.91*
Time (minutes)	***21***	***22***	***23***	***24***	***25***	***26***	***27***	***28***	***29***	***30***
UPTD	*77.61*	*81.31*	*85*	*88.7*	*92.39*	*96.09*	*99.78*	*103.48*	*107.18*	*110.87*
Time (minutes)	***31***	***32***	***33***	***34***	***35***	***36***	***37***	***38***	***39***	***40***
UPTD	*114.57*	*118.26*	*121.96*	*125.66*	*129.35*	*133.05*	*136.74*	*140.44*	*144.13*	*147.83*
Time (minutes)	***41***	***42***	***43***	***44***	***45***	***46***	***47***	***48***	***49***	***50***
UPTD	*151.53*	*155.22*	*158.92*	*162.61*	*166.31*	*170*	*173.7*	*177.4*	*181.09*	*184.79*
Time (minutes)	***51***	***52***	***53***	***54***	***55***	***56***	***57***	***58***	***59***	***60***
UPTD	*188.43*	*192.18*	*195.87*	*199.57*	*203.27*	*206.96*	*210.66*	*214.35*	*218.05*	*221.74*

Unit Pulmonary Toxic Dose - PP oxygen 3.0 ata

Time (minutes)	***1***	***2***	***3***	***4***	***5***	***6***	***7***	***8***	***9***	***10***
UPTD	*3.82*	*7.65*	*11.47*	*15.29*	*19.12*	*22.94*	*26.77*	*30.59*	*34.41*	*38.24*
Time (minutes)	***11***	***12***	***13***	***14***	***15***	***16***	***17***	***18***	***19***	***20***
UPTD	*42.06*	*45.88*	*49.71*	*53.53*	*57.35*	*61.18*	*65*	*68.83*	*72.65*	*76.47*
Time (minutes)	***21***	***22***	***23***	***24***	***25***	***26***	***27***	***28***	***29***	***30***
UPTD	*80.3*	*84.12*	*87.94*	*91.77*	*95.59*	*99.41*	*103.24*	*107.06*	*110.89*	*114.71*
Time (minutes)	***31***	***32***	***33***	***34***	***35***	***36***	***37***	***38***	***39***	***40***
UPTD	*118.53*	*122.36*	*126.18*	*130*	*133.83*	*137.65*	*141.47*	*145.3*	*149.12*	*152.94*
Time (minutes)	***41***	***42***	***43***	***44***	***45***	***46***	***47***	***48***	***49***	***50***
UPTD	*156.77*	*160.59*	*164.42*	*168.24*	*172.06*	*175.89*	*179.71*	*183.53*	*187.36*	*191.18*
Time (minutes)	***51***	***52***	***53***	***54***	***55***	***56***	***57***	***58***	***59***	***60***
UPTD	*195*	*198.83*	*202.65*	*206.48*	*210.3*	*214.12*	*217.95*	*221.77*	*225.59*	*229.42*

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Many documents that have been used for this handbook are not listed here but can be found on our website bookstore with other papers through this link: https://diving-rov-specialists.com/.

They can also be downloaded from the following sections of the website:

- Scientific documents
- Diving & ROV procedures
- Historical diving documents