

Edmonds' Diving Medicine

DMFSD

DMFSD 2



Free diving medical
text for
Scuba divers
Free Divers
Dive Instructors
Diving Paramedics
Doctors new to Diving

Diving Medicine for Scuba Divers, 3rd ed. 2010

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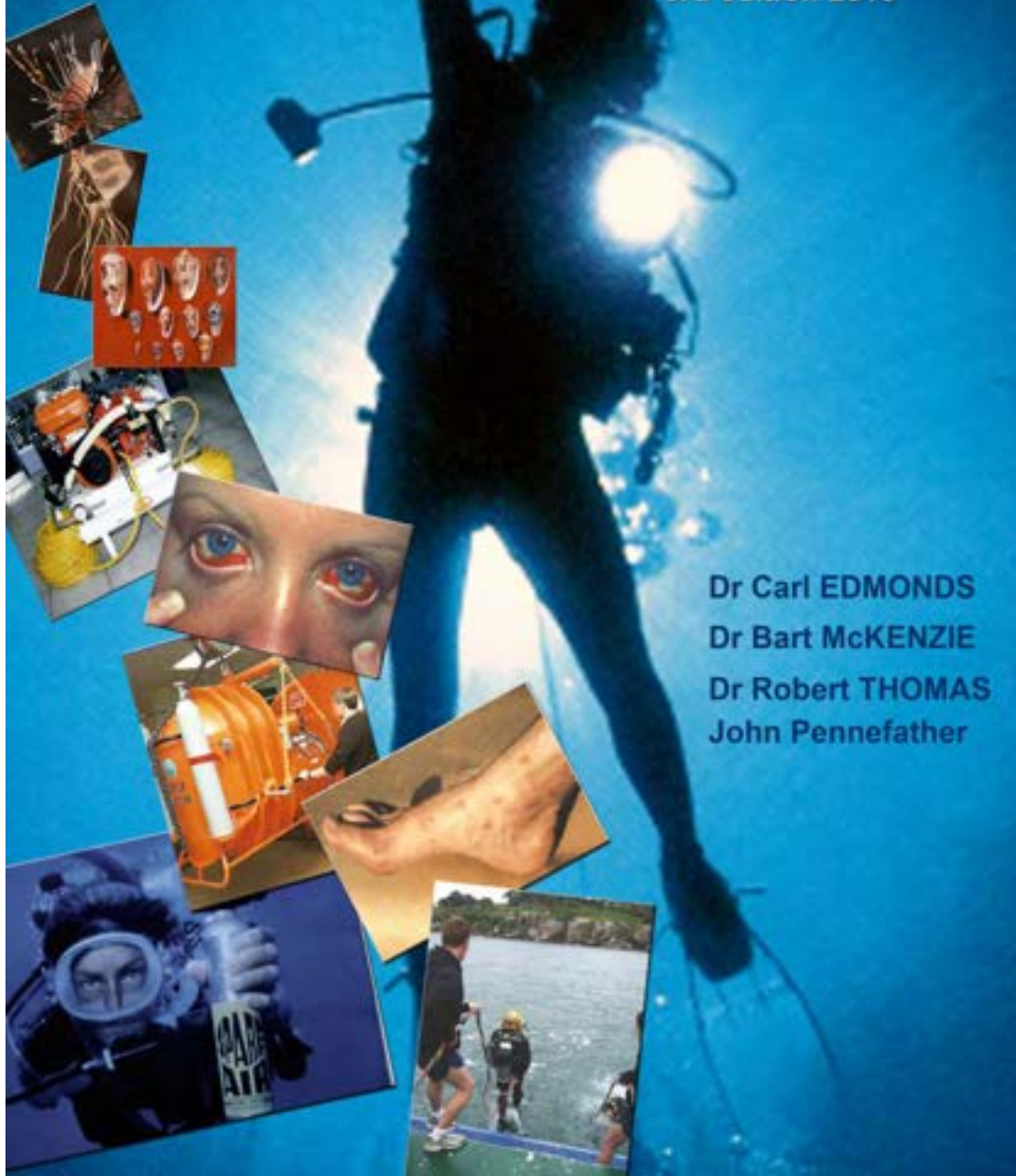
either individual Chapters, or complete text

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Diving Medicine for SCUBA Divers

3rd edition 2010



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Published by Carl Edmonds
Ocean Royale, 11/69-74 North Steyne
Manly, NSW, 2095 Australia
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First edition, October 1992
Second edition, April 1997

National Library of Australia Catalogue

1. Submarine Medicine
2. Scuba Diving Injuries
3. Diving – physiological aspects

Copyright: Carl Edmonds
Title 1 of 1 - Diving Medicine for Scuba Divers
ISBN: [978-0-646-52726-0]

FORWARD

"Diving and Subaquatic Medicine", in its third and highly respected edition, has provided a cornerstone of knowledge for the diving medical professional. Now, "Diving Medicine — for Scuba Divers" is a condensed, simplified and lighter publication for the general diving population. The authors — Drs Edmonds, McKenzie and Thomas, have done an excellent job of providing a comprehensive, useful and up to date resource base for the diver in the field.

The presentation of the material reflects the fact that the authors are experienced divers as well as specialists in diving medicine. Their thinly disguised sense of humour is reflected throughout the text in emphasising important issues and occasionally just lightening the academic loading on the reader. Their treatment of areas of controversy reflects their experience and background in treating diving emergencies. Some readers will find information which may be inconsistent with their teachings. It is strongly suggested that the reader pay attention to the advice that is presented by the authors. Their many years of cumulative experience is reflected in their advice.

This text represents the broadest coverage of diving medical issues that has been focussed upon the general diving public. It does an excellent job of bridging the gap between the "dive rescue" type materials put out by the various agencies, and the other excellent medical texts which focus primarily on physician education. This text should serve the reader well since its intent is clearly designed to stimulate the diving population at large to become better informed on current diving medical issues. Additional resource materials are presented for those who wish more detailed information.

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Addendum:

The success of this text, together with its Japanese and Korean editions, has inspired me to revise and update the material, even though the person I originally designed it for, my young son Mark, is now well into middle age.

The 2nd edition is well over a decade old, and it had run out of print. With the assistance of my esteemed co-worker, John Pennefather, and life long friend, Clarrie Lawler, the 3rd and final edition is now finished.

My intent is that it be made available freely (at least on Internet) to all divers, conscientious dive instructors, paramedics and doctors new to this fascinating and rewarding subject.

Carl Edmonds, 2010

ACKNOWLEDGMENTS

For the 1st and 2nd editions of this text

To my diving buddy and wife, Cindy, for her invaluable criticisms, suggestions and proof-reading. To my son, Mark, who inspired the predecessor of this text, "A Diving Manual for Mark", which I prepared for him. The motivation was to ensure that he had more factual information than that available from his open water scuba diving course. He is now a competent and perceptive diver, partly from his understanding of this text. Thanks also to Patricia Larke and Enid Page for their secretarial services.

Carl Edmonds.

I would like to thank my wife, Ann, for tolerating my on and off affair with a word processor, and also my daughter Deborah, Des Lund, Tammy Lye, Shirley Warner, and Joanne Wright who helped me in the gestation of this book.

Bart McKenzie

This author wishes to gratefully acknowledge the assistance given by my daughter, Natalia, to producing some of the diagrams used within the book. I also wish to thank my wife, Denise, for her tireless support and patience regarding my often frustrating use of the Macintosh computer and noisy printer in completing this text — something she never believed would really ever eventuate!

Bob Thomas

For the 3rd edition,

John Pennefather, who assisted Carl Edmonds in reviewing and updating the text

Clarrie Lawler, who supplied many excellent diagrams

John Lippmann, CEO, DAN Asia-Pacific.

Richard Pyle for technical diving diagrams Cis-Lunar Development
Laboratorie

DIVING MEDICINE FOR SCUBA DIVERS

BACKGROUND

1. History of Diving
2. Physics
3. Physiology
4. Breath-hold Diving
5. Equipment
6. Diving Environments
7. Stress Disorders — Panic and Fatigue
8. Female Divers

SPECIFIC DIVING DISEASES (PRESSURE RELATED DISEASES)

9. Ear Barotrauma
10. Sinus Barotrauma
11. Pulmonary Barotrauma
12. Other Barotraumas
13. Decompression Theory & Physiology
14. Decompression Procedures Tables
15. Decompression Sickness Clinical
16. Decompression Sickness Treatment
17. Dysbaric Osteonecrosis
18. Nitrogen Narcosis
19. HPNS
20. Hypoxia
21. Oxygen Toxicity
22. Carbon Dioxide Problems
23. Carbon Monoxide Toxicity
24. Breathing Gas Contamination

AQUATIC DISEASES

25. Drowning
26. Salt Water Aspiration Syndrome
27. Cold and Hypothermia
28. Infections
29. Dangerous Marine Animals

GENERAL DIVING RELATED MEDICAL PROBLEMS

30. Hearing Loss
31. Disorientation
32. Miscellaneous Disorders –
Pulmonary Oedema,
Contact Lenses
Muscular Cramps
Other Ear Problems
Headache
Sunburn
Seasickness
Jaw Joint Arthritis
Explosions
33. Unconsciousness in Divers
34. Why Divers Die
35. Sudden Death Syndrome
36. Psychological Disorders
37. Drugs and Diving

TREATMENT and PREVENTION

38. Medical Examinations for Divers
39. First Aid Kit
40. Oxygen Therapy Techniques
41. Training and Safety Checklist
42. Resuscitation Review

RECENT DEVELOPMENTS

43. Technical Diving

APPENDICES

- A. Diving Medical Library
- B. Emergency Contact Numbers
- C. In-water O₂ Recompression Therapy
- D. Divers Alert Network

Chapter 1

HISTORY of DIVING

Historians are unable to identify the first divers. Probably the techniques they used were similar to those of the native pearl and sponge divers. They may have used a stone weight to ensure rapid descent, but it is unlikely that they could dive deeper than 30 metres, or spend longer than 2 minutes underwater. Later, diving was employed for military purposes (such as destroying ships anchoring cables, boom defences, etc.) and for salvage work. Divers took part in great naval battles between 1800 BC and 400 BC. Alexander the Great was said to have descended in a diving bell (circa 330 BC) but the details are scarce and some of the stories of the descent are fanciful.

Commercial diving evolved through the 19th and 20th centuries and encompassed salvage and shell diving, extending into exploration, deep diving, off shore oil rigs, aquaculture, ecology and most importantly for you – recreational diving.

The history of diving evolved in two directions. The first is the development of diving equipment – described in this chapter. The second is the understanding of diving physiology and medicine – described in the rest of this text.



Fig. 1.1

A Roman historian, Pliny, recorded the earliest use of surface supplied breathing air by divers in AD77, when a breathing tube connected the diver to the surface. This possibly represents an

early "schnorkel". Its use was limited to very shallow dives, since man's respiratory muscles cannot draw air very far down from the surface – maximum half a metre. It was also depth limited due to the excessive volume of the breathing tube.

Leonardo da Vinci sketched several designs for diving equipment and submarines. Many diagrams of divers' hoods can be found in other historical texts from 1500 AD onwards, but much of this equipment would not have worked at depths greater than a few feet. They did, however, attest to man's desire to remain below the surface for extended periods. In 1680 Borelli, an Italian, designed a diving set which purported to be a self-contained diving apparatus. Although it was impracticable, the idea was revolutionary at that time. Despite the fact that much diving equipment was primitive and rarely functioned adequately, diving bells were used with success from the 17th century onwards.



Fig. 1.2

Diving with a helmet (the equivalent of an upturned bucket which enclosed the diver's head) gradually became an accepted method. It contained air that was pumped down from the surface following the development of efficient air pumps around 1800 AD. Bellows were used to force air down to the divers. This allowed longer and deeper dives and brought to light the many physiological problems caused by the undersea environment.



Fig. 1.3
Siebe's helmet.

In 1837 Augustus Siebe marketed the first effective standard diving dress. This incorporated an air-supply line connecting a pump or compressor on the surface to a diving helmet. The helmet was attached by an airtight seal to a flexible suit that enclosed the diver and was filled with air.



Fig 1.4

Rouquayrol and Denayrouze, Diving suit 1863

The development of **self-contained** air supplies was impeded by the lack of sufficiently powerful compressors and reservoirs. In 1863 the Frenchmen, Rouquayrol and Denayrouze, invented the first satisfactory demand regulator for self-contained underwater breathing apparatus (SCUBA), but due to lack of suitable high pressure air compressors and cylinders, it was limited to surface air supply lines.

In 1878 H. A. Fleuss made a workable self-contained (closed-circuit) oxygen breathing apparatus utilising caustic potash to remove exhaled carbon dioxide. "Closed" refers to the absence of an outlet for gas (i.e. no bubbles) and means that the exhaled gas is rebreathed. This was the forerunner of modern closed-circuit diving units.

Divers in the late 1800's were capable of reaching depths in excess of 50 metres, but the effects of decompression sickness (or bends) caused much concern and many injuries to divers. Paul Bert, a French scientist, was the first to explain that the disease was caused by the formation of nitrogen bubbles in the body and proposed the idea of a slow ascent to the surface. It was not until the early 20th century that Dr J. S. Haldane derived satisfactory mathematical decompression tables to overcome this physiological problem of deep diving. The first successful tables were based on the assumption that decompression sickness could be avoided by not exceeding a 2:1 pressure reduction between stops. It reflected a mathematical model of inert gas behaviour in a body and was to be the forerunner of current decompression tables. Later observations showed this principle to be incorrect in many cases, but these early tables and the later modified versions, prevented many divers from developing the bends.

Diving research this century has led to a great improvement in all forms of diving equipment and since 1940 the use of such equipment has increased greatly. The design by Cousteau and Gagnan in 1943, of a proper demand-regulated air supply from compressed air cylinders worn on the back has developed into modern day **scuba**.

The scuba equipment used today, with the high-pressure regulator on the cylinder and a single hose to a demand valve in the mouth, was invented in Australia and marketed by an engineer named Ted Eldred in the early 1950s, under the *Porpoise* trademark.

Closed-circuit rebreathing apparatus using, oxygen or oxygen/nitrogen mixtures, has also been improved considerably since the early units used by Italian Naval divers in their attacks on shipping in Gibraltar in 1941. With the advent of deeper diving, gas economy has become a major problem and for this reason closed-circuit systems have achieved even greater importance.

Diving to depths in excess of 100 metres required not only the development of specialised closed or semi-closed circuit rebreathing apparatus, but also the use of other inert gas mixtures mixed with oxygen. Nitrogen, because of its narcotic effect at depth, has been replaced largely by other gases such as helium and hydrogen. These are not used without complications – as all gases cause specific physiological problems and no ideal mixture yet exists.



Fig. 1.5
"Jim" one-atmosphere diving suit.

The advent of **saturation diving** has completely revolutionised the ability to dive and work at great depth, and for lengthy durations, and this is economically rewarding. The system is based on saturation at any depth of all the diver's tissues by the inert breathing gas. Once this is achieved the body is incapable of absorbing further amounts of gas, no matter what the duration of exposure at this depth. Hence, further exposure does not lengthen decompression times. This practice is now adopted for most diving with extended bottom times at depths in excess of 100 metres.

In an attempt to reduce the risks in deep diving, **one-atmosphere diving suits** (ADS) have been developed out of strong lightweight alloys. These suits are fitted with articulated joints and use mechanical levers or claws for "hands". Some even have mobility and propulsion, but all require backup 'rescue' facilities. They are equipped with a self contained rebreathing apparatus and are often used at depths of 200–300 metres. Although somewhat bulky and requiring hoisting gear at the surface, divers can achieve a reasonable degree of movement at depth with the latest models. These suits are also useful for inspection-type work, although much of this is now done by non-manned **Remote Operated Vehicles** (ROV's) with video surveillance.

Fig 1.6 The Royal Australian Navy Clearance Diving Team, 1955, with oxygen breathing equipment and wearing the famous "Clammy Death" suit.



Chapter 2

PHYSICS

To understand the physical and physiological problems which can confront a diver, it is helpful to recall a few basic physical laws of nature. Only a brief and simplified review of the physics of diving is given in this text. For more detailed explanations, refer to the diving manuals.

PRESSURE

Some of the major physical hazards are related to the effects of pressure. Pressure is defined as force per unit area. i.e.

$$\text{PRESSURE} = \frac{\text{FORCE}}{\text{AREA}}$$

If a force is spread over twice the area, the pressure is halved.

This explains why, for example, wide tyres are preferable for driving on beaches. The weight of the vehicle (force) when spread over a large area causes less pressure on the sand. This vehicle is less likely to sink into the sand than one with narrow tyres.

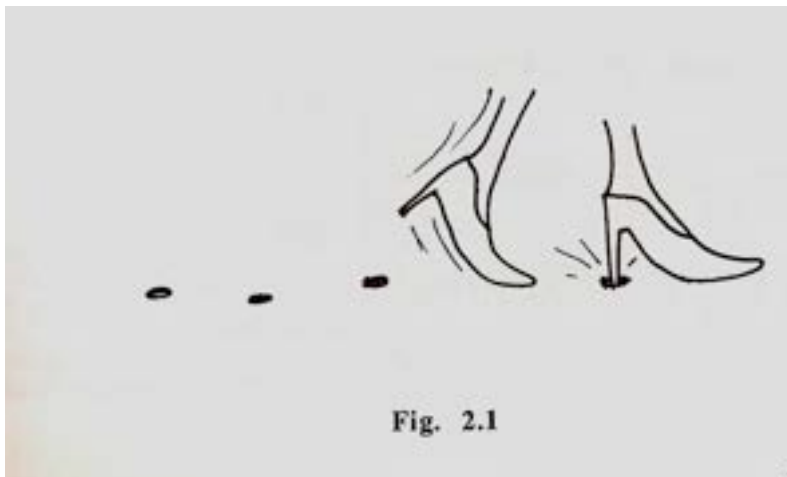


Fig. 2.1

The effect of stiletto heels on soft wooden boat decks

Gases exert pressure because they are made up of lots of fast moving molecules. The greater the number and the faster they move, the greater the pressure.

Pressure on a Submerged Diver

The pressure acting on a submerged diver has two components:

1. The atmosphere above the water, termed **atmospheric pressure**,
2. The weight of the water above the diver, termed **hydrostatic pressure**.

Divers' depth gauges are calibrated only to read the hydrostatic pressure (the depth of water) and so they read zero at sea level. They do not read the 1 atmosphere (1 ATA) above them. Thus the "gauge pressure" is always 1 atmosphere less than the true or "absolute" pressure. We will now elaborate.

Atmospheric Pressure

The atmosphere above the earth is some 150 km high. Although air is very light, this amount of air has significant weight and exerts substantial pressure on the earth's surface.

ABSOLUTE PRESSURE	GAUGE PRESSURE	DEPTH of SEAWATER
1 ATA	0 ATG	Surface
2 ATA	1 ATG	10 metres (33ft)
3 ATA	2 ATG	20 metres (66ft)
4 ATA	3 ATG	30 metres (99ft)

Atmospheric pressure at sea level is referred to as "one atmosphere" or "one bar". It is the same as 101.3 kPa, 1 kg/cm², 760mm Hg and 14.7 psi. At higher altitudes, atmospheric pressure is reduced, a factor which has a significant effect on diving in mountain lakes (see Chapter 6).

Table 2.1
Pressure at Depth

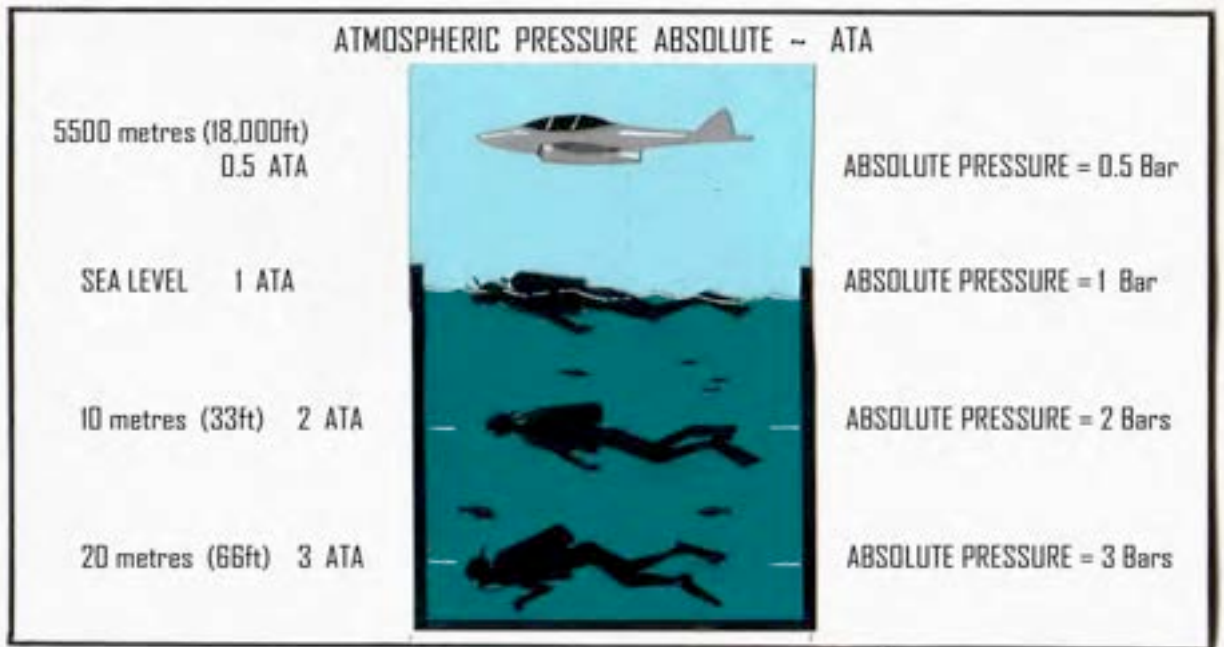


Fig. 2.2 Atmospheric and Hydrostatic Pressures (depth) added and thus converted to Absolute Pressure

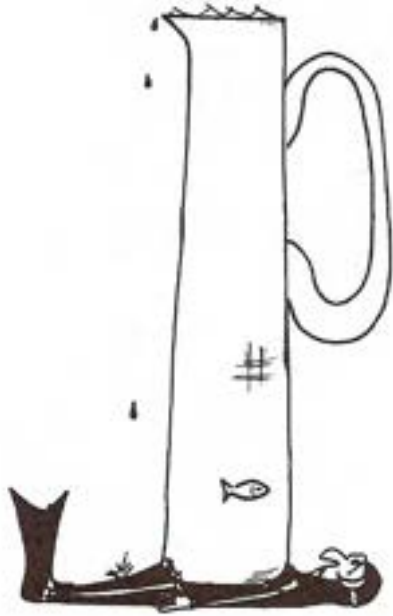


Fig. 2.3

Water is much denser than air and 10 metres (or 33 ft) of sea water exerts the same pressure (weight) as the whole 150 km of atmospheric air i.e. 1 ATA. For every additional 10 metres the diver descends, the water will exert a further pressure, equivalent to another atmosphere (1 ATA).

Common units of pressure (approximately):

<p>1 ATMOSPHERE = 10 metres sea water = 33 feet sea water = 34 feet fresh water = 1 kg/cm² = 14.7 lbs/in², psi = 1 bar = 101.3 kilopascals, kPa = 760 millimetres mercury, mm Hg.</p>
--

Absolute Pressure

The total pressure exerted on a diver at depth will be the pressure due to the atmosphere acting on the surface of the water (atmospheric pressure) plus the pressure due to the depth of the water itself (hydrostatic pressure).

The total pressure acting on the diver is termed the "absolute pressure". It is often expressed in terms of atmospheres and is called "atmospheres absolute" or "ATA".

To calculate the absolute pressure acting on a diver at a given depth in terms of atmospheres, divide the depth in metres by 10 (since every 10 m. sea water exerts 1 atmosphere pressure) and add 1 (the pressure of the atmosphere above the water).

e.g. the absolute pressure at 40 metres is $[40 \div 10] + 1 = 5$ ATA

(The depth in feet, divided by 33 + 1 also calculates absolute pressure, for those in the USA, e.g. the absolute pressure at 99 ft is $99/33 + 1 = 4$ ATA).

Gauge Pressure

As described above, hydrostatic pressure in diving is generally measured by a **pressure** or **depth gauge**. Such a gauge is normally set to register a pressure of zero at sea level and so it ignores the pressure due to the atmosphere (1ATA).

The pressure registered by a gauge at 10 metres sea water depth would thus be one atmosphere gauge (1ATG) or equivalent units. Gauge pressure is converted to absolute pressure by adding 1 atmosphere pressure.

Partial Pressure

With a mixture of gases, the proportion of the total pressure contributed by each of the gases is termed its partial pressure (its part of the pressure). The partial pressure contributed by each gas is proportional to its percentage of the mixture. Each gas contributes the same proportion to the total pressure of the mixture, as is its proportion in the composition of the mixture.

e.g. air at 1 ATA contains 21% oxygen, hence the partial pressure of oxygen is 0.21 ATA and air at 1 ATA contains 78% nitrogen, hence the partial pressure of nitrogen is 0.78 ATA.

GAS LAWS

Gases behave in nature and in diving according to several laws. Knowledge of these laws is important to the diver because they influence the duration of the air supply and affect the gas containing spaces in the body such as the ears, sinuses and lungs. They also cause other diving illnesses.

Boyle's Law

This defines the relationship between pressure and volume. It states that the **volume of a given mass of gas varies inversely with the absolute pressure (if the temperature remains constant)**.

Stated simply, for a given amount of gas, if the pressure is increased, the volume is proportionally decreased and vice versa. This means that if the pressure is doubled, the volume is halved and vice versa.

Stated mathematically: V varies as $\frac{1}{P}$ (where V = volume and P = pressure)

It follows that for a given amount of gas, the volume multiplied by the pressure always has a constant value.

i.e. $P \times V$ is constant.

So if a sample of gas has an original volume of V_1 and an original pressure of P_1 , and either the pressure or volume are changed, the new volume V_2 and the new pressure P_2 will multiply out to the same value.

i.e. $P_1 \times V_1 = P_2 \times V_2$



Fig. 2.4

This law can easily be demonstrated by a piston and cylinder such as a bicycle pump. If the piston is pushed into the cylinder half way, and the escape of gas prevented, the pressure in the cylinder will be found to have doubled. By this process, many litres of air can be crammed into a bicycle tyre but at the cost of an increase in pressure in the tyre (and hard work). Compressors work in this way, squeezing 2000 or more litres of air into a scuba cylinder – but at a high pressure.

Since water pressure increases with depth, the consequent reduction in gas volume becomes very important to the diver because his body has numerous air spaces.

Descent Problems: The air in the diver's middle ear and sinuses will contract in volume as the diver descends. If these volume changes are not compensated for by adding more air ("**equalisation**"), then pressure damage (**barotrauma**) to the tissues will result. For example:

If a 6 litre bag is filled at the surface (1 ATA) and taken to 20 metres depth (3ATA), the volume will be reduced by a factor of 3, to 2 litres.

$$\begin{aligned}
 P_1 \times V_1 &= P_2 \times V_2 \\
 \therefore 1 \times 6 &= 3 \times V_2 \\
 \text{i.e. } V_2 &= 2 \text{ litres}
 \end{aligned}$$

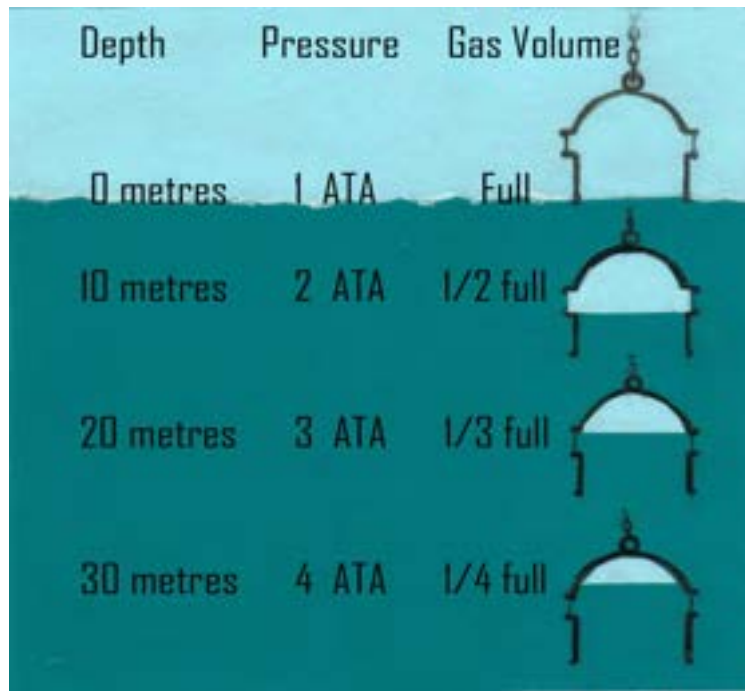


Fig. 2.5

In the same way, if a **breath-hold diver** takes a full breath at the surface and descends to 20 metres (3 ATA), the volume of air in his lungs may be reduced from 6 litres to 2 litres. The chest and lungs cope with compression better than distension. The limit for breath-hold diving is not known, but now has been shown to exceed 150 metres in certain individuals.

Ascent Problems. An average male diver's lungs may contain about 6 litres of gas. If a diver takes a full breath at 20 metres (3 ATA) from his scuba set and returns to the surface (1 ATA) without exhaling, the volume of gas in his lungs will increase from the 6 litre total lung capacity to 18 litres (6 × 3 litres).

This can be easily calculated this way:

$$\begin{aligned}
 P_1 \times V_1 &= P_2 \times V_2 \\
 P_1 &= 3 \text{ ATA}, V_1 = 6 \text{ litres}, P_2 = 1 \text{ ATA}, \\
 V_2 &= ? \text{ litres} \\
 V_2 &= \frac{P_1 \times V_1}{P_2} \\
 &= \frac{3 \times 6}{1} \\
 &= 18 \text{ litres}
 \end{aligned}$$

The lungs would have to expand to 18 litres to accommodate this volume – well beyond their rupturing point, causing **burst lung (pulmonary barotrauma of ascent)**.

An important practical observation of Boyle's Law is that **the greatest volume changes take place near the surface. This means that the greatest danger from barotraumas is near the surface — and this applies with descent as well as ascent.**

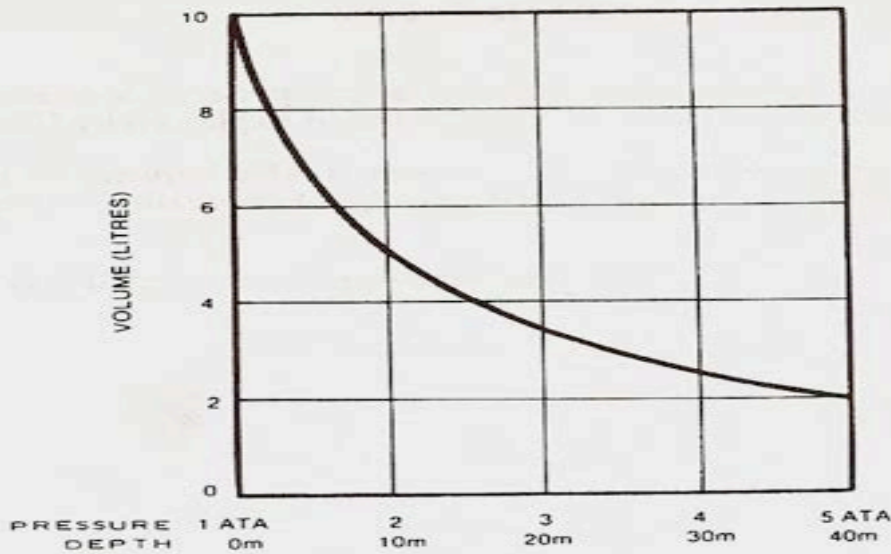


Fig. 2.6

This diagram shows changes in gas volume caused by pressure change at various depths. Note that maximal volume change is near the surface.

For example, if diver has a maximum of 4 litres of air in his lungs at 40 metres depth (5 ATA) and ascends 10 metres without exhaling (to 4 ATA), the volume in the lungs will increase to 5 litres:

$$\begin{aligned}
 P_1 \times V_1 &= P_2 \times V_2 \\
 5 \times 4 &= 4 \times V_2 \\
 \therefore V_2 &= 5 \text{ litres}
 \end{aligned}$$

Some people could possibly accommodate this expansion without lung damage.

If the same diver started at 10 metres depth (2ATA), and then ascended 10 metres to the surface (the same ascent distance as before), without exhaling, the pressure would change from 2ATA to 1ATA. The air in the lungs would expand from 4 to 8 litres. This would rupture his lungs.

Although the dives involved the same ascent distances, the volume change, and hence the danger, in response to Boyle's Law, is much greater near the surface.

Many divers are not aware of this and have a fallacious belief that if they confine their diving to shallow depths they will minimise the risk of barotrauma.

Buoyancy compensators are similarly affected by depth changes in response to Boyle's Law. **Wet suits** are also affected and lose their buoyancy and insulating properties with depth.

Charles' Law

Most divers will have noticed that bicycle pumps and air compressors become hot during use. As the volume of gas is compressed, heat is produced. This is explained by Charles' Law.

This Law states that **if the pressure remains constant, the volume of a given mass of gas varies directly with the absolute temperature** (absolute temperature is obtained by adding 273 to the temperature in degrees Celsius).

In other words, at a fixed pressure, if gas is heated it expands, and if gas is cooled its volume contracts.

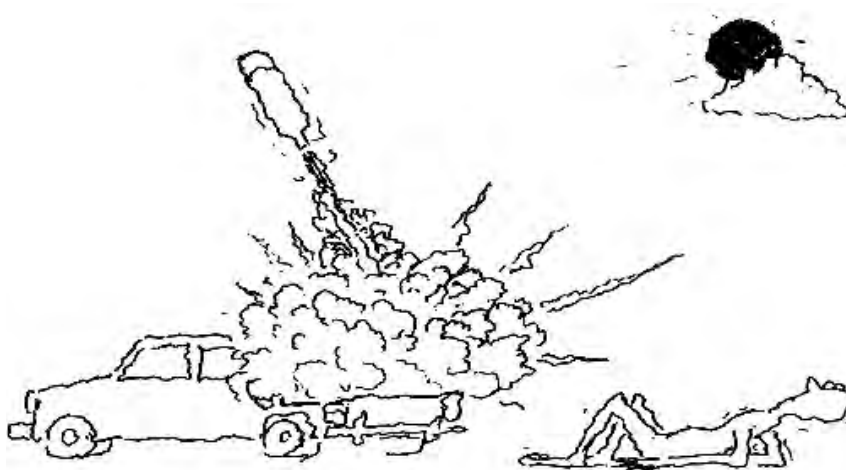


Fig 2.7

Charles' and Boyle's laws can be combined into the **General Gas Law** : $\frac{PV}{T}$ is constant

For the non-mathematically minded this means that for a given amount of gas, the pressure multiplied by the volume, divided by the temperature, always comes to the same value – so if one of these factors is varied, it has an effect on the other two.

If a gas sample having $\frac{P_1V_1}{T_1}$ has one of these factors changed,

the new set of values $\frac{P_2V_2}{T_2}$ will multiply out to the same answer

$$\text{i.e. } \frac{P_1V_1}{T_1} = \frac{P_2V_2}{T_2}$$



Stated in another way; if a gas is compressed, its volume decreases and it gets hotter. If the gas is heated and the volume is prevented from expanding, the pressure rises.

The consequence of this law has led to the demolition of several perfectly good automobiles (and divers!) following the storage of full scuba cylinders in the boot (trunk) in hot weather. Similarly, inflatable dive boats are often pressurised to the maximum and are then left in the sun. As the temperature rises, the pressure of the contained air progressively increases and then suddenly reduces – when the volume increases and when the boat explodes.

If gas is allowed to expand rapidly, it cools. Cooling from the expansion of previously compressed air, as it is breathed from a scuba cylinder, can lead to the regulator freezing up during cold water diving.

Problem: If the temperature of a scuba cylinder is 37°C after being disconnected from the compressor. Its pressure gauge reads 199 ATG, what is the pressure after it has cooled to 17° C?

$$\frac{P_1 V_1}{T_1} = \frac{P_2 V_2}{T_2}$$

now because V_1 and V_2 are the same (the cylinder volume is unchanged), the equation can be written:

$$\frac{P_1}{T_1} = \frac{P_2}{T_2}$$

and this can be rearranged to:

$$P_2 = \frac{P_1 T_2}{T_1}$$

Substituting the figures: (note that the cylinder pressure is in ATG and needs to have 1 atmosphere added to get ATA, also that the temperatures have to be converted to degrees absolute by adding 273 degrees)

$$\begin{aligned} \therefore P_2 &= \frac{(199+1) \times (273+17)}{(273+37)} \\ &= 187 \text{ ATA} \end{aligned}$$

Dalton's Law

With a mixture of gases, the total pressure exerted by the mixture, is the sum of the pressures that would be exerted by each of the gases if it alone occupied the total volume. That is, the total pressure is the sum of the partial pressures.

As the overall pressure increases (with descent underwater), so the partial pressure of each constituent gas increases.

e.g. if air contains approximately 21% oxygen (O₂) and 78% nitrogen (N₂), then in a sample of air at a given pressure, O₂ will contribute 21% of the total pressure and N₂ will contribute 78%.

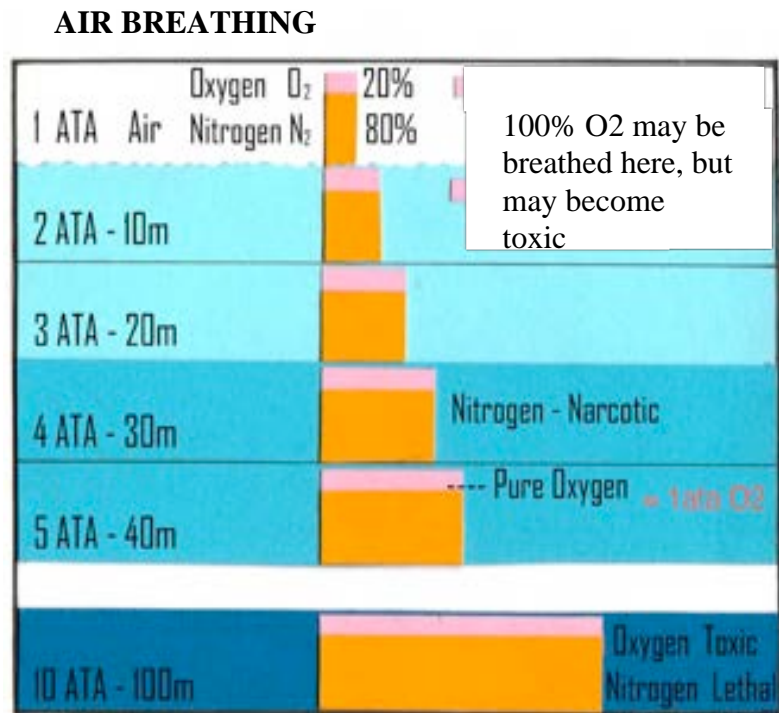


Fig. 2.9

At atmospheric pressure the partial pressure of O₂ in air is $\frac{21}{100}$ of 1 ATA. = 0.21 ATA
 while the partial pressure of N₂ is $\frac{78}{100}$ of 1ATA = 0.78ATA.

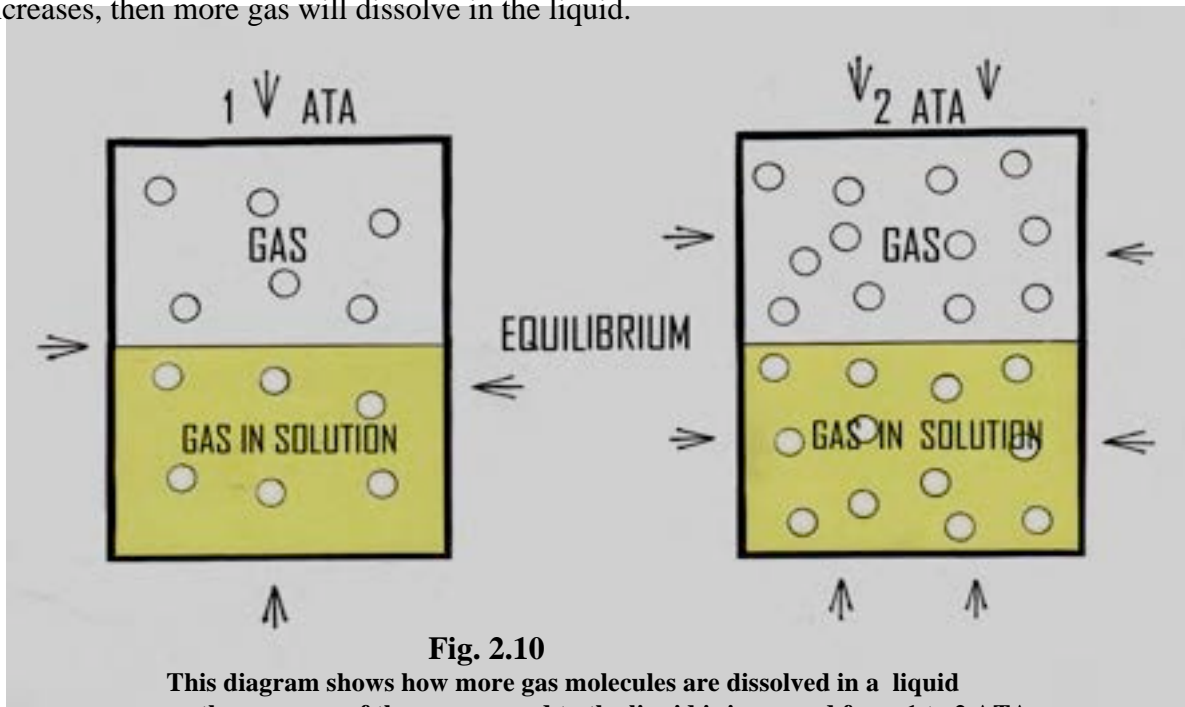
To calculate the partial pressure of a gas, multiply the percentage of gas by the absolute pressure.

This law is important when considering the toxic effect of gases at depth or the use of O₂ for treatment purposes.

Problem: Since O₂ can cause convulsions when breathed at greater than 1.8 ATA, would it be safe to breathe a mixture of 50% O₂ and 50% N₂ at 30 metres (4 ATA) ?
 The partial pressure of O₂ = 50% × 4ATA = 2ATA
 This oxygen / nitrogen mixture would be potentially toxic at this depth.

Henry's Law

This law describes the dissolving of gas in a liquid and states that the **quantity of gas which will dissolve in a liquid at a given temperature is proportional to the partial pressure of gas in contact with the liquid**. This means that if the pressure of gas exposed to a liquid increases, then more gas will dissolve in the liquid.



An example of this law can be seen whenever a fizzy soft drink bottle is opened. During the manufacture of these drinks, carbon dioxide is dissolved in the liquid under pressure and the lid on the bottle maintains the pressure. When the bottle is opened and the pressure released, the liquid will not allow as much gas to be dissolved and so the excess gas is released from solution in the form of bubbles.

At sea level (1ATA) the human body contains approximately 1 litre of N_2 dissolved in the tissues. Whenever a diver breathes compressed air at depth, more N_2 will dissolve in the body because the partial pressure of N_2 in the air being breathed is increased. This is the cause of **nitrogen narcosis**.

Under certain circumstances, when the diver returns to the surface this N_2 can come out of solution in the form of bubbles. These bubbles cause tissue injury which is the basis of **decompression sickness** ("bends").

Fig. 2.11

Diffusion of Gases

If a diver were to pass wind in a confined room, all the occupants of the room would soon be aware of the fact but, fortunately, not necessarily the source.

This process of distribution of gas is termed diffusion. It is caused by the rapid random movement of gas molecules to all parts of a contained space. Gas molecules, being only single or small groups of atoms, are able to easily diffuse through watertight membranes such as blood capillaries or cell walls. This process allows O₂ and other gases to pass from the lungs to the blood and tissues, and then back.

GASES OF IMPORTANCE TO DIVERS

Air

Air consists of a mixture of O₂ + N₂ + a trace of carbon dioxide (CO₂), and minute amounts of rare gases. Rare gases such as Neon (Ne), Argon (Ar) and Xenon (Xe), and Hydrogen (H₂) exist in trace amounts only.

The approximate composition of air is:

Oxygen (O₂)	—	21% by volume
Nitrogen (N₂)	—	78% by volume
Carbon Dioxide (CO₂)	--	0.04% by volume
Others	--	< 1 % by volume

Some less reputable suppliers of air fills for scuba tanks provide free additives to the compressed air, such as dust, oil, hydrocarbons, rust, water vapor and carbon monoxide (CO).

Oxygen – O₂

This is a colourless, odourless, tasteless gas which is indistinguishable from air to breathe.

It is essential for metabolism and maintenance of life yet in quantities exceeding those in air it is **toxic** to man. Its proportion in air (21% or more specifically, a partial pressure of 0.21 ATA at sea level) is critical. A little more than this causes O₂ toxicity, a little less will not support human life. For this reason most gas mixtures breathed by deep divers contain an inert gas – usually either N₂ or helium (He), mixed with O₂ to ensure that the O₂ composition is maintained at a partial pressure close to 0.2 ATA (0.16 – 0.40 ATA).

O₂ supports combustion vigorously and can cause normally non-flammable substances (such as the occupants of a recompression chamber) to burn brilliantly if it is present at a sufficiently high partial pressure.

Divers should be aware of the potentially explosive and combustible properties of oxygen, as they may require to use it in first-aid, or be inadvisably enticed into diving with high oxygen mixtures.

Nitrogen – N₂

This gas, which is the major constituent of air, is also colourless, odourless and tasteless. N₂ dissolves well in body fluids and tissues, causing **narcosis** at depth and **decompression sickness** when it bubbles out of solution, after ascent.

It is termed an "**inert gas**" because it does not take part in human biochemical processes. The Creator appears to have included this gas in air to prevent us from developing O₂ toxicity, and to reduce the fire hazard.

Divers vary this N₂/O₂ ratio (in Nitrox, oxygen enriched air or mixed gas diving) in an attempt to improve on nature, extend diving durations, and reduce narcosis.

Carbon Dioxide – CO₂



This gas is also colourless, odourless and is said to be tasteless. However if a diver inhales a mouthful of CO₂ from a buoyancy vest inflated from a CO₂ cartridge it will be found to taste very nasty, due to its formation of carbonic acid in water.

CO₂ is a by-product of cellular metabolism and we exhale approximately 5% of CO₂ in our breath.

If a diver rebreathes some of his exhaled gas by using faulty breathing equipment or an excessively long snorkel the CO₂ will accumulate in the body leading to toxicity. These effects are discussed further in Chapter 22.

Carbon Monoxide – CO

This gas is colourless, odourless and tasteless. It cannot be detected by a diver and even in trace amounts can cause loss of consciousness or death.

It is usually produced as a product of incomplete combustion of carbon containing compounds and is a constituent of internal combustion engine exhausts and cigarette smoke.

Air contaminated by carbon monoxide, if supplied in scuba cylinders or by surface supply to divers, may have lethal results (see Chapter 23).

Helium – He

This is a colourless, odourless, tasteless gas, which is very light and very expensive. It is obtained from underground natural gas sources found in North America and elsewhere.

It is used to dilute O₂ in gas mixtures breathed at great depths because it has little tendency to produce narcosis (e.g. Heliox may be 90% He + 10% O₂, or any other proportion).

Due to its very low density it readily escapes through small leaks in pipes and valves making it difficult to retain. It is also a very effective conductor of heat, causing serious problems with **hypothermia**.

The low density of He alters the normal process of speech production causing "**Donald Duck**" like speech when a diver breathes this gas.

Hydrogen – H₂

This is a very lightweight gas that can replace N₂ to reduce narcosis at depth. Unfortunately it can combine explosively with O₂ and the resultant water (H₂O) is not sufficient to 'put out the diver'. It is sometimes used with very low O₂ percentages, at great depths, by skilled professional divers. It shares many problems with He.

Inert Gases:

**Neon – Ne, Argon – Ar,
Radon – Rn, and Xenon – Xe**

These are more biologically inert gases which are present only in trace amounts in the atmosphere. They are of no importance to recreational divers.

Oil Gases

Because of lubrication needs in the compressor, oil vapors and hydrocarbons can be produced which may then contaminate the air supply. See Chapter 24.

BUOYANCY

It is important for divers to understand the factors affecting buoyancy. These are:

Density

Density is defined as **mass per unit volume** (density = mass \div volume).

For our purposes, mass can be considered to be the same as weight, so density is equivalent to weight per unit volume.

A substance is more dense than another if the same volume has more weight. Try lifting a bucket of water and then a bucket of lead, to illustrate this.

Specific Gravity

Specific gravity (S.G.) is the density of a substance compared to the density of fresh water which is given a value of one.

Lead has a specific gravity of 13.5 so it is 13.5 times as dense as water.
e.g. 1 litre of water will weigh 1 kg., while the same volume of lead will weigh 13.5 kg.

The concept of specific gravity is important since the specific gravity of a substance determines whether it will float or sink in water.



Fig. 2.13

A substance with a specific gravity greater than 1 (i.e. denser than water) will sink. Lead, with a specific gravity of 13.5, does not float well, whereas oil, with a specific gravity of 0.8, floats easily — producing an oil slick.

The human body has a specific gravity of slightly greater than 1, depending on its content (fat has a specific gravity less than 1, and bones are greater than 1) but the air content of the lungs provides enough buoyancy to allow most people to float.

Archimedes Principle

The ancient Greek, Archimedes (apparently while reclining in his bath), discovered that when an object is immersed in a fluid, it appears to be lighter, and that the apparent loss of weight (or **buoyancy**) is equal to the weight of water displaced by the object.



Fig. 2.14

That is – the buoyant effect will be equivalent to the weight of fluid of equal volume to the immersed object.

Depending on whether the weight of fluid displaced is greater than, equal to or less than the weight of the object, an object immersed in the fluid will either float, remain suspended or sink. Even an object which sinks will still appear to be lighter than it would out of the fluid.

Sea water is denser than fresh water because of the salt content, so a greater weight of sea water will be displaced by an object. Hence objects in sea water are more buoyant than in fresh water.

Air (in the abdomen, buoyancy compensator and wet suit) contributes to buoyancy. Unfortunately air in these compartments varies in volume in response to the pressure changes with varying depth, making constant buoyancy adjustments necessary. This is usually accomplished by adding air to, or releasing it from, the diver's buoyancy compensator.

Divers go to considerable lengths to vary their buoyancy to help them submerge, to stay at a given depth, or to ascend or stay afloat in an emergency.

PHYSICAL EFFECTS OF THE ENVIRONMENT

Temperature

Body heat is a form of energy, the level of which can be estimated by measuring the body temperature.

Heat energy flows from areas of high temperature to areas of low temperature. The heat transfer which is important to the diver is **thermal conduction** (or transfer of heat by direct contact), and may cause **hypothermia** (low body temperature).

Since normal body temperature is 37°C and oceanic water temperature is commonly 12–20°C, the diver is almost always immersed in water at a lower temperature than his body. Usually

the water temperature decreases with depth, but there may be layers of water at different temperatures (thermoclines) – especially in still water.

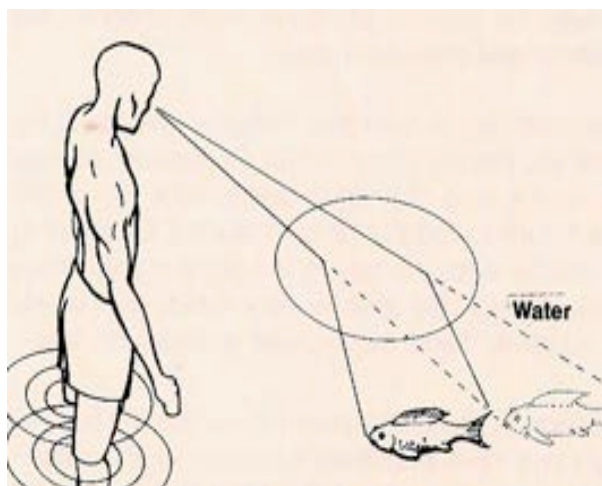
Cold water creates a strong temperature gradient along which heat flows from the body, resulting in a continuous heat loss into the water. This process is assisted by water having a high capacity to conduct and absorb heat.

Since the maintenance of normal body temperature is essential for physiological functioning, the diver needs to take steps to minimise heat loss. This may be achieved by inserting a layer of air (which is a poor conductor of heat) between the diver and the water. It is conveniently contained in minute cells in a wet suit or under a rubber skin in a dry suit.

Light and Colour

Substances that transmit light have a tendency to slightly alter the path of the light rays which pass through them. This process is termed **refraction**. The degree to which they do this is termed the refractive index. Each time light passes through an interface between substances with different refractive indices, its path is bent.

When a diver views objects underwater, light must pass through the water, the face mask glass, and the air in the mask before it reaches his eyes. The light rays are refracted at each of these interfaces and the distortion makes objects appear larger and closer by a factor of about 25%.



Until the diver adapts to this distortion, it may be difficult to judge size and distances. This creates practical difficulties with simple tasks such as spear fishing.

Light rays are scattered by particles in the water making shadows less pronounced and reducing the ability to see clearly over large distances.

Fig 2.15 The fish appears closer, because the light rays are refracted at the air/water interface

Clear focusing of the eye depends heavily on the refraction of light rays passing between the air in front of the eye and the cornea (the clear surface at the front of the eye). If the eyes are opened underwater without a face mask, the absence of this air/cornea interface results in very blurred vision.

Water absorbs colours to differing degrees. In clean oceanic water, red is absorbed in the first metre, orange in the first five metres, yellow in the first ten, and green and blue at greater depths. This explains why most things, regardless of their colour on the surface, appear to be coloured shades of blue or green at depths beyond about ten metres.

Inshore waters often contain yellowish products of vegetable decay which absorbs most colours except green. As a result, clean oceanic water appears blue, while inshore and estuarine water appears green from the surface.

Because deep water is lit mainly by blue and green light, coloured corals and fish at these depths look less brilliant unless illuminated by a torch or camera flash.

For safety reasons, it is advisable to wear conspicuously coloured diving equipment. However, the absorption of light underwater needs to be considered when choosing these colours. Red, for instance, which is easily visible on the surface, appears black at depth because of the significant absorption of red light by the water.

Fluorescent orange or **yellow** paint or fabric affords better visibility because the fluorescent dye actively emits light of its own colour and also provides a good contrast against natural aquatic backgrounds.

Sound

Sound waves in air are usually reflected at the air–water interface, and therefore shouting instructions to submerged divers is not of much value. Underwater, the sound wave travels much faster than in air, and this makes localisation of the source much more difficult. An example of this is the concern experienced when divers hear outboard engines, but cannot identify the distance or direction of the boat.

Altitude

If exposed to altitude (less than 1 ATA) a variety of effects may endanger the diver. Some equipment may be affected e.g. pressure gauges, and the diving profile needs to be modified to prevent pulmonary barotrauma and decompression sickness (see Chapter 6).

Chapter 3

PHYSIOLOGY*

A basic understanding of the bodies' processes is needed to grasp the physiological effects of diving and the application of diving medicine. The cardiovascular and respiratory systems are described here while the physiology of some other organs, such as the ear, are considered in specific chapters.

METABOLISM

The Need for Energy

Energy is a fundamental requirement for all life processes. It is needed for growth, repair, movement and all the active functions of the body. The fuel for this energy comes from **carbon compounds**, which are incorporated in complex molecules in the food we eat. This is biochemically dismantled in the digestive tract into simple chemical compounds which are absorbed and carried by the blood stream to the cells. Here they undergo further biochemical processing until ultimately the carbon is combined with oxygen (O₂), forming carbon dioxide (CO₂) and releasing energy.

This is similar to the energy formation which takes place in an automobile engine or a fire, where carbon in fuel or wood is combined with O₂ to produce energy. The body processes will only function under strict conditions of O₂ availability, temperature and acidity.

The body needs a means of transferring food products to the cells, together with delivery of O₂ and removal of CO₂. This is performed by the blood, in the vascular system. It comprises **arteries** which take blood to the tissues, a vast network of microscopic **capillaries** that bring the blood into contact with all the cells of the body, and **veins** which return blood to the heart.

The blood is circulated through the blood vessels by a muscular pump – the heart, and the whole system is called the **cardiovascular** system. It brings O₂ from the lungs to the cells and eliminates CO₂ through the respiratory system.

*** Reading this chapter can be delayed if time does not permit, and the reader is in a hurry to commence diving**

RESPIRATION

Anatomical Structure

The respiratory tract begins at the mouth and nose and ends in the microscopic air sacs called the **alveoli**, in the lungs.

The **nose**, apart from its decorative function, warms and humidifies the air that we breathe. It also filters large particles which might otherwise be inhaled. If the nose is bypassed by breathing through the mouth, a snorkel or scuba regulator, the lung then has to cope with drier, colder, unfiltered air.

After passing through the mouth or nose, the air then enters the throat where the **larynx** (or voice box) is situated. This is recognised as the "Adams Apple". The larynx produces the sounds of speech as well as helping to protect the lungs from inhalation of foreign material.

When sea-water from a flooded snorkel or scuba regulator enters the larynx, a trap-door like structure called the epiglottis closes over the opening and the vocal cords shut to prevent the foreign material from entering the lungs. If any material passes these structures, the cough reflex, activated by foreign material touching the inside of the air passages, may cause a **coughing** reaction which tends to expel whatever has been inhaled.

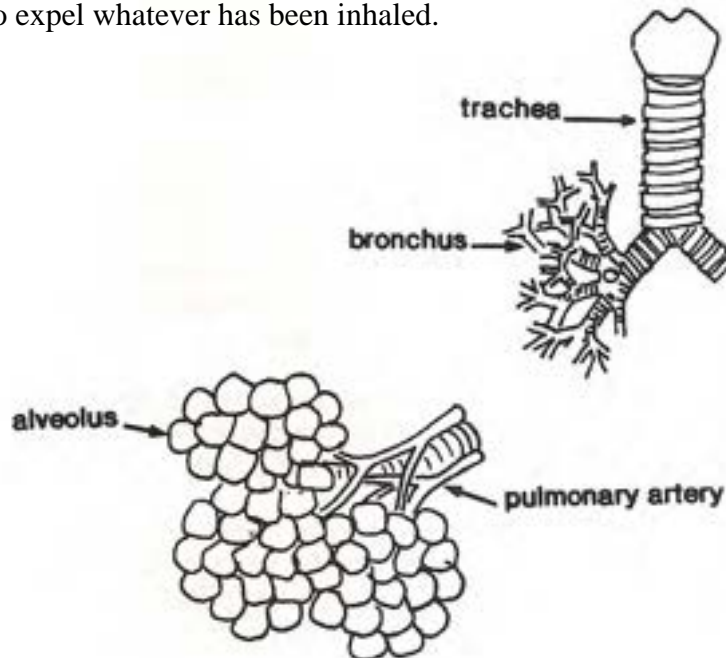


Fig. 3.1

Diagram showing the trachea and bronchial tree, terminating in the alveoli. The relationship between the bronchus, alveoli and branches of the pulmonary artery can be seen.

Below the larynx the air passes through a tube called the **trachea**. This is about as thick as the average snorkel and branches inside the chest into two tubes, the **bronchi**, which lead to the lungs. Those air passages are lined with cells covered with microscopic hairs (cilia) which move a sheet of secreted mucous slowly upwards towards the larynx. Small pieces of foreign material such as dust eventually find their way to the larynx, along with this mucous sheet. It is then either coughed-up or swallowed. The cilia may be damaged by smoking or infection, causing retention of mucous and inhaled material which may eventually obstruct the air passages.

The bronchi divide repeatedly into progressively smaller passages rather like the branches of a tree. These passages have encircling muscles in their walls which, by contraction or relaxation, can vary the diameter of the air passage.

In **asthma** the muscles of the small bronchi become oversensitive and overactive, causing excessive narrowing and obstruction of these air passages. This can occur in response to exercise, allergy, cold, infection, anxiety, smoking or other inhalants such as sea water. At the same time, the cells lining these passages produce excessive and thickened mucous. The combination of these factors causes airway narrowing which has serious repercussions for a diver.

The smallest branches of the bronchi end in bunches of microscopic air sacs called **alveoli**. The vast number of alveoli are packed together into the two sponge like organs, the **lungs**. There are about 300 million alveoli in the lungs and the combined surface area of all the alveoli in the lungs is equal to about half a tennis court. The alveoli are lined by a thin layer of fluid containing a detergent-like substance called **surfactant**. This acts as a wetting agent to prevent the alveoli from collapsing from surface tension.

The surfactant lining of the alveoli can be damaged in disease or by inhalation of water, leading to collapse of the lungs and serious respiratory difficulty.

Each alveolus is surrounded by a network of blood capillaries. These bring the blood into close contact with the air in the alveolus, with only the microscopically thin walls of the alveolus and capillary separating the two.

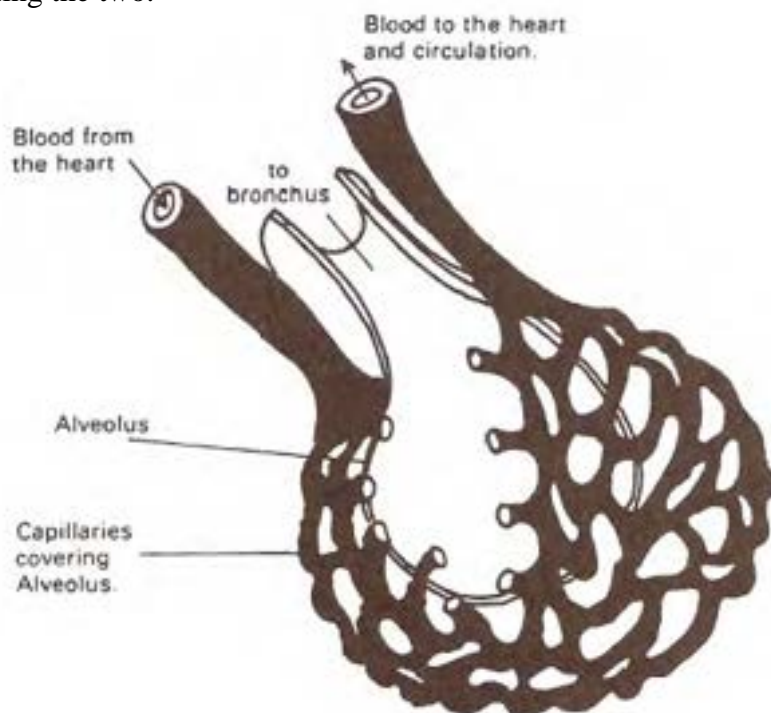


Fig. 3.2
This diagram illustrates an alveolus with its surrounding meshwork of capillaries.

If the wall of an alveolus is ruptured, as it may be in **pulmonary barotrauma** ("burst lung"), then air from the alveolus is able to enter the blood stream where it may cause blockage of distant vessels such as those in the brain. This is called an **air embolism**.

The lungs occupy a cavity about the size of a football on each side of the chest. The lung is covered by a thin membrane coating, called the **pleura**, and the inside of the chest wall is lined by a similar membrane. Between the two pleural layers is a narrow space which contains a small amount of lubricating fluid to minimise friction as the lungs expand and contract during breathing. If the outer surface of the lung tears, as it may in pulmonary barotrauma, then air can enter this pleural space causing the lung to collapse. This disorder is called **pneumothorax**.

The chest wall which encloses the lungs is made up of ribs with muscles between them - known as intercostal muscles. At the base of the chest cavity lies a large thin dome shaped muscle called the **diaphragm**. When the diaphragm contracts, it flattens and has a piston like effect, reducing the pressure in the chest cavity and increasing the volume of the lungs. The reduced pressure draws air into the lungs through the air passages.

Contraction of the diaphragm is the main method of **inhalation** in the resting state. It is assisted by contraction of the muscles between the ribs which rotate the rib cage upwards and outwards, enlarging the chest cavity and reducing the pressure in the chest. A group of neck muscles which are attached to the rib cage can also assist respiration when maximal breathing is required.

At the end of inhalation the elasticity of lungs and rib cage causes the lungs and chest wall to contract and **exhalation** takes place. With quiet breathing, this does not require muscular effort. With heavy breathing, exhalation can be assisted by the abdominal and chest muscles.

Respiratory Function

During quiet respiration in adult males, about 500 ml of air is moved in and out of the respiratory tract with each breath. The volume per breath is termed "**tidal volume**". During extremely heavy exercise, the tidal volume can increase 10 fold, up to about 5 litres.

The total amount of air that can be held in the lungs (**total lung capacity** or **TLC**) in adult males is approximately 6 litres. Only about 10% of the air in the chest is exchanged with each breath during quiet respiration. The **vital capacity (VC)** is the maximum volume that can be exhaled in one breath, and the **forced expiratory volume (FEV_{1.0})** is the maximum volume that can be exhaled in one second.

The **flow of air** through the respiratory passages varies at different stages of respiration. It reaches a peak about midway through inspiration — and during quiet breathing this peak flow rate is approximately 30 litres per minute. This value increases during exercise to 600–700 litres per minute.

Any breathing system (such as a snorkel or demand valve) which the diver is using, should be capable of handling these large air flows without significant resistance. If this does not occur, then the diver must exert extra effort during respiration in order to overcome this resistance. This problem is compounded when the diver is breathing compressed air at depth because the increased density of the gas will further increase the resistance to airflow in both the equipment and the lungs.

Gas Uptake and Loss

Air, which contains approximately 21% oxygen (O₂) and 78% nitrogen (N₂), is inhaled into the alveoli where it is brought into contact with the blood in the capillaries. This blood contains a lower partial pressure of O₂ than the air in the alveolus and a higher partial pressure of CO₂, since it has just returned from the body, which has been using O₂ and generating CO₂. Consequently, there is a pressure gradient causing O₂ to diffuse from the alveoli to the blood, and CO₂ to diffuse from the blood to the alveoli, where it is then exhaled. There is no net movement of N₂ since the N₂ in the alveoli and in the blood is in equilibrium, except when diving, altitude exposure or breathing different gases.

If the diver breathes air (78% N₂) or another inert gas such as helium, while descending or remaining underwater, this inert gas will pass from the alveoli to the blood because the partial pressure of the gas in the lungs is increasing as the diver goes deeper.

On ascent, the partial pressure of inert gas in the lungs will reduce, and this allows inert gas to move from the blood (returning from the tissues) to the alveoli, and be exhaled.

Respiratory Control

The partial pressures of CO₂ and O₂ in the blood are kept within very strict limits by a sensitive control system. There are sensors in the brain which detect small changes in the blood CO₂. If this increases, then the sensor causes stimulation of the **respiratory centre** within the brain, leading to faster and deeper respiration to eliminate more CO₂.

When a snorkel diver holds his breath, the CO₂ level in his blood increases. This produces respiratory stimulation which compels the diver to take a breath — hopefully after he has had time to return to the surface.

The sensors for blood O₂ pressure are in the carotid arteries which supply the brain. A reduction in the blood O₂ level also leads to respiratory stimulation, but this effect is not as powerful as that caused by CO₂ changes.

Smoking

The ingenious habit of rolling tobacco into a tube of paper, setting fire to it and inhaling the smoke, sabotages the complex respiratory and circulatory process at several points.

As well as predisposing to lung cancer and emphysema, noxious tars in the smoke precipitate out in the bronchi producing chronic irritation, narrowing of the bronchi and cause a persistent outpouring of mucous. This ultimately results in chronic bronchitis. The tar also poisons the cilia, which conduct the mucous up the airway to the larynx, resulting in retention of old mucous in the lungs (smell the breath!).

Various toxins in the smoke ultimately cause destruction of the alveolar walls producing cavities in the lungs and destruction of the lung architecture, resulting in the disease called **emphysema**. This, combined with obstruction of the air passages, makes the smoking diver less physically fit and more liable to air trapping in the lungs and pulmonary barotrauma (see Chapter 11).

The carbon monoxide content of the smoke reduces the capacity of the blood to carry O₂, thereby reducing oxygenation of the tissues.

Some of the chemical constituents of the smoke are absorbed into the blood stream producing changes in the walls of the blood vessels supplying the heart, brain and limbs. Ultimately these become obstructed. In later life this can cause heart attacks, strokes and peripheral vascular disease (gangrene).

CARDIOVASCULAR SYSTEM

Blood

Arteries take blood from the heart. **Veins** return blood to the heart. Arterial blood (which has absorbed O_2 as it passed through the lungs), is then pumped to the periphery by the heart and is brought close to all the cells in the body by the capillary system. Here the O_2 diffuses into the cells and the CO_2 diffuses out of the cells into the blood.

The blood transports O_2 and CO_2 . The O_2 is mainly carried by an iron containing compound called **haemoglobin (Hb)** contained in the red cells. 100 ml of blood will transport approximately 20 ml of O_2 . If the red blood cells are removed, blood plasma (the liquid part of blood) will transport only 0.3 ml of O_2 per 100 ml blood. A drop of blood contains approximately 300 million red cells.

In arterial blood, the haemoglobin is almost 100% oxygenated when the blood leaves the heart to go to the tissues. It is bright red in colour. If for any reason the arterial blood is not adequately oxygenated, it causes the blue colour of the skin and tongue (cyanosis) seen in **hypoxia** (see Chapter 20).

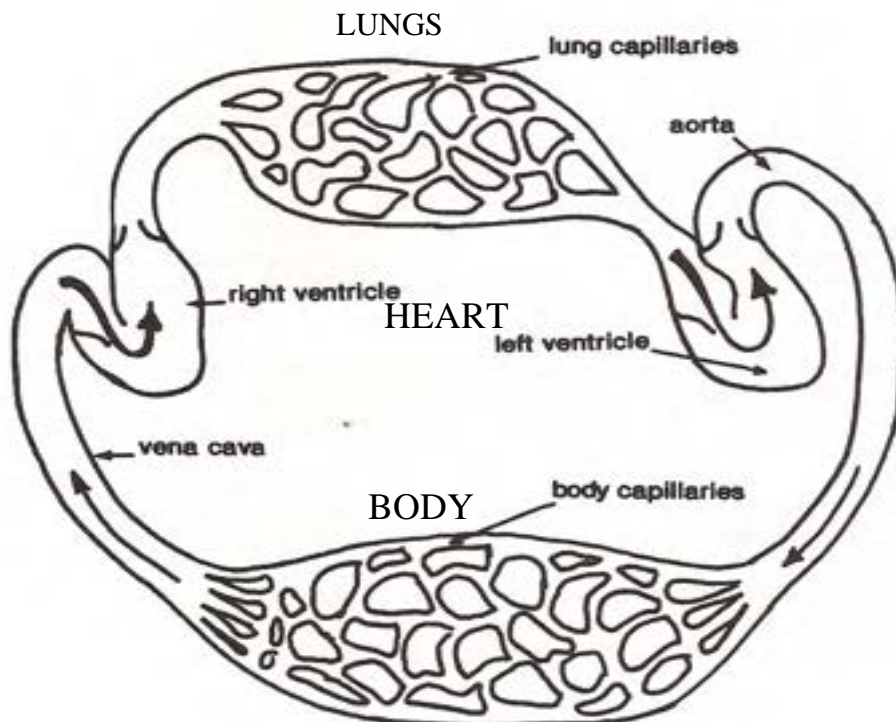


Fig. 3.3

A diagram showing the relationship between the circulations produced by the right and left sides (ventricles) of the heart.

In venous blood, the haemoglobin in red blood cells returns to the heart with 75% of its O₂ load still attached. It is then a more bluish colour.

The tissues need only 25% of the O₂ carried in arterial blood. This allows a reserve supply of O₂ which can be used during exercise or breath holding.

CO₂ is carried from the tissues by the blood in the veins, back to the lungs. Some of it is dissolved in blood plasma and some bound to the protein of the haemoglobin molecules. Although the CO₂ dissolved in the blood forms carbonic acid, the acidity of the blood is prevented from rising to excessive levels by a system of buffering compounds.

It is possible to increase the O₂ carrying capacity of blood by the use of **hyperbaric oxygen**. In recompression chambers, increased amounts of O₂ can be physically dissolved in the plasma, even though the haemoglobin is fully saturated with O₂.

Heart

The heart is a large muscular pump (about the size of a man's fist) located in the centre of the chest. See fig 3.4. It is composed of two functionally separate pumps which maintain two distinct circulations. The **right side** of the heart receives venous blood from the body and pumps this blood through the lungs where it picks up O₂ and eliminates CO₂. The **left side** of the heart receives this oxygenated arterial blood from the lungs and pumps it through the body.

Each side of the heart is essentially a two-stage pump which is not unlike a two-stage compressor. The **atrium** is the first or low pressure stage of the pump and it has a thin muscular wall. It receives blood from the veins at low pressure. When it contracts, it propels this blood into the second or high pressure stage – the more thickly walled and stronger ventricle.

The **ventricle** has two “one-way” valves, one valve preventing blood from flowing back into the atrium, and the other valve preventing blood flowing back into it from the arteries. When it contracts, it pumps blood into the arteries.

Occasionally there may be openings between left and right sides of the heart (patent foramen ovale, septal defects). In divers this allows bubbles to pass from the venous system to the arterial, causing serious manifestations of decompression sickness from dives that should otherwise be safe. People with significant heart abnormalities should not undertake scuba diving.

The heart, being a muscle, requires its own blood supply. This is provided by the **coronary arteries** which originate in the aorta, the main artery of the body. Any obstruction of these coronary arteries will cause damage to the heart muscle – a heart attack.

Partial obstruction of the coronary arteries may produce **angina** (which is pain or discomfort arising from insufficient O₂ in cardiac muscle), because it is receiving insufficient blood supply. Since a heart attack can take some of the fun out of a diving expedition, it is important for divers to have skilled medical examinations to exclude this problem or to help predict which divers will be susceptible to such heart conditions (coronary artery disease).

The resting output of the heart is about 5 litres of blood per minute. The heart has considerable reserve and if the tissues require it, can increase this output several fold by increasing its rate and strength of contraction.

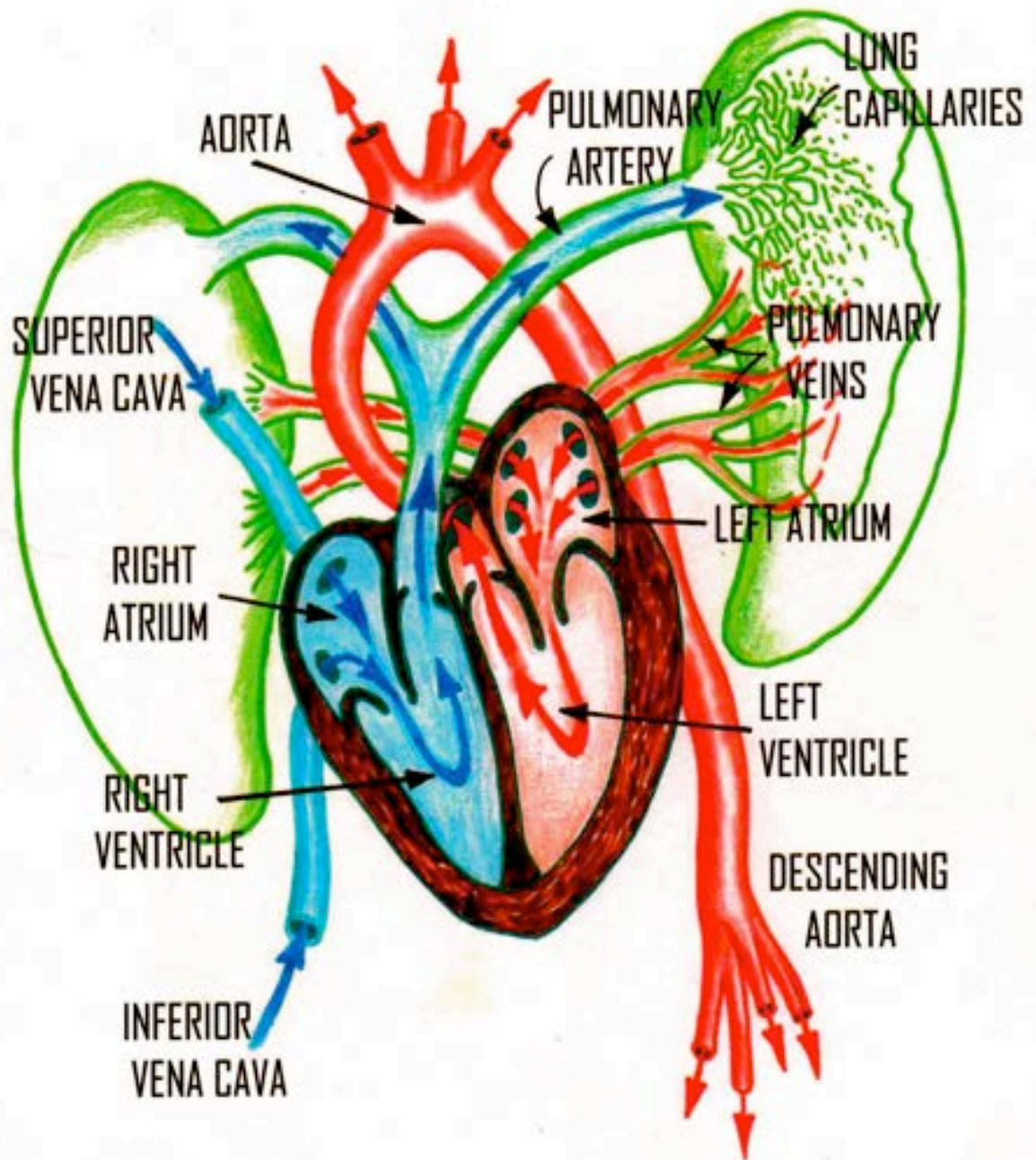


Fig. 3.4

This diagram shows a cutaway drawing of the heart to illustrate the flow of blood from the vena cava through the chambers of the heart and the lungs to the aorta.

Circulation

The blood flow from the heart is pulsatile and the **blood pressure** varies depending on the stage of heart contraction. The higher blood pressure during the heart's contraction is called the **systolic blood pressure** and has a normal value of around 100–140mm mercury (mm Hg). The pressure when the heart is not contracting is the **diastolic blood pressure** which has a normal value of around 60–90mm Hg. Blood pressure is normally recorded as Systolic / Diastolic – e.g. 130/90.

Blood vessels can change their internal diameter under the control of the nervous system. This allows for some variation in blood flow to parts of the body depending on specific circumstances. For instance, during exercise the blood vessels dilate allowing more blood flow to the muscles, while under cold conditions the blood vessels to the skin constrict, reducing the blood flow to the skin (appearing pale) and so minimising heat loss.

The constriction or dilatation of the blood vessels also influences blood pressure. Excessively high blood pressure (**hypertension**) can ultimately cause damage to the blood vessels and an excessive strain on the heart. High blood pressure requires treatment, often with drugs which dilate the blood vessels but which may interfere with safe diving.

Blood pressure is constantly maintained by a sophisticated sensing and feedback mechanism. Variations in blood pressure caused by physical activity or standing from a reclining position are quickly compensated for by changes in the diameter of the blood vessel walls.

When a person is in a reclining position, blood pressure is maintained easily and the effect of gravity does not have to be opposed by the contraction of blood vessels. When standing up quickly from this position, blood pressure in the upper part of the body may fall. Occasionally, even in normal people, the heart and blood vessels cannot compensate rapidly enough and fainting or light-headedness can result. This is known as **syncope** or **postural hypotension**.

The cardiovascular system is able to compensate for changes in blood volume, such as those associated with severe bleeding (**haemorrhage**), by constricting the blood vessels and diverting blood from non-essential organs to essential organs such as the brain and heart.

In **pulmonary barotrauma**, air can gain access to the blood as it passes through the lungs. Air bubbles may be carried to vital organs such as the brain and heart, obstructing their blood flow and leading to serious consequences (**air embolism**). In **decompression sickness**, gas bubbles may also be transported by the blood stream.

COMPRESSED-AIR DIVING

Scuba allows the diver considerable freedom but has its own limitations. It has all the potential problems of free diving, but adds special physiological problems of its own.

Resistance to Breathing

A major limitation to diving with scuba is resistance to breathing. During maximal exertion, a diver can consume over 70 litres of air per minute at the surface – but the peak flow rate during

inspiration is about three times this value. Some regulators may have difficulty delivering gas at this rate, adding considerable resistance to breathing.

This problem is magnified at depth because the greater pressure increases the density of the inhaled gas, especially at depths in excess of 30 metres when air is breathed. The same effect is seen at about 200 metres depth when helium /O₂ (heliox) mixture is breathed, because heliox is less dense than air. It is likely that resistance to breathing will ultimately limit the depth to which divers can reach.

An idea of the respiratory loads which the diver faces can be gained from the following table :

SCUBA SWIM	SPEED*	OXYGEN CONSUMPTION	RESPIRATORY MINUTE VOLUME
Slow scuba swim	0.5 knots	0.8 litres / minute	18 litres / minute
Average scuba swim	0.8 knots	1.5 litres / minute	28 litres / minute
Fast scuba swim	1.0 knots	1.8 litres / minute	40 litres / minute
Maximum scuba swim	1.3 knots	3–4 litres / minute	70–100 litres / minute

*. a knot is equal to 1 nautical mile per hour, or 1.85 km / hr

Table 3.1

Air Consumption

O₂ consumption is virtually the same for a given amount of exercise whether it is performed at the surface or deep under water. Because compressed air is being breathed at depth, more O₂ will be supplied than is needed by the diver. The actual volume of gas breathed at any depth will be the same as that which would be breathed at the surface. However, since the gas being breathed at depth is at greater pressure, the volume breathed, if converted to atmospheric (surface) pressure, will also be greater.

For example, during maximal effort a diver may consume 70 litres of air per minute at the surface. If he is performing an equivalent amount of effort at 20 metres depth (3ATA), he will still be breathing 70 litres per minute from his scuba regulator at 20 metres, but this will be equivalent to :

$$70 \text{ (litres)} \times 3 \text{ (atmospheres)} \\ = 210 \text{ litres per minute at surface or atmospheric pressure.}$$

So, the endurance of an air supply decreases with depth.

The regulator may not be able to meet the respiratory demands of a diver when certain conditions apply (see Chapter 5). Under these conditions, the diver may be aware of an inadequate air supply and either panic or take other dangerous action, such as a rapid ascent or omission of decompression requirements.

Skip Breathing

It is possible for a scuba diver to minimize his air consumption by deliberately slowing his breathing rate. This type of breathing pattern obviously limits the reserve of O₂ which will be stored in the divers lungs and haemoglobin, and may lead to retention of CO₂ and acidosis. It

reduces the safety margin in the event of air supply failure as well as increasing the likelihood of pulmonary barotrauma, and is recommended only for those with suicidal tendencies. Excess CO_2 also acts as a narcotic, so the diver may make less sound decisions

Other Effects on a Scuba Diver

The physiological effects of scuba diving may parallel those of breath-hold diving (see Chapter 4). Hyperventilation, breath holding, the diving reflexes and the effects of immersion, may all be provoked. The dehydration effect of immersion is of importance in aggravating decompression sickness.

The many pathological problems developing from scuba diving are referred to later in this text. They include pulmonary barotraumas (see Chapter 11), respiratory decompression sickness (see Chapter 15), oxygen toxicity (see Chapter 21), breathing gas contamination (see Chapter 24), the drowning syndromes (see Chapters 25 and 26) and scuba divers' pulmonary oedema (see Chapter 32).

Gas Pressures

Because of the increased pressures on the diver, high nitrogen partial pressures cause nitrogen narcosis (or "narks"). Similarly, higher O_2 partial pressures may produce O_2 toxicity in very special circumstances. Gas coming out of solution in the divers body may cause decompression sickness and/or dysbaric osteonecrosis (or "bone rot").

Chapter 4

BREATH-HOLD DIVING

Free, breath-hold or snorkel diving is a prerequisite for successful scuba diving. The diver can thus become comfortable in the aquatic environment and gradually learn the swimming and snorkeling skills that may later be used in reaching dive sites and remaining safe even after the scuba set has been exhausted. One extension of snorkeling is free diving, used to spear fish, prolong underwater explorations, retrieve equipment, check anchors and many other activities .

It is not difficult for a diver to perform a breath-hold dive for a duration of one minute or more. This is possible because there is a reservoir of oxygen (O_2) stored in the lungs (about 1 litre O_2 when the lungs are full), in blood haemoglobin, and in myoglobin in the muscles.

With these reserves the diver is able to hold his breath for some time without the blood level of O_2 becoming dangerously low. Below a threshold blood O_2 partial pressure (about 30mm Hg – less than half the normal value), the brain ceases to function properly, causing loss of consciousness. At about this stage, the heart also becomes seriously starved of O_2 causing cardiac damage or disturbances of rhythm.

During a breath-hold dive, O_2 is consumed and carbon dioxide (CO_2) produced, decreasing the blood level of O_2 and elevating that of CO_2 . Both effects may stimulate respiration but the CO_2 is the more dominant. Usually the diver develops an overpowering desire to breath (he reaches the **break point**) before the arterial O_2 level falls to a dangerous value. The urge to breath eventually becomes irresistible and the diver may even take a breath under water, if he is unable to reach the surface in time.

Breath-holding can be extended considerably, with experience and will-power but the break point is eventually reached. This is nature's safety mechanism to prevent people from losing consciousness from excessively prolonged breath-holding (see Case Histories 33.2 and 33.3).

ACCIDENTS and DEATHS

Breath-hold divers suffer from the same problems as scuba divers, except for those related to compressed gas inhalation. The common problems include environmental hazards, some equipment limitations and medical diseases such as the barotraumas, marine animal injuries,

infections, hypothermia, panic and fatigue, cardiac disorders and the drowning syndromes. These are discussed in the chapters dealing with scuba diving medicine.

In an Australian series of snorkeling deaths, the causes were dominated by drowning (45%), Cardiac disease (30%) and hypoxic blackout (20%). The first two were mainly in aquatically unfit, older tourists, and the last in younger, aquatically fit and experienced free divers. The inevitable predominance of time on the surface makes the breath-hold diver more susceptible to sunburn, boat injury and tidal currents.

The other problems more associated with breath-hold divers are discussed here.

Lung Squeeze (Pulmonary Barotrauma of Descent)

During a breath-hold dive the chest and lungs are compressed by the increasing pressure of water. As the air in the lungs is compressed, the volume is replaced to a limited degree by expansion and engorgement of the lung's blood vessels. Lung injury from this mechanism is known as lung squeeze, or pulmonary barotrauma of descent (see Boyle's Law, Chapter 2).

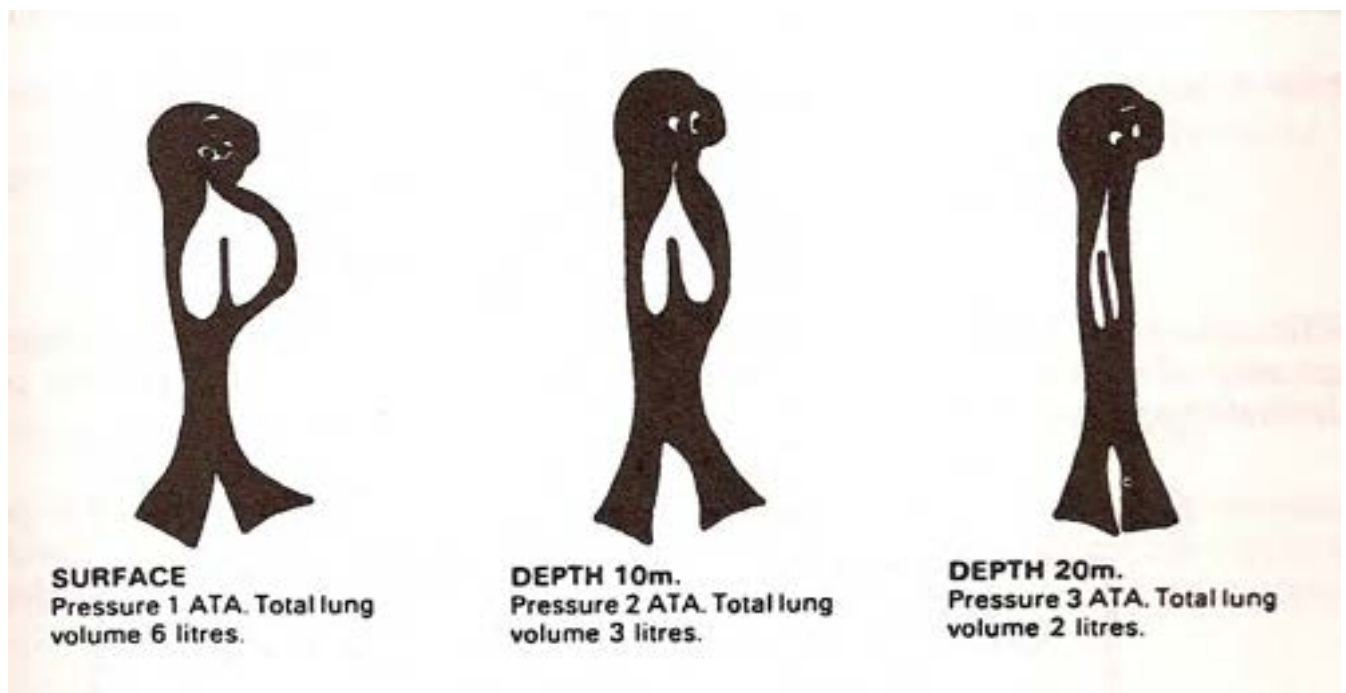


Fig. 4.1

Theoretically, the maximum safe depth for most divers should be about 30 metres (4ATA), but it probably varies between individuals, as much deeper breath-hold dives have now been performed - in excess of 200 metres.

Immersion

A neutrally buoyant diver is exempt from the main effects of gravity and this produces physiological changes in the body. The return of blood flow to the heart and lungs is increased. The body interprets this as an excess blood volume and compensates by **increasing urine production** (which may then lead to **dehydration**).

Cold water exposure produces many reflexes, including a desire to urinate. Temperature regulation is more difficult. The pressure variations may influence lung function with head out or vertical positions. Spatial orientation processes are disrupted. Trauma, in the form of physical injury from water movement, marine infections, dangerous marine animals, barotraumas, drowning etc. are dealt with in separate chapters.

Dive Reflex

Aquatic mammals display a reflex known as the "**dive reflex**". This is associated with profound slowing of the heart and redirection of the blood flow away from the muscles and non-essential organs to give a better blood supply to the heart and brain. It allows for longer submersions. This reflex is present to a less degree in humans and can be produced by immersing the face or head in cold water. The heart slowing component of the reflex has been used by physicians to treat certain cardiac disorders associated with a rapid heart rate. It can also result in heart arrhythmias.

Other potentially harmful reflexes can be induced by cold, Valsalvas, breath-holding *etc.*

Hypoxic Blackout

This loss of consciousness, due to an inadequate supply of **O₂** to the brain, usually develops without any warning. Underwater this leads to aspiration of water and drowning. It is a frequent cause of deaths amongst breath-hold divers. Sometimes the diver arrives on the surface still alive but in a state almost unconscious, and with some brain damage. This is called LMC (Loss of Motor Control) and causes unsteadiness and clumsiness. It also may occur after the diver has been rescued while unconscious or semi-conscious. At other times it may result in dementia, severe muscle impairment, visual damage or epileptic convulsions. There are two main types of hypoxic blackout, although they can occur together.

a. Hypoxic Blackout due to Hyperventilation and Breath-holding

There are some people who find the flaunting of safety mechanisms an overwhelming challenge. They may be trying to swim or stay underwater for as long as possible (such as swimming the length of a swimming pool, or to impress their peers or girl friend). The break point can be delayed by **hyperventilating** (taking a succession of rapid deep breaths) before a dive. This reduces lung and arterial CO₂ so that it takes longer for the blood level to reach the break point during a dive. During this delay, the blood O₂ level may fall below that necessary to maintain consciousness and the diver may become unconscious without any warning. This is one cause of **Hypoxic Blackout**. This can occur at any depth, such as in a one metre deep pool. Using this method some divers have been able to prolong their breath hold dives for extended periods — until the body is found!

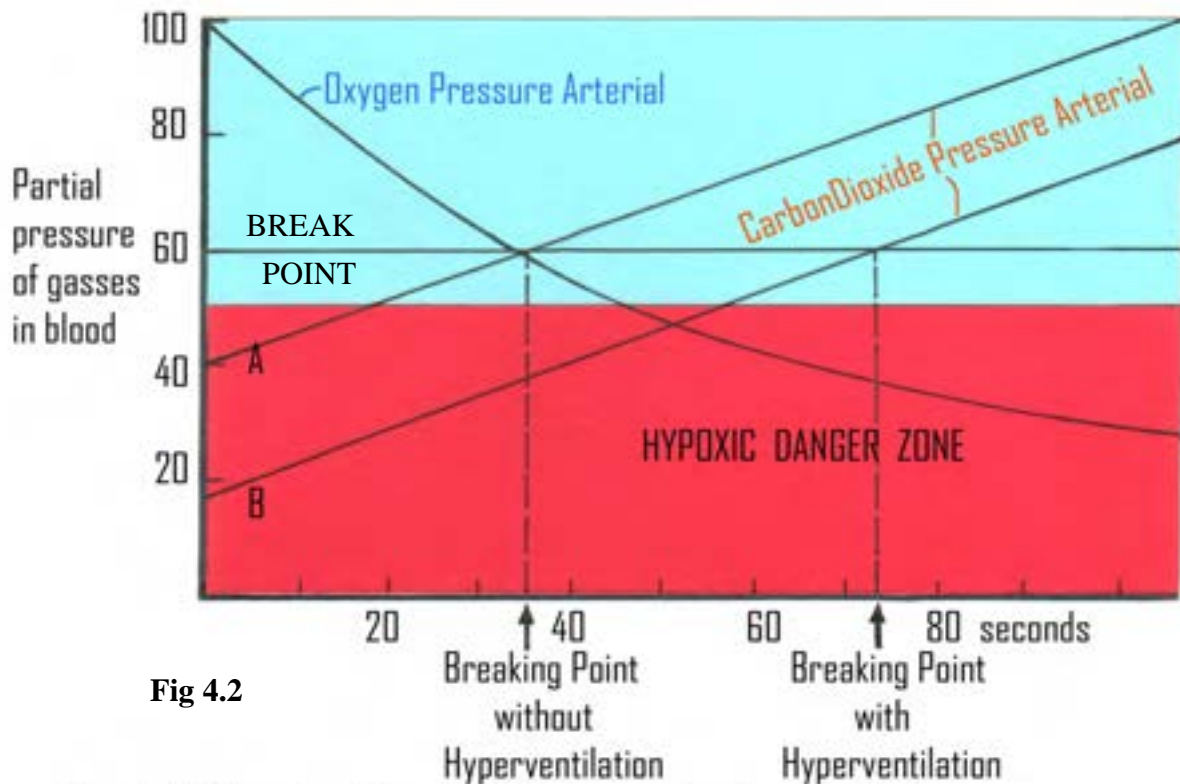


Fig 4.2

Breath hold breaking point- Carbon dioxide } A without preceding hyperventilation
 B with preceding hyperventilation

This diagram shows the relationship between the fall of oxygen and carbon dioxide levels in the blood with breath-holding. Normally, with breath-holding, (A) the breaking point is reached before the hypoxic zone is reached. After hyperventilation and breath-holding, (B) the breaking point is in the hypoxic zone.

b. Hypoxic Blackout due to Hypoxia of Ascent

Most divers will have noticed during a breath-hold dive that the desire to breathe often decreases with depth. This is probably due to the partial pressure of O₂ in the lungs increasing as they are compressed. There is a corresponding rise in the partial pressure of O₂ in the blood which will reduce the hypoxic stimulus to breathing. At depth the diver continues to exercise and use up his O₂ reserves. As the diver ascends however, the lungs will expand and the partial pressure of O₂ in them will correspondingly decrease. This produces an abrupt reduction in the O₂ partial pressure in the blood. It may fall below the threshold and cause unconsciousness during or immediately after ascent. This phenomenon is termed **hypoxia of ascent**. It may even occur after a diver has taken the first breath after surfacing, because of the delay between taking this breath and the replenished oxygen reaching the brain.

Some divers, especially those who are spear fishing, taking photographs or deep breath-hold diving for other reasons, will not only hyperventilate first, but then dive deep until they feel the necessity to breath (the break point). These are exposed to the dangers both of hyperventilation and breath-holding, and also to hypoxia of ascent. Death in such breath-hold divers is common, as unconsciousness occurs without warning.

Hypoxic Blackout - Prevention

1. Do not hyperventilate before breath-holding
2. Do not dive deep
3. Avoid excessive exercise
4. Avoid competitive behaviour resulting in the above activities
5. Ensure buoyancy near the surface
6. Dive with a buddy or have a safety observer/rescuer
7. Educate breath-hold divers of this hazard

Snorkel Diving

All the difficulties associated with breath-hold diving occur with snorkel diving – which is just breath-hold diving with a snorkel. Snorkel breathing is a convenient way of obtaining air whilst on the surface and with the head immersed, however it has several physiological and physical limitations due to the snorkel's structure (see Chapter 5).

MEDICAL CHECKLIST FOR SNORKELLERS

Have you ever had any of the following medical conditions :

- | | | |
|--|-----|----|
| 1. Any cardiovascular disease?
(Heart, blood pressure, blood, etc). | YES | NO |
| 2. Any lung disease?
(Asthma, wheezing, pneumothorax, TB, etc.) | YES | NO |
| 3. Any fits, epilepsy, convulsions or blackouts? | YES | NO |
| 4. Any serious disease? (Such as diabetes) | YES | NO |
| 5. Serious ear, sinus or eye disease? | YES | NO |
| 6. Any neurological or psychiatric disease? | YES | NO |

Over the last month have you had any of the following :

- | | | |
|---|-----|-----|
| 7. Operations, illnesses, treatment? | YES | NO |
| 8. Drugs or medications? | YES | NO |
| 9. If female, are you pregnant? | YES | NO |
| 10. Can you swim 500 metres without aids? | NO | YES |
| 11. Can you swim 200 metres in 5 minutes or less? | NO | YES |

NAME :

DoB :

ADDRESS :

(If under 16 years, guardian to sign.)

Note : If the candidate indicates an answer in the left hand column, then further investigation or action is required before snorkelling is considered as safe.

Chapter 5

DIVING EQUIPMENT

This chapter has been included to explain the operating principles and the limitations of some of the equipment currently used in free and scuba diving.

FREE DIVING EQUIPMENT

Mask

The reason we go diving is to view the spectacular underwater world. Unfortunately, our eyes are designed to see through air. In water the view is blurred and distorted – and somewhat magnified. The latter is the reason why divers exaggerate the size of the fish they see. Fishermen, who examine their catch on the surface, do not have such an excuse for their blatant lying.

The way we compensate for the water/air interface distortion, is to add an air space in front of our eyes. This can be achieved by air-filled contact lenses, swimming goggles or face masks. The last of these is used by divers.

The variety of face masks on the market suggests that the ideal mask has not yet been developed for the multitude of different face shapes.

The mask should cover the eyes and nose but not the mouth. Having the nose included allows the diver to exhale into the mask to compensate for the changes in water pressure, and so prevent **face mask squeeze** (see Chapter 12). The ability to exhale into the mask is also essential to clear a face mask flooded with water. The mask should be shaped so that the diver's fingers can reach and block his nostrils, to make ear equalising easier.

Ideally, the mask should have a small air volume so as to reduce the effort needed to equalise water pressure during breath-hold diving. The face plate should be close to the eyes, to maximise the field of vision. With a basic face mask this is limited by the nose. This problem could possibly be overcome by radical nasal surgery, but is best achieved by an indented rubber nose piece which allows the glass to be brought closer to the eyes. Clear plastic or glass side panels may also possibly help. Although this arrangement generally improves peripheral vision, the nose piece still restricts downward vision – obscuring the harness, weight belt and emergency gear.

Some face masks are fitted with a one-way exhaust or “purge” valve on the undersurface to aid elimination of water. This makes clearing the mask of surplus sea water much easier, but the valve can be an annoying source of water leakage into the mask, if it does not function correctly. Another source of leakage is a moustache or beard. These are still able to permit a seal if adequately wetted beforehand.

The body of the mask may be made of plastic, rubber, or silicone. The material needs sufficient rigidity to maintain the basic shape of the mask but a soft flanged edge (or skirt) is necessary to allow the mask to adapt to the contours of the face and provide a watertight seal. If the mask is excessively rigid it will not accommodate to water pressure changes, making face mask squeeze more likely. Masks made of silicone rubber are available for use – essential for divers with rubber or plastic allergy.



Fig. 5.1
Mask, snorkel and fins – basic free diving equipment

The viewing plate of the mask can be made of either glass or plastic. Masks, like the windscreens of cars, should be made of “tempered “ or safety glass, thereby preventing shattering and damage to the face and eyes, in the occasional event of trauma. Then it will shatter into small cubes rather than sharp splinters when broken. Good quality plastic is less likely to break, but is prone to scratching



**Fig. 5.2
Silicone
masks**

A mask can be chosen by fitting the mask to the face and gently inhaling through the nose. A mask which seals adequately will then adhere to the face without any air leak, and not fall off. The mask should seal properly without excessive tension from the mask strap. Some mask straps are broadened at the back of

the head to distribute the tension over a greater area, as narrow bands are less secure and often cause local tenderness or headaches.



CAN YOU EASILY SEE?



Fig. 5.3

Diving masks with corrective lenses incorporated into or glued onto the faceplate.

For those divers who are myopic, they may wear soft or fenestrated contact lenses, or have negative corrected lenses built into the face plate. Lenses of differing refractive index are now available which slot directly into some masks, replacing the "blank" lenses. For the "oldies", who need spectacles to read their gauges and see the miniscule aquatic animals and plants, they may stick a positive lens onto the face plate, low down and on one side only.

Snorkel

The snorkel allows the diver to breathe while floating, with the face submerged. Otherwise he would have to disrupt his view of the underwater world to turn his head to breath – like swimmers. It can also be used during periods of surface swimming before or following a scuba dive, to conserve compressed air or return to safety without relying on the scuba cylinder, which may be near empty.

Because of the limited strength of the respiratory muscles and the effect of water pressure, it is not possible to breath through a snorkel at a depth in excess of about 50 cm. The length of the snorkel should be sufficient to allow the diver to swim face down, to look around and to swim through choppy water without the snorkel flooding. It should not be excessively long as this increases

breathing resistance and respiratory "dead space". The optimum length is about 30–35 cm.



The snorkel should be of maximum diameter to reduce breathing resistance but not wide enough to create excessive dead space. The optimum internal diameter is approximately 1.5–2 cm. It should have a minimum of angles and curves, and the interior should be smooth. Corrugated tubing or sharp angles increase breathing resistance. Mouthpieces are sometimes made to swivel and rotate in order to minimise drag on the mouth and permit a comfortable hold. The latter can be assisted by individual "bite moulding" of the mouthpiece.

Fig. 5.4

A range of snorkels. Some of these have "purge" valves for eliminating water.

The breathing resistance of a poorly designed snorkel is usually not noticed during quiet breathing, but may prevent the diver from exercising to his maximum capacity when needed. With moderately heavy breathing, as with anxiety-produced hyperventilation or swimming at >1 knot, snorkels restrict the breathing capability of divers engaged in surface swimming.

Several peculiar devices have been invented to prevent water entering the snorkel during a dive. These usually employ buoyant objects such as a table-tennis ball or cork which floats into, and obstructs the end of the snorkel when it is submerged. This requires an extra U shaped bend in the snorkel which increases resistance and dead space. These devices are unreliable and unnecessary, and often catch on other objects. Divers learn to expel water from the snorkel after returning to the surface by turning their head towards the side with the snorkel and blowing hard and fast.

Some snorkels are now fitted with a small purge valve near the mouth piece and which allows most of the water to drain from the snorkel automatically. It reduces the amount of water which needs to be expelled by the diver, and therefore the effort required. It is also a potential source of leaks.

If used excessively or gripped too tightly, sometimes the jaw becomes sore after a long dive. See Chapter 32.

Fins (Flippers)

The use of fins considerably improves the diver's swimming efficiency. There are several designs available.

Fins have two basic types of foot fitting — one has a shoe integrated with the fin (enclosed heel), and the other has a half shoe fitting and a heel strap (open heels) which allows the diver to wear neoprene boots. These boots can be used for protection when walking over reefs, and offer some thermal insulation to the feet.

The blades of the fins vary in size and rigidity and some types are fitted with vents (for a venturi effect). Studies of various fin types have shown that no one type is ideal for all divers. Fins with larger, more rigid blades, allow a more powerful action but require greater strength and are more difficult to manoeuvre. Muscular cramps can result from inappropriate powerful fins. In general, fins of medium size and medium flexibility are suitable for most recreational divers.

The way fins are used is important. Traditionally, a narrow, straight-leg kicking stroke has been taught. A less graceful wide-kicking stroke using bent knees is more efficient. This comes from directing the thrust of the fin along the direction of movement of the diver. Beginners may need coaching to avoid a bicycle peddling action, which is ineffective as a swimming stroke.



Fig. 5.5

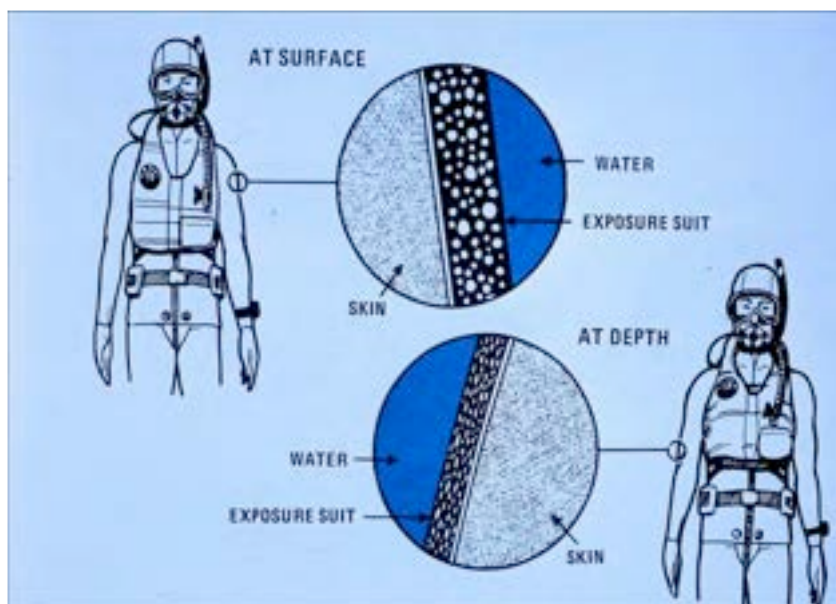
Fins with integrated shoes can often cause blistering and abrasions ("fin ulcers") on the foot or ankle, around the rim of the shoe fitting. This can be reduced by the use of socks until the diver becomes more accustomed to the particular fins. Correct fitting of the fins is necessary. If too loose, their loss will endanger the diver. An excessively tight fit may cause muscular cramps and fin ulcers.

New divers tend to rely on hand movements for propulsion. This is not effective underwater and they have to learn to employ fin propulsion

Wet Suit

The wet suit provides protection, comfort and safety. It is made from rubber or neoprene which incorporates tiny air bubbles, to provide good thermal insulation. It also provides protection from scratching, abrasions and stinging animals.

On the surface and in shallow water, these suits give great buoyancy. To overcome this effect and therefore submerge, accessory weights are usually necessary. At depth however, the air bubbles in the wet suit are compressed (remember Boyles Law?), reducing its thickness, buoyancy and insulating properties. The variations in buoyancy at different depths may need to be offset by the use of a buoyancy compensator.



A poorly fitting wet suit can cause chafing, especially around the neck and arm-pit. A wet suit with an excessively tight neck can also compress the blood vessels to the brain leading to dizziness and fainting. Tightness around the chest may cause difficulty in breathing. Zippers allow for easier access and exit, but contribute to water leakage and reduced thermal reliability.

Fig.5.6

A wet suit variant containing inflatable gas compartments can partly overcome these problems. These suits can be inflated orally

or directly from a scuba tank. Careful venting on ascent is necessary in order to prevent too rapid an ascent with these suits as the gases expand. They are a modern version of the traditional "dry suit". The latter is used in colder waters, when a layer of gas is injected between the rubberised suit and an insulating undergarment. A gas cylinder adds air into this space during descent, and the air is exhausted during ascent, to maintain neutral buoyancy. Because of the added buoyancy problems, special training is needed to use dry suits. See Chapter 12 for suit "blow up". Urinating in dry suits is problematic, and the P-Valves that permit urinating into the ocean can cause rare but serious problems with retrograde flow of sea water, air and infections into the genito-urinary tract

There are **hybrid suits** that include characteristics of both wet and dry suits, and some include fluids and malleable solids that replace the gas spaces and avoid the variable buoyancy effects with depth.

Weight Belt

A weight belt is used to offset the buoyancy of the body, wet suit and other items of equipment. Ideally the diver should use enough weight to produce neutral buoyancy at the surface (without reliance on a buoyancy vest) or at a shallow depth, about 3-5 metres – where a safety stop is often indicated. The correct amount of weight is found by trial and error and this should be done in shallow water. As the diver descends, compression of the wet suit makes the diver less buoyant. This effect can be offset by the use of a buoyancy compensating vest (B.C.).

A diver without a wet suit will usually require less than 2 kg (5 lbs) weight and many divers will require no weight at all. A diver wearing a wet suit may require about 1 kg weight for each 1 mm wet suit thickness, with an extra 1 kg for neoprene booties or hood. Inexperienced divers tend to use more weights than experienced divers, and are therefore more at risk from buoyancy problems.

Fig. 5.7

The deliberate attachment of up to 10 kg of lead weight, or more, to an otherwise neutrally buoyant air-breathing creature in the water has obvious safety consequences. It aids in descent, but may impair the





ability to surface safely. "Lead poisoning" is a common contributor to recreational scuba diver deaths

Most weights are moulded lead shapes through which the belt is threaded. For comfort these are sometimes curved, and some newer belts incorporate zippered compartments filled with lead shot for better fit to the body. Weights are usually sold in 1, 2, or 3 kg. sizes.

Fig 5.8. Being over weighted is one of the most common faults of new divers.

Fig. 5.9
Sitting on the gunwale with a weight belt on, is living dangerously.



The weight belt should be fitted with a quick release buckle, preferably one which is separate from the scuba harness release. Exceptions to this requirement are found in saturation diving and in cave diving where a sudden ascent due to inadvertent release of the weight belt could have catastrophic consequences. The buckle should be easily identified by feel and therefore different from the harness buckle. The strap should not be too long or it will hinder quick release

The weight belt should fit firmly around the waist. If it does not, compression of the wet suit at depth may result in it becoming loose and rotating around the body, with the buckle becoming inaccessible. Some new belts are made from elastic material which conforms regardless of depth.

In a significant proportion of diving accidents, the diver fails to release the weight belt at the time of the emergency. Training of divers is required to ensure that release of the weight belt is routine in an emergency. When ditching the weight belt, the diver should release the buckle with one hand and hold the weight belt well clear of the body with the other, before dropping it — otherwise

entanglement with other equipment is possible. Unbuckling the weight belt *per se* will not necessarily cause it to fall.

The attachment of weights to the diver using rope or an ordinary belt buckle which cannot be rapidly released, has sometimes proved more permanent than the diver would have wished.

The weight belt should always be the last item of equipment put on before entering the water, and the first removed before leaving the water. If this advice is followed, then an inadequately equipped diver who does fall back into the water, is more likely to float and not sink or drown.

Diving Knife



Contrary to the popular Hollywood image, the diver's knife has limited usefulness in fighting marauding sharks. It is, however, an essential item of safety equipment which can be used to cut the diver free from entanglements such as rope, kelp, fishing lines and nets. Scissors may be more effective for this.

Although stainless steel blades resist rust, inferior quality steels do not hold a cutting edge well. The knife should be of robust construction and of a reasonable size. It should be strapped to the diver at a location where it will not cause snagging (e.g. the inner surface of the calf or arm), and easily accessible. It should not be attached to any item of equipment, such as the weight belt or scuba harness, which may be ditched in an emergency.

Fig. 5.10

Spear Guns



Fig 5.11

Although there is usually some value to specific bits of diving equipment, with inevitable problems accompanying them, the only piece of common diving equipment which can be universally condemned, is the spear gun. The senior author is so scared of divers carrying this equipment, that he departs the water as soon as one is observed. Sometimes the injury is to other divers, sometimes to the spear fisherman himself. The latter was the case in the fig 5.11. He managed to spear himself with the spear (seen as the white rod in the X-ray) penetrating his soft palate, the optic chiasm (nerves to the eye), the sinus and a lot of the frontal part of his brain (which he probably was not using, as he possessed this implement).

COMPRESSED GAS DIVING EQUIPMENT

The use of this equipment has given divers a high degree of freedom underwater and the capacity to go deep and stay there for long periods of time.

Strictly speaking, the term "**scuba**" refers to all **self contained underwater breathing apparatus** but these days it is generally restricted to open-circuit transportable air equipment only (initially called the "Aqualung"). With this equipment the diver breathes compressed air from a cylinder carried on his back, and then exhales into the water.

Other equipment used by divers includes **surface supply compressed air breathing apparatus** (Hookah or SSBA) and **rebreathing apparatus** (semi-closed or closed circuit). **Closed circuit** and **semi-closed circuit rebreathing apparatus** allows a diver to rebreathe some of his exhaled gas. It includes a chemical "scrubber" or absorber to remove exhaled CO₂. By re-using exhaled gas it makes economical use of the gas supply, as well as minimising bubble release into the water. They have obvious advantages for military, technical and commercial operations. See Chapter 43.

SCUBA

There are two basic forms of this system — the **twin-hose system** and the **single-hose** type. The twin-hose system is rarely used now.

The single-hose unit uses compressed air contained within a steel or aluminium cylinder ("**scuba tank**"). It is usually filled to a pressure of 150–200 Bar (2250–3000 psi). Some systems developed in Europe improve endurance by utilising cylinder pressures of approximately 300 Bar (4500 psi). New cylinders manufactured from alloy-mix materials permit greater pressures, and are smaller and lighter. In most countries, laws require that all cylinders are visually and hydrostatically tested every 1–2 years.

A **cylinder valve** fitted with a mechanical tap and connecting fitting is threaded into the neck of the cylinder. Standards require the fitting of a "**burst-disc**" to this valve so that this will burst before the cylinder in the event of overpressure.

A "**first-stage**" **pressure reducing regulator** attaches to the cylinder, usually by a universal screw-on or clamp fitting. This regulator reduces the pressure of the gas in the cylinder of 150–200 Bar to an intermediate pressure of 7-10 Bar (100-150 psi) above the local pressure and supplies air at this pressure to an **air hose** which passes over the diver's shoulder. The first stage regulator is thus designed to adjust the pressure in the air hose to the water pressure at the depth the diver is swimming (the environmental or "ambient" pressure). It automatically maintains this pressure differential as the diver changes depth.

Open-circuit Scuba

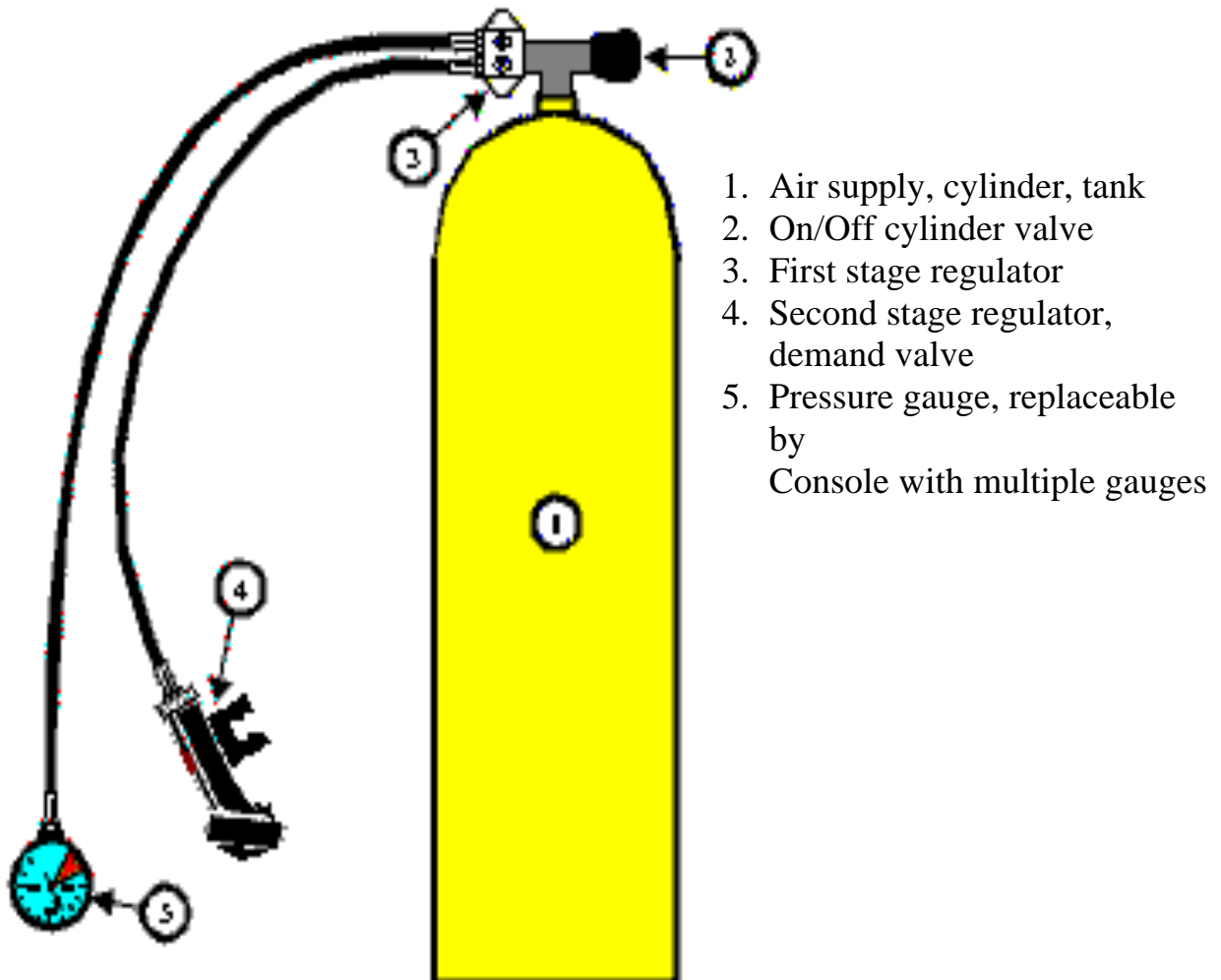


Fig. 5.12

A basic scuba set, without harness, console, octopus reg. or buoyancy compensator

The **air hose** is a small diameter flexible tube made of pressure resistant material which carries air from the first stage regulator to a **second stage regulator** (or **demand valve**) which in turn supplies air to the diver through the mouthpiece. With inhalation, a diaphragm moves to open a valve in the demand regulator, and air passes from the air hose to the diver, at the environmental pressure.

The diver exhales directly into the water through one or more one-way valves which should prevent water from entering the demand valve during inhalation.

It is important that the pressure of the air supply to a diver does not vary as the scuba cylinder empties, otherwise it will become progressively more difficult to breath as the tank pressure falls. Modern first stage regulators are much improved on the earlier models and have incorporated devices such as "balanced" valves to reduce this problem to some degree.

With most equipment, it is necessary for the diver to create a slight negative pressure in the mouthpiece during each inhalation in order to activate the demand valve mechanism. This negative pressure should be minimal or breathing becomes tiring. A regulator which is easy to breathe from at the surface, may not necessarily be able to deliver the large gas flows required during exertion at depth. When choosing a regulator, divers should refer to independent (e.g. U.S.Navy) testing.

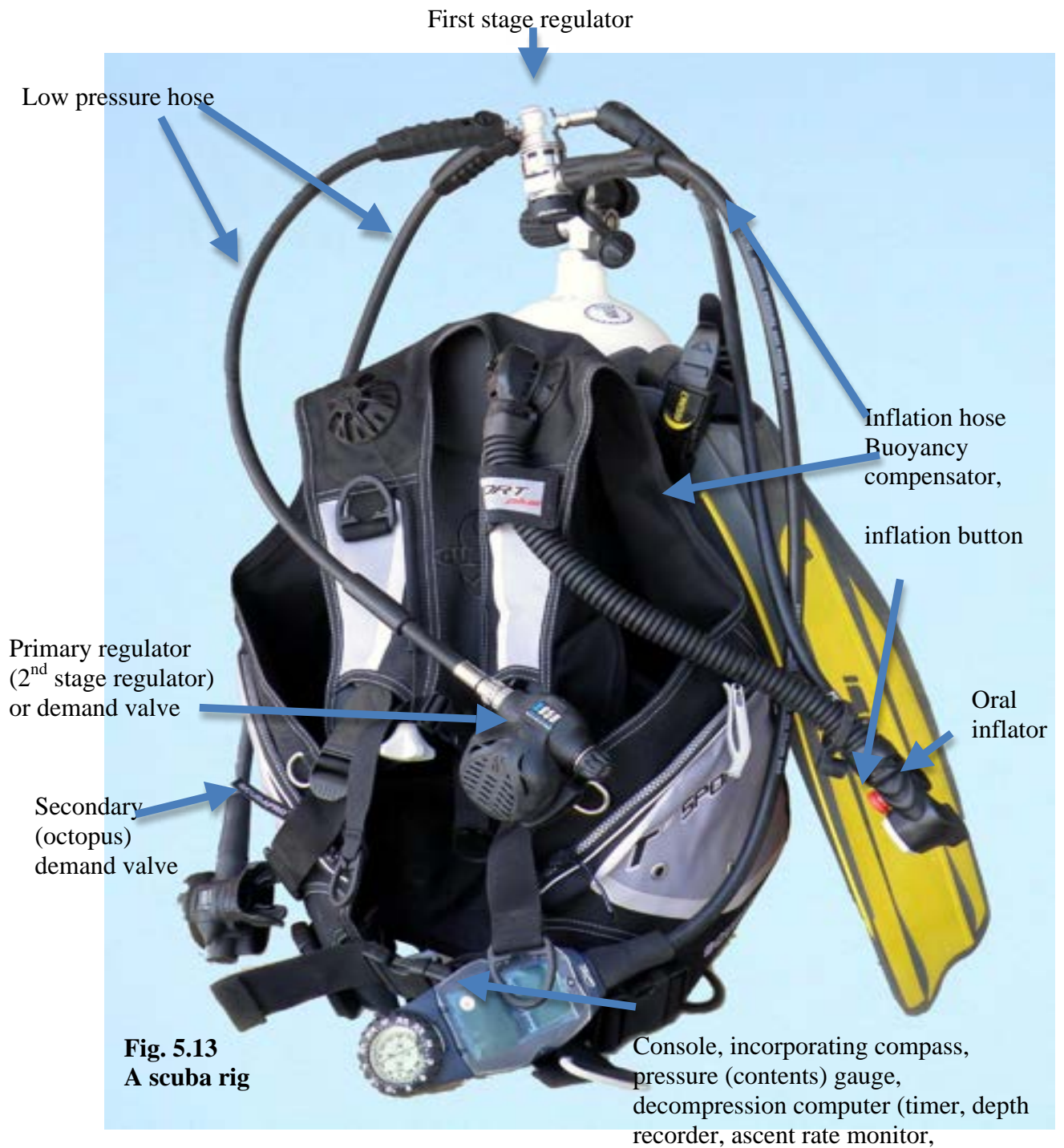


Fig. 5.13
A scuba rig

Difficulties are still encountered in obtaining adequate air supply with reasonable respiratory efforts under the following conditions :

- low cylinder pressures (observable on contents gauges), < 50 Bar
- cylinder valve not fully opened
- resistance in the first or second stage regulators (poor design or inadequate maintenance)
- increased respiration (exertion, hyperventilation, negative buoyancy etc.)
- at greater depths where the air breathed is more compressed (dense), > 30 metres
- with other demands on the air supply (inflating buoyancy compensator, octopus reg. etc.)

Some demand valves are bulky and quite heavy, requiring continual tension on the bite and the jaw to retain the mouthpiece. This can lead to painful spasm of the jaw muscles and a dysfunction of the jaw (temporo-mandibular) joint (see Chapter 32). Malleable plastic mouthpieces are available which attempt to spread the load evenly over the teeth. A soft silastic mouthpiece may be more valuable. Lugs attached to the mouthpiece are designed to keep the mouth open slightly, in a comfortable position and to locate and retain the demand valve correctly in the mouth. It should not be necessary to grip the lugs tightly.

Cylinder Valve

The gas outlet from the cylinder to the regulator is controlled by a high pressure valve or tap. High pressure "burst discs" are fitted by law to all scuba cylinder valves to minimise the risk of explosion if the tank is over-pressurised.

A common problem with divers is when they open the valve to check tank pressure, then close it to prevent accidental air loss *en route* to the dive site. The high pressure remains in the hoses and so the pressure gauge reads "full". There is enough gas in the hose to permit a breath or two as the diver descends. Very soon he finds the supply of gas suddenly depleted, the pressure gauge then reads "zero", or nearly so, and a rapid and embarrassing ascent is required.

Twin Hose Scuba

The twin or dual hose unit has both a first and second stage reducing valve combined in a single module attached to the cylinder yoke. Air is delivered by an intake hose to the diver's mouth at a pressure equal to the surrounding water. An outlet hose exhausts the exhaled air to the regulator for release to the water.

Since the diver's exhaust gas bubbles are released behind the head from the regulator, they tend not to interfere with vision. The twin hose apparatus has the disadvantage of requiring two bulky corrugated air hoses of around 2.5–3 cm diameter, and it is more difficult to purge the system of water. These units are rarely used today, except by photographers and in sites where regulators may freeze. The twin hoses were very prone to perishing and leakages.

HOOKAH and SSBA

Air can be supplied to the diver by a hose from the surface, either from a compressor (**hookah unit**) or from a cylinder or bank of cylinders (**surface supply breathing apparatus - SSBA**).

The air from SSBA is supplied directly to the demand valve at a pressure which is manually preset according to the depth at which the diver is operating. The first stage reducing valve (regulator) is located on the cylinder at the surface, and can be adjusted according to the diver's depth. This system can allow almost unlimited diving duration, which poses a risk of decompression sickness if the depth and time of the dive is not monitored.



Fig. 5.14

A hookah compressor and motor with capacity for two divers.



Fig 5.15
Surface supply

If the gas pressure in the hose from the surface fails due to a hose rupture, compressor failure or an empty cylinder, a pressure gradient can rapidly develop between the diver's respiratory tract and the failure site. Unless a non-return valve is incorporated in the gas supply line, near the diver, this pressure gradient can result in parts of the diver returning to the surface through his air hose. See Chapter 12.

Surface hookah units usually include a small pressurised reservoir as an emergency supply for breathing in case of compressor failure. Many divers carry small compressed air cylinders with them underwater ("pony bottles" or "bail-out bottles") which are able to be operated manually in the event of a main supply failure.

STANDARD DRESS or HARD HAT

This traditional piece of equipment uses compressed air delivered by a flexible hose to a rigid brass or copper helmet, usually connected to a heavy duty dry suit. The depth of the dive determines the pressure of the delivered air. A continuous air flow is supplied to the helmet at a rate sufficient to supply the diver's oxygen needs and to flush out exhaled gas. Originally hand powered compressors were used, later superseded by motorised compressors. A bank of compressed air cylinders can also be used, as with SSBA.

This system is bulky and requires heavy lead weights (usually boots and chest corsets) to offset the buoyancy of the helmet and the suit. Failure of the gas supply to keep up with the diver's rate of descent, or loss of the air supply (in the absence of a non-return valve), can lead to the diver being compressed into the helmet — causing head or body barotrauma. See Chapter 12.



Fig. 5.13

Fig. 5.16
A "hard hat" or
standard dress rig.



A modern variant of this system is used today in deep diving. It utilises a smaller, light-weight fibreglass or aluminium helmet or mask in conjunction with a dry or warmed wet suit, enabling the diver to swim and move more freely. The tethering line may go to the surface or a diving bell. The diver usually breathes gas mixtures which include helium, to prevent the development of nitrogen narcosis.

Fig. 5.17

A modern professional diving mask in rear with Standard Dress helmet in the foreground.

Closed and Semi-closed Circuit REBREATHING APPARATUS

With this equipment some or all of the diver's exhaled gas is passed through a carbon dioxide absorber ("scrubber") and then rebreathed from a breathing bag ("counterlung"). This minimises gas usage, produces fewer bubbles and allows smaller cylinders to be used for an equivalent dive duration.

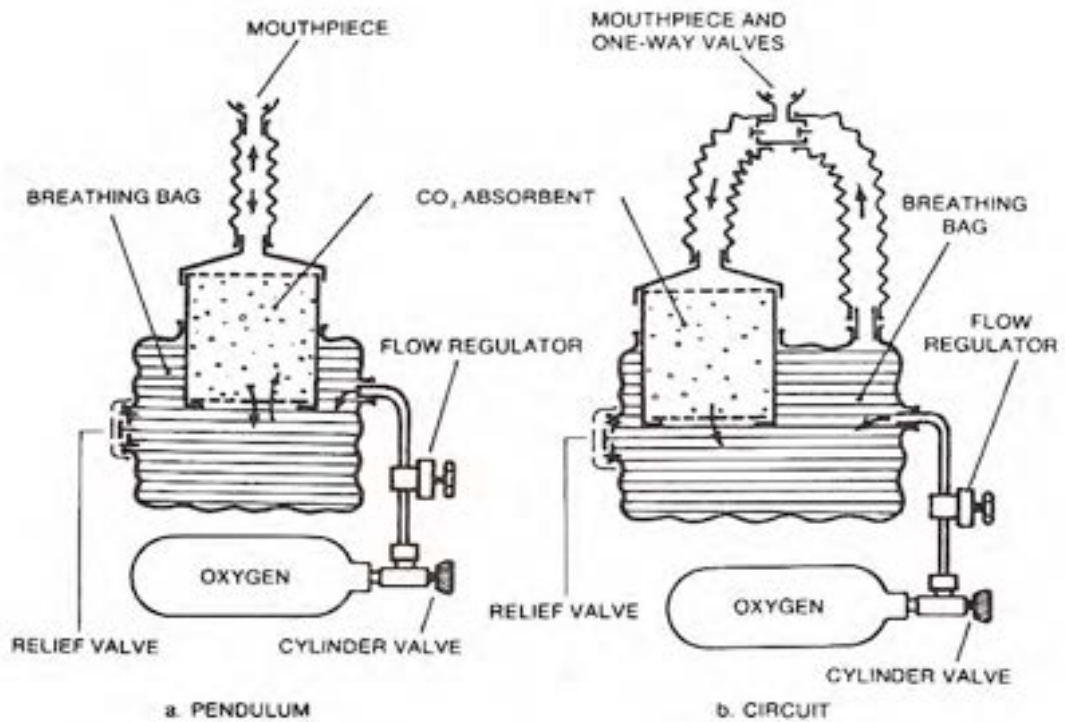


Fig. 5.18

Diagram of two types of closed circuit oxygen rebreathing sets.

A military system, using 100% oxygen in a closed circuit, is employed for clandestine operations (blowing up ships, examining potential landing sites, etc.). Because the diver rebreathes 100% oxygen, there is a risk of oxygen toxicity, so these sets have a practical depth limit of 9 metres.

Some of these sets have a demand-type system, where gas is supplied automatically when the volume in the counterlung is reduced. Others have a continual gas supply, with excess being exhausted.

Closed circuit mixed-gas rebreathing systems are used in technical and deep diving operations. These are further described in Chapter 43, but are not recommended for use by other than very experienced and meticulously trained divers.



Fig. 5.19 Military diver wearing oxygen rebreathing sets.

ANCILLARY DIVING EQUIPMENT

Buoyancy Compensator, Buoyancy Vest, B.C.

This device was originally devised as a modified life jacket to provide emergency flotation for the diver at the surface. Its value in compensating for changes of buoyancy due to wet suit compression with depth, was realised and it was modified to allow the gas content to be varied during the dive, depending on the buoyancy needs. It was also variously called a B.C.D. or B.C.V. (buoyancy compensating device or vest) or A.B.L.J. (adjustable buoyancy life-jacket)

Desirable features. When inflated the B.C. positive buoyancy should be sufficient to offset the negative buoyancy of the submerged weight of the diver and his equipment. It should support an unconscious diver so that his face is clear of the water. Ten kilograms (22 lbs) of buoyancy is more than adequate to achieve this. Most B.C.'s have excess capacity.

The B.C. should have a means of oral inflation, as well as a means of manually inflating with gas from a compressed air cylinder. With modern B.C.s the latter usually takes the form of an auxiliary "direct low pressure feed" line from the first stage or reducing valve. This direct-scuba-feed allows

the B.C. to be inflated using air from the scuba tank. This may provide insufficient or slow inflation with a low tank pressure at depth, especially if air is also needed for breathing.

Ideally there should be a separate emergency supply of inflating gas — this may be either a CO₂ cartridge or a small compressed air bottle. If a CO₂ cartridge is used it should have the ability to fully inflate the vest at depth, which usually requires at least a 20 gram capacity. The CO₂ cartridge triggering device is especially prone to corrosion and needs to be regularly maintained and inspected before each dive. The toggles which operate these cartridges can snag on passing obstructions, accidentally inflating the vest. This can have disastrous consequences in cave diving, saturation and decompression dives and other situations.

Some B.C.s are fitted with a small compressed air bottle for emergency inflation. This is activated by a rotating valve which will not open accidentally. The bottle can also serve as an emergency source for a few breaths of air if a modified demand valve is fitted to the vest. These bottles are usually charged from the main gas cylinder at the surface just prior to fitting the B.C.

The B.C. should have a pressure relief valve to prevent rupture from over-inflation on ascent. There also needs to be an easily accessible air-dumping valve to allow quick release of gas. The direct scuba feed line should also have an easily operated "quick-release" fitting at the B.C. end in case of a jammed inflator valve causing greater inflation than can be released by a dump valve.

The B.C. should be designed so that it will not ride up onto the throat when inflated. This was traditionally accomplished by fitting a crotch strap or attachment to the scuba harness. B.C.s are becoming increasingly more complex and expensive, and may contribute to diver errors and therefore injuries. Operating a B.C. requires repeated training.

Jacket B.C.s incorporating a scuba-tank backpack have become popular in recent years. These are comfortable and convenient to use, do not compress the chest and eliminate many of the straps associated with a traditional scuba-tank harness. With most of these units, however, it is difficult in an emergency to ditch an empty scuba tank on the surface without losing the B.C.



Fig. 5.20 This is the type of B.C. you do not want. One that tips the unconscious diver face down in the water

Contents Gauge

It is essential to monitor the air content of the scuba tank during a dive, to allow a sufficient air reserve for return to safety, emergency use and for decompression.

The pressure observed in the contents gauge overestimates the air available, because a substantial pressure is required just to drive air through the regulator. Thus something like 40 Bar should be deducted from the reading to calculate the remaining air available for the dive.



Fig. 5.21.
Depth and contents gauges (calibrated in feet of sea water and psig, respectively).

"Reserve" valves are not adequate substitutes for contents gauges since they may be inadvertently opened before or during the dive, and have been observed to leak or fail under operational conditions.

To gain maximum advantage from the contents gauge the diver should refer to it frequently, and should be aware of the values in respect to his own diving air consumption at that depth.

Alternate Air Source

The **octopus regulator** is a second-stage demand valve which can be used by the diver in the event of failure of the main demand valve, or which may be used by another diver who has an equipment failure or air exhaustion. The hose for the octopus or second reg. is longer than the primary reg so that it can be used easily by the OOA/LOA (out-of-air, low-on-air) diver. This facility eliminates the need for buddy breathing from a single demand valve, which can be difficult and dangerous to perform in high stress situations or between inexperienced divers.



Obviously, two divers using the same scuba system will halve the endurance of the tank. An alternative is to carry a complete separate emergency "**spare air**" unit with an adequate supply of air to reach the surface. At depth, and with a low tank pressure, insufficient air may be available for simultaneous use of the demand valve and the octopus regulator. Other alternative air sources include twin scuba cylinders (and independent regulators) and air breathing from a B.C. supply.

Fig. 5.22
A Spare Air unit

Diving Watch

A reliable, accurate, waterproof watch or dive timer is an essential piece of scuba diving equipment, in order that decompression requirements can be calculated.

The device should include some means of measuring elapsed time. A rotating bezel on the face of the watch is a simple and popular way of achieving this. It is not essential, but it is traditional, for divers to wear black-faced watches. Digital watches with elapsed time counters are also used.

Electronic dive timers, which are automatically triggered after a shallow descent, may not only record the dive duration but also the time between dives (surface interval).

Depth Gauge

It is necessary for the scuba diver to have an accurate knowledge of his depth exposure so that decompression requirements can be calculated. A depth gauge should be easily read under all visibility conditions. There are several types of depth gauge currently available. The simplest type uses an air-filled **capillary tube**. As the air in the tube is compressed during descent, water enters the capillary tube and the position of the water interface on a calibrated scale indicates the depth. This type of gauge is very accurate at depths down to about 10 metres but it is inappropriate in excess of 20 metres, due to the small scale deviations available on the gauge at these depths.

A **Bourdon tube gauge** incorporates a thin curved copper tube which straightens slightly as increased water pressure compresses the air within the tube. The movement of the tube is magnified by a gearing system which moves a needle across a scale. This type of gauge may become inaccurate due to salt obstructing the water-entry port, repetitive mechanical damage and altitude exposure.

Another type of gauge has a flexible **diaphragm** incorporated into the casing of the gauge. The diaphragm moves a needle through a magnifying gear system. This type of gauge has the advantage of relative simplicity and reliability.

Modern micro-processor technology has produced digital depth gauges which measure depth using a **pressure transducer**. This type of gauge is dependant on an adequately charged battery with water-tight integrity for reliable operation.

A device which records the **maximum depth** attained (maximum depth indicator or M.D.I.) is recommended as the diver may fail to note the greatest depth attained during a dive. This knowledge is necessary in calculating decompression requirements.

A depth gauge should be regularly recalibrated to ensure its accuracy. Some depth gauges incorporate a capillary depth gauge which will provide a cross check of calibration at shallow depth. Often depth gauges are contained in "consoles" which also contain cylinder contents gauges, timers and compasses.

Compass

Possibly one of the least appreciated pieces of equipment, until one needs it to navigate both underwater and on the surface.

Decompression Meters (D.C.M.) (see Chapter 14)

A decompression meter or computer uses a mechanical or electronic model of the inert gas uptake and elimination by the diver. The dive computers (D.C. or D.C.M.) are based on decompression theories (or the principles on which the tables were developed) but often omit some of the safety factors incorporated in the formal tables. It is impossible for them to exactly duplicate the very complex gas uptake and elimination from a living diver and to allow for individual variation. They do however, accommodate the divers need to undertake both repetitive diving and multi-level diving in a much more manageable manner than the formal decompression tables.

Most current meters also incorporate accurate devices for recording times, depths, ascent rates, cylinder contents and even water temperatures. Some provide "print-out" capabilities or connections to a computer. These enable accurate graphical representations of a diver's dive profile, and are useful to diving physicians treating cases of decompression sickness and to demonstrate where the diver went wrong. Unfortunately, sometimes it is the dive computer that goes wrong, not the diver.

Communication Systems

The safety of the buddy system of diving depends on the two divers being in constant communication. Divers who are not in constant communication are in reality only diving in the same ocean and may or may not be available to assist their buddy in an emergency. Even when they do, third party rescue is often needed. Buddy lines and buddy diving are discussed in Chapter 34.

Surface detection aids

The purposes of this class of personal equipment are to:

- allow the support boat to monitor and find divers on the surface during or after a dive
- prevent the diver being struck by boat traffic
- mark the diver's position when drift diving or while at the decompression stop
- help rescue services in lifeboats and helicopters to locate the diver



• Fig. 5.23

An inflatable safety sausage or "divers condom".

Surface detection aids include:

- Surface marker buoy, Decompression buoy, Delayed SMB, safety sausage or blob
- Red or yellow collapsible flag - high visibility, robust, bunged to cylinder
- Glow stick - for night diving
- Whistle - cheap, will only be heard by people far from engine noise
- Torch/flashlight - if at sea after night fall
- Strobe light - needs long-lasting batteries
- High pressure whistle - expensive but effective
- Orange water dye - increases diver's visibility from search helicopters
- Mirror, such as a used compact disc, to reflect sunlight or searchlights
- Red or Orange Pyrotechnic flares -for helicopters and lifeboats
- Emergency Position-Indicating Rescue Beacon (EPIRB)

A **whistle** may be of value on the surface, in attracting support from the boat crew or other divers. Another system of drawing attention and demonstrating the divers position on the surface, where most accidents either commence or end up, is a depth-resistant **distress signal** (smoke for daytime, flare for night).

A 2 metre orange plastic tube, able to be inflated by scuba or mouth, is of value and is marketed as the **Safety Sausage**. If erect, it is easily seen from aboard boats. Aircraft can identify it more easily when it is laid flat on the water surface. It is also known as the "Diver's Condom".

Underwater a diver can be contacted by a variety of transmitting and homing devices. **Lights** are of real value at night, if the visibility is good.

It is a sad fact that most divers' bodies are retrieved only after a search — and usually death occurs without the buddy-diver's knowledge. Many deaths could possibly be prevented by the proper use of such simple and cheap systems of communication.

Most divers rely on diving close to each other, with visual communication only. Variations, such as one diver leading the other or diving with a group, results in an antithesis of the buddy system – as there is no clear and complete responsibility of each diver for the other.

A **buddy-line** keeps a pair of divers in close contact. It consists of a short length of cord (2–4 metres in length and preferably of floating line) which is attached to each diver's arm by a detachable strap. Any emergency affecting one diver will soon become apparent to the buddy even if he is not watching. Possibly the only instance where the buddy line should be discarded is when snagging is likely, or if a large shark takes a serious interest in one's buddy

Chapter 6

DIVING ENVIRONMENTS

When the aspiring diver is given his formal training, it is usually in a controlled and pleasant environment. He is trained to dive in that environment – not in other environments. He often has no concept of the demands and dangers of the other environments. He may subsequently encounter these dangers without an appreciation of the consequences of his actions, until it is too late. A perusal of this chapter may introduce him to some of the problems he could face and that need to be overcome. He needs specialised training and supervision when extending his diving profile to encompass these new environmental situations,

We will present only a brief overview. Reference should be made to diving manuals and texts for further details (see Appendix A). Divers are advised to obtain expert tuition from diving organisations specialising in these environments, before they contemplate venturing into them. Even then, they should be chaperoned by divers with specific experience of these environments

WATER MOVEMENTS

Tidal Currents

Currents of several knots* are commonly found in estuaries and ocean sites frequented by divers. These cannot be matched by the relatively puny swimming speeds achievable by a diver. For a short burst, a diver can manage about 1.5 knots, but a sustained speed of about 1.2 knots is the maximum which a fit diver can reach. For a relatively relaxed dive, a current of less than half a knot is acceptable. The problems posed by currents can be lessened by correct dive planning.

Firstly the diver needs to be aware that specific currents are a factor in any planned diving location. At other times they may be predicted by the tidal charts. This information is best obtained from local divers and/or maritime authorities. Currents can sometimes be identified by the behaviour of the dive boat at anchor, with the bows usually pointing into the current.

* 1 knot = 1 nautical mile per hour = 1.85 km per hour

The "Half Tank Rule". The best technique is to plan to swim into the current for the first half of the dive and use it to drift back to the boat during the second half. The dive is divided into halves on the basis of the air supply. After subtracting the air pressure needed to drive the regulator and as a reserve, allow half the remaining gas for the swim into the current and return using the second half of the supply. In tidal areas, it is necessary to anticipate any change in direction as the tide turns, or both halves of the dive may be into current. The best time to dive in tidal areas, for both ease and visibility, is usually during slack water - between tides.

As an example, if there is 200 ATA (or Bar) in the scuba cylinder, 40 ATA would be needed as a driving pressure for the regulator. That leaves 160 ATA for the dive. Eighty ATA could be used to swim into the current, and that leaves 80 ATA to drift back to the boat, allow for navigational errors and perform a safety stop. If one had allocated gas for emergencies, this would reduce the dive times accordingly.

The **anchor line** can be used to advantage. It is much easier to make headway against a current by pulling along a rope or chain, than by swimming. If a rope is attached loosely to the anchor line at the surface and run around the side of the boat, divers can enter the water holding onto this and use it to pull themselves to the anchor line. By using the anchor line, divers can pull themselves down to within 2 metres of the bottom, where the current is often less strong. Avoid swimming onto or dislodging the anchor, which can cast the boat adrift or lift and injure the diver. Swimming around the anchor allows the diver to check the anchor's security before continuing upstream.

Another rope (a **floating** or **Jesus line**) should drift with the current from the back of the boat, for 50 metres or more. This should be supported at regular intervals by buoys or floating plastic containers. This line has earned its name by "saving the sinners" who have missed the boat or surfaced down-current.

Another technique used in locations with strong currents is **drift diving**. Because of the fast currents, all equipment should be firmly attached and snagging on environmental hazards and other divers must be avoided. A **float** is towed to mark the diver's position and allow for signals to be sent to the surface craft. A rescue or pickup boat must drift with the divers and the current to another location, where the divers are hopefully recovered. Any such boat should have a **propeller guard** if it is to be used to rescue the divers.

Divers being left and lost at sea is unfortunately not a rare event, and it is often difficult for boatmen to find their divers if the sea surface is choppy (bubbles not detectable) and waves or swells block the diver from the low gunwaled boat. A divers float or a "**safety sausage**" (a long fluorescent inflatable plastic float) is a useful backup for a lost diver after an ocean or drift dive, and can be seen for a kilometre or so. A whistle can be used to attract attention, but is difficult to hear over engine and ocean noises. Other means of attracting attention of boatmen are low pressure horns (with >100dB sounds, for > 1km.), signal mirrors (if the sun is shining, for many km.), and waterproof **smoke flares** (lasting a minute or so, and visible for up to 10 km.). EPIRB and other electronic signalling devices may send emergency signals to commercial transports, including aircraft, over many km. See Chapter 5 for information on equipment.

Surge

In shallow water affected by waves, a **to-and-fro surge** which is too strong to swim against, may be encountered. This is best dealt with by gripping the bottom (with gloves) during the adverse surge, and moving with the favourable one. A diver contending with a powerful surge can become disoriented from the violent movement, injured from impact with rocks and can succumb to panic.

Surf

A surf entry without proper technique can be hazardous. The fully equipped diver presents a large vulnerable target to waves which can quickly divest him of essential equipment while engulfing him in unbreathable, non-buoyant foam.

The recommended technique of surf entry is to approach the water backwards after donning all equipment including fins before entry. The fins and mask must be firmly attached as they are easily lost and the regulator is attached to the vest by a clip in a readily accessible position. The diver watches oncoming waves over his shoulder while keeping an eye on his buddy, who is using the same procedure. Waves in shallow water should be met side-on to present the smallest surface area. The diver adopts a wide stance and leans into the wave. The diver should descend and swim while breathing through the regulator as soon as possible. Thus he avoids turbulence by diving under oncoming waves. After passing the waves break line, ascent allows reconnection with his buddy.

Floats are towed behind the diver on entry, and pushed in front when returning. Exit is achieved by the opposite process and by using incoming waves to help with progress towards the beach.

ENTRAPMENT

A variety of ropes, cords, fishing lines, nets, kelp and other material can easily snare the diver or his bristling array of equipment. Entrapment of this type can be safely dealt with by a calm appraisal of the situation and a sharp knife. Some divers prefer to use scissors (similar to wire cutters) instead of a knife, as they are more effective in cutting through particularly tough lines made of synthetic fibres. The limited field of view inherent with all face masks complicates these problems and makes the assistance of a buddy invaluable in tracing and untangling or cutting the causes of entrapment.

Kelp

This is a giant seaweed growing in forests from as deep as 30 metres and reaching the surface. It has a long trunk with branching fronds near the surface. It occupies cooler waters and provides a fascinating but potentially dangerous diving environment.

A diver can easily become entangled and drowned in kelp, especially near the surface where the fronds are thickest and special diving techniques are necessary for safe kelp diving.

Divers help minimize projections, which cause entanglement, by wearing the knife on the inside of the leg, use flush-fitting buckles and tape over protruding equipment. The scuba cylinder can be worn "upside down" to reduce regulator entanglement. The water is entered feet first and an attempt is made to push a hole in the kelp fronds, through which the diver passes. Divers should avoid twisting and turning in the kelp. A good kelp diver is a slow diver. The area near the bottom of the kelp causes the least likelihood of entanglement.

It is important to return to the surface with an ample reserve of air to ensure that the passage through the surface kelp is careful and unhurried. If entangled, be careful when cutting kelp stalks with a diameter similar to the regulator hose – you never know...

Enclosed Environments

Caves, wrecks, under-ice and even diving beneath large overhangs are potentially hazardous environments which should not be entered without special training and planning. The following outline is by no means comprehensive. Specialised training and equipment are needed.

• Caves.

A diver in a cave usually cannot return directly to the surface in the event of an equipment malfunction or emergency. Even without these problems, it is easy to become lost and be unable to find the surface before the air supply is exhausted. The main problems are – panic, loss of visibility and navigational difficulties. The roof of a cave may collapse after air (expired from scuba) replaces the previously supporting water.

Caves are usually dark and lined with fine silt which is easily stirred into an opaque cloud by the use of fins. This is reduced with small fins, slow movements and avoiding the floors and roof. With silt, the natural or artificial illumination sources become valueless, reflecting the light back towards the diver.



All essential equipment and lights are duplicated. A compass is mandatory. Cave divers carry a spare tank and regulator attached to a manifold, with the spare regulator on a long hose so it can be used by another diver following in a narrow passage, if necessary. Totally separate emergency air supplies are recommended.

Probably the safest diving equipment to use in caves, if not deep and the penetration distance not long, is a surface supply. Then return can follow the hose, which is being withdrawn by the surface tender.

Return to the entrance of the cave is marked by a line which is dispensed from a reel by the dive leader, who goes in first. The diver follows the leader into the cave.

Fig. 6.1

This allows the way-out to be found by following the line, away from the leader. Vertical passage to the surface is marked by a heavier shot line which is less likely to entangle the diver ascending in haste.

- **Wrecks.**

Wreck diving shares many of the problems of cave diving (requiring similar preparations and precautions) as well as presenting some unique problems. In many areas the enduring wrecks are deep, adding the risk of decompression sickness and nitrogen narcosis to the general hazards.

Wrecks frequently contain physically or chemically unstable cargo, explosives and ordinance, toxic chemicals and unfriendly marine animals. Disturbed silt deep in a wreck and sharp jagged metal edges can make navigation through a labyrinth of ladders and passageways difficult. A compass may be of little help as the metal in the wrecks is often magnetised.

- **Ice diving.**

Diving under ice requires special equipment and know-how. It shares many of the hazards and precautions of cave diving but has the added complication of freezing conditions. Being trapped under ice can be an alarming experience for a diver with a frozen and therefore non-functioning regulator. Full reliance should not be placed in specialised "ice diving" regulators – in which the water is replaced by oil, alcohol or air. These can also freeze especially on the surface, using octopus regulators and with over-breathing. Attention has also to be paid to the exit procedure, as holes can "ice-over" rapidly. Protection may also be needed for surface tenders, as they may be exposed to wind and much colder temperatures than the diver, who is only at zero degrees C.

ENVIRONMENTAL VARIANTS

Cold Water

This can disrupt the performance of both the diver and his equipment. Diving in cold water requires the insulating qualities of a thick wet suit or dry suit, with gloves, boots and a hood. The wet suit, unfortunately, loses its efficiency when the insulating air layer is compressed with depth.

The cooling effect of compressed air expanding in the regulator, added to the low temperature of the water, makes freezing of the regulator a significant problem. Modified regulators which reduce these occurrences are available but cannot be fully relied upon.

Night Diving

This is not for everyone. The concept holds real fears for some divers who are perfectly comfortable diving in daylight. Because of the dangers of anxiety reactions and panic, night diving should be avoided by divers who are claustrophobic or feel excessively anxious at the prospect. The lack of visual cues can cause disorientation and imagination runs rife.

Lights well above the waterline, should be displayed on the boat and the shore exit. Torches should not be shone into a diver's face — it blinds him temporarily (destroys night vision) — but they may be directed to display one's own hand signals.

The problems centre on impaired visibility. Vision is dependent on artificial light which is very restricted and can easily fail. It is important for the night diver to be able to find and use all items of equipment by touch alone.

Detecting and rescuing divers who develop problems and surface some distance away, may be difficult. An emergency flare, strobe light or chemical light stick (e.g. "cyalume") attached to the diver's tank valve is worthwhile carrying for this eventuality, as is a whistle.

Deep Diving

Dives deeper than 30 metres have an increasing number of complications, possibly with inappropriate responses to these.

The endurance of the scuba air supply is severely limited at greater depths while the decompression requirements increases almost exponentially, adding a sense of urgency to the dive in the face of a diminishing reserve-air safety margin.

Decompression stops become obligatory for even short dives to depths in excess of 40 metres and requires the provision of extra air for this purpose. Unfortunately, the decompression tables, even if followed exactly, become less reliable as the depth increases, raising the possibility of serious decompression sickness even after a faultlessly executed dive.

Nitrogen narcosis can occur at less than 30 metres (100 feet) and progressively impairs judgement, attention, perception and an appropriate response to adversity as the depth increases. At depths in excess of 45 metres (150 ft.) mental stability, cognition and judgement are seriously impaired. See Chapter 18)

Equipment becomes more difficult to manage at these depths. Breathing through the regulator becomes harder. The buoyancy compensator takes much longer to inflate and uses more of the limited air supply. Wet suit compression reduces its insulating properties at the same time as the diver passes into colder deep water. This compression also progressively decreases buoyancy.

The environment beyond 30 metres is dark, colourless, cold, relatively devoid of marine life (although the fish and sharks are often larger), and replete with physiological hazards. In spite of this, some recreational divers feel compelled to experience it, albeit briefly because of the limited air endurance.

The authors recommend that, in view of the increased hazards and the limited diving satisfaction available in deep dives, recreational divers regard 30 metres (100 ft.) as the maximum recommended safe depth. Uneventful dives beyond this depth often impart a false sense of capability – which is then shattered when one or more things go wrong. It is then that the effects of narcosis are demonstrated.

Altitude Diving

Diving in waters located above sea level (e.g. a mountain lake or dam) introduces some potential hazards which are related to the cold temperatures at altitude and buoyancy problems with fresh water (see below). Other variations with altitude are much more important, but not immediately obvious.

Consider a dive in a mountain lake where the atmospheric pressure is half that at sea level (this would be at an unlikely altitude of about 6000 metres or 18,000 ft. elevation, but it makes the calculations easy). The pressure at the surface of the lake is that of the atmosphere, 0.5 ATA. Assume it is a salt water lake (fresh water is slightly less dense and so exerts slightly less pressure at a given depth).

The water in the lake will exert the same pressure at this altitude as it would at any other altitude.

That is, 10 metres of water will still exert a pressure of 1 ATA.

The pressure at 5 metres depth therefore will be 1 ATA, consisting of 0.5 ATA contributed by the atmospheric pressure, and 0.5 ATA contributed by the water.

The pressure at 10 metres will thus be 1.5 ATA.

One might think initially that this would give the diver a safety margin since the pressure at a given depth in a mountain lake is less than that in the ocean. The critical difference, however, is that the diver in the lake is returning to a lower surface pressure.

This can be illustrated by referring to one of Haldane's hypotheses (see Chapter 13). He indicated that a diver could spend an unlimited time at 10 metres (2 ATA) and return to the surface (1 ATA) without developing decompression sickness. In other words, a diver could return to a pressure of half the original pressure (i.e. a 2 : 1 ratio) without developing nitrogen bubbles in the tissues.

In the mountain lake, because the surface pressure is only half that at sea level (0.5 ATA), the diver need dive to only 5 metres (1 ATA) and return to the surface to encounter the same 2 : 1 "safe" ratio. A 10 metre dive exceeds the "safe" decompression ratio. This makes dive tables designed for sea level unreliable at altitude unless considerable corrections are made.

Decompression at altitude is further complicated by difficulties in estimating depth. Digital electronic gauges must be calibrated for altitude.

A mechanical depth gauge calibrated for sea level is likely to be unreliable at altitude. The gauge simply measures pressure and registers this as depth. Since the pressure at the surface of the lake is 0.5 ATA (half that of sea level), the gauge will be straining its mechanism and possibly bending the needle, trying to get its pointer past the zero stop to register what it interprets as negative depth. The gauge may only start to register a depth after it has returned to 1 ATA. This would not happen in the mountain lake until the water pressure and atmospheric pressure added up to 1 ATA – a depth of about 5 metres.

Even a capillary depth gauge, calibrated at sea level, will not really read accurately. At sea level, the air-to-water interface in the capillary will move half way along the capillary at 10 metres, since the pressure there is twice that at the surface. In the mountain lake with a surface pressure of 0.5 ATA, twice the surface pressure will be encountered at about 5 metres depth. So the capillary gauge will reach the "10 metre depth mark" (the half volume mark) at 5 metres.

The lower surface pressure also means that gas volume changes with depth are different. The gas in a diver's lungs will double in volume between 5 metres and the surface in the lake, instead of between 10 metres and the surface as would occur in the ocean. Ascent rates thus need to be reduced if the risk of barotrauma is not to increase. The gas expansion in a buoyancy vest will also be greater near the surface in the lake, which can lead to buoyancy changes unexpected by a diver used to ocean diving. Hyperventilation is more likely at altitude because the air is less dense, and over-breathing the regulator is more likely.

Flying after Diving

This creates some similar problems to altitude diving. The decompression tables were calculated on the assumption that the diver would be returning to a pressure of 1 ATA. If the diver then goes to altitude either in an aircraft or on a high mountainous road, with nitrogen still in his tissues, bubble formation is more likely because of the lower pressure experienced, and existing bubbles are liable to enlarge. Special "post diving flying rules" apply.

Diving in Freshwater and Dams

Buoyancy is less in freshwater than saltwater. Depth estimations and calculations are similarly disrupted (10 metres of seawater = 10.3 metres of freshwater). Freshwater is often still, and therefore develops dramatic thermoclines. Trees and other sources of entanglement tend to accumulate and not be destroyed as rapidly as in the sea. Some freshwater currents may cause difficulty. Chemical and sewerage pollution can be a major problem, and some specific freshwater organisms are very dangerous (e.g. *Naegleria* causing amoebic encephalomyelitis).

Dams have a specific problem with outflow below the surface. A diver may be unaware of the pressure gradient that can develop if part of the body covers an outflow orifice. Such a gradient will tether the diver underwater and may cause grotesque injuries as it forces the diver into and through the opening.

Conclusion

In this chapter, we have reviewed only some of the problems of diving in various environments and only some of the measures that can be taken to reduce the dangers. It is hoped that the reader will review this information whenever he is invited to join a diving activity different to one for which he has been trained. He may then be encouraged to undertake more specific training appropriate to his proposed dive program.



Fig 6.2 For some divers, there are few safe environments

Chapter 7

STRESS DISORDERS, PANIC & FATIGUE

INTRODUCTION

The diving equipment and facilities of the 1950's and 1960's were often spartan and the divers were to some extent influenced by the difficulties produced by this equipment and the environment. Diving was not easy, safe or comfortable and only the dedicated few were involved in the sport. The divers who survived tended to be capable and well adapted to the environment. They were “water people”.

The advent of more user-friendly equipment, together with the marketing and general popularity of diving, has seen the introduction into the sport of some divers who are less naturally suited to the environment. These divers may be more prone to **stress syndromes** when confronted with some of the threatening aspects of the marine world.

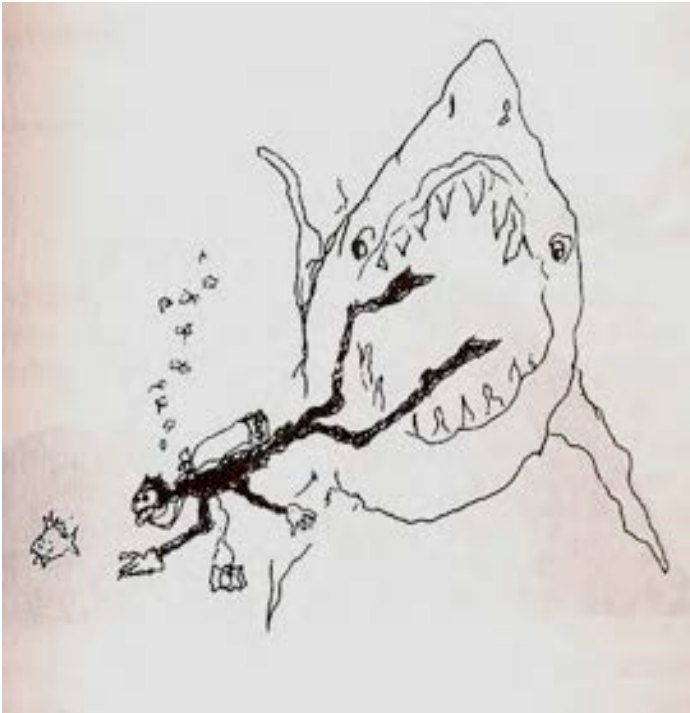
Some of the factors influencing the divers ability to cope with the diving equipment and environment will now be considered.

PERSONALITY FACTORS

Some personalities are better suited to scuba diving than others.

Military diving requires exacting physical and psychological standards and this is reflected in the high failure rate, generally about 50%. Many professional diving courses have a similar requirement and failure rate. This prompted researchers to look at the personality characteristics of successful trainees in an attempt to select out those who were not suitable.

In general, successful military divers were psychologically stable, not anxious about the dangers of diving, intelligent, practical, physically fit, self sufficient, good swimmers, capable and confident in the aquatic environment.



There is little data available for features which characterise successful and safe recreational divers. While the exacting requirements of a military diver probably are not as necessary, it would seem likely that similar characteristics would be shared by the most competent divers of both groups.

Fig. 7.1

Although there is a high failure rate with military and commercial diving courses, the failure rate in many recreational diving courses is close to zero. The standards set by some diving organisations is a source of concern, as it is possible that they may be overly influenced by commercial considerations. Between 5–10% of deaths in recreational divers occur while under training. Possibly this is the result of a combination of unsuitable people being trained in an unsafe manner. It is uncommon for a trainee to accept that he is not suited to diving, or for a diving organization to admit to less than ideal teaching methods.

STRESS RESPONSES

We all have an inbuilt automatic response to threats in the environment. This involves activation of the nervous system which prepares the body to confront the challenge or flee – the so called "**fight or flight**" response.

When this response is triggered, the sympathetic nervous system releases adrenalin into the body, stimulating the heart, increasing blood flow to the muscles, alerting the brain and increasing respiration. For example, a person suddenly confronted by a mugger is automatically primed to fight or run away. If the mugger is armed, the sensible victim usually considers the safest option is to quietly hand over money. This is an intellectual decision appropriate for survival and overrides the autonomic response. Logic can over-ride emotion.

Some divers may respond to certain levels of stress in ways inappropriate to survival. These potentially dangerous stress responses are :

- **Panic** – a psychological stress reaction characterised by excessive anxiety
- **Fatigue** – a physical stress response to exertion
- **Sudden Death Syndrome** – a lethal cardiac response to stress (see Chapter 35).

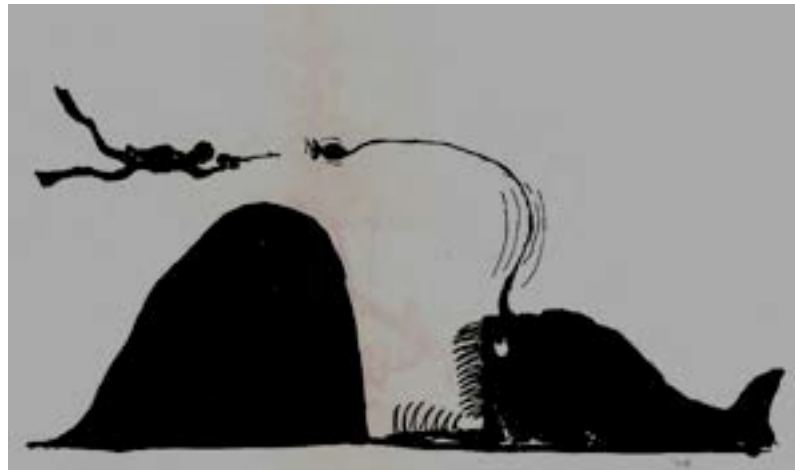


Fig. 7.2

PANIC

Panic is probably the most common contributor to death in recreational scuba diving. Studies have implicated panic as a contributor to between 40–80% of such diving deaths.



Fig 7.3

Panic is an extreme and inappropriate response to a real or imagined threat. Behavioural control becomes lost. Some readers will have experienced, or been near to, panic in some real life situations.

In general, the more naturally anxious a diver is, the more likely he is to panic.

As panic develops, the capacity to think rationally and solve the emerging problem deteriorates. The diver becomes more and more narrow minded and eventually may focus on only one goal e.g. reaching the surface – to the exclusion of other vital factors, such as exhaling during ascent.

Consider the following scene, which has been gleaned from several diving fatalities, to illustrate some of the factors contributing (in *italics*) to a panic-related death.

Case history.

Harry was a recently qualified diver who had *borrowed* equipment to undertake an open ocean dive in an *unfamiliar* area. His borrowed wetsuit was a little *tight* around his chest, restricting his breathing.

He decided to use two *extra weights* on his weight belt to help him descend in the ocean conditions, which were somewhat foreign to him. He was *inexperienced* at open ocean diving and the conditions were regarded as marginal so he felt a little *uneasy* about the dive.

His companions were more experienced than him and he was unsure of his ability to make his *air supply* last as long as his buddies. After all, he did not want to be the first to run out of air and force his buddies to shorten their dive.

During the dive he was sure he was using more air than the others but he had no way of checking this as his borrowed scuba set did not have a *contents gauge*.

He became a little more *apprehensive*. They seemed to have swum a long way both from the dive boat and the shore. But he did not want to *inconvenience* his buddy or *embarrass* himself by ascending and checking his distance from shore or inquire about his buddies air supply. He had no idea how much air he had left but he felt that there probably wasn't much.

He became a little more *anxious* and his *breathing rate* increased. He noted some *restriction* to breathing. Was this just *resistance in his regulator* or was he now running out of air?

He activated his reserve valve. Perhaps this would improve the restricted gas flow. It didn't.

There was a tidal current running, which slowed their progress to the planned end of the dive – the safe exit point.

He was hoping that his companions were also running *out of air*, as he appeared to be.

He was becoming more *anxious*. His heart was pounding and his *breathing rate* was increasing. It was becoming harder to get sufficient air from his demand valve.

The difficulty in obtaining enough air settled the matter. He decided to get to the *surface*, fast. In spite of his rapid ascent, he still did not seem to be getting more air from his demand valve. He must be out of gas.

He burst through the surface, gasping for breath. He wrenched off his *face mask* and *demand valve* and gasped air.

The water was choppy and waves washed over his face. He kicked hard with his *fins* to stay on the surface. One of the ill-fitting borrowed fins came off. A wave washed over his face and he *inhaled water* and started coughing. It was a real *struggle* to stay on the surface, he was becoming *exhausted*. He wondered how long he could keep this up. He tried to keep his head well above the waves, but could not.

His buddy noticed he was missing and after a brief search, surfaced. Harry was no where to be seen. An organised search later found his body on the bottom, immediately below where he had surfaced.

His *weight belt* was still fastened, his *buoyancy vest* uninflated. There was ample air in his cylinder and testing of his demand valve revealed normal functioning, but demonstrating the usual resistance with high gas flows.

The autopsy report read "**drowning**". The **real cause was "death from panic"**.

The above story illustrates some of the factors which combined to develop the anxiety which leads to panic and illustrates the irrational responses in a panicked diver. An appropriate logical response at any one of the steps that led to the disaster, would have prevented or relieved the situation.

Prevention.

If anxiety is an important precursor to panic, reducing anxiety is an effective counter measure. The most effective way to reduce anxiety is to have confidence in, and familiarity with, the task. This is achieved by **knowledge, training** and repeated **practice** of diving and safety procedures.

A good example is seen in the training of commercial airline pilots. They are required to fly a minimum number of hours per month and to practice and demonstrate emergency procedures at regular intervals. They spend many hours practicing emergency drills in a flight simulator. The usually cool and appropriate performance of these professionals in emergencies is a testimony to the success of this approach.

Another important preventive measure is for the diver to know his **limitations** and to dive within them. A diver may be comfortable, confident and competent in one diving situation but not in another. The first allows for safe diving, the second for a panic scenario. Panic is more likely when the diver is extending his dive parameters, especially if without competent training and supervision.

FATIGUE

Studies of recreational diver deaths show that **fatigue contributes in about 28% of cases.** This fatigue comes about from a combination of personal, equipment and environmental factors.

- **Personal.**

A high level of physical fitness is an important survival factor in diving. Even the calmest water dive can degenerate into a situation requiring maximal physical exertion due to unforeseen circumstances, such as currents, rescue requirements, etc.

During severe exertion, fatigue and its associated apathy will come sooner to physically unfit divers. Also, fatigue is experienced sooner by anxious or neurotic divers.

As a general rule, scuba divers should be able to swim 200 metres in < 5 minutes, without equipment. A fit diver will complete this in 4 minutes and a very unfit diver may take over 5 minutes.

- **Equipment.**

Much of the diver's equipment, the buoyancy compensator, tank, facemask, and wet-suit either increases resistance to swimming or restricts movement. Excessive weights make swimming more strenuous. Even the best regulators have appreciable resistance to airflow at high flow rates, significantly restricting breathing. All these factors accelerate fatigue.

- **Environment.**

Fully equipped, a diver cannot swim for long against a current of more than about 1 knot (see chapter 6). Rough water and cold exposure will make this even harder.

Chapter 8

THE FEMALE DIVER

Up to the 1960's, diving was almost exclusively a male domain with a certain associated macho image. Since then, an increasing proportion of female divers have come to enjoy this sport and have proven themselves equal to male divers in every regard.

Despite this, women are not the same as men and there are important consequences of this dissimilarity in diving activities. Unfortunately, much of the following information is based on very inadequate data.

History of Women in Diving

Probably the most famous of female diver groups are the **Ama** shell divers of Japan and Korea. These divers were originally men but the work was taken over by women, possibly because of their better tolerance to cold – the men only dived in summer while women were able to dive all year round. Another theory is that the men believed that diving impaired their virility.

The Ama underwent some remarkable physiological adaptations. During the winter months they increased their metabolic rate by 30%, which allowed them to generate more internal heat. Also, they reduced their skin blood flow by 30%. The fat content beneath their skin was increased. Both these changes improved their insulation.

There have been numerous famous women diving personalities. In the 1940's, Simone Cousteau dived alongside her husband Jacques Cousteau. In Australia, Valerie Taylor and Eva Cropp became well known because of their diving exploits. In America, Eugenie Clark was known as the 'shark lady' because of her brilliant work in this field. In 1969, Sylvia Earle led the first all woman team of aquanauts in the Tektite II habitat experiment.

Until recent years **diving instruction** was almost exclusively a male occupation. Many of these instructors basked in a 'superman' role and possessed more experience than knowledge.

In recent years women have become recreational diving instructors and have proven to be diligent and highly competent. In general they have been more keen to impress their students by knowledge and skills, rather than strength and bravado.

Women divers must be doing something right. Diving statistics show that females comprise 34% of trainees, but account for only 10-20% of fatalities (depending on the survey).

Scuba Training

In Western society, women are generally regarded as being less mechanically and mathematically adept than men. This prejudice is reflected in attitudes to diver training. In many cases, women are patronised by well-meaning male instructors and male companions.

Until recently, culturally acquired lack of assertiveness on the part of many women led them to refrain from asking what appeared to them to be naive questions of their instructors. On the other hand, prejudices by instructors led them to assume women would not be interested in, or understand, the intricacies of equipment functioning or decompression planning. This information tended to be directed towards the males in a training group. Women would often turn to a male friend or buddy rather than the instructor for answers to questions which arose during training. The information that they received was not always accurate.



A woman who has her equipment assembled and checked by a male companion, who has the equipment carried to the water and who is assisted into and out of the water, is overall less likely to become a competent and self sufficient diver.

The old stereotype of the woman in a dependent role can lead to problems in diving practice. Thus having men and women buddied together for basic training may be inappropriate. The concept, introduced by PADI some years ago, of an all-women class has much to commend it.

Fig. 8.1

Anatomical Differences

Womankind has been described as the "weaker sex." While it is generally true that on average women are less physically strong than men, there is not a vast difference in their performance in aquatic sports. For example, in the 1988 Olympics there was only a 10–12% difference in times between women and men for swimming events.

For the same physical size, men on average have greater physical strength than women. This is because men have a greater muscle mass per unit body weight. This minor difference in strength is much less significant in the weightless aquatic environment.

Being physically smaller, the woman has a lower requirement for oxygen at a given level of physical activity and will produce less carbon dioxide. With smaller lungs, women also take smaller breaths. Thus, women can often manage with less air than a male diving companion and so can use a smaller, lighter scuba cylinder. This can offset the apparent disadvantage of diminished body size and strength.

Because of differences in body shape, women have different equipment design requirements. There can be difficulty in obtaining appropriate sized or shaped wet suits, fins, boots, and gloves, in less developed countries.

Particular problems arise with male sized equipment, especially face masks which may not fit well, and large scuba cylinders which are unnecessarily bulky or heavy. Backpacks can be too long and so cover the weight belt, making the release of the belt in an emergency difficult. Over sized buoyancy compensators designed for males may give excessive buoyancy and drag with females.

Diving Activity

It is anecdotally believed that males tend to dive deeper, longer, more frequently and engage in more rapid ascents. Whether this is true, is unknown, but they certainly do engage in more risk-taking activities than females in all general epidemiological surveys, and so perhaps it is correct. They also suffer many more accidents and traumatic deaths than females – at all ages.

In one study, females dived mostly between 19-40m (61-68% of the activity) and much less between 41-60m (16-26%) or greater than 60m (1-2%). Young females dived at a more varied depth but also deeper than older females.

Thermal Variations

Women are better insulated than their male counterparts. They have a fat layer beneath the skin some 25% greater than men. They also have a better ability to constrict the blood flow to their limbs, reducing heat loss. These factors allow women to conserve their heat more effectively in a cold water environment while producing natural buoyancy, which improves their swimming and floating ability.

Unfortunately women tend to expose themselves less to demanding environmental temperatures, and so may not become as well adapted to cold exposure, and initially react more to this situation.

Menstruation

Some women perceive their ability to dive safely during the menstrual cycle may be impaired, and that the activity of scuba diving may alter the menstrual cycle. Statistically there is some justification for believing that diving related incidents are more frequent around the time of menstruation.

During menstruation, the average woman is likely to lose 50–150cc blood and cellular debris. There are some physical and physiological consequences of menstruation which will be discussed, but usually there is no reason why women should not dive during menstruation. For convenience, most women today prefer to wear internal protection such as tampons rather than

menstrual pads. In the early days, there was some concern that menstrual blood-loss may act as an attractant to sharks. In fact, females have a much lower incidence of shark attacks than males. This may be related to their different diving behaviour, or that haemolysed blood is a shark repellent.

Hormonal changes before and during menstruation tend to cause fluid retention and swelling. There is a theoretical possibility that this might encourage the development of DCS, and one study of altitude DCS suggested that DCS was more likely earlier in the menstrual cycle. This has not been validated for divers. There is minimal experimental data to support this association, but it may be wise for women, at least during this time, to add a safety margin to their decompression requirements.

Some women have significant psychological and physical problems around the time of menstruation, with abdominal pain, muscle cramps, headaches, nausea and vomiting. These may impair their diving ability. Women who suffer from severe problems of this nature, are advised to avoid diving at this time. The psychological disturbances associated with pre-menstrual tension and anxiety, may sometimes warrant the avoidance of diving during this time.

Some female migraine sufferers, have an increased likelihood of migraines around the time of menstruation. The problems associated with migraine are discussed further in Chapter 32 and the recommendations there should be followed.

Repeated exposure to high pressure environments seems to have no significant effect on hormone regulation, ovulation or menstruation.

Oral Contraceptives (the "Pill")

The physiological and psychological consequences of these hormonal tablets may have similar implications to those described above, under "menstruation". In theory, the increased coagulation effects from the pill could initiate or aggravate DCS. In practice this has not been observed. There is no relationship between the development of DCS and the taking of oral contraceptive tablets *per se*. It was considered prudent to cease oral contraceptives in the female team who undertook a long saturation dive during the Tektite No. 2 project. However, the absence of males probably made this decision an uncomplicated one.

Decompression Sickness (DCS)

Several studies have shown an increased incidence of DCS in women. Some studies of women divers showed a more than 3 times increased incidence of DCS compared with men who were exposed to the same dive profiles. Other studies have not shown this and perhaps the different observations are due to different cohorts with different dive profiles. For example, females in the space program seemed to be more susceptible to altitude DCS, and develop more serious DCS, than men.

Studies on female divers indicated that those who did "reverse profile" dives had more DCS symptoms than those who did their deeper dives first. In a large series of DCS cases it was shown that the men so affected dived deeper than the women (almost 3 metres) and it is possible that their diving exposures and ascent rates were greater.

The weight of evidence does tend to suggest that there might be an increased incidence of DCS among women. There are several possible explanations for this.

Women are frequently less physically fit than men and physical fitness is negatively related to DCS. Women usually have a higher proportion of subcutaneous body fat (+ 10%) for a given weight than men, and the body fat has a 4-5 times higher capacity for absorbing nitrogen. Fat tissue is slower to absorb and to eliminate the nitrogen. Logically, because of the different fat distribution between the sexes, studies that dealt with certain dive profiles (longer dives) could have more nitrogen absorbed and a greater incidence of DCS in females. Both reduced physical fitness and higher fat content probably increase the incidence of DCS .

Navy decompression tables were designed for and tested on physically fit, healthy young male divers. Strictly speaking, the tables should only apply to this population.

Because of this increased risk, it is wise for women divers to apply extra safety factors when using the dive tables: e.g. by reducing the allowable bottom time for any depth or by decompressing for a greater duration. Decompression computer programs should be on the more conservative settings.

A modern decompression problem has emerged with **breast implants**. Fortunately, gas filled implants are no longer used, as the barotrauma consequences of diving with these would be disturbing. However, even silicone filled implants do absorb nitrogen during a dive and a 4% expansion in the size of these implants has been recorded after dive profiles commonly used by women sports divers. This is not likely to cause a problem with the implants. However, if these women were involved in saturation diving there is the potential for significant volume changes which could lead to damage or rupture of the implant during or after ascent.

Other Diving Diseases

In a number of reports on **scuba divers pulmonary oedema**, a preponderance of females has been noted. Others have suggested an increased susceptibility to **dysbaric osteonecrosis**.

The relative risk of **oxygen toxicity** for women compared to men was 1.6 times for pulmonary toxicity and 2.9 times for neurological toxicity.

Panic is a common and potentially serious problem even amongst experienced divers. Although females reported this more frequently than males, the latter waited longer to react and so it was more likely to progress to a life threatening situation in them.

A study of dive masters and instructors found that female instructors and dive masters reported more diving-related **ear and sinus** symptoms than males. This relation was consistent even when controlling for potential confounders.

PREGNANCY

There has been considerable controversy over whether pregnant women should dive. This question arises because most women divers are in the child-bearing age group. The controversy hinges on the conflict between restricting the freedom of an individual and the risks (which have not been fully evaluated) to the unborn child. The potential problems of diving during pregnancy are as follows:

Maternal Effects

❑ Vomiting.

In the second and third months of pregnancy, many women are prone to vomiting – often manifest as "morning sickness". They are more prone to seasickness and to nausea and vomiting underwater during certain conditions. This predisposes to serious diving accidents.

❑ Barotrauma.

From the fourth month onwards, fluid retention and swelling of the lining of the respiratory tract, makes sinus and ear equalisation more difficult and predisposes to barotrauma.

❑ Respiratory function.

There is a decline in respiratory function as pregnancy progresses. There is an increase in resistance to air flow in the lungs. Later, the enlarging baby presses up into the chest, limiting breathing capacity. This combination impairs the pregnant woman's ability to cope with strenuous activity which may be required in an emergency, and may predispose to hypoxia or pulmonary barotrauma.

❑ Decompression sickness.

There are major alterations in blood volume and circulation during pregnancy. This may increase the uptake and distribution of nitrogen and may make the woman more prone to DCS.

❑ Infection.

It has been claimed that there is an increased risk of vaginal infection in pregnant women who dive. In the later stages of pregnancy some women develop minor leaks of the amniotic sac, which surrounds the baby with fluid. There is a possibility of infection of this fluid from organisms entering from the water before birth or directly into the womb after birth.

Effects on the Baby

The developing foetus is uniquely at risk from some of the physiological hazards associated with diving. The potential risk primarily consists of DCS, but hyperoxia, hypoxia, hypercapnia and increased nitrogen pressure may also be involved.

❑ Development of the foetus.

The foetus begins as a single cell organism and up until after the fourth month it is smaller than a mouse. A small bubble, such as develops during DCS, could have catastrophic effects.

The **circulation in the foetus** is unique and critical. In an adult diver, venous blood returning from the body passes through the lung capillaries, which filter out the bubbles frequently formed during or after ascent. In the foetus, the blood by-passes the lungs (since the foetus does not need to breathe) and passes directly to the left heart without passing first through this filtering network. Even one bubble forming in the tissues then veins of a foetus will be transported directly to the arterial circulation and will embolise to somewhere in its body.

In one study it was shown that diving >30 metres in the first trimester was associated with a 16% risk of foetal abnormality, but the numbers were small. Other studies have produced conflicting data.

□ Hypoxia.

The outcome of many non-fatal diving incidents is hypoxia, most likely to be caused by salt water aspiration or near drowning. The pregnant diver will not only expose herself to hypoxia in this situation but will also expose the much more susceptible foetus to this.

□ Hyperbaric oxygen.

Divers are likely to be exposed to hyperbaric oxygen in two situations. By simply breathing compressed air at depth they are inhaling elevated partial pressures of oxygen. In addition, if divers develop DCS or air embolism they will be given hyperbaric oxygen therapy for treatment of the condition.

Some foetal tissues are very sensitive to high partial pressures of oxygen. Great care is taken with newborn premature babies to avoid administration of high concentrations of oxygen because of the danger of retrolental fibroplasia, which causes blindness. The eye of the unborn baby is probably even more sensitive to high partial pressures of oxygen.

The circulation of the foetus contains a channel (the ductus arteriosus) which allows blood to by-pass the lungs. This channel closes after birth under the influence of a raised partial pressure of oxygen in the blood. There is a danger of premature closing of this and other shunts if the foetus is exposed to hyperbaric oxygen because of treatment given to the mother.

□ Decompression sickness.

As mentioned earlier, women may have increased susceptibility to DCS and there are theoretical reasons to believe that pregnant women are even more susceptible. It is known from Doppler studies that showers of bubbles are regularly formed in the veins of divers ascending from many routine non-decompression dives. These bubbles do not usually cause any symptoms.

Some experiments in pregnant animals suggest that the foetus is more resistant to bubble formation than the mother but that bubbles do form after some dives, especially those deeper than 20 metres. Because of the unique circulation of the foetus even a few bubbles in the foetal circulation can have disastrous consequences.

Experiments with pregnant animals have produced conflicting results. One study on pregnant sheep (which have a placenta similar to a human) showed that the foetus developed bubbles in its circulation even after dives of less than 30 metres (100 ft.) and within the US Navy no-decompression limits. These results are disturbing when considering the vulnerability of the foetus to any bubble.

Other studies have shown an increased incidence of abortion, birth defects and still-births in pregnant animals after decompression.

Exposure to hyperbaric oxygen has also been shown in some studies to cause birth abnormalities and death.

Human Data

Japanese female divers, the Ama, often dived until late pregnancy, and had a 44% incidence of premature delivery with a high incidence of small babies when compared with non-diving women from the same area. Another survey on modern day Ama, who were not exposed to the same stresses, did not reflect this association.

Margaret Bolton from the University of Florida carried out a survey on 208 women who dived during pregnancy. She found an increased incidence of abortion, still-birth, low birth weight and death of the infant within the first month. Of the 24 women who reported diving to 30 metres (100 ft) or more, three had children with congenital defects. This contrasts with an incidence of 1 in 50 in the general population. One of the infants had an absent hand, a very rare abnormality.

An Australian case report, showing multiple grotesque abnormalities after diving, suggested that the effects and vulnerable time frame may be similar to taking congenital-abnormality producing drug, Thalidomide, during pregnancy.

It is difficult to draw firm conclusions from these studies, or others that have been conducted, because the numbers are too low for statistical validation, or are retrospective. They are however consistent with many of the animal studies.

The Bottom Line ---

There is considerable evidence suggesting that diving during pregnancy is harmful to the foetus. It is generally accepted that unnecessary drugs, alcohol and smoking should be avoided during pregnancy because of the risk to the foetus and we recommend a similar conservative approach to diving. The sacrifice of not diving during pregnancy may be easier to cope with than the guilt, valid or not, associated with abortion or giving birth to a malformed child.

Evidence in western societies suggest that female divers have accepted the above restriction and now do avoid diving when pregnant, or when attempting to become pregnant, making the collection of more data difficult, but good for the kids.

One interesting issue to consider by women who contemplate diving during pregnancy is that the foetus, who will have to live with any birth defect which may result, cannot be consulted when the decision to dive is made.

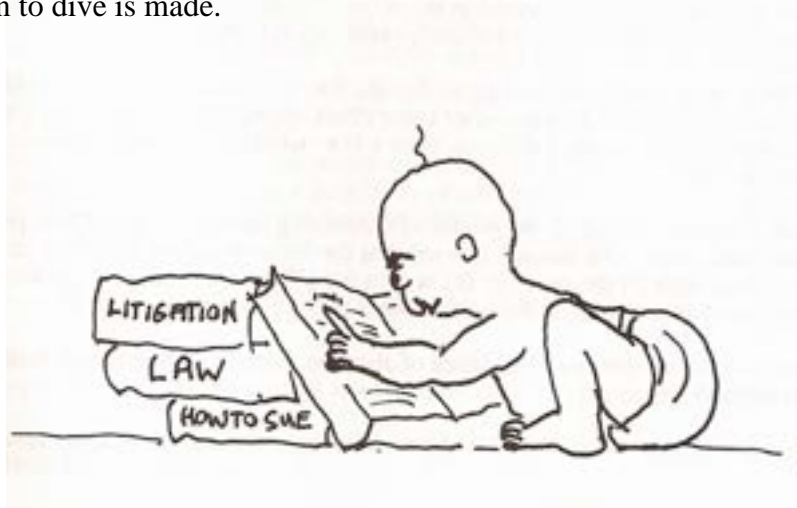
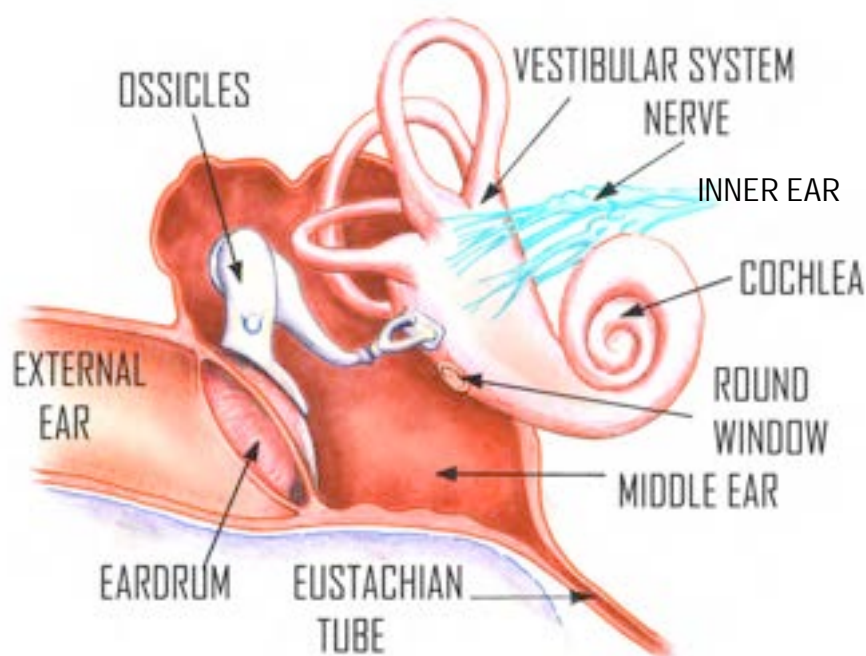


Fig. 8.2

Chapter 9

EAR BAROTRAUMA

ANATOMY OF THE EAR



The ear is divided anatomically into the outer (external), the middle and the inner ear.

The **Outer Ear** comprises the visible part of the ear (the pinna) and the external ear canal. The pinna gathers sound waves and reflects them into the ear canal and onto the ear drum.

The **Middle Ear** is a pea sized cavity enclosed in a solid bony part of the skull. It is separated from the ear canal by the paper thin **ear drum**. There are several structures opening into the middle ear space.

Fig. 9.1
Anatomy of the Ear

- The **Eustachian Tube** joins the middle ear with the throat, allowing air to enter the middle ear cavity.
- The **Mastoid Sinus** (air pockets in the mastoid bone) also come off the middle ear.

There are two openings on the inner bony surface of the middle ear space called the **Round** and **Oval Windows**, because of their shape. These openings connect the middle to the inner ear. The oval window is a tough membrane attached to the end of one of the three interconnecting middle ear bones (ossicles), while the round window is closed by a thin delicate membrane.

The **Ear Drum (or Tympanic Membrane)** is connected by the three tiny ear bones or *ossicles* - the malleus, incus and stapes - to the oval window across the middle ear space. This bony chain, which is barely visible to the naked eye, transmits the sound vibrations from the ear drum to the inner ear.

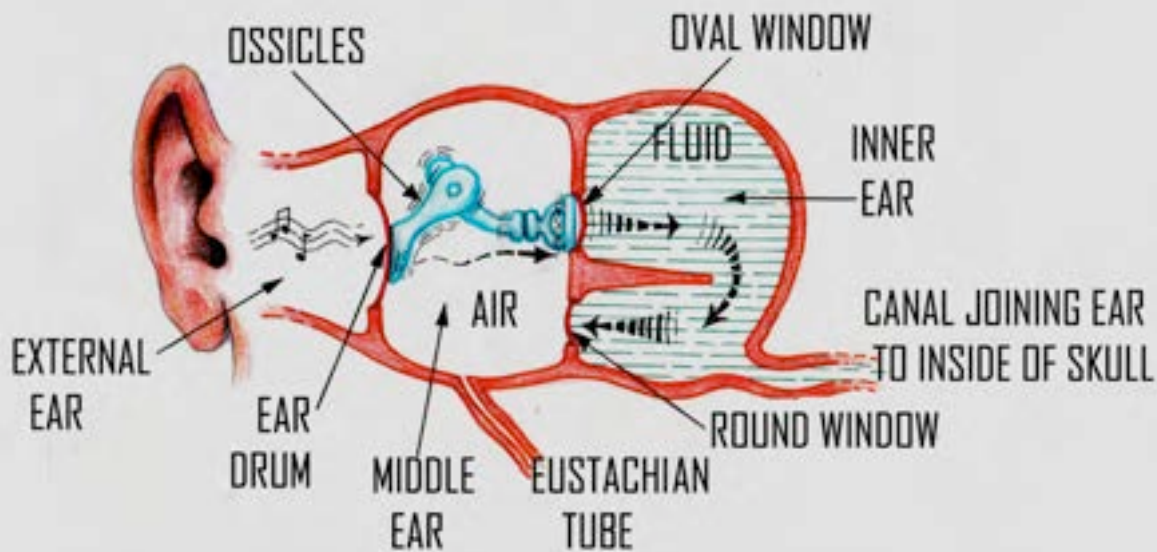


Fig. 9.2

**Middle ear communicates with external and inner ear
Transmitting sound from the exterior to the inner ear**

The **Inner Ear** contains the **Hearing** and **Balance organs**. It is entirely encased in bone and filled with fluid. The hearing organ (the **Cochlea**) is a spiral shaped structure containing fluid which surrounds nerve cells sensitive to sound vibrations.

A system of **3 semi-circular canals** is also filled with this fluid, and is the balance organ which is sensitive to position and movement. It is also called the **Vestibular System** (or **Vestibular Apparatus**).

THE MECHANISM OF HEARING

The hearing system works in an ingenious way. Sound vibrations, caught and reflected by the pinna, are directed down the ear canal causing the ear drum, at the end of the ear canal, to

vibrate. These vibrations are transmitted and magnified by the bony chain system of levers, to the oval window. The outer (air) and middle ears (ossicles) thus **conduct** sound waves to the inner ear.

Damage to the outer and middle ears interferes with the conduction of sound waves to the inner ear, and so causes **conductive deafness**. This affects the low frequency sounds (those commonly used with speech, 250-4000 Hz).

At the oval window, the sound waves are converted to pressure waves in the hydraulic fluid in the cochlea. As fluid is incompressible, the inward and outward vibrations of the oval window are compensated for by the round window bulging outward and inward, respectively.

The cochlea is tuned so that vibrations of various frequencies transmitted through it resonate in specific areas, allowing the ear to distinguish between differing frequencies of sound. This stimulates sensory nerve fibres within the cochlea and the impulses are perceived as sound when they reach the brain. Damage to this system causes **sensori-neural deafness**.

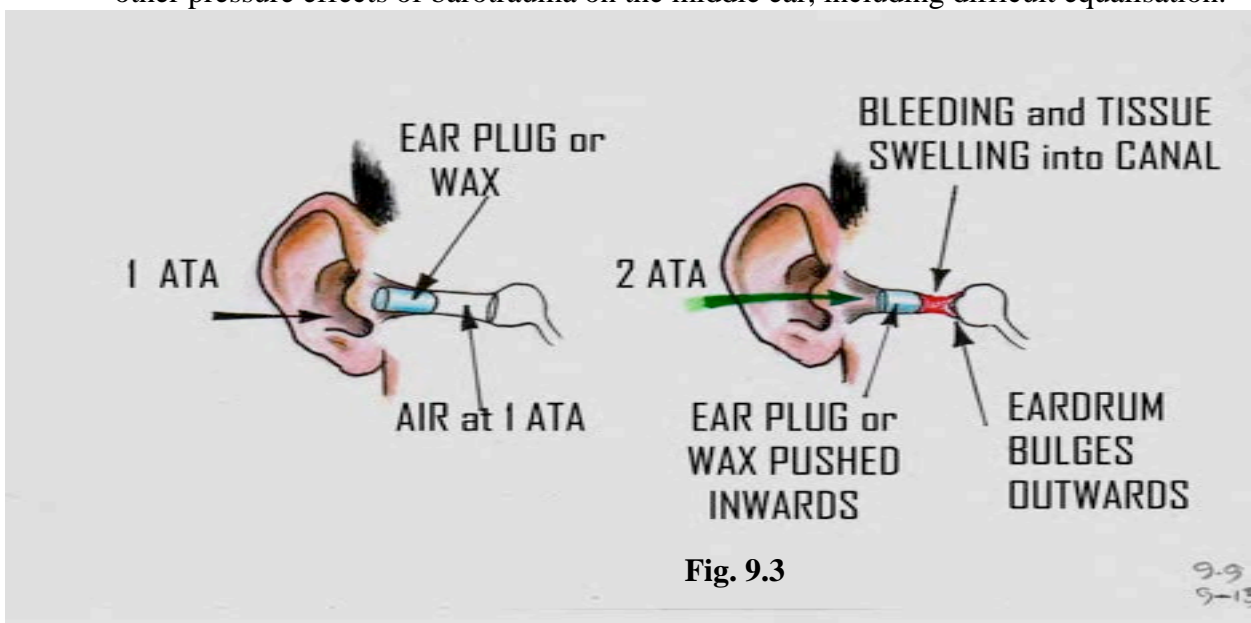
EXTERNAL EAR BAROTRAUMA

(EXTERNAL EAR SQUEEZE)

If the external ear canal is obstructed, the enclosed gas will be compressed and so reduce in volume during descent (Boyle's Law, again!). This will cause an outward bulging of the eardrum and swelling and bruising of the skin lining the ear canal.

Obstruction of the canal can be caused by a tight fitting **hood**, **wax** in the ears, **bony growths (exostoses)** in the ear or the wearing of **ear plugs**. As this condition can be encountered at depths as little as 2 metres, **ear plugs should not be worn during any type of diving**.

The symptoms include discomfort and pain on descent, bleeding from the external ear and the other pressure effects of barotrauma on the middle ear, including difficult equalisation.



MIDDLE EAR BAROTRAUMA OF DESCENT

(MIDDLE EAR SQUEEZE, AEROTITIS MEDIA)

The main risk of barotrauma to the ears is encountered on descent and the commonest site is the middle ear. About one quarter of diving trainees experience this, to a variable degree.

Water pressure around the diver increases as he descends. This pressure is transmitted to the body fluids and tissues surrounding the middle ear space causing compression of the gas space in the middle ear (Boyle's Law). The diver is aware of this sensation of pressure and voluntarily compensates for the reduction in middle ear gas volume by "**equalising the ear**" "auto-inflation" or "**clearing**". In this manoeuvre, air is blown up the Eustachian tube to replace the volume of gas compressed in the middle ear space. The ways of doing this are described later.

If the diver fails to equalise, water pressure will force the ear drum inwards, stretching it and escalating the sensation of pressure into one of pain. At the same time, reduced gas volume in the middle ear is compensated by blood and tissue fluid, swelling of the lining (mucosa) of the middle ear space. Ultimately, the blood vessels become over distended and rupture, bleeding into the ear drum and the middle ear space. This tissue damage takes days or weeks to resolve. Sometimes the ear drum itself will tear or rupture.

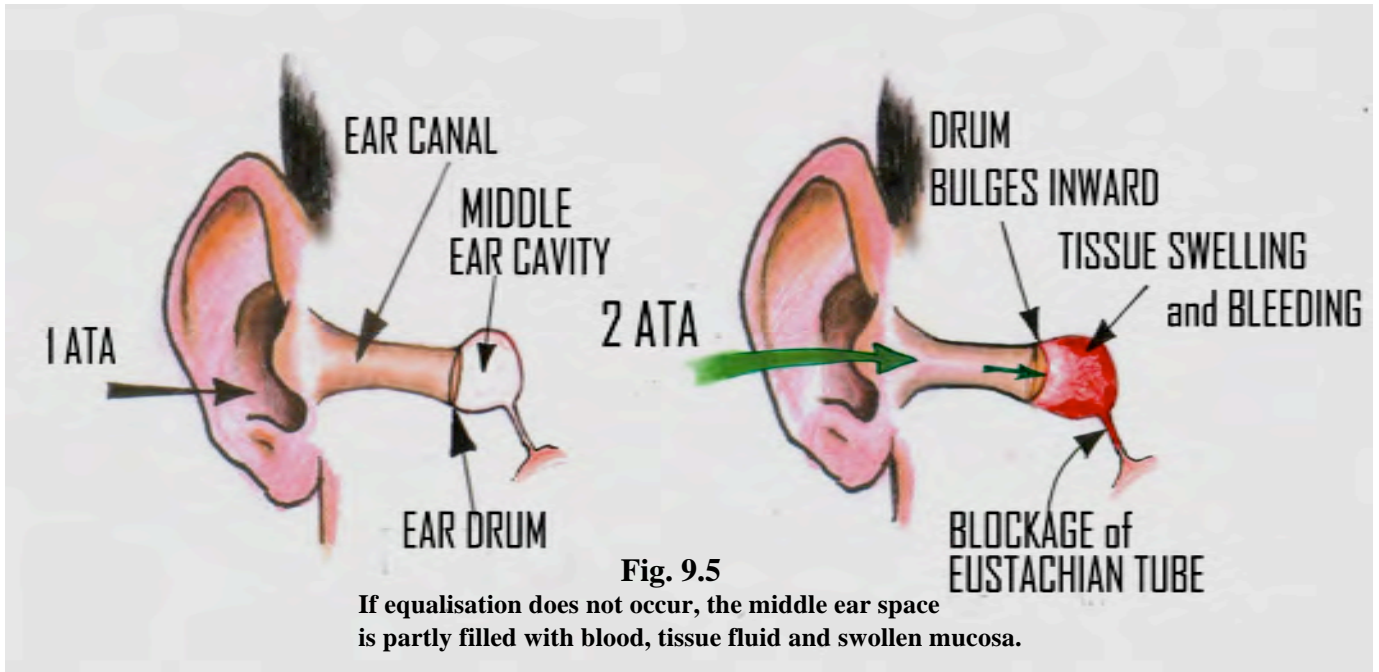
The depth at which this damage occurs depends on the size of the middle ear space and the flexibility of the ear drum. It is normally reached at 1–2 metres and if the diver does not equalise by the time he has reached this depth, barotrauma of the ear is likely.

The commonest diving problem!

**If the diver can equalise his ears on the surface,
the problem is due to incorrect diving technique**



Fig. 9.4



Clinical Features

A sensation of **pressure** is the first symptom of damage to the ear. This pressure sensation may develop into **pain**, which is usually severe, sharp and localised to the affected side. It increases as the diver descends, unless he equalises the middle ear spaces.



If the diver continues descending until the **ear drum ruptures**, he will experience relief of the pressure or pain, followed by a **cold feeling** in the ear. This is due to the sea-water which enters the middle ear space, cooling the bone and tissues near the balance organ. Thermal currents may be produced within the balance organ, causing stimulation and dizziness. Fluid may also be felt trickling down the throat, after running down the Eustachian tube from the middle ear space.

Fig. 9.6
Large perforation of the ear drum from a single dive to 8 metres without adequate equalisation.

Middle ear barotrauma, especially when associated with rupture of the ear drum, may be accompanied by **dizziness**. This sensation is termed **vertigo**. It may also be accompanied by **nausea** and **vomiting**. Vomiting underwater is a skill not frequently practiced and it can block the air supply and lead to aspiration of sea water (and vomitus) and drowning.

With lesser degrees of barotrauma, pain or discomfort in the ear may also be felt after the dive. There is often a **feeling of fullness** (or "water") in the ear and sounds may appear muffled. Crackling noises may also be heard (especially with chewing, swallowing or with jaw movements), caused by bubbles of air in the blood/body fluid mixtures within the middle ear.

Occasionally blood from the middle ear is forced down the Eustachian tube when the middle ear gas expands on ascent (Boyles Law). After surfacing the diver may notice **small amounts of blood** coming from the nose, on the same side as the barotrauma, or running down the throat.

Other symptoms due to the ear barotrauma include; a "**squeaking**" sound during equalisation (due to a Eustachian tube narrowed by mucosal swelling, or scarring from repeated episodes of barotrauma), or an **echo** sensation and/or a mild **ache** and **tenderness** over the ear/mastoid area following the dive.

Occasionally a diver has a naturally reduced pain appreciation, so that considerable barotrauma damage can be done despite the absence of much discomfort. These divers are vulnerable to permanent damage, as the usual warnings of middle ear barotrauma are absent. Others can suffer significant damage after exposure to even small pressures, e.g. in a swimming pool – especially if the submersion and pressure exposure is for many minutes duration.

The really serious problem from middle ear barotrauma in extension of the pathology to the inner ear with haemorrhage, or rupture of the round window and permanent hearing loss.

Treatment

A diver who has experienced middle ear barotrauma needs an examination by a diving **physician** to diagnose the condition, and check for complications such as a perforated eardrum or inner ear damage. Assessment of the cause, and advice on prevention of future difficulties is important (see later). **Audiograms** (hearing tests) are essential to test for damage to, and function of, the middle and inner ears.

Occasionally, the doctor may prescribe an oral **decongestant (or nasal spray)** to help open the Eustachian tube, while **antibiotics** may be prescribed if **infection** is present in the nose or throat area, or develops in the blood pooled in the middle ear cavity. This usually presents as a recurrence of pain hours or days after the barotrauma. **Ear drops** do not reach the middle ear and are of no value. They may be harmful if the ear drum has ruptured.

Once serious complications have been excluded, active treatment is usually unnecessary. In order to rest the ear and allow healing, diving, flying and middle ear equalisation should be avoided until the barotrauma has resolved. This commonly takes from 1–2 days up to 1–2 weeks.

The length of time away from diving depends on the severity of the barotrauma. The diver should not return to diving or flying until the physician has confirmed resolution of the barotrauma and the ability to equalise the ears. **Understanding why the barotrauma developed is necessary in order to prevent it happening again.**

If the ear drum was **perforated**, complete cure may take 1–3 months, even though it may appear to have sealed over within days. Early return to diving predisposes to recurrent perforation. Occasionally the drum fails to heal and requires surgical "patching" or "grafting" by a specialist. Later, diving may be permitted if repair is complete and easy voluntary equalisation of the ear is demonstrated.

It is necessary with the recurrence of symptoms, to perform repeat audiograms to confirm that no inner ear damage has occurred. About half of these divers may have ENT pathology, which may be treated by an otologist, and half will be recurring because of marginal Eustachian tube narrowing and failure to perform the equalisation techniques correctly. Both groups will gain from learning the correct "equalisation ahead of the dive" techniques.

PREVENTION OF BAROTRAUMA

Equalisation

Adequate equalisation of pressures in the middle ear space will prevent middle and inner ear barotrauma. This equalisation is necessary whenever increasing depth in the water. It should be performed frequently and before any ear discomfort is felt. It is necessary to equalise more frequently near the surface since the volume changes are greatest there (as explained by Boyle's Law). Equalisation should always be gentle to avoid damage. The technique of ear equalisation is a skill which improves with practice. Some divers can equalise without any apparent effort or action.

Upper respiratory tract infections (URTIs) cause congestion of the throat and Eustachian tube openings, making equalisation difficult or impossible. **Hay fever, allergies, snorting drugs or cigarette smoking** have a similar effect. Diving with these conditions is risky. A deviated nasal septum may also predispose to aural barotrauma as well as sinus barotrauma (see Chapter 10).



There are several ways of active and voluntary middle ear equalising before and during descent:

- **Valsalva manoeuvre.**

This technique is most frequently used because it is easy and effective. The diver **holds his nose, closes his mouth and blows gently against the closed nose and mouth**. This raises the pressure in the pharynx, forcing air up the Eustachian tubes into the middle ear. He hears the ear drums "pop". If they produce a longer squeak, the tube is partially obstructed.

If there is infected material in the throat this can also be blown up the Eustachian tube into the middle ear, leading to **infection**. This is another reason why divers are advised against diving with an upper respiratory tract infection.

Fig.9.7

To supplement this manoeuvre, **opening of the Eustachian tubes** can be facilitated by wriggling the jaw from side to side or thrusting the lower jaw forward as the manoeuvre is performed (**Edmonds Number 1 technique**).

A drawback of the Valsalva technique is that if it employed too forcefully, it is theoretically possible that the inner ear may be damaged. Another drawback is that the nose must be held closed with the fingers, which is not always easy with some professional diving helmets or full-face masks.

- **The Toynbee manoeuvre.**

This involves **holding the nose and swallowing simultaneously**. This usually causes the Eustachian tubes to open momentarily, allowing air to enter or leave the middle ear.

The Eustachian tubes open only briefly with this manoeuvre and it causes a negative pressure in the pharynx, so only smaller amounts of air are able to pass into the middle ear space. Consequently, this manoeuvre is not as effective as the Valsalva manoeuvre, but it is used successfully by many divers.

- **Others.**

Voluntary Opening of the Eustachian tubes (BTV technique) can be performed at will by many experienced divers, by contracting certain muscles in their throat. This technique can be performed by holding the nose, closing the mouth, and then trying to lift up the larynx (Adams Apple), which can be viewed in a mirror. A clicking can be heard in both ears if the procedure is successful. This technique is difficult to describe but if it can be mastered it is convenient and effective, there is little force involved and the manoeuvre can be performed repeatedly.

If any difficulty is encountered, the **Lowry technique** ("swallow and then blow at the same time" – a Toynbee + Valsalva combination) or the **Edmonds Number 2 technique** ("sniff and blow" – suck the cheeks in with a sniff against the closed nostrils, immediately followed by a Valsalva), may be used.

Diving Technique

Anyone who has problems with middle ear equalisation should gently practice this procedure a few times each day, on land. Practice improves performance and makes perfect. Voluntary controlled equalisation becomes easier with repetition. Some may even need to use nasal decongestants to assist this, at first, but they should not be used when diving. Any diver who has difficulty with middle ear barotrauma should practice using the Valsalva, Lowry or Edmonds techniques, as they cause a positive pressure in the middle ears. They should not rely on swallowing, Toynbee or BTV techniques or any others that result in negative middle ear pressures, even though they may open the Eustachian tubes.

Many divers suffer hearing loss because they do not equalise their ears correctly.

Ideally on the day of a proposed dive, the diver should confirm that he is able to equalise easily before setting out on a diving expedition.

All divers should equalise on the surface immediately before descending. This confirms that equalisation is possible and the ear drums balloon slightly outwards, causing a slight positive pressure in the middle ear and allowing some margin for error if the diver becomes distracted and forgets to equalise during the first metre of descent.

The diver should then **equalise every metre or less as the descent proceeds**, so that no sensation of pressure is felt. This is called "**equalising ahead of the dive**" and is much safer than waiting until the pressure sensation (or actual pain) is felt. By that stage the middle ear mucosa is already swollen and obstructing the Eustachian tube, making equalisation more difficult.

If any difficulty is encountered, it is unnecessarily dangerous to descend further as equalisation will become more difficult - until the middle ear becomes congested with tissue fluid and blood. Instead the diver should either abort the dive or, if the dive is an important one, immediately ascend a little and repeat the equalisation manoeuvre. The diver should not persist with this "yo-yo" technique, or remain at a depth at which a "pressure sensation" is present. The middle ear is filling ("equalising") with blood. This is not a sensible situation. If the ears do not equalise easily, abort the dive. That way, he will be back diving much more quickly, having not damaged the middle ear.

If descent is continued, a '**locking effect**' on the **Eustachian tube** may develop. This is caused by the pressure difference between the middle ear and the throat. Equalisation is then impossible.

Descending 'feet first' makes equalisation considerably easier, and is best done on an anchor or shot line. This allows accurate control of the descent rate and depth while allowing the diver to concentrate on equalisation without the distractions of swimming and depth control. At least keep the head vertical when equalising, assisting the passage of air up the Eustachian tubes.

The novice diver and the diver who has difficulty with his ears should use a face mask which allows easy access to the nose to facilitate the various manoeuvres. If one ear causes more problems, then cock this ear to the surface when equalising (this brings that Eustachian tube even more vertical, and air travels upwards, under water).

Surgical correction of nasal septal deviations, cessation of smoking and adequate treatment of URTIs and allergies may be needed by those who have these predisposing causes.

Medication

Medication has been used to facilitate equalisation when there is some disorder of the ear, nose or throat. Topical nasal decongestant sprays and drops such as **phenylephrine and oxymetazoline** shrink the lining of the nose and Eustachian tube, reducing congestion and opening the air passages. Some oral medications such as **pseudoephedrine** have a similar effect.

While these drugs can make diving possible when it otherwise would not be, **they assist the diver to dive with conditions which should preclude diving**, such as upper respiratory tract infections. There is an added risk of the drug predisposing to barotrauma during the ascent - which is far more dangerous than ear barotrauma of descent. These drugs often relieve obstruction of the throat end of the Eustachian tube, improving equalisation during descent. Unfortunately they do not influence the middle ear end of the Eustachian tube, thus they do not assist release of gas from the middle ear during ascent. Decongestants may also permit diving despite minor descent barotrauma but can wear off during the dive, resulting in congestion of the pharyngeal end of the Eustachian tube and further obstruction to the outflow of gas from the middle ear (see below, "middle ear barotrauma of ascent").

Because these drugs mask the symptoms of conditions which would otherwise preclude diving, divers are advised that it is **better to avoid diving than to continue while taking these drugs**. See Chapter 37, last page, for more on the problems of drug treatment. They make other diving disorders more likely e.g. Sudden Death Syndrome.

•Middle Ear Equalisation (ME=) Client's Problem Check List

- **I descend a bit slower than my buddies.**
- **If there is any pressure, I halt my descent and wait a bit. Or,**
- **I may ascend until the ear clears. (Yo-Yo)**

Why do these procedures? If you are not ME= promptly or sufficiently, then these procedures merely allow the ME to fill with blood or tissue fluids, and thus allow further descent with less pain or discomfort. This is not a sensible way to ME=. It results in middle ear congestion, Eustachian tube obstruction and other pathology which may be temporary or permanent.

- **I am trying to use swallowing to ME=**

If you have any difficulty with ME=, employing techniques that result in a relatively negative ME pressure causes ME congestion and Eustachian tube blockage. Use the positive pressure Valsalva technique (or Lowry, or Edmonds) , prior to and during descents.

- **I dive down the shot line.**

This requires greater force to inflate the ME, as you are trying to force air down the Eustachian tube. Descend feet first and you can blow air up the Eustachian tube. Air travels easier up than down, in the water. Remember bubbles? They rise.

- **If there is any water in my ears (fullness, crackling) after the dive, I use alcohol ear drops to dry them out.**

It is likely that the “water” in your ear is really fluid in your ME!

- **I sometimes have a bit of blood from my nostril (or in my throat).**

Although it may be from your sinus, following expansion of air with ascent, it is more likely from the middle ear on that side. In either case, ME= correctly (“ahead of the dive”) may well fix both. See previous pages.

- **When I dive and ME=, I hear a squeaking sound in my ear.**

This suggests a narrowed Eustachian Tube, possibly from inadequate ME=. The sound you should hear when you equalise, and the ear drum moves outward, is a click or pop. It takes a split second to achieve. Not a long drawn out sound.

- **I can often dive once, without problems, but cannot ME= on other dives.**

You have probably produced some middle ear congestion in the first dive, but continued the dive. By the second dive, you start off with significant middle ear congestion, and so ME= is more difficult.

- **One ear equalises before the other**

Not a problem. It is normal. You may wish to assist the slow ear by pointing it towards the surface as you ME=.

If reasonable, avoid the use of hoods which cover the external ear. If it is necessary to use these hoods, then it is preferable to have holes inserted over the ear, which will allow air and water movement and pressurisation. This is not, of course, necessary or possible with diving helmets or certain dry suits. Special ear plugs and masks used for prevention of ear barotrauma probably only work by delaying damage and therefore inducing fewer symptoms - not by preventing damage.

MIDDLE EAR BAROTRAUMA OF ASCENT

(ALTERNOBARIC VERTIGO, REVERSE SQUEEZE)

This condition is relatively uncommon by itself, but it is often a complication of a middle ear barotrauma of descent and/or the use of nasal decongestants. During ascent, air in the middle ear space expands and must escape. The air normally escapes down the Eustachian tube to the throat without any conscious effort by the diver. If very observant, he may actually hear or feel it escape from his ear.

Occasionally the Eustachian tube may obstruct this flow of air, with subsequent air distension and increased pressure sensation in the middle ear cavity during ascent. This causes bulging and possible rupture of the ear drum. There may also be damage to the inner ear, leading to hearing loss (see below, and Chapter 30). The increased pressure in the middle ear may also stimulate the nearby balance organ producing vertigo and its associated symptoms.

Clinical Features

Increasing pressure and pain is sometimes felt in the affected ear as the diver ascends. Often there may be **vertigo** as well as **nausea** and **vomiting**. After surfacing the diver may feel fullness or dullness in the ear. **Tinnitus** or **hearing loss** may indicate serious damage (inner ear barotrauma).

Vertigo may develop after only a metre or so of ascent (see Chapter 31, Case History 31.2). Many of these symptoms can be hazardous, especially as ascent may be prevented by the symptoms.

First-Aid

If a diver encounters ear pain or vertigo during ascent, he should **descend a little** to minimise the pressure imbalance and attempt to open the Eustachian tube by holding the nose and swallowing (Toynbee, or other equalisation manoeuvre). If successful, this equalises the middle ear by opening it up to the throat and relieves the distension in the affected middle ear.

Occluding the external ear by pressing in the tragus (the small fold of cartilage in front of the ear canal) and suddenly pressing the enclosed water inwards, may occasionally force open the Eustachian tube. If this fails then try any of the other techniques of equalisation described previously, and attempt a slow ascent.

Treatment

Uncomplicated cases resolve quickly but eardrum rupture or inner ear damage may need **specialised care. All cases need expert diving medical assessment, for diagnosis and advice.** Unless it can be understood and prevented, diving should be avoided.

INNER EAR BAROTRAUMA

A serious consequence of ear barotrauma is inner ear (hearing and balance organ) damage. The inner ear can be damaged in several ways.

- **Round window fistula (or "leak").**

If the diver fails to equalise the middle ear adequately, water pressure will bulge the eardrum inwards. Since the eardrum is connected to the oval window by the bony chain, this window is forced inwards and the round window bulges outwards. If these movements are excessive, the small end-bone can be pushed through the oval window or, more commonly, the round window may tear. After these injuries, the window may then leak the inner ear fluid (perilymph) into the middle ear.

Round window fistula may also be associated with an excessively **forceful middle ear equalisation manoeuvre**. Increased intravascular pressures in the head associated with this manoeuvre may be transmitted to the cochlea fluid, causing bulging and then rupture of the round window. Alternatively, the sudden displacement of the eardrum after an equalisation manoeuvre may set up a pressure wave in the inner ear fluid which tears the round window. The fluid which leaks out is crucial to the healthy function of the **cochlea** and its loss leads to damage to the hearing organ. Permanent severe **hearing loss** may follow if the fluid loss is not interrupted by healing of the round window or surgical repair,.

The same fluid also bathes the balance organ, and damage to this organ may also result in **dizziness (vertigo), nausea and vomiting.**

- **Other pathology.**

Permanent hearing loss or balance disturbance, unrelated to round window fistula, can be caused by direct cochlea damage from inner ear barotrauma. The cause may be haemorrhage (or bleeding), inner ear membrane rupture, or air entering the inner ear (from a stretched round window). This hearing loss may be temporary or permanent depending on the degree of damage and its management.

Clinical Features

The cardinal features of inner ear barotrauma are :

- **tinnitus** (ringing or buzzing noises in the ears)
- **hearing loss**
- **vertigo** (a feeling of being pulled, rotation, rocking or unsteadiness)
- nausea or vomiting
- rarely, **dysacusis** (painful hearing)

One or more of these must be present to make the diagnosis. Fluid may be noted in the middle ear.

Tinnitus is a ringing, buzzing or musical sound in the ear, usually high pitched, due to damage or irritation of the nerve cells of the cochlea.

Hearing loss is due to damage of the cochlea. It may improve, stay the same or deteriorate. Audiograms may differentiate this type of hearing loss (sensori-neural) from that due to middle ear barotrauma (conductive). See Chapter 30.

Vertigo is the spinning or pulling sensation due to balance organ damage (see Chapter 31).

Treatment

A diver presenting with any of these symptoms needs **immediate assessment** by a diving physician. In the interim, he should avoid any exertion, middle ear equalisation, altitude or diving exposure, sneezing or nose blowing.

The physician will examine the ear and perform **serial audiograms** to detect any hearing loss, which may not be obvious to the diver. **Tests of balance organ function (ENGs)** may be necessary.

Aspirin, nicotinic acid (vitamins), other vasodilators or anti-coagulants should not be taken.

An expert diving medical opinion concerning **future diving** should be sought if the diver has sustained permanent hearing loss, tinnitus or balance disturbance, as it is probable that further episodes of inner ear barotrauma will cause additional and possibly permanent disastrous effects. Training in correct middle ear equalisation techniques is essential if a return to diving is contemplated.

- **Round window fistula.**

This condition can usually be managed conservatively with **absolute bed rest in the sitting position**. Straining, sneezing, nose blowing, sexual activity, loud noise and middle ear equalising should be avoided, to prevent pressure waves in the inner ear.

The round window fistula often heals spontaneously within a week or two with this regimen but if hearing loss progresses or the other features persist, it may be necessary to resort to **surgery** to patch the round window leak.

Once an oval or round window fistula or cochlea injury has healed, the diver's future in this sport is bleak. Flying should be completely avoided for some months to allow complete healing of the injury or the surgical repair.

- **Cochlea damage.**

In the absence of a round window fistula, no specific treatment is available for this type of injury. Rest in a head elevated position, repeated pure tone audiograms, and avoidance of exertion, equalisation attempts and further exposures to barotrauma (flying or diving), is necessary until the condition has stabilised.

Chapter 10

SINUS BAROTRAUMA

ANATOMY OF THE SINUSES

The sinuses are air filled cavities contained within the bones of the base and front of the skull. Apart from causing inconvenience to divers, their exact function is unknown.

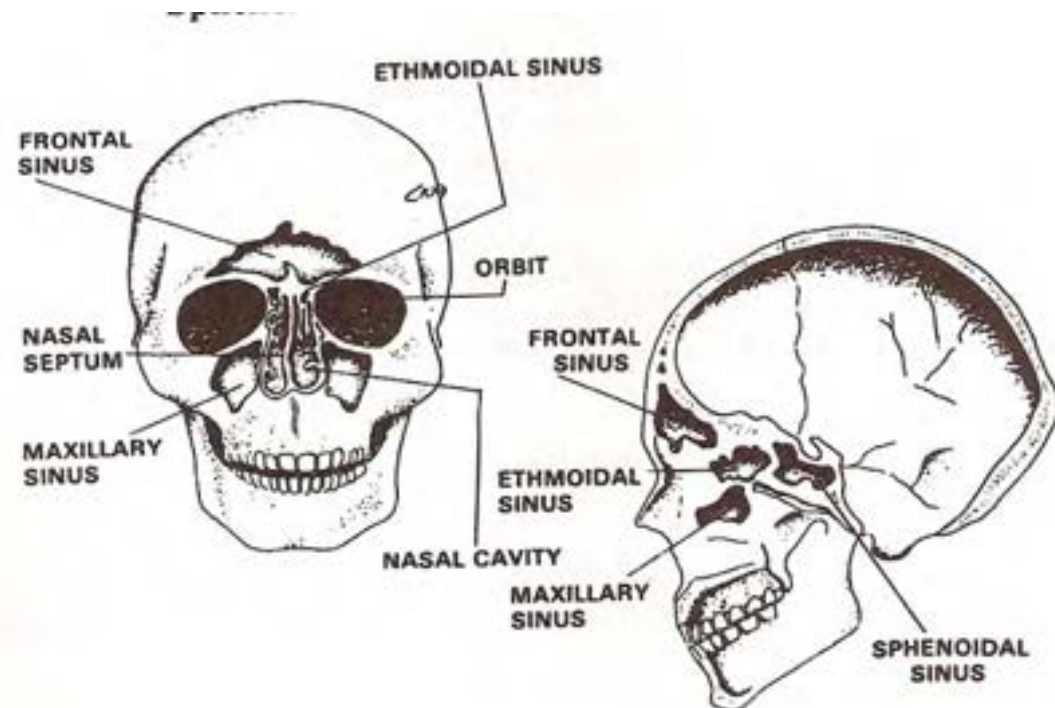


Fig. 10.1
Location of sinuses in the skull. They are connected by canals to the nose.

There are four main groups of sinuses, with openings into to the nose :

- Maxillary sinuses in the cheek bones
- Frontal sinuses in the skull above the eyes
- Ethmoid sinuses in the thin bone at the base of the nose
- Sphenoidal sinuses situated deep inside the central part of the skull.

All the sinuses are lined by a soft mucous-secreting tissue which is richly supplied with blood vessels. Each sinus communicates with the nose by its own narrow opening called the ostium, and through these, the sinuses are permanently open to the atmosphere.

The mastoid sinus or antrum is a similar structure that opens into the middle ear cavity. It more often reflects the pathology of the middle ear and reference should be made to Chapter 9 for this.

THE MECHANISM OF SINUS BAROTRAUMA

As the water pressure changes during a dive, the sinuses normally equalise automatically by free passage of gas into or out of their openings.

Problems are inevitable, however, if these openings become obstructed. Obstruction can be due to congestion associated with allergy, smoking, respiratory tract infection or the overuse of topical decongestants and other drugs. Other causes of ostia obstruction include chronic sinus inflammation (sinusitis), nasal inflammation (rhinitis), folds of tissue (polyps) and plugs of mucous.

When the sinus is blocked, during descent the gas in the sinus is compressed (according to Boyles' Law) causing sinus barotrauma of descent. The shrinking volume is replaced by swelling of the sinus lining, tissue fluid or bleeding – partly filling the sinus.

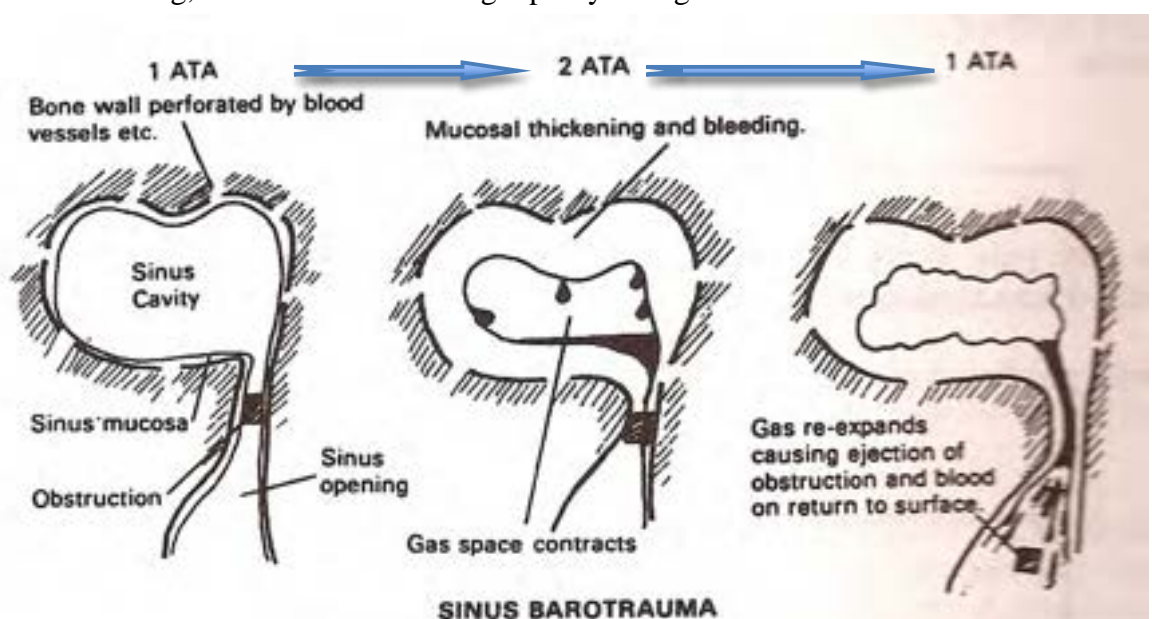


Fig. 10.2 A blocked sinus cavity on the surface, after descent to 10 m. then re-surfacing.

This tissue fluid and blood, which may take days or weeks to absorb, represents a rich nutrient medium for bacterial growth, promoting sinus infection (see Chapter 28).

During ascent, blood and tissue fluid from the sinus barotrauma of descent may be discharged into the nose or back of the throat by the gas expanding in the sinus, causing an apparent nose bleed from the same side as the injured sinus. Alternately, it may be spat out or swallowed.

If the sinus opening becomes obstructed during ascent, the expansion of gas flattens the sinus lining against its bony wall, causing pain and injury to this delicate tissue. This is called sinus barotrauma of ascent.

Sinus barotrauma of descent is more common than ascent, but they often coexist.

CLINICAL FEATURES

Sinus Barotrauma of Descent

This condition usually presents during descent with a sensation of pressure, developing into a pain in the region of the affected sinus. It is usually felt over the eye (frontal or ethmoidal), the cheek bone (maxillary), or deep in the skull (sphenoidal) depending on which sinus is involved.

Maxillary sinus barotrauma can also present as pain in the upper teeth.

The pain may settle during the dive, as blood and other fluids equalise the pressure differential, or it may recur as the damaged sinus causes a dull pain or headache afterwards.

A small amount of blood issuing from the nose during or after ascent is a frequent accompaniment of sinus barotrauma.

Occasionally the maxillary nerve may be involved, causing numbness over the cheek.

Sinus Barotrauma of Ascent

This presents with pain in the affected sinus during and after the diver's ascent. Bleeding from the sinus frequently drains through the nose or can be spat out.

Severe headache persisting or developing hours after the dive suggests either inflammation or developing infection (i.e. sinusitis) or sinus tissue damage.

Rarely, the bony walls of the sinus may rupture, with the expanding gas passing into the eye socket (orbital emphysema), the brain cavity (pneumocephalus) or tracking elsewhere. Any such severe manifestation must be treated as a medical emergency

TREATMENT

Any case of suspected sinus barotrauma accompanied by headache after a dive requires medical assessment, because decompression sickness and many other conditions can also present as headache. See Chapter 32.

Normally sinus barotrauma resolves without any treatment. Significant bleeding into the sinus may drain more rapidly if topical nasal and oral decongestants are used.

The diagnosis may be confirmed by X-Rays, CT scans or MRI (preferred) of the sinuses. The sooner this is done after the dive, the more likely it is to demonstrate the pathology.

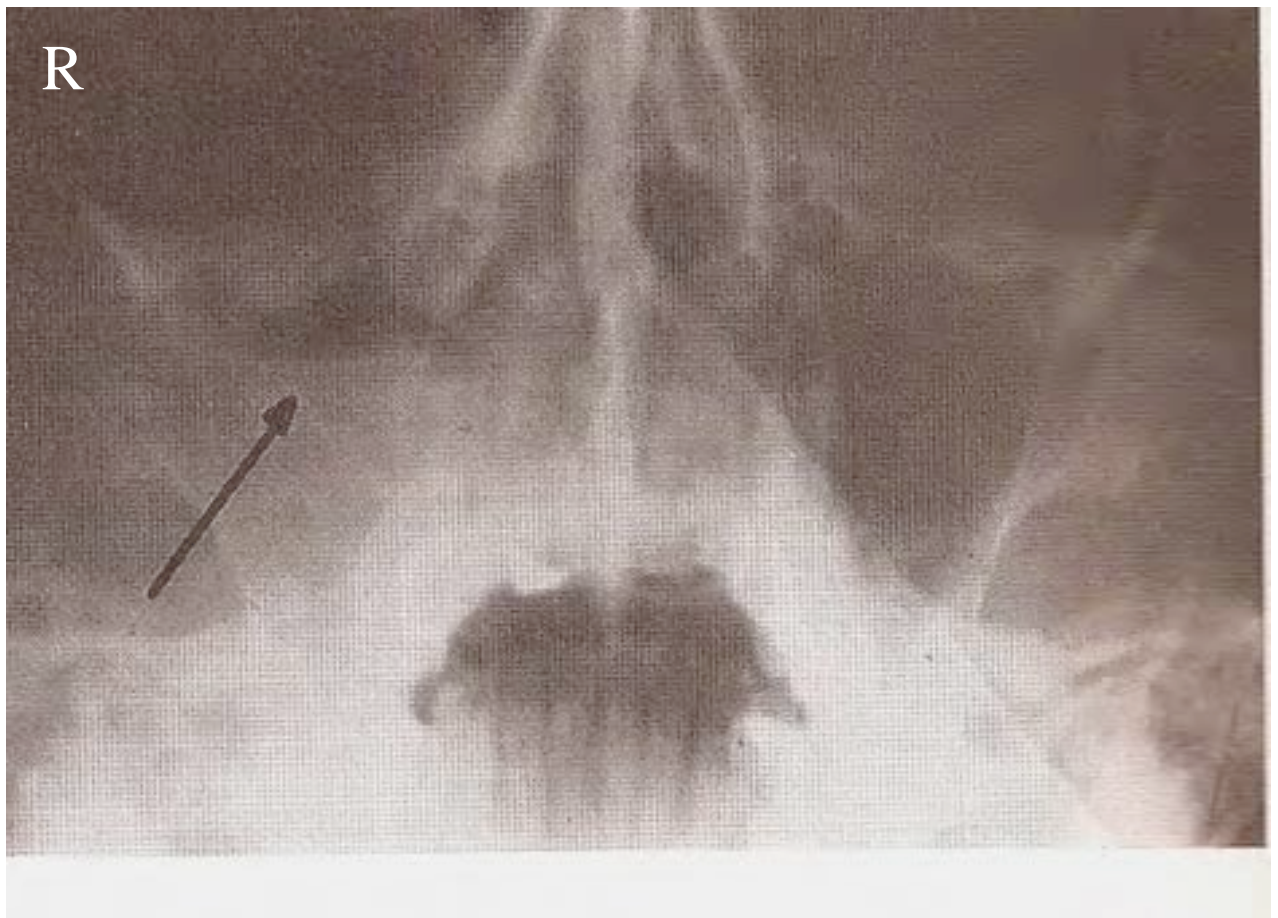


Fig. 10.3

Sinus x-ray showing fluid level (opaque area almost filling cavity below right orbit) in right maxillary sinus after barotrauma of descent. Left sinus cavity appears clear and filled only with air (black).

Increasing pain in the sinus, with fever or malaise developing after the dive suggests infection which is treated with decongestants and antibiotics.

Diving and flying should be suspended until the condition has resolved, usually from 2–10 days.

PREVENTION

Active and frequent middle ear equalisation, using positive pressure techniques such as the Valsalva, fortuitously assists by forcing air into the sinuses during descent and preventing barotrauma of descent. The “equalising ahead of the dive” technique is applicable (Chapter 8).

Diving should be avoided if the diver is suffering from any upper respiratory tract infection, to reduce both the risk of barotraumas and the infection complications. Smoking and allergic nasal congestion (hay fever) increases the risk of sinus barotrauma by obstructing the sinuses. A deviated nasal septum may also contribute to the development of sinus barotrauma, and if so, it can be surgically corrected.

Not all patients with chronic sinusitis need avoid diving. If the ostia is open and there are no polyps or other obstructions, the sinuses may get a good “washout” with air travelling into and out of the sinuses as the diver descends and ascends. This medical procedure is free to divers.

Nasal decongestants used at the time of diving tend to reduce the congestion of the sinus ostia (at least at the nasal end), but may not prevent sinus barotrauma of ascent. For this reason they should be avoided. It is better for the diver to be prevented from descending (sinus barotrauma of descent) than to be prevented from ascending (sinus barotrauma of ascent). See Chapter 37, last page, for a discussion on the effects of drugs used to prevent sinus barotrauma.

Repeated minor sinus barotraumas can result in progressive scarring of the ostia, causing obstruction and intractable sinus barotrauma. Then the diver has to choose between ceasing his diving career or problematic endoscopic surgery.



Fig. 10.4

Chapter 11

PULMONARY BAROTRAUMA

(LUNG BAROTRAUMA)

Pulmonary barotrauma is lung injury caused by pressure changes (Boyle's Law again). In divers it can occur on ascent or descent. Barotrauma of ascent is relevant to scuba diving, and barotrauma of descent to free diving (breath-hold).

PULMONARY BAROTRAUMA OF ASCENT

("BURST LUNG")

This is second only to drowning as a cause of death in young recreational scuba divers.

The lungs of a male diver normally contain about 6 litres of air, contained in the alveoli and air passages. If a diver takes a full breath at 20 metres (66 ft.) and returns to the surface, that 6 litre volume expands to 18 litres since the pressure at 20 metres is 3 ATA and at the surface, 1 ATA.

In this situation, to avoid over distension of his lungs, the diver must exhale 12 litres of air (measured on the surface) before or during his ascent. If he does not exhale this air, the expanding gas will distend his lungs, like a balloon, and even some normal lungs will rupture if they are distended more than 10%.

When near the surface, this 10% distension can be produced by an over-pressure of about 80mm Hg. - equivalent to the pressure difference between one metre depth (less than 4 ft.) and the surface, making pulmonary barotrauma a real possibility even for a scuba diver in a swimming pool. Divers have died from pulmonary barotrauma in shallow swimming pools

Even if the diver does exhale correctly, he can still encounter this problem if there is some obstruction to the venting of air from some part of the lung i.e. some form of lung pathology.

Like all other barotraumas, this is more a disease of the shallows, than of great depths. Gas volumes change more nearer the surface, for each alteration in depth.

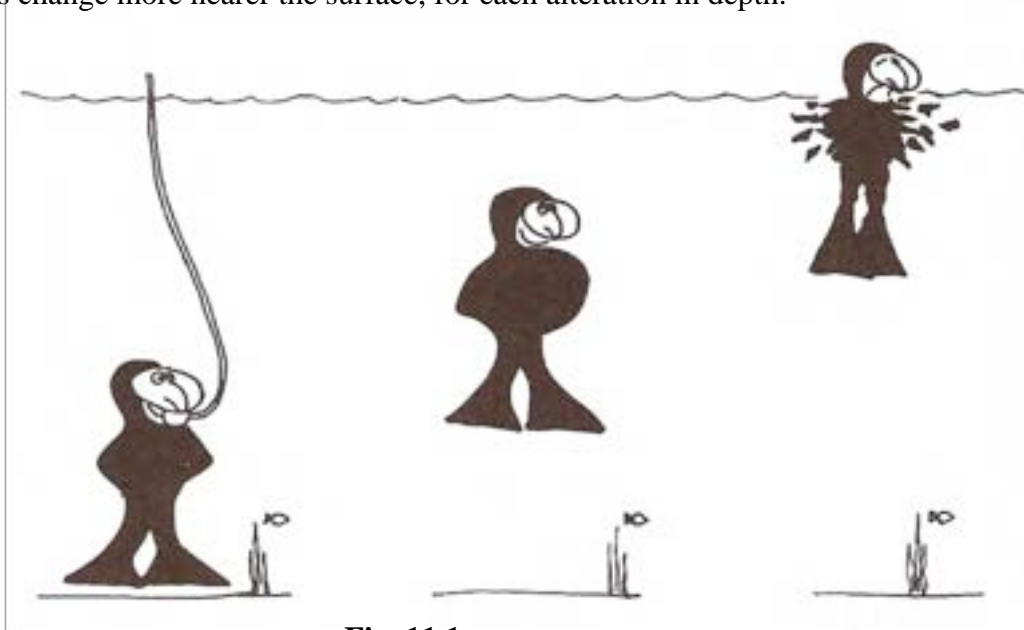


Fig. 11.1

Clinical Features of Pulmonary Barotrauma

If the lungs rupture due to excessive volume expansion, any or all of four consequences can follow:

- **Lung tissue damage**
- **Emphysema (gas in the tissues)**
- **Pneumothorax (gas in the chest cavity)**
- **Air embolism (gas bubbles in the blood)**

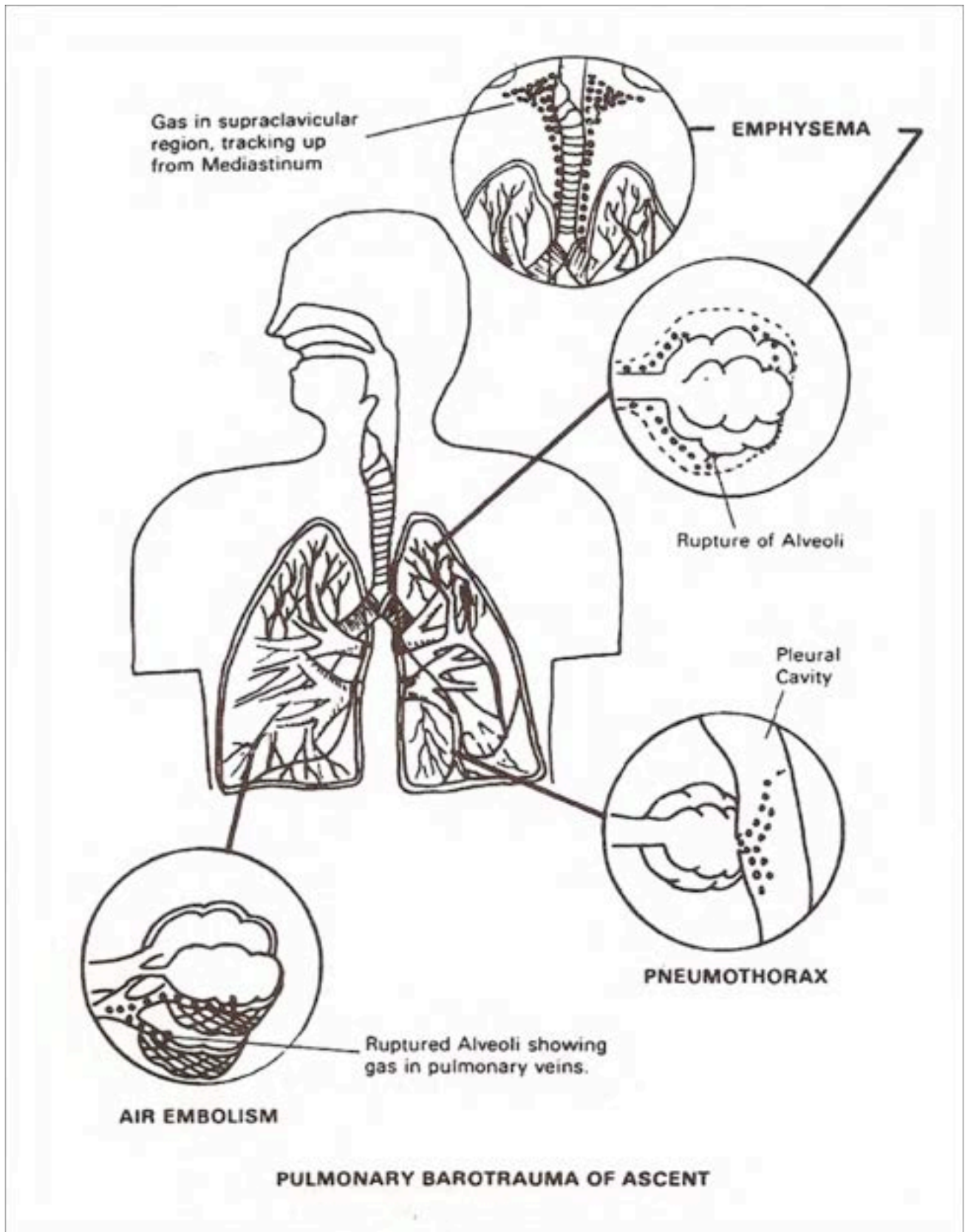


Fig. 11.2

The various clinical manifestations of a ruptured lung on ascent.

Lung Tissue Injury

If the lungs are over distended, generalised tearing of the lung tissues with severe diffuse damage to the lung structure is likely. Bleeding, bruising, and generalised destruction to the lungs causes severe breathing difficulties.

❑ Clinical features.

Shortness of breath, pain when breathing, coughing, coughing-up blood, and shock are the principal manifestations. Death may follow rapidly.

❑ Treatment.

The diver should be examined and treated for other manifestations of pulmonary barotrauma. Lung tissue damage alone has no specific first aid treatment apart from basic resuscitation measures (see Chapter 40 and 42). The patient should be given oxygen and taken immediately to hospital.

Surgical Emphysema

Tearing of the alveoli allows gas to escape into the tissues of the lung. Air tracks along the lung tissues to the **mediastinum** in the midline. From here it migrates into the **neck** or, in severe cases, tracking around the heart sac (pericardial sac) or even into the **abdominal cavity**.

If the diver has performed a long or deep dive and still has a nitrogen load in his tissues, nitrogen will continue to diffuse into these air spaces to expand them over the next few hours, with increasing symptoms.

The presence of the air in the tissues causes damage by compressing the blood vessels, nerves, larynx, or oesophagus. In severe cases air can compress the heart, causing malfunction.

❑ Clinical features.

It may take some time for symptoms to develop, as the air migrates slowly through the tissues. Air in the mediastinum and around the heart may cause **chest pain** and **shortness of breath**. Air in the throat leads to **voice changes** (the voice developing a "tinny" or "brassy" note), shortness of breath and/or **swallowing difficulties**. A "**crackling sensation**" may be felt under the skin around the neck – and especially just above the collar-bones (supra-clavicular space). It feels like "rice bubbles beneath the skin" or "cellophane paper", on pressing. The diver may complain of a sensation of **fullness in the throat**.

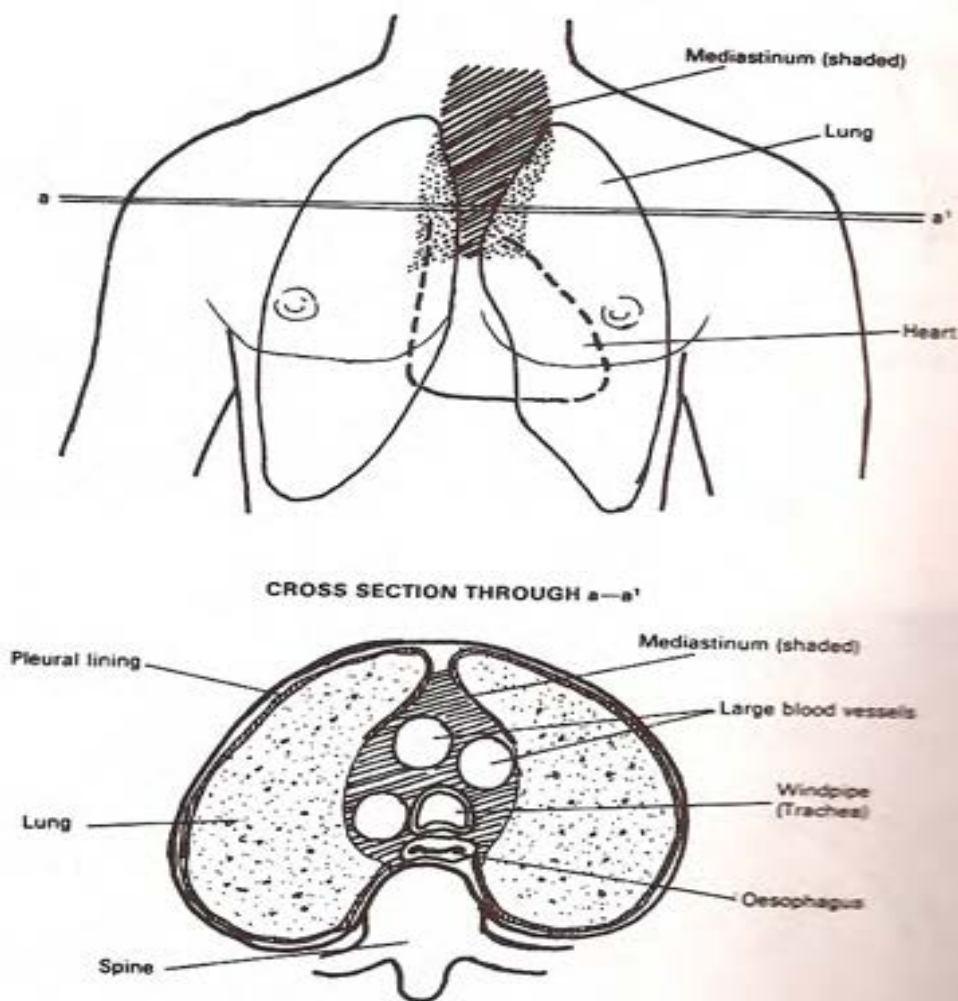


Fig 11.3

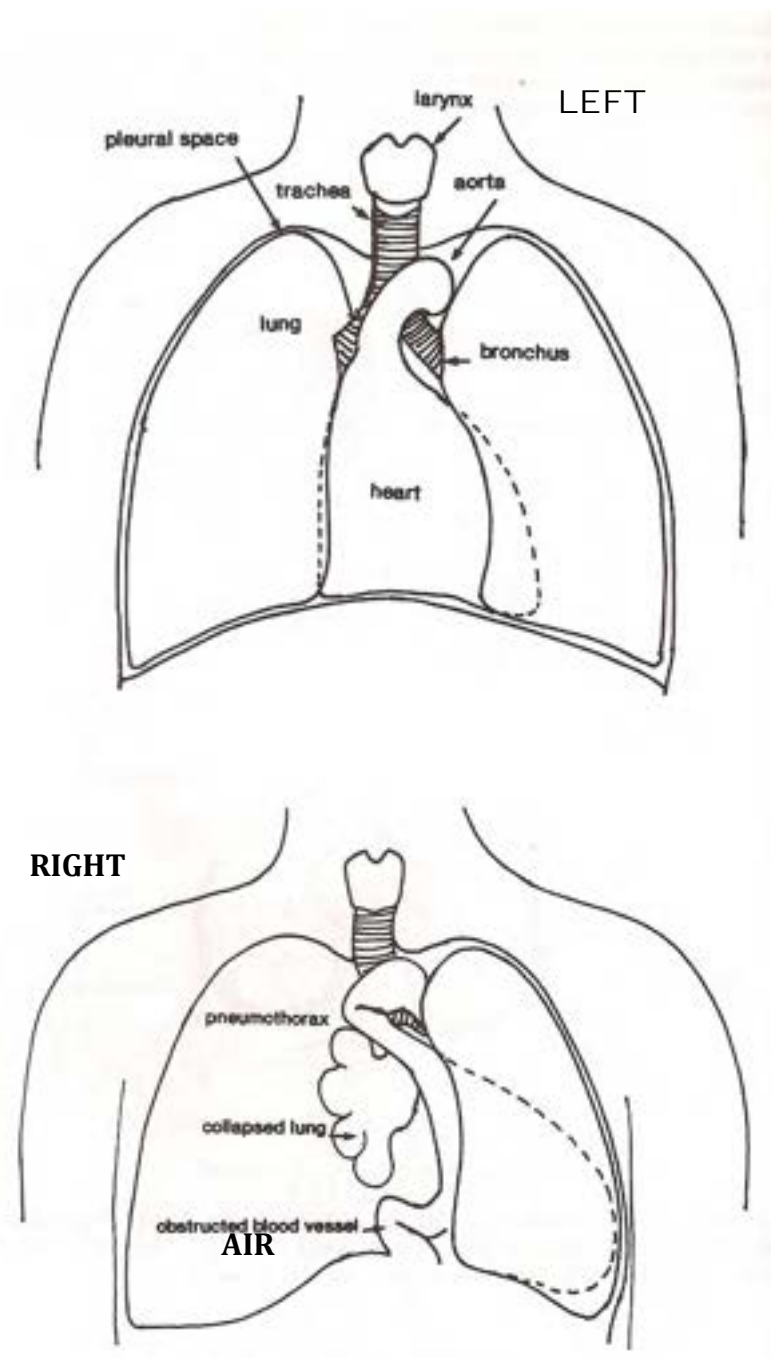
The location of the mediastinum can be seen from this frontal view and cross section. It is located deep in the chest between the lungs and above the heart, and its connection with the neck tissues can be noted.

□ Treatment.

The diver should be examined and treated for the other manifestations of pulmonary barotrauma. Mild surgical emphysema alone responds to **100% oxygen**. (see Chapter 40). This causes a diffusion gradient for nitrogen (between the air space and the nitrogen-free blood) which eliminates the air bubbles. If not treated, the condition will slowly resolve, but it may last many days.

Severe surgical emphysema, especially if causing compression of the airway or blood vessels, will respond to **recompression** in a recompression chamber, compressing the emphysema. If air is breathed, more nitrogen may diffuse into the tissues, making the surgical emphysema even worse when the diver is decompressed. Breathing oxygen, especially under pressure in a chamber, produces a diffusion gradient of nitrogen out of the air spaces, with rapid resolution.

Pneumothorax



If the lung ruptures near its surface, air gains access to the pleural space, between the lung and the chest wall (pneumothorax). The elasticity of the lung causes it to collapse like a burst balloon and the lung tissue within the chest cavity is replaced by an air pocket.

The air pocket cannot escape.

Fig. 11.4

These schematic diagrams of a normal chest (as seen on x-ray) at the top, and a pneumothorax below, with collapse of the right lung. As air pocket in the right side expands with further ascent, it pushes the heart and midline structures towards the left side of the chest, causing a "tension" pneumothorax on the right.

Occasionally a valve effect allows air to pass from the air passages into the pneumothorax but prevents its return. As more and more air collects in the pneumothorax, the pressure in the thoracic cavity rises and forces the contents of the chest (including the heart and lungs) to the opposite side. This is called a **tension pneumothorax** and its effect on cardiac function is catastrophic and rapidly fatal if the air is not released.

If the lung rupture occurs at depth, the air in the chest cavity expands with ascent (Boyles Law) and this may also cause a tension pneumothorax

Bleeding may take place into the pneumothorax, leading to a **haemo-pneumothorax**.

❑ Clinical features.

A pneumothorax is usually heralded by **chest pain**, often made worse by breathing, and causes **shortness of breath**. Respiration becomes rapid and the heart rate increases.

With a **tension pneumothorax**, as the mediastinum is pushed to the opposite side, the trachea can be felt to be displaced to that side. The patient becomes increasingly short of breath and may become cyanotic (blue) and shocked. The pulse is difficult to feel as the blood pressure falls.

With severe cases of burst lung, a pneumothorax will be evident very soon after the diver reaches the surface, but in milder cases, the symptoms of pneumothorax may be delayed for many hours. Symptoms may be brought on by coughing or altitude exposure (e.g. mountain range, travel in aircraft, more diving).

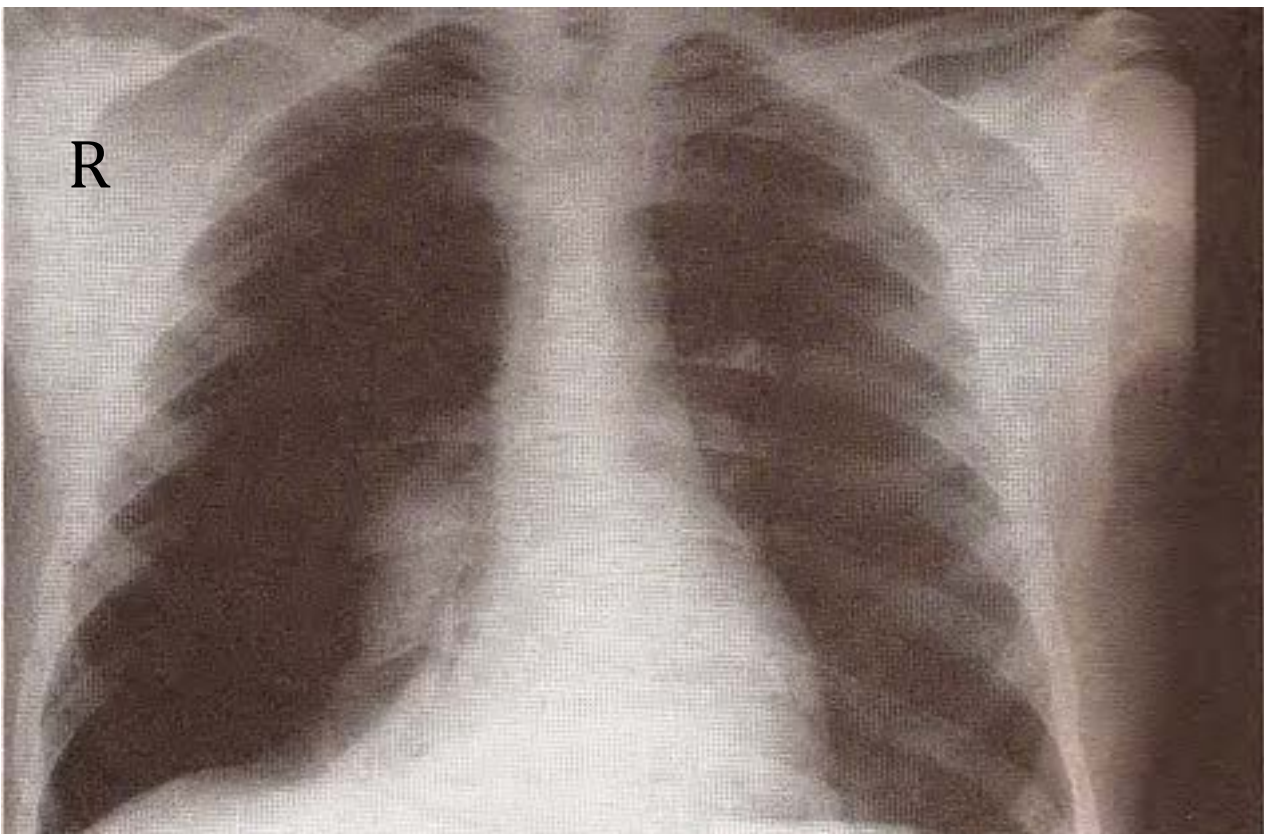


Fig. 11.5

X-ray of diver's chest after suffering pulmonary barotrauma of ascent with a right sided pneumothorax. The right chest cavity appears "black" due to its being filled with air and the collapsed lung (white) can be seen low, near the midline.

□ Treatment.

A pneumothorax requires **urgent medical attention**. The extent of lung collapse is assessed clinically and confirmed by a chest x-ray. A large pneumothorax is treated by placing a **tube into the pleural air space** and connecting it to a one-way valve such as a Heimlich valve or an underwater drain. This allows air out of the pneumothorax but prevents its return. The placement of tubes in the chest is usually beyond the capability of untrained personnel as there are important structures, like the heart, which can be injured in the process. After a period of hours or days the tear in the lung usually heals and the lung slowly re-inflates.

A minor pneumothorax (less than 25% lung collapse) may be treated by the diver **breathing 100% oxygen** (see Chapter 40).

A **tension pneumothorax** is a medical **emergency**. The pressure in the pneumothorax must be relieved by the insertion of a needle or tube through the chest wall, into the pneumothorax.

If the diver is aware of the possibility of a pneumothorax, he may be able to alert a physician to the possible diagnosis if any of these clinical features are present. The physician may release the air by inserting a needle into the second intercostal space in the mid-clavicular line, or through the 5–6 intercostal space in the mid-axillary line. Both have potential complications.

Air Embolism

When the lungs rupture, tears in the alveoli walls (and contained blood capillaries) can allow air to enter the blood circulation. This air is conducted to the left side of the heart, from whence it is pumped through the arterial circulation.

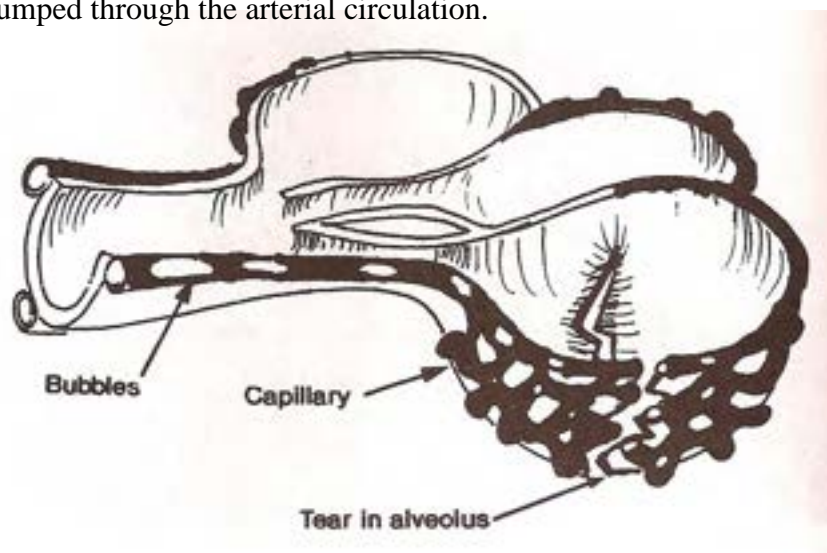


Fig. 11.6

Diagram of a ruptured alveolus and capillary vessel from pulmonary barotrauma of ascent. Air bubbles (emboli) are entering the veins carrying blood back to the left atrium of the heart.

The air bubbles obstruct or damage blood vessels in vital organs such as the heart and brain, leading to impairment of function, serious disability or death.

❑ **Clinical features.**

Symptoms present abruptly, usually immediately or within 10 minutes of the diver reaching the surface. Air bubbles lodging in the brain may cause **loss of consciousness, fits or confusion**, a pattern of symptoms similar to a "cerebral stroke". This is called **Cerebral Arterial Gas Embolism** or **CAGE**.

There is often **loss of function** of parts of the causing :

- **disturbances of sensation** such as **numbness** or **tingling**
- **disturbances of movement** including **paralysis** or **weakness**
- **disturbances of vision**
- **disturbances of speech**
- **disturbances of balance** or **co-ordination**
- **disturbances of intellectual function**

Air bubbles lodging in the coronary arteries which supply the heart with blood may lead to symptoms resembling a **heart attack** including **chest pain, shortness of breath** and **palpitations**. Air bubbles lodging in the circulation of the skin cause white or purplish patches (**marbling**).

If a diver surfaces after a deep dive and develops disturbances of brain function it is also possible that he might have **cerebral decompression sickness**. It may not be possible immediately to distinguish between air embolism and cerebral decompression sickness, now both called **acute decompression illness**. Fortunately the initial treatment is similar for both (see Case History 33.5).

❑ **Treatment.**

Air embolism causes hypoxic damage by obstructing important blood vessels with air bubbles. **Recompression therapy** in a chamber reduces the size of the bubbles and allows them to flow on, into smaller less-important blood vessels. The bubbles ultimately pass into the venous system, may be trapped in the lungs, and are eliminated as nitrogen diffuses out of them.

Divers who develop air embolism after free ascent or in submarine escape training, where a recompression chamber is available nearby, are immediately recompressed to reduce the air bubbles from their original size and so reduce the brain damage. The depth chosen will depend on the severity of the condition. High oxygen pressures are administered to reduce the bubble further. Otherwise, **transport** to a chamber must be arranged urgently.

There is some controversy about the best way to **position** the patient. Divers used to be taught to place the patient in the 30 degrees head down position, to keep the rising bubbles away from the brain. This caused difficulty with resuscitation and transport however, and the increased venous pressure in the head worsened the cerebral oedema (brain swelling) which accompanies injury. It is no longer advised.

A more reasonable approach is to place the patient **horizontally, on their side** (the left side is theoretically preferable but this is probably not critical) without a pillow. This will place the head slightly lower than the heart. The patient is likely to be unconscious or drowsy and this position is also good for patency of the airway. This is often called the **coma position**.

The patient should **not be allowed to sit up or stand up** once this position is adopted as there are some case reports of divers rapidly deteriorating after sitting up, even some time after the barotrauma event. This may be due to bubbles rising to the brain.

The patient should be given **100% oxygen** to breathe. After an hour of breathing 100% oxygen, it is probably safe to allow him to assume a more comfortable position.

If the patient is unconscious the principles of airway management, breathing and circulation management (**A–B–C**) take precedence and must also be followed (see Chapter 42).

The **other complications** of burst lung, such as pneumothorax or emphysema, must also be looked for and treated, if present.

The definitive treatment is **recompression therapy** in a well equipped chamber. Urgent transport is necessary in order to minimize brain or other essential organ injury. Unfortunately, even in the best facilities, full recovery is not always possible.

Predisposing Factors

❑ **Breath-holding.**

This may be due to failure to read "*Diving Medicine for Scuba Divers*", **panic, ignorance, forgetfulness or spasm of the larynx after inhalation of water.** The first rule that any aspiring scuba diver should learn is to exhale ("blow bubbles") during ascent. Breath-holding during ascent can lead to excessive distension of the lungs, and their rupture.

❑ **Air trapping.**

Anything preventing air from leaving all or part of the lungs can lead to pulmonary barotrauma.

Several factors may predispose to air trapping. Obstruction of the bronchi is frequent in; **asthma, acute and chronic bronchitis, respiratory tract infections.** This obstruction may allow air to enter the lungs but restrict exit of air – a ball valve effect. Other conditions which can cause this include; **tuberculosis (T.B.), tumours of the lung, calcified glands, cysts in the lung and emphysema. Heavy smoking may cause mucous plug obstructions.**

❑ **Disorders of lung compliance.**

Lung compliance is a measure of the stretching ability of the lungs. One published study investigated pulmonary barotrauma in Navy divers who had correctly exhaled during ascent and were previously medically fit. Studies of the lung compliance of these divers showed their lungs to be more "stiff" than normal, and therefore presumably more prone to tearing when slightly over-expanded.

Divers with **scars or fibrosis in the lungs** may have localised reduction in lung compliance which may cause shearing forces and tearing in these areas. Fibrosis of this type may be found after inflammatory lung disease such as **sarcoidosis, tuberculosis, lung abscess** or even some **severe pneumonias.**

Lung tearing has occasionally been described in **breath-hold divers.** These divers developed pneumothorax and mediastinal emphysema during breath-hold dives — the tearing of the lungs being caused by the diver taking very large breaths, with excessive respiratory pressures. On investigation, these divers were found to have relatively small lungs and relatively large chest cavities. Full expansion of the chest cavity in these divers led to over-expansion of the lungs and subsequent tearing.

❑ **Rapid ascents**

Any partially obstructed airway may restrict airflow. This can be overwhelmed by the massive volume changes which occur during rapid ascents. This risk can be reduced by adhering to the recommended slower ascent rate of 9 metres or 30 ft per minute, upon which most new decompression tables are based.

A slow ascent rate (9 metres or 30 feet/min, or slower), as in most Diving Manuals) is strongly recommended by the authors.

This rate may also help to reduce the risk of developing serious decompression sickness. The bottom time should be reduced accordingly



Fig. 11.7

❑ Emergency ascents.

The sudden failure of gas supply, especially at considerable depths, tends to alarm even the most sanguine diver. The subsequent emergency ascent is often undertaken with rapid ascent rates. Breath-holding due to anxiety together with a rapid gas expansion, greatly increases the likelihood of pulmonary barotrauma.

❑ Free ascent training (or "Emergency swimming ascent training").

A "free ascent" is a manoeuvre in which the diver breathes from compressed air equipment, takes a breath and then returns to the surface without breathing further from the equipment. Naturally, he must exhale to exhaust the expanding gas – but he may still encounter several problems during ascent. Most divers, aware of the dread consequences of breath-holding, tend to exhale excessively and may run out of breath before surfacing.

In many navies free ascent training, pulmonary barotrauma, salt water aspiration syndrome and near drowning were not uncommon accompaniments of these exercises. Thus, a **recompression chamber** and a **specialised diving physician** had to be available immediately adjacent to the practice ascent site. Perhaps because they are of less value, civilians do not impose the same standards.

Unless closely supervised, the rate of ascent is usually excessive, especially from greater depths since the diver knows that he only has one lung full of air to sustain him until the surface is reached. In fact he has the equivalent of 3 lungs full of air when compressed air is breathed at 20 metres depth. The excessive rate of ascent causes rapid gas expansion and damage if airways are partially obstructed.

This outmoded training technique was designed to prepare the divers to cope with an "out-of-air" situation in days before contents gauges, octopus rigs, and emergency air supplies were common.

Unfortunately deaths from this procedure made it a questionable practice. Also, the tendency of divers to over-inhale before commencing the ascent made the procedure more hazardous and not at all similar to the genuine out-of-air situation, where lack of air is usually detected after exhalation. Thus, the lungs in a real "out-of-air" situation are not fully inflated. As this 'real situation' usually happens without any inspiratory capability, it is presumably safer.

❑ Submarine escape.

Escape from a sunken submarine usually involves a rapid, buoyancy-assisted free ascent. This technique is practiced by most navies from depths of 20–30 metres in especially made submarine escape training facilities (SETF). Ascent rates are very fast and pulmonary barotrauma can occur in spite of good training and thorough preliminary medical examinations.



The emphasis on escape procedures demonstrates the optimistic outlook of submariners since the submarines often operate in water depths which exceed the crush depth of the submarine's hull.

Fig. 11.8

The Royal Australian Navy's S.E.T.F. in Western Australia.

❑ **Buddy breathing.**

Sharing a single regulator with a buddy, when his regulator fails, is not easy to achieve and unsuccessful attempts at buddy breathing, especially during ascent, are often followed by the diver abandoning the procedure and undertaking a free ascent to the surface. Either diver may tend to over-inhale prior to handing over the regulator and then breath-hold during ascent, while waiting for its return. These conditions are conducive to pulmonary barotrauma.

Employing an octopus regulator (a spare regulator with a longer hose) or another air supply system, is now almost universal, to facilitate safer buddy breathing. See Chapter 5.

Prevention of Pulmonary Barotrauma of Ascent

❑ **Medical fitness.**

Divers should be carefully screened to ensure there are no respiratory problems that predispose to pulmonary barotrauma (asthma, fibrosis, cysts, pneumothorax, infections, etc). Divers who do burst their lungs and survive are much more likely to have recurrences, which are then more severe – often with fatal consequences. Thus an episode of pulmonary barotrauma usually precludes further diving.

❑ **Diving techniques.**

Divers are advised to avoid situations which could lead to them having to perform an emergency free ascent. Such situations include greater **depth**, **reduced air supply**, **overweighting** and/or **excessive buoyancy**.

The use of well maintained **good quality equipment** (e.g. regulator), a **contents gauge**, an **octopus rig** or better still, and an **emergency air source** (to avoid the need for buddy breathing) are common sense measures which can be employed.

It is important for scuba divers to remember to **keep breathing normally** at all times, as a relatively small ascent in shallow water while the diver is holding his breath can lead to over-pressure (distension) of the lungs and pulmonary barotrauma. "**Skip**" (controlled or reduced) **breathing** is dangerous because it increases the time when a diver is not breathing.

Pulmonary barotrauma is a not uncommon accompaniment of "**free ascent training**" (also called "**emergency swimming ascent training**"). Included in this category is "**ditch and recovery**" drill, where the diver performs a free ascent as he returns to the surface after ditching his gear on the bottom. The greatest volume changes due to Boyle's Law take place near the surface, so that free ascents from even shallow depths are not safe.

The concept of training novice divers in "free ascent" technique is controversial. Obviously it is desirable for all divers to be familiar with safe "free ascent" principles in case of equipment failure. However, if a diver develops a serious air embolism after a free ascent, a fatal outcome is likely, unless a recompression chamber with an experienced diving physician is available at the site of the dive. This facility is rarely available in sport diver training. Even a few minutes delay in instituting recompression has a significant negative influence on the outcome of treatment.

Studies conducted on submarine escape trainees in Sweden showed an almost 4% incidence of EEG (electroencephalogram or "brain wave") changes in these divers indicating sub-clinical brain damage, presumably due to minor air embolism. Studies of free ascents by trainees in the U.S. Navy showed an incidence of pulmonary barotrauma of 1 in 3000.

❑ **Out of Air (OOA) and Low on Air (LOA) Situation.** (See Chapter 34.3)

All scuba divers should surface with at least 50 ATA air remaining. If not, the dive procedure needs reappraisal. Most of the OOA problems are due to failure to comply with this rule.

Without an air supply, hypoxia is inevitable. Whatever the excuse, running out of air underwater is hazardous. Alternately, failure to surface is uniformly fatal. Thus the diver should have a planned "bail out" procedure to be used if OOA, to reach the surface.

The more effort used in an OOA swimming ascent, the more the diver risks unconsciousness from hypoxia, panic and carbon dioxide build up.

From the OOA diver's point of view, a procedure that could be followed, is as follows (in priority):

1. At the first sign of any problem with the air supply, signal to a buddy.

Do not chase after him unless he is very close or is between you and the surface. Any unnecessary effort will further deplete your already limited air supply.

2. Commence a controlled ascent to the surface.

3. Unless in an enclosed space (a wreck, cave, etc.), ditch the weight belt – or unclasp it and hold it away from your body, so that in the event of more problems arising, it will be ditched automatically.

4. If the buddy responds to the signal by offering an air supply to you, in the form of a separate regulator, then remove your non-functioning regulator and accept his.

Unless you are well trained in buddy breathing and have practised this frequently with the rescuer, then it is usually not worth while to attempt an ascent while sharing a single regulator. Occasionally this may be necessary (such as when one is in an enclosed area).

5. If there is no secondary air supply available either from your own equipment or your buddies, then leave the regulator in your mouth, as some more air may become available due to the decreasing environmental pressure with ascent. It may also reduce the likelihood of salt water aspiration.

6. It may be necessary, depending on your equipment, to inflate the buoyancy compensator, but this is often neither prudent nor possible, because of the inadequate air supply. Inflating

the BC takes excessive time at depth and could also result in an accelerated ascent as the air in the BC expands, with ascent. More reliable and consistent buoyancy is obtained by ditching weights.

7. In the OOA situation there is little time available – but usually adequate if it is not wasted. Unnecessary and difficult underwater dialogue, especially followed by reassurance from narcotic rescuers-to-be, does not compensate for an air supply. Contacting everybody (no matter how important they may be) and evaluating the situation, is a topside indulgence. In some circumstances it is necessary to perform a rapid ascent, risking the possibility of decompression sickness and barotrauma, in order to avoid the inevitability of drowning .

**If totally OOA, it is often preferable to ditch the weight belt and surface rapidly,
exhaling if and as required.**

From the **buddies** point of view it is prudent to :

1. Supply a secondary air source to the OOA diver. This is usually done via an octopus regulator or an alternative air source. In a panic situation, this may be done by giving him your own regulator, because this is what he grabs, because he can see it and knows it is functioning, and for you to use one of the secondary regulators.

2. Control the ascent of the OOA diver, assisting this by ditching his weight belt. It is preferable not to ditch your own weight belt as it may be necessary for you to descend later. As your buddy is already OOA, this requirement will not be needed by him. If necessary to obtain adequate buoyancy, it may sometimes be necessary to ditch your weight belt also.

3. Once you reach the surface, retain a secure hold on the OOA diver, as he could lose consciousness from CAGE, within the subsequent few minutes, and sink. Also, once you are on the surface, inflate his BC, either by the inflator button or orally. This will ensure his buoyancy and reduce his anxiety.

4. Attract attention and assistance by the use of many of the techniques already referred to in this text e.g. whistle, flare, safety sausage, etc.

PULMONARY BAROTRAUMA OF DESCENT

(LUNG SQUEEZE)

There is a slight risk of pulmonary barotrauma during descent as well as ascent, although from a different mechanism.

A diver descending during a breath-hold dive will have the air in his chest and lungs progressively compressed in accordance with Boyle's Law. Eventually a lung volume is reached when the compression of gas can no longer be accommodated by a further reduction in lung volume, and is instead compensated by the engorgement of blood vessels in the lungs. The lung blood vessels have only a limited ability to distend, and can be expected to rupture once this limit is exceeded, causing pulmonary haemorrhage.

A rapid descent when standard dress equipment is used, or failure of a surface-supply gas pressure in the absence of an effective non-return valve, are also possible causes of pulmonary barotrauma of descent. It is theoretically possible whenever a surface-supply of air is used e.g. standard dress, surface supply from a compressor or compressed air tanks, or pumping the air supply from the surface in commercial devices.

It is more likely in breath-hold diving, but case reports of this condition are infrequent and poorly documented. It is more likely in deep, record breaking attempts. The theoretical basis of the condition was severely tested when a world record descent to beyond 200 metres was made by a breath-hold diver several years ago.

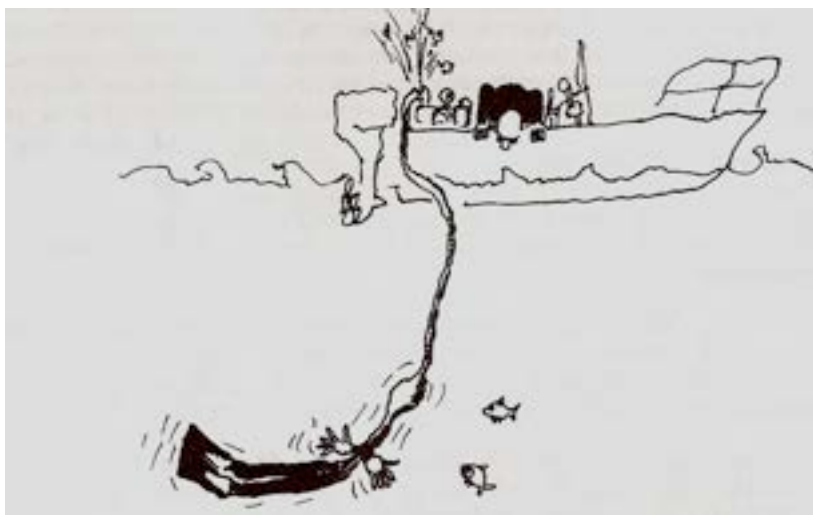


Fig 11.9

SSBA with no non-return valve, when the compressor stops

Chapter 12

OTHER BAROTRAUMAS

Barotrauma can develop wherever there is an enclosed gas space adjoining tissues. With descent, the space contracts, pulling tissue and blood into the space (implosions). With ascent, the space expands and disrupts tissue (explosions). Because gas changes are greater near the surface, barotrauma is more likely in the shallower depths.

The spaces may be within the body or between the body and the equipment. They include :

- **Facial (mask) squeeze**
- **Skin (suit) squeeze**
- **Body squeeze and "blow up"**
- **Gastro-intestinal barotrauma**
- **Dental barotrauma.**

Barotraumas dealt with earlier include lung, ear and sinus barotraumas.

FACIAL BAROTRAUMA OF DESCENT

(MASK SQUEEZE)

During descent the airspace inside a face mask is compressed and the contraction in volume of the gas space is accommodated by flattening of the mask against the face, and later by congestion of the facial skin and eyes.

It can lead to bleeding into the soft tissues under the skin and produce a characteristic **bruised** facial appearance under the mask area. The whites of the eyes may be grossly haemorrhagic ("**red eye**"). It may take 1–3 weeks to clear up.

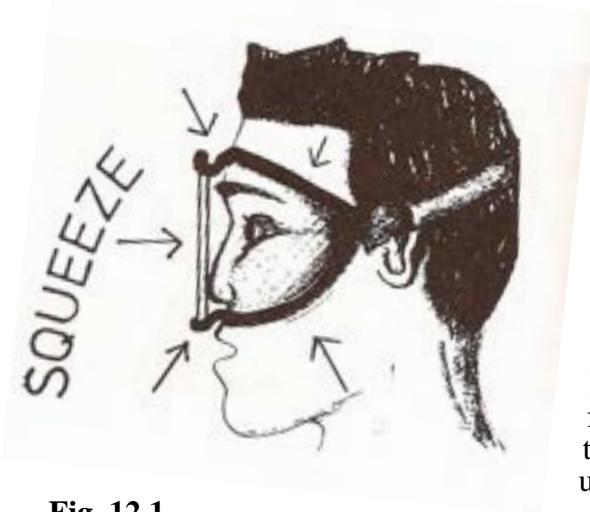


Fig. 12.1

This condition is easily prevented by exhaling into the face mask during descent, to equalise the mask with the water pressure. More cases have developed since rigid plastic face masks replaced the soft rubber ones. Expanding gas automatically vents around the edge of the face mask during ascent.

Divers using swim goggles run the risk of a similar form of barotrauma on a smaller scale involving the tissues around the eye. In the past, if goggles were used to dive, a method of equalising the space around

the eye, such as that shown in diagram 12.2, was employed.



Fig. 12.2



Fig. 12.3 Facial barotrauma from diving with goggles – note the bleeding into the sclera (white parts) of both eyes. Face masks can cause similar eye bleeds, but then the surrounding facial tissues are also bruised.

SKIN BAROTRAUMA OF DESCENT

(SUIT SQUEEZE)

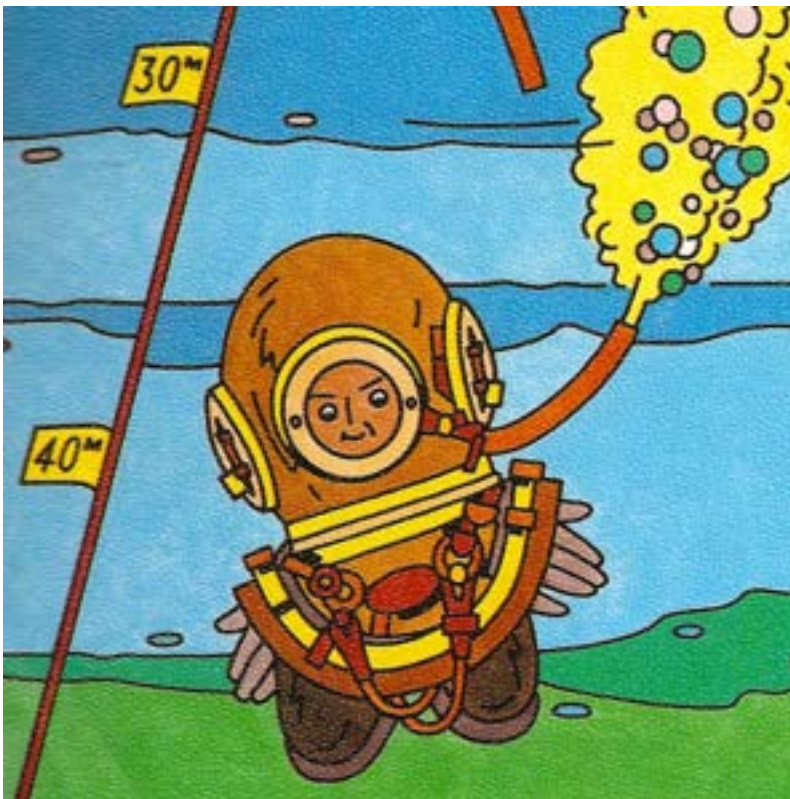
Divers using a **dry suit** or a **loose wet suit** may experience this problem. During descent, pockets of gas can be trapped in folds under the suit. Where the suit has folds, the contraction of the gas space is accommodated by the skin being sucked into the space leading to strips or welts of bruising.

It may cause discomfort at the time. After surfacing the diver may notice bruising over the skin, corresponding to the folds.

BODY BAROTRAUMA OF DESCENT

(BODY SQUEEZE)

With the solid metal helmet used in standard diving there is a possibility of the diver descending and the pressure in the air hose not keeping up with the environmental pressure. If this occurs, the diver's body may be forced up into the helmet, and crushed.



This can also happen at a constant depth, when a non-return valve is not used (or is not functional) and the air supply fails. The only treatment for this bizarre injury is to wash out the helmet with a good antiseptic.

Even the modern plastic helmets, used in deep and helium diving, can cause minor variants of this condition.

Fig. 12.4

SUIT BAROTRAUMA OF ASCENT

("BLOW UP")



With either dry suit or standard dress, the gas in the suit can expand with ascent, causing increasing buoyancy, more rapid ascent, etc. and a vicious circle develops where the diver may hurtle to the surface and be imprisoned in a balloon-like inflated suit. Special training and emergency procedures are needed for recreational divers who wear this equipment. As well as the physical injury that may result, other barotraumas and decompression sickness are likely.

Fig 12.5

GASTROINTESTINAL BAROTRAUMA

Gas is normally present in the gastro-intestinal tract. This finds its way into the atmosphere from time to time, as those who consume prunes, baked beans or cabbages will attest.

During a dive, gas may be swallowed when the diver equalises his ears, especially if in the inverted (head-down) position. It may accumulate in the stomach and gastrointestinal tract without initially causing any discomfort to the diver. During ascent however, this accumulated gas increases in volume, and can result in cramping colicky abdominal pain, belching and vomiting. Rare cases of stomach rupture have even occurred.

Divers are advised not to equalise their ears in the "head down" position.

Several cases of severe gastric discomfort have been reported during chamber dives when the divers drank carbonated beverages while under pressure. One amusing account relates the opening of a new hyperbaric facility which was toasted by champagne at 20 metres depth. The occupants were disappointed that the champagne appeared to be flat, but they drank with relish anyway because it tasted good. Their discomfort was exceeded only by their embarrassment during ascent as the gas in the champagne came out of solution and expanded in their stomachs.

DENTAL BAROTRAUMA

This uncommon form of barotrauma has on occasions been given sensational publicity, causing some divers to believe that they carry potential bombs set into their jaws.

Decayed teeth can occasionally contain a small air space which may lead to the tooth crushing inwards (implosion) during descent or fragmenting painfully (exploding) during ascent. The latter happens when there is an opening sufficient to allow air to enter during descent, but insufficient to allow it to escape during a fast ascent. As divers age, they develop small air spaces around their teeth, resulting in dental pain on descent. Because the air space is constant, the depth at which the barotrauma occurs also tends to be constant.

The explosive potential of this occurrence during descent or ascent has been overrated.

Diving within several days of a tooth extraction may occasionally allow air to enter the tissues through the tooth socket from the positive air pressure generated by breathing through a regulator. This results in air tracking into tissues around the face (**tissue emphysema**). This is rarely serious and is treated by the diver breathing 100% oxygen for several hours to eliminate the air.

Diving after any tooth extractions should be avoided until the tooth socket has healed — usually this takes about a week to ten days.

Chapter 13

DECOMPRESSION PHYSIOLOGY and SUSCEPTIBILITY

Decompression Sickness (DCS) is an illness caused by the effects of gas coming out of solution to form bubbles in the body after diving. It is due to the effect of Henry's Law (see Chapter 2) following diving exposures. Understanding decompression theories is difficult if not impossible, so the average diver may well bypass most of this chapter, if he is not technically inclined.

In sport divers the main gas formed in bubbles is nitrogen (N_2) because these divers almost always breathe air. However, the same principles apply to other inert gases, such as helium (He), which may be breathed by deep commercial and technical divers.

GAS UPTAKE

When a diver breaths air from scuba equipment at depth, N_2 is breathed at an increased partial pressure. Because gas diffuses from areas of high concentration (high partial pressure) to areas of lower concentration, N_2 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 N_2 , and therefore the amount of N_2 absorbed. Early in the 20th century, Haldane applied this concept to decompression.

The speed of N_2 distributing to the tissues depends on their blood flow. Tissues with high metabolic needs such as the brain, heart, kidneys and liver receive most of the blood pumped from the heart. They will also receive most of the N_2 carried in the blood and will have a **rapid N_2 uptake**. Such tissues are termed "**fast tissues**" because of their fast N_2 uptake.

Because blood passing through the lungs immediately equilibrates with any change in inspired N_2 partial pressure, blood is the fastest tissue of all.

Other tissues such as ligaments, tendons and fat, with a relatively small blood flow, have a relatively slow N_2 uptake. These tissues are termed "**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 N_2 in a tissue is **exponential** i.e. it varies depending on the amount of gas already taken up by the tissue. As the tissue takes on gas, the uptake slows because the partial pressure gradient decreases.

The filling of a scuba cylinder is an example of an exponential process. When an empty cylinder is connected to a high-pressure source, the cylinder initially fills quickly, but the flow slows as the pressure in the cylinder increases and approaches that of the gas source.

The uptake of gas in any tissue is initially rapid but slows with time. Accordingly, it may take a long time for a tissue to become fully saturated with gas, but fast tissues become saturated sooner than slow tissues.

Since the exponential uptake takes a long time to reach completion, even if it starts rapidly, the concept of **tissue "half times"** is used to compare tissues. The half time is the time taken for a tissue to reach half its saturation level. A fast tissue may have a half time as little as a few minutes, while a slow tissue may have a half time of some hours.

GAS ELIMINATION

N_2 is eliminated in a reverse of the uptake process. As the diver ascends there is a reduction in the partial pressure of N_2 in the air he breathes, allowing blood to release N_2 into the lungs. The decrease in the blood level of N_2 causes N_2 to diffuse into the blood from the tissues. Fast tissues naturally unload N_2 quicker than slow tissues.

Theoretically, tissues should lose N_2 exponentially, and most decompression tables are calculated on this assumption. While large amounts of N_2 are lost initially, the process slows with time and it may take 24 hours or longer for all the N_2 taken up during a dive to be released. Diving again during the time of N_2 elimination will mean that the diver will start his second dive with a **N_2 retention** in some tissues. Adjustments are provided in the decompression schedule to allow for this and are incorporated as the **repetitive dive tables**.

If there is diminished circulation to a tissue during decompression, gas elimination will be reduced and thus bubble formation will be more likely.

In practice, even during routine conservative dives, bubbles of N_2 frequently form in the blood and tissues, interfering with N_2 elimination. It has been estimated that as much as 5% of N_2 taken up by the body after some dives is transformed into bubbles on decompression. These are often termed "**silent bubbles**" since they usually do not produce any symptoms. They do however have a profound and unpredictable influence on the decompression requirements for repetitive diving, because it takes much longer to eliminate gas bubbles in tissues than it does gas in solution.

SATURATION

When tissues are subjected to an increased partial pressure of inert gas during a dive, they take up dissolved gas in accordance with Henry's Law. However, there is a limit to the amount of gas which can be dissolved by a tissue exposed to any given partial pressure of gas (i.e. depth of dive). When this limit is reached the tissue is said to be **saturated**.

Our bodies are normally saturated with N₂ at atmospheric pressure and contain about one litre of dissolved N₂. If a diver were to descend to 20 metres (3 ATA) and remain there for a day or more, his body would take up the maximum amount of N₂ possible at that pressure and would then be saturated at that depth. His body would now have about 3 litres of N₂ dissolved in it.

Once the body is saturated with inert gas at a given depth, it will not take up more of that gas, no matter how long the diver spends at that depth. Consequently, once the diver is saturated the decompression requirement does not increase with time. This economy of time is exploited in **saturation diving**, when the diver is kept at depth for very long periods of time (days, weeks, months) but then needs only the same lengthy decompression.

BUBBLE FORMATION

The process of bubble formation can be demonstrated easily by opening a bottle of beer (or champagne, depending on taste and income). In a carbonated beverage CO₂ is dissolved in the liquid at a high pressure, which is then maintained by the lid. When the lid is opened, the pressure over the liquid becomes atmospheric and the partial pressure of CO₂ in solution exceeds the critical limit for bubble formation, causing bubbles to form. This could be avoided if the pressure was reduced slowly (decompressed).

During ascent, the pressure surrounding the diver (the environmental pressure) is reduced. Eventually, the pressure of N₂ dissolved in the tissues may become greater than the environmental pressure. The tissue is then said to be **supersaturated**.

The tissues are able to tolerate a certain degree of gas supersaturation. Nevertheless, Haldane explained that if the pressure of N₂ in the tissues exceeds the environmental pressure by a critical amount, then bubble formation is likely. The pressure differential needed to cause this varies between tissues but with most scuba diving it equals or exceeds 2 : 1 (i.e. the partial pressure of inert gas in the tissues should not be more than twice the environmental pressure). This explains why DCS under recreational diving conditions is unlikely after an isolated dive to less than 10 metres — the pressure at 10 metres is 2 ATA, while the pressure at the surface is 1ATA — a 2:1 ratio..

Gas bubbles in the tissue and blood are the cause of DCS. The exact mechanism of bubble formation is complex. It is likely that microscopic gas spaces (**bubble nuclei**) exist in all body fluids and that these form a nucleus for bubble formation during decompression.

Bubbles can form in any tissue in the body including blood. The pressure in each bubble will be the same as the environmental pressure (if it was not, the bubble would expand or contract until it was) and the bubble size is governed by Boyle's Law as the pressure changes.

At the onset of DCS, the pressure of N_2 in the tissues is supersaturated (greater than the environmental pressure) so there is an immediate diffusion (pressure) gradient of N_2 which then diffuses into any bubbles (or nuclei) present, causing them to expand.

A bubble of DCS contains mainly N_2 if the diver has been breathing air, but the other gases present in the tissues, such as carbon dioxide (CO_2), oxygen (O_2) and water vapour, also diffuse into it.

Once a bubble has formed its behaviour depends on several factors. Any increase in pressure such as diving or recompression will reduce its size while any decrease in pressure such as ascent in the water, over mountains or in aircraft, will expand it. The bubble will continue to grow in any tissue until the N_2 excess in that tissue has been eliminated. Once this has occurred (which may take hours or days) the bubble will begin to decrease in size but it may take hours, days or weeks to disappear. In the meantime the bubble can damage the tissues around it.

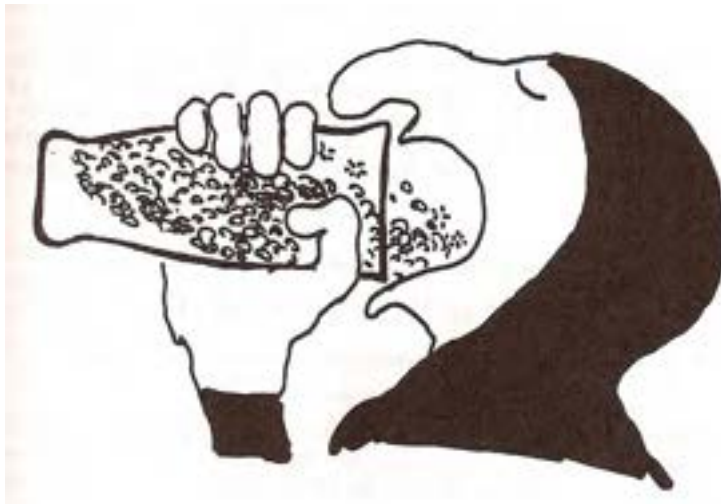


Fig. 13.1

There is good evidence that bubbles frequently form in tissues and blood of recreational divers after routine no-decompression dives, even when the tables have been faithfully followed. These bubbles do not usually cause symptoms but certainly cause doubt about the validity of the tables.

Tissue damage by a bubble results from several factors. Bubbles in the blood obstruct blood vessels in vital organs such as the brain, while bubbles forming in the tissues may press on blood vessels and capillaries obstructing their blood flow. Bubbles in the blood can also stimulate the clotting process causing the blood to clot in the blood vessels, obstructing blood flow to vital organs, and reducing the ability of the remainder of the blood to clot adequately. In the brain, spinal cord and other tissues, bubble pressure in or on nerves may interfere with nervous system function.

DIVE PROFILES

The type of dive has a significant bearing on where and when bubble formation takes place. **Short deep** dives (i.e. deeper than 30 metres) **tend to cause bubbles in the fast tissues** (blood, brain and spinal cord) while **long shallow dives tend to produce bubbles in the slow tissues** (like the joints). Long deep dives cause bubbles everywhere.

This distribution occurs because:

- in short dives, only the fast tissues take up enough N_2 to form bubbles on ascent and
- after shallow dives, fast tissues eliminate their relatively modest N_2 excess before a critical pressure differential develops.

It can thus be seen why it is important to ascend slowly. The slower the ascent, the longer the time for fast tissues to eliminate N_2 through the lungs, before a critical N_2 pressure-differential develops.

Diving folklore contains a myth that a diver using a single 2000 litres (72 cu. ft) tank cannot develop DCS. The air supply available was said to limit the diver to safe dive profiles. This is only true at very shallow depths and even then only partly so, e.g. for a dive to 20 metres, the average endurance may be about 30 minutes, which is within the no-decompression time given by most tables. Remember though, as mentioned previously, that any dive in excess of 10 metres can produce DCS.

The myth may become more apparent for deeper dives. For example, a single 2000 litre tank will give around 10 minutes duration for a 50 metre dive. According to most decompression tables, a 10 minute dive to 50 metres will require 10 minutes of decompression — but there will be no air remaining to complete these stops. Even if there was sufficient air, dives to this depth have a significant risk of DCS despite the tables being followed correctly.

FACTORS INFLUENCING DCS

DCS is unpredictable. In general, anything that increases blood flow to an organ will increase the rate of N_2 loading. Anything that interferes with blood flow from an organ will reduce the capacity to off-load N_2 . These alterations may explain the possible predisposing factors that increase the likelihood of DCS.

□ Depth/duration.

Any dive deeper than 10 metres can produce DCS although in general, the deeper the dive, the greater the risk. The longer the dive, the more gas absorbed (until saturation). The more the DCS.

□ Individuals.

Some people appear to be more susceptible to DCS than others. Even an individual may vary in susceptibility at different times, and DCS can develop after a dive profile which has been safely followed on many previous occasions. Others frequently get DCS despite conservative diving.

□ Adaptation.

Repeated dives to similar depths over a period of time reduce the incidence of DCS. This may be due to the elimination of bubble nuclei. A diver returning to these dives after a 2 week break loses the benefits of this adaptation or acclimatisation.

□ Age.

Older divers tend to be more predisposed to DCS (an old diver can be defined as anyone older than the senior author of this text). This age factor probably comes into play after the 3rd decade.

□ Obesity.

This appears to be a predisposing factor probably due to increased N₂ solubility (4-5 : 1) in fat compared to water (obesity is defined as anyone heavier than the biggest author). This may be relevant for those with a BMI of > 25.

□ Debilitation.

Factors causing the diver to be unwell such as **dehydration**, **hangover** or **exhaustion** tend to predispose to DCS.

□ Injury.

DCS, particularly involving the musculo-skeletal system and joints, is more likely with recent bruises, strains or chronic injuries.

□ DCS.

A previous episode of DCS, especially if it was unexpected from the dive profile, or if it damaged tissue (as in neurological DCS), makes the diver predisposed to similar subsequent episodes.

□ Patent Foramen Ovale.

One reason for some people to have an increased susceptibility to DCS is that they have a small hole in their heart. All of us had a hole in our heart when we were a foetus. In about a third of the population some remnant of this hole remains, it is called a patent foramen ovale, or PFO. These people have an increased susceptibility to DCS, the likely reason is that bubbles that would normally be trapped in the lungs without causing symptoms pass through the hole, by-passing the lung filter, and on to other parts of the body, where they cause noticeable symptoms. However, most results suggest the risk from a PFO is not great enough for it to be appropriate to test all divers for this.

❑ Cold.

Diving in cold conditions makes DCS more likely, especially when the diver is inadequately insulated. More precisely, coldness during the dive inhibits inert gas uptake (because of restricted circulation) but allows more N_2 to dissolve in body fluids — whilst coldness during decompression inhibits inert gas release. Theoretically, it may be better to be cold during the dive and warm on decompression, unless bubble formation occurs. Warming will then reduce gas solubility and increase bubble growth and DCS.

The association between cold exposure and DCS is complex and contentious. During decompression and post-diving the cold environment may cause peripheral constriction of blood vessels and more bubble formation. Alternately, taking hot showers also tends to cause increased bubble formation and DCS.

❑ Alcohol and other drugs

It has been observed that divers who over-indulge in alcohol, or perhaps take other **drugs** or medications, may be especially susceptible to DCS. In the case of alcohol, especially taken the night before, the effects may be due to the associated dehydration or the vascular dilatation (remember the throbbing headache and “hangover”), increasing N_2 take-up.



❑ Exercise.

Strenuous exercise during a dive is likely to increase the N_2 uptake by increasing blood flow to muscles, increasing gas uptake and favouring DCS development. Gentle exercise during decompression, by promoting circulation from the tissues probably aids in N_2 elimination. Strenuous exercise after the diver has returned to the surface makes the development of DCS, particularly in the musculo-skeletal system, more likely by promoting bubble formation. Strenuously exercising (shaking) a beer can, before opening it, aptly illustrates this phenomenon. During the first hour after a dive, particularly when there has been a large N_2 uptake, it is best to rest quietly as this is the period of maximal N_2 elimination.

Fig. 13.2

❑ Physical Fitness.

The less physically fit the diver, the more likelihood of DCS, probably because more energy is used and more blood flow is required for the same outcome – transporting more N_2 .

❑ Gender.

There is some evidence that women have a higher incidence of DCS for certain dive profiles. There are subtle differences in physiology and body composition which could explain this. The decompression tables in current use only evolved after extensive testing on men alone (see Chapter 8).

❑ **Dive profile.**

Deep dives (greater than 18 metres), **long dives**, **decompression dives** and any dives exceeding the **limiting line** (in RN based tables) all have a higher incidence of DCS.

❑ **Rapid ascents.**

These allow insufficient time for N₂ elimination from fast tissues, thus encouraging bubble formation.

❑ **Multiple ascents.**

Multiple ascents during a dive imply multiple decompressions and often involves rapid ascents. Bubbles in the blood (fast tissue bubbles) are likely to form during these ascents. The bubbles may not be adequately filtered by the lungs, passing along into the tissues, or may be reduced in size during the second or subsequent descent, allowing them to escape through the pulmonary filter into the tissues. DCS is then more likely.

❑ **Repetitive dives.**

Each repetitive dive begins with a N₂ load of some degree from the previous dive. Since bubble formation even after routine dives is common, a repetitive dive will often start with the diver carrying N₂ bubbles from the previous dive. These bubbles will be supplemented by N₂ taken up during subsequent dives, and make DCS more likely.

Also with repetitive dives there may be the residual physiological effects of the previous dive, increasing the likelihood of decompression sickness. These physiological effects may include a lower body temperature, dehydration from immersion and recent exercise.

❑ **Reverse Dive Profiles**

Divers are advised to do their deep dive first, in repetitive dives, and to dive to progressively shallower depths when multi-level diving. If this order is not followed, DCS is more likely.

❑ **Flying after diving.**

The jet age often finds divers flying home after a dive holiday within hours of their last (sometimes literally) dive. International airliners are pressurised to an altitude of about 2000 metres (6500ft.) above sea level. This means a pressure reduction on the diver of about 25% with a corresponding increase in the degree of N₂ supersaturation as well as a corresponding increase in the size of any bubbles he may be carrying. The increase in size of critical bubbles may be sufficient to provoke symptoms.

❑ **Decompression meters/computers** (see Chapter 14).

Using computers that are based on largely invalidated **theories** (as opposed to practical diving and decompression table experience) may result in a diver getting much more time underwater while diving — and in the recompression chamber during treatment. Both can be included in his log book if he survives.

❑ Multi-Factorial Effect.

Often there is more than one factor increasing the likelihood of DCS. Thus in one large Australian series over half the cases engaged in multiple dives, deep dives (greater than 30metres) and/or had ingested alcohol within 8 hours. Another 20% were precipitated by aviation exposure. Thus many of these divers would have had at least 2 factors increasing their likelihood of DCS.

Note: The senior (elder) author believes that the only explanation for most cases of DCS lies in the random application of Chaos Theory, which he also does not understand, or string theory which no-one understands.

Chapter 14

DECOMPRESSION TABLES & METERS

Since the work of **J.S.Haldane** (a British physiologist) early last century, decompression tables have been based on mathematical models of gas uptake and elimination in the body. He believed (wrongly) that the exponential rates of uptake and elimination of gases would be equal, and that body tissues could tolerate a supersaturation pressure gradient of air equal to 2:1, without causing bubble formation. He experimented on goats – the closest animal model that he could get to the diver – to determine this gradient. Thus a goat could tolerate 2 ATA of air pressure in its tissues, even when walking around at 1 ATA! Or, it could ascend from 6 to 3 ATA, 4 to 2 ATA and from 2 to 1 ATA, without bubble formation.

Haldane devised mathematical equations (later referred to as models or algorithms) that would represent a diver, and applied this to dive exposures. He postulated 5 hypothetical tissues (with half-times of 5, 10, 20, 40, 75 min) and decompressed the diver so that the supersaturation gradient in each of these 5 tissues would never exceed 2:1. Once any tissue reached that ratio, the diver would stop his ascent and “stage” there until the tissue “off-gassed” to allow him to ascend to the next stop. Thus bubble formation would be avoided by this decompression.

Although used by the Royal Navy for 50+ years, it was evident that some of Haldane’s dive stops, or stagings, were too conservative and others too radical. Also, the assumptions on which the procedure was based were wrong.

The acclaimed 1957 **U.S. Navy Tables** are based on Haldane's theories. These include equal exponential uptake and elimination of inert gases and supersaturation gradients – as described in Chapters 1 and 13 – but have been modified by experimental trials and practical diving experience. In an attempt to overcome the flaws in Haldane’s tables and to make decompression safer, they increased the number of hypothetical tissues to 6 (5, 10, 20, 40, 80 and 120 minutes), and calculated different maximum safe supersaturation ratios (now called **M values**) for the different tissues at different depths.

Following experiments and reviewing of the established decompression tables, between 1960 and 2000 a whole series of innovations and modifications were introduced. Dr Bruce Bassett (a USAF physiologist) concluded that the US Navy Tables resulted in an excessive incidence of about 6% DCS, when pushed to the no-decompression limits. Merrill Spencer in Seattle verified this observation and supported it with extensive Doppler monitoring, showing that bubbles developed in many routine dives – implying inadequate decompression. Many others observed similar inadequacies and in an attempt to cope with this new information, tables

based on modified Haldane principles were developed by Bassett, Huggins, NAUI, PADI, and many others.

The main alterations to improve safety were in;

- reducing the acceptable no-decompression times by 10-20%
- reducing ascent rates from 18 m/min to 9-10 m/min (at least in the top 30m)
- Adding a “safety stop” of 3-5 min at 3-5 m.

The Swiss decompression expert, Professor **Buhlmann** produced the Swiss model, which includes 16 theoretical tissue compartments with widely ranging half-times. The testing of these tables at altitude became more extensive than for most other tables, and he extended the concept into repetitive dives. Much later the US Navy E/L model assumed an exponential uptake of gas and a linear loss to eliminate gas

In the UK, Hempleman introduced a slab diffusion concept, which later evolved into the RNPL/BSAC tables. A cylinder of tissue was used by Hills in Australia. Other groups assumed the presence of bubbles or bubble forming nuclei, bypassing the traditional gas-in-solution supersaturation concepts, and decompressed in such a way as to keep the bubbles at a tolerable level. These are sometimes referred to as “**bubble models**”

Yount in Hawaii developed tables designed to keep gas nuclei from forming larger bubbles, and the allowable pressure gradients across these nuclei were less than Haldane’s supersaturation ratios. Thus the no-decompression times were less, the initial decompression stops were deeper and the ascent rates were slower. Weinke developed these concepts further in the Reduced Gradient Bubble Model, and has collected some diving data based on these tables.

The Canadians (**DCIEM**) considered the transfer of gas between adjoining tissues as a major principle, and produced a very well researched table, popular amongst conservative recreational divers. Although based on decompression theory, it has been modified by extensive human testing in cold water and hard working conditions, with Doppler (ultrasound) monitoring. The single no-decompression times, and most repetitive dives, are more conservative than the US Navy tables and are often recommended for recreational diving.

Another approach involves the collecting of a vast number of well-documented dive profiles, some safe and some unsafe, and letting the computer devise the safe tables. Extending this concept, the US Navy, and now DAN, use such statistical analyses to determine the probable DCS risk with each dive profile and so “probabilistic” tables that accept a defined DCS incidence of 1%, 2.3% or 5% can be derived – in theory.

When the formal tables were pushed to the limits (decompression diving, deep diving and dives approaching non-decompression limits), there was an unacceptable 1–5% incidence of DCS. Even in divers who appeared unaffected, Doppler (ultrasound) studies often showed bubbles in the major veins during decompression. Nevertheless, in both recreational and navy diving there is only a 1/5,000 to 1/10,000 incidence of clinical DCS. This paradox is explained by the fact that most dives do not approach decompression or depth limits and so do not really test the tables. Also, divers include conservative modifications or “fudge factors”, making their diving safer. Even the US Navy has modified its “gold-standard” table by reducing ascent rates and incorporating safety stops, making it marginally safer than it was.

It is not possible to make a perfect mathematical model of the decompressing human body. Even under normal conditions, the blood flow to some capillary beds shuts off from time to time, while flow to other areas increases. During decompression, nitrogen elimination will virtually cease from an area of shutdown capillaries. As decompression proceeds, this area will have a much higher N₂ concentration than predicted. No mathematical model can predict biological phenomena such as this.

When one considers the ill-defined complexities of gas transport through the range of different tissues, all with different solubilities, different supersaturation tolerances, different nuclei quantity and location - all at different depths, durations and ascent rates - the accurate representation of this with mathematical equations, models or algorithms, is impossible at this time.

Any table that offers longer durations underwater, deeper or more dives, will result in a greater incidence of decompression illness.

The imminent release of "new" tables is a permanent rumour. The difficulty in producing mathematical models which truly reflect human decompression physiology upon which to base the tables, as well as the difficulty and expense of testing them, makes the development and validation of tables based on truly new models unlikely. However, tables based on recycled mathematical models do frequently arise and often there is an amalgamation of decompression theories.

Towards the end of last century recreational diving involved more repetitive dives and multi-level diving. It reflected the interests of recreational divers, taking diving holidays, on live-aboard boats, exploring reefs and drop-offs. This was considerably different to the pre-planned single-depth/designated-duration dives of navies and commercial divers. With much recreational diving, relatively little time is spent at the maximum depth. Attempts to accommodate for these different diving profiles were attempted, with ingenious innovations by Graver, Huggins, the PADI RDP and Wheel, etc., with variable success. Others, such as the Buhlmann tables were more easily modified to incorporate the greater number of variables with multi-level and repetitive diving.

For any given nitrogen loading, multi-level diving from deep to shallow should produce less DCS than the single-depth dives documented in the formal dive tables. However, decompression from the infinitely variable multi-level diving is almost totally based on theory. There have been no adequate and comprehensive trials performed to show the tables reliability.

For both theoretical and practical reasons, most dive authorities advised divers to dive to progressively shallower depths, when repetitive or multi-level diving. This is referred to as a "Forward Dive Profile". About a decade ago there was a misplaced academic campaign to accept "Reverse Dive Profiles" as having equivalent decompression obligations, as an acceptable diving practice, without incurring decompression penalties. Both experimental evidence and diving experience subsequently showed that the traditional "deep dive first" concept is correct, in both multi-level and repetitive diving.

DECOMPRESSION METERS (DCM)

With the preponderance of multi-level diving, repetitive diving and multi-day diving, the expedient introduction of decompression meters (DCM) and decompression computers (DC) resulted in a gradual reduction in the use of established tables and an acceptance of the new technology.

These meters are based on three different principles:

❑ Mechanical models of gas transfer.

The early meters in the 1960s relied on the movement of gas through small orifices to simulate uptake and elimination of nitrogen by compartments of the body. They are obviously a gross oversimplification of the gas transfer in divers but one, the **SOS meter**, was moderately safe for single shallow non-decompression dives to less than 24 metres.

❑ Electronic models of existing tables.

These devices of the 1970s recorded the depth and time of a dive and related this to one of the existing decompression tables which was stored in its memory. One was even incorporated into a wristwatch. They saved the diver the difficulty of recording his depth and time, reading and remembering the tables.

These meters were not been very popular, because they did not offer any decrease in decompression obligation compared to the conventional tables and represented quite an expense merely to save the diver the trouble of applying a decompression table.

❑ Decompression Computers (DC)

These are electronic models based on decompression theory. Microprocessors developed for computer use were incorporated into the diving world with the Edge (USA) and Decobrain (Switzerland), in the 1970s. Instead of following the tables, with their inevitable rounding up of depths and durations and the associated safety factors involved in overestimating pressure exposure, they were far more precise in their measurements and calculations. This meant that they could meticulously track the dive exposure and use algorithms to calculate in real time the nitrogen uptake and decompression required, according to any predetermined decompression theory. The safety of the DC then depends on the validity of the theory.



DCs are of great value to divers, if they are used correctly. They have more accurate gauges (depth, duration) than previously available and this information is stored in a more reliable computer than the divers' narcotised memory. The incorporation of an ascent meter and alarm is invaluable in warning of rapid ascents.

Fig. 14.1

These meters are programmed with one of the mathematical models (theories) on which the conventional tables have been based (usually the US Navy, Buhlmann or “bubble” tables) and this allows for multi-level and repetitive diving calculations. Because of this, and the avoidance of the rounding-up safety features inherent in the tables, the DCs usually permit longer dive durations, more flexibility in dive depths and repetitive dives, shorter surface intervals and less intellectual demand than “table diving”. There are currently over a dozen different algorithms determining decompression in DCs. In general medicine, there is an axiom that the more treatments that there are available, the less likely it is that any work well. Same with diving medicine and DCs .

Flexibility can be incorporated and with the more advanced DCs, there are optional safety factors and restrictions. One can determine whether the DC behaves more conservatively than the theoretical algorithm normally calculates, allowing for increased age, weight, importance etc – but at the inevitable cost of a reduced dive duration. Many also indicate altitude (flying) restrictions post dive.

Probably over 90% of divers now use DCs instead of decompression tables, and considering the poor comprehension of the tables, this is probably a good thing. It certainly means that when an accident does occur, a comprehensive dive profile can be downloaded onto a computer for analysis, in retrospect – and often to the embarrassment of the diver who pleads innocence.

❑ Disadvantages of decompression meters/computers.

Sophisticated electronics, batteries and sea water are sometimes not compatible. Machines and technology sometimes fail. Back up plans are advisable.

The price paid for more dives and longer durations is increased DCS.

Most DCs employ algorithms that have not been adequately validated. When each of the conventional decompression tables was first formulated from a theoretical model, it had to be extensively modified based on human testing. Essentially, the meters are offering the tables as they were, before they were modified with safety factors. With the removal of the tables rounding-up safety factors, the price divers pay for their extended underwater durations is that they can dive much closer to the DCS margin.

Most of the new DCs are (like the old SOS meters) conservative for short single, shallow dives. The dangers increase with longer, repetitive or deeper diving. They are also probably less safe with some multi-level profiles, if reverse profiles are used.

Nevertheless, decompression meters will continue to be popular. They are perceived as an alternative to personal responsibility and are easy solutions to a difficult problem (decompression calculations). To question the value of any specific computer is interpreted as casting aspersions on the owners and brings a deluge of unsolicited testimonials and threats of legal action by the manufacturers.

❑ Safety suggestions (the DC Ten Commandments).

If you rely on DCs, the following recommendations are made:

1. Do not dive in the 24 hours before using the DC.
2. The DC should be used on **all** dives, if it is used on **any**.
3. Ensure a back-up documentation of the dive profile and details.
4. In multi-level dives, the depths should be progressively shallower.
5. Repetitive dives (on the same day) should be progressively shallower.
6. Repetitive dives should have surface intervals of at least 2 – 4 hours (longer with greater depths and longer dives).
7. With multiple days diving, every fourth day should be non-diving.
8. Add an extra decompression safety stop for 5 minutes at 5 metres, on each dive in excess of 15 metres depth, if practicable.
9. Do not do dives that require decompression or go into the decompression mode. Stay as far away from those dives as possible.
10. Do not presume that the DC is accurate for diving at altitude or for altitude exposure (flying).

DCs are preferably used at depths of less than 30 metres and definitely depths of less than 40 metres. Pseudo-science accompanied the technology in the promotion of these DCs, and many claims of excellence were more applicable to the colour brochures and marketing than any research or development activities.

For those who are more susceptible to DCS (age, gender, fitness, weight, medications, injuries, DCS history, etc.) or undertaking more hazardous dives (depth, duration, decompression obligation, temperature, currents, repetitive, multiple ascents, etc), more conservative options should be chosen on the DC.

Chapter 15

DECOMPRESSION SICKNESS CLINICAL FEATURES

ACUTE DECOMPRESSION ILLNESS

This term (ADI) was introduced for divers or paramedics who were unable to differentiate between cases of decompression sickness (DCS) and air embolism (secondary to pulmonary barotrauma of ascent). Sometimes the differential diagnosis is difficult, and rarely the diseases may even co-exist. As the treatment of neurological DCS and arterial gas embolism from pulmonary barotrauma is usually the same, differentiating these illnesses is not essential initially, and so the term is tolerated.

Eventually, in planning future advice to the diver, it is necessary to be more specific.

CLASSIFICATION OF SYMPTOMS

In the past it was traditional to describe decompression sickness (DCS) as Type 1 (minor – musculo-skeletal or joint) or Type 2 (serious - cardio-pulmonary and neurological) DCS. Now the convention is to classify the clinical features according to the organ or system involved (e.g. neurological, musculo-skeletal etc.). It is prudent to include with this diagnosis an indication of the evolution of the symptoms (as described later), as both influence the treatment required

ONSET OF SYMPTOMS

The clinical features of DCS are seen during or after ascent. In the majority of cases, symptoms will be evident within six hours, and 50% within the first hour of the dive. Less commonly, a delay in onset of 24 hours or greater has been described. Divers often deny the reality of their symptoms, or will rationalise them. In one large series, only half the divers requested assistance within 12 hours of the first symptom developing.

The time of onset of symptoms depends to some degree on the type of dive. Deep dives (greater than 30 metres), especially those that require decompression or are close to the no-decompression limits or in which decompression has been omitted, are likely to present early. In extreme cases, symptoms may present during ascent or at the decompression stops. In general, the earlier the symptoms, the more potentially serious the DCS.

Symptoms may be initiated or aggravated by exposure to altitude (driving over mountains, air travel), exercise or breathing certain gases. Divers should be advised of the serious complications of flying after diving.

JOINT PAIN (MUSCULO-SKELETAL DCS, BENDS)

Pain in or near one of the muscles or tendons around the joints is the **most common presenting feature of DCS from shallow diving**. The shoulder is most often affected while the elbows, wrists, hand, hips, knees, ankles are less frequent. It is not unusual for two joints to be affected, commonly adjacent ones e.g. the shoulder and elbow on the same side. It is rare for multiple joints to be affected in a symmetrical pattern.

Symptoms may begin with discomfort or an abnormal feeling in or near the joint. Over the next hour or two, pain and other symptoms may develop. The pain is generally of a constant aching quality (like a toothache), but occasionally may be throbbing. The diver may hold the joint in a bent position to reduce the pain. The stooping posture, that was adopted by Caisson (tunnel) workers affected by DCS near the hip, led to the term "bends".

The joint is usually not tender to touch but movement may aggravate the pain. Pressure, as from a blood pressure cuff (sphygmomanometer), may relieve the pain.

If not treated, pain usually continues for several uncomfortable days before slowly subsiding. In mild cases, minor and fleeting discomfort lasting only a few hours ("**niggles**") may be the only manifestation.

Symptoms are often found around a joint which has been subjected to unusual exertion or strain during or after the dive or which has been involved in a recent or chronic injury.

There may occasionally be difficulty distinguishing between DCS and other causes of a painful joint such as strain, injury or arthritis. In the latter conditions, the joint is usually tender to touch and may be red and swollen. Generalised arthritis is often bilateral and

symmetrical and involves smaller joints, and local pressure application produces no relief. These signs are uncommon in DCS.

In general, any pain in or near a joint after compressed air dives in excess of 10 metres (or shallower with repetitive or prolonged dives) must be assumed to be DCS until proven otherwise.

NEUROLOGICAL DCS

DCS can affect the **brain, spinal cord and/or peripheral nerves**. The clinical features are due to disturbance of activity in the nervous system, interfering with one or more of its five principal functions:

- **sensation**
- **movement** (including balance and co-ordination)
- **consciousness** and intellectual functions
- **autonomic** functions
- **reflexes** (e.g. knee jerk, cough reflex).

Of these, the first four are easier for the layman to assess.

The Senses.

These include sight, hearing, smell, taste, pain and touch. Numbness and tingling (paraesthesiae) are frequent symptoms. Other abnormal signs include loss of sensation.

Movement.

This includes the ability to move any muscle, the strength of the movement and the ability to co-ordinate it.

Higher function of the brain.

The important intellectual functions are consciousness, orientation (awareness of time, person and place), thinking, speech and memory. Epileptic fits (convulsions) and confusion are possible. See the AMTS box at the end of this chapter for a rapid, simple method of assessing higher function brain involvement.

Autonomic functions.

Interference with the control of breathing and heart function may cause shock and collapse. Bladder and bowel malfunction usually causes progressive abdominal discomfort and tenderness until the bladder or bowels are opened.

In **CEREBRAL (Brain) DCS**, the bubbles of DCS may be located in or near the blood vessels supplying the brain, causing obstruction of blood flow and direct pressure on the neurological tissues. The brain swells like any other tissue when injured, but because it is

confined within the solid bone of the skull, the pressure in the skull rises, further impairing blood supply to other parts of the brain. Swelling of the brain (cerebral oedema), as well as expansion of the nitrogen bubbles themselves, often leads to a steady worsening of this condition.

The onset of cerebral DCS is often heralded by **headache** — probably due to brain swelling. **Numbness** or **tingling (paraesthesiae)**, **weakness** or **paralysis** affecting a limb or one side of the body, difficulty with **speech**, **visual** disturbances, **confusion**, loss of **consciousness** or **convulsions** are all possible presenting symptoms of this serious disorder.

The part of the brain responsible for co-ordination (the cerebellum) may also be affected causing incoordination known as "**staggers**". The position sense and the balance organs can also be affected.

SPINAL DCS has a common association with DCS bubbles in the blood and lungs, commonly known as "**chokes**". It also may be preceded by "**girdle pains**" — or pain around the chest or abdomen. Disturbances in movement such as **weakness** or **paralysis** or disturbances in sensation such as **numbness** or **tingling** are also common. Interference with nerve supply to the bladder and intestines, may lead to **difficulty in passing urine, or opening the bowel**. **Paraplegia** or **quadriplegia** may develop.

INNER EAR DCS

The cochlea (hearing) or vestibular (balance) organs may be involved. This type of DCS is more commonly associated with **deep diving, breathing a helium-oxygen mixture**. **Hearing loss, ringing noises** in the ears (tinnitus), and/or **vertigo, nausea** and **vomiting** are the usual presenting features. This condition must be distinguished from the other major cause of these symptoms in divers – inner ear barotrauma (see Chapter 9).

LUNGS or PULMONARY DCS

Nitrogen bubbles are frequently found in the veins of divers ascending after deep dives, without necessarily the development of overt clinical DCS. When large numbers of these bubbles form, they may become trapped in the small vessels of the lungs, obstructing the blood flow. If excessive bubbles occur, this leads to a disturbance of lung function and a feeling of **breathlessness**, known as the "**chokes**".

Clinical features also include a **tight feeling in the chest, chest pain, difficult** and **rapid breathing**. Coughing is often observed, and may be precipitated by smoking, hyperventilating or exercise. Frequently the symptoms come on very soon after ascent, from relatively deep dives (over 30 metres) or after prolonged dives. Death may supervene.

HEART or CARDIAC DCS

The nitrogen bubbles that commonly form in the veins of divers after deep dives are usually filtered by the lung vessels.

There is a condition affecting the heart (**patent foramen ovale – PFO**) which is sometimes claimed to be present in about 30% of the population, and in which there is a potential communication between the right and left sides of the heart – between the right and left atria. It is a flap valve, normally kept closed by the naturally higher pressure on the left side (left atrium) of the heart. When large amounts of nitrogen bubbles obstruct the lungs, the back pressure in the right atrium can exceed the pressure in the left atrium. This flap may then open allowing gas bubbles to pass from the right to the left side of the heart, and then be pumped and distributed to any part of the body (similar to arterial gas embolism resulting from a burst lung – see Chapter 11).

Bubbles passing through a patent foramen ovale (PFO), or any other cardiac defects, tend to rise because of buoyancy. They can be easily carried into the blood vessels supplying the brain because it has a large blood supply and is located above the heart. This is one cause of cerebral DCS after apparently so called "safe" dives. It may be called cerebral arterial gas embolism or **CAGE**, but is due to DCS, and not pulmonary barotrauma. The term **acute decompression illness** covers both causes.

Bubbles can occasionally pass down the coronary arteries, which supply the heart, restricting the blood supply to the heart itself. In severe instances this can lead to a fatal destruction of heart muscle (myocardial infarction) just as for a "heart attack" in a non-diving person. In other cases, life threatening disturbances in cardiac pumping and rhythm may result.

Cardiac symptoms include chest pain, palpitations and shortness of breath.

GASTROINTESTINAL DCS

Obstruction of blood flow to the intestines by nitrogen bubbles can occasionally affect the gut. Clinical features are not common, but can include **vomiting** or **diarrhoea**, **cramping abdominal pain** and **haemorrhage** into the gut. Severe cases can show clinical shock, and can bleed to death.

SKIN MANIFESTATIONS of DCS

These are not common in scuba divers who wear wet suits.

Itching of the arms and legs, sometimes with a rash, is not uncommon after deep recompression chamber dives and with dry suits. This condition is probably due to gas passing from the surrounding high pressure atmosphere into the skin. The condition is not serious and requires no treatment.

In more severe DCS, nitrogen bubbles in the blood can obstruct blood supply to the skin, causing patchy white, blue and pink areas – "**marbling**". Obstruction of the lymphatic system (drainage of tissues) may produce **localised swelling of skin**.

GENERAL SYMPTOMS of DCS

Apathy, tiredness, malaise, and a **generalised weakened state** are common observations in many cases of DCS. In very severe cases there may be **generalised internal haemorrhages, shock,** and/or **death**.

DELAYED SYMPTOMS of DCS

Prolonged symptoms may be due to damaged nerve, spinal cord or brain (see above), body tissues or bone (see Chapter 17). In these cases there can be a persistence or recurrence of symptoms. Various psychological problems can also supervene on DCS. Even the stressful treatments and peer recriminations can exact an emotional toll on divers. Anxiety states and post-traumatic stress disorders are not uncommon sequelae

EVOLUTION OF SYMPTOMS

In general, the slower symptoms are to develop after ascent, the less serious the DCS, and *vice versa*. Also, the longer the delay between ascent and symptoms (say, 6-24 hours), the fewer and slower subsequent symptoms are to develop. The corollary of this is:

With symptoms developing soon after the ascent, the DCS is likely to be more dangerous.

In assessing DCS, the time of onset of symptoms should be related to the time of ascent. The clinical manifestations and their evolution should be described, together with any aggravating factors.

The manifestations may be progressive (getting worse), static or improving. They may also relapse.

Aggravating factors include not only those that predispose to DCS, but also those that precipitate it (see Chapter 13).

A major observation, supporting the diagnosis of DCS, is the favourable response of DCS to raised environmental pressure (re-immersion in the water, treatment in a recompression chamber) and, with a slower response, administration of 100% O₂.

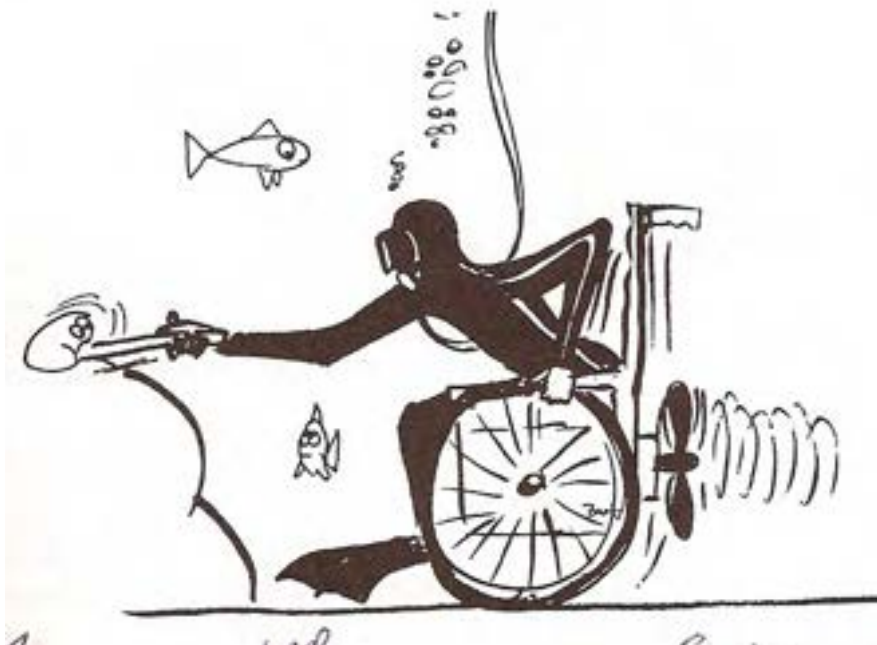


Fig. 15.1

The **ABBREVIATED MENTAL TEST SCORE (AMTS)** can be used to assess mild delirium, confusion and other cognitive impairment.

The following questions are put to the patient. Each question correctly answered scores one point. A score of 6 or less indicates impairment of brain function. A score above 6 does NOT mean that there is no cognitive impairment

Question

What is your age? (1 point)

What is the time to the nearest hour? (1 point)

Give the patient an address, and ask him or her to repeat it at the end of the test. (1 point)
e.g. 36 Smithsonian Street

What is the year? (1 point)

What is the name of the hospital or number of the residence where the patient is situated? (1 point)

Can the patient recognise two persons (the doctor, nurse, home help, etc.)? (1 point)

What is your date of birth? (day and month sufficient) (1 point)

In what year did World War 1 begin? (1 point)

(other well known dates can be used, with a preference for dates some time in the past.)

Name the present monarch/prime minister/president. (1 point)

(Alternatively, the question "When did you come to [this country]?" has been suggested)

Count backwards from 20 down to 1. (1 point)

As well as a "spot check", this test can be used to indicate improvement or deterioration, if repeated. It is not as comprehensive as others, which may be employed by paramedics and doctors e.g. the Mini-Mental State Examination, or the TYM.

Chapter 16

DECOMPRESSION SICKNESS TREATMENT

FIRST AID

□ Expert advice.

Expert advice from a diving physician, a diving medical organisation (such as DAN) or a hyperbaric facility, should be sought as soon as possible. **Appendices B and D** contain a list of sources for such expert advice. In the interim, first aid treatment must be applied

The principles of first aid management of decompression sickness (DCS) are:

- **basic first aid, A–B–C** — Airway, Breathing, Circulation . (see Chapter 42).
- **100% oxygen (O₂)**. (see Chapter 40).
- **positioning and rest**
- **fluid replacement.**

□ Oxygen therapy

If the diver breathes 100% oxygen (O₂), nitrogen (N₂) is removed from the lungs. The breathing apparatus must supply to the lungs as close to 100% O₂ as possible. This means an **anaesthetic type mask** or an O₂ diving regulator as used in technical and commercial diving. It is not the simple plastic hospital oxygen mask that does not produce an air tight seal. 100% O₂ in the lungs results in a high diffusion (pressure gradient) of N₂ from the blood to the lungs, causing the increased elimination of N₂ from the blood and tissues – and also from any bubbles there.

Unfortunately 100% O₂ can be toxic to the lungs if given for 18–24 hours on the surface. This may complicate the hyperbaric O₂ which is later given to the diver in a recompression chamber (RCC). Ideally O₂ therapy should be given under the supervision of a diving physician however, if expert advice is unavailable, then **all suspected case of DCS should be given 100% O₂** from the outset, before and during movement of the patient and transport to the recompression chamber.



Fig. 16.1

Photograph of diver with DCS breathing near 100% O₂ from a resuscitation bag. Note reservoir bag (lowermost) and high constant flow (>14 litres per minute) through an oxygen delivery tube.

Unconscious divers, if not breathing of their own accord, will require assisted ventilation (intermittent positive pressure respiration or I.P.P.R.). Conscious patients can be treated with continuous flow or demand type masks. In such cases, treating personnel must always check the breathing mask on themselves first in order to ensure the oxygen system is working, and that resistance to breathing is not excessive.

□ Position and rest.

An unconscious diver should be placed on his side in the coma position, to protect the airway at all times (see Chapter 42). If there is any likelihood of air embolism, the diver is best placed horizontal - preferably on the left side, but this is not essential. Some clinicians recommend that cases of cerebral DCS should be managed with the patient on the side, without a pillow, to prevent buoyant gas bubbles reaching the brain through the circulation.

The Trendelenburg (30 degree head down, feet up) position is no longer recommended. Having the head lowered raises the pressure in the brain – and this can aggravate the brain injury.

Due to the buoyancy of bubbles, sitting or standing may be dangerous in patients with air embolism or cerebral decompression sickness where the bubbles are still in the blood stream. As a general rule, 100% O₂ should be given for at least an hour before allowing the patient to sit or stand. After this, the diver can be allowed to adopt any comfortable position but should be kept relatively still and on 100% O₂. A diver with "chokes" will be more comfortable sitting up.

□ Fluid replacement.

Severe DCS results in loss of blood and fluids into the tissues. It may be valuable to replace this fluid orally or intravenously. Oral fluids may be given if the diver has no abdominal pain, nausea or vomiting. Water or electrolyte fluids (eg. "Gastrolyte") may be given – with the type and volume recorded. Acidic (orange juice) and glucose drinks should be avoided. If there is no bladder involvement (i.e. the patient can urinate), a litre of clear fluid every 2-4 hours should suffice. This fluid intake should be modified by the patient's thirst.

Some authorities have recommended large volumes of oral fluids while the diver is being transported to a RCC in an attempt to replace this deficit. One problem with this (as any party goer will attest) is that the fluid load will promote a vigorous urine flow, so the diver arrives

at the recompression facility with a stomach and bladder full of fluid. A patient with spinal DCS may be unable to empty the bladder and will therefore be in considerable pain.

The patient will usually be treated on a hyperbaric O₂ (HBO) treatment table. There is a very real risk of nausea, vomiting and convulsions as complications of this treatment. A full stomach can then possibly result in regurgitation of the stomach contents and aspiration into the lungs – further complicating treatment.

If the brain or spinal cord is involved and the patient has difficulty in voiding urine, an **in-dwelling urinary catheter** should be inserted whenever possible by a trained physician or nurse. If this is not feasible, care must be taken not to overload the patient with fluids.

Anyone who is trained to institute and monitor an intravenous infusion can be expected to be able to assess the state of hydration and determine the desirability and quantity of intravenous fluids, remembering that glucose fluids can aggravate cerebral oedema.

□ Drugs.

Aspirin as a first aid measure has not been demonstrated to be of value in DCS. It may interfere with blood clotting and cause haemorrhage (bleeding) – especially in the stomach. Haemorrhage is already a major pathology in spinal cord and inner ear DCS.

The authors have seen one patient with severe DCS bleed to death from an internal haemorrhage just before he was to be given an "experimental last-ditch" anti-clotting agent. We are therefore reluctant to advocate the routine use of aspirin either for pain relief or to inhibit clotting in any DCS case.

Joint pains of DCS can be significantly eased without the risk of serious side effects by the administration of **paracetamol (acetaminophen)** – 1000 mg (or two tablets) 4 hourly. NSAID drugs may be requested by a diving physician, but are not routinely needed.

Other drugs such as steroids, diuretics and special intravenous fluids such as "Rheomacrodex" have been advocated but have not been proven to be beneficial. Anti-epileptics and other drugs such as diazepam ("Valium") may be needed to control fitting (convulsions), and for confusional states.

TRANSPORT OF PATIENT WITH DCS

With mild DCS, or if there has been a delay of 12-24 hours or more, and there is no progressive deterioration, local treatment with rest, monitoring and breathing 100% O₂ may be all that is necessary. This decision is best made by a diving physician.

With more severely affected divers, or those that are deteriorating or need medical attention, transport should be expedited. The diver should be transported with **minimum agitation** and as close as possible to sea level or at **1 ATA**. Mountainous roads should be avoided whenever an evacuation route by land is planned. 100% O₂ should be breathed before and during transport (see Chapter 40).

Transportation in aircraft presents problems. Apart from movement which aggravates DCS, environmental pressure decreases with altitude, causing DCS bubbles to expand (Boyle's Law) and more gas to pass from the tissues into any bubbles.

If the patient is evacuated by air, unpressurised aircraft should endeavor to fly at the lowest safe altitude. Even an altitude of 300 metres (1000 ft.) can make the symptoms of DCS worse. However, maintaining such an altitude can be alarming when flying over 297 metre terrain.

It should be remembered that most commercial "**pressurised**" aircraft normally maintain a cabin pressure of around 2000 metres (6000 ft.), which could seriously aggravate DCS.

Whenever possible the cabin altitude should be maintained at 1ATA. This is attainable by many modern commercial jet aircraft, executive aircraft such as the King Air and Lear Jet, and some military transport aircraft (Hercules C-130). This requirement is not popular with the commercial airlines since it necessitates the aircraft flying at lower than its most efficient altitude, resulting in excessive fuel consumption. This requirement may also limit the range of certain aircraft.

Breathing 100% Oxygen before and during the flight may be of value, especially from closed or semi-closed circuit equipment. There are risks to the aircraft and its inhabitants if open circuit O₂ is used as many airlines recirculate the cabin atmosphere and fire/explosion is possible.



Fig. 16.2

A portable patient treatment chamber connected to a second compartment for the exchange and transfer of attendants and medics. The larger chamber also allows for transfer of patient to a larger recompression facility.

DEFINITIVE TREATMENT OF DCS

This is best controlled by diving physicians, hyperbaric facilities or specialised diving medical organisations, such as DAN.

□ Therapeutic recompression.

This is the most effective treatment for DCS. Delay increases the likelihood of a poor final result. The diver is placed in a **recompression chamber (RCC)** and the pressure is increased according to a specified recompression treatment table.

The increase in pressure reduces the bubble size (Boyle's Law) and usually relieves the clinical features. It also increases the surface area to volume ratio of the bubble, which may collapse. The increased pressure in the bubble also enhances the diffusion gradient, encouraging nitrogen to leave the bubble.

The therapeutic recompression schedule determines the depth and duration of the treatment profile. The table selected may depend on factors such as the time elapsed since the onset of symptoms, the depth of the original dive and the type and severity of symptoms, as well as the capability of the treatment facility and the various breathing gases available. The treatment may be amended depending on the patient's response.

In recent years the **Oxygen Treatment Tables** are preferred because of their increased effectiveness. The injured diver is usually compressed to an equivalent depth of 18 metres (60 ft.) and is decompressed over 3–5 hours. He breathes oxygen from a mask while the rest of the chamber is filled with air. If the whole chamber is filled with oxygen, the **fire risk** increases dramatically.

Because the attendant breathes chamber air, great care must be taken to monitor his dive profile to avoid the embarrassing predicament of an attendant emerging from the RCC with DCS.

The diver breaths oxygen for the duration of the treatment except for 5 minute periods of air – or sometimes heliox (He-O_2) breathing – every 25 minutes, to minimise oxygen toxicity to the **lungs**. The regimen is not without hazard as there is a significant risk of **cerebral oxygen toxicity** causing convulsions in such persons.

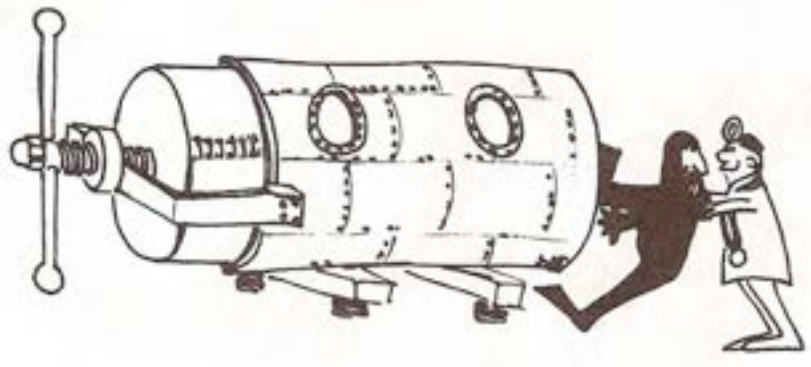


Fig. 16.3

Other tables are available which involve compressing the diver to a depth equivalent of 30–50 metres sea-water (100–165 ft.) in **air**, **heliox** and **nitrox** mixtures and then decompressing over periods ranging from several hours to several days depending on the severity of the symptoms.

Many other investigations and treatment modalities will be employed by experienced physicians in the RCC, including fluid balance, medications, etc, which need not concern the average diver.

A further emergency procedure, **Underwater Oxygen Treatment**, has been devised for use under expert supervision in remote localities. The diver is **recompressed in the water** to a maximum of 9 metres while breathing 100% O₂. Details of this procedure are outlined in Appendix C.

Treatment in water with the diver **breathing air** has been used in many parts of the world and water treatment tables are contained in some Navy diving manuals. While success has often been reported and delay in treatment can be avoided, this form of treatment has serious theoretical and practical difficulties which can result in worsening of the diver's condition.

The deep water air requirements (30 metres initially, and decompression for many hours)) renders the patient and accompanying divers prone to cold (hypothermia), narcosis, gas exhaustion, tide and other current changes. Attending divers may well develop DCS from extra exposures. This form of treatment is not generally recommended, unless other options (RCC, underwater oxygen treatment, etc.) are unavailable.

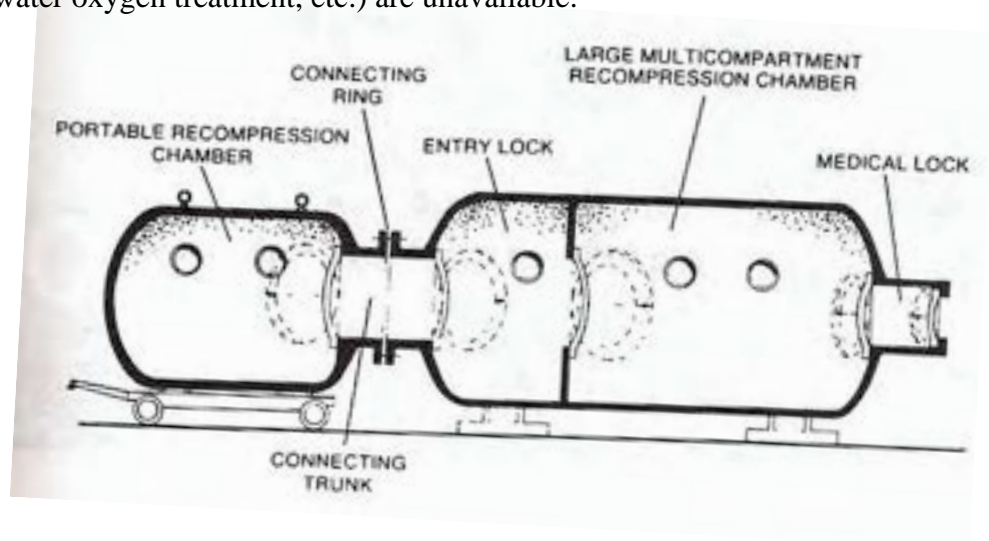


Fig. 16.4

Schematic outline of large static RCC treatment facility showing linking of a small portable chamber to the larger fixed unit. Both chambers are pressurised to identical pressures for a "transfer-under-pressure" (TUP).

□ Hazards of therapeutic recompression.

While therapeutic recompression generally produces a dramatic relief of symptoms, it has several serious hazards. These include **oxygen toxicity** in the patient, the risk of **fire** and the risk of producing **DCS in the attendant**. It should only be used under the close supervision of a medical officer experienced in its use.

PREVENTION OF DCS

There are a number of factors which predispose to DCS. These are described in Chapter 13. Obviously these should be avoided wherever possible but some are unavoidable. Apart from falsifying a birth certificate there is little an individual can do about the predisposing factor of age.

Because of the unreliability of the currently available dive tables and DCs, and the unpredictability of the development of DCS, it is possible for even the most careful, well trained and conscientious diver to develop this condition. The following suggestions will help reduce the risks.

It is important that the diver never exceeds the no-decompression limits and ascent rates. In spite of this, all the dive tables, and especially the dive computers so far devised, have a significant failure rate.

□ "Fudge factors".

The rate of development of DCS varies from less than 1% to as much as 5% depending on the table used, the depth and the duration of the dive, and if tables are pushed to their limits. The apparent safety of the tables is probably improved by intelligent divers incorporating "fudge factors" of their own. This is especially required for older, fatter and less fit divers. They imply ascending before it is theoretically necessary – reducing bottom times and depths.

□ Accurate depth & time.

It is essential that the diver knows accurately the depths and durations of his diving. A depth gauge which indicates the maximum depth attained is useful, because it is common for divers to descend deeper than they realise. An underwater **watch** or, better, a **bottom timer** is valuable, as is a D.C.

□ No-decompression diving.

Although the tables are not totally reliable, they are less reliable for deep diving (greater than 30 metres). It is advisable to avoid pushing the tables to the limits when a no-decompression schedule is followed and to avoid dives requiring decompression.

□ Slow ascent rates.

A slow ascent is prudent and the diver should certainly not ascend faster than the rate recommended by the tables. Preferably a slower ascent rate should be employed (8–10 metres or 25–33 ft. per minute is an acceptable safe rate) and the extra time taken is deleted from the bottom time. i.e. ascend earlier than permitted by the tables.

□ Routine decompression stops.

Most authorities recommend a routine minimum safety ("decompression") stop at **3-5 metres for 3-5 minutes**, after a no-decompression dive greater than 15 metres, to allow partial nitrogen elimination and trapping of venous emboli in the lung vessels.

□ Dive planning.

When repeated dives or multi-level dives are planned, the **deeper dives** should always be **performed first**. With **multi-day diving**, a rest day is included after each 3 continuous diving days. With deep diving, gradual build up (**acclimatisation**) is achieved by progressively deeper exposures.

□ Post-dive restrictions.

It is advisable to **rest** for an hour or more after a deep or long dive to ensure elimination of nitrogen from the fast tissues. Surface intervals should be > 2-4 hours. Flying and significant altitude exposures within 12–24 hours of diving is not recommended.

In addition to the above precautions, the diver is also advised to buy a good quality waterproof rabbit's foot. A healthy scepticism towards reliance on any mechanical equipment, especially if promoted by a glossy brochure or a dive computer salesman, also has good survival value.

□ Dive computers (See Chapter 14).

Chapter 17

DYSBARIC OSTEONECROSIS

**(DIVERS BONE DISEASE,
AVASCULAR NECROSIS OF BONE,
ASEPTIC BONE NECROSIS,
BONE NECROSIS, BONE ROT,
CAISSON DISEASE OF BONE)**

This was first noticed in caisson (tunnel) workers in the 19th century, and was described as being an area of localised bone death, predominantly occurring in the long bones of the arms and thighs.

If this area of dead bone is located beneath the joint surface of the bones in the hip or shoulder joints, pain and symptoms of arthritis, along with a reduction in mobility of the joint is a common consequence – often occurring in mid or later life.

The exact cause of the disorder is probably a delayed effect of damage caused by gas bubbles produced during a dive (see Chapter 13). In this sense it is a delayed form of decompression sickness.

Cause

Bone is a living organ containing bone cells which constantly absorb and lay down new bone. It has a cleverly designed structure which resembles reinforced concrete or fibreglass and contains fibres of a sinew-like substance called collagen, embedded in a concrete-like calcium material. This is traversed by numerous vessels which supply the blood to the bone cells embedded in the bone. The bone cells permit the repair of fractures and allow the bone to change its structure to accommodate stresses which may vary during the person's life.

If the blood vessels supplying the bone cells are blocked by gas bubbles or any other cause, the bone cells die and the self-repairing ability of the bone stops. It becomes unable to fix the

repeated minor trauma which is common around joints and eventually the bone structure collapses causing permanent damage to the major load bearing joints, such as the hips or shoulders.



Fig. 17.1

The reported incidence of this condition varies from less than 1% in some Navy series, to 80% in Chinese commercial divers. This variance is probably due to factors such as different diagnostic criteria and differing dive patterns and frequency.

Predisposing factors which are commonly associated with osteonecrosis include:

- **age greater than 30 years**
- **inadequate decompressions**
- **experimental dives**
- **deep dives**
- **decompression sickness**
- **long duration dives**

X-Ray changes have been seen as soon as 3 months after a dive and it has been reported following a single dive to 30 metres. When joint involvement does occur, the onset of symptoms is usually delayed for many years, reflecting the time required for joint destruction. Fortunately, in most cases the disease does not cause any serious damage to the joints and so produces no symptoms.

Occasionally bone pain may increase during recompression treatment, or may persist after treatment. Some of these progress to dysbaric osteonecrosis over the following months.

Classification of Bone Necrosis

The lesions are classified into two groups:

- **Type A lesions** – which are near the joint surface (juxta-articular).
- **Type B lesions** – which are remote from the joint surface (head, neck and shaft).

□ **Type A lesions.**

With these, the joints may become involved as the under-lying bone is destroyed and the joint surface collapses. This may produce symptoms which are potentially crippling. Hips and shoulders are more frequently affected.

□ **Type B lesions.**

These rarely cause symptoms and are generally of little clinical importance, except to suggest more conservative diving procedures. The most common areas affected are the long bones of the thigh, leg and upper arm. Occasional cases of bone cancer have developed in these lesions.

Clinical Features

When Type A lesions injure the joint, common symptoms are pain, which is usually aggravated by movement, in the affected joint and accompanied by a restriction of joint movement. As the condition progresses, severe osteoarthritis develops and the joint may eventually become frozen and incapacitating, due to pain.

Investigations

The lesions can also be identified in excellent detail, using MRI (Magnetic Resonance Imaging) scanning within days of the incident (but some of these may disappear). This is expensive but has no associated risks of irradiation. Early lesions can also be identified with radioactive Technetium ("bone scans"), which will bind to an osteonecrotic area and can be detected with a scanner within 2 weeks of the injury.

X-Rays have been the traditional investigative method but these will only reveal lesions once bone changes have developed. This may take months or years.

Treatment

The pain associated with movement can be reduced with an anti-inflammatory drug such as NSAIDS. Severe cases may require the fusion of a joint or its replacement with a synthetic joint made of either metal or plastic. While this procedure relieves the pain and increases mobility, a synthetic joint is never as robust as the "natural model" and its endurance is limited. Other techniques involve replacement from the divers healthy bone, or realignment of articular surfaces.

As the disease is regarded as an occupational hazard of diving, workers compensation claims may help off-set expensive medical costs.



Replacement hips may cause problems.

iously desirable. Most sensible y they should; avoid dives deeper than 40 metres, avoid dives requiring decompression, not approach the no-decompression limits and ascend slowly. It is likely that the longer duration dives permitted with many dive computers, together with the increased number of dives and the ability to dive close to the edge of decompression commitment, now makes this disorder more likely for recreational divers.

For divers who develop decompression sickness, a follow-up bone scan after 2–4 weeks should detect areas of bone damage. MRI imaging can often be better used to determine the extent of the lesion.

Divers with high risk factors who develop unexplained joint pain should be assessed to exclude this condition.

Occupational divers and other divers who are at increased risk because of their diving practices may require regular routine screening assessments. Since X-ray investigations can involve worrisome exposure to radiation, their frequency must be weighed against the risk of osteonecrosis development, and so MRIs are preferred. Divers who are likely to be at risk are required to have a baseline investigation performed before they are employed. For some susceptible occupational divers, follow-up assessments at 5 year intervals are recommended.



Replacement hips may cause problems.

Fig 17.3

Chapter 18

NITROGEN NARCOSIS

(COMPRESSED AIR INTOXICATION, RAPTURE OF THE DEEP, INERT GAS NARCOSIS, NARCS)

Intoxication in divers is not confined exclusively to beach barbeques and hotel bars. When breathed under pressure, nitrogen (which makes up 78% of air) has an intoxicating effect which, like alcohol, is variable and may lead to pleasure or disaster.

This phenomenon was regarded as an annoyance to the helmet diver who could be pulled to the surface by his attendant if he behaved irrationally, but the consequences to the scuba diver, who's safety is dependent on a buddy exposed to the same effect, can be more serious.

It will be present in all divers breathing air at a depth in excess of 30 metres, although some will notice it earlier. Others may not be aware of the effect, as judgment and perception are affected. The severity of symptoms and the exact depth of their onset varies between individuals. Because of narcosis, diving on air beyond 30 metres (100 feet) is not prudent, and 40 metres is considered unsafe for most recreational divers. A 50 metres depth is considered the maximum safe depth for experienced professional divers breathing air.

CAUSES OF NITROGEN NARCOSIS

The exact cause of this narcotic effect is uncertain. Nitrogen is classified as an inert gas because it does not participate in any chemical reactions within the human body. The influence of nitrogen on narcosis must therefore be due to some physical reaction.

When other inert gases such as neon, xenon and argon were investigated, it was found that their narcotic effect at depth correlated approximately with the relative weights of their individual molecules (i.e. their molecular weights). An increased molecular weight caused a greater narcotic effect. It was further shown that the inert gases which were more soluble in fat than water, tended to have a greater narcotic effect. There were unfortunately several inconsistencies in the behaviour of these gases, including hydrogen, which cast some doubt on these generalisations.

Other theories have been proposed implicating oxygen or carbon dioxide toxicity, lipid solubility and enzyme changes in the brain.

CLINICAL FEATURES



Fig. 18.1

The narcotic effect usually becomes effective within a few minutes of reaching a particular depth and does not worsen as exposure continues at this depth. Rapid descents may increase the effect, but with ascent it is dissipated.

The higher brain functions such as reasoning, judgment, memory, perception, concentration and attention tend to be the first affected by narcosis. This often leads to a feeling of well-being and stimulation in a diver secure in his surroundings. In a novice or an apprehensive diver, a panic reaction may follow. Some degree of tolerance develops at a given depth or with repetitive exposures.

The influence of narcosis may not be evident if the dive is uneventful, thus giving a false impression that the diver is in control of the situation. Memory and perception deficits may only be evidenced by a failure to follow instructions or the dive plan, or being inattentive to buoyancy, air supply or buddy signals. When a problem develops, the diver may be unaware of this – attention and perception being focused elsewhere (perceptual narrowing or "tunnel vision"). Thus emergency signals will go unheeded, emergency air supplies will not be offered, weight belts will not be released, rescue attempts will be crude and amateurish. Survival instincts and responses may be dampened. The safety of both the diver and his buddy are compromised.

Death may supervene due to errors provoked by impaired judgment or perception, and by over confidence. Loss of consciousness may happen without warning and be unnoticed by the diver's buddy. At great depths the diver may lose consciousness from the narcosis itself or the interaction between it and other factors such as sensory deprivation, carbon dioxide or oxygen toxicity.

Martini's Law - Table

20-30 metres	Mild impairment of performance on unpracticed tasks, mild euphoria
30-50 metres	Overconfidence and inadequate responses to danger. Perceptual narrowing, fixation on a particular function or exercise. Judgment impairment, affecting; air supply, buoyancy control. navigation, decompression obligations, ascent rates etc. Anxiety
50 metres	sedation , loss of judgment. Hallucinations possible.
50-70 metres	In a chamber, depending on conditions - talkative or terrified.
70 metres	Poor reasoning ability. Very poor response to signals or instructions.
70-90 metres	Poor concentration and mental confusion, stupefaction and loss of memory.
> 90 metres	Hallucinations and loss of consciousness.

Factors which are known to **increase** the effects of nitrogen narcosis include:

- **low intelligence**
- **fatigue or heavy work**
- **anxiety, inexperience or apprehension**
- **cold (hypothermia)**
- **recent alcohol intake or use of sedative drugs (includes seasickness medications), marijuana etc.**
- **poor visibility**

Factors which tend to **reduce** the effects of narcosis include:

- **strong motivation to perform a given task**
- **acclimatisation following prolonged or repeated exposures**
- **tolerance to heavy alcohol intake**

A diver who can "hold his liquor" is said to have a greater tolerance to nitrogen-narcosis. A plea of "acclimatising to narcosis" is generally not accepted by the courts however, as defence for an alcoholic intoxication charge.

The effect of nitrogen narcosis has been likened by some to that of drinking one martini on an empty stomach for every 10 metres depth (**Martini's Law**). The "olive" appears to be optional.

Case History Examples:

1. A group of divers descended into a deep clear freshwater cave in order to savour the pleasant intoxication of narcosis. Their bodies were found some weeks later in a deep confine of the cave. They were victims of over-confidence and impaired judgment induced by nitrogen narcosis.
2. Another diver became so elated during his dive that he removed his regulator and offered it to the other marine inhabitants.
3. A diver developed problems with his air supply but, possibly because of the 40 metre depth and narcosis, he did not attempt to ditch his weight belt. He triggered the dump valve of his BC instead of the inflation valve, and drowned with minimal struggling.

PREVENTION

Avoidance of compressed air diving to depths known to cause narcosis is a good policy. This implies a depth limit of 30 – 40 metres (100 – 130 feet) depending on the diver's experience, his tolerance to narcosis and the task performed. Safe diving beyond 30 metres requires an awareness of the ever increasing risk of this condition and its effects on human performance and judgment. Some experienced professional divers may be able to perform certain practised tasks at depths up to 60 metres with competency, but dives greater than 30 metres should be a source of concern for recreational divers and greater than 50 metres should be regarded as excessive even for professionals.

TREATMENT

A diver incapacitated by narcosis should be protected from injury and inappropriate behaviour, and brought to a shallow depth with a controlled ascent, bearing in mind decompression requirements. Symptoms clear rapidly as the nitrogen pressure is reduced. Any other symptoms present on surfacing (e.g. salt water aspiration and near drowning, decompression sickness etc.) are due to complications of experiencing narcosis at depth and not narcosis *per se*.

Chapter 19

HIGH PRESSURE NEUROLOGICAL SYNDROME

(HIGH PRESSURE NERVOUS SYNDROME, HPNS, HELIUM TREMORS)

This condition is a problem for deep commercial diving operations where helium/oxygen (Heliox) mixtures are breathed at depths in excess of 130 metres (430 feet). It causes a serious limitation to very deep diving and gets worse as the depth increases.

It is not a problem to recreational divers unless a grave (sic) miscalculation of depth or buoyancy has been made.

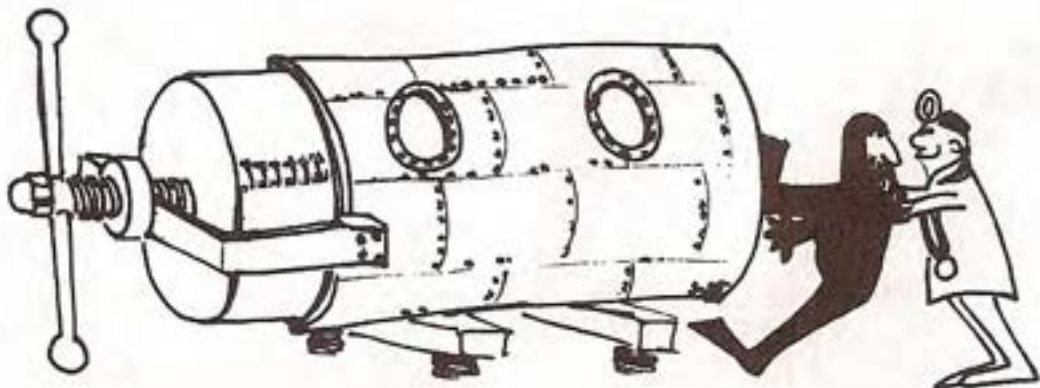


Fig. 19.1

CLINICAL FEATURES

The first sign is usually a mild uncontrollable tremor, with muscle twitching and difficulty in coordinating movements. If the diver then continues with the descent, confusion, drowsiness, disorientation and unconsciousness may follow. Respiration may also be affected by the neurologically induced incoordination of muscle activity. A tremor particularly affects the hands and arms and resembles the shivering due to cold (which helium breathing may also produce).

The condition is aggravated by rapid descent and thus a slow descent rate is a requirement in all deep diving operations using exotic gas mixtures.

The cause of HPNS is not fully understood. It is probably due to an excitation of a part of the brain by the direct mechanical effect of pressure. Evidence for this is that drug induced anaesthesia in animals can be reversed by simply compressing the animal to depths which provoke HPNS.

TREATMENT AND PREVENTION

Since the effects of HPNS resembled an excitation of the brain, early researchers reasoned that an agent which caused sedation might reverse the condition. Nitrogen narcosis is a common cause of sedation during diving, and so divers affected by HPNS were given small concentrations of nitrogen to breathe. This produced an effective reduction of some of the symptoms of HPNS. This helium-nitrogen-oxygen mixture (Tri-mix) is now used in most deep diving and by some technical divers. Small percentages of nitrogen have been included to reduce the HPNS – but not enough to cause significant nitrogen narcosis.

The added nitrogen in Tri-mix also permits better speech comprehension (because helium distorts sound production in the human larynx) and may slightly reduce the rapid heat loss with helium.

Chapter 20

HYPOXIA

(ANOXIA)

Hypoxia refers to an inadequate level of oxygen (O_2) within the cells. **Anoxia** implies there is no O_2 left at all in the cells, and is uncommon. Without O_2 most cells, especially those of the brain, die within a few minutes. This is the final outcome of many diving accidents and is often the ultimate cause of death.

Hypoxia is caused by an interruption in the chain of physiological processes (see Chapters 3 & 4) which bring O_2 from the outside air (or breathing gas) to the body's cells. There are **four links** in this chain where interruption can cause hypoxia, supplying a logical classification.

CLASSIFICATION OF HYPOXIA

HYPOXIC HYPOXIA

Inadequate Oxygen content in Arterial Blood

In diving, the most common form of hypoxia is **hypoxic hypoxia**. This usually follows an inadequate air supply and/or salt water aspiration or drowning. Either there is inadequate O_2 getting to the lungs because the diver has, for a variety of reasons, only water to breathe, or the lungs are unable to convey inhaled O_2 to the blood because of alveolar damage due to near drowning.

An inadequate air supply can also arise from an inadequate concentration of O_2 in the breathing gas (e.g. a gas mixture in which O_2 has been inadvertently omitted or an internally rusty scuba cylinder which extracts O_2). It may develop from equipment failure or obstruction somewhere in the respiratory tract between the nose or mouth and the alveoli, due to :

- upper airway obstruction due to unconsciousness
- tracheal obstruction from inhalation of vomit and
- alveolar damage from salt water inhalation (see Chapters 25 and 26).

Pulmonary barotrauma is another cause. A less common, but often catastrophic, cause is decompression sickness, when the gas bubbles are carried to the lungs in such quantities that the lungs cannot cope with them – resulting in “chokes”.

STAGNANT HYPOXIA

O₂ Taken Up by the Blood Fails to Reach the Tissues

This is generally due to failure of the heart to pump the blood adequately to the tissues (e.g. from a heart attack or air embolism). Poor circulation to the extremities in cold conditions can cause localised hypoxia to these areas without generalised hypoxia.

ANAEMIC HYPOXIA

Inability of the Blood to Carry O₂ in the Presence of Adequate Circulation

This is generally due to inadequate amounts of circulating functional haemoglobin, usually from blood loss or carbon monoxide poisoning (see Chapter 23).

HISTOTOXIC HYPOXIA

Inability of the Cells to Use the O₂

This is caused by certain poisons including carbon monoxide (see Chapter 23).

HYPOXIA IN BREATHHOLD DIVING

Drowning Syndromes

See Chapters 25 and 26. The inhalation of water into the lungs is the commonest cause of hypoxia in all types of recreational diving.

Hyperventilation

As explained in Chapter 4, hyperventilation before a breath-hold dive reduces the urge to breathe during the dive and may cause the diver to lose consciousness from hypoxia while still underwater (see Case Histories 33.2, 33.3) and with little or no warning. Drowning frequently results from this.

Hypoxia of Ascent

The partial pressure of O₂ in the lungs falls as they expand during ascent from a breath-hold dive. In some circumstances, this can cause loss of consciousness from hypoxia during ascent. Details are explained fully in Chapter 4.

HYPOXIA IN COMPRESSED GAS DIVING

Scuba

Exhaustion of the air supply, equipment malfunction, regulator resistance or loss of the demand valve will leave the diver with nothing but water to breathe – inevitably resulting in hypoxia due to salt water aspiration or drowning. Panic and poor dive techniques are often precursors to these problems.

Diving induced asthma, pneumothorax (from pulmonary barotrauma) and decompression sickness (chokes) can also interfere with breathing sufficient to cause hypoxia.

Rebreathing Equipment

This type of equipment shares the same causes of hypoxia as scuba equipment, but has some additional hazards.

A hypoxic gas mixture can be breathed if the **wrong gas** or **wrong mixture** is used (i.e. a gas mixture containing insufficient O₂). A specific example of this is when a gas mixture intended for use at great depth (e.g. one containing 5% O₂) is breathed near the surface. With rebreathing equipment using a constant flow of gas, the **flow of O₂** must be sufficient for the energy needs of the diver, and this will increase with **exertion**. Thus, O₂ concentrations too low, inadequate flow rates or excessive O₂ consumption may all lead to hypoxia in rebreathers.

Dilutional hypoxia is a particular problem with rebreathing equipment. When the diver first begins to breathe from the diving set, a significant amount of nitrogen may be displaced from the lungs and body into the counterlung (breathing bag) of the equipment. If this is not vented after a few minutes breathing, the diver is likely to rebreath almost pure nitrogen from the rebreathing bag. Oxygen is consumed by the diver and the carbon dioxide produced is

absorbed by chemicals used in the equipment. Because the counterlung will still contain gas (mostly nitrogen) the diver will be unaware of the danger. The O₂ % may be adequate at depth, but not near the surface.

CLINICAL FEATURES

In most cases of hypoxia, the diver is unaware that there is anything wrong and therefore can lose consciousness without warning.

Mild hypoxia starves the brain of O₂, causing confusion, impaired judgment and clouding of consciousness. The diver is frequently unaware that there is a problem and may even become over-confident. Sometimes he may notice a loss of colour vision, but this is infrequent. An observer should notice a deterioration of performance.

More **profound hypoxia** causes unconsciousness and in some cases, muscular jerking and spasms (especially of the jaw muscles) or epileptic type fits. **Severe hypoxia** results in rapid death.

Hypoxia makes the blood blue in colour. Hypoxic blood in the body capillaries gives the skin a blue appearance, and is termed **cyanosis**. It is not easily detected under the blue water, but is often obvious when the diver is surfaced

There is a form of localised cyanosis (stagnant hypoxia) associated with **cold** which does not denote generalised hypoxia. This is seen in the fingers and ears, due to (peripheral) blood vessel constriction causing inadequate circulation in these areas, in response to cold. It can be distinguished from the cyanosis of generalised hypoxia (hypoxic hypoxia) by looking at the colour of the tongue. The tongue is blue only in the generalised hypoxia.

TREATMENT

If hypoxia is due to insufficient O₂ in the cells, treatment should aim to reverse this. The basic resuscitation principles should be applied first (see Chapter 42).

- A. Clear the airway.
- B. Establish or maintain breathing.
- C. Establish or maintain circulation.

Give the patient the highest possible O₂ concentration to breathe and use positive pressure if needed (see Chapter 40).

All diving operations should be undertaken with emergency O₂ equipment available, sufficient to last until the diver can access more formal medical facilities. A rule of thumb is to allow for 15 litres of O₂ per minute. There should always be at least one person, who is not diving, cognisant of and trained in the application of the O₂ equipment. This is usually the boatman or dive operator.

PREVENTION

Most diving deaths are ultimately caused by the hypoxia associated with drowning, regardless of the initial problem. In many cases this can be avoided by the **buddy system**, which permits the diver to be rescued and employ positive **buoyancy** for the victim. A good buoyancy vest should keep even an unconscious diver's face clear of the water.

Avoid prolonged or deep **breath-hold dives**. **Never hyperventilate** before a breath-hold dive and avoid exertion as much as possible.

With **scuba**, maintain equipment adequately and check it before a dive. **Monitor the air supply** continuously, using a contents gauge.

With **re-breathing equipment**, check the O₂ concentrations before diving, also the gas flows and pressures. Avoid excessive exertion and flush through with appropriate gas before ascending.

Chapter 21

OXYGEN TOXICITY

Oxygen (O₂) is toxic when breathed at a partial pressure in excess of 0.4 ATA (40% O₂ at atmospheric pressure) for sufficient time. The greater the concentration and pressure, the shorter the time. The two common forms of O₂ toxicity affect the lungs and the brain.

When O₂ is breathed at partial pressures between 0.4 and 1.6 ATA it is eventually toxic to the lungs. At partial pressures in excess of 1.6 ATA, it is toxic to the brain as well as the lungs. The effects are more pronounced and more rapid as the inspired partial pressure of O₂ increases.

MECHANISM

The exact cause of O₂ toxicity is unknown. It is generally considered that hyperbaric O₂ interferes with the activity of enzymes in the cells and that this disrupts the biochemical functions, particularly in the brain and lungs.

In the lungs, damage to the cells lining the alveoli causes a general thickening and stiffening of the lung tissues.

In the brain there is a reduction in the amount of certain nerve transmission chemicals as well as generalised damage to the nerve cells. If cerebral O₂ toxicity is allowed to develop, convulsions eventually follow.

The sensory organs are really neurological outposts. Thus vision, hearing and touch, may also be affected.

PREDICTION OF O₂ TOXICITY

To calculate the inspired partial pressure of O₂, multiply the percentage of inspired O₂ by the ambient pressure in atmospheres absolute and divide by 100.

e.g. the partial pressure of O₂ in room air is :

$$21\% \times 1\text{ATA} \div 100 = 0.21\text{ATA.}$$

The risks of O₂ toxicity increase with increasing partial pressure. In general it is usually possible to breath 100% O₂ (1ATA) for 12–24 hours without developing significant pulmonary O₂ toxicity. This duration reduces as the O₂ pressure increases. If therapeutic recompression is contemplated, a maximum period of only 6-12 hours breathing 100% O₂ may be acceptable since the subsequent therapeutic recompression will generally involve the use of hyperbaric O₂, and this will summate with existing O₂ toxicity. The amount of pre-treatment of diving casualties with O₂ will preferably be discussed with the diving physician responsible for the therapeutic recompression.

Oxygen toxicity results from a combination of O₂ pressure and duration of exposure. Both must be considered and tables are available to indicate the maximum values allowable for different pressures and durations, for both respiratory and cerebral O₂ toxicity.

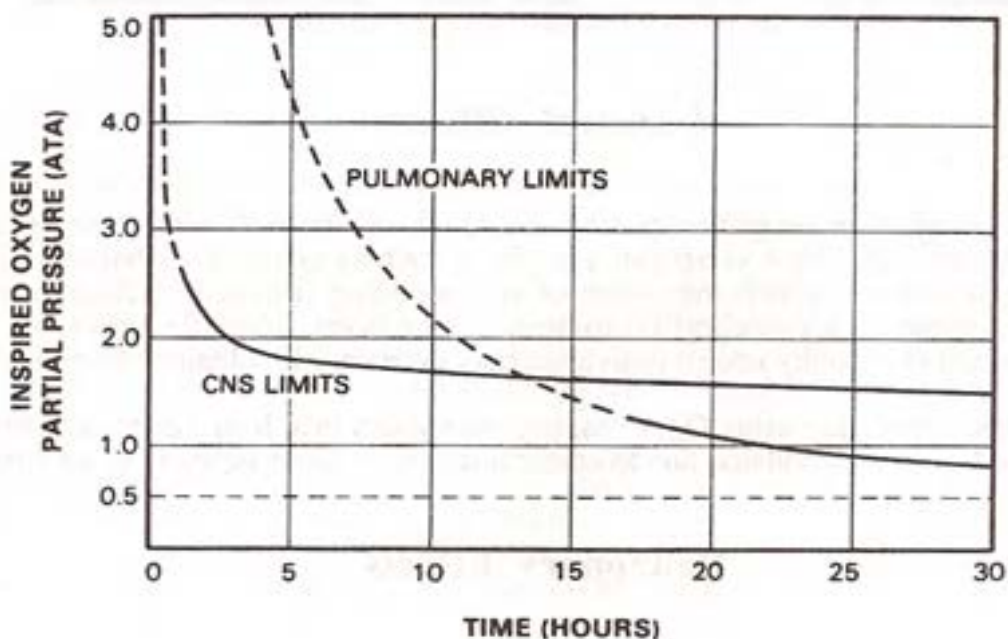


Fig. 21.1

This graph shows the predicted pulmonary and cerebral toxicity limits of exposure to varying partial pressures of oxygen. It can be noted that oxygen can be tolerated for much longer periods at lower partial pressures.

CAUSES OF O₂ TOXICITY

For **resuscitation**, **100% O₂** should be used for hypoxic diving casualties without any fear of O₂ toxicity. As mentioned above, the treatment of decompression sickness and air embolism cases includes 100% O₂, even before consultation with the diving physician regarding the long term toxic effects.

Oxygen re-breathing equipment should be restricted to military, commercial and trained technical divers use and diving with this should not be attempted by recreational divers. O₂ diving sets have an absolute depth limit of 9 metres for resting dives and 8 metres for working dives in order to reduce the risk of convulsions. Rebreathing and scuba sets employing nitrogen/O₂ (**nitrox**) mixtures are limited to depths which produce an inspired O₂ partial pressure of no more than 1.6 atmospheres.

In **deep diving** operations, gas mixtures of helium, nitrogen and O₂ should have the composition adjusted so that the inspired partial pressure of O₂ never reaches the toxic range.

Therapeutic recompression using O₂ tables often involves the compression of the diver to 2.8 atmospheres while breathing 100% O₂. There is a significant risk of both pulmonary and cerebral O₂ toxicity and these tables should only be employed on the advice of diving medical experts.

CLINICAL FEATURES

Cerebral Effects

In this case the earliest symptom may be a convulsion which can develop without any warning signs. It may sometimes be preceded by a variety of symptoms such as facial pallor, visual or auditory disturbances, tunnel vision, faintness, or facial twitching – which may warn of an impending convulsion. Nausea, retching and even vomiting are common with cerebral O₂ toxicity, as are anxiety and palpitations. There is considerable individual variation in susceptibility to cerebral O₂ toxicity and an individual may vary in his tolerance from day to day. It may be increased by anything that increases carbon dioxide levels, such as exercise, immersion, resistance from breathing equipment and nitrogen narcosis.

During therapeutic recompression using O₂ tables, any convulsion in a diver due to cerebral O₂ toxicity must be distinguished from a convulsion due to cerebral decompression sickness or air embolism. Sometimes the convulsions occur soon after the O₂ supply is removed (the “off effect”).

Pulmonary Effects

The early symptom is an irritation deep in the central part of the chest, progressing to pain and a burning sensation which is aggravated by inspiration and accompanied by coughing. As the condition develops, shortness of breath ensues and a pneumonia type illness supervenes. Although the early symptoms are reversible, progressive serious symptoms may cause permanent lung damage or even death.



Fig 21.2

TREATMENT

Cerebral Effects

Whilst undergoing therapeutic recompression, if warning signs of cerebral toxicity develop, the patient should be encouraged to hyperventilate and then be given air to breath until the symptoms abate. Modification to the O₂ treatment table may then be necessary.

If the patient convulses he should be placed on his side to protect the airway from obstruction or aspiration of stomach contents. He should be protected from injuring himself on nearby solid objects. A padded mouth piece may be gently placed between the teeth to protect the tongue. After the convulsion has ceased the patient may be unconscious for a short time. His airway should be protected and he should be managed according to the principles outlined in Chapter 42. See Case Report 24.2.

Pulmonary Effects

These effects will usually resolve spontaneously if the supplementary O₂ administration is ceased as soon as symptoms develop. If it is essential to continue O₂ therapy however, a reduction in the partial pressure of O₂ given will slow the development of toxicity. Short periods of 'air breathing' (or Heliox), 5 minutes every half hour, are often used by experienced doctors to delay oxygen toxicity during O₂ therapy.

CONCLUSIONS

1. Recreational divers should not use O₂ enhanced diving equipment. Technical divers should not expose themselves to O₂ pressures greater than 1.6 ATA or durations that could cause respiratory manifestations.
2. Resuscitation training with O₂ equipment is of great value to divers and dive boat operators. In diving accidents, the delayed risks of O₂ toxicity are outweighed by the benefits of treating the hypoxic diving casualty.
3. The use of O₂ in the first-aid treatment of decompression sickness and pulmonary barotrauma should always be undertaken whilst bearing in mind the prospect of eventual pulmonary oxygen toxicity. Breathing air for 5 minutes after 25 minutes of O₂ is one way of reducing the risk of pulmonary toxicity, but this should be discussed with the diving physician who will ultimately manage the case.
4. During therapeutic recompression using O₂, the use of short air or Heliox breaks during the treatment reduces cerebral and pulmonary O₂ toxicity.
5. There are other logistical problems with the use of oxygen, and some of these are discussed in Chapter 40.

Chapter 22

CARBON DIOXIDE PROBLEMS

Carbon dioxide (CO₂) is the gaseous by-product produced when the body consumes oxygen to fuel its metabolic processes. The body has an efficient way of disposing of CO₂, mainly through buffering systems in the blood and exhalation from the lungs.

CARBON DIOXIDE INSUFFICIENCY (HYPOCAPNEA OR HYPOCAPNIA)

Hypocapnea refers to a blood carbon dioxide (CO₂) level below normal. The CO₂ partial pressure in the blood is normally maintained within narrow limits by a biological feedback mechanism. Voluntary or involuntary hyperventilation (overbreathing) will overcome this regulatory mechanism and lower the blood CO₂ level. The most common cause for this is the rapid sighing respiration associated with hysterical and anxiety states – the feeling one experiences on confronting a great white shark eye to eye.

A number of divers (fewer each year, due to natural selection) deliberately hyperventilate to lower their blood CO₂ level, before a breath-hold dive, in order to prolong the dive. They often succeed beyond their wildest dreams. The lethal consequences of this practice are explained in Chapter 4.

Clinical Features

A person hyperventilating from anxiety is not usually aware of an altered breathing pattern, although it may be evident to an observer. Hyperventilation causes increased resistance to breathing with scuba, and this causes more anxiety.

Symptoms include tingling or "pins and needles" (paraesthesiae) of the fingers, dizziness and light headedness, an altered conscious state or confusion. Muscular twitching or spasms can occur in extreme cases.

Treatment

The simplest treatment for hypocapnea is to reduce the breathing rate and depth. This restores the blood CO₂ level and cures the symptoms. On land, doctors often advise the patient to breathe in and out of a brown paper bag (rebreathing), but underwater most divers are not prepared to replace their regulator with a soggy paper bag.

Alternative Diagnoses

It is important to exclude other serious conditions such as decompression sickness, air embolism, carbon monoxide poisoning and salt water aspiration, whenever a diver presents with these symptoms. These illnesses can in themselves, also cause apparent hyperventilation and can mimic anxiety states.

CARBON DIOXIDE TOXICITY (HYPERCAPNEA)

CO₂ toxicity is due to accumulation of CO₂ through excess production or inadequate ventilation (breathing).

The excess production is usually due to metabolism from increased exertion. Whereas only 0.5 litres/minute of CO₂ is produced at rest, this can rise to over 3 litres during maximal exercise.

Inadequate ventilation can be caused by breath-holding, breath control ("skip breathing"), rebreathers or extension of the respiratory passages ("dead space") with snorkels, etc.

The effect of depth on inspired partial pressure is important. While 3% inspired CO₂ may be tolerated at atmospheric pressure without significant symptoms, the same percentage at 20 metres (3 ATA) is the equivalent of 3 × 3 or 9% at the surface – a level which will cause serious toxicity.

Re-breathing exhaled CO₂ is the most common cause of CO₂ toxicity in divers. Hence, CO₂ toxicity is most commonly encountered with rebreathing equipment, but it can sometimes occur in diving helmets, compression chambers, saturation complexes (habitats) or possibly even scuba.

Causes of CO₂ Toxicity

❑ Rebreathing equipment.

Some types of military and technical diving equipment conserve gas and reduce bubble formation by allowing the diver to rebreathe his exhaled gas. A canister of CO₂ absorbent (soda lime) is included in the circuit to remove the CO₂ which the diver exhales (see Chapters 5 & 43).

This mechanism can fail due to exhaustion of absorbent material, salt water contamination, improper packing, excessive CO₂ production due to exertion, or improper assembly of the equipment.

❑ Diving helmet problems.

With a standard-dress helmet or with some helmets used in deep diving, the diver can partly rebreathe his exhaled gas if the fresh gas flow in the helmet is insufficient to flush out exhaled CO₂.

❑ Chambers and habitats.

CO₂ which is exhaled by chamber occupants must be removed by constant flushing of the chamber with fresh air or by the recirculation of the chamber gas through a CO₂ absorbent (scrubber). If either of these mechanisms is inadequate, the occupants can develop CO₂ toxicity by rebreathing their own exhaled CO₂.

❑ Scuba.

Since rebreathing is not possible with scuba equipment, CO₂ toxicity is not generally a problem for scuba divers unless there is resistance to breathing (regulator resistance, increased gas density at depth) or a reduced respiratory response of the diver to CO₂ (possibly due to voluntary control or “skip breathing”, adaptation, nitrogen narcosis, or high oxygen levels).

Clinical Features

These depend on the rate of onset and the actual partial pressure of the inspired CO₂.

A rapid accumulation of CO₂ may cause unconsciousness before any symptoms are experienced.

A slower build-up causes a variety of symptoms, including :

- **shortness of breath**, or air hunger.
- **flushing of the face** and **sweating** (sweating is not easy to detect underwater).
- **repetitive activity**, such as swimming, without awareness of this.
- **light headedness**, muscular **twitching**, jerks, tremors or **convulsions**.
- **impaired vision**.
- **unconsciousness**.
- **a splitting or throbbing headache**, usually at the front of the head. This may be severe and start after the CO₂ levels have been corrected. It often lasts for hours.
- **death**.

CO₂ toxicity may increase the likelihood of decompression sickness, oxygen toxicity, nitrogen narcosis and resistance to breathing (because of increased respiration). As with oxygen toxicity, there is sometimes an “off effect” whereby the symptoms of CO₂ toxicity are temporarily worsened when a diver suddenly resumes breathing normal gases after partially adapting to a high CO₂ pressure.

Treatment

Any diver, diving with rebreathing equipment, who experiences symptoms of CO₂ toxicity should immediately **cease exertion, inform his buddy, flush** the rebreathing system with fresh gas, then return to the surface by a **buoyant ascent** and **breathe air**.

Attendants of a surfaced diver suffering from CO₂ toxicity should isolate him from the source of CO₂ rebreathing, give **100% oxygen** by mask, and administer cardiopulmonary **resuscitation** if appropriate.

Other causes of headache and breathing difficulties such as pulmonary barotrauma, decompression sickness, carbon monoxide toxicity etc. should also be excluded (see Chapter 32).

The severe headache which follows CO₂ toxicity should be treated with a simple analgesic such as paracetamol (acetaminophen).

Chapter 23

CARBON MONOXIDE TOXICITY

Carbon monoxide (CO) is a gas produced by the incomplete combustion of carbon containing compounds. It is a component of the smoke from **engine exhausts**, slow combustion **stoves** and **cigarette** smoke. It is also produced in divers' air compressors (see Chapter 24).

CO breathed in anything more than trace amounts can be lethal. It binds avidly to the oxygen (O₂) binding sites of haemoglobin (Hb) in the blood, preventing the haemoglobin from carrying O₂. CO bound to haemoglobin forms carboxyhaemoglobin (HbCO). If a sufficient number of the O₂ binding sites are occupied by CO, death from hypoxia ensues (see Chapter 20).

CO also binds with components of the energy-producing biochemical pathways in the cells, interfering with fundamental cellular function.

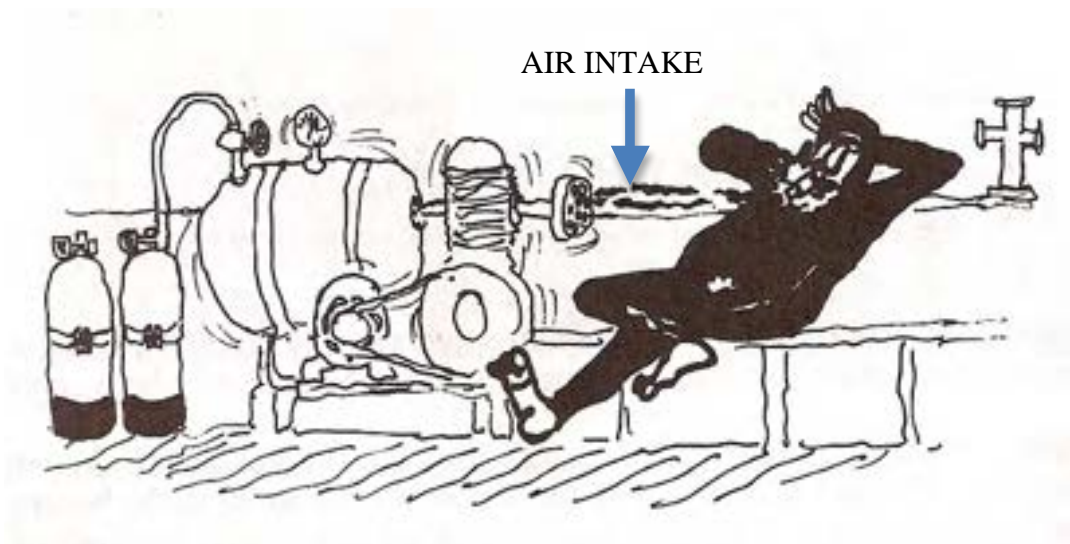


Fig. 23.1

CLINICAL FEATURES

Symptoms are those of progressive hypoxia due to the reduction in the oxygen transport by the blood. They vary with the **carboxyhaemoglobin** content of the blood as shown in the following table:

Concentration of CO in Breathing Gas	% Carboxy-haemoglobin	Effects on a Diver
400 parts per million (ppm)	7.2%	Nil or slight
800 ppm	14.4%	headaches dizziness, nausea breathlessness with exertion
1600 ppm	29.0%	confusion, vomiting, collapse.
3200 ppm	58.0%	paralysis, or loss of consciousness
4000 ppm	72.0%	coma
4500 ppm	87.0%	death

Table 23.1

The effects of CO are cumulative and are related to the concentration breathed and the duration of exposure. A concentration of 400 ppm may produce symptoms in an hour while 1200 ppm will need only 20 minutes. As the carboxyhaemoglobin (HbCO) level falls, following removal of the CO contamination, the clinical state may lag due to CO bound in the tissue, or from enzyme or protein damage. The classical "cherry pink" colour is only seen in the acute and early cases, before respiratory failure develops. Despite the above table, HbCO levels may not correlate directly with mortality or morbidity.

The effects of CO poisoning are greatly increased by increased pressure at **depth**, if the oxygen pressure is kept consistent. A 400 ppm contamination which would not produce clinical effects at atmospheric pressure will be equivalent to 4×400 ppm (or 1600 ppm) at 30 metres depth (4 ATA), a concentration sufficient to cause serious toxicity. Because the oxygen partial pressure reduces with ascent, the symptoms of mild CO poisoning may only become serious during or after ascent.

Serious **brain damage** is a frequent complication of significant CO toxicity due to prolonged hypoxia of the brain. (See Case History 24.1)

Because the contaminated compressed air may be given to other divers who employ the same supplier, it is possible that they may also be affected. This may have implications not only on the differential diagnosis of the victim, but for prevention of other casualties.

TREATMENT

The diver should be rapidly **isolated** from the contaminated gas and have **100% O₂** administered by mask. The ABC resuscitation principles (see Chapter 42) should be applied where appropriate.

Hyperbaric O₂ (HBO) is the treatment of choice. The high partial pressure of O₂, which occurs in a hyperbaric chamber, will dissolve enough O₂ in the blood plasma to meet the bodies needs without participation of the haemoglobin system. Oxygen is breathed at a partial pressure of 2 ATA or more to sustain life while the CO slowly detaches from the haemoglobin and is breathed out through the lungs, allowing the haemoglobin to resume its normal O₂ transport role.

If hyperbaric O₂ is to be of value it should be instituted as soon as possible, preferably within 6 hours of poisoning. Delay in treatment may result in irreversible and progressive brain damage.

PREVENTION

The major danger to any diver is from carbon monoxide contamination of the compressed air supply. Sources of contamination include:

Direct contamination by CO from gasoline engine exhausts.

This may come either from the compressor motor itself, or from other nearby motors or gas exhaust outlets. The classic case occurs where the compressor air inlet hose is located downwind from the compressor motor exhaust.

Contamination produced by the breakdown of unsuitable lubricants.

The incorrect use of hydrocarbon-based lubricants used to lubricate an air compressor is a common cause, however it may also result from overheating of the compressor. Both carbon and nitrogen oxides can be formed.

The intake of polluted atmospheric air to fill air cylinders.

It is important for suppliers of compressed air to regularly check the quality of the air being compressed, to ensure that this and other pollutants are not included in divers' air supplies. Adequate filtration systems are necessary on all compressors, and these should always be properly maintained.

Chapter 24

BREATHING GAS CONTAMINATION

The supply of uncontaminated breathing gas (air) is of vital importance to the diver because of the magnifying effect on contamination by the partial pressure rise with increasing depth. For example, 5% contamination of gas at atmospheric pressure is equivalent to 20% at 30 metres depth (4 ATA).

Contamination usually arises either from impurities in the air taken into the compressor or from contaminants generated by the compressor itself.

PREPARATION OF COMPRESSED AIR

Atmospheric air is taken into the compressor and is compressed by one of two methods. Most dive shops use a piston and cylinder compressor which raises the pressure of the gas in several stages. A more advanced compressor uses a diaphragm pump similar in principle to that in a refrigerator.

Ideally the compressed air should be treated by passing it through several purifying cartridges (or filters) to remove contaminants. Silica gel is used to remove **water** vapour, activated charcoal removes **oil** and **hydrocarbons**, a molecular sieve removes **water droplets and dust particles** and a catalyst converts **carbon monoxide** to carbon dioxide, which can be absorbed. Less scrupulous air suppliers have been known to substitute women's sanitary pads, instead of filters.

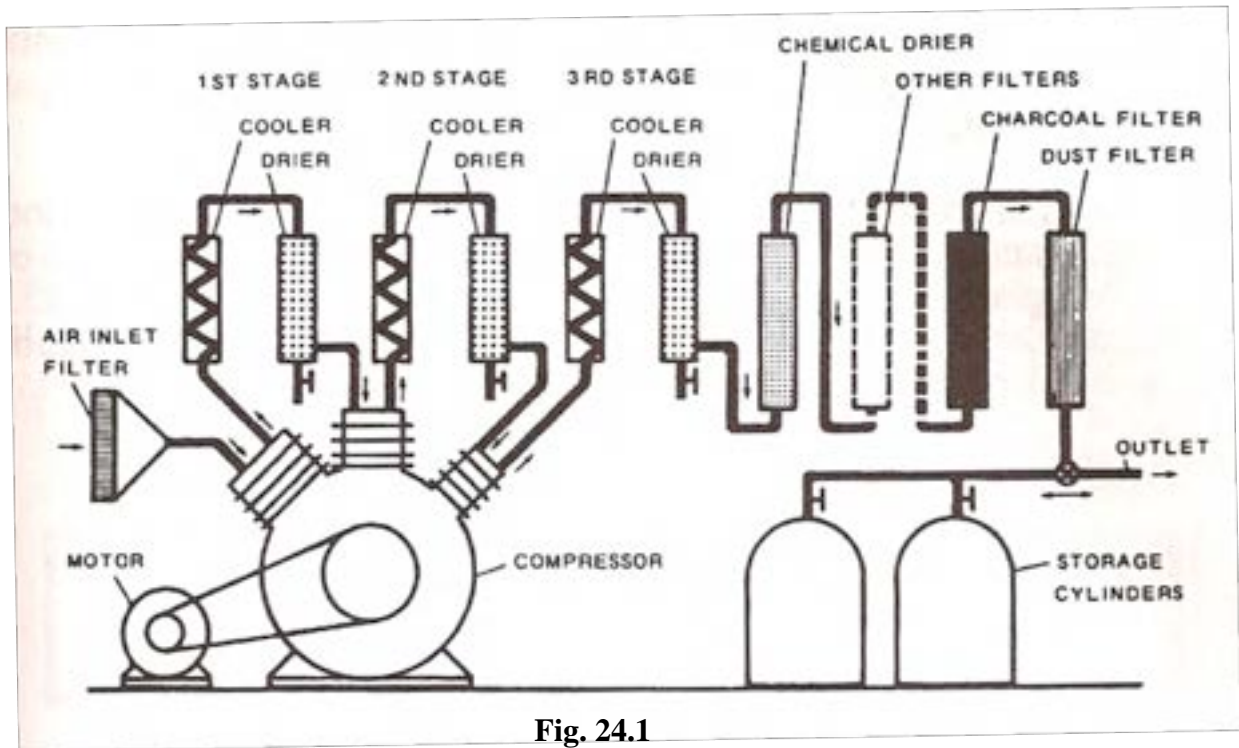


Fig. 24.1

Schematic diagram of a compressor system with filters.

GAS PURITY STANDARDS

Authorities such as the US Navy, NOAA and Standards Australia specify minimum standards of purity for breathing gas.

Case History 24.1 In an area subject to tidal currents an experienced diver planned to dive at slack water. He anchored his boat almost at low tide. The hookah compressor he used was correctly arranged with the air inlet upwind of the exhaust and the dive commenced. After 90 minutes at 10 metres the diver felt dizzy and lost consciousness but was fortunately pulled aboard by his attendant and resuscitated.

Diagnosis — Carbon monoxide poisoning.

Explanation — as the tide turned, so did the boat. This put the compressor **air inlet downwind of the motor exhaust** and carbon monoxide from the exhaust was breathed under pressure by the diver.

Compressors can also generate some lethal contaminants internally. The compressor piston requires lubrication and this is usually achieved by the use of special **oils**. In some circumstances, such as where there is excessive wear of the compressor, high temperatures can be generated and this may decompose the lubricating oil into toxic products such as **oxides of nitrogen** or **carbon monoxide**, which are then pumped into the diver's air tank. Poor maintenance of the compressor can also lead to an **oil** and **hydrocarbon** mist escaping into the air supply.

If the compressor is operated in an unclean environment, **dust** (and chemical vapours) can find its way into the diver's air causing abnormal wear on the moving parts of both the compressor and the regulator.



Fig. 24.2 A modern air compressor

Water vapour must be removed from the air delivered from the compressor or it can condense in the scuba cylinder causing rust, or allow the regulator to freeze up during diving in cold conditions.

Most compressors have a **filtration system** both on the inlet side to prevent the intake of dust, and on the outlet side to filter out oil and water vapour. Their efficiency depends on regular maintenance and the absence of over-loading.

Occasionally contamination comes from the destruction of the filters and lubrication systems. Non-hydrocarbon based lubricants with high "flash points" are preferable. The problems of oil lubrication can be overcome by using a compressor which is lubricated with water or dry Teflon materials. Unfortunately the expense of these is beyond the reach of many air suppliers. Diaphragm pumps also avoid the problem of oil lubrication but are also very expensive.

CLINICAL FEATURES

Contaminated air may have an unusual taste or smell, or alternately, it may appear quite normal.

As many of the divers may obtain their air supply from the same source, it is not uncommon for similar symptoms to be spread amongst the diving group. As only the most serious case may present, the other divers should be interrogated for similar but less severe symptoms.

- **Oxides of nitrogen** cause lung damage, which is likely to cause coughing, wheezing, shortness of breath and/or tightness in the chest.
- **Carbon monoxide** causes headache and unconsciousness – a detailed description can be found in Chapter 23.
- **Oil** can cause chest pain, shortness of breath, coughing and pneumonia.
- **Trichloroethylene and other aliphatic halogens** can cause respiratory and gastrointestinal symptoms.

TREATMENT

If a diver is affected by breathing contaminated air he should be separated from the source, managed according to first-aid principles outlined in Chapter 42, and gas from his scuba cylinder should be sent for analysis to a chemical or gas testing laboratory.

PREVENTION

The diver should breathe from his equipment before entering the water and should not use air which has an unusual taste or smell.

As the expertise of compressed air (and other breathing gases) suppliers vary, divers are well advised to obtain air fills only from a reputable supplier. Regular checks by local authorities on the quality of the air are advisable, and in many places are now mandatory. It can be tested by chemical detector tubes that determine the level of each specific contaminant. Drager (a gas and medical equipment company) supply these tubes in a Drager Gas Detection Kit. Many others are available.

Following any diving accident, suspect air can be tested by commercial gas suppliers and State Health authorities.

Case History 24.2

A diving club had for many years been filling their cylinders from an air bank made up of large cylinders, the source of which had been lost in the mists of time. It was decided to return the bank of cylinders to a major industrial gas supplier for testing. The cylinders had their original paint in good condition – black cylinders with a white collar. The gas company tested the cylinders, found them to be sound and refilled them according to the colour code on the cylinders. Unfortunately, this was the standard colour code for pure oxygen and that is what the company filled them with, having no idea that they would be ultimately used to fill scuba tanks.

A member of the dive club took delivery of the cylinders and reinstalled them in the bank. He did not know the significance of the colour coding and assumed that because he was using the cylinders to store air that the company would refill them accordingly. Because they were already full there was no need to fill them from the compressor and the bank was immediately used to fill several sets for a dive the following day. Two divers used tanks from this source on a dive to 20 metres. One abruptly convulsed 10 minutes into the dive and was fortunately rescued by his buddy before he too convulsed.

Some clever detective work performed by the rescuing diver, and the diving physician they consulted, established the cause of the problem as oxygen toxicity. Swift action by the police to round up all the contaminated scuba tanks before they could be used, averted a major disaster. In this case, breathing from the cylinder at the surface before the dive would not have disclosed any detectable difference from air.

Chapter 25

DROWNING SYNDROMES

It is vital for divers to understand the management of near-drowning because it is the final outcome of a large number of diving accidents.

GENERAL

Drowning is defined as the death of an air breathing animal due to immersion in fluid. When patients lose consciousness due to immersion and aspiration, but subsequently recover, the term 'near drowning' is used. When symptoms are not severe enough to classify as near drowned, another term the 'aspiration syndrome', is employed. There is a continuous progression between aspiration, near drowning and drowning cases.

Aspiration syndromes merge with near drownings - often the intensity of the symptoms and the degree of consciousness depending on various circumstances, the activity of the victim and the administration of oxygen.

"Near drowning" cases sometimes die hours or days later, thereby being re-classified as secondary or 'delayed drowning'.

Some of the apparently "drowned" victims, because of enthusiastic CPR and capable intensive care, surprisingly recover without serious sequelae.

The incidence of death by drowning appears to be diminishing in the more civilised countries, from 7 per 100,000 in 1970s to around 2 per 100,000 now. Drowning is second only to motor vehicle trauma as a cause of accidental death in Australia and the USA, and is the major cause in some age groups (since the introduction of seat restraints in motor vehicles).

There is an over-representation of young males in most drowning series and there is a predictable age distribution for specific types of drowning. Most swimming pool deaths

occur in the very young, surf deaths mostly in teenagers and young adults, ocean deaths of sailors and fishermen throughout the whole adult range, and bathtub drownings are in either babies, the infirm or homicides.

When a fully conscious human accidentally falls in the water, he usually fights to survive, involving a panic reaction with violent struggling followed by automatic swimming movements. There may be a period of breath holding and swallowing of large gulps of water. Vomiting may occur, followed by gasping and aspiration of water and stomach contents. Blood stained froth develops in the airways and may be coughed up. Finally the patient convulses and then dies from cerebral hypoxia.

Drowning was traditionally associated with a "fight for survival" response but in other circumstances drowning may proceed in a quiet and apparently unemotional manner. In these cases loss of consciousness occurs without any obvious warning, and the underwater swimmer/diver then aspirates and drowns quietly. Examples of *quiet drownings* include:

1. Hyperventilation and hypoxia of ascent in breath-hold diving. This is a common cause of drowning in otherwise fit individuals who are good swimmers. See Chapter 4.
2. Hypothermia and/or cardiac arrhythmia cases.
3. Drugs and alcohol effects. These increase the incidence of drowning by impairing judgment and reducing the struggle to survive. It is likely that nitrogen narcosis may have a similar effect in divers.
4. Diving equipment problems may produce hypoxia. These include the dilution and ascent hypoxias with rebreathers and carbon monoxide toxicity, interfering with oxygen metabolism. They are all likely to cause loss of consciousness without excess CO₂ accumulation, dyspnoea or distress. See Chapters 6, 20 & 43.
5. The salt water aspiration syndrome of divers. See Chapter 26.
6. Other causes of unconsciousness in divers, leading to drowning, have been described in Chapter 33, e.g. cerebral arterial gas embolism, some marine animal envenomations, coincidental medical illnesses such as epilepsy, cerebral haemorrhage etc.

Fresh or salt water entering the alveoli (air sacs of the lung) appears to wash out or damage the surfactant lining them, causing alveoli to collapse and become unavailable for gas exchange. Damage to the walls of the alveoli also causes the capillaries to leak blood and protein into the lungs. This interacts with air and water producing a foam which the victim may cough up in copious amounts. This is called pulmonary oedema.

The **sequence of events** in a near-drowning diving incident often goes as follows:

The degree of **panic** behaviour is variable, and may be reduced by such factors as personality, training, drug intake and nitrogen narcosis. If some air is still available from the regulator, the diver may persist with attempting to breathe from this (even at the cost of aspirating some water), and request assistance. Even if an alternative air supply is made available, **hypoxia** may still develop because of the water aspirated. Coughing and gasping may be voluntarily suppressed until the diver

reaches the surface. If the diver is totally deprived of his air supply for some reason, he initially breath-holds until the "break point" is reached and then takes an involuntary breath.

The resulting inhalation of a bolus of water usually provokes **coughing** and **closure of the larynx** producing involuntary breath-holding followed by unconsciousness. It is unusual for large amounts of water to 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 sea water, which makes the diver susceptible to vomiting and aspiration.

CLINICAL FEATURES

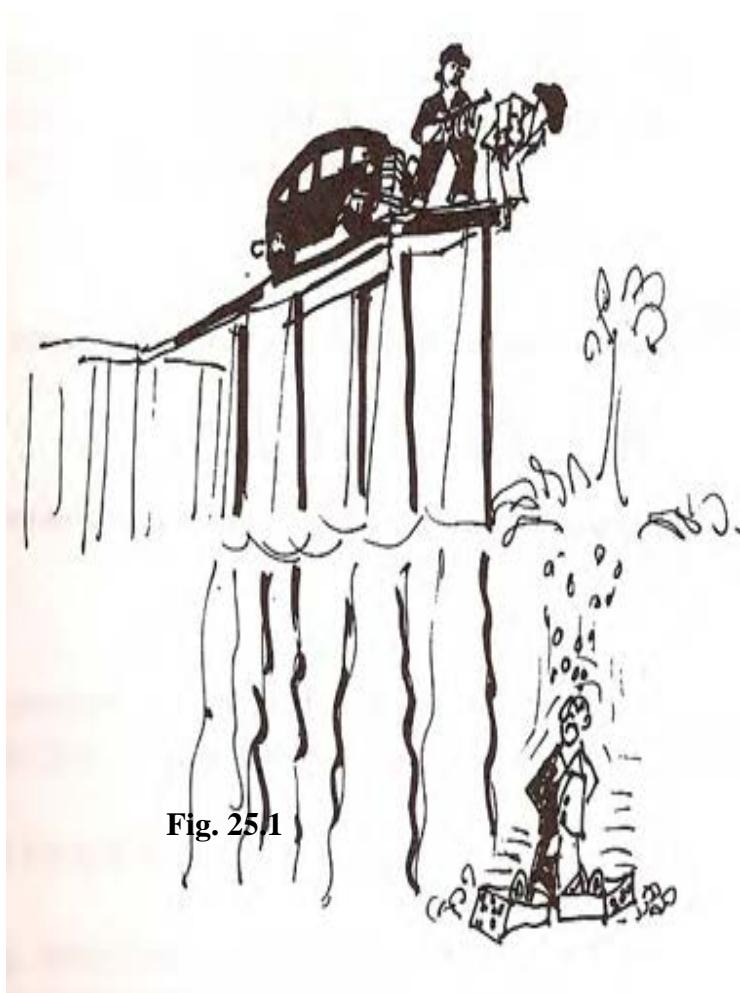


Fig. 25/1

Fig. 25.1

The commonest ultimate cause of death in recreational scuba divers is drowning.

For example, any loss of consciousness or capability when engaging in terrestrial activities is unlikely to cause death. It would do so more frequently if the victim was diving under water.

When first rescued the condition of the near drowned victim may vary from fully conscious to unconscious, with normal, laboured or absent respiration. The initial effects are on the respiratory system, but later damage is due to hypoxia on the brain.

If the victim is breathing, the stiff lungs cause laboured respiration and it is common for foam, often copious and blood stained, to be coughed up or to exude from the nose or mouth. Vomiting is also common, as is aspiration of stomach contents, either spontaneously or during resuscitation attempts. Cyanosis (bluish coloration of lips, tongue, ears) from hypoxia is frequent.

TREATMENT



Fig. 25.2

Rescue and recovery from the water is the top priority and much time will be spent in the diving course in this training. Success is reliant on buddy behaviour, acquiring positive buoyancy during ascent and on the surface (including ditching of weights) and attracting attention.

Treatment at the scene of an accident will often determine whether the victim lives or dies. The standard of first aid and resuscitation training of the rescuers therefore influences outcome.

The temperature of the water and thus the degree of hypothermia may also be a factor. Poorer results are achieved in warm bath water drownings. Other factors which influence outcomes include: the

presence of chlorine and other chemicals and foreign bodies, the aspiration of stomach contents, the subsequent development of pneumonitis, respiratory infection and lung damage, haemolysis, renal failure and coagulopathies. These complications are for the intensive care physicians to cope with.

In exceptional circumstance, near drowned victims have fully recovered after periods of total immersion of over 15-45 minutes (especially so in children in cold waters), so it is worth attempting resuscitation even in apparently hopeless cases.

The resuscitation principles of airway management, restoration of respiration and circulation (A-B-C as outlined in Chapter 42) should be employed immediately. **Oxygen** in the highest concentration

available should be given by mask to offset hypoxia. Masseter spasm (“jaw clenching”) is a common feature of hypoxia, and may obstruct artificial respiration.

Near drowned cases are liable to deteriorate many hours after making an apparent recovery, so all near drowned victims should be taken to **hospital** and must remain there for at least 24 hours under observation.

PREVENTION

It is paradoxical that drowning, which causes more than 80 times the number of deaths in recreational divers than either decompression sickness or contaminated air, does not rate more than a paragraph or two in some diving medical texts.

A normally functioning diver, with adequate equipment in a congenial ocean environment, is protected from drowning as he carries his own personal life support system with him - his scuba. Drowning only occurs when there is;

- diver fault (pathology, psychology or technique),
- failure of the equipment to supply air, or
- hazardous environmental influences.

A survey was conducted of 100 recreational scuba deaths from drowning and compared these with near drownings, and it demonstrated that simple measures were available to avoid the fatal drowning cases. They were:

1. Diver fitness.

Ensure both medical and physical fitness, so that there is no increased likelihood of physical impairment or loss of consciousness, or difficulty in handling unexpected environmental stresses.

2. Experience.

Ensure adequate experience of the likely dive conditions (become trained and dive under the supervision of a more experience diver, when extending your dive profile).

3. Equipment.

Absence of appropriate equipment is a danger, but not as much as equipment failure and misuse. The latter includes the practice of overweighting the diver, and his over reliance upon the buoyancy compensator.

4. Environment.

Hazardous diving conditions should be avoided, using extreme caution with tidal currents, rough water, poor visibility, enclosed areas and excessive depths.

5. Neutral buoyancy (during the dive).

Ensure neutral buoyancy whilst diving. This implies not being overweighted and not being dependent on the buoyancy compensator.

6. Air supply.

An inadequate supply of air for unexpected demands and emergencies may convert a problematic situation into a fatal one. It also forces the diver to experience surface situations that are worrying and conducive to anxiety, fatigue and salt water aspiration. Equipment failure is not as common a cause of LOA/OOA as failure to monitor the contents gauge and/or a decision to breathe the tank down to near-reserve pressure.

7. Buddy diving.

Use traditional buddy diving practice - 2 divers swimming together. Solo diving, for the whole or part of the dive, is much more likely to result in an unsatisfactory outcome in the event of diving problems. It is the divers who are committed to the traditional buddy diving practices who are likely to survive the more serious drowning syndromes.

8. Positive buoyancy (post incident)

Positive buoyancy is frequently required if problems develop.

Failure to remove the weight belt during a diving incident continues to be a major omission, and must reflect on training standards. In most situations, unbuckling and then ditching (if necessary) the weight belt is the most reliable course of action once a problem becomes evident.

Buoyancy compensators cause problems in some emergency situations, and not infrequently will fail to provide the buoyancy required. They are of great value in many cases - but are not to be relied on.

9. Buddy communication.

If feasible, inform the buddy prior to ascent. If correct buddy diving practice is being observed, the buddy will automatically accompany the injured or vulnerable diver back to boat or shore.

10. Rescue

Employ the rescue, water retrievals, first aid facilities (including oxygen) and medevac systems which were planned before the dive. See Chapters 5 & 39.

These factors differentiate a drowning fatality from a successful rescue.

Chapter 26

SALT WATER ASPIRATION SYNDROME

This condition was first described in Royal Australian Navy divers in the late 1960's. Some divers were repeatedly presenting for treatment with a brief condition characterised by shortness of breath, sometimes a pale or bluish (cyanosis) skin colour, mild fever accompanied by shivering, malaise, anorexia, and generalised aches and pains. Chest X-rays sometimes showed an appearance similar to a patchy pneumonia and blood gases consistently verified hypoxia.

Close questioning of the divers revealed that nearly all the cases had aspirated a fine mist of seawater from a leaking or flooded demand valve. "Volunteer" experiments confirmed the association between aspiration of sea water and the development of the syndrome.

CLINICAL FEATURES

There is often a delay of half an hour or more between aspiration of the water and the major symptoms. The onset in mild cases is often provoked by exercise, movement or cold exposure. Others may develop after movement, such as arising from bed the next morning.

The diver has some or all the following symptoms:

- initial coughing, sometimes with expectoration, after surfacing
- fever with shivering (induced by cold exposure),
- malaise with anorexia, nausea or vomiting,
- shortness of breath, coughing, cyanosis
- headache and generalised aches and pains.

TREATMENT

The condition is self limiting and resolves without treatment within 2-24 hours. **Rest** and the administration of **100% oxygen** by mask for several hours until the symptoms have abated, is of considerable value. The oxygen not only relieves the hypoxia but produces dramatic resolution of the symptoms of this syndrome.

Because of the nature of the symptoms, it is necessary to distinguish the salt water aspiration syndrome from other serious conditions such as decompression sickness (chokes), pulmonary barotrauma (burst lung), severe infection and pneumonia – which can all present with some or all of the features of this condition.

PREVENTION

The condition can be prevented by avoiding situations which will result in the aspiration of seawater. Buddy breathing from a single regulator can be a fruitful source of the syndrome if the shared demand valve is not adequately cleared of water. Others include a towed search, poor regulator performance, exhaustion of air supply and free ascent practice. Proper maintenance of the demand regulator and its exhaust valves, is important.

Other cases develop on the surface, after divers remove the demand valve to talk – as they are wont to do. Similar symptoms are observed in surfers and victims rescued from the sea (especially by helicopters, which produce a strong down draft causing a sea water spray).

Some divers are especially vulnerable based on hyperactive airways, with a history of hay fever or asthma. In respiratory laboratories, aerosol inhalations of hypertonic saline (sea water) are used to provoke these breathing difficulties and demonstrate susceptibility to the syndrome.

Chapter 27

COLD & HYPOTHERMIA

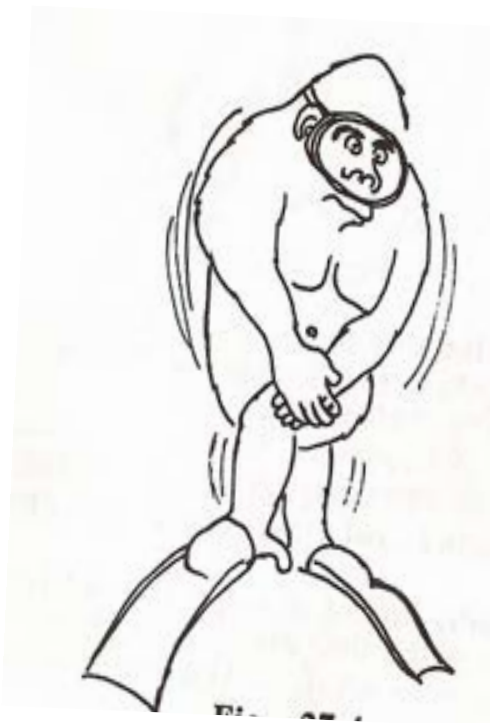


Fig 27.1

Recognition of the early clinical features of hypothermia may convince a diver to leave the water before a serious problem arises.

A diver is usually immersed in water which is considerably colder than the normal body temperature of 37°C. Unfortunately, water is particularly efficient at removing body heat, having a conduction capacity 25 times that of air and a specific heat (the amount of heat necessary to raise a given volume by a certain temperature) 1000 times that of air.

Without insulation, a diver will lose body heat much faster in water than in air at the same temperature. This can cause **hypothermia**, a harmful drop in body temperature to below 35°C.

The body can reduce temperature loss by generating heat through metabolism, exercise and shivering, and by restricting blood flow to the skin. The rate of heat loss also depends on factors such as the temperature of the water, the thickness of body fat, presence a wetsuit or other insulation, and the posture of the diver.

CLINICAL FEATURES

All divers will have experienced the early features of cold — numbness, blueness or pallor of the skin (especially in peripheral areas such as the fingers, toes and earlobes), and shivering.

If the body temperature falls by about 2°C, loss of co-ordination and uncontrollable shivering may impair the ability to swim and render the performance of finely coordinated movements (like manipulating equipment and assisting buddies) impossible.

After a body temperature drop of 3–4°C, the diver may become weak, apathetic, confused and helpless. Drowning is a real possibility at this stage. A body temperature less than about 30°C results in unconsciousness. This may be confused with other causes of unconsciousness in divers. Often the diver appears to just lose consciousness without other obvious clinical manifestations.

A victim who is unconscious from severe hypothermia may have a very slow respiratory rate, and a barely detectable pulse, and may appear dead to the inexperienced observer. It is important to not assume the worst in this situation. Do not presume that he is dead, unless he is warm and dead.

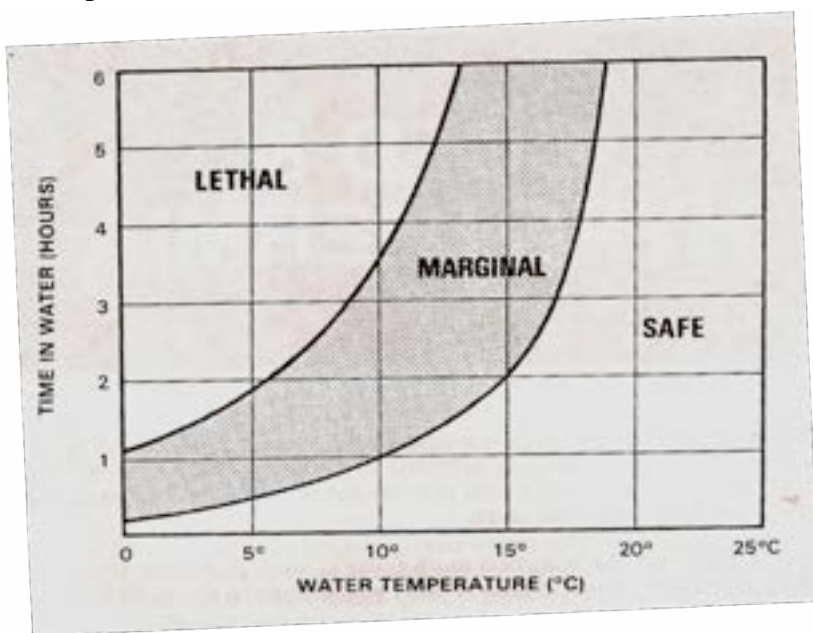


Fig. 27.2

This graph gives an indication of approximate survival times of an uninsulated human in water of various temperatures. These figures are overestimates – a diver would be severely incapacitated well before he reached the limits of survival. It is obvious that survival times of less than one hour can be expected without insulation in water temperatures found in many countries.

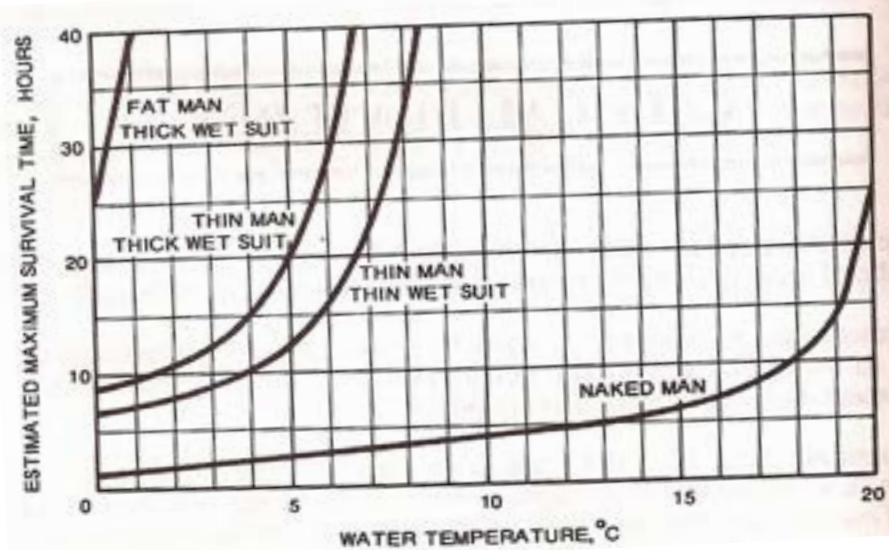


Fig. 27.3
Graph illustrating survival times in varying water temperatures for divers

FIRST AID TREATMENT



The diver must be handled gently. Both active and passive movements are to be avoided, as these tend to trigger serious or lethal cardiac arrhythmias. While the patient is hypothermic, ensure that he remains horizontal, as the vertical position can result in death. Always clear the airway, check for any evidence of heartbeat or respiration, and begin resuscitation if necessary. If medical or hospital facilities are available, many other treatments are more effective than the warm water immersion regime described below.

If required, the basic first-aid principles of management of airway, breathing and circulation, (**A-B-C**: see Chapter 42), take precedence. It is recommended that expired air resuscitation (**EAR**) and external cardiac compression (**ECC**) be performed at half the normal rate in cases of hypothermia because body metabolism is slowed. However, unless the rescuer is confident that hypothermia is the sole cause of the victim's collapse, the usual resuscitation techniques and rates are probably indicated.

Fig. 27.4

The aim of management is to keep the victim alive, while returning the body temperature to normal. This is most simply achieved by **immersing the victim in a warm bath at a temperature of 37–38°C**. A warm shower is a less efficient alternative and certainly not with the patient standing. A pleasantly warm bath or shower is approximately the right temperature.

Some workers recommend that only the torso (trunk) should be immersed in a bath initially, and the limbs kept out of the water, warming the torso first while allowing the limbs to stay cool. The reason for this is that the limbs will have restricted blood flow in response to the hypothermia, and this will be reversed by warming. Increased blood flow to the limbs at this critical stage may lower the blood pressure as well as divert heat from the vital organs to the less important periphery. In an emergency, this ideal may not be necessary or practical and it is reasonable to immerse the whole body.

Facilities to warm a diver are usually limited at a dive site and improvisation may be required. **Wrapping the victim in blankets with other divers** is an efficient and often acceptable way of transferring body heat to a mildly hypothermic diver. Warm diver buddies, especially of the opposite sex, may be sought by some unscrupulous divers.

A reflective **survival blanket** over normal blankets improves their efficiency. Blankets used alone, without a heat source, are less effective as they do not generate heat and the victim's heat output is very slow. Wrapping in a **plastic (garbage) bag**, or even **newspapers**, may also help with insulation by reducing air flow over clothes and skin.

The **engine room of larger vessels** is often warm enough to be of value in the management of hypothermia and engine cooling water may be a source of warm water in an emergency. Treatment can be suspended when the patient reaches 37°C, or starts to sweat.

Although **alcohol** produces a warm inner glow it actually worsens hypothermia by increasing blood flow to the skin, accelerating heat loss. It should not be given to severely hypothermic patients. Stimulating drinks such as tea and coffee should also be avoided. Warm water, glucose or electrolyte drinks may be given to fully conscious patients.

PREVENTION

Even in tropical waters, loss of body temperature during a dive is likely if the diver is not insulated.

The most popular and convenient insulator is the **wet suit** (see Chapter 5). Air bubbles enclosed in synthetic rubber provide an insulating barrier between the diver and the water without the need for the suit to be waterproof – hence the term "wet suit". They are available in various thicknesses depending on the expected water temperature. Wet suits have the disadvantage of compression of the air cells at depth which reduces their insulation and causes inconvenient changes in buoyancy.

This problem is overcome in deep professional diving operations by the use of a "**dry suit**" which uses air as the insulating material. Other variations include electrical, chemical or hot water warming procedures, or even an inflatable air pocket enclosed in a wetsuit.

When immersed and in a survival situation, heat loss in an uninsulated person can be minimised by floating in a curled-up posture ("foetal" position) with the knees near the chest and the arms by the side, so covering the body areas which lose heat the most (axilla and groin). This can obviously be done only if the diver has a flotation aid. Huddling together with other survivors may be of value. Restriction of movement will also minimise heat loss.

To reduce heat loss, it is best not to swim more than a short distance, as although swimming generates some metabolic heat, this is more than offset by heat lost into the water during movement.

OTHER REACTIONS TO COLD

There are a variety of other problems which can eventuate from cold exposure. These include the following:

❑ Reflex Responses.

Exposure to cold can produce a number of adverse effects on the function of the heart and lungs. These reactions can kill a swimmer/diver on entering the water and can cause a problem before he develops hypothermia. It can cause arrhythmias and interference with the coronary artery blood flow – with possible angina or myocardial infarction. It can also have reflex effects on the lung function, causing the diver to inhale excessively, causing aspiration of sea water, resistance to breathing, electrolyte changes, etc. Another problem is that a diver may respond to the cold stimulus with intense contraction of the blood vessels, causing a rise in blood pressure and a “stroke”.

Usually these changes are not noticed by the average diver and are of more interest to the diving physician investigating unusual diving accidents. (See Chapter 35)

❑ Cold Urticaria.

Some rare divers are particularly sensitive to the inhalation of cold air (due to the drop in pressure across the first and second stages of the regulator). This can cause difficulty in breathing as the cold air irritates the respiratory passages, producing an asthma-like syndrome. Cold water exposure on the skin can also produce an allergy-like effect, with a skin reaction similar to hives (urticaria). It can lead to generalised effects on blood vessels and blood pressure (shock), and it is important that the diver ceases to expose himself to cold, and obtains medical advice.

❑ Sinus and Ear Pain.

Exposure to cold seems to produce a reflex pain in susceptible divers, similar to the "ice cream headache". The site of pain may be related to an over-sensitive area of the skin being stimulated by the cold. Similar symptoms may develop during skiing or surface swimming in cold water.

If the external ear is affected, then ear plugs can be worn for surface swimming and fenestrated ear plugs for diving. Alternately a hood can be used with a small aperture over the ears. This will reduce the cold stimulus effect by retaining the water warmed to body temperature. Others instill oil or wax to reduce this effect.

Occasionally this disorder has been confused with barotrauma of descent, as it tends to occur within a few minutes of immersion, while the diver is descending.

Chapter 28

INFECTIONS

There are a variety of both exotic and mundane infections to which divers are exposed. Some are terrestrial and are the same as experienced by non-divers. Others are caused by specific marine organisms and require special methods of identification.

ABRASIONS AND INFECTED CUTS

Divers are frequently subjected to minor injuries including cuts and abrasions. These injuries are more prone to infection than those encountered in terrestrial pursuits because of the unusual bacteria encountered in the aquatic environment and because cuts and abrasions on divers frequently remain moist for long periods of time.

Cuts and abrasions which are not due to coral or other marine life do not require aggressive cleaning unless they are obviously contaminated. Bleach, antiseptic or antibiotic cream or powder should be applied as soon as possible. When out of the water they should be kept dry and loosely covered to prevent further contamination.

Coral Cuts

Coral often causes minor cuts and abrasions in unprotected divers in tropical waters. These cuts are particularly prone to infection, probably because of the large numbers of marine bacteria on coral and the retention of coral particles and slime in the wound.

They frequently become infected within hours. Even minor cuts or abrasions can become red, swollen, tender and painful. Later there may be a discharge of pus from the area.

A severe infection may spread to the lymphatics and blood stream, with fever, chills and tender swollen lymph glands in the groin or armpit, depending on the site of the injury.

❑ Treatment.

All coral cuts should be washed with bleach or soapy water as soon as possible and the surface of the cut or abrasion should be thoroughly cleaned. This removes foreign material that may be the source of inflammation. All cuts should then have local antibiotic powder, cream or ointment applied every 6 hours until healed. The senior (elderly) author, who has a tendency towards cowardice, relies more on the antibiotics than vigorous cleansing. Suitable topical antibiotics include neomycin or bacitracin.

Early attention to every coral cut in this way will usually prevent serious infections. If treatment is delayed, or if systemic effects occur, oral broad-spectrum antibiotics may be needed. The development of a chronic inflammation may cause severe itching over the next few weeks, but this usually responds to local steroid (cortisone) ointments.

❑ Prevention.

It is wise when diving on coral reefs to always wear protective clothing or a wet suit, gloves and booties. Modern lightweight "lycra suits" afford some protection and may be worn in very warm tropical waters. These provide no flotation or thermal insulation properties, and diving must be adjusted for this.

EAR INFECTIONS

Otitis Externa (Swimmer's or Tropical Ear')

This outer ear infection is one of the most common and troublesome problems in divers. It is especially likely to occur in **hot humid conditions** (e.g. tropical climates, standard diving dress, compression chambers) or when **water is retained** within the ear after immersion, especially if with **contaminated water**. Small bony outgrowths (**exostoses**) are commonly found in the ears of swimmers and divers, and these may be large enough to cause retention of water, wax, debris and organisms. **Local injury** induced by scratching the ear canals (with a match or hair pin), or by clumsy attempts to remove **wax** (often using cotton buds) frequently precipitates the infection. Sometimes an underlying **skin disorder** is present such as eczema or dandruff. Many bacterial organisms have been incriminated, as well as fungi.

❑ Clinical features.

Mild infection causes **itching** of the ear which encourages the diver to scratch the ear canal, further breaking down the protective barrier and aggravating the infection. This has prompted the good advice that "nothing smaller than your elbow" should be inserted into the ear canal.

Serious infection may appear as a local boil in the ear canal, or as a diffuse inflammation with narrowing of the canal and an offensive smelling discharge. **Pain** with movement of the jaws or pulling on the ear is common. Occasionally a **mild hearing loss** or **dull feeling** in the ear may be noticed, and **dizziness** during diving is a possibility if one canal is completely blocked.

❑ Treatment.

The condition may be difficult to cure and treatment should be supervised by a doctor. Mild cases may only require careful cleansing of the ear canal followed by local (topical) **antibiotic + steroid** ear drops three times per day. More severe cases will need pain killing tablets such as paracetamol — two tablets four hourly as required, along with packing of the canal with special antibiotic + steroid ointments e.g. "Kenacomb otic". Oral antibiotics may be required in severe cases. Diving, along with further exposure to any water, should cease until the infection resolves.

❑ Prevention.

This can be achieved by the use of olive oil drops in the ears prior to diving, or the application of a few drops of a solution of 5% acetic acid in 85% isopropyl alcohol in each ear after a dive to ensure adequate drying. Commercial solutions include "AquaEar" and "Otic Domeboro". Scratching the ear canal with matches, hair pins, cotton buds and the like, although tempting, should be avoided.

Otitis Media (Middle ear infection")

Middle ear infection is not very common in adult divers, but may occur after **middle ear barotrauma** or following upper respiratory tract infections (**URTIs**) or allergies. It may also follow an uneventful dive. Most infective organisms enter the middle ear cavity via the Eustachian tubes, which lead from the throat to the middle ear cavity, during middle ear equalisation. Occasionally a **perforation** in the ear drum will allow direct entry of contaminated water.

❑ Clinical features.

Clinically there may be a constant and/or stabbing **pain in the ear, fever, ringing** noises (tinnitus) and often a slight **hearing loss**. In this case the ear will not usually be tender to touch.

❑ Treatment.

Treatment is urgent and will include oral broad spectrum antibiotics, pain relieving tablets such as paracetamol and decongestants (such as pseudoephedrine). Ear drops are not advised. No diving or flying in aircraft should occur until resolution — usually 5-7 days.

GENERAL INFECTIONS

There are many diseases that can be caught while diving and swimming. Some are generalised and serious, and the organisms are especially adapted to water environments. Others, such as infectious hepatitis, poliomyelitis, typhoid, cholera, gastro-enteritis, etc., are common diseases and are found especially in contaminated water. Diving in contaminated water needs special clothing and protection as well as post-dive cleansing techniques.

□ **Vibrio and other Aquatic Organisms**

Generalised manifestations such as fever, chills, septicaemia, shock etc. may be accompanied by respiratory symptoms, muscular pains, headaches, coma and death.

□ **Key West Scuba Divers Disease**

This condition is not restricted to the city where it was described, in Key West. It is due to contamination of regulators (especially older ones with twin hoses) by bacteria and tends to occur in multiple divers— such as in diving courses where regulators are shared. It can also develop from breathing bags. It is similar to influenza with respiratory symptoms, but usually clears after 3 days.

□ **Naegleria (Amoebic Meningitis).**

This lethal condition is encountered by divers or swimmers bathing in fresh water lakes, streams, hot springs, spas or hot tubs. It is caused by a microscopic amoeba which usually enters water by **faecal** contamination. It may survive in **warm fresh water** (not in sea water). The amoeba enters the body through the nose from where it burrows through the olfactory nerve to enter the brain. After an incubation period of about a week it causes meningitis and encephalitis, which is difficult to treat and is usually fatal.

The condition is usually manifest by a progressively worsening headache, fever, vomiting, discomfort on looking at bright light, neck stiffness, confusion and finally, coma. Death usually follows after 5-7 days.

There is very little that can be done to treat this dangerous condition apart from intensive nursing care in a major hospital and aggressive intravenous therapy with several antibiotics – none of which are very effective.

Because the organism enters through the nose, infection can be prevented by not immersing the head in fresh water, which is at risk of contamination. Such waters should be avoided if possible, however if diving is essential in these areas (police underwater searches, mining or drainage assessments etc.) then only diving equipment incorporating helmets which totally enclose the head and face should be used, and these rinsed off thoroughly prior to undressing after the dive. Heavy chlorination will kill the organism as will cold temperatures and seawater, eventually.

SINUSITIS

Because air passes into the sinuses during descent (see Chapters 2 and 10), if the diver has an **upper respiratory tract infection** and goes diving with this, then organisms will be transferred to the sinuses as he equalises pressures. Because of the overwhelming infection that is then produced, it is common to develop symptoms within hours or days of the dive exposure.

As a general rule, the more descents carried out, the greater the amount of infective material which passes into the sinuses. Also, if there is any sign of **sinus barotrauma** (especially on descent) then there is blood and fluid in the sinus at body temperature, which makes an ideal medium for the growth of organisms.

❑ Clinical features.

With sinusitis there is not only a feeling of **fullness over the area of the sinus** (usually maxillary, frontal, ethmoid, sphenoid or mastoid), but there is **pain** which is likely to increase in severity. If there is any significant obstruction of the sinus ostium, then pressure develops within the sinus as infection flares. There may be severe systemic signs – similar to that of an abscess, thus the diver may be **feverish**, feel ill and may look sick.

❑ Treatment.

This usually involves oral broad spectrum antibiotics, pain relief (paracetamol) and decongestants (pseudoephedrine). Sometimes a fluid level can be seen on scans or X-Ray or MRI and rarely, surgical drainage is necessary.

Because infections tend to produce scarring, sinusitis must be avoided as much as possible by divers – otherwise the openings of the sinuses can become scarred and narrowed. This means that the diver is much more likely to develop sinus barotrauma in the future – thereby limiting his diving career.

❑ Prevention.

Avoid sinus barotrauma (see Chapter 10). The rapid and effective treatment of infections that do develop in the sinuses will be of some preventative value. Of more importance is the avoidance of diving during times in which there is any inflammatory disease of the upper respiratory tract (nose, throat), such as hay fever, rhinitis or upper respiratory tract infection.

SWIMMER'S ITCH

Swimmer's itch is a localised skin infection caused by a bird parasite (*Schistosoma cercaria*) which can be encountered by persons swimming or wading in lakes or lagoons frequented by water birds. The parasite, which is present in the water, burrows through the skin and then dies, causing an inflammatory reaction under the skin. It causes multiple small, raised, red itchy lumps, which may last for a week or so.



The lesions usually resolve without treatment. Occasionally, more severe reactions may follow in individuals who are allergic to the parasites and may require medical attention by way of oral antihistamines and even topical or oral steroids (cortisone).

Fig 28.1

SWIMMING POOL GRANULOMA

Also called **Swimmer's Elbow**, this infection is due to an organism (marine *vibrios*) entering the skin via an underwater abrasion from a swimming pool, ship's hull etc. **Red swellings** covered with fine scales may develop 3–4 weeks after injury over bony prominences such as the elbows or knees. Thick pus may be found if the swelling is incised and spontaneous resolution may take up to a year or more. **Diagnosis** may only be confirmed by microscopic examination of a piece of the ulcer or lesion, and culture of the organism involved.

TINEA PEDIS – "TINEA" (or "ATHLETE'S FOOT")

This is a common fungal infection which affects the feet of divers and swimmers exposed to repeated wet and warm conditions, such as shower rooms. It causes itchy, scaly or raw areas between the toes and on the feet.

Many divers suffer from this infection, and are the source of cross-infection to others. The fungus can be found in many areas and makes the condition difficult to prevent because of repeated exposures.

Fortunately it responds readily to modern topical anti-fungal agents such as imidazole derivatives, (tolnaftate or undecylenic acid). The solution or cream should be applied twice daily and continued for two weeks after the condition appears to be cured. Attempts should be made to keep the feet as dry as possible, and drying with tissues between the toes after bathing or swimming is helpful. A light application of an anti-tinea powder (e.g. econazole dusting powder) daily may also be beneficial in preventing recurrence. In severe or resistant cases, oral anti-fungal medication such as ketoconazole or griseofulvin may even be necessary. Towels and footwear should not be shared.

PITYRIASIS VERSICOLOR (or "TINEA VERSICOLOR")

This mild fungus infection of the skin may either cause itching or no symptoms at all. With exposure to the sun however, a diver will notice coin-sized spotty areas which do not tan on the chest, back and arms. A fine scales on the skin will be seen on close inspection. It is best treated with topical anti-fungal lotions or creams such as clotrimazole or econazole applied twice daily. An alternative is 20% sodium thiosulphate (or photographer's 'Hypo' solution – but this stains clothing).

HIV/AIDS

More than 25 million people have died of AIDS, and there are 35 million people living with HIV/AIDS. Half are under the age of 25 years. The potential duration of the disease may be 1-2 decades, or longer. During this time, inevitably some will experiment with scuba diving. The risks to the diver or his companions are not known with certainty. The following is a general discussion based on the current information.

The virus must gain entry to the bloodstream to produce infection and is generally unable to enter through intact body surfaces such as skin or mouth lining. It can gain entry through small breaks in tissues such as cuts, abrasions or ulcers. Tissue fluid oozing from breaks in the skin or mouth lining can contain viruses in infected individuals. The virus is sometimes present in blood (viraemia) and saliva, making transmission a possibility (albeit probably only slight) in some situations encountered during diving. The viraemia is also the danger period to the patient, as this is the time when the virus can attack the central nervous system, especially the brain

□ Sexual.

This is the most common mode of transmission. It can be eliminated by celibacy and reduced by using condoms ("safe sex" practices), avoidance of both promiscuity and anal intercourse. A wet suit should prove an effective barrier if worn at all times.

□ Blood.

Blood from infected people can transmit the virus to others. This usually follows the sharing of needles or transfusions of infected blood, but there have been a few reports of transmission by infected blood splashing onto the skin — usually skin which has been broken. In diving, infection in this way could theoretically follow the first-aid treatment of an injured diver after trauma, cuts or marine animal injury. The sharing of mouthpieces and regulators, such as during buddy breathing, and which have not been adequately cleaned after each individual use, could pose a potential risk of infection through oral abrasions.

□ Resuscitation.

Expired air resuscitation often requires mouth-to-mouth or mouth-to-nose contact. There is a theoretical risk of transmission of the virus during resuscitation, especially if either the victim or rescuer has ulcers or bleeding in the mouth. The risk from saliva is probably small but it would be wise, if a known AIDS carrier was involved, to use a mouth-to-mask or resuscitation tube technique. If the rescuer is trained in its use, an O₂ or air resuscitator bag would be even better. See Chapter 40.

□ Sharing equipment.

It is common for equipment to be shared in diving schools. Since the virus is known to be present in the saliva of infected people, there is a theoretical risk of transmission of the disease from the sharing of demand valves (including buddy breathing practice) and snorkels. The risk is probably slight and there have not been any cases of such transmission yet reported.

Until the risk is excluded it would seem wise to disinfect shared equipment between uses by soaking in a solution known to be lethal to the virus. Check with the manufacturer beforehand to ensure that the chosen solution will not damage the equipment.

❑ **The HIV positive or AIDS infected diver.**

These individuals could pose a risk to their fellow divers in the situations outlined above. It would be considerate for them to take care to avoid situations which might bring their blood or other body fluids into contact with others. Breathing equipment should not be shared.

HIV positive cases (those with the virus infection but no obvious symptoms) have recently been shown to have neuropsychological abnormalities not obvious to themselves, but which could be detrimental to the normal intellectual functioning and judgment needed for scuba diving.

Infected divers may be exposing themselves to added risks by diving. Depression of the immune system makes them more susceptible to infection from coral cuts and abrasions, from exotic marine bacteria, and possibly to infections acquired from shared breathing equipment. It is shown by some researchers that hyperbaric environments and hyperbaric oxygen (as occurs with diving and diving treatment respectively) may reduce the integrity of the blood brain barrier and allow the extension of the virus into the brain, causing the dreaded neurological AIDS. The influence of some of the other physiological effects of increased pressure on AIDS infected divers is unknown.

❑ **Prevention.**

Both divers and non-divers should avoid contamination of the skin by other people's blood where possible, and use disposable plastic or latex gloves whenever possible. If it is unavoidable, wash the blood off as soon as practicable with soap or antiseptic solutions.

If medical attention is sought in **underdeveloped countries**, try to ensure that only single-dose vials, new or disposable syringes (i.e. totally unused) and needles are used, and that re-used instruments have been properly sterilised. Transfusions of blood and blood plasma in some of these countries carries a significant risk of HIV/AIDS or hepatitis infection. Artificial blood expanding solutions such as polygeline ("Haemaccel") should be used whenever available in such countries.

HEPATITIS

This is a highly contagious viral condition which infects and damages the liver. There are three or more variants of the virus but hepatitis-B probably poses the greatest potential threat to divers.

Hepatitis-B virus can cause a fatal infection in up to 10% of cases and fatal liver cancer or cirrhosis may develop after many years in apparent survivors. It is usually transmitted by infected blood.

There is potentially a small risk of infection in divers by sharing breathing equipment (as mentioned previously for HIV/AIDS), but the greatest risk comes from contamination of the skin by the blood of an infected person. In this regard it is similar to, but far more infectious than, HIV/AIDS. The virus is also far more "hardy" and can remain infectious for some time. Old dried blood in syringe needles found lying about can still infect anyone 'pricked' several weeks after being used by a carrier or actively infected person.

Prevention is along similar lines to HIV/AIDS.

INFECTIONS IN HYPERBARIC ENVIRONMENTS

Underwater habitats and compression chambers are humid areas which have a high concentration of oxygen.

This favours the growth of certain types of organisms, both in the chamber and on the skin of inhabitants. Outer ear infections (otitis externa), described earlier, are particularly common in underwater habitats and pressure chambers because of these environmental conditions, and divers occupying these environments are frequently given prophylactic ear drops to prevent these infections.

Should severe infections occur, treatment may prove difficult within such environments. These infections include sinusitis, bronchitis, pneumonias and skin infections. An acute attack of appendicitis occurring in a diver whilst decompressing from a saturation dive may even require surgery within the pressure chamber. The administration of general anaesthesia, along with the sterility necessary for such surgery, render this normally simple procedure much more difficult.

Chapter 29

DANGEROUS MARINE ANIMALS

There are many marine animals which are dangerous to eat, to be eaten by, or to touch. The diver who is content to observe or photograph the creatures of this undersea environment will rarely have his safety threatened by them. Of necessity, this chapter is an oversimplification, with many significant omissions. The photographs are copied from *Dangerous Marine Creatures*, by Carl Edmonds.

SHARKS

Although encounters with sharks are commonplace in diving, shark attacks on skin and scuba divers are not common. Many of the attacks recorded have been associated with spearfishing or shell harvesting, situations in which vibrations and chemicals given off by the wounded marine animal are likely to attract sharks.

In a large proportion of attacks on divers the victim was unaware of the presence of the shark until he was actually bitten. Several behaviour patterns preceding shark attacks have been documented. In some cases the **shark circles** the victim and occasionally bumps him (presumably to gain some sensory information about the nature of this unfamiliar but potential food source), before attacking.

In many tropical species, **sharks may exhibit a threat display (agonistic)**, apparently in response to a territorial invasion by the diver. This is characterised by the shark swimming with an irregular jerking motion, accompanied by an arched back, head up and pectoral fins

pointed downwards. This type of behaviour is the signal for the diver who wishes to experience old age, to depart the area.

The Great White shark has a "**bit and spit**" technique in which a single sudden powerful attack is made, with the shark then retreating until the victim (seal, dolphin, diver) haemorrhages in the water and loses consciousness. The shark can then feed without fear of damage from a counter attack.

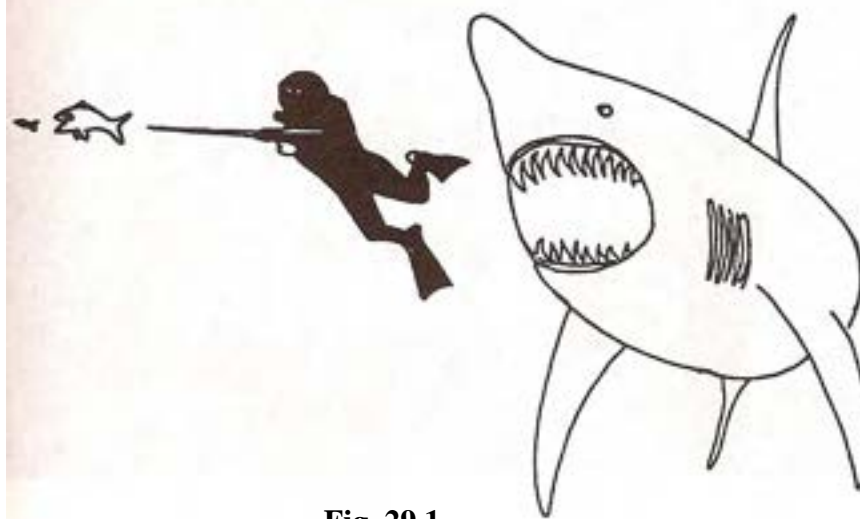


Fig. 29.1

Clinical Features

The seriousness of the injury depends on the size of the shark and the ferocity of the attack. Sharks larger than 2 metres in length have extremely powerful jaws equipped with razor sharp teeth which are easily capable of severing limbs or biting large pieces out of the torso. In spite of this, there have been many instances of divers surviving bites from sharks in excess of 4 metres in length. In some of these, the divers sustained severe lacerations from the puncture wounds of the teeth but no further injury. A shark of this size could easily bite a diver in two, so it appears that in some cases the shark will maul a victim and then not persevere, perhaps due to distaste for wet suit material or other items of the divers paraphernalia. Some divers may be as distasteful to sharks as they are to non-divers.

The blood loss from the massive lacerations accompanying shark attack is severe and immediate. Major blood vessels are frequently torn and generalised bleeding issues from the tissue laceration. Blood loss is often torrential and pulsates from severed arteries.

The victim will display clinical features of severe blood loss — pale clammy skin, a rapid weak pulse, low blood pressure and rapid respiration. Fatality occurs in 25% of cases.

Treatment

The principles of successful management of shark attack victims were first described by Australian and South African authorities following their combined experiences. They are:

□ Stop the blood loss.

This must be done by rescuers **at the site** of the attack. Bleeding which is oozing or welling up from a wound can be stopped by applying a clean cloth pad to the wound and pressing firmly with the hand or applying a tight bandage. Spurting arterial bleeders up to about 3 mm in size can also be stopped by a pressure bandage or pad. Larger arterial bleeders can be stopped by the application of pressure by a finger or thumb. Bleeding from major blood vessels (the size of a finger) can be stopped by pinching the end of the vessel between finger and thumb, or a tourniquet if a limb is involved. Tourniquets have to be released every 10–20 minutes to let blood return to normal tissues.

It is important that pads, used to stop bleeding, have pressure applied to them to force the blood vessels closed. It can be disastrous when rescuers merely cover bleeding areas with a dressing, without any pressure application. This soaks up and conceals the blood loss, without stopping it. Any clean material such as toweling, clothing or handkerchiefs are satisfactory in the first-aid situation.

□ Resuscitate the victim at the site of the attack.

Immobilisation is advised. Once the victim is in a place of safety, (boat or shore) it is vital that he not be moved further. Bundling a victim into the back of vehicle for a bumpy ride to hospital has resulted in death of the victim on many occasions.

The victim should be kept lying horizontal at the rescue site and resuscitation equipment and personnel brought to him (see Chapter 42 for **A–B–C** of resuscitation).

Resuscitation involves replacing the patient's blood loss by the **intravenous infusion** of blood or blood substitutes such as plasma, saline or other intravenous fluids. It is not safe to move the victim until a satisfactory circulating volume has been established. Evidence for this is a relatively normal pulse (rate less than 100) and blood pressure.

This management principle is sometimes difficult to accept by rescuers who understandably wish to dispatch the victim to hospital (anywhere!) as soon as possible. However, once the victim reaches there, exactly the same management as should have taken place at the shark attack site will be needed. i.e. arrest of the blood loss accompanied by the administration of intravenous fluids.

Major hospitals in shark attack prone areas have a shark attack protocol along the lines mentioned above. Equipment may be available for immediate transportation to a shark attack site. Shark attack is so rare, however, that practice at implementing this protocol is sometimes neglected.

In spite of the severity of the injuries, it is common for the patient not to experience significant pain for some time after the attack. This phenomenon is frequently seen in other forms of severe injury such as motor vehicle and war injuries. If the patient is suffering significant pain or shock, the rescuing medical team will administer morphine in an appropriate dose.

Nothing should be given by mouth to the victim, as an anaesthetic may be required.

Prevention

Since vibrations and chemicals given off by speared fish and other forms of marine life commonly attract sharks, the avoidance of fishing should lessen the risk to the diver. The carrying of speared fish near the diver's body underwater invites a close inspection by an interested shark.

The well publicised practice of diving with a buddy should, on statistical grounds alone, reduce the likelihood of a shark attack on oneself by at least 50%.

Case Report 29.1 A young swimmer was attacked by a shark which amputated his leg above the knee. He was pulled from the water by his companions and the bleeding stump was wrapped in a blanket. He was noted to be pale and clammy with a weak thready pulse and was semi-conscious. He was placed in the back of a car and rushed to the nearest hospital which was 15 miles away.

A subsequent newspaper report read: "shark attack victim died while being rushed to hospital". It should have read: "shark attack victim died **because** he was rushed to hospital".

Swimmers are protected by swimming in enclosed or shark meshed areas. They are advised not to swim where shark attacks have occurred or where fish or meat products are ditched (fish markets, abattoirs etc.). It is also safer to swim with groups of people and to avoid swimming at dusk (feeding time for sharks) or in areas of low visibility. Urine and blood are claimed to attract sharks and thus should not be released into surrounding water. Women who are menstruating, produce haemolysed blood which is not an attraction to sharks.

Divers are given the same advice, with the additional recommendation to avoid deep channels and drop-offs, and if diving with sharks, to carry an implement that can be used to fend them away (shark billy). A chain mail suit gives good protection, but it very heavy and thus dangerous for recreational divers. Ultrasonic, electrical, chemical and bubble deterrents are probably not effective against dangerous animals, but are enthusiastically marketed.

BOX JELLYFISH OR SEA WASP

This deadly stinging creature is found in the tropical waters of the Indian, Pacific and Atlantic oceans during certain seasons. The season for North Australia is October to March, but may be year long nearer the equator. They are rare in the temperate regions. The animal is an active swimmer which may be found even in very shallow water around beaches.

Its numerous tentacles may trail for up to 3 metres behind the body, which grows to 20 cm along each side of the cube. The tentacles cling to the victim's skin and contain thousands of microscopic stinging cells (nematocysts) which can inject venom. The innumerable tiny doses of venom injected combine to form a large injection of toxin into the victim. The amount of venom injected depends on the length of tentacle in contact with the victim, and the area stung, as well as the thickness of skin.

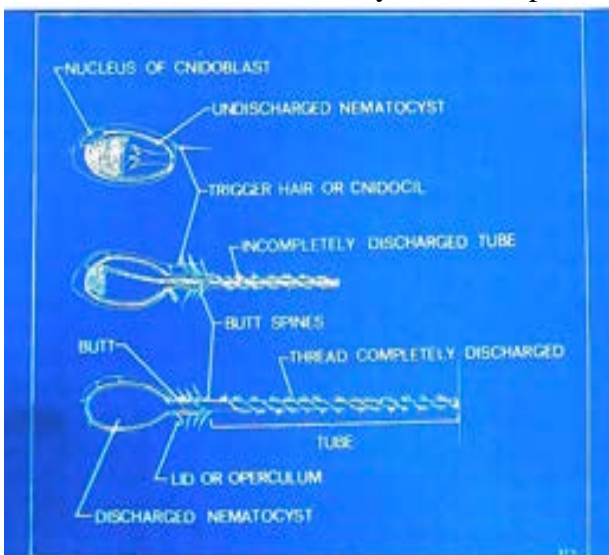


Fig. 29.2 Nematocysts from jellyfish

The venom has its most serious effects on the heart and the respiratory system. It paralyses the respiratory muscles leading to death.

Weakening of cardiac contraction, as well as cardiac rhythm disturbances, compounds the problem. The venom exerts a local effect producing agonising pain.

Clinical Features

The victim experiences **immediate agonising pain** on contact with the tentacles. With a large sting, sudden **collapse, cessation of breathing, cyanosis, unconsciousness** and **death** may follow rapidly. These effects are particularly dangerous in small children or old frail swimmers.

If the victim recovers, severe pain still persists for many hours, and **scarring** is common in the stung areas due to local tissue destruction.

Treatment

Rescue the victim from the water and prevent drowning. This takes immediate precedence.



The first-aid principles of **A–B–C** (airway maintenance, breathing support and circulation maintenance) as described in Chapter 42 should be applied.

Apply copious amounts of ordinary **household vinegar** to the tentacles and gently remove the tentacles from the victims skin. The tentacles cannot sting effectively through the thick skin of the palm of the hand and fingers so this may be safer than it sounds. It is important not to rub or damage the tentacles as this will encourage the injection of further venom into the victim.

Alcohol application is no longer advised, as there is some evidence that this may cause the discharge of further venom into the victim. If it is of good quality, the alcohol may be more beneficial to the rescuer, once the victim has been taken safely to hospital.

The cause of death in box jellyfish sting is usually respiratory arrest. However, this may be transient if the victim is kept alive by **expired air resuscitation (EAR)** or other artificial ventilation during this period. The victim should be transported to hospital urgently.

Fig 29.3 Chironex – Box Jellyfish

An **antivenom** against the Chironex box jellyfish neutralises the venom present in the victim's body. It has been developed by the Commonwealth Serum Laboratories (CSL Australia) and should be used in severe cases or where significant local scarring is threatened. It may not be as effective against other box jellyfish.

Prevention

The practice of covering as much exposed skin as possible by the wearing of a face mask, wet suit and hood, overalls or a Lycra suit, prevents the access of tentacles to the skin. This protection also reduces the risks of stings from other jellyfish and injuries from corals. Even water repellent skin preparations, such as sun-burn oils and creams, may reduce the danger.

OTHER JELLYFISH STINGS

Several other stinging jellyfish such as the **Portuguese Man-of-War**, **fire coral** and **stinging hydroids** can produce painful and sometimes incapacitating stings, although they are unlikely to be lethal.

The same technique of general management as described for box jellyfish should be followed. However, different local applications seem to work for different species. Some degree of pain relief can be afforded by the application of **local anaesthetic** (e.g. lignocaine ointment) to the stung area. Other preparations which have a variable effect, include "Stingose", "Stop-Itch", Tannic Acid Spray, etc. Any **anti-burn** preparation, including ice packs, may give some relief. More recently, application of heat (about 45°C. as for fish stings – see later) has been advocated.

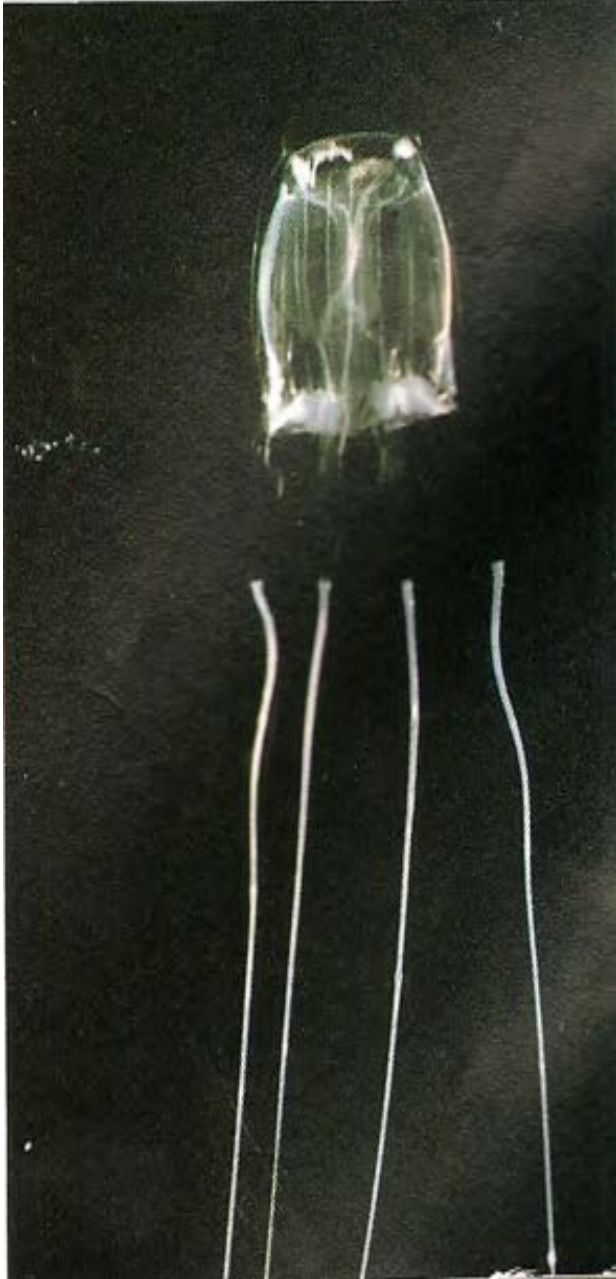


Fig 29.4 Physalia (Blue Bottle, Man-of-War)



Fig 29.5 Stinging Hydroid

IRUKANDJI SYNDROME



This disorder was first described as a result of an almost innocuous small box jellyfish (*Carukia barnesi*) sting with big after-effects. It is now known to be a possible complication of many other jellyfish stings. It has been the cause of severe cardiac illness and has been confused with both an acute abdomen and cardio-pulmonary diseases, because of its various symptoms.

The victim may, or may not, be aware of the sting. If not, there still may be a red patch visible where the sting occurred. After a latent period of between a few minutes and two hours, severe muscular cramps and pain develops (abdominal, spinal, limbs, chest). The diver becomes anxious, restless, sweating and may have gastro-intestinal and respiratory symptoms. There may be increased pulse rate and high blood pressure recorded. Cardiac damage and pulmonary oedema can develop.

Because of the latent period, the relationship to the jellyfish sting may not be appreciated, leading to many other medical diagnoses, including decompression sickness.

First aid treatment may involve copious vinegar application and/or pressure bandage+immobilisation (see later) if identified early. Successful medical investigations and therapies are available, but deaths have been reported.

The injury can be prevented by wearing protective clothing (see above).

Fig. 29.6 *Carukia barnesi*. This is the most frequent animal incriminated in the Irukandji syndrome. Its bell is only a couple of centimetres (one inch) long, but the tentacles, which are not always easily seen, are up to a metre long.

VENOMOUS CONE SHELLS

A small number of species of the cone shell family are capable of delivering a lethal venom. This is injected by a tiny dart shot from a tubular appendage which the animal can direct to any part of its shell. This apparatus is normally used by the animal to kill its prey (usually small fish), but it will use it as a weapon against a human who is careless enough to handle it.

Expert knowledge is required to differentiate venomous from harmless cone shells, and divers are advised to avoid handling them at all. Reef walkers, being less valuable than divers, may do as they wish.

Clinical Features

The initial sting may or may not be painful. It can penetrate cloth and skin. They should not be handled or put in pockets. The toxin affects the heart, skeletal and respiratory muscles. Muscle spasms develop. Death is usually from respiratory arrest.



Fig. 29.7 A collection of venomous cone shells

Treatment

The application of a **pressure bandage and immobilisation** (see later) should delay the spread of venom from the wound, although there have been no clinical case reports to verify this.

The **A–B–C** resuscitation measures (see Chapter 42) may keep the patient alive until the respiratory paralysis has worn off. This may involve many hours of artificial respiration.

BLUE RINGED OCTOPUS

This attractive little animal is found in rock crevices along the water's edge of many islands in the Pacific and Indian oceans, as well as in deeper water. If annoyed it will display a colourful array of blue or purple rings on its skin. This may arouse the curiosity of a potential victim, especially a child.

Unfortunately it can inflict a small, relatively painless, bite and inject venom through a beak at the base of its tentacles. The bite may go unnoticed by the victim until the major effects of the venom are felt.

The injected venom can produce muscular paralysis within minutes, leading to cessation of breathing. The victim will normally remain fully conscious but will be unable to



communicate with bystanders due to the paralysis. Death is due to the respiratory failure, unless treatment is given.

Fig. 29.8 Blue Ringed Octopus

This dangerous little animal should not be handled.

Treatment

Artificial respiration must be continued until recovery (**4 – 12 hours**). This is necessary because of the respiratory muscle paralysis.

A **pressure bandage** and **immobilisation** (see later) should be applied to delay spread of the venom, until full resuscitation measures have been implemented.

Case Report 29.2. A diver found a small octopus with attractive iridescent blue rings - hiding in a shell. She placed it under her wet suit vest, intending to show it to her companion later. After the dive she complained of double vision and respiratory difficulty. When she showed the octopus to her buddy, the buddy correctly diagnosed the problem and kept the victim alive by mouth to mouth respiration until hospital was reached. The victim later pointed out that she was not encouraged by comments such as "it looks as though she is not going to make it " from bystanders who had not realised that she was fully conscious, in spite of being paralysed.

SEA SNAKE

Sea snake bites are not uncommon in the Indo-Pacific ocean waters. In certain areas, sea snakes will approach divers underwater. These advances may be inspired by curiosity, as it is rare for sea snakes to bite divers without provocation. They will retaliate if grabbed.

The venom of sea snakes is more potent than that of the cobra. Even when bites occur, the presence of short fangs at the back of the mouth deprives many sea snakes of an efficient way of delivering this venom into humans. Often venom is not injected, despite the biting.

Fig. 29.9 Yellow Bellied Sea Snake (*Pelamis platurus*)



Clinical Features

If envenomation occurs, symptoms may become evident within minutes to hours after the bite. Muscle weakness leading to paralysis, including **respiratory muscle paralysis** and **asphyxia**, and finally **cardiac failure** may follow the bite.

Occasionally the sea snake bite itself results in severe **lacerations** and **blood loss**.

Treatment

The **pressure bandage + immobilisation** technique (see later) will delay the symptoms until medical assistance, resuscitation facilities and antivenom can be acquired.

A–B–C first-aid measures should be instituted where necessary (see Chapter 42). Mouth to mouth respiration is the major requirement. The victim should be taken to hospital as soon as possible. Serious cases should be treated with sea snake **antivenom** (made by CSL – Australia).

STONEFISH

This is the most venomous fish known. It is extremely well camouflaged and may not move away when approached, as is implied by its name.

It is capable of inflicting severe stings by means of 13 poisonous spines along its back. The spines are able to penetrate rubber soled shoes or neoprene boots. At the base of each spine is a venom sac which empties its contents into the victim's wound.



Fig. 29.10 Stonefish

Clinical Features

Envenomation results in severe **agonising pain at the site** of puncture. Extreme **swelling** and **local paralysis** develops rapidly. The venom can lead to **respiratory distress, cardiac disturbances** and syncope (**fainting**) with a reduction in **blood pressure**. Death is uncommon except in children or the infirm.

Treatment

Immersion of the stung area in hot water about **45° C** (first tested by the attendant's hand, to ensure against scalding) often gives significant pain relief and should be employed as soon as possible as a first-aid measure. Elevating the wound may reduce swelling.

The severe pain of the sting can be relieved by the **injection of local anaesthetic** (with no added vasoconstrictor agent such as adrenalin) **into the puncture sites**. This treatment may need repeating several times before the pain stops recurring as the effects of the local anaesthetic injection wear off. A physician may prefer to block the nerve supply to the region with local anaesthetic as an alternative. Cleansing of the wound and antibacterial treatment is required.

Resuscitation (A–B–C supportive measures, see Chapter 42) should be instituted if necessary. **Antivenom** from the Australian CSL Laboratories is now available and its use may be necessary in severe cases.

OTHER SCORPION FISH

Other members of the scorpion fish family such as the **fortescue**, **lionfish** (or **butterfly cod**) and **bullrout**, produce painful stings similar to that of the stonefish, although both the local and generalised effects are usually not as severe. **Cat fish** have a similar effect.



Fig. 29.11 Butterfly Cod



Fig 29.12 Fortescue

Pain relief can be obtained by **immersing the area in hot water** at about **45°C** (previously tested by immersing an unaffected limb in the water) as for the Stonefish sting (above), while more sustained relief can again be obtained by **injecting the punctures with local anaesthetic (no adrenalin)**. Cleansing of the wound and antibacterial treatment may be required, and the wound should be elevated.

STINGRAY

These flattened relatives of the shark have one or more long bony spines, which are intended for self defence, at the base of the tail.

The animals often bury themselves in the sand where they can inadvertently be stood upon, or otherwise disturbed, by an unsuspecting diver. The stingray defends itself by swinging its tail quickly over the top of its body, driving the spine into anything which happens to be above it.

The spine may produce a puncture and deposit venom. Its serrated edge can cause serious or even lethal lacerations. Parts of the spine, marine organisms and a toxic slime may be left in the wound to cause infections and local inflammation.

Clinical Features



Fig. 29.13 Typical manner in which a stingray injury occurs

Pain caused by the toxin is **immediate** and **very severe**. **Swelling** is rapid. Toxin may be absorbed into the body producing **generalised symptoms** of syncope (**fainting**), weakness, palpitations, low blood pressure and disturbances of cardiac rhythm. **Death** is rare – except in cases where a vital organ such as the heart have been pierced by the spine.

Despite initial improvement, there can be a deterioration in the clinical state some days later, if there is any

foreign material or organisms left in the wound, or if damaged tissue becomes necrotic. For this reason, all cases should be referred for medical assessment.

Treatment

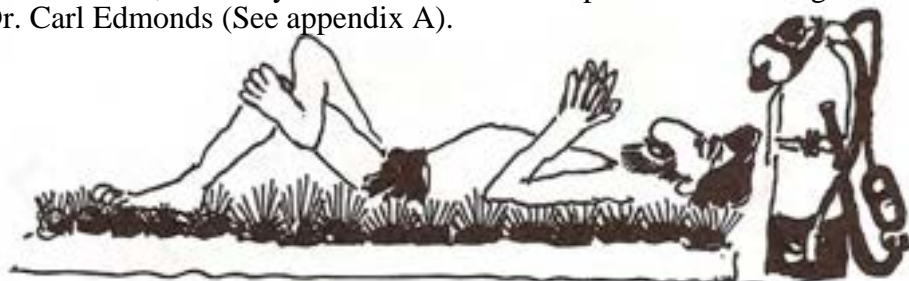
The **hot water** immersion treatment and the use of **injected local anaesthetic**, as described for stonefish injury, are useful. The wound should be **cleaned** to remove any foreign body or venom. An X-ray, ultrasound, CT or MRI may demonstrate an embedded spine, which needs to be removed surgically. Local antibiotic cream, and often oral antibiotics (such as doxycycline), are needed.

Prevention

Shuffling the feet while wading in areas frequented by stingrays will usually allow them to move away. Footwear may not be adequate to protect the feet or lower legs from these injuries. Diving into shallow waters where these animals inhabit could be dangerous. Divers should swim well above the sea bed.

OTHER MARINE ANIMALS

Many other marine animals may cause major or minor injuries, and require different first-aid treatments. These, together with more detailed descriptions of the potentially lethal animals and those poisonous to eat, are fully discussed in the companion text "Dangerous Marine Creatures" by Dr. Carl Edmonds (See appendix A).



PRESSURE BANDAGE + IMMOBILISATION TECHNIQUE

This is used to delay the absorption of venom from a wound. A bandage (preferably stretchable) is applied over the bite and then wrapped around the limb (and extending up the limb) tight enough to block the drainage vessels (lymphatics). The pressure is approximately the same as that used to treat a sprained ankle.

Care must be taken not to put the bandage on so tight that it causes pain and cuts off circulation. For this reason the technique is not applicable to painful, swollen bites that already have circulation impairment.

The limb should then be immobilised with a splint to prevent any local muscle movement (this spreads the venom despite the bandage).

The pressure bandage+immobilisation of a limb should be continued until the victim has knowledgeable medical personnel and facilities available to cope with the envenomation. This happens as the bandage is released and the venom moves into the bloodstream. The doctors may well administer antivenom (if available), before removing the bandage.

The technique is especially applicable to sea snake, blue ringed octopus and cone shell bites. A variant may be used if the bite is on the torso, with a pad and bandage to produce the pressure.

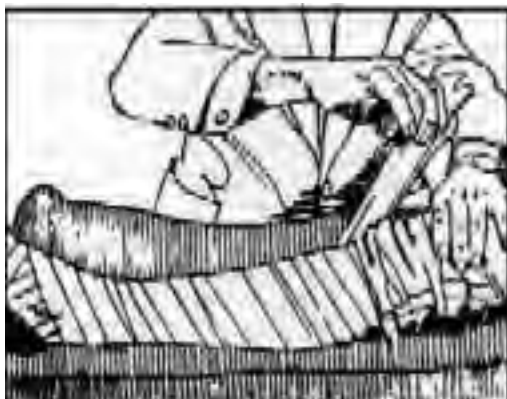
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Chapter 30

HEARING LOSS

This chapter may be easier to understand if the structure and function of the ear, as outlined previously in Chapter 9, is reviewed. All cases of hearing loss should be assessed by a diving physician.

Divers frequently complain of a sensation of hearing loss which cannot be verified when hearing tests (pure tone audiometry) are performed. It is likely that currently available hearing tests, such as speech discrimination, are not sophisticated enough to detect such subtle alterations in the sensation of hearing.

The **causes** of demonstrable hearing loss fall into two categories:

- **Conductive hearing loss** – where there is some impediment to the conduction of sound vibrations (usually in the external and middle ear) *en route* to the hearing organ.
- **Sensorineural (nerve) hearing loss** – where sound vibrations reach the hearing organ (cochlea) in the inner ear, but the sound is not perceived due to damage of the cochlea or its nerve.

CONDUCTIVE HEARING LOSS

The likely causes of conductive hearing loss are in the external or internal ear.

External Ear Obstruction

Any obstruction to the outer ear such as wax accumulation, plugs or hoods, outer ear infections (see Chapter 28) or exostoses (see Chapter 32) can cause this.

Tympanic Membrane Damage

This membrane can be torn by:

- **Excessive stretching** during descent (middle ear barotrauma). See Chapter 9.
- A **shock wave** passing down the ear canal, such as an **underwater explosion** or a **pressure wave from a fin** passing close to the divers ear.
- An excessively forceful **Valsalva** manoeuvre has also been known to rupture the tympanic membrane from within.

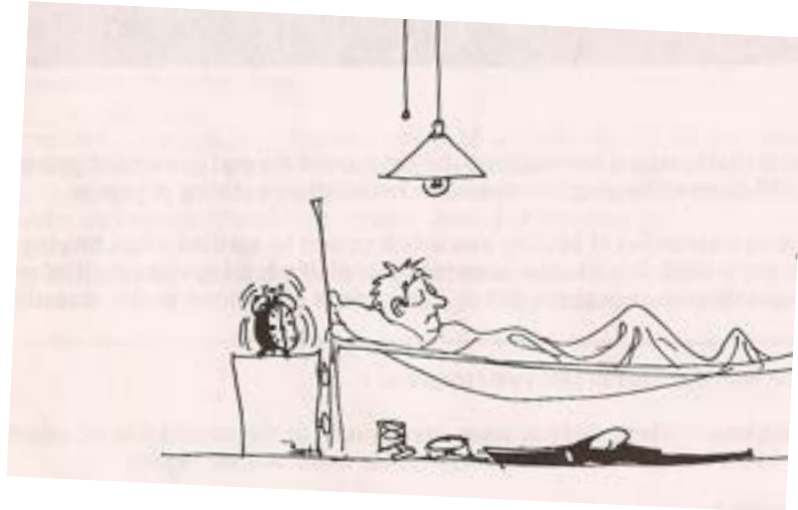


Fig. 30.1

Case History 30.1. A diver swimming closely behind his buddy suddenly felt pain in his left ear as his buddy's fin swept past his ear. Dizziness followed but soon settled. He surfaced and noticed a small amount of blood coming from his ear.

Diagnosis: Rupture of the ear drum caused by a pressure wave from a fin. The dizziness was due to cold water entering the middle ear through the ruptured ear drum. The blood was extruded by gas expanding in the middle ear, during ascent.

Case History 30.2. An old professional hard hat diver who smoked cigarettes and had suffered repeated tympanic membrane ruptures from barotrauma was in great demand at parties because of his ability to blow smoke from his ears. He claimed that during the latter part of his career he no longer needed to equalise.

Diagnosis A: Chronic perforation of the ear drums. His unusual talent was made possible by smoke passing from his throat to his ears through the Eustachian tubes, after he takes a drag from the cigarette and performs a Valsalva manoeuvre. His ears became self equalising later in his career because of permanent holes in his ear drums. Although in demand at parties, unfortunately he often misheard the directions and turned up at the wrong address.

Diagnosis B: Poor hearing due to recurrent barotrauma and chronic perforation of tympanic membranes.

Middle Ear Disorders

Disturbances of the middle ear impair conduction of sound vibrations from the ear drum, through the bony chain to the cochlea. Causes include:

- **Middle ear barotrauma** which produces bruising and swelling of the middle ear tissues and bleeding into the middle ear space. Both factors dampen sound transmission (see Chapter 9).
- **Middle ear infection (otitis media)** which causes swelling and inflammation. This fills the middle ear space with pus, which impairs sound conduction (see Chapter 28).

SENSORINEURAL HEARING LOSS

This is often accompanied by tinnitus (ringing in the ears) and sometimes by disorientation. Tinnitus can sometimes be more incapacitating than hearing loss. In recent hearing loss, early treatment increases the likelihood of improvement in hearing.

Noise Induced Deafness

Repeated exposure to loud noise may produce a progressive hearing loss which usually affects high frequency hearing first. This loss may be noticed by hi-fi enthusiasts who will complain that music has lost its sparkle. It is often insidious and may not be noticed for many years. Occasionally, a single exposure to loud noise can cause noticeable hearing loss immediately. Rock concerts and discos are also incriminated.

Noise induced hearing loss may be transient in the early stages but repeated exposure leads to permanent deafness, which worsens with more exposure. Industrial noise usually affects the ears symmetrically, but other noise such as gunfire, commonly affects only one ear (the one exposed to the noise or blast).

The diving environment is often a noisy one. Recompression chambers, compressors, boat engines, helmets and compressed air leaks are often loud enough to present a threat to the hearing of those in their vicinity. Divers should take care to protect their ears when necessary by the use of industrial protective ear muffs or ear plugs (but not when diving).



Fig. 30.2

High frequency hearing loss also renders consonants such as "S" or "CH" difficult to hear – hence the story of the yacht owner who was delighted when the curvaceous blonde diver with sensorineural deafness accepted his invitation to "crew" on his yacht.

Barotrauma

Inner ear barotrauma or associated round window fistula may lead to temporary or permanent hearing loss (see Chapter 9).

Decompression Sickness

Inner ear damage is an uncommon complication of decompression sickness (see Chapter 15) in air breathing divers. It is more common in deep helium or mixed gas divers.

OVERVIEW OF HEARING LOSS

- **All prospective divers must have their ears examined to exclude ear problems** likely to predispose to barotrauma.
- **All divers should have a baseline audiometry** performed, to enable the physician to detect early hearing loss, to make assessment of future hearing problems much easier and to allow early and more knowledgeable treatment to be administered in the (not uncommon) event of a diver presenting with hearing loss.
- **Any case of hearing loss in a diver should be assessed as soon as possible by a diving physician.** The doctor will take a history of the condition, examine the ears, test the hearing by pure tone audiometry at least, and possibly perform other specialised investigations such as bone conduction, speech discrimination, impedance audiometry, diving tympanogram, electro-nystagmograms and brain stem evoked auditory responses.

The cause is usually fairly obvious and **management** of the specific conditions is covered in other chapters.

- **Divers with pre-existing hearing loss** should realise that any deafness arising from barotrauma will be added to the loss they already have. It is also believed that people with hearing impairment are more susceptible to further damage than others. Divers who are aware of hearing loss should discuss the implications with a diving physician.
- **Occupational implications are raised.** Those who need excellent hearing for their livelihood, such as musicians, cardiologists, sonar operators and airline pilots, should consider whether the small but real risk of hearing damage associated with diving is worth taking.
- **Hearing loss is sometimes associated with abnormalities of the body's balance mechanism,** which might have safety implications with diving (see Chapter 31).

Chapter 31

DISORIENTATION

Accurate orientation whilst underwater is important for the diver so that he can find his way back to the surface. On land the diver uses a combination of vision, the feeling of gravity on his body, and the balance organs (vestibular system) to tell him which way is up.

When underwater, the diver becomes virtually weightless, depriving him of the sensation of gravity and making him reliant on vision and his balance organs for spatial orientation. With poor visibility, even the visual cues are lost, leaving the diver almost totally reliant on his balance organs for this orientation. A sensation of disorientation requires investigation by a diving physician.

The experienced diver can acquire some clues about his body position from:

- the way heavy objects such as the weight belt or other metal objects hang,
- the direction his bubbles are going,
- the direction of a life-line or hookah hose.

Inexperienced or panicking divers are often unable to use this subtle information. If the diver becomes disoriented he is likely to experience anxiety. Panic can easily ensue.

VERTIGO — OR "DIZZINESS"

This is a false sensation of spinning or moving. The diver may either have a sensation of himself spinning or the environment spinning about him. It happens because the balance organ (**vestibular system**) can be unreliable underwater – it was designed to work on land. Under certain circumstances it can supply the brain with misleading information which is falsely interpreted as movement.

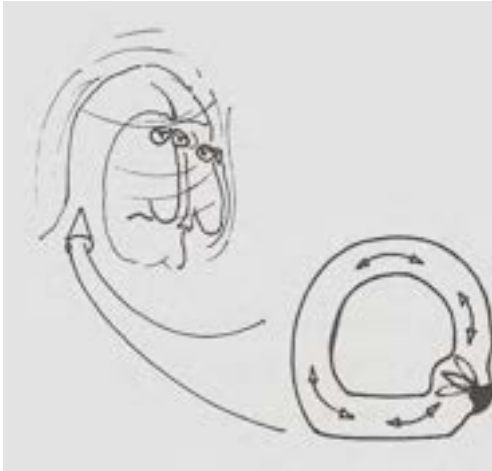
The sensation of vertigo is bad enough, but it is often accompanied by **nausea** and **vomiting** which can threaten a diver's life. These symptoms may vary from mild to very severe. Vomiting into, and then breathing from, a demand valve is not easy underwater.

Function of the Vestibular System

The balance or vestibular system comprises two marble-sized structures located in the skull above and behind the middle ear space on either side of the head. Each vestibular apparatus has two parts. Abnormalities of either cause vertigo and disorientation. They are:

□ A system of three interconnecting tubes (semi-circular canals).

These are aligned at right angles to each other and filled with fluid. They detect movement in all three planes. If the body rotates, the fluid in these three canals tends to lag behind, due to its inertia. The differential movement of the body and the fluid is detected by nerve endings – hair like projections into the fluid (hair cells) at the base of each canal.



The semi-circular canals are located close to the ear canal. Cold water entering the ear canal can cool them slightly, causing convection currents in the fluid. The movement of the fluid is detected by the hair cells and, if this does not synchronise with stimuli from the other side, causes vertigo. This is termed **caloric** induced vertigo and is usually associated with the diver being in a near horizontal position.

Fig. 31.1

□ The Otolith organ.

These other fluid-filled structures have a viscous base which contains minute calcium granules. Hair like projections of nerve cells penetrate this gel and detect any movement of the granules. Because of their weight, the granules tend to move in response to **gravity** and **acceleration**. The hair cells detect this and continuously inform the brain about which way is up and the direction of any acceleration.

Pressure changes can cause **barotrauma** induced vertigo, and it is possible that this could be due to stimulation of the otoliths or the semicircular canals and is usually associated with the diver being in a more vertical position.



Fig. 31.2

CAUSES OF VERTIGO

Problems arising from the vestibular system fall into two principle categories:

- unequal vestibular stimulation and
- unequal vestibular response.

Unequal Vestibular Stimulation

If both vestibular systems are equally sensitive but are stimulated unequally, then vertigo may result due to the unequal responses received by the brain.

Any condition causing more cold water to enter one external ear more than the other causes unequal **caloric** stimulation. Wax blocking one ear, an air bubble, otitis externa, exostoses, ear plugs or a ruptured ear drum will all have this effect.

With middle or inner **ear barotrauma** (see Chapter 9) affecting one side, or **decompression sickness** (see Chapter 15) on one side, unequal vestibular stimulation may result in vertigo.

Failure of the ears to equalise pressures to the same degree can stimulate the vestibular system unequally. This is not uncommon on ascent, as the pressure of the expanding gas in the middle ear spaces can become greater on one side than the other due to differences in patency of the Eustachian tubes releasing it. This is termed **Alternobaric Vertigo** or **middle ear barotrauma of ascent** (see Chapter 9). It is very common and is often noticed as the diver ascends a metre or so, from depth. He may even be aware of the sensation of one Eustachian tube opening before the other, or of the expansion of air in the other middle ear.

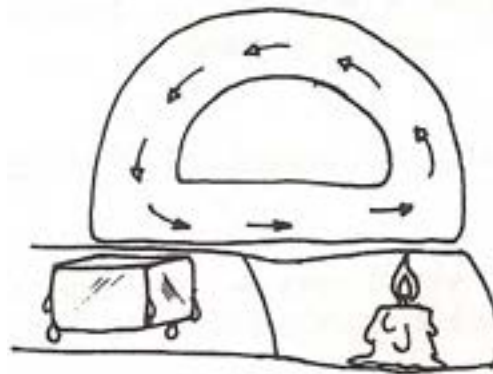


Fig. 31.3
Caloric stimulation producing "convection" current flows in inner ear fluids.

Case Report 31.1. A diver using hookah apparatus on a training dive at night lost contact with the bottom. He was unable to see his bubbles and had no idea which way was up, but by feeling the direction of his air hose, he was able to establish where the surface was.

Diagnosis: disorientation due to the reduced sensory input (decreased vision) of night diving.

Case Report 31.2. An inexperienced diver had difficulty equalising his middle ear during descent. He continued to descend in spite of this. The pain was abruptly relieved and he became aware of a hissing sound and cold sensation in his ear as his ear drum perforated. Seconds later he developed a severe sensation of spinning which was accompanied by nausea. He clung onto his shot line and was relieved when the vertigo gradually passed off after several minutes.

Diagnosis: Vertigo due to one sided vestibular stimulation (caloric) from cold water entering the middle ear when the ear drum ruptures. As the water warmed to body temperature, the cooling effect to the vestibular system subsided.

Unequal Vestibular Response

The two vestibular systems are normally equally sensitive to any stimulus such as movement. Some people may have unequal sensitivity. This can be due to a slight imbalance (either overactive or underactive function) which has been present from birth or to damage to one side from causes such as ear barotrauma or some medical conditions. In this situation, the same stimuli cause a greater response from one side than the other, and is experienced by the person as vertigo.

People with this problem unconsciously adapt by avoiding sudden movements of the head or body. They learn by experience to avoid gymnastics and roller coaster rides, but are usually not aware of the dangers posed to them by the extreme stimuli which are commonplace in diving.

Water entering the ear canals is a potent cause of vertigo in these people. As mentioned above, this water can cool the fluid in the semi-circular canals setting up convection currents (**caloric stimulation**). If the vestibular systems on each side are not equally sensitive, a stronger response will be produced from one side. The brain will interpret this information as indicating movement, and the diver will experience vertigo and feel disorientated.

During ascent or descent, equal **pressure changes** in the middle ear can also produce vertigo and disorientation in those with unequal vestibular response.

Other Causes of Vertigo

If the diver is deprived of normal visual cues in conditions of **poor visibility** or at night, it is possible for the resulting disorientation to culminate in vertigo, especially in inexperienced divers.

Nitrogen narcosis may aggravate vertigo. Vertigo has also been recorded as a symptom of other conditions not commonly encountered by recreational divers. These include **oxygen toxicity**, **carbon dioxide toxicity**, **carbon monoxide toxicity**, and **high pressure neurological syndrome**.

PREVENTION

A thorough diving medical examination before a prospective diver undertakes training can exclude some of the factors predisposing to vertigo, and is advised for all divers.

A diver experiencing vertigo under water should avoid unnecessary movement and hold onto a fixed object if one is available. Fortunately, in most cases, the vertigo is short lived. If the vertigo fails to abate and there are no obstacles above him, ditching of weights, possibly with cautious inflation of a buoyancy compensator, should return the diver to the safety of the surface.

Any diver who experiences vertigo under water and survives should abandon the dive, **consult a diving physician** to investigate, identify and correct the cause before diving again.

Occasionally the diver may be very astute and be aware of the cause of the vertigo, and may then correct the problem e.g. with middle ear barotrauma of ascent (see Chapter 9).

CONCLUSIONS

While **disorientation** under water can be unpleasant and dangerous, **vertigo** can be life threatening because of the risk of vomiting or panic.



Fig. 31.4

MISCELLANEOUS DISORDERS

PULMONARY OEDEMA

Scuba Divers Pulmonary Oedema is usually described as an uncommon disorder, often in apparently healthy individuals. In a survey of scuba divers about 1% described it. An individual predisposition for pulmonary oedema is a likely factor since a diver or swimmer with pulmonary oedema may have other episodes previously or subsequently.

It presents clinically with difficulty with breathing, with fast shallow respirations and a sensation of crackling sounds in the chest. It may be associated with fatigue, cough, sometimes blood-stained, and possibly a bluish tinge to the lips, tongue and face (cyanosis).

Symptoms usually resolve rapidly (some hours) after the immersion, but deaths have been reported. Treatment includes oxygen inhalation and medical assessment is required to verify the illness and exclude any predisposing features, such as undiagnosed heart disease.

Whether the disease is due to the individual diver, the dive profile, environmental conditions or the dive equipment, is unknown. It is more common in older divers, possibly aggravated by the effects of immersion, breathing against a inspiratory resistance from the regulator, cold exposure, hypertension, heart disease or using certain drugs (especially beta blockers).

Other diseases that can produce pulmonary oedema and cause diagnostic confusion are the salt water aspiration syndrome, drowning, respiratory oxygen toxicity, gas contaminations, cold urticaria and asthma. Pulmonary decompression sickness, pulmonary barotrauma and the so-called 'deep diving dyspnoea' are diving disorders that may cause diagnostic confusion. Anxiety produced hyperventilation may also cause some diagnostic confusion, but this has none of the other respiratory manifestations.

CONTACT LENSES

Contact lenses are a convenient alternative to spectacles but can be a source of problems to the diver. The most common of these is **loss** of the expensive lens during removal of the face mask. The eyes should be shut while removing the mask underwater or on the surface.

In certain circumstances, especially during long or deep dives or in compression chambers, it is possible for **gas bubbles** to form behind the contact lens (particularly with hard, non gas-permeable lenses) causing pressure and damage to the cornea of the eye. If this happens, the diver may experience discomfort in the eye, blurred vision and the appearance of halos around bright lights. Long term effects could include scarring of the cornea.

Gas bubble damage can be overcome in the hard contact lens by an optician drilling a small hole in the centre of the lens (a **fenestrated** lens) which allows gas bubbles to escape. This has no effect on the visual performance of the lens. Soft contact lenses are usually not a problem because of their gas permeability and flexibility.

It is now relatively easy to have corrective lens ground into or attached to the diver's face mask, as an alternative to contact lenses (see Chapter 5).

MUSCULAR CRAMPS

Cramp is a painful spasm of a muscle group. It is common in divers and can cause a dangerous diversion or incapacity. The muscles most commonly affected are those in the sole of the foot, the calf and the thigh, but other muscles can also be involved.

Unusual exertion of the muscles, due to changes in fins or equipment, makes cramp more likely, especially if the diver is generally unfit. Cold water is another predisposing factor.

Cramp is managed by slowly stretching and maintaining tension on the muscle involved. Sometimes this may require the diver to actually stand and push down with his toes onto some firm underwater surface in order to stretch the muscle. Ditching of weights underwater or inflation of the buoyancy vest on the surface may be helpful in an emergency, avoiding the need to continue swimming.

This condition can be inconvenient or even dangerous if the diver is simultaneously coping with environmental problems such as white water, strong currents or tidal flows.

It is best **prevented** by maintaining a high level of physical fitness, using familiar and comfortable fins and having adequate insulation from cold water.

EAR PROBLEMS

Wax (Cerumen)

Ear wax (cerumen) is a protective substance which coats and waterproofs the external ear canal. Occasionally the ear produces excessive wax which accumulates and obstructs the ear canal, or contributes to water retention with subsequent otitis externa (see Chapter 28). It may produce curable hearing loss, or caloric induced vertigo if water is able to enter only one ear (see Chapter 31). Divers may try to remove this wax with cotton-tipped "buds", but unfortunately this often results in infection or the wax being compacted even tighter in the canal, precipitating total obstruction.

The excessive wax is easily removed by a diving physician using an instrument or syringe. This leaves the ear canal somewhat open to infection however (otitis externa - see Chapter 28), and therefore should not be done unless the wax totally occludes the canal. Ear drops are readily available (Cerumol, Waxsol, olive oil etc.) which help to soften wax so that the normal self cleaning function of the canal can proceed more easily. Diving itself aids in wax removal.

Exostoses

The inner part of the external ear canal passes through bone. People who swim or dive regularly, especially in cold water, sometimes develop outgrowths of this bone, known as exostoses, bulging into the ear canal. These can cause partial obstruction which may lead to the accumulation of wax and the retention of water causing infection or hearing loss.

Large troublesome exostoses can be removed surgically, however this is not usually necessary.

Others

Infections (otitis externa, otitis media) are discussed in Chapter 28, hearing loss in Chapter 30, vertigo and disorientation in Chapter 31, barotrauma in Chapter 9 and decompression sickness in Chapter 15.

HEADACHE

Headache during or after a dive is a frequent complaint and can be caused by conditions ranging from trivial to life threatening. It always requires careful assessment.

The most likely cause of the headache can usually be deduced from the past medical history, location of the pain, dive history, mode of onset and progression.

Details of clinical and diagnostic features can be found in the relevant chapters elsewhere. Although most headaches are not serious, the more serious causes will be discussed first.

Decompression Sickness and Pulmonary Barotrauma

Air emboli and bubble development in the brain can cause brain injury and swelling which often presents as headache. This may start within a short time after surfacing, or may be delayed for several hours. Headache followed by confusion or loss of consciousness is very suggestive of this dangerous disorder. The dive profile is helpful in diagnosing headaches of this type (see Chapters 11 and 15).

Sinus Barotrauma

This condition usually affects the various sinuses located around the eyes, or the maxillary sinuses in the cheek bones. Sharp pain in the affected sinus may be experienced during descent or ascent, or a more dull pain in the region of the sinus may be felt after the dive (see Chapter 10). A more serious and difficult-to-diagnose sinus headache can develop in the sphenoidal sinuses, a deep and central headache. The barotrauma headache is not usually long lasting.

Pain may be referred from the sinus to the upper teeth or behind the eyes. After minor barotrauma, an infection (**sinusitis**) can develop hours or days after the dive, causing a headache in similar sites to those mentioned (see Chapter 28).

Migraine

This condition can be a worrisome problem in divers. It is common in the general population.

Clinical features.

These may include an "aura" before the onset of the headache, with visual effects ranging from flashes of light, shimmering lines, partial loss of a visual field to mild blurring of vision. A severe headache aggravated by bright lights, usually accompanied by nausea and vomiting, and sometimes numbness, tingling, weakness or paralysis of the limbs, most often follows the visual aura.

Migraine headaches can be trivial or can be associated with vomiting, severe incapacity and neurological symptoms (visual disorders, numbness or 'tingling sensations' in arms or legs etc.). These more severe symptoms lead to diagnostic confusion with air embolism and decompression sickness and may result in an emergency evacuation and inappropriate treatment.

A severe migraine developing during a dive can incapacitate the diver or induce vomiting underwater with subsequent drowning.

For reasons which are not well understood, mild migraine sufferers can sometimes have very severe and unusual migraine attacks precipitated by diving. It may be that this is a response to bubbles within the cranial extravascular system. Migraine may also result from excessive exercise and carbon dioxide/oxygen pressure variations. Cold and exertion are also possible aggravating causes.

For these reasons migraine sufferers are not encouraged to dive. Nevertheless, some have no "neurological" features and are very infrequent and mild. Then if they do dive, they are usually **restricted to non-decompression dives** and to **less than 18 metres** and long surface intervals

(i.e. dives that do not typically produce intra-arterial bubbles or cerebral decompression sickness).

Tension Headache

Diving and training for diving can be a stressful experience which can cause headaches in susceptible individuals from excessive muscular tension. These individuals will often recognise the headache as similar to those associated with other stressful experiences. Most are frontal or involve the neck and back of the head.

Mask Strap Tension

Inexperienced divers often tighten their mask strap excessively in the hope that the alarming prospect of loss of the mask underwater, can be avoided. Excessive tension of this strap interferes with the blood supply to muscles around the skull, causing a headache similar to tension headache. The pain is prevented by slackening the strap. As the diver gains confidence in his ability to deal with a flooded or displaced face mask, the need to keep the strap excessively tight disappears. Some headaches are related to the design of the strap (ie. wide single straps verses narrow split straps.) Trial and error may sort out this type of problem.

Carbon Dioxide Toxicity

This is a frequent problem with re-breathing equipment, but is sometimes observed with scuba. It can develop as a consequence of a breathing resistance from faulty regulators, possibly from excessive depth (when the air is more dense) or, more likely, from a voluntary inhibition of breathing (slowed or shallow breathing, “skip breathing”) in an attempt by the diver to reduce his air consumption. This popular explanation still awaits experimental or clinical proof.

The headache is often severe, throbbing and unresponsive to analgesics. It may last for an hour or more. See Chapter 22.

Other Types of Headache

Cold water entering an ear canal can cause headache (or earache) when it comes into contact with the ear drum (see Chapter 27). This is easily prevented in swimmers with normal ear plugs. These cannot be used safely by divers. Special ear plugs designed for divers, with perforations, may work. The best prevention is a neoprene hood, which allows the trapped water to warm to body temperature. Some masks are designed to include the ears.

There are many **other causes** of headache, including neurological, barotraumatic, thermal, orthopaedic and vascular mechanisms, that are too complex to be assessed here. Any headache associated with diving deserves investigation, before its consequences during future diving become more serious than those occurring while on land. Exertional headaches cause particularly difficult diagnostic problems.

SUNBURN

Sunburn, especially in tropical areas, is a common problem for divers. It is caused by ultraviolet radiation from the sun. This radiation is scattered by the atmosphere and reflected from water so that even sheltering in shade does not provide complete protection.

The **clinical features** of sunburn have been experienced by almost all divers and do not require elaboration.

Treatment.

This is essentially symptomatic. Further exposure to sunlight (even indirectly) should be avoided. A soothing or cooling lotion is often of value in relieving the pain, and steroid (cortisone) creams may be beneficial in severe cases. Blisters should not be ruptured as this invites secondary infection.

Prevention.

Protection can be afforded by covering the skin by clothing, by wearing a hat and by the use of a broad spectrum UV screening cream or lotion. Snorkel divers are advised to wear one of the lightweight protective Lycra suits, which also give protection against marine stingers and coral cuts.

Ultraviolet screening agents are now coded by a SP number which gives an approximate indication of the degree of protection compared with unprotected skin e.g. SP 10 cream will protect the skin from burning for a period 10 times longer than unprotected skin. Unprotected skin can begin to burn in 15 minutes in strong sunlight so that a sun screen with this level of protection can be expected to protect for 2.5 hours if an adequate thickness is maintained and the screen is not washed off. SP 15+ creams are even more effective and are advised.

Prolonged exposure to sunlight is associated with an increased incidence of skin cancer and premature skin ageing.

SEASICKNESS

This is a distressing and potentially hazardous problem for divers. It usually develops in susceptible individuals in the dive boat but can also develop underwater, during decompression on a shot line, in rough conditions or with underwater surge. On the boat, less attention is paid by the sea sick diver to dive planning and equipment preparation.

The associated vomiting causes dehydration on the boat and requires considerable skill to cope with underwater, if the diver is to continue breathing through his demand valve. It does have the advantage of attracting all sorts of fish homing in for a free feed.

Another potential problem relates to the sedating effect which is produced to some degree by most of the available anti-seasickness medications. This will affect judgment and aggravate nitrogen narcosis.

Prevention.

General measures to be taken include:

- remain in the centre line of the boat, but not near the bow (reduce spatial movements),
- positioning in the boat so that head movement is minimised, remain still (lie down),
- either keeping eyes closed or focusing on the distant horizon (avoid reading),
- if in an enclosed cabin, ensure air circulation with a fan if possible.

If mildly seasick, swimming or snorkeling around on the surface of a sheltered area for a short while will often settle symptoms. The diver can then reboard the boat to don gear and start the dive.

Short acting anti-seasickness tablets such as cyclizine are effective if taken 1 or 2 hours before boarding the boat. These last about 4 hours.

Another effective preventative measure is to take promethazine tabs (a well known oral antihistamine), 25 mg. at bed time the night before. It will cause sedation during the night but this corresponds to the normal sleep time. One dose at night will provide some resistance to sea sickness for the early part of the following day, with minimum sedation. The **depth** of diving should be **limited to less than 30 metres** (100 ft.), maximum, and preferably less than 18 metres (60 ft.) if drugs are used. A cup of coffee (caffeine) beforehand reduces seasickness and counters sedation in some.

In all cases, medication should have been tried previously (a "dry run") to ensure adverse side effects are not produced. It should not be taken if alcohol has been consumed because of additive effects.

Transdermal ("Scop") skin patches are not recommended for diving due to side effects and variable absorption, but may be effective for sailors.

Acupuncture (via acupressure pads) and ginger, although currently fashionable, are really only of psychological value.

TEMPORO-MANDIBULAR (JAW JOINT) ARTHRITIS

Novice divers tend to be apprehensive underwater, especially about the reliability of their air supply. They therefore clamp their jaws tightly on the mouth piece, causing excessive stress on the joint between the upper and lower jaw. This can cause minor injury to the joint, manifested by spasm of the jaw muscles, pain, tenderness over the joint (in front of the ear), and inability to fully open the jaw.

In recreational divers this condition is usually temporary and is reversible by correcting the cause. The diver is encouraged to grip the demand valve less tightly with the jaws. Some older demand valves are heavy and bulky, placing undue stress on the jaw, while other types may be positioned so that the air hose pulls the jaw to one side, causing uneven and excessive strain.

In some older divers, permanent arthritic changes to the joint can occur, from this cause. Individually mouldable lugs on the mouthpieces of snorkels and regulators may help minimize these effects in some cases.

EXPLOSIONS - UNDERWATER BLAST



Fig. 32.1

are in contact with air - all gas containing spaces within the body can be affected.

This topic is included only as a warning for recreational divers not to use explosives underwater. Military divers are particularly at risk from these hazards, even in training - because of the use of "scare charges" which are designed to discourage underwater saboteurs and are sometimes used in the vicinity of trainee divers to toughen them up.

When an underwater explosion is observed from the surface, a sudden explosive projection of water and foam into the air can be seen immediately after the explosion. This is the effect of the pressure wave emanating from the blast when it meets an air-water interface.

A similar effect is produced at air-tissue interfaces in the body as the shock wave travels through the diver. This can shred tissues such as lungs, intestines, sinus cavities and the middle ear spaces, which

Clinical features.

The organs worst affected are the lungs and intestines. Rupture and bleeding of the tissues in the lungs and bowel cause:

- chest pain
- shortness of breath
- vomiting or coughing up of blood
- passage of bloody or black bowel motions.

Damage to the ears and sinuses causes features similar to barotrauma. Ruptured ear drums and deafness are particularly common.

If a diver is caught in the water where an explosion is inevitable, some protection can be afforded by attempting to float on his back, on the surface - this will remove some of the air containing tissues from contact with the water.

Chapter 33

UNCONSCIOUSNESS IN DIVERS

There are many causes of a diver losing consciousness in the water but the final outcome is very often the same – **drowning**. This chapter provides an overview of the underlying causes and basic treatment.

Unconsciousness on land rarely leads to death. Underwater, it frequently does. Because of the hazardous nature of diving in a state of impaired consciousness, great care must be applied to ensuring divers are medically fit and have no increased propensity to loss of consciousness. Also, once consciousness is lost, the adherence to a genuine "buddy system" is of demonstrable value.

When an unconscious diver is rescued and the first-aid measures necessary for all these cases are then instituted (see Chapter 39, 40 and 42), the remainder of the management depends on the cause of the unconsciousness. It is therefore important to be able to identify the likely causes.

They are best classified according to the type of diving being performed, and the equipment used. More information can be found on each topic elsewhere in this book.

CAUSES OF LOSS OF CONSCIOUSNESS

The causes **common to all types of diving** are:

- Hypoxia (from a diversity of causes)
- Salt water aspiration or near drowning
- Cold
- Marine animal injuries
- Vomiting and inhalation of vomit or sea-water
- Underwater explosions
- Miscellaneous medical conditions

In addition to these the causes associated with **scuba diving** are:

- Hypocapnea
- Decompression sickness
- Air embolism from pulmonary barotrauma
- Nitrogen narcosis
- Carbon monoxide toxicity
- Hypoxia due to faulty equipment or gas contamination

In addition to these, causes associated with **rebreathing** or **mixed gas diving equipment** (not commonly used by recreational divers) include :

- Hypercapnea
- Oxygen toxicity
- Hypoxia due to ascent, dilution or excessive consumption.

The more **common causes** of unconsciousness are as follows:

<p style="text-align: center;">HYPOXIA (SEE CHAPTER 20)</p>
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Hypoxia of the brain associated with near-drowning is the final event in many diving accidents and is the most common cause of unconsciousness in divers. It may follow events as diverse as breath-holding in free diving, inadequate air supply, salt water aspiration, equipment faults or misuse, inhalation of vomit, pulmonary barotrauma, gas contamination, etc. It is frequently associated with panic and physical exhaustion.

Case History 33.1 A diver breathing from a semi-closed breathing apparatus lost consciousness shortly after leaving the surface. He was brought back to the surface and revived with 100% oxygen. His slightly bluish face turned red later on when it was discovered that he had filled his cylinders with pure nitrogen.

Diagnosis: Hypoxia due to inadequate (i.e. nil) inspired oxygen.

HYPOXIA DURING BREATHHOLD DIVING

(SEE CHAPTER 4)

□ Hyperventilation.

Hyperventilation before a breath-hold dive makes hypoxia more likely because the urge to breathe is suppressed. This technique was used by some divers to increase the duration of dives. The practice is gradually dying out, along with the divers who use it.

□ Hypoxia of ascent.

A diver can lose consciousness from hypoxia of ascent during deep breath-hold dives. The partial pressure of O₂ in the lungs can fall dangerously low during ascent or immediately after surfacing.

Case Report 33.2. A young diver was attempting to beat a local swimming pool underwater distance record. He was seen to hyperventilate before the dive. Midway through the second lap, he ceased to swim and sank to the bottom. Luckily, he was quickly pulled from the water and revived by mouth to mouth respiration. He was discharged some weeks later, with permanent brain damage.

Case Report 33.3. An experienced diver taking part in a spear fishing competition was found dead on the bottom with a speared fish nearby. Post-mortem revealed no abnormality apart from drowning. He was known to practise hyperventilation, push himself to the limit and to dive deep.

NEAR DROWNING

(SEE CHAPTER 25)

This is the consequence of many diving accidents. The hypoxia associated with near drowning can render a diver unconscious, or the diver can become unconscious first, and then drown.

COLD OR HYPOTHERMIA

(SEE CHAPTERS 3, 27 AND 35)

Exposure to cold water can cause an progressive and excessive fall in body temperature which can make a diver initially confused (at a body temperature of around 34°C) and then unconscious (below 30°C). A diver suddenly entering cold water can sometimes develop a hypertensive spike or cardiac rhythm disturbances which can produce immediate unconsciousness from cardiac or cerebral accidents (heart attack or stroke).

MARINE ANIMAL INJURIES

(SEE CHAPTER 29)

Venomous animals can cause unconsciousness either from the direct effect of the venom on the brain, from hypoxia due to respiratory paralysis, or due to inadequate cerebral circulation from a lowering of blood pressure. Shock from blood loss after shark attack can also cause unconsciousness.

Case History 33.4. A group of divers on their first dive on a tropical reef eagerly took to the water. They returned later with speared fish, coral and shells among which were several varieties of venomous cone shells which they had handled, and in some cases, carried under their wet suits. A member of the boat crew recognised the cone shells and advised the divers of their narrow escape.

Diagnosis: potential loss of consciousness, or "accidents looking for somewhere to happen".

DECOMPRESSION SICKNESS

(SEE CHAPTERS 14–16)

Cerebral Decompression Sickness can lead to unconsciousness. It is more likely after deep dives and repetitive diving.

Case Report 33.5. A 26 year old diver undertook a 55 metre (180 foot) dive with a bottom time of 8 minutes. He returned rapidly to the surface when his contents gauge indicated an almost exhausted air supply. While climbing into the boat, he complained of numbness down one side, and developed slurred speech. He then had a convulsion and lost consciousness. He died en route to a recompression chamber 600 km. away.

Diagnosis: the probable diagnoses would include cerebral decompression sickness or air embolism (CAGE) from pulmonary barotrauma (burst lung). Autopsy verified the diagnosis.

**AIR EMBOLISM FROM
PULMONARY BAROTRAUMA
OF ASCENT**
(SEE CHAPTER 11)

Cerebral arterial gas (air) embolism (CAGE) can arise either during or soon after ascent, from any depth. It can cause abrupt loss of consciousness. It is sometimes associated with pneumothorax, which needs special management.

**CARBON MONOXIDE
TOXICITY**
(SEE CHAPTER 23)

Divers breathing compressed air are vulnerable to this problem if the air source is contaminated, often from the exhaust of a nearby internal combustion engine.

OXYGEN TOXICITY
(SEE CHAPTER 21)

Military divers using oxygen equipment, technical or professional divers breathing mixed gases are at risk from convulsions due to oxygen toxicity under certain circumstances. A convulsion can be followed by unconsciousness and confusion.

GENERAL MEDICAL CONDITIONS

A variety of medical emergencies including hypoglycaemia (low blood sugar) in diabetics, heart attack, stroke, epileptic fit, drug overdose, head injury, severe infection and shock can cause unconsciousness. Many conditions with the potential to cause unconsciousness require exclusion in a diving medical examination and people with these conditions would normally be advised against diving.

Case Report 33.6. A 30 year old diver using scuba at 10 metres (33 foot) became unconscious ten minutes after the start of the dive while swimming strenuously on the bottom. He was brought rapidly to the surface by his buddy. He remained unconscious on the boat and was pale and sweaty with a rapid pulse. He was breathing adequately and 100% Oxygen breathing on a mask produced no improvement. Those present were at a loss for a diagnosis until the diver's wife informed them that he was a diabetic taking insulin. He was successfully treated in hospital by intravenous glucose.

Diagnosis: Hypoglycaemia (low blood sugar level) due to unexpected exertion, even though the diver took a reduced Insulin dosage. He was advised that diving and diabetes requiring medication was a suicidal combination and he agreed to take up a sport less dangerous to diabetics

RESCUE AND FIRST AID TREATMENT

(SEE CHAPTERS 39–42)

First **ditch the diver's weight belt**. The **diver must be brought to the surface and then removed from the water as rapidly** as possible. Emergency buoyant ascent takes precedence over concern for burst lung (pulmonary barotrauma) when the recreational diver is unconscious. These unconscious divers will usually exhale passively during ascent as the lung volume expands.

After surfacing the airway must be cleared and if there is no respiration, expired air resuscitation commenced. Details of the rescue and resuscitation technique are explained in Chapters 39 and 42. After securing the essential **A–B–C** (airway, breathing and circulation) the cause must be sought and **specific treatment** started. Diving medical advice should obviously be sought as soon as possible.

Diagnosis of the cause is made by a logical process of elimination, taking into account the medical history of the diver, the equipment used, the type of diving, the dive profile, the events leading to the unconsciousness and the appearance of the diver. Assume the most serious and treatable diagnosis.

Contact medical authorities (see Appendices A and B) with full details (see Check List Chapter 39) and follow their advice. Transport may be required to a medical facility or formal medevac may be instituted. Divers may be made worse by transport in certain circumstances (shark attack, decompression sickness, pulmonary barotrauma, etc.) and so this decision is best left to experienced diving physicians cognisant with the prevailing circumstances. If aviation transport is employed, the type of aircraft and its ability to be pressurised, is also to be considered.

While waiting for assistance, administer 100% O₂, if needed or if in any doubt about the diagnosis.

Keep detailed records and ensure that these accompany the patient. Retain the equipment for future assessment.

Chapter 34

WHY DIVERS DIE

INTRODUCTION

Experience of life suggests that anything which is fun tends to be either illegal, immoral, fattening, or dangerous. Recreational diving partly conforms to this universal law, ranking below hang gliding and parachuting but above most sports as regards the risk of a fatal accident.

Diving statistics from the USA, UK, Canada and Japan all show diving death rates of 15–30 per 100,000 divers per year, with the statistical chance of a fatality being about 2-3 per 100,000 dives.

These figures tend to contradict the misinformation issuing from some sections of the diving industry (fatalities of < 4 per 100,000 divers) which would have us believe that diving is a very safe recreation. It is not, but then we accept risks every day. Even driving an automobile to a dive site carries an appreciable (but much less) risk of death - a possibility which we generally regard with equanimity.

This chapter will show that many diving deaths should be preventable and that a diver ought to be able to minimise his chances of becoming a statistic by understanding and influencing the factors which are now known to be associated with diving deaths.

STATISTICAL EVIDENCE

The information presented here is mainly based on data gathered by valuable studies involving recreational diving fatalities. They have been conducted in different countries, but show strikingly similar results. The USA recreational diving deaths, originally compiled by John McAniff of the University of Rhode Island and then NUADC, are now collected and reported on by DAN, which recently analysed 947 open circuit scuba divers. The DAN survey also included technical divers, who dive deeper, longer and with gases other than

compressed air. The BSAC do a similar job in the UK and Project Stickybeak is the Australian compiler. Unfortunately significant data is frequently not available and so relevant causal factors are often underestimated. Another Australian approach (the ANZ series of diving fatalities) was to select and analyse only the accidents in which sufficient data was available to make the analysis credible, and to determine what factors materially contributed to the fatality. Most of our statistics come from this source and are rounded up, for simplicity.

OVERVIEW

❑ Diving Fatality Data

- 90% died with their **weight belt on**.
- 86% were **alone** when they died.
- 50% did **not inflate their buoyancy** vest.
- 25% encountered their difficulty first on the **surface**,
50% actually died on the surface.
- 10% were **under training** when they died.
- 10% were advised that they were **medically unfit** to dive.
- 5% were cave diving.
- 1% of “rescuers” became a victim.

❑ Age.

The recorded deaths range from children (pre-teens) to septuagenarians. Some decades ago the average age of the deceased was in the early 20s. Then there developed a small increase in the middle ages (45-60 years). This bimodal curve has now become distorted on the other side, and the average scuba death age is now 43 years. The reasons for this increasing age of death are:

- The “youngsters” from the 1970-80 scuba diving boom are now older
- Cardiac disease, the sudden death syndrome, affects the elderly and diving introduces more cardiac hazards than many other sporting activities
- Diving is becoming a life-style option for the increasingly active and affluent elderly, with more older people taking up this sport

❑ Gender.

In the 1990s 1 in 10 of the fatalities were women. The actual percentage of women in the overall diving population was about 1 in 3, suggesting that women are safer divers than men. Even now females account for only 20% of the deaths.

❑ Diving Experience.

In most series, 1/3 were inexperienced, 1/3 had moderate experience and 1/3 had considerable experience. The most dangerous dives were the first dive and the first open water dive. In half the cases the victim, based on witness statements and previously logged dives, was extending his diving experience (depth, duration, environment, equipment etc.) and thus did not have the experience to undertake the final dive. For this reason, any diver extending any of his dive parameters (depths, durations, environments, equipment) is advised to do this only with more experienced supervisors.

❑ Major Causes of Death identified at Autopsy.

According to death certificates, most divers ultimately drowned (over 80%), but a number of factors usually combined to incapacitate the diver before this terminal event. Drowning is

really only the final act in a sequence of events that lead up to this. It is a reflection of the medium in which the accident happens, more than the accident itself. Often it obscures the real cause of death. Unless there are other factors, drowning should never happen to a scuba diver, as he carries his own personal air supply with him! Drowning develops because of preceding problems, such as cardiac disease, pulmonary barotrauma, the stress disorders, unconsciousness from any cause, salt water aspiration, trauma, equipment difficulties or environmental hazards, etc. These are referred to in the following sections and in other chapters.

CONTRIBUTING FACTORS

Deaths usually followed a combination of difficulties, which alone may have been survivable. The factors contributing to deaths are easier to understand when classified, and we have categorised them into the following groups:

- **Diving Techniques** (Inadequate air supply, buoyancy, buddy system)
- **Human Factors** (medical, physiological, psychological)
- **Equipment Factors** (misuse, faults)
- **Environmental Factors.**

DIVING TECHNIQUES

Inadequate Air Supply

In the ANZ survey in **half the deaths (56%), critical events developed when the diver was either running low or was out-of-air (LOA, OOA)**. When equipment was tested following death, few victims had an ample air supply remaining. The DAN survey found 41% in this situation.

Most problems arose when the diver became aware of a low-on-air (LOA) situation. Some divers then died while trying to snorkel on the surface, attempting to conserve air (8%).

Concern about a shortage of air presumably impairs the diver's ability to cope with a second problem developing during the dive, or causes the diver to surface prematurely and in a stressed state of mind, where he is then unable to cope with surface conditions. In many cases the LOA diver faced these difficulties alone, as his buddy who had more air, continued the dive oblivious to the deteriorating situation (see later). LOA situations should be avoidable by adequate dive planning, using a cylinder with ample capacity for the planned dive, and frequent observation of the contents gauge.

A particularly dangerous technique was to intentionally use all the available air (breathing the tank dry). Then there is much less opportunity to cope with unexpected eventualities and greater likelihood of emergency ascent and salt water aspiration. The dive should always be completed with at least 50 ATA remaining.

A description of the methods of preventing and coping with an OOA and LOA situation is in Chapter 11.



Fig. 34.1

In some cases the diver was using a smaller cylinder than the usual 2000 litre (72 cu.ft). A 1400 litre (50 cu.ft) cylinder has much less endurance than a conventional cylinder, and allows fewer breaths once a LOA situation develops at a significant depth. Also, a diver using a smaller cylinder will usually run out of air sooner, encouraging separation from his group.

Buoyancy

In the ANZ survey, half **the diving victims (52%) encountered buoyancy problems**. Most of these were due to inadequate buoyancy, but some (8%) had excessive buoyancy. The DAN survey buoyancy problems were the commonest adverse event leading to death.

The **buoyancy changes peculiar to wet suits** were a significant factor. The considerable buoyancy offered by a wet suit at the surface needs to be compensated by weights. An approximate formula for this is:

- 1 kg for each 1 mm thickness,
- 1 kg for "long john" extensions and a hood,
- 1 kg for an aluminium tank,
- \pm 1–2 kg for individual body variations in buoyancy.

Based on the above formula, **40% of divers who perished were found to be grossly overweighted** at the surface. This factor would have been greater at depth. When weighted according to this formula, a diver should be neutrally buoyant at or near the surface. In this state, descent or ascent are equally easy.

During descent, the wet suit becomes compressed, making the diver negatively buoyant. This is where the **buoyancy compensator (B.C.)** comes in. It is inflated just sufficiently to restore neutral buoyancy. This is why it is called a buoyancy compensator.

Evidently, **some divers deliberately overweighted on the surface**, using this excess weight to descend more easily and were then using the B.C. to maintain depth and then later to return to the surface. This places excessive reliance on the B.C.. This dangerous practice is unfortunately promoted by some instructors. It has advantages from a commercial point of view, as it expedites training. Groups of divers can be quickly taught to descend with minimum skill. The technique is less advantageous in terms of longevity of the diver.

In another fatality survey on buddied divers who ran into LOA/OOA situations, it was of interest that irrespective of who became OOA first, the overweighted diver was the one who died – at a 6:1 ratio. See Chapter 5, dealing with weights, buoyancy compensators, etc.

In spite of being heavily reliant on their B.C., many divers then misused them. Examples of this include accidental inflation or over-inflation causing rocket like ascents ("Polaris missile effect"), confusion between the inflation and dump valves, and inadequate or slow inflation due to being deep or LOA. The drag induced by the inflated B.C. (needed in many cases to offset the non-discarded weight belt) was a factor contributing to exhaustion in divers attempting to swim to safety on the surface.

There are other unpleasant consequences of buoyancy problems. The American Academy of Underwater Sciences, in a symposium in 1989, reported that **half the cases of decompression sickness were related to loss of buoyancy control**. After acquiring the initial open-water certificate, possibly the best course to undertake would be on buoyancy control.

Ditching of Weights

This was omitted by most victims (90%). This compelled them to swim towards safety carrying many kilos of unnecessary weight, and made staying on the surface very difficult in these cases. This critical and avoidable factor should be easily remedied by restoring the traditional weight belt ditching drills.

Earlier diving instructors taught that the weight belt was the last item put on, the first taken off. It was to be removed and held at arm's length in the event of a potential problem. The diver then had the option of voluntarily dropping the belt if the situation deteriorated, or replacing it if the problem resolved. When problems did develop, the belt was dropped automatically! Some current diving students now question the validity of dropping these lead (? dead) belts – perhaps the high cost of replacement is worth more than their lives. "Lead poisoning" is a frequent contribution to fatalities.

When ditched, the belt is held at arms length to avoid falling and fouling on other equipment. This entanglement occurred in some of the reported fatalities. In other cases, the belt could not be released because it was worn under other equipment (e.g. B.C., backpack harness, scuba cylinder etc.), or the release buckle was inaccessible because a weight had slid over it, or it had rotated to the back of the body. In some cases the belt strap was too long to slide through the release buckle. Other fatalities have occurred where release mechanisms have failed, due to the use of knotted belts (which could not be untied), or lead balls contained within a backpack.

In an emergency requiring either ascent or buoyancy, to keep the diver afloat on the surface, several kilograms of flotation are immediately available by simply discarding the weight belt. This action also results in a more consistent, controlled ascent than with an inflated B.C..

Buddy Diving System

The value and desirability of the buddy system is universally accepted in the recreational diving community. Two maxims have arisen in diving folklore from this concept:

- "Dive alone – die alone"
- "Buddies who are not in constant and direct communication are not buddies, — merely diving in the same ocean".

In spite of this, **only 14% of divers who perished still had their buddy with them**, and in the Hawaiian series it was 19%. In 33% of the ANZ cases, the deceased diver either dived alone or voluntarily separated from his buddy beforehand, 25% left their buddy after a problem developed, and 20% became separated by the problem. Of those who started diving with a buddy in the DAN series, 57% were separated at the time of death.

A common cause of separation was one diver (the subsequent casualty) having inadequate air, OOA or LOA. In this case, the buddy often continued the dive alone, or accompanied the victim to the surface, before abandoning him and continuing the dive.

There were many misapplications of the buddy system. In some cases more than two divers 'buddied' together, leading to confusion as to who was responsible for whom. A particular variant of this is a training technique in which a group of inexperienced divers follows a dive leader. When one becomes LOA, he is paired with another (usually another inexperienced diver) in the same situation, and the two instructed to return to the surface together. Often the heaviest air consumers are the least experienced and are over-breathing through anxiety. Two such inexperienced, anxious divers, both critically low on air, are then abandoned underwater by the dive leader and left to fend for themselves!

In others, the buddy was leading the victim and therefore not immediately aware of the problem. Generally, the more experienced diver took the lead, affording him the luxury of constant observation by his buddy, while he gave intermittent attention in return. In this situation, unless a "**buddy line**" is used, the following diver (upon developing a problem such as LOA or OOA) has to expend precious time and energy and air, catching his buddy to inform him of the difficulty. Often this was impossible, and the first indication the leading diver had of the problem was the absence of his buddy, who by this time was unconscious on the sea bed or well on the way to the surface.

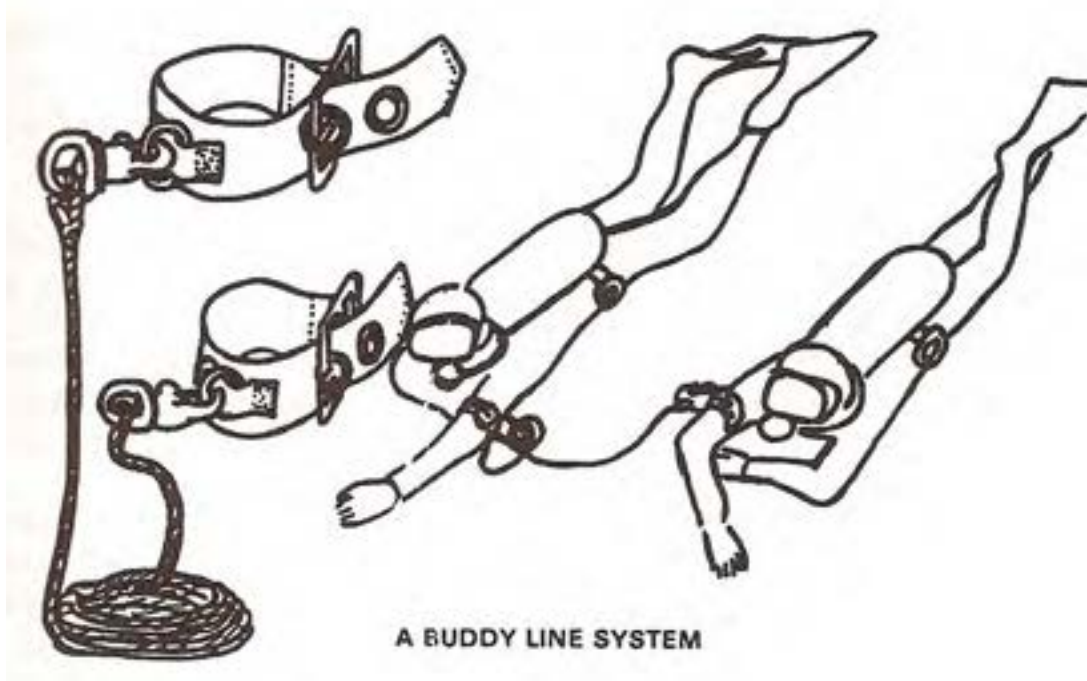


Fig. 34.2
A buddy line may be life saving



Fig 34.3
But not always

❑ Buddy rescue.

In only a minority of cases was the buddy present at the time of death. Most divers ultimately died alone, usually because of poor compliance with the principles of buddy diving. In only 1% of cases did the buddy die attempting rescue, indicating that adherence to the buddy principle is reasonably safe for the would-be rescuer.

❑ Buddy breathing.

4% of fatalities were associated with failed buddy breathing. In a study of failed buddy breathing conducted by NUADC, more than half were attempted at depths greater than 20 metres. In 29% the victim's mask was displaced and the catastrophe of air embolism occurred in 12.5% of cases.

One in 8 victims refused to return the demand valve, presumably to the righteous indignation of the donor. In one reported instance, knives were drawn to settle the dispute! Nevertheless, donating a regulator rarely results in the donor becoming the victim.

The use of an **octopus rig** or (more sensibly) a complete **separate emergency air supply** (e.g. "Spare Air") would appear to be a more satisfactory alternative, having the added advantage of providing a spare regulator for the owner in the (not so rare) event of a failure of the primary air supply.

HUMAN FACTORS MEDICAL, PSYCHOLOGICAL AND PHYSIOLOGICAL

In at least **25% of cases, the diver had a pre-existing disease which should have excluded him from diving** (compared to 8-10% in the potential diver trainee population). The diseases either killed the diver or predisposed him to the diving accident.

In assessing the cause of scuba fatalities, it is too easy to ignore the disorders which have no demonstrable pathology, such as panic and fatigue, but to do so results in less understanding of the incident. Drowning obscures many other pathologies and some, such as asthma or the sudden death syndrome, may not show up at autopsy.

Panic

39% of deaths were associated with panic. Panic is a psychological stress reaction of extreme anxiety, characterised by frenzied and irrational behaviour. It is an unhelpful response which reduces the chance of survival. This topic is covered in detail in Chapter 7.

Evidence of panic was derived from witness accounts of the diver's behaviour, in the Australasian series. Other studies suggest a 40–60% incidence of panic.

Panic was usually precipitated when the diver was confronted by unfamiliar or threatening circumstances such as LOA, OOA, poor visibility, turbulent water, unaccustomed depth, buoyancy problems (usually insufficient buoyancy), or separation from diving companions.

After panicking, the diver frequently behaved inappropriately by actions such as failure to ditch weights or inflate the B.C., rapid ascent, or abandoning essential equipment such as the mask, snorkel and regulator.

Fatigue

In 28% of cases fatigue was a factor. Fatigue is a consequence of excessive exertion, and limits the diver's capacity for survival. Physical unfitness aggravates it.

It commonly arose from a variety of circumstances including attempting to remain on the surface while overweighted, long swims in adverse sea conditions or swimming with excessive drag from an inflated B.C..

The fatigue factor was not restricted to unfit divers — under special circumstances any diver will become fatigued. In some cases the fatigue was associated with salt water aspiration syndrome, cardiac complications or asthma.

Salt Water Aspiration

This factor was present in 37% of cases. It refers to inhalation of small amounts of sea water by the conscious diver.

In many cases this was the result of; a leaking regulator, aspiration on the surface after removing the regulator, and buddy breathing. In most cases salt water aspiration was a pre-terminal event as the situation became critical. It frequently predisposed to the development of panic, fatigue, respiratory and other complications.

Pulmonary Barotrauma

13% of deaths had autopsy evidence of pulmonary barotrauma (burst lung). In some cases it was a complicating factor rather than the initial cause. Factors promoting the barotrauma were diverse, including panic, rapid buoyant ascents, asthma and regulator failure. Half the cases had an identified cause for the illness. The other half were unexplained.

Cardiac (Sudden Death Syndrome)

In these cases there was either gross cardiac pathology or a clinical indication of cardiac disease (See Chapter 35). In the DAN series, 26% of deaths were due to this. Of the cardiac deaths, 60% complained of chest pain, dyspnoea or feeling unwell before or during the dive.

Victims tend to be older – cardiac causes explain 45% of the scuba deaths in those over 40 years. They tend to be more experienced divers, often with a history of known cardiac disease (arrhythmias or ischaemia) or high blood pressure - often under control with medication (especially beta blockers).

They usually die quietly and the pathophysiology is probably a cardiac arrhythmia (ventricular fibrillation). Resuscitation is difficult or impossible under these environmental conditions. The trigger factors producing this very rapid ineffective heart beat include the following; exercise, drugs, hypoxia from salt water aspiration, respiratory abnormalities from breathing under dysbaric conditions through a regulator, cardio-respiratory reflexes and cold exposure.

Asthma

In at least 9% of deaths the diver was asthmatic in the ANZ survey, and in at least 8% of cases asthma contributed to the death. In some other surveys (especially those with less data on each fatality, or those that do not specifically check the previous medical history), this data is not so obvious.

Asthmatics should normally be excluded by a competent medical examination. Even so, surveys have shown that between 0.5 and 1% of divers are current asthmatics. When this figure is contrasted with the 9% of fatalities who have the condition, it implies that **asthma is a significant risk factor.**

There was often a series of adverse contributors to death in this group, including panic, fatigue and salt water aspiration. The ultimate pathology was usually drowning or pulmonary barotrauma.

The risk of pulmonary barotrauma is predictable, considering that asthma narrows and obstructs airways. Added to this is the possibility of an incapacitating asthmatic attack during the dive. A considerable number of divers in the survey died this way, some as they were returning to get their medication (aerosol inhalers). Others took it before the dive!

The diving environment can aggravate asthma in several ways:

❑ Salt water aspiration.

Respiratory physicians use nebulised salt water to provoke an asthmatic attack in cases of questionable asthma. Divers immerse themselves in such a solution and often breathe a fine mist of seawater through regulators.

❑ Cold dry air.

Breathing this air precipitates attacks in some asthmatics. Divers breathe this type of air continuously. It is carefully dried by the filling station before being used to fill scuba tanks, and cools as it expands in the regulator.

❑ Exertion.

This aggravates many attacks. Even the most routine dive can require unexpected and extreme exertion, due to adverse environmental factors such as rough water or currents.

❑ Hyperventilation.

The effects of anxiety cause hyperventilation and changes in respiratory gases. This will have little effect on normal lungs. It provokes asthma in those susceptible.

❑ Breathing against a resistance.

Many of the cases first notice problems at depth, where the air is more dense, or if there is increased resistance in the regulator – such as with a LOA or OOA situation.

A study from Denver showed that although normal divers did not show any change in respiratory function with exercise or breathing through scuba regulators, asthmatics had decreases of 15% and 27% respectively.

Vomiting

Apart from the cases that vomited during resuscitation – and there were many – in 10% vomiting initiated or contributed to the accident. It was often produced by sea sickness or salt water aspiration, but ear problems and alcohol over-ingestion also contributed.

Nitrogen Narcosis

This was an effect of depth, and contributed in 9%, but was never the sole cause of death in the ANZ series.

Respiratory Disease

A further 7% of casualties had chronic bronchitis, pleural adhesions, chest injury or other respiratory conditions. Because divers with these conditions are in a minority, they appear to be over represented in the deaths.

Drugs

Alcohol and cannabis (marijuana) are well known contributors to drowning. Cocaine is an established cause of sudden death in athletes. What surprised us was the apparent association between drugs taken for hypertension and the deaths from the sudden death syndrome. Anti-asthma drugs seemed to have the same association.

Decompression Sickness

The dread of DCS is prominent in the minds of most divers. Perhaps this is why there are no deaths due to this condition in the ANZ studies, and less than 1% in the NUADC. Hawaiians reached 4%, due to deep diving for black coral. The DAN survey has 2.5%, probably because of the inclusion of technical divers, who often dive deeper – the mean depth being 68 metres (226 ft) in that study.

While DCS is an important cause of serious disability (such as paraplegia) in all divers, it is not a frequent cause of mortality in recreational divers. This is not, however, true for professionals.

EQUIPMENT PROBLEMS

A significant proportion of deaths were associated with equipment malfunction (35%) or misuse (35%). There was some overlap in the equipment faults and the equipment misuse categories. In spite of the advanced technology available, modern equipment still frequently fails and divers need to be prepared for this possibility (see Chapter 5).

Regulator

In 14% of deaths there was a regulator fault, and in 1% it was misused. Subsequent testing of the regulators showed the majority of problems were due to leakage allowing inhalation of salt water, but in some cases there was excessive breathing resistance following a mechanical dysfunction. In a few cases, the regulator failed catastrophically, or the hose 'blew out'.

The difficulty of obtaining useable air from the regulator was often complicated by other factors such as panic or exhaustion.

Fins

13% of cases lost one or both fins. In some cases this was due to defective or ill fitting fins, but in the majority of instances the cause was not obvious.

A likely explanation is that the fin(s) was lost because of vigorous swimming efforts during attempts to stay afloat with inadequate buoyancy, or during an attempt to swim to safety. Once a fin is lost swimming efficiency is drastically impaired. Panic and fatigue probably had a significant role in these situations.

Buoyancy Compensator

In 8% of cases the B.C. malfunctioned. Usually this was due to failure of the inflation system, but some B.C.s did not remain inflated.

In 6% of deaths, the B.C. was misused. Some divers confused the inflation and dump valves, usually causing over-inflation of the B.C. and precipitating an uncontrolled ascent. Others pressed the wrong button and sank when they wanted to float.

Scuba Cylinder

12% of deaths had problems with the cylinder, usually from misuse. These included under-filling, using a cylinder too small for the dive, the cylinder being dislodged from its harness, and failure to turn on the cylinder valve.

Other Equipment Problems

In 5% or less of deaths, problems were experienced due to failure or misuse of:

- **weight belt** – usually inability to discard it (see Chapter 5)
- **harness** – design faults or covering the weight belt
- **mask** – loss, flooding, and broken straps
- **protective suit** – ill fitting, usually too tight
- **lines** – entanglement
- **gauges** – faulty readings, blow offs

ENVIRONMENTAL PROBLEMS

Environmental factors contributed to 62% of deaths (see Chapter 6).

Deaths near the Surface

25% of the accidents commenced on the surface, and 50% of the divers died at the surface. This may seem surprising as most divers would regard the surface as a safety zone. In many cases they were compelled to surface because of exhaustion of the air supply.

Turbulent (White) Water

Difficult water conditions caused problems in 36%. These included excessive **current**, **rough** water, **surf** and **surge** around rocks, **underwater surge** from wave movement, and impaired **visibility** caused by these conditions.

These unfavourable conditions often assailed the diver who was forced prematurely to the surface, OOA or LOA, and who was also frequently overweighted and hampered by the drag of his inflated B.C.. Exhaustion or panic then resulted in drowning.

Depth

Excessive depth was a factor in 12%. Often the fatal dive was the deepest ever for the victim. Deep water is a more gloomy and dangerous environment.

The dangers of excessive depth are predictable. They include; increased air consumption, impaired judgment from nitrogen narcosis, colder water, reduced visibility, slow or failed response to B.C. inflation, excessive air consumption, resistance to breathing, and a prolonged ascent in the event of problems.

Other Environmental Problems

Factors which contributed to less than 10% of fatalities included:

- **cave** dives – sometimes causing multiple deaths
- **marine animal injury** – including shark and other animal bites, marine stings (3–6%)
- **difficulties entering and exiting** the water
- **cold**
- **entanglements** with ropes, lines and kelp
- **entrapment** – under caves, ledges, or boats
- **night** diving.

DEATHS IN PROFESSIONAL DIVERS

Professional divers have a much higher death rate than recreational divers, especially when operating from deep sea oil rigs. Death rates up to 4.8 per thousand divers per year have been reported. Technical divers, likewise, are more vulnerable.

The causes of death are different from recreational divers. DCS and CAGE account for up to 28% of deaths. These divers not only frequently develop DCS, but sometimes die from the disease.

Because of the inhospitable environment in areas like the North Sea, cold and heavy seas were a significant factor in deaths, as was increased depth and duration in technical diving. Enclosed diving, such as in caves, salvage, wrecks, and under ice, were also hazards to which the recreational diver is not usually exposed.

Other important factors were equipment failure (saturation divers are highly dependent on equipment integrity for their survival), and the use of more complex gas mixtures and equipment.

Surprisingly, in spite of legislation requiring careful medical supervision, 6% of deaths had a contributing medical factor.

SUMMARY

Diving fatalities generally arise from a combination of factors, none of which alone would have caused disaster.

The contributing factors show an emerging pattern which needs to be addressed by diver education and training. For example, the majority of deaths were in divers who were medically unfit to dive or had a **LOA or OOA element**.

These should be largely preventable by **adequate dive medical examinations** prior to commencing diving, and proper planning and air supply monitoring. Most of these were preventable.

Case Report 34.1 A composite diving fatality might unfold as follows:

A young, inexperienced, slightly overconfident, indifferently trained, male diver undertakes a dive in open water under conditions with which he is relatively unfamiliar. He is healthy but does no regular exercise apart from occasional diving. He has a vague dive plan which he does not discuss with his equally casual buddy. He is mildly anxious because of the unfamiliar conditions. He follows his usual practice of using a generous number of weights, initially inflating and then deflating his B.C. on the surface, to allow his weights to help him descend. Fascination with the environment leads him and his buddy to descend to 40 metres, deeper than they originally intended. He checks his contents gauge and is alarmed to find he is close to his reserve. His anxiety is increased by the realisation that there may be a decompression requirement for this dive, but he may have insufficient air to complete even a safety stop. He is unsure of the decompression requirement, if any, and he did not bring any tables with him. He had not chosen any of the more conservative options on his decompression meter.

He activates the inflation valve on his B.C. but gets so little response that he swims for the surface. He heads for the surface alone with some urgency, unable to communicate with his buddy who is some distance away and preoccupied with other marine life. His air supply runs out during the ascent and he arrives at the surface in a state of panic.

He has extreme difficulty staying afloat but in his frenzied state, neglects to ditch his weight belt or orally inflate his B.C.. His predicament is aggravated by inhalation of sea water and the loss of one of his fins. He becomes exhausted trying to remain on the surface, because of his negative buoyancy and reduced propulsion.

A search team later found his body on the bottom – directly below where he surfaced. They have difficulty in surfacing the body, until they release the weight belt.

The most significant factors in recreational diving fatalities are:

- **diving with disqualifying medical conditions**
- **panic**
- **fatigue**
- **water movement**
- **buoyancy problems**
- **LOA or OOA**
- **adverse sea conditions**
- **failure to ditch the weight belt when in difficulty**
- **ignoring or misapplying the buddy system**
- **improper use of equipment**
- **failure of equipment.**

PREVENTION

Many of the factors associated with diving deaths are avoidable.

Some changes in the emphasis of diver instruction, aimed at better education concerning the high risk areas of diving, would be helpful. Divers who are extremely knowledgeable of decompression theory and practice, are running out of air and drowning in solitude, with their excessively laden weight belts still firmly attached.

Before diving is attempted, aquatic skills including unassisted swimming and snorkeling should be acquired. Scuba training should be from professional diving instructors, both for the initial open water training and subsequent courses on buoyancy control, rescue and advanced diving. Supervision is needed while extending diving activities involving different environments, equipment or dive parameters.

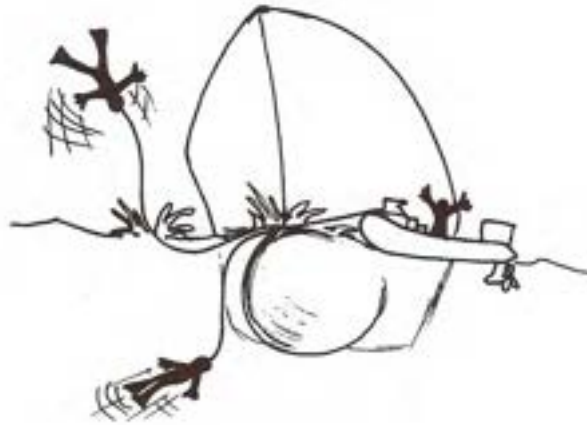


Fig 34.4

Chapter 35

SUDDEN DEATH SYNDROME (Cardiac Death)

PATHOLOGY

Sudden death in divers, especially middle aged divers, is not a rare event. The usual cause is cardiac — either a fatal disturbance of cardiac rhythm (arrhythmia), heart muscle death from a blockage of a diseased coronary artery (coronary occlusion causing ischaemia, myocardial infarction or "heart attack"), or a disease of the heart muscle itself (myocarditis, cardiomyopathy).

Statistical studies on deaths in diving show a disturbingly high incidence of death attributed to heart disease, and rising. In the DAN series, 26% of deaths were cardiac and they contributed to 45% of the scuba deaths in those over 40 years. They tend to be in more experienced divers, associated with known cardiac history, arrhythmias or ischaemia, hypertension or the use of cardio-active drugs.

Cardiac Arrhythmias

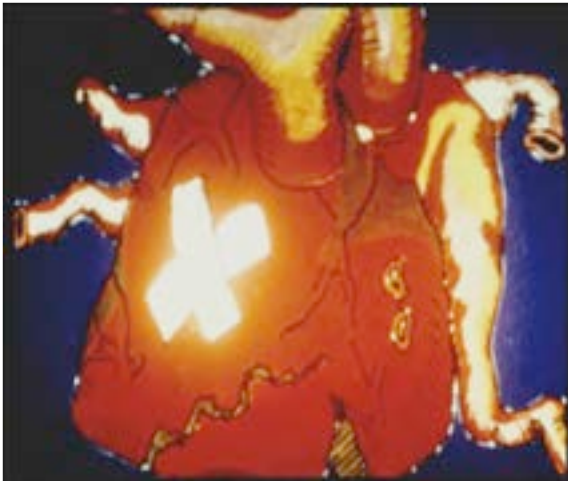
The heart normally beats in an orderly and regular way (see Chapter 3). The atria contract, first propelling blood into the ventricles which then in turn contract, ejecting blood into the major arteries.

If this rhythmic contraction is disturbed (an arrhythmia or "irregular heart beat"), the efficiency of cardiac function is impaired and the heart has to work harder, requiring more oxygen and blood flow of its own. Impaired efficiency may also cause lowered blood pressure, which can reduce blood flow to the brain, causing unconsciousness. The

arrhythmia which causes sudden death is called **ventricular fibrillation**, and this usually results in unconsciousness within a few seconds and death within a few minutes.

Severe arrhythmias tend to occur in divers who already have less severe arrhythmias, those with cardiac ischaemia, cardiomyopathies or who take cardio-active drugs (such as for hypertension, asthma, stimulants etc.)

Coronary Artery Disease



The heart receives its own blood supply from the coronary arteries. Its requirement for blood increases when it has to perform more work, for example during exercise. For a given level of exercise the heart has to work even harder if the blood pressure is elevated, or if the heart has to beat too rapidly, or if the resistance to blood flow is increased. Arrhythmias also increase the cardiac workload.

Fig.35.1

The heart is less able to cope with extra demands for work if the coronary arteries are obstructed, since the blood flow to the heart is reduced. When the coronary arteries do not supply sufficient blood and oxygen to the heart muscles, the latter becomes painful and produces central or left sided chest pain ("angina") or breathlessness (dyspnoea). This may be temporarily remedied by reducing the exercise and the demand for oxygen, by resting. If this deprivation of oxygen to the heart muscle is severe enough, heart muscle dies, and this is then called a myocardial infarction ("heart attack"). In divers, the first sign of this may be at autopsy.

Heart Muscle Disease

Some forms of heart muscle disease (cardiomyopathy – hereditary, alcoholic, or toxic) may affect its function and can occur at all ages. In non-divers who become aware of these diseases, heart transplants are often the only successful treatments. Divers may be unaware that they have these diseases. Viral infections sometimes involve the heart muscle (myocarditis), often without the patient being aware of this, and these infections predispose to cardiac deaths.

CONTRIBUTING CAUSES

There are a number of ways death or incapacity from cardiac diseases can come about and they are usually precipitated by one or more **trigger factors** encountered while diving. Some of these are:

Exercise

Severe exercise can cause sudden death by a number of mechanisms. Probably the most well known example was the death of the first marathon runner who dropped dead after running from Marathon to Athens to deliver the news of the Greek victory over the Persians. In reality, his death was probably due to heat stroke or heat exhaustion. Usually exercise will cause cardiac deaths only in those with some cardiac disease or malfunction.

A diver is at a disadvantage in some ways during exercise. During exertion on land, the cardiac output increases to meet the metabolic demands of the exercising muscles. In doing this, the work of the heart is made easier by blood vessel dilatation in the peripheral circulation, reducing the resistance to blood flow. In an exercising diver however, the skin blood vessels do not dilate because they are trying to conserve heat in response to the surrounding cold water. The diver's heart has to pump against an increased resistance and so work harder for a given amount of exercise, compared to a land athlete.

One of the limitations to exercise on land is the inability to disperse the metabolic heat of exercise. With the diver, much of this heat is conducted away by the water. As a result, it is possible to exercise in the water to a greater degree without the "hot and sweaty" discomfort.

It is therefore possible to exercise to a great degree in the water, with less discomfort but at a greater strain on the heart. In a trained athlete with a healthy heart this probably is only of academic interest. In a middle aged (i.e. over 40 years) diver with some degree of coronary artery disease ("narrowing of the arteries") the diver can overload the heart without realising it. This can result in sudden death.

Exercise, even in fit healthy divers, has been shown to cause significant arrhythmias with diving. It is much more likely to cause incapacitating or fatal arrhythmias in divers with cardiac disease.

Psychological and Personality Factors

Some personalities are more susceptible to cardiac disease than others. The so called **Type A** or **Type D personality** is believed to be most prone to cardiac disease. These individuals are intensely competitive, aggressive and as a result, by society standards, usually successful. They drive themselves hard and do not give up. They are twice as likely to develop coronary artery disease than others, and when they develop it they are likely to push their diseased heart beyond its limitations.

Traditionally this has been a **male personality trait**, but in a more competitive and equal society it is probable that a similar disease pattern will emerge in women competing in previously male dominated areas. Sudden death is not uncommon in Type A personalities.

Anxiety can have threatening cardiac consequences. Anxiety typically causes internal release of adrenalin, one of the stress hormones, which stimulates the heart to contract more forcefully, beat faster, and makes it more prone to arrhythmias. A fast beating heart has less time to replenish its own blood supply and becomes relatively starved of blood.

In the peripheral circulation, adrenalin causes constriction of blood vessels to the skin and internal organs, increasing the resistance to blood flow and the work of the heart. The stressed anxious individual thus has a fast beating heart with a poor blood supply which is more prone to arrhythmias and which has to work harder for a given exercise load.

A condition analogous to fainting (known as **vasovagal syncope**) is commonly seen in individually threatening situations such as a blood donation or the receiving of injections. A nervous response through central stimulation of the vagus nerve causes profound slowing of the heart. The end result is inadequate blood pressure and reduced cerebral circulation causing the diver to lose consciousness (“faint”).

Cold

Sudden incapacity and death of divers soon after entering cold water has been frequently reported. The body has several immediate responses to cold water which could explain this.

During **cold water immersion** there is an increased sympathetic nervous system activity resulting in the release of adrenalin. This causes the potentially deleterious cardiac effects described above. A greater sympathetic response has been described in individuals who are **not adapted** to cold water exposure **or** who are **unfit**.

Sudden death from **vagal stimulation** associated with the **diving reflex** can occur after immersion of the face in cold water, although it can also be produced by immersion of the trunk in cold water. See below.

Sudden immersion in cold water is thought to be associated with a sudden death syndrome associated with reflex **coronary artery spasm, fatal arrhythmias** or **myocardial infarction**.

Divers will be familiar with the involuntary over breathing which can accompany sudden immersion in cold water or even a cold shower. In experimental animals, and also in man, the heart becomes more prone to arrhythmias caused by the reduction in blood carbon dioxide from this **involuntary hyperventilation**.

Hypothermia also makes the heart more prone to arrhythmias and may combine with some of the other problems mentioned above to cause sudden death.

Reflexes Associated with Diving

□ The Diving Reflex.

Diving mammals such as whales are able to hold their breath for an hour and attain amazing depths. They are able to do this partly because of the evolution of the dive reflex. When the mammal leaves the surface there is a profound stimulus of the vagus nerve which slows the heart to about a fifth of its normal rate. At the same time, there is intense constriction of the blood supply to the skin and most organs with the exception of the heart, lungs and brain. This conserves oxygen reserves for use by the organs which need it most. The diving mammal maintains a normal blood pressure, but the output and work of the heart is dramatically reduced.

This reflex is present to some extent in humans. When a human is immersed in cold water there is vagal stimulation which slows the heart, as well as sympathetic nervous stimulation which constricts blood vessels to the skin and other organs. Because the reflex is only incompletely developed in man, there is often a rise in blood pressure but minimal or no fall in cardiac output. This increases, rather than reduces, the work of the heart.

The result of this process in man is increased work of the heart as well as the development of cardiac arrhythmias. Studies conducted on the traditional breath-hold Ama who showed an incidence of arrhythmias of 43% in summer, and an even higher incidence in winter.

❑ Carotid Sinus Syndrome.

The Carotid arteries, on each side of the neck, are the main arteries which supply the brain with blood, and these have a pressure sensing organ – the carotid sinus – in their walls at about the level of the larynx. External pressure on these carotid sinuses causes the cardiac control centre of the brain to mistakenly assume that the blood pressure has suddenly risen. This leads to a **reflex slowing of the heart** and **reduced blood pressure**. This can cause faintness or even loss of consciousness.

A similar effect is caused by pressure from the collar of a tight fitting wetsuit or dry suit neck seal, on the carotid sinus. The problem is especially likely in wet suits without a front zip fastener or having tight "crew necks".

In a series of 100 carefully documented diving deaths in Australia, only one case was thought to be due to this carotid sinus syndrome. In other cases however, distressed divers were seen to pull a constricting wetsuit away from the neck. This may have been a response to respiratory difficulty from cardiac causes, a tight neck opening in the suit, or the carotid sinus syndrome.

Hyperbaric Exposure

Studies in experimental subjects breathing air at pressures similar to those experienced by sports divers showed a significant incidence of arrhythmias caused by the hyperbaric exposure. This may be partly due to the elevated partial pressures of oxygen breathed at these depths.

Immersion

Simply immersing the body in water causes an increased return of blood to the heart, due to the change from a gravity influenced circulation to weightlessness. This rush of blood to the heart can rapidly double its workload until stability is returned. In redistributing blood from the periphery to the lungs, there may be a predisposition to pulmonary oedema (see Chapter 32)

With immersion, the effect of gravity on the blood vessels is removed. Sudden exposure to a gravity effect on the blood vessels, such as climbing up the ladder and out of the water, increases the possibility of hypotensive syncope. This is a good reason to not hover underneath divers on ladders.

Aspiration

Aspiration of seawater – always a possibility in diving – can cause immediate cardiac effects by a mechanism akin to the diving reflex (see above), It can also be followed by delayed effects due to hypoxia as the lungs are involved, as in the salt water aspiration syndrome (see Chapter 26).

Drug Effects

A large variety of drugs have arrhythmic and other effects on the heart, which may predispose to sudden death. Many can be purchased 'over-the-counter' in pharmacies or supermarkets. Some are contained in 'cold cures' and 'cough mixtures' and may be inadvertently used by divers. Some of these drugs include:

- Alcohol
- Nicotine – cigarette smoking
- Caffeine – coffee and tea, stimulant drugs to overcome sleepiness
- "Social" drugs such as cocaine, weight reducing and stimulant drugs such as amphetamines
- Blood pressure controlling drugs (e.g. calcium channel blockers, beta blockers)
- Drugs used to suppress arrhythmias (e.g. beta blockers)
- Drugs that change electrolyte concentrations in the blood – diuretics and electrolytes
- Sympathomimetic drugs (e.g. decongestants such as pseudoephedrine, anti-asthma medications such as salbutamol, and some anti-seasick drugs).
- Others that may cause arrhythmias –antidepressants, digoxin, some anti-malarials, local anaesthetics.

Cardiac Disease

❑ Coronary artery disease or CAD.

This heart disease (causing narrowing or obstruction of the coronary arteries), while considered to be a disease of middle and older age, is probably present to some degree even in some young adults.

It would appear from post-mortem studies done during the Korean and Vietnam wars that coronary artery disease begins in early adulthood, but usually only causes symptoms and death from heart attack after 40 years of age. The older the diver, the more significant this is likely to be. Divers with this disease are more prone to sudden death due to arrhythmias or myocardial infarction secondary to impaired blood supply to the heart muscle.

❑ Coronary artery bypass grafts.

Some blockages of the coronary arteries can be bypassed by blood vessel grafts – usually using arteries or veins. This reduces cardiac pain and improves cardiac performance but does not cure the underlying disease, which affects the many coronary arteries not improved with the by-pass. People with such grafts are still more prone to arrhythmias and cardiac dysfunction and should not dive, unless proven to be cured. A similar situation exists in those people who have already suffered a myocardial infarction or "heart attack" and those needing or possessing pacemakers

□ Myocarditis.

Some viral infections which produce a flu-like illness can temporarily affect the heart muscle, impairing its performance and making it susceptible to arrhythmias. Sudden deaths from this often insidious condition (myocarditis) are occasionally reported in very fit athletes and in divers. It is unwise to dive or perform heavy exertion when suffering from a viral infection, for this reason.

Sometimes the heart is permanently and irreversibly damaged by such viruses. It is then similar to a cardiomyopathy. In terminal cases the only effective treatment is a heart transplant.

PREVENTION

All candidates should be carefully examined by an experienced diving physician before dive training. Those with known cardiac disease or a tendency to arrhythmias cannot dive safely. Middle aged divers, and those with high coronary risk factors need regular assessments.

Coronary risk factors include:

- a family history of heart disease at a similar age to the diver
- cigarette smoking
- hypertension
 - cardio-active drugs (see earlier)
- obesity
- high cholesterol – hyperlipidaemia
- physical unfitness
- diseases such as diabetes and alcoholism.

Diving situations often require extreme physical exertion, which stresses the heart. A high standard of physical fitness brought about by regular exercise will improve the hearts ability to cope with this exertion.

While jumping into cold water gets the discomfort over quickly, it maximises the physiological stress. Enter cold water slowly to minimise these physiological stresses.

The combination of performance anxiety, transport stress, inadequate sleep, excessive alcohol, coffee and other drugs which often accompany a "high-living" diving holiday may be possible contributors to cardiac arrhythmias, and some deaths in divers.

Chapter 36

PSYCHOLOGICAL DISORDERS

Psychological Traits of Successful Divers

This topic has not been extensively researched, but the few studies which have been done on the psychological make-up of divers have shown the following — successful divers tend not to be anxious people; they are self sufficient, intelligent and emotionally stable. Their tolerance to stress often allows them to continue to function during difficulties which would incapacitate many non-divers. This may be helped by their tendency to use "denial", a mental mechanism by which they ignore the hazards which may confront them.

In spite of their overall stability, divers, like anyone else, can suffer from psychological disturbances (see Chapter 7 for a full description of stress and panic responses).

Anxiety States

It is quite normal for divers to feel concerned with the very real hazards of diving – and this book is full of them. Some people, however, develop an excessive and inappropriate anxiety to diving hazards which may become a **phobia** – an irrational fear. This may be the result of a previous traumatic event (such as near drowning during childhood) or may be an exaggerated reaction to some diving danger. Phobias may relate to diving in general or to a specific diving hazard (such as an excessive fear of sharks) or a situation (e.g. claustrophobia with the face mask, night diving, poor visibility).

Phobias can be treated by psychological de-conditioning therapy.

Most people who are anxious about diving are aware of this early in their training and quite sensibly desist. Why continue a recreational activity which causes apprehension? Unfortunately some continue because of peer pressure, ego challenge or other personal reasons. These divers tend to have a high baseline level of anxiety (**neuroticism** or "**high trait anxiety**") when diving and are more prone to panic when confronted with real or imagined hazards.

Flora Fisk Quiz for Recreational Divers:

Question: Is recreational diving supposed to be fun? Answer: Yes

Question: Are you having fun? If "yes" — continue diving.

If "no" — STOP DIVING.

Some divers experience a general anxiety reaction under special circumstances. It is an aquatic manifestation of a general medical (psychological) disorder produced by sensory deprivation - called the **Blue Orb Syndrome** by high altitude aviators. It usually happens to a lone diver in deep water, where there are no visual references. The diver develops an anxious feeling of being alone in the vastness of the ocean. This can lead to mounting anxiety and panic. The panicked diver may rush to the surface, omitting decompression or develop pulmonary barotrauma from failure to exhale adequately on ascent. The symptoms usually subside if the diver can establish visual contact with concrete objects such as the sea bed, a dive boat or even another diver, or by concentrating on diving instruments, such as a watch or depth gauge.

This syndrome can be avoided by diving with a buddy who provides reassuring company and a visual reference. Avoidance of deep water where there are no visual references, is also helpful.

Panic

This frenzied and irrational behaviour is the end result of a number of diving difficulties. It is more likely to occur in anxiety prone divers and frequently results in a diving accident or fatality. It is an important topic for divers to understand and is covered in detail in Chapter 7 and Case History 7.1

Psychological Disturbances due to Medical Causes

Brain function can be disturbed by physiological factors (such as **nitrogen narcosis**, **hypothermia**) and by other diving related illnesses.

Cerebral decompression sickness and **air embolism** can cause alteration of brain function during both the acute event and recovery. A diver may act irrationally because of these diseases, and not just his basic personality. Suspect this if he is acting "out of character".

Near drowning, **hypoxia** and the **gas toxicity diseases (oxygen, carbon dioxide, carbon monoxide**, etc.) may also cause temporary or permanent brain damage.

Symptoms include confusion, irritability and irrational behaviour. This should always be borne in mind if a victim of a diving accident is unreasonably reluctant to undergo treatment. People who know the diver well will normally be the best judges of whether the behaviour is out of character.

Dementia

This is a deterioration of intellectual capacity and memory which is common in the elderly and has a variety of causes. Alzheimer's ("old timers") Disease is a severe form of dementia. Diving folklore holds that divers suffer an increased incidence of dementia. This belief has been supported by media reporting and anecdotal accounts of divers suffering from the condition. Some even believe that only "demented" persons would take up diving!

There are plausible theoretical reasons why divers could sustain brain damage sufficient to cause dementia from conditions such as repeated subclinical, or overt, cerebral **decompression sickness, air embolism, near drowning** or **carbon monoxide poisoning**, to name a few.

There are also some scientific studies which show evidence of at least transient brain damage in some divers. A study in Sweden showed 3.5% of free ascent trainees to have **EEG** ("electrical brain wave") abnormalities after free ascent training, and in another survey, EEG abnormalities were found in 43% of a group of Polish professional divers compared to 10% in a normal population.

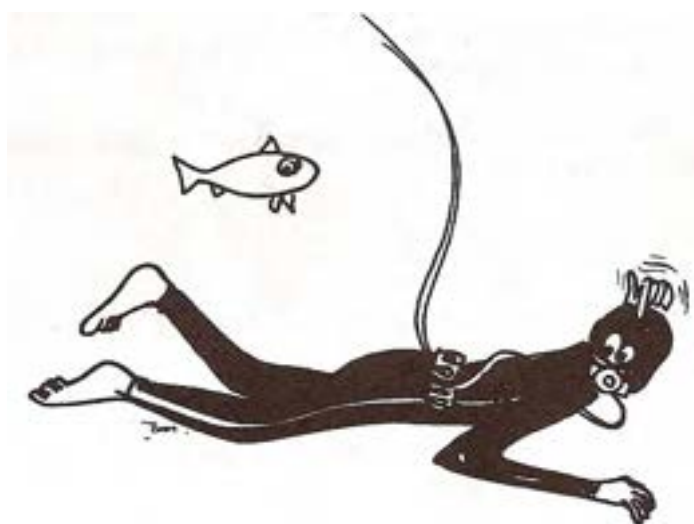


Fig. 36.1

In Australia, a group of divers, studied after treatment for **decompression sickness**, showed **neurological, psychological** and **EEG abnormalities** for some weeks after treatment, even in those who had no symptoms of neurological decompression sickness.

There have been several studies worldwide which appear to show deterioration of intellectual performance and psychological disturbances in divers suffering from neurological decompression sickness or "near miss" diving accidents. Unfortunately the methodology of these studies was grossly inadequate, making the conclusions unreliable.

To clear up some of the controversy a study of all 152 professional **abalone divers**, from a closed community, was undertaken in Australia by Edmonds and others in 1988. The divers in the study had diving exposures which would generally be regarded as extreme. On average they had been diving for 16 years and had been professional abalone divers for 12 years. They averaged 5 hours underwater per day on Hookah equipment for 105 days per year at an average depth of 15 metres

(50 ft) and admitted to being "bent" four times. Many other incidents of decompression sickness went unrecognised and untreated. Half of the divers used a dive profile which would, according to conventional dive tables, require decompression but which they omitted. Of the 69 cases of decompression sickness in this group which were actually treated, at least half were neurological.

It would seem that if there was any group of compressed air divers prone to brain injury after excessive diving exposure, it would be this one.

The divers were subjected to a wide battery of tests including **intelligence tests**, **psychometric investigations** to detect psychological abnormalities, **memory tests** and studies designed specifically to detect early dementia, **EEG studies** and **neurobehavioural tests**. The divers were compared with control groups taken from non-diving fishermen in the same localities.

The results showed the divers studied were within the normal range for the general population and displayed no evidence of brain damage or dementia. This implies that air divers, in general, have no greater risk of dementia or brain damage than non-divers. If brain damage does occur, it is either rare or so mild that it could not be detected by conventional testing.

Since the diving practices of this group were extreme, it seems reasonable to conclude that divers following more conservative practices, as well as other, more conventional, professional compressed air divers, have no greater risk of dementia or progressive brain damage than non-divers, unless they suffer a major diving accident affecting the brain (such as those mentioned above).

The signs of brain damage which have been described in studies performed soon after minor diving events are presumably temporary in nature.

Chapter 37

DRUGS AND DIVING

It is common for divers to enter the water under the influence of drugs. These may vary from paracetamol taken for a minor headache, to alcohol or marijuana from a beach party the night before, or a therapeutic drug for an illness such as high blood pressure.

Since some drugs are innocuous while others can have potentially lethal effects with diving, it is important to know something about them.

Problems can arise from effects of the drugs themselves, but commonly the condition for which the medication is taken poses a greater threat to the diver. For instance, most antibiotics have no harmful influences on divers, but a diver being treated for bronchitis with an antibiotic, has a significant risk of developing pulmonary barotrauma until the condition resolves.

We will consider commonly used drugs under four categories:

- Drugs taken for treatment of illnesses
- Drugs taken for prevention of illness (prophylaxis)
- Recreational or social drugs
- Drugs used for diving related illnesses.

TREATMENT DRUGS

In many cases the drug which is used to treat an illness can be a greater hazard to the diver than the illness itself. Some drugs may have frequent and predictable effects on diving activities and these are summarised here, but much more information can be sought by an Internet search.

The idiosyncratic effects of other therapeutic drugs on an individual are unpredictable. Thus, if drugs are to be used when diving, they should be introduced to the diver long before the diving

is undertaken. Then a drug reaction is less likely to complicate the interpretation and treatment of potential diving diseases.

Cardiac and Blood Pressure Medications

❑ Beta-blockers.

A variety of these drugs (e.g. atenolol, metoprolol) are used to treat high blood pressure or pain from coronary artery disease (angina). Their main action is to block the effect of the cardiac stimulant, adrenalin, on the heart. Adrenalin acts on specific drug receptors in the heart known as "beta receptors", thus giving rise to the term beta-blocker.

By inhibiting the action of adrenalin, beta-blockers reduce the force of contraction of the heart muscle. This diminishes the work it performs and so reduces angina symptoms, while the reduced output of blood lowers the blood pressure.

A diver taking beta-blockers has a significant limitation of the reserve pumping capacity of the heart. If a large blood supply is required by the muscles to extract the diver from an emergency e.g. heavy wave action or an adverse current, it may not be available.

Beta-blockers have been incriminated in the production of both arrhythmias and pulmonary oedema in divers.

Beta-blockers also act on the muscle lining of the bronchi and may unmask asthma in some individuals, exposing them to the dangers of both asthma and burst lung. Even those beta-blockers which are described as "cardio-selective" can still have these effects.

These drugs are often used in eye drops for the treatment of glaucoma. Significant amounts can sometimes be absorbed into the body causing generalised effects. Divers using these drops should seek medical advice to ensure that they are not affected in this way. They can avoid this complication by using a eye-drop technique called lacrimal compression.

The conditions for which the drugs are taken can cause difficulties as well. For example, a diver under treatment for high blood pressure is also at high risk of coronary artery disease, and may already have a sub-clinical form of this disease. The drugs may summate with, or potentiate, other causes of heart rate reduction associated with diving, and provoke arrhythmias and the sudden death syndrome (see Chapter 35).

❑ Other blood pressure drugs.

Apart from beta-blockers, blood pressure lowering drugs fall into two broad categories — blood vessel dilators and diuretics (stimulators of urine production).

- **Blood vessel dilators (vasodilators)** reduce blood pressure by widening peripheral blood vessels, where most of the resistance to blood flow occurs. These include prazosin and felodipine.

Some can inhibit the body's ability to compensate for changes in posture, causing fainting on standing. This is an undesirable side effect in a diver attempting to ascend a ladder to leave the water, especially in adverse sea conditions or if he is preceding his buddy.

A newer drug, the ACE inhibitors, can produce a dry cough which can be troublesome in the diving environment. Others, (such as calcium channel blockers like verapamil) may affect the nerve conduction of the heart, making it more susceptible to the sudden death syndrome (see Chapter 35).

- **Diuretics** stimulate the production of urine and tend to dry the body out. This reduces blood volume and so tends to lower the blood pressure, but there are probably other mechanisms acting as well. The effects on diving are not clear but there are potential problems.

Reduction of the blood volume may affect blood viscosity and the dynamics of blood flow and so increase the dangers with bubble formation. In addition, changes to regional blood flow may alter the pattern of gas uptake and elimination (decompression).

Some diuretics tend to lower the blood potassium level, making the heart more prone to disturbances of rhythm. These arrhythmias may be unmasked by the effects of cold, the dive reflex, heavy exertion and the other causes of the sudden death syndrome (see Chapter 35).

One group of diuretics, carbonic anhydrase inhibitors, is also used as treatment for glaucoma. These cause increased paraesthesia especially in the cold-exposed hands of divers, and may lead to a mistaken diagnosis of decompression sickness.

Psychotropic Drugs

□ **Tranquillisers and sedatives.**

This group of drugs includes **benzodiazepines**, of which diazepam ("Valium") is a common example, and sleep making drugs like barbiturates. A significant proportion of the population takes drugs to relieve anxiety. Excessive anxiety alone is a significant risk factor in diving, and the drugs taken to relieve it further complicate the problem.

Another class of tranquillisers, like **phenothiazines**, (e.g. chlorpromazine – "Largactil") and its modern equivalents, are used to treat serious psychiatric disorders such as schizophrenia. Apart from the side effects of these drugs, people suffering from this disorder often have a tenuous grip on reality and this can seriously impair their ability to make safe judgments related to diving.

Tranquillisers and sedatives cause; drowsiness, impaired judgment, slowing of thought processes and reduction in problem solving ability. These effects are intensified by nitrogen narcosis, but they are potentially dangerous at all depths.

□ **Antidepressants.**

Depression is not an ideal state of mind for an active diver. Even when successfully treated with antidepressants, the diver is left with potentially harmful side effects from the drugs. Some of the antidepressants cause sedation, but the principal problem is the tendency of others to cause potentially lethal disturbances of heart rhythm and epilepsy. Some can react with certain foods and other drugs to affect blood pressure and consciousness.

□ **Anticonvulsants (anti-epileptic drugs).**

These have similar sedative side effects to the tranquillisers as well as some other specific complications. Any form of epilepsy can have disastrous effects on cerebral activity, with loss of consciousness being common. The influence of nitrogen narcosis on these drugs is unknown. Some diving conditions (stress, glare and flickering light, high or low oxygen and carbon dioxide levels) can precipitate convulsions, despite these medications. Epilepsy and medications used for its control (e.g. phenytoin or carbamazepam), preclude safe diving.

Antihistamines

These are usually taken to treat allergic conditions. Pharmacologically, many are closely related to the psychiatric drugs and share a common side effect, sedation. They cause the same potential hazards to diving as other sedatives. In addition, if antihistamines are taken to treat hay fever, there is a strong possibility of the diver developing ear or sinus barotraumas. These drugs seldom completely cure the nose and throat congestion. Other recently developed drugs are less sedative, but may provoke cardiac arrhythmias or bronchospasm (asthma).

Antibiotics

These have a large number of side effects, but few of specific relevance to diving. Tetracyclines can occasionally cause photosensitivity, a condition resembling sunburn caused by enhancing sensitivity to sunlight. Many antibiotics increase susceptibility to **vomiting**.

The condition for which the antibiotic is taken is usually of more concern. This especially applies to respiratory tract infections which make the diver prone to barotraumas.

Analgesics

A diver suffering from pain which warrants the use of pain killers should generally not be diving. Apart from the adverse interactions some diseases can have on diving, the commonly used analgesics can have undesirable side effects. There is also the **diagnostic confusion** that may result between the painful condition aggravated by diving, and decompression sickness.

❑ Aspirin.

This commonly used analgesic causes an inhibition of the clotting ability of blood, with just one dose and lasts for many days.

This is usually not a problem in everyday use – in fact the blood effect is used to prevent heart attacks and strokes. However, it can have implications if the diver develops **inner ear barotrauma** or serious **decompression sickness**. The increased bleeding tendency can result in haemorrhage into injured tissues, such as the spinal cord, with greater consequences. Ulcer-like erosions can also occur in the stomach, with vomiting and occasionally bleeding from the gut

It may also cause bronchospasm, like **asthma**, in some divers.

❑ Paracetamol (acetaminophen).

If a diver needs to take analgesics for minor pain (hopefully after excluding diving related illnesses as a cause), it is better to use paracetamol which has fewer side effects than aspirin. Paracetamol does not effect blood coagulation and avoids the stomach upsets, common with aspirin.

❑ Strong analgesics.

Preparations containing codeine or dextropropoxyphene (both narcotic derivatives) and other strong analgesics are sometimes prescribed for severe pain.

These drugs have comparable sedative effects to the tranquillisers and can have similar adverse interactions with diving. People with pain of this intensity should not be diving.

Insulin and Anti-diabetic Agents

People taking these drugs are prone to sudden depression of the blood sugar level which produces **anxiety**, **confusion** and then **unconsciousness**. This is particularly likely during exercise. This complication in the water often has a fatal outcome. Because of this possibility and other potential physiological complications (e.g. acidosis and hyperventilation), diabetics are generally advised against diving.

Bronchodilators and other Asthma Medications

Asthma is an inflammatory condition of the air passages of the lungs. It causes swelling of the lining of the airways, spasm of the muscles in the airways (bronchospasm) and obstruction of airflow through them. The bronchospasm can be reduced by aerosol sprays containing drugs such as salbutamol ("Ventolin") or oral bronchodilators. These can disturb cardiac rhythm and precipitate the sudden death syndrome during diving, because of the multiple trigger factors (see Chapter 35).

While the use of these and other asthma medications will improve some of the airway flow and thus relieve symptoms, it does not cure the condition. Asthmatics have airways which are excessively sensitive to irritants, reacting with bronchospasm to stimuli such as cold dry air and sea water inhalation. There is usually a degree of obstruction in some of the airways most of the time. This makes them susceptible to pulmonary barotrauma or death from the diving sequelae of asthma - panic and drowning.

Some oral bronchodilators (theophyllines) can cause pulmonary vasodilatation – which could potentially allow asymptomatic venous bubbles from normally safe dives to enter the arterial circulation as gas emboli, even without pulmonary barotrauma. **Asthma and these medications are incompatible with safe diving.**

Implanted Drug Delivery Systems

Implanted **reservoirs** are now being used to deliver drugs which cannot be taken orally and which need to be used over prolonged periods. Many of the conditions for which these reservoirs are used are incompatible with scuba diving.

Implants form a potential site of bubble formation during decompression. If bubbles form inside or around the reservoir, expansion in response to the gas laws may lead to excessive delivery of the drug. As experience with these devices in diving is limited, divers fitted with them are advised to seek expert medical advice concerning the possible complications.

PROPHYLACTIC (PREVENTION) DRUGS

Statins

These are frequently used to reduce the harmful effects of cholesterol, but may have other beneficial effects. Patients who need these drugs are likely to have an increased risk of cardiac disease, and so should be assessed more completely by their diving physician.

Some recipients will respond to statins with serious muscular and other disorders and so diving should be delayed for some weeks or months to ensure these problems are not experienced.

Oral Contraceptives — the "Pill"

These preparations can have serious side effects, even without the complication of diving. In older high dosage drugs, excessive blood clotting resulted in occasional deaths from pulmonary embolus or strokes.

Occasionally the pill produces serious psychological sequelae, migraine, nausea and vomiting, which may make diving more hazardous.

The newer low dose oral contraceptives have a lower incidence of these disorders. The concern with diving is the possibility of more congealable blood interacting with gas bubbles during decompression. There is no overwhelming evidence to either confirm or refute this theoretical risk, despite a number of surveys on female divers.

Anti-Sea Sickness Drugs

See Chapter 32.

Antimalarial Drugs

Tropical countries offer some spectacular diving locations but also frequently have endemic diseases, including malaria.

The chances of contracting this potentially lethal disease are reduced by the use of anti-malarial drugs such as chloroquine and pyrimethamine ("Maloprim"). Unfortunately many countries now have strains of malaria which are resistant to conventional anti-malarial drugs, making their use as a preventative measure not fully reliable. As well as being fallible, these drugs can have serious side effects including suppression of white blood cell production, anaemia, and eye damage.

One of the anti-malarials, mefloquine ("Lariam"), can cause coordination disturbances and vertigo which may have alarming implications and cause diagnostic problems for divers.

A diver intending to visit a malaria endemic area should seek expert medical advice regarding prophylaxis for malaria in that area, as well as other more exotic tropical diseases. A diving physician should also be asked about possible interactions of the prescribed drugs with diving.

RECREATIONAL (SOCIAL) DRUGS

Alcohol

Diving culture has traditionally included substantial use of alcohol. Like other drugs, it can have adverse interactions with diving.

There is no safe blood alcohol level for diving and few people in their right mind would consider diving while under its influence. Some may not be aware that the liver has a limited capacity to metabolise this drug, so it is possible to have an appreciable blood alcohol level on the morning after a night of heavy consumption. Traffic police are well aware of this. They frequently apprehend drivers going to work with an illegal blood alcohol level on the morning after.

The danger of alcohol consumption associated with aquatic recreations is well documented — 80% of **drownings** in adult males are associated with alcohol use, in Western countries. The hazards are predictable. Alcohol intoxication, as well as **impairing judgment and coordination**, causes **cardiac rhythm disturbances**, **impairs the pumping ability of the heart**, **reduces the blood volume** due to excessive urine production, and increases **heat loss** through the skin (**hypothermia**).

The disturbed physiology – otherwise known as a "hangover" – after excessive alcohol consumption is well known to the younger authors of this text. Used to excess, this drug is a toxin, damaging the liver, heart and brain. In divers, the vascular and metabolic dysfunction after heavy consumption is a probable risk factor for the development of **decompression sickness**. Increased susceptibility to both **sea sickness** and **vomiting** is frequently observed. Soporific effects may summate with those of **nitrogen narcosis**.

Tobacco

The gentle art of inhaling burnt tobacco leaf has some unfortunate side effects. The associated risks of lung cancer, heart and vascular disease are well known. There are more subtle effects.

A smoker inhales carbon monoxide, which binds to the haemoglobin and reduces the blood's ability to transport oxygen by up to 10%. This reduces the capacity for exertion and impairs the physical ability to respond to an emergency (e.g. fatigue from a surface swim). The nicotine in the tobacco also stimulates the heart, making it prone to **rhythm disturbances** (arrhythmias).

Airway narrowing caused by chronic smoke irritation impairs exercising ability and increases the risk of **pulmonary barotrauma**. A similar chronic irritation of the upper respiratory tract predisposes to **ear** and **sinus barotrauma**.

Marijuana — Cannabis or "pot"

Chronic use of this drug causes many of the diving related respiratory problems attributable to cigarette smoke, and **chronic bronchitis** is especially common in heavy users. This predisposes to **pulmonary barotrauma**.

Marijuana causes altered perception, **impaired judgment**, and mood alterations, which are incompatible with diving safety. As with other drugs, these effects are compounded by the effects of nitrogen narcosis. It also is said to increase the likelihood of **hypothermia** by blocking blood vessel response to cold. The allegedly "beneficial effects" of marijuana are negated by pressure!

Cocaine ("coke") and Other Stimulants

These drugs have similar physiological effects to adrenalin, stimulating and irritating the heart, causing potentially lethal **cardiac rhythm disturbances**, and elevating the blood pressure. Sudden death in young people from the cardiac effects is common, especially in athletes who exercise after taking cocaine.

The mental stimulation and mood elevation **impair judgment** and encourage **risk taking**. Its use while diving, apart from being illegal, is very risky.

Caffeine

This drug is found in coffee, tea, cola, and many natural foods. Even chocolate drinks, periodically given to children at bedtime, contain it. It is one of the more innocuous drugs which is used almost universally.

When used to excess it can irritate the heart causing **rhythm disturbances** which are a potential problem in diving or other strenuous exercise. It also stimulates urine production which discourages some divers from lending their wet suit to known caffeine abusers.

Narcotics

The **sedative** and **judgment impairing** qualities of these drugs makes their use during diving even more dangerous and destructive than their use as a recreational drug.

Intravenous drug users have a significant risk of being infected with the **hepatitis** and **HIV (AIDS)** virus, which should be born in mind by their diving companions (see Chapter 28).

DRUGS FOR DIVING DISEASES

Anti Sea Sickness Drugs

See Chapter 32

Sinus and Ear Problems

Many inexperienced divers have difficulty equalising the ears and sinuses to pressure changes. Often this difficulty is associated with congestion of the lining of the nose, generally due to allergy (hay fever) or infection (URTI). Poor technique is a contributing factor.

Nasal congestion can be relieved to some extent by the use of tablets such as pseudoephedrine ("Sudafed"), or nasal decongestant sprays such as phenylephrine or ephedrine. They all have a **disruptive effect** on the **heart's conduction system** and may thus increase the likelihood of the sudden death syndrome.

These agents used in proper doses on land have few harmful generalised effects. However their activity on the nasal tissues can be unpredictable. Prolonged use causes localised tolerance to the drug, eventually aggravating the congestion which they are supposed to relieve. This applies particularly to nasal sprays. Their effect can wear off during the dive, allowing a trouble free descent, followed by sinus or ear barotrauma during ascent (see Chapters 9 and 10).

These drugs are sometimes used by divers to overcome the temporary nasal congestion of an upper respiratory tract infection (a cold or URTI). A safer approach is to avoid diving during the course of these infections.

If the decongestant is somewhat effective, it may result in the diver avoiding barotrauma of descent (as the beneficial effect is on the nasal lining) but have little or no effect on the "internal" opening of each of the air passages (Eustachian tube, sinus ostia, etc.). Thus the diver is now vulnerable to an internal blockage which manifests during ascent and produces **barotrauma of ascent**. This disorder is far more dangerous than the disease for which he is taking it, as it prevents ascent to safety. Barotrauma of descent merely stops him diving.

Self medication with these drugs is unwise and divers with "congestion" problems should seek the advice of a diving physician.

Medication for use in Decompression Sickness

In view of the relative unreliability of the decompression tables, researchers have experimented with drugs to inhibit the development of bubbles and speed gas elimination from the body. While some experimental drugs now allow laboratory animals to dive with a much greater margin of safety, no agents useful to human divers have yet been convincingly demonstrated.

Chapter 38

MEDICAL EXAMINATIONS FOR DIVERS

This chapter is not adequate to instruct a medical practitioner on the complexities of performing diving medical examinations. Special courses and qualifications are needed for this purpose.

Because of the unique physical and physiological conditions encountered in diving, medical standards for divers differ considerably from those of other sports. As a result it is sometimes necessary for a diving physician to advise a prospective diver against diving because of a disqualifying condition. Often the recipient of this advice is supremely physically fit, and some have been of Olympic standard. These individuals understandably find it difficult to comprehend how a physically fit athlete is not necessarily fit to dive, medically.

To those with more knowledge of diving physiology it becomes obvious that even the highest standard of physical fitness will not protect a diver with lung cysts or asthma from a diving death.

The examining physician must consider many factors when conducting a diving medical examination. Almost 10% fail the medical and 10–15% incur specific diving limitations or advice, for safety reasons.

PSYCHOLOGY

The ideal diver is probably the cool James Bond like character we would all like to be - stable, calm under stress, able to endure physical and mental pressure, not prone to anxiety, able to conveniently ignore danger, slightly overweight and perhaps not surprisingly, a fluent liar.

Psychological stability is difficult to evaluate during the medical examination. Some clues may be gained from the history of sporting activities and occupation. Often the diving instructor is best able to evaluate the diver's psychological make-up during the course of instruction.

AGE



Fig. 38.1

Ideally the trainee diver should be aged between 18 and 35 years although exceptions can be made at both extremes of age. Divers over 45, if complying with the medical standards should be acceptable, but may require special tests such as a cardiac risk assessment.

Divers younger than 16 require very careful supervision during and after training because of their often smaller stature, limited strength and (most importantly) emotional immaturity. A buddy line to an experienced adult diver is recommended during the training of youthful divers. The mature and experienced buddy of an adolescent diver should take control of the dive and remember that his buddy may be an unreliable rescuer if difficulties arise. Most reputable medical authorities will not certify divers under the age of 15–16 years, without qualification. This does not prevent younger divers being given a limited "diving experience" by qualified diving instructors under very strict and controlled conditions, and provided they are medically fit.

OCCUPATION

Pilots and aircrew are advised of the risks associated with flying after diving. Musicians, sonar operators, cardiologists, pilots and others reliant on excellent hearing for their livelihood are informed of the small but real risk to their hearing, or development of tinnitus, should they suffer ear barotrauma.

MEDICATION

Any illness requiring drug treatment needs careful consideration because either the illness or the drug may compromise diving safety. Sedatives, tranquillisers, antidepressants, antihistamines, anti-diabetic drugs, steroids, anti-hypertensives, anti-epilepsy drugs, alcohol and hallucinatory drugs such as marijuana and LSD all place the diver at risk. See Chapter 37 for more specific details.

Some antibiotics may have no direct adverse effect on diving, but the condition for which they were prescribed may have.

Experience and experiments indicate that many drugs which affect the brain have unpredictable effects on a diver exposed to the very high pressures encountered in deep diving.

HEART

Most heart diseases or abnormalities of heart rhythm are incompatible with safe diving and are disqualifying conditions. They can often be inferred from the personal or family history, clinical examination, biochemical tests or electrocardiograms (ECGs). The blood pressure should be normal for the age of the diver. See Chapter 35.

OBESITY

The overweight person is more prone to decompression sickness when air diving and is likely to have a reduced level of physical fitness. Most physically fit obese individuals may dive safely with appropriate reductions of the allowable durations of dives.

LUNGS

Lung disease is a disqualifying condition. The diver needs normal lung function to allow a reserve of respiratory function to cope with exertion and to permit easy air flow from the lungs to avoid pulmonary barotrauma. The lungs must be very elastic to enable them to stretch during sudden volume changes on ascent. A history of asthma, chronic bronchitis, bronchiectasis, fibrosis, cysts, spontaneous pneumothorax, chest injury or chest surgery are disqualifying conditions.



Fig 38.2

The doctor may be able to detect localised airway obstruction (which can lead to a burst lung) by listening to sounds made in the chest when the diver breathes deeply and rapidly. The history and **respiratory function tests** (expiratory spirometry) aid in the assessment. Occasionally radiological screening (Chest X-ray, CT scan etc.) may be necessary.

There was a dramatic drop in the incidence of burst lung in Navy divers after the institution of these standards.



Fig. 38.3

Diving candidate blowing into a "Spirometer" to assess lung function. These devices have been largely replaced by digital expiratory spirometers, standardised for the specific population being tested.

EAR, NOSE AND THROAT

The ears, nose, throat and sinuses account for most diving induced illnesses. Any acute infection such as a cold will temporarily disqualify a candidate. A history of chronic or recurrent allergies, hay fever, sinusitis, tonsillitis, or tooth decay needs special assessment. Diving should be avoided while so affected. A deviated nasal septum (often appearing as a crooked nose) can cause obstruction of the sinus openings. All these factors can predispose to **sinus** or **ear barotrauma**.

The ears are carefully examined. The outer ear must be free from infection and not blocked with wax. The eardrum must be seen to be moved voluntarily during the **Valsalva**, or other equalising manoeuvre. An eardrum which has been scarred from previous perforation may be weakened. The examining physician, by viewing the ear drum while the diver attempts middle ear equalisation, can advise on correct techniques to be used when diving.

The hearing function test (**pure tone audiogram**) measuring hearing up to 8000 Hz is performed. Any significant hearing loss is regarded seriously since there is a risk of further hearing loss if **barotrauma** to the ears occurs during the diver's exposures.

Damage to the hearing organ may also be associated with disturbance of the balance organ. A special type of balance test is used to detect this, called the Sharpened Romberg, and further investigation is by an electronic measurement (electronystagmogram) if necessary. It is important to detect any balance organ dysfunction since it can lead to **vertigo** and **vomiting** underwater.

EYES

Good **vision** is essential for the diver to see his boat or buddy, if he surfaces some distance away. A diver who has impaired vision can have **corrective lenses** included into his face mask, but should always dive with a visually fit buddy in case the mask is lost or broken during the dive. See Chapter 5.

Contact lenses can pose problems and advice is needed about these. Hard lenses can trap bubbles between them and the cornea, causing pressure damage. Soft lenses are susceptible to loss – especially during mask removal. These divers are advised to keep the eyes closed when removing the mask, either underwater or on the surface. See Chapter 32.

The operation of **radial keratotomy**, used to surgically correct short sightedness, can cause problems. With this procedure, the cornea is cut radially in a sunburst pattern to change the curvature of the cornea. These cuts weaken the cornea which is prone to burst if the eye is bumped or subjected to external pressure reduction. If such a diver develops face mask squeeze (see Chapter 12), the eyeballs may actually rupture. Anyone who has undergone this operation should not dive. Most modern techniques, such as Laser resections for myopia, involve only minimal damage to the cornea, and are not a problem.

Colour vision is of lesser importance, apart from a few professional diving situations involving colour coded cylinders or wires (involving explosives).

BRAIN

Any disorder of the nervous system will complicate and confuse diagnosis and treatment of diving illnesses such as cerebral air embolism and decompression sickness.

Epileptics, even if controlled by drugs, should not dive as an epileptic fit underwater could prove fatal. The higher partial pressures of oxygen encountered during a scuba dive may render these persons more vulnerable to such attacks. Hypoxia, hyperventilation and sensory deprivation can aggravate fits. Many divers have had their first fit underwater.

Migraine is often made worse by diving (see Chapter 32). Severe migraine attacks leading to incapacity have occurred during dives in previously mild sufferers. It may also complicate recompression treatments. If certain precautions are observed some migraine sufferers can engage in limited diving in reasonable safety.

GENERAL CONDITIONS

Other diseases of the body such as **diabetes mellitus** (see Case History 33.6), severe **kidney** or **liver** disease also increase the risks of diving.

Muscle, bone and **joint** diseases or injuries can predispose to decompression sickness and make diagnosis and treatment of this disorder more difficult. Fatigue may be induced more easily.

Professional divers or those who frequently undertake decompression diving may require **long bone radiology or scanning** (see Chapter 17) to establish a baseline in the event of bone abnormalities developing, and for legal reasons. Because of the low risk of dysbaric osteonecrosis, the cost and the potential hazards posed by radiation exposure, these are not usually recommended for recreational divers.

A history of **motion sickness** is significant because it interferes with safe diving and it is difficult to vomit through a demand valve. Divers with a propensity to this condition need advice from the physician on remedies for seasickness which are compatible with safe diving (see Chapter 32).

Smoking diminishes physical fitness and can predispose to lung, sinus and ear barotrauma.

Pregnancy should preclude diving (see Chapter 8).

PHYSICAL FITNESS

This refers to the strength and speed, so necessary to athletes. It includes muscular, cardiac and respiratory capabilities. It is important to divers, as they are often called upon to exert themselves, to survive. One reasonable standard is to require an ability to swim, unaided, a distance of 200 metres in less than 5 minutes for recreational divers who do not subject themselves to difficult conditions. For professional or competent divers, this could be reduced to 4 minutes.

Medical fitness for diving refers to the freedom from illness likely to prejudice diving safety. **'Physical fitness' does not necessarily equate with 'diving medical fitness'.**

It is not uncommon for physically fit young individuals to feel quite distressed when advised against scuba use by diving medical practitioners.

MEDICAL EXAMINATION FORMAT

There is little doubt amongst responsible diving instruction groups and diving medical associations, that mandatory full and comprehensive medical examinations should be performed on all divers before commencing scuba training. It is also needed before using scuba apparatus – even in such shallow and apparently safe locations as a swimming pool.

During a recent workshop on diving medical examinations, the following consensus was achieved with this advice for recreational divers:

- All diving candidates must be examined according to an established diving medical Standard. An example is the South Pacific Underwater Medicine Society (SPUMS) Medical Format (included in this Chapter) prior to commencing any use of scuba apparatus – even if only in a pool.



Fig 38.4

- The medical examiner must have been trained appropriately (at a recognised course) in diving medicine.
- Should any doubt exist as to the 'fitness' of an individual, then that person must be referred to a specialist diving medical practitioner (i.e. one with extensive training and experience in diving medicine).

This textbook is not aimed at instructing medical practitioners in Diving Medicine – although it will serve as a useful primer for those interested in this type of medicine. A list of recommended courses of instruction and reading texts is included in Appendix A.

A copy of a typical **Diving Medical Format** follows. It is suitable for candidates wishing to experience Scuba diving or to subject themselves for diver training. It must be performed and interpreted by a physician trained in diving medicine by an accredited body.

It comprises 3 sections:

- (1) **Medical history**
- (2) **Diving [and diving medical] history**
- (3) **Clinical examination and investigations.**

Each is necessary and every item except for identification data, is of relevance to diver safety and diving limitations.

APPENDIX B
PRE-DIVE MEDICAL FORM FOR PROSPECTIVE ENTRY-LEVEL SCUBA DIVERS
The first two pages to be completed by candidate.

1	Surname	Other Names	2	Date of Birth
			4	Sex: Male Female
3	Address		5	Telephone (Home)
6	Principal Occupation		7	Telephone (Work)
8	Intended Dive School			
9	Do you participate in any regular physical activity?		Yes	No
10	Description of activity			
11	Do you smoke?		Yes	No
12	Do you drink alcohol?		Yes	No
13	How many drinks a week?			
14	Are you taking any tablets, medicines or drugs?		Yes	No
	List:			
15	Do you have any allergies?		Yes	No
	Details:			
16	Have you had any reactions to drugs or medicines or foods?		Yes	No
	Details:			

Have you ever had or do you now have any of the following? Tick Yes or No.

	Yes	No
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Notes on History

	Yes	No
54	Chronic or persistent cough	
55	TB	
56	Pneumothorax ("collapsed lung")	
57	Frequent chest colds	
58	Asthma or wheezing	
59	Use a puffer	
60	Other chest complaint	
61	Operation on chest, lungs, or heart	
62	Indigestion, peptic ulcer or acid reflux	
63	Vomiting blood or passing red or black motions	
64	Recurrent vomiting or diarrhoea	
65	Jaundice, hepatitis or liver disease	
66	Malaria or other tropical disease	
67	Severe loss of weight	
68	Hernia or rupture	
69	Major joint or back injury	
70	Limitation of movement	
71	Fractures (broken bones)	
72	Paralysis or muscle weakness	
73	Kidney or bladder disease (cystitis)	
74	In a high risk group for HIV or AIDS	
75	Syphilis	
76	Diabetes	
77	Blood disease or bleeding problem	
78	Skin disease	
79	Contagious disease	
80	Operations	
81	In hospital for any reason	
82	Life insurance rejected	
83	A job or licence refused on medical grounds	
84	Unable to work for medical reasons	
85	An invalid pension	
86	Other illness or injury or any other medical conditions	
Have any blood relations had		
87	Heart disease	
88	Asthma or chest disease	
89	TB	
Females Only		
90	Are you now pregnant or planning to be?	
91	Do you have any incapacity during periods?	

92 Date of most recent chest x-ray

Previous Diving Experience		Yes	No
93	Can you swim?		
94	Have you ever had any problem during or after swimming or diving?		
95	Have you ever had to be rescued?		
96	Do you snorkel dive regularly?		
97	Have you tried scuba diving before?		
98	Have you had previous formal scuba training?		
99	Year		
100	Approximate number of dives		
101	Maximum depth of any dive		
102	Longest duration of any dive		

I certify that the above information is true and complete to the best of my knowledge and I hereby authorise Dr. _____ to give medical opinion as to my fitness, or temporary or permanent unfitness to dive to _____ (Diveshop). I also authorise him or her to obtain or supply medical information regarding me to other doctors as may be necessary.

Signed:

Date:

Chapter 39

FIRST – AID KIT

Certain drugs and equipment are of value in a diving accident and a diving team could reasonably be expected to acquire and carry these on diving expeditions. Training in the use of these, as well as in resuscitation, is of great importance.

FIRST-AID MATERIALS

For shark attack or trauma, large sized thick **cotton pads** (more than 20 cm square) with 10 cm **crepe bandages** (6 of each) are useful to make **pressure dressings** to stop bleeding and also for pressure bandages to reduce venom absorption. If obtainable, **shell** dressings of the type used by the military are ideal for this purpose. They can sometimes be obtained from army disposal stores.

A **rubber bandage** 10 cm wide ("esmark" bandage obtainable from a medical equipment supplier) for use as a **tourniquet**. When wrapped tightly around the limb this is the best form of tourniquet. It covers a wide area, effectively stopping blood flow to the limb while minimising damage to tissues under the tourniquet.

Small adhesive skin **dressings** such as Elastoplast or Band-Aids.

Surgical instruments — scissors, artery forceps, fine forceps, disposable scalpel blade, disposable syringes and needles.

An aluminised **thermal blanket** such as a "Space blanket" to protect divers suffering from hypothermia.

Heat packs — of value in treating fish and jellyfish stings.

Cold packs — of value in treating jelly fish sting and general muscular strains

Eye irrigation solution.

Torch, pen and paper (for recording purposes).

RESUSCITATION EQUIPMENT

- **Airways (Guedel type)** in two adult sizes are useful if a victim loses consciousness and develops airway obstruction, or if artificial respiration is needed. A positive pressure air system (such as an AMBU Bag) is of value in combination with the airway for prolonged artificial respiration.
- **Oxygen Resuscitation Equipment.** A supply of oxygen and equipment to administer it can be lifesaving in some diving accidents. Devices as described in Chapter 40 should include a complete oxygen supply and delivery system in a robust portable container.
- A **large oxygen cylinder** with appropriate adaptors should be available if diving at a distance from diving medical facilities and recompression chambers.

An **underwater oxygen** system (appendix C) for recompression therapy by more sophisticated groups, in remote areas.

MEDICATIONS FOR DIVING PROBLEMS

- Household **vinegar**, preferably in a one litre container, to neutralise adherent stinging cells of box jelly fish and some other jelly fish. Household **bleach** is useful for sterilising coral cuts.
- **Local anaesthetic spray or ointment** (lignocaine) to relieve the pain from minor stings from animals such as Portuguese man-o-war and other jellyfish stings. **Solacaine** or other anti-burn preparations such as **Tannic acid** sprays may be efficacious for this purpose.
- Topical **antibiotic powder** to prevent infection from coral cuts and other minor injuries.
- **Skin antiseptic solution** such as chlorhexidine for cleaning wounds contaminated with dirt.
- Broad spectrum **antibiotic tablets** (e.g. erythromycine, doxycycline) to initiate treatment for serious infections, otitis externa, otitis media, sinusitis, and coral cuts etc.
- **Prophylactic ear drops** such as commercial preparations of Aqua Ear, Vosol or Otic Domoboro.
- **Therapeutic ear drops**, including antibiotic and steroid combination, for outer ear infections.
- **Local anaesthetic for injection** such as lignocaine 1% (without adrenalin) for wounds from stone fish and other fish stings. Up to 15 ml of this solution can be injected into the stung area in an adult and repeated every 2 hours if necessary.
- **Antivenoms** — depending on the geographical location.

GENERAL MEDICATIONS

- **Anti-diarrhoea** tablets such as diphenoxylate ("Lomotil") or loperamide ("Imodium").
- **Analgesics** (pain killers) such as paracetamol (acetaminophen). Aspirin, or drugs containing this substance, may be unpredictable and hazardous and are best avoided.
- **Ultra-violet** blocking sunscreen (SP15+ or greater). A **1% hydrocortisone cream** is useful to treat sunburn, allergic dermatitis or itching.
- **Anti-Seasickness** tablets (see Chapter 32).
- **Decongestants** — pseudoephedrine tablets, and topical nasal sprays.
- **Topical antibacterial and antifungal** preparations, such as Cicatrin or Neosporin.

TRAINING

A diving team venturing to a remote locality should have at least one member (preferably two in case that one becomes the victim of an accident) trained in first aid relevant to divers. Resuscitation and oxygen administration requires expert training and supervision. Training in the use of injections is an advantage, both for the administration of local anaesthetics, antivenoms and other drugs under the direction and advice of a distant medical specialist.

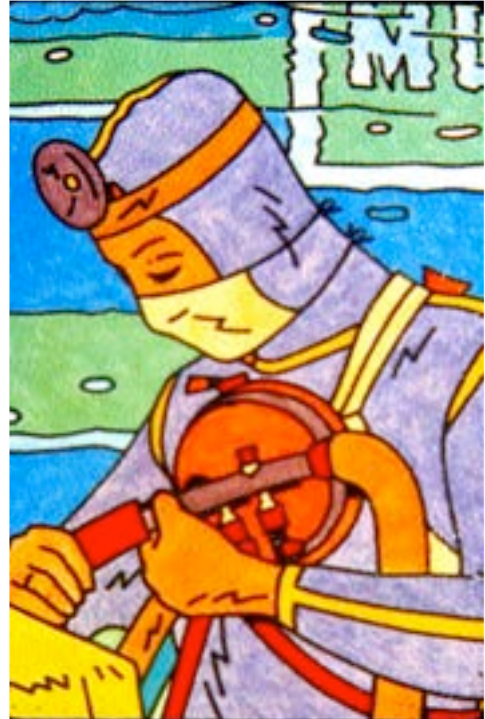
MEDICAL INFORMATION

Perhaps the most valuable addition to any first aid box is a source of information. This should include diving **medical texts** (see appendix A) and general **contact numbers** (see appendix B & D) for both medical assistance and recompression chamber availability. This should be supplemented by local contacts and phone numbers of knowledgeable divers and diving physicians.

A copy of **this book** should remain with the First-aid kit.

Also in the kit should be a list of its **contents**, including purchase and expiry dates of the drugs.

**INFORMATION NEEDED
about a DIVING ACCIDENT.
CHECK LIST**



Name of informant

Name and age of victim

TELEPHONE NUMBER to return call, or if disconnected

Geographical location (+ local medical facilities or RCCs)

Case history

Initial symptoms, including time of onset

Description of clinical symptoms + progress

Signs of illness

Negative findings (symptoms NOT present e.g. micturition, dyspnoea, skin wounds)

First aid given (including oxygen, amount and method)

Dive details: Profile, gases, deco,(+ recent dives)

Personal history

Diving

Illnesses – diving and general

Medications and surgery

Any other persons injured

First aid facilities and/or medical personnel available on site

Follow up arrangements

Medevac possibilities

Chapter 40

OXYGEN THERAPY TECHNIQUES

100% Oxygen (O₂) therapy is an essential technique in first-aid treatment of many diving emergencies. Unfortunately, the correct use of O₂ equipment is often poorly understood by divers. See Chapter 16 for details of the use of O₂ in decompression sickness.

OXYGEN BREATHING EQUIPMENT

The apparatus for administering O₂ is not unlike a scuba system. It comprises a **cylinder** which holds the oxygen at a pressure comparable to scuba tank pressure, and a pressure **reducing valve (regulator)** connected either to a **demand valve** or to a **constant flow system**. There is a need to avoid the use of flammable lubricants (such as oils and silicone grease) which can cause explosions in the presence of O₂.

Oxygen can accelerate burning, cause spontaneous combustion, and can make ordinarily non-combustible materials burn furiously, so its use requires a strict fire prevention attitude. Oxygen should not be administered near heat sources, and smoking near O₂ is even more hazardous to your health than usual. Oxygen should not be used in poorly ventilated areas where high concentrations can build up. All equipment must be kept clean and the cylinder valve should be turned on slowly and the system purged before O₂ is given to the patient. If the patient is unconscious, always check that the apparatus is working by you or an attendant breathing on it first.

The O₂ can be delivered in high or low concentrations depending on the apparatus used. With a constant flow system, the O₂ can be delivered to the patient through either a cheap disposable (usually loose fitting) plastic **oxygen mask**, **nasal prongs**, a **nasal catheter** or a **bag-valve-mask resuscitator system**.

Where the administration of 100% O₂ is required (e.g. in first aid for decompression illness) this means that nothing but O₂ is inhaled by the diver. Many of the O₂ masks commonly used

in medicine have 100% O₂ delivered to the mask, but, the patient inhales only about 25-50% O₂ because the design of the mask allows this O₂ to mix with air, which dilutes the final breathing mixture. The disposable plastic O₂ masks in common use in ambulances and hospital casualty rooms are of this type.

These are **NOT** adequate for treating serious diving injuries.

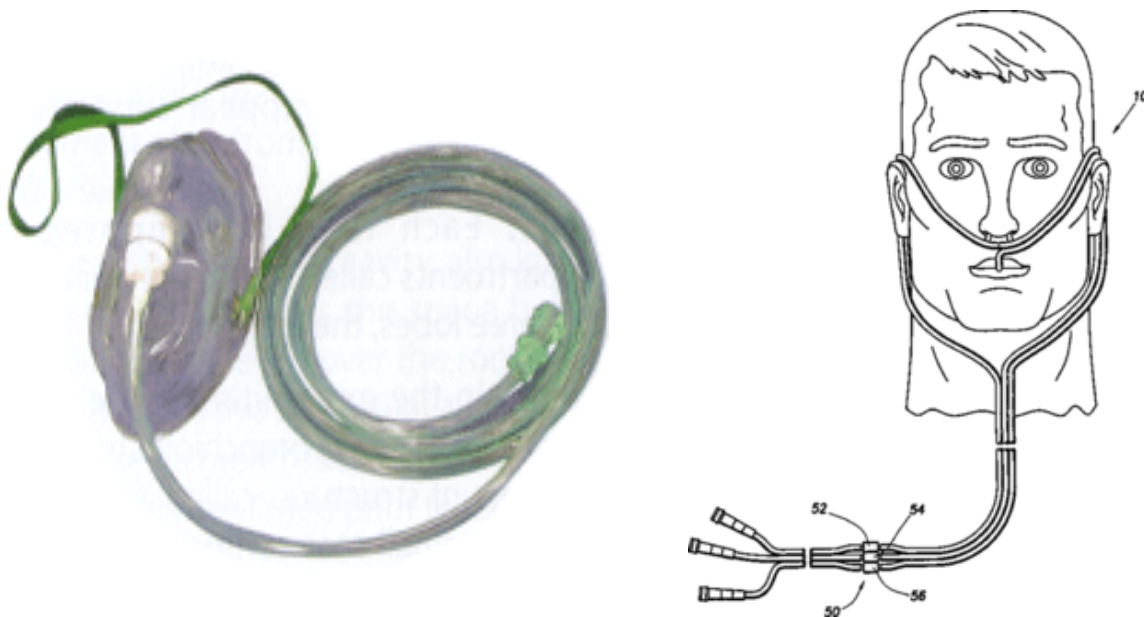


Fig. 40.1
These oro-nasal plastic masks, catheters and nasal prongs
are not adequate for treating divers with 100% O₂.

As a general rule, **decompression sickness**, **gas embolism** (and other manifestations of **pulmonary barotrauma**), carbon monoxide poisoning and **near drowning** cases should be given 100% O₂ from the outset.

Other diving accidents which produce shock or hypoxia can sometimes be helped with lower concentrations of O₂. In general, if the patient is cyanosed (blue) they need O₂ in a sufficient concentration to make them pink again. Usually, 100% O₂ is needed for treatment of diving accidents.

CONSTANT FLOW SYSTEMS

Such devices deliver a constant flow of O₂ to a mask or an alternative delivery system.

There are several types of plastic **disposable oxygen masks** available, the **Hudson** mask being typical. Each is normally accompanied by instructions which specify the correct O₂

flow to use, most masks using a flow of about 4–6 litres per minute. These masks allow the O₂ flow to be diluted by inspired air, the flow of which varies during inspiration and peaks at about 30 litres per minute. Because of this, the concentration of O₂ which the patient actually inhales is reduced from 100% to about 40 or 50%.

To increase the inspired O₂ percentage, increase the O₂ flow. Unless this is increased beyond 30 litres per minute an inspired percentage of 100% is not attainable. Such high flow rates rapidly deplete most divers' O₂ supply.

A device known as a **nasal prong** (see above photo) is available which delivers O₂ by small tubes directly into the patient's nostrils. An elastic head strap holds the prongs in place. This system has a similar efficiency to the common loose fitting O₂ mask but is more comfortable and more effective for long term use because it is less likely to dislodge when the patient sleeps.

These systems are acceptable when O₂ supplementation in low concentrations is all that is required. Cases in this category would include typical general medical conditions found in hospitals such as heart attack, and mild recovering cases of near drowning, salt water aspiration syndrome and shock associated with serious trauma or shark attack.

HIGH CONCENTRATION OXYGEN SYSTEMS

When near 100% O₂ delivery is required in cases such as decompression sickness, pulmonary barotrauma or near drowning, a more efficient O₂ delivery system is necessary.

This can be achieved by a **demand valve**, an O₂ circuit incorporating a **rebreathing bag**, or a bag–valve–mask device with an O₂ inlet and a reservoir bag with a very high O₂ flow rate.

Demand Valves

The simplest and most effective way to deliver 100% O₂ is via a **demand valve** - like a second stage regulator.

Some demand valves have been specially designed for O₂ administration. Some, such as the Oxiden and LSP demand valves, are designated to provide O₂ only to spontaneously breathing patients. Others, such as the Robert Shaw (which is used on the Oxy Viva, marketed in Australia) and the Elder demand valves, can produce 100% O₂ to a spontaneously breathing patient as well as provide O₂ resuscitation to a non-breathing casualty via a manual trigger. DAN also supplies an oxygen kit with this type of valve.

These demand valves are usually used with a tight fitting anaesthetic type mask. However, some can be fitted with a scuba mouthpiece to provide O₂ to a breathing diver (in this case the diver's nose should be sealed with a nose clip or by some other means).

In an emergency in remote locations, certain demand valves can be adapted to deliver O₂, with an adaptor connected to an O₂ reducing valve, or alternatively by connecting the diver's first stage regulator to an O₂ cylinder, using the specialised adaptor. It is essential if this system is used that all components in the breathing system (including the lubricants) are O₂

compatible. Otherwise the diving illness may be complicated by fire, explosion, or shrapnel injuries. When using this type of system the regulator should be tested for safety by purging it before anyone breathes from the system.

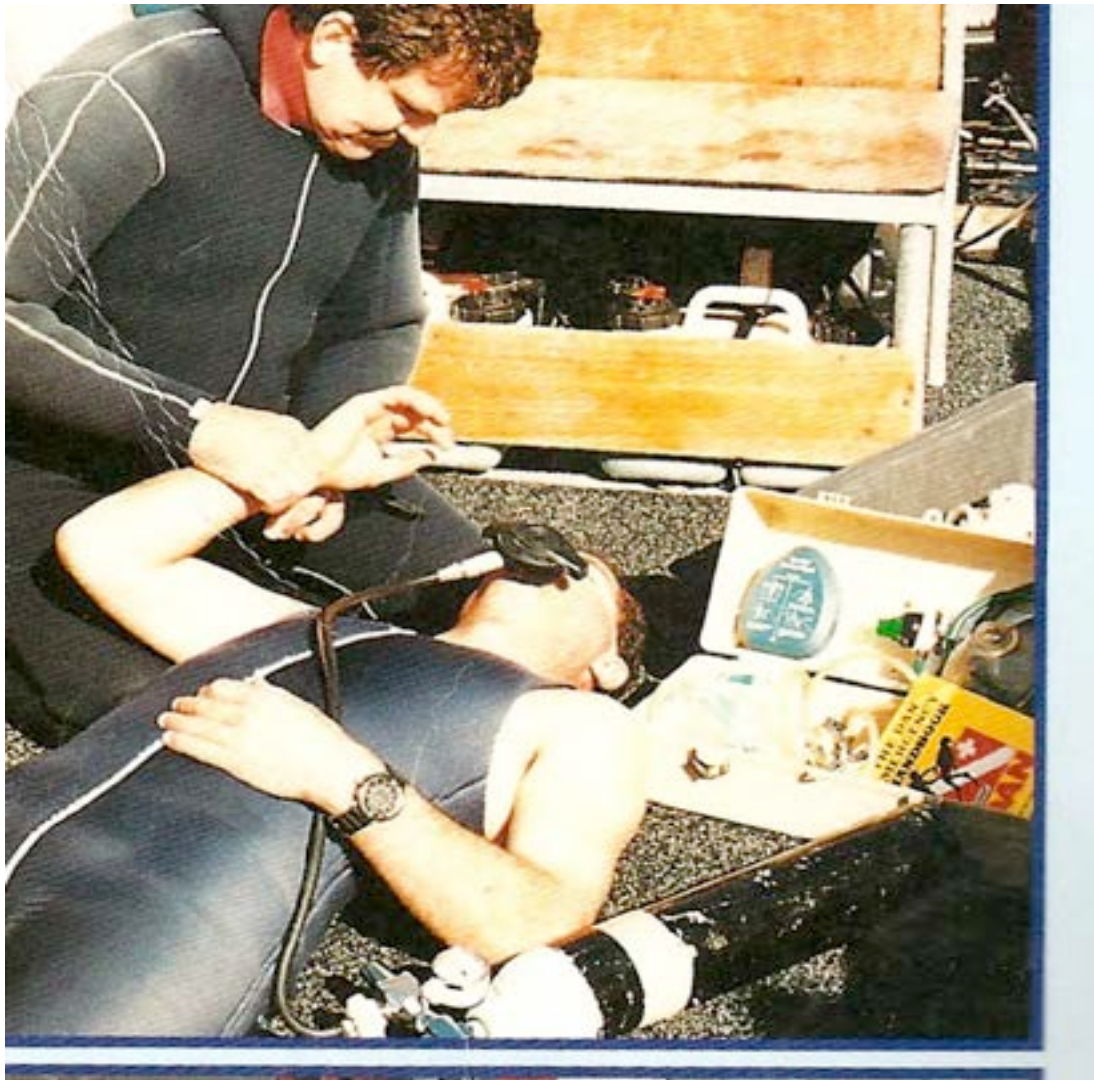


Fig. 40.2

A system designed by DAN (USA) specific for divers and incorporating a demand valve for 100% O₂ inhalation. The diver must be breathing spontaneously.

Rebreathing System

Oxygen use can be reduced if the diver breathes from a system which permits him to rebreathe some of his exhaled gas. This requires the nitrogen to be flushed from the breathing circuit after a few minutes rebreathing, and the exhaled CO₂ to be absorbed in a soda line (or similar chemical) canister. There are commercially available units using this system. Technical divers frequently use diving sets that can be used for 100% O₂.

PSA Oxygen Concentrator

These devices use a molecular sieve to filter nitrogen from air and deliver high concentrations of oxygen. They are available in a portable size and can be battery powered.

Bag-Valve-Mask Respirator

These devices comprise a self inflating rubber or plastic bag and a valve system which allows the patient to be ventilated with air through a mask by squeezing the bag. Oxygen can be piped into the circuit to increase the inspired O₂ concentration. If a reservoir bag is attached to the inflation bag, the inspired O₂ concentration can be increased to almost 100% by using a large O₂ flow (approx. 12–14 litres per minute), providing a good seal is achieved and the patient's respiratory volume is not excessive. The valve system also allows the patient to breath spontaneously from the device. Details of the operation of these devices vary between manufacturers and are outlined in the instruction manual supplied.



Fig. 40.3

A resuscitator showing anaesthetic-type mask, hand-squeezed breathing bag, oxygen reservoir bag and head strap to secure mask to diver's face.

Multiple Systems

Most countries have a variety of multi-purpose near 100% O₂ delivery systems available. Aga, AG, CIG, Ambu, LSP, Drager and Laerdal and DAN manufacture such units. Self-contained O₂ therapy units and resuscitation devices are now available with a demand valve allowing the patient to either breathe spontaneously out of the device, be given positive pressure ventilation by manual pressure, or use a breathing bag. A flow meter can be fitted to another part of the system, to allow O₂ to be delivered to a mask at a high continuous flow rate. The details vary between resuscitators and are explained in instructions supplied with them. The entire device should be contained in a sturdy rustproof metal or heavy duty plastic box which is compact, water resistant and easy to carry. It should have an adaptor and be able to be supplemented by connection to a larger O₂ cylinder, if necessary.

GENERAL INFORMATION

Oxygen Toxicity

(See Chapter 21). 100% O₂ will cause some reversible damage to lungs after 18–24 hours. This has to be balanced against the benefit of the condition being treated and this will usually be physician's decision. If medical advice cannot be obtained, in decompression sickness and gas embolism cases, or with near-drownings or carbon monoxide toxicity, O₂ toxicity is generally the lesser of two evils, and it is usually best to continue giving 100% O₂ until expert advice to the contrary is given.

Oxygen toxicity is generally not a consideration with low concentration devices as they do not delivery much more than 40% O₂, which is probably below the threshold for toxicity for the estimated duration of exposure.

Contraindications to O₂ Therapy

In addition to the problems of O₂ toxicity, there are some problems associated with oxygen usage in the general community.

There is a theoretical risk to premature babies (eye damage) and to sufferers of emphysema (respiratory depression). Neither of these groups are numerous in the diving population.

In recent years a problem of sensitivity to O₂ has emerged in cancer sufferers who have been treated with the drug **Bleomycin**, and similar substances. These people can suffer from severe, permanent lung damage if given O₂ in concentrations greater than 21% (air). This problem is not likely to be frequently encountered, as these people should be excluded from scuba diving.

Practicalities of O₂ Administration

The main disadvantage of resuscitation apparatus is the limited O₂ supply available from the contained O₂ cylinder, because sufficient O₂ must be supplied to allow for transport of the diver from the accident site to an appropriate medical facility. This can be overcome by carrying additional cylinders or by an adaptor which allows a connection to a larger O₂ cylinder. Estimate the rate of consumption of O₂, the supply available, and plan accordingly. During transport, and whenever O₂ is given, attention must be paid to adequate ventilation to prevent O₂ build up to dangerous levels.

All divers are encouraged to undergo additional training in resuscitation and O₂ administration. This should be mandatory for dive masters and dive instructors.

Cases such as gas embolism and decompression sickness generally require **100% inspired O₂** from the outset. A system capable of delivering 100% O₂ must be chosen. Even then, an ill fitting mask may allow air to be breathed around the seal, diluting the O₂. Attention to the mask fit and attachment is necessary to prevent this. It is especially likely if the patient sleeps. If a demand valve is used, make sure the patient does not breathe air through the nose at the same time. This can be prevented by using a nose clip (improvise if necessary) or a diver's face mask.

Whenever O₂ is administered there is a serious **fire hazard** since increased concentrations of O₂ accelerate burning and can make ordinarily non-combustible materials burn furiously. The area where the O₂ is administered should be well ventilated and sources of ignition and combustible materials (including cigarettes) should be avoided. The system should be turned on slowly and should be tried and running before it is applied to the patient's face.

Further information may be obtained from the text "Oxygen First Aid for Divers"— by John Lippmann, J.L. Publications, Australia.



Fig 40.4

A portable oxygen resuscitator. This system allows positive pressure ventilation with 100% O₂ for a non-breathing diver, or passive demand supply for a breathing diver. A long supply hose can be fitted to a larger cylinder containing 100%O₂ for prolonged use. The airways and suction appliance are included

Chapter 41

TRAINING & SAFETY CHECKLIST

SAFETY INFORMATION AND BACKUP

- Diving manuals and library (see Appendix A).
- Diving medical and safety manuals and texts (see Appendix A).
- Diving medical organisations (see Appendix B & D).
- Medical Insurance — DAN has organised this for North Americans, world wide.

DAN.SEAP covers the area from the eastern Indian ocean to the western Pacific ocean
BSAC arranges insurance from the UK/Europe.

Most diving shops and organisations advise on the relevant local contacts.

DIVER TRAINING

- Physical fitness – 200 metres unassisted swim in less than 5 minutes.
- Medical examination fitness for diving by a qualified diving physician.
Includes pure tone audiogram and expiratory spirometry.
- Entry level diving certificate. Qualified by a diving instructor from a reputable training organisation.
- Advanced level training, specialty courses such as buoyancy training.
- Diving rescue and resuscitation training.
- Specialised courses for the specific diving environments (cave, deep, etc.).

DIVE PLAN

- Boat operator's safety and equipment check.
- Dive plan — includes terrain information, navigation and emergency plan, maximum depth, air supply, ascent rate, safety and decompression stops.
- Basic diving equipment check and maintenance.
- Dive responsibility, documentation and buddy system.
- Diving rescue equipment — includes BC, emergency air supplies, alarm signals, etc
- Lost diver strategy, including EPIRB in open ocean.
- Diving rescue plan - includes boat / buoy / diver backups.
- First-Aid kit and resuscitation equipment. (See Chapters 39 and 40).
- Emergency contact numbers (local and elsewhere).

FOR DIVE OPERATORS

- Register trip plan with local maritime or port authority
- Rescue and resuscitation equipment and training
- Onboard observer and recorder (diver in/out times)
- Communication system. Satellite phone, radio etc
- A good lawyer!

CHECK LIST FOR DIVING ACCIDENTS

INFORMATION NEEDED if reporting a DIVING ACCIDENT.
for both reporter and recipient

Check List

- NAME of informant
- NAME AND AGE of victim
- TELEPHONE NUMBER to return call, or if disconnected
- Geographical LOCATION (+ any local RCCs)
- CASE HISTORY
 - Initial SYMPTOMS, including time of onset
 - Description of clinical symptoms + progress
 - Signs of illness
- NEGATIVE FINDINGS (symptoms that are NOT present)
- FIRST AID given (including oxygen, amount, method and time)
- DIVE DETAILS Profile, gases, deco, (+ recent dives)
- PERSONAL HISTORY
 - Diving
 - Illnesses – diving and general
 - Medications and surgery
- Any other persons injured
- First aid FACILITIES and/or medical personnel available on site
- Follow up ADVICE and ARRANGEMENTS
- Possibility of medevac

n.b You may think that you have read this before, in Chapter 39. You have. In emergencies, either chapter may be accessed.

Chapter 42

RESUSCITATION REVIEW

It is not possible to learn the techniques of resuscitation from a book. To acquire these skills, the authors recommend that all divers undertake a resuscitation course from one of the many organisations worldwide which teach these techniques. Once learnt, the skills need to be practised regularly, just as do diving emergency procedures.

The protocol used here is meant as a reminder to divers who have already been trained in resuscitation. It is based on that recommended by the Australian Resuscitation Council. Organisations in other countries may have slightly different protocols, but if they come from reputable organisations they should be equally effective.

WHAT IS RESUSCITATION?

Resuscitation is the restoration or preservation of life using maintenance of the A–B–C — Airway, Breathing, and Circulation — to preserve oxygenation to vital tissues. The most important tissue to protect from hypoxia is the brain.

Expired Air Resuscitation (EAR) is usually the best method of initially ventilating the lungs. If the rescuer has the equipment and skill to ventilate the victim with an Oxygen Resuscitator, then that is preferable.

ASSESSMENT OF THE DIVING CASUALTY

In addition to the following measures, it is important to protect the victim from further injury and to control bleeding if present.

Is the Victim Conscious?

Most problems arise in an unconscious victim. If the victim appears unconscious, confirm this by shouting at him and squeezing the shoulder. If the victim does not respond, he is unconscious.

If the victim is **conscious** he will normally take care of his own airway and breathing. Exceptions to this are the sea snake, blue ringed octopus or box jellyfish (Chironex) envenomation, where the victim may be conscious but paralysed. In these cases the victim will not respond to shouting, so the management for an unconscious victim (which is appropriate) will be undertaken.

If the victim is **unconscious** he will be in danger of hypoxic hypoxia from obstruction of the airway or inhibition of breathing, or of stagnant hypoxia from lack of circulation (see Chapter 20).

With an unconscious victim, take care of the following systems:

- **AIRWAY**
- **BREATHING**
- **CIRCULATION**

This is easily remembered by the mnemonic — A B C.

Maintenance of airway, breathing and circulation takes precedence over other forms of care. Without these functions, the victim is certain to die.

A — AIRWAY

An unconscious victim loses muscle control. Loss of control of the muscles of the throat and tongue can cause the airway to become obstructed. This is particularly likely when the victim is lying on his back, mainly due to the tongue falling backwards into the throat, due to gravity.

The airway can be further obstructed by vomit, saliva or foreign material. This would normally be swallowed or spat out by a conscious person, while any material which entered the larynx or trachea would elicit the reflexes of coughing and laryngeal closure. These reflexes may be lost in the unconscious patient.

Clear the airway

In **cases of near drowning** and **gas embolism** (both cases probably best managed initially on the side)

To prevent airway obstruction, turn the victim on his side (left side down for gas embolism).

Sweep any foreign material from the mouth with the fingers.

Position the mouth slightly downward to allow any fluid to drain out. (In these cases, there is frequently fluid, vomit or other material in the airway).

The side (lateral) position with the mouth turned slightly downward will help any fluid to drain from the airway.

In **other cases** secure the airway with the victim on their back. (See the chapter on pulmonary barotrauma for special considerations in this condition).

Tilt the head backwards, to open the airway, if necessary apply the chin lift maneuver (taught in resuscitation courses) if the airway remains obstructed. If there is a possibility of spinal injury, further injury by unnecessary movement of the spine should be avoided when positioning the patient, but clearing the airway takes precedence.

B — BREATHING

Even with a clear airway the victim may not breathe because of respiratory muscle paralysis, cerebral injury, hypoxia, cardiac arrest or other reasons.

Check for breathing – look, listen and feel.

Look and feel for respiratory movements of the chest and abdomen. Listen and feel for air moving from the nose and mouth. Occasional gasping respirations should be treated as not breathing. If the victim is making respiratory movements ensure that the airway is clear.

Recognition of airway obstruction

If the airway has been cleared of foreign material, it can still be obstructed.

Signs of **obstructed airway** are snoring sounds, crowing sounds or movements of the chest and abdomen with no air moving from the nose and mouth.

If there is airway obstruction, look to improving the head tilt and chin lift maneuver.

If the victim is breathing, put him or leave him on his side and keep the airway clear.

If the victim is not breathing:

Turn onto the back, commence expired air resuscitation (EAR).

The theory and practice of this are covered in a resuscitation course. The steps are:

- clear the airway
- tilt the head back, apply chin lift
- use mouth to mouth or mouth to nose expired air resuscitation
- look for the chest rising with each breath (if it is not, clear and open the airway)
- look, listen and feel for exhalation

If the rescuer has the equipment and skills, help secure the airway using an airway device (eg guedel airway), and ventilate using mouth to mask, a self inflating bag (eg Ambubag) with or without oxygen, or an oxygen inflating device (eg Oxyviva).

Give two full inflations of one second each then:

Check the circulation

C — CIRCULATION

If the victim is unconscious, unresponsive and not breathing it can be assumed that there is no circulation and external cardiac compression (ECC) should be started (Australian Resuscitation Council recommendation).

Note: Previous protocols recommended feeling for a pulse at this stage. This has now been omitted as it is often difficult to feel a pulse in these circumstances, even by medical personnel, and feeling for a pulse and then deciding there is none delays the start of life saving ECC.

Advanced resuscitators such as doctors or paramedics may check for circulation by feeling for a carotid pulse before commencing ECC. But this may be unreliable even in experienced hands and no more than 10 seconds should be used checking for a pulse. If there is no pulse, or any doubt, commence ECC.

When there is no circulation, the sooner ECC is commenced, the better the outcome.

Commence external cardiac compression (ECC) and Cardiopulmonary Resuscitation (CPR)

External cardiac compression. Circulation sufficient to keep the victim alive can be restored with this technique. The chest is compressed by pressure on the sternum, forcing blood into the major arteries and producing some circulation to the vital organs. It takes numerous compressions to establish a blood pressure adequate to provide circulation. If compressions are stopped, the blood pressure immediately falls and the process has to be restarted. Thus the number of times compressions are stopped should be minimized.

The combination of artificial respiration and external cardiac compression is called CPR.

CARDIOPULMONARY RESUSCITATION (CPR)

When the heart stops, so does the breathing (except for occasional gasping respirations in some cases). It is necessary to maintain respiration and cardiac function simultaneously. CPR is EAR and ECC combined.

Give two EAR (expired air resuscitation) breaths of one second per inspiration each, then

Start external cardiac compression (ECC)

Practical details of this are covered in a resuscitation course. Remember, compress:

- vertically
- over the lower half of the sternum (the centre of the chest)
- with the heel of the hand, both hands locked together
- with no pressure on the ribs
- to a depth of 4–5 centimetres (1.5–2 inches or 1/3 the depth of the chest) in adults
- a rate of 100 per minute (almost 2 per second)
- time of compression equal to relaxation – allow complete relaxation

CPR with one rescuer

2 respiratory inflations to 30 cardiac compressions.

CPR with two rescuers

2 respiratory inflations to 30 cardiac compressions

Co-ordinate the inflation with the relaxation phase of compressions. Pause the compressions to allow the inflations.

Continue ventilation and chest compressions at a 30 to 2 ratio until the patient recovers.

Do not stop resuscitation to check for signs of life. If the victim recovers it should be obvious.

If multiple rescuers are present, rotate chest compression duty every 2 minutes as it is very tiring.

If rescuers are unable or unwilling to give expired air resuscitation, chest compressions alone are better than nothing.

If an AED (Automated External Defibrillator) is available attach it and follow the prompts.

Some causes of cardiac arrest (e.g. ventricular fibrillation) can be fixed by giving an electrical shock through the chest. An AED can analyse the cardiac electrical activity and automatically give a shock if appropriate.

The AED should be used as soon as possible after initial CPR.

CPR should be continued until the AED is attached.

If the device detects a shockable rhythm and delivers a shock, CPR should be continued for a further two minutes - the heart takes a few minutes to recover its strength. The machine should then be used to recheck the rhythm.

Duration of CPR

Continue CPR until:

signs of life return
more qualified help arrives
rescuers are exhausted.
an authorized person pronounces the patient dead.

Recovery checks

Frequent recovery checks (stopping cardiac compressions to feel the pulse) are no longer considered appropriate. Interruptions to cardiac compressions result in a poor outcome. Lay people often have difficulty feeling a pulse. Continue CPR until there are signs of life.

Chapter 43

TECHNICAL DIVING

Most recreational SCUBA divers use compressed air in a single cylinder, with a single-hose regulator, to depths up to 30-40 metres, and avoid any decompression staging obligation (although routinely a safety stop is included for dives exceeding 12 metres).

Technical diving is a term used to describe extended diving or where the gas and/or the equipment is different from the original “Aqualung” concept, which only used open circuit, compressed air. The purpose of this technical diving is to extend durations or depths. The simplest modification is the use of a different gas in the scuba cylinder. Then comes the use of several different gases, in multiple cylinders. To reduce the increasing bulk of this diving apparatus, as well as to reduce gas loss, complex rebreathing equipment can be added.

The increasingly complex equipment bestows some advantages, but with increased risks. Some industry representatives and other enthusiastic advocates have promoted this technical diving to the recreational diving community as a safer alternative, enticing others who may not be as adept. It is a more complex and more risky activity than recreational diving and requires expensive equipment and extensive training – two reasons why it appeals to the diving industry.

Most of the diving accidents and deaths which occur in recreational scuba diving, are not due to decompression sickness. Indeed the major causes include the ocean environment (Chapter 6), the stress responses on the individual (Chapter 7), equipment failure or misuse (Chapter 5 & 34) and some diving practices which are especially hazardous – exhaustion of the air supply, buoyancy problems, and failure to follow buddy diving practices (Chapter 34). Technical diving techniques do not reduce and often increase these risks.

The complexity and novelty of technical diving has attracted many established divers, mainly men. Possibly the element of danger and the *avant garde* nature of the activity combine to offer an enticing challenge, extending diving experiences and excitement.

The technical diver, often studious and attracted to risk taking behaviour, operates on a reduced safety margin but usually with a quiet confidence in his skills and equipment.

He may have commercial interests, being involved in wreck salvage, equipment manufacture, marketing and sales, diver training, or other related activities. An appreciable number of high-profile experts in technical diving have died undertaking this activity, and their deaths have hopefully served to caution many younger and less experienced divers.

DEFINITION

1. USING GASES OTHER than COMPRESSED AIR

e.g. OXYGEN

NITROX, (Oxygen Enriched Air),

HELIOX, (Helium & Oxygen)

TRIMIX, (Helium, Nitrogen & Oxygen)

2. DECOMPRESSION DIVING

3. DEEP DIVING (> 40 metres)

4. REBREATHING EQUIPMENT

Technical diving refers to diving in excess of the usual range for recreational scuba divers. This may involve an extension of duration at any depth, the depth itself (in excess of 30–40 metres), changing the gas mixtures to be used, or using different types of diving equipment. All these fall into the realm of technical diving.

It is important, when discussing technical diving, to specify which type, as the relative risk varies from little or no additional risk (compared with recreational diving) to a high one, such as with rebreathing equipment.

Decompression dives and deep diving using only compressed air have added risks and have already been dealt with in previous chapters.

The other risks increase as the gas mixture deviates from normal air and with increased complexity of the equipment.

Diving on 32% oxygen, 68% nitrogen instead of air in a scuba cylinder, to a maximum of 40 metres on a no-decompression conventional air profile, could possibly incur slightly less risk than a recreational scuba air dive.

THE TECHNICAL DIVE

1. DIVE PROTOCOLS, PROFILES and GAS MIXTURES
2. EQUIPMENT COMPLEXITY
3. PHYSIOLOGICAL ASSUMPTION
 - EAD / O₂ / CO₂ / INERT GAS TRANSFER
4. ENVIRONMENTS
5. ACCIDENT & RESCUE IMPLICATIONS

1. DIVE PROTOCOLS, PROFILES and GAS MIXTURES

The diver attempts to select the theoretically ideal gas mixture for the ascent and descent (travel mixes), the bottom (bottom mix) and the decompression staging (usually oxygen or a high oxygen mixture).

The simplest form of technical diving has the diver breathing a mixture of 32% or 40% oxygen (O₂) in nitrogen (N₂). With the increased O₂, there is proportionately less N₂. That means less decompression obligation (and less N₂ narcosis). For the same decompression risk, the dive can therefore be prolonged and this is highly desirable in some dive trips. The additional risk of oxygen toxicity must be appreciated.

Using a single O₂ enriched gas mixture limits the technical diver to shallower dives than with compressed air. A series of gas mixtures in separate cylinders, with diminishing O₂ percentages, allows the technical diver to reach greater depths. Substituting helium (He) for N₂, either in Heliox (He/O₂) or Trimix (N₂/He/O₂) allows the technical diver to descend further, while avoiding or reducing N₂ narcosis. During ascent the changes of gas mixtures is reversed until nearing the surface, when higher O₂ percentages may be breathed to expedite the elimination of inert gas (He or N₂).

When there are various gas mixtures being breathed, the safe profile of the dive may be very complex and errors may be made in the choice of gas breathed. Nevertheless, using open circuit equipment and several gas mixes, dives to over 100 metres have been safely performed.

The use of rebreathing equipment enormously increases the potential hazards (see later), while attempting to control and monitor the gases breathed and the decompression required.

2. EQUIPMENT COMPLEXITY

Technical diving involves more complex equipment for producing, supplying and delivering the various breathing gases, other than air. With an increase in the complexity of the equipment there is an associated increase in the likelihood of human error at all these 3 stages.

The handling of mixtures with higher than normal oxygen percentages implies greater risk of fire and explosions. Gas mixtures may not be as compatible as the "normal" oxygen/nitrogen mix in air, and the heat generated during compression must be appreciated. Although not common, explosions associated with high oxygen percentages are very destructive.

Problems and mistakes develop from the use of multiple gases and complex equipment:

- Mixing, labelling and transport of gas;
- Handling it at the dive site;
- Analysing the gases and confirming that they are the ones appropriate for the dive to be performed;
- Selection of appropriate gases during the dive.
- Different gases require different cylinders together with the various attachments; manifolds, O rings, contents gauges, high pressure hoses, and often, separate regulators.

Because of the added complexity of the equipment, the use of multiple gas mixtures and the increased support facilities, there are substantial initial capital outlays, operating and maintenance costs.

3. PHYSIOLOGICAL ASSUMPTIONS

There is considerable doubt regarding some of the physiological assumptions on which technical diving is based. It is claimed that the equivalent air depth (EAD) calculation can be used to determine the different influence of the gas mixture on the diver, and this has been applied to both nitrogen narcosis and decompression sickness (DCS). There is, in fact, no really good evidence that this EAD is a strictly accurate concept. Experience in highly controlled navy diving has been reassuring, and the implication is that the EAD concept is a valid approximate assumption.

Divers using O₂/N₂ mixtures decompress using tables of EAD. These calculate of the actual partial pressure of N₂ for the dive and from this calculate the depth of an air dive that has the same N₂ pressure. The diver then decompresses as if he had done an air dive to the calculated depth, the EAD.

Thus a diver breathing 40% O₂ at 30 metres (60% of 4 ATA = 2.4 ATA of N₂) has an EAD of 20 metres (80% of 3ATA = 2.4 ATA of N₂). So, after this dive to 30 metres, our diver decompresses as if he had dived to 20 metres on air.

For the same depth/duration dive, the O₂ enriched diver may have less DCS risk and less N₂ narcosis, but he will have more risk of O₂ toxicity, than the air breathing diver. There are the physiological implications of breathing oxygen at varying partial pressures, as well as increased carbon dioxide retention with both increased oxygen diving and deep diving.

Inadequate factual information is available regarding the physiological interactions of multiple gases. The rate of inert gas transfer between the breathing gases in the lungs, the body tissues and any gas spaces (including decompression bubbles) varies both with the gas and the depth. Thus the selection of different gas mixtures is likely to influence the transfer of inert gases in many ways, far more complex than can be deduced from a simplistic formula. Anyone who doubts this should peruse one of the more sophisticated texts on such topics as nitrogen narcosis and the counter diffusion of gases.

Decompression procedures and algorithms are often unproven. Even with the vast data available on air diving to 40 metres, there are many inexplicable decompression accidents. Adding the vagaries of extended depths and durations, multiple gas mixtures and computer modelling, makes for greater uncertainty in technical diving. Technical divers should question the origin and validity of the decompression schedules they are encouraged to use. Some have had to be altered to reduce their incidence of DCS. The lack of controlled trials have caused some to compare the promoters of these decompression protocols to a pharmaceutical company marketing a drug without testing it and then expecting the consumers to determine the correct dosages.

4. ENVIRONMENTS

The main purpose of technical diving is to extend the environments into which diving is performed. This usually results in an increase in the hazards associated with such environments. The exception is a reduction of the N₂ narcosis of deep diving, by the substitution of helium. Most of the other problems with deep diving are aggravated. Not only can the depth or duration of the dive be extended, but so can the actual diving terrain. This is the reason why many wreck divers and cave divers have embraced this activity.

5. ACCIDENT & RESCUE IMPLICATIONS

For the above reasons, the mixed-gas diver often wears a large amount of equipment, complex and bewildering (especially when problems develop during the dive). The likelihood of superimposed equipment problems is thus compounded. Difficulties include those of buoyancy and entrapment. Depth control requires greater discipline and skill as the margin between the "safe" depth and the oxygen toxicity depth, is much reduced. Sometimes a full facemask is indicated so that drowning becomes less likely and rescue more possible.

Because of the different equipment and gases, and the extension of the environments, the procedures for accident management and rescue may have to be altered to take into account the specific problems – such as difficulty in removal of heavy and cumbersome gear. With each variation from the conventional scuba system, there is a price to pay, and a possible modification of the first aid and treatment procedures.

OXYGEN PRESSURE

1. CNS AND RESP. TOXICITY
2. EFFECT ON RECOMPRESSION THERAPY
3. ? NITROGEN NARCOSIS & DCS
4. MIXING & HANDLING DANGER
5. HYPOXIC MIXTURES
6. CO₂ BUILD UP
7. EQUIPMENT CHANGES

There is little concern about oxygen toxicity when diving with compressed air within recreational diving limits. Neurological and respiratory oxygen toxicity are virtually impossible. Also, the amount of oxygen exposure is unlikely to significantly influence any recompression treatments that may be needed for decompression accidents. Neither statement can be applied to technical diving.

It had been assumed that oxygen, by virtue of its replacement of nitrogen, would to some degree reduce the severity of nitrogen narcosis and decompression sickness. Although this is possibly so in theory, the scant experimental evidence that there is available, would suggest that oxygen may actually contribute to nitrogen narcosis. The possibility that O₂ could contribute to DCS has been proposed, but this is unproven.

Inadequate mixing can result in oxygen pressures being higher or lower than intended. This has implications regarding the safety of the dive profile.

Higher oxygen levels are also likely to interfere with carbon dioxide transport in the blood. This has implications as regards carbon dioxide and oxygen toxicity, nitrogen narcosis and possibly decompression sickness.

The handling of gas mixtures, where oxygen is used or is added to air or other gases, can produce some hazards. Oxygen increases the risk of fire and explosion.

Some divers have observed that O₂ aggravates the deterioration of soft materials, such as O-rings and other materials that comprise the diving equipment. It can also accelerate corrosion in cylinders.

See The Technical Dive, above.

OXYGEN ENRICHED AIR or NITROX (EAN_x)

CONFUSION of TERMINOLOGY and JARGON ——— SPECIFY or DIE

40/60 = 40% O₂ (EUROPE) or 60% O₂ (USA)

NOAA 36% + 32% O₂ — max. 1.6 ATA O₂ pressure

NURC, NC — max. 1.45 ATA "

SWEDEN — max. 1.4 ATA "

DAN (R. VANN) — max. 1.2 ATA "

BUT O₂ TOX = ?CNS, ?RESP, ?CO₂. Specify which.

"EAD" = EQUIVALENT N₂ PRESSURE

Much of the technical diving now performed involves the use of nitrogen/oxygen mixtures in which the oxygen concentration is greater than that of compressed air. Under these conditions it is very important to specify exactly how much oxygen is being used. Such phrases as 40–60 or 60–40 are not only confusing but often misleading. In Europe 40–60 could imply 40% oxygen, whereas in the USA it is more likely to imply 40% nitrogen.

The actual percentages used in technical diving do vary with different countries and establishments but NOAA in the USA have chosen 32% oxygen and 36% oxygen as their two major mixes. These should not be referred to as Nitrox 1 or Nitrox 2, as this could also be misleading.

The EAN_x refers to enriched air (nitrox) with the x = oxygen percentage. Thus EAN 32 should mean 32% oxygen and not 32% nitrogen! Do not rely on jargon. Specify the exact mixture, in full.

Any EAN_x diving has a safe depth range less than air, due to oxygen toxicity.

The oxygen pressures that are considered acceptable vary with different authorities, and in many cases there is confusion between the neurological oxygen toxicity (which can result in nausea, vomiting, seizures, etc.) and respiratory oxygen toxicity, which tends to only occur with prolonged exposure. Also, many of the pressures being quoted in the literature refer to the oxygen pressures observed with rebreathing equipment, when the carbon dioxide levels have not been measured – complicating considerably the actual cause of symptoms. Most of the work carried out during World War 2 and soon after, failed to measure the carbon dioxide levels and therefore their conclusions regarding safe oxygen limits, are questionable.

NOAA states that the maximum oxygen pressure acceptable is 1.6 ATA. The National Undersea Research Centre in North Carolina recommends 1.45 ATA. The Swedish authorities have recommended 1.4 ATA and Dr Richard Vann of the Divers Alert Network has suggested 1.2 ATA. The US Navy gives a much greater range, and relates it to the duration of the exposures.

The claimed advantages of EANx diving include a probable reduction in decompression sickness incidence, and a possibility of reduced nitrogen narcosis.

On a theoretical basis, presuming nitrogen pressure as the sole cause of nitrogen narcosis, a 20% oxygen mixture (air) at 23 metres could be replaced with 36% oxygen at a depth of 30 metres. to give an equivalent "narcotic effect". Experimental verification for belief in this theory has been sought, but it was unable to be verified.

A common claim is made that there is less post-dive fatigue with EANx than there is with air. This has not yet been verified.

LOW RISK NITROX DIVING

NITROX (EAN_x) REPLACES AIR. SAME EQUIPMENT
(Same Profile as AIR DIVE). RANGE 15 – 40 metres depth.

ADVANTAGES

1. LESS DCS
2. ? LESS NITROGEN NARCOSIS
3. ? LESS POST DIVE FATIGUE

DISADVANTAGES

1. GAS MIXING PROBLEMS
2. LESS MAX DEPTH (O₂ TOXICITY)
3. ? DETERIORATION OF DIVE EQUIPMENT
4. ? MORE CO₂ RETENTION

It is possible to use EANx to obtain possible advantages, with relatively few disadvantages, under certain conditions.

In this type of technical diving, the nitrox mixture, usually 32% or 36% oxygen, replaces air, but the same equipment is used and the same decompression profiles permitted, within the 15 – 40 metre range. Others use 28–40% oxygen, the latter with appropriate depth reduction.

It has been claimed that there is deterioration in the dive equipment by using high oxygen mixtures but this has not been verified. It is believed that halofluorocarbon O-rings (e.g. Viton) are less

likely to oxidise and have a higher ignition point – and are thus frequently preferred by technical divers.

It is likely, because of the higher oxygen levels inhaled that there will be a concomitant degree of carbon dioxide retention, based on the common and competitive pathways for the transfer and transport of these gases.

HIGHER RISK NITROX DIVING

NITROX (EAN_x) REPLACES AIR — PROFILE AS FOR E.A.D.

ADVANTAGES

RANGE 15 – 40 metres

1. INCREASED DURATION of NO-DECO DIVE

or LESS DECO STOPS

or GREATER DURATION/DEPTH of DIVE for SAME
DECO

2. DECO VALUE — IF AIR STOPS FOLLOWED (LESS N₂)

DISADVANTAGES

1. GAS MIXING, HANDLING & CORRECT USAGE

2. MAX DEPTH LIMITED (O₂ TOXICITY)

3. ?ALTERATION OF DCS & RECOMPRESSION

THERAPY

4. ? DYSBARIC OSTEONECROSIS (SLOW TISSUES
AFFECTED BY LONGER DIVES)

In this type of diving (EAN_x) the profile of the dive is altered to make allowance for the high oxygen, lower nitrogen levels, based on the EAD or similar calculations. Thus the diver is likely to increase the duration of his no-decompression dive, reduce the decompression stops required or increase the duration or depth of the dive for the same decompression time commitment. Whether this calculation is justifiable under all conditions, has yet to be demonstrated.

The probable only genuine safety advantage of this kind of diving occurs if "air stop" times are followed during decompression, whilst using EAN_x.

There is a possibility of an increased risk of decompression sickness, due to the effects of oxygen contributing to this disorder, or because of the use of untested algorithms used in commercial nitrox decompression profiles. The "bent" diver is also more likely to have had a higher oxygen dose, contributing to respiratory damage during the recompression therapy, than his air breathing colleague.

There may well be an alteration in the type of decompression sickness sustained with this form of diving because of the increased duration that it frequently entails. Thus the slower tissues are more

likely to be affected, and this should be considered during the subsequent recompression therapies, and of a possible increased susceptibility to dysbaric osteonecrosis.

HIGH RISK, HELIUM DIVING

LESS DENSE, LESS SOLUBLE, FASTER DIFFUSION, HEAT CONDUCTIVITY

ADVANTAGES

1. LESS NARCOSIS — GREATER DEPTH
2. LESS BREATHING RESISTANCE — GREATER DEPTH
3. REDUCED CO₂ RETENTION
4. LESS DECO (for LONGER DIVES)

DISADVANTAGES

1. DEEPER DIVING
2. MORE DECO (for SHORT DIVES)
3. HEAT LOSS (ENVIRONMENT, ? RESPIRATORY)
4. VOICE DISTORTION 5. MIXING 6. HPNS

There are significant differences in the way the body handles helium and nitrogen. Both are inert gases, but helium is much less dense and is also less soluble in some tissues than nitrogen. It does, however, have a much greater speed of diffusion and also conducts heat more rapidly.

The real advantage compared to nitrogen is that it does decrease the incidence of nitrogen narcosis. For dives in excess of 30 – 40 metres, the risks of nitrogen narcosis can be proportionately decreased as helium replaces nitrogen. It thus tends to be used for dives of greater depths. An additional factor is the reduction in breathing resistance due to its decreased density and other factors, also allowing dives to greater depths.

The effects on decompression likelihood are more complicated. It is probably likely to produce less decompression requirement for the longer dives, but may well require more decompression for shorter dives. Many of the helium and Trimix decompression tables are less well validated than the air tables, and herein lies a major difficulty with helium diving.

The main aggravating problem is that the divers are diving deeper with helium and Trimix than with compressed air, and therefore are exposed to all the associated problems of depth (other than nitrogen narcosis and breathing resistance). Barotrauma and DCS risks are aggravated. The environmental difficulties associated with depth include poor visibility, buoyancy implications, excess gas consumption, stress factors and the increased problems with first aid, rescue and resuscitation.

There is also a greater conductive heat loss from helium, even though there is some question regarding the respiratory heat loss. Heliox feels colder to breathe, and in a helium environment the heat is lost more rapidly. Increased depth also aggravates heat loss.

Voice distortion can produce communication problems. At greater depths the high pressure neurological syndrome (HPNS) also becomes relevant.

The difficulties with mixing gases, referred to above, are also present with helium and are complicated by the different compressibility of helium, as well as the risk of ascending with low oxygen pressures – which are commonly used with deep helium diving.

Comparison with the commercial deep divers is noteworthy. These experts usually require a surface supply of gas, full facemasks, communication systems, a standby diver, a wet bell and a recompression chamber on site. Experience has demonstrated the need for these. The less trained amateurs appear to have no such requirements.

VERY HIGH RISK. RE-BREATHERS or CIRCUIT SETS

Rebreathing equipment has been in use for more than a century, causing many deaths and cases of unconsciousness. Despite the recent electronic mechanisms, the essential problems of rebreathing equipment remain. It is very much a high risk strategy to employ for specific reasons, by professionals.

The value of rebreathing equipment is that it produces fewer bubbles, and is therefore quieter. This is of use both in clandestine military operations and for marine photography. It is more economical on gas, as the gas is recycled through the diving equipment, in a "circuit". It can also be constructed with low magnetic materials, which are useful if one is working around magnetic mines.

The disadvantage that is inherent in all types of rebreathers is the failure of the carbon dioxide absorbent system to work effectively under all diving conditions. This may occur for various reasons. One is an inappropriate canister design. The early absorbent canisters were inadequate for maximal exertion. It is surprising how few improvements the manufacturers have included in some of the carbon dioxide absorbent canisters in the sets now being promoted. Also, the absorbent itself is not always reliable. It frequently varies in efficiency, and each absorbent batch needs to be tested. This is not feasible for the individual diver. The handling and storing of absorbent may result in deterioration in efficiency, as will the degree and type of wetting that may occur.

When diving in sea water, hypertonic saline can enter the system, causing a great reduction in efficiency. The absorbent itself, when combined with carbon dioxide, produces water as a by-product, which can also influence the efficiency. Water traps are incorporated in some sets.

REBREATHERS

ADVANTAGES

SILENT, ECONOMICAL, +/- MAGNETIC

DISADVANTAGES

1. CO₂ TOXICITY
2. DILUTION HYPOXIA, HYPEROXIA
3. CAUSTIC COCKTAIL
4. INITIAL and MAINTENANCE EXPENSE

OXYGEN RE-BREATHERS :

DEPTH LIMIT 8 – 9metres

CONSTANT FLOW. O₂ = FLOW vs. ENERGY

OXYGEN MONITORS = FAILURE. DCS?

Oxygen Rebreather

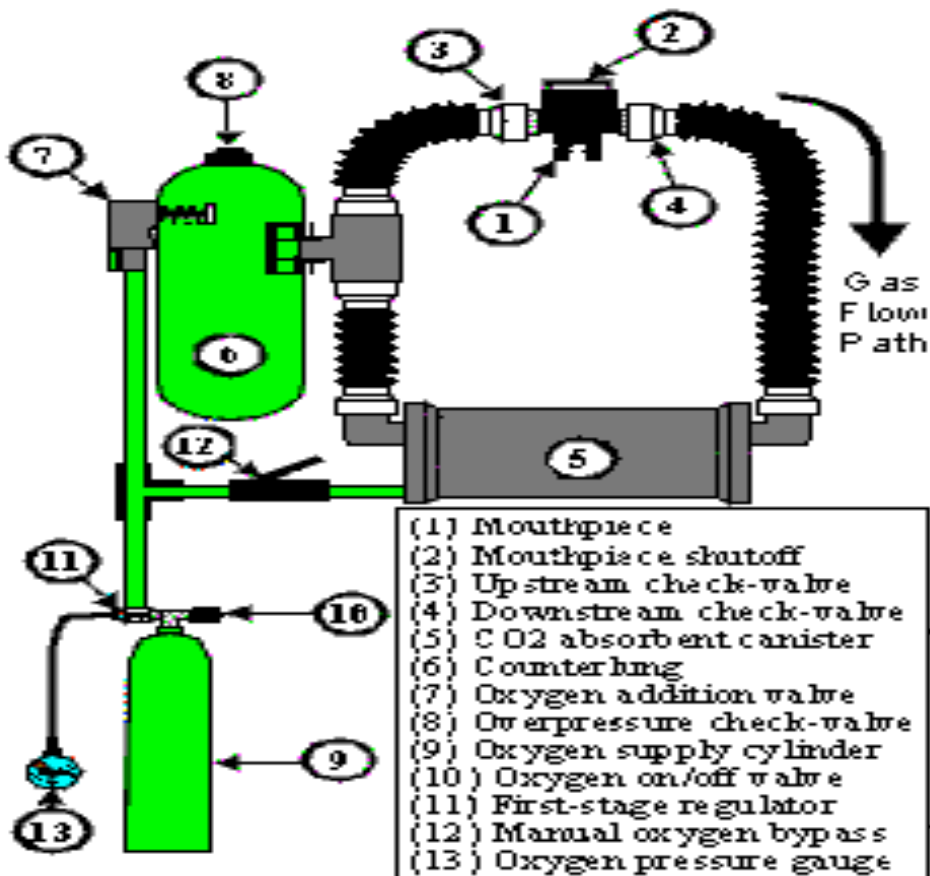


Fig. 43.1

The carbon dioxide absorbent must be packed correctly into the canister. This is an acquired skill and requires training. The density of packing influences the efficiency. Lower temperatures also reduce the efficiency of the absorbent. Some absorbents can be pre-packaged.

Often absorbent canisters will work very well at a moderate work load, but when exertion is required, the absorbent canister will eventually fail – especially if it has been in use for a considerable time.

The manufacturers' claims regarding the safe duration of carbon dioxide absorption in their diving equipment are usually very optimistic, and do not apply to emergency situations where the diver is exerting himself maximally (such as when swimming against a current, or trying to rescue and tow a companion – even on the surface).

When water gets into the rebreathing set, it may collect some of the alkali from the absorbent and enter the divers mouth and lungs, which can be very unpleasant. This is called a "caustic cocktail".

A rebreathing set can cause dilution hypoxia, usually by incorrect technique and failure to "clear the set" (and the lungs) of the inert gas. This is more likely when the supply gas is on demand, compared to the old fashioned constant flow sets. It can also occur if there is a small amount of inert gas in the gas cylinder, and especially so when there is a considerable amount of nitrogen or helium, such as with nitrox, heliox or trimix diving. It may be induced by an incorrect mix, a leak from or obstruction to the inflow, or low cylinder pressure. It can even occur in 100% O₂ sets, especially those that supply gas on demand.

Sometimes the hypoxia will only be noticed during ascent. A lower oxygen percentage at depth may translate to a dangerously low oxygen partial pressure nearer the surface.

Re-breathers require specialised diving protocols, when rescue and resuscitation are needed. It is not just a matter of removing a mouthpiece and replacing it with another. Companion diver drill needs to be tailored for each type of re-breather.

The problems of gas mixing and handling, as described above, also relate to this equipment.

Semi-closed Rebreather

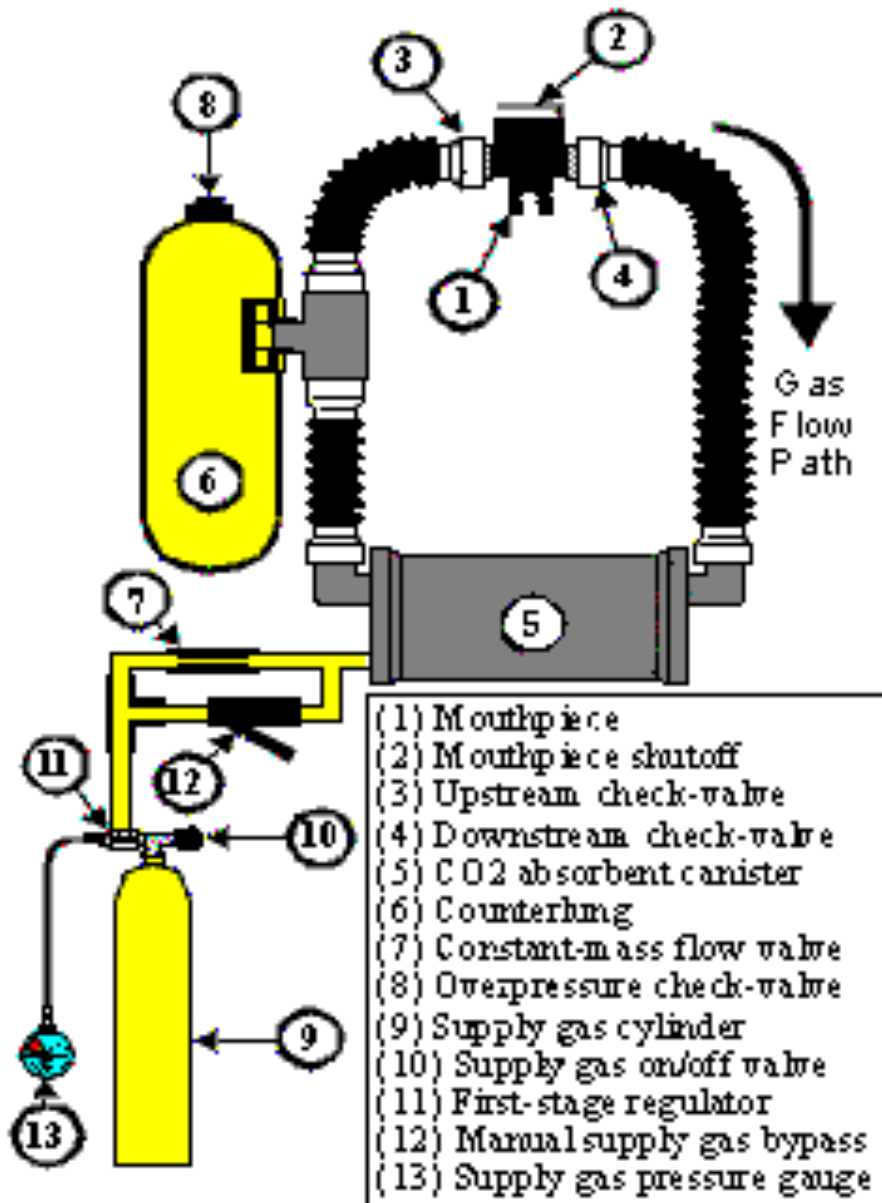


Fig 43.2

- The oxygen rebreathers are closed circuit sets, used to a maximum depth of about 8-9m, are usually restricted to specialised navy divers. They have resulted in many cases of unconsciousness and death.

Occasionally photographers use this equipment, but they would be considered unwise to do so. The companion rescue drill is often required and marine photographers are not gregarious beasts.

- Some rebreather sets have a constant flow of nitrox, heliox or trimix gas. They are usually semi-closed circuit sets. With these, the oxygen level in the breathing bag or inspiratory tube may vary according to two major factors. The first is the flow of oxygen into the set, and the second is the amount lost from the set. The inspiratory oxygen range can be a variable quantity, and should be designated pre-dive. The relevant factors determining inspiratory oxygen include:
 - the volume and mixture of the incoming gas
 - the energy utilised in metabolism (oxygen consumption)
 - the volume and mixture of gas released as bubbles (e.g. with ascents).

The interaction between the input and output of oxygen will result in a variable oxygen percentage and ascent or descent will then determine the oxygen pressure. These sets are especially likely to cause dilution hypoxia and hypoxia of ascent.

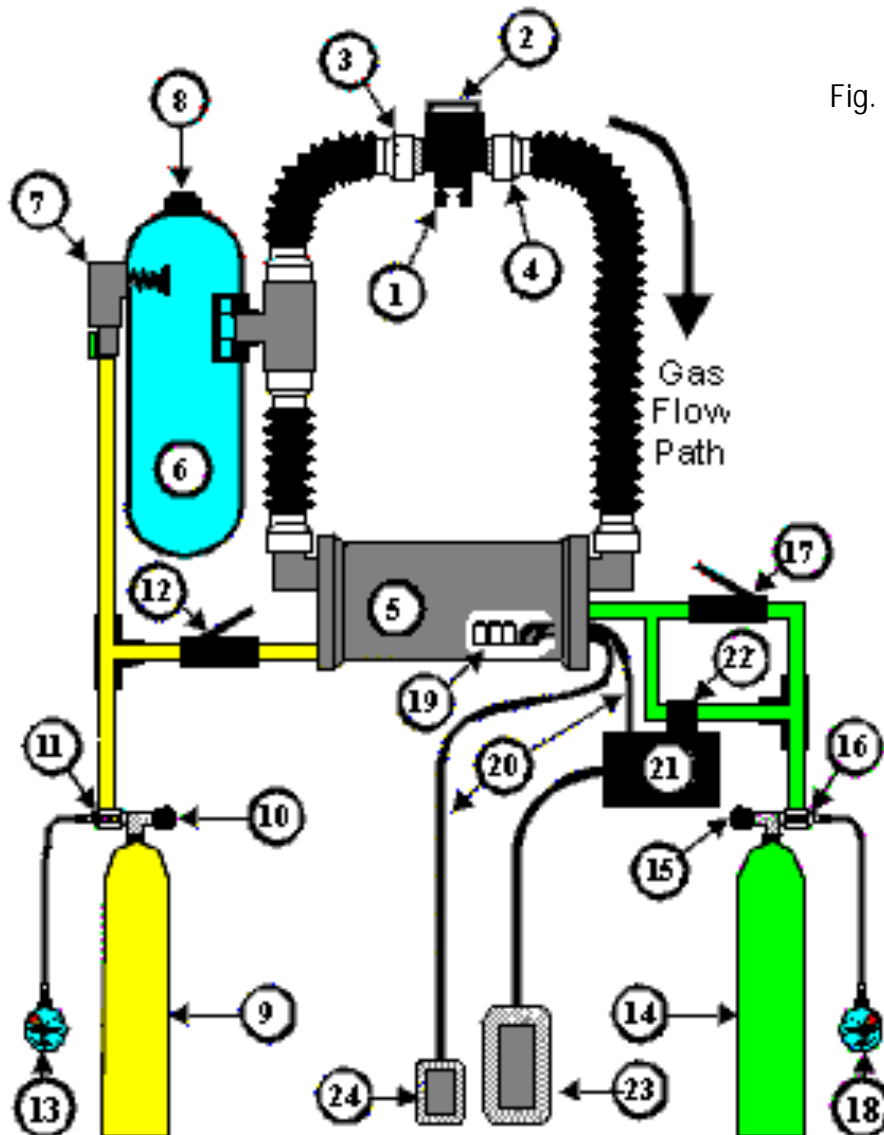
As hypoxia usually produces no warning prior to it causing unconsciousness, the use of constant flow rebreathing sets would be considered unwise. Close attention to the cylinder pressure, ensuring an adequate inflow of gas, and a replacement with fresh gas prior to ascent (a "flush-thru") is essential

- The more expensive closed circuit mixed gas rebreathing sets use analysers and solenoids to measure and control the oxygen pressures during the dive and a feed back system adds oxygen or a diluent gas (nitrogen, helium, mixtures) as required – to ensure that the oxygen partial pressure remains within a certain range. This equipment is extremely expensive, often not reliable and should only be used by those with faith in this technology.

N.B. Anyone who uses a rebreather should be aware of the much greater risk of unconsciousness. Without a full facemask, this problem usually converts to subsequent drowning and death.

Closed-Circuit Rebreather

Fig. 34.3



- | | |
|--------------------------------|--------------------------------|
| (1) Mouthpiece | (13) Dihydrogen pressure gauge |
| (2) Mouthpiece shutoff | (14) Oxygen supply cylinder |
| (3) Upstream check-valve | (15) Oxygen on/off valve |
| (4) Downstream check-valve | (16) Oxygen regulator |
| (5) CO2 absorbent canister | (17) Manual oxygen bypass |
| (6) Counterlung | (18) Oxygen pressure gauge |
| (7) Dihydrogen addition valve | (19) Oxygen sensor |
| (8) Overpressure check-valve | (20) Oxygen sensor cables |
| (9) Dihydrogen supply cylinder | (21) Main electronics |
| (10) Dihydrogen on/off valve | (22) Oxygen solenoid valve |
| (11) Dihydrogen regulator | (23) Primary display |
| (12) Manual dihydrogen bypass | (24) Secondary display |

CONCLUSION

There are few problems in understanding the general concepts of technical diving, using different gas mixtures, different equipment and extending limits beyond those of recreational diving. The principles are relatively simple. The devil is in the application.

Perhaps the most important thing about technical diving is to realise that the majority of the diving deaths that occur in recreational divers occur for reasons which will be aggravated by the use of more complex equipment, in more hazardous environments. Technical diving is therefore, by its very nature, likely to have greater risks than normal recreational diving, other factors being constant.

The margin for error in this type of diving is appreciably less, and therefore it should only be employed by divers with enormous experience, detailed training and meticulous attention to equipment selection, maintenance and use. The advocates of technical diving tend to lay great stress on certain aspects of safety - which are relatively unimportant compared to the others referred to in Chapter 34.

To overcome some potential equipment related problems, technical divers may stress the need for redundancy, redundancy, redundancy. Complexity is an unintended accompaniment.

They will stress the importance of decompression sickness, and the physiological advantages of oxygen, but may ignore the more frequent causes of diving deaths, such as exhaustion of gas supply, buoyancy problems, stress responses, etc. They will also tend to ignore the areas in which the "technical advances" have been meagre e.g. the efficiency of carbon dioxide absorbents, to focus in preference on high-tech oxygen sensors and theoretical decompression algorithms.

The leaders in this field will be experienced, highly skilled, very fit, entrepreneurial divers often with a high public profile. It is not for occasional, pleasure seeking divers who anticipate a relaxed, hassle free, unencumbered dive and an automatic expectation of survival at the end of the day.

Appendix A

DIVING MEDICAL LIBRARY

DIVING TEXTS

- **Physics, Physiology and Decompression Theory** for the Technical and Commercial Diver – Bruce Wienke
- **Deeper Into Diving** – John Lippman and Simon Mitchell
- **High Altitude Diving** - Bruce Wienke
- **Scuba Equipment Care and Maintenance** - Farley and Royer
- **Oxygen and the Scuba Diver** - Hendrick and Thomson
- **Oxygen First Aid** – John Lippmann
- **The Application of Enriched Air Mixtures** - Betts
- **Sport Diving in Depth** - Griffiths
- **The Physics and Engineering of Diving** - Dickens
- **The Underwater Handbook**, a Guide to Physiology and Performance for the Engineer (1976) — by C. W. Shilling, M. F. Werts & N. R. Schandolmeier.
- **Mastering Rebreathers** - Jeff Bozanic
- **Nitrox Manual** - Dick Rutkowski
- **Recreational Nitrox Diving** - Robert N. Rossier
- **Technical Diving In Depth** - Bruce R. Wienke
- **Snorkeling and Free Diving** - Tom Scott, Jim Flagg
- **Cold Water Diving** - John N. Heine
- **Scuba Diving** - Dennis K. Graver

DIVING MANUALS

- U S Navy Diving Manual, Revision 6, (Vol 1 Air Diving) - Naval Sea Systems Command, Supervisor Of Diving. Free download on internet
- British Subaqua Club (BSAC) Diving Manual <http://www.bsac.com/>
- National Oceanic and Atmospheric Administration (NOAA) Diving Manual
- Professional Association of Diving Instructors (PADI) – Open Water Diving Manual.
- National Association of Underwater Instructors (NAUI) – Diving Manual.

NEWSLETTERS

• **Undercurrent** – this doubles both as a consumer guide to holiday dive sites, as well as a forum for the presentation of recent technical data, diving accidents, etc. :
Address : PO BOX 1658 Sausalito, California 94965, U.S.A.

• **Alert Diver** – a quarterly newsletter of the Divers Alert Network. An excellent informative newsletter regarding diving medical safety and accident information, produced by DAN :
Address : BOX 3823 Duke University Medical Centre, Durham, North Carolina 27710, U.S.A.

• **Pressure** – a bi-monthly newsletter of the Undersea and Hyperbaric Medical Society, with information of a social, academic and educational nature, relevant to the Society and its members – mainly physicians and paramedics:
Address : Undersea and Hyperbaric Medical Society 9650 Rockville Pike, Bethesda Maryland 20814, U.S.A.

• **Diving and Hyperbaric Medicine Journal** – a bi-monthly journal/newsletter of the South Pacific Underwater Medical Society and the European Undersea Biomedical Society. This contains both original diving medical articles and reviews, and also summaries of other diving medical research conducted throughout the world:
Address: **SPUMS, c/- ANZCA, 630 St Kilda Rd, Melbourne Vic, 3004, Australia**
email spumsadm@bigpond.net.au

- **Undercurrent.** A USA newsletter with excellent independent information on equipment and dive travel sites.
<http://www.undercurrent.org/>
- **X-Ray Mag. A free internet magazine.** More into ecology, marine animals, photography, etc. URL: <http://www.xray-mag.com/>
- **Advanced Diver.** More into high tech diving and equipment.
<http://www.advanceddivermagazine.com/>

NB. Many other countries have their own variations of the above newsletters, from local institutions.

DIVING MEDICAL TEXTS

- ***Diving Medicine – for Scuba Divers** — (2010) by Carl Edmonds, Bart McKenzie, John Pennefather and BobThomas .
- ***The DAN Emergency Handbook** — by John Lippmann & Stan Bugg.
- ***Dangerous Marine Creatures *** — by Carl Edmonds,
- *** Oxygen First-Aid for Divers** — by John Lippman, J.L. Publications,
- **Diving And Subaquatic Medicine (4th Edition)** Carl Edmonds, Christopher Lowry, John Pennefather, And Robyn Walker. Arnolds Publ.
- **Bove and Davis’ Diving Medicine**— A.A. Bove by, published by Saunders, Philadelphia.
- **Bennett and Elliott’s Physiology & Medicine Of Diving-5th Edition** - Alf Brubakk, Tom Neuman
- **The Physicians Guide to Diving Medicine** (1984) — by Shilling, Carlston and Mathias. Published by Plenum Press, New York.
- **Diving Accident Management** (1990) — the 41st Undersea and Hyperbaric Medical Workshop, edited by Bennett and Moon, and published by the Undersea and Hyperbaric Medical Society,
- **Assessment Of Diving Medical Fitness For Scuba Divers And Instructors** - Peter B. Bennett, Frans J. Cronje, Ernest S. Campbell
- **Encyclopedia Of Underwater Investigations** - Robert G. Teather

* = Suitable for recreational divers

NOTE Most of the above can be acquired over the internet, and most texts can be obtained from:

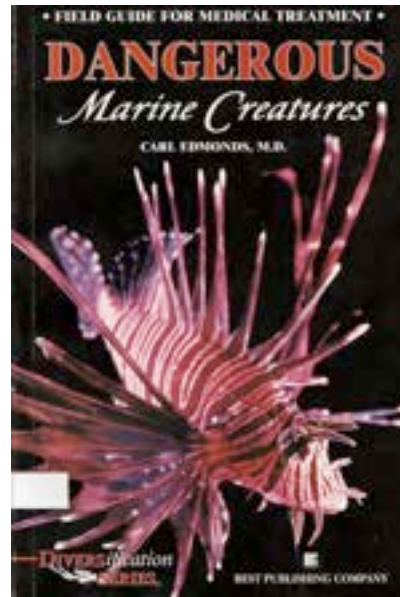
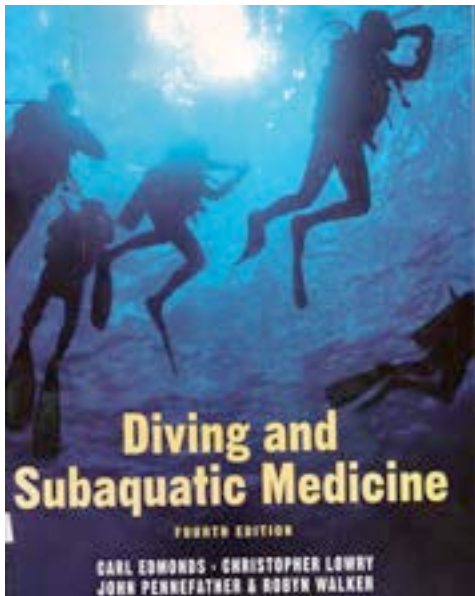
Amazon.com

Best Publishing

Divers Alert Network.

Some can be accessed from the internet, free. (such as the US Navy Diving Manual) and

Diving Medicine – for Scuba Divers — (2010) by Carl Edmonds, Bart McKenzie, John Pennefather and BobThomas . www.divingmedicine.info



DIVING MEDICAL REFERENCES

Apart from the Diving Medical Societies (see Appendix B) and in the medical texts referred to above, the best source of diving medical references is:

<http://rubicon-foundation.org>

In the past there have been excellent sources of references and reports. They include

A Bibliographical Sourcebook of Compressed Air, Diving and Submarine Medicine. Vols 1, 2 and 3. U.S. Govt publication.

Key Documents of the Biomedical Aspects of Deep-Sea Diving, Vols 1-5. Undersea Medical Society publication.

Underwater Medicine and Related Sciences. A Guide to the Literature. Vols 1-6. Undersea Medical Society publication.

It is possible to conduct research without access to these publications, but I do not see how. CE

Appendix B

INFORMATION CONTACTS

SOCIETIES

Apart from the instructor organisations (NAUI, PADI, SSI, YMCA, BSAC, CMAS, IANTD, etc.) there are certain societies which would be very useful for any diving paramedic to belong to.

These include the following :

- **Divers Alert Network (DAN)** — BOX 3823 Duke University Medical Centre, Durham, NC 27710 (USA and the American continent, offshore and USA islands and trust territories). www.diversalertnetwork.org
- **Divers Alert Network, Asia Pacific(DAN.AP). P.O. Box 384, Ashburton, Vic, Australia.** www.danasiapacific.org Email: info@danasiapacific.org
- **Undersea and Hyperbaric Medical Society** —Maily American. www.uhms.org
- **South Pacific Underwater Medicine Society (SPUMS)** — C/- The Australian and New Zealand College of Anaesthetists. Mainly the Indo-Pacific region, centred in Australia). www.spums.org.au
 - **European Underwater and Biomedical Society.** www.eubs.org
- **British Subaqua Club (BSAC)** — (mainly around the U.K. and Europe). www.bsac.org
- **CMAS Confédération Mondiale des Activités Subaquatiques.** Mainly European. <http://www.cmas.org>
- **IANTD**An organisation that publishes information for technical divers. <http://www.iantd.com/iantd2.html>

See **Appendix D** for all **DAN** regional offices and emergency contacts

**EMERGENCY TELEPHONE
NUMBERS**

Your Area : _____

Telephone : _____

Telephone : _____

Telephone : _____

Others : _____

Telephone : _____

Others : _____

Telephone : _____

Others : _____

Telephone : _____

Others : _____

Telephone : _____

Others : _____

Telephone :

Appendix C

IN-WATER O₂ RECOMPRESSION THERAPY

AUSTRALIAN UNDERWATER OXYGEN TABLE

Notes :

- 1. It is recommended that the application of this therapy technique usually be limited to trained and experienced diving medical practitioners and paramedics.**
- 2. This technique may be useful in treating cases of decompression sickness in localities remote from recompression facilities. It may also be of use while suitable transport to such a centre is being arranged.**
- 3. In planning, it should be realised that the therapy may take up to 3 hours. The risks of cold, immersion and other environmental factors should be balanced against the beneficial effects.**
- 4. The diver must be accompanied by an attendant at all times.**

❑ Equipment.

The following equipment is essential before attempting this form of treatment :

1. Full face mask with demand valve and surface supply *or* helmet with free flow.
2. Adequate supply of 100% oxygen for patient, and air for attendant.
3. Wet suit for thermal protection.
4. Shot with at least 10 metres of rope (a seat or harness may be rigged to the shot).
5. Some form of communication system between patient, attendant and surface.

❑ Method.

1. The patient is lowered on the shot rope to 9 metres, breathing 100% oxygen.
2. Ascent is commenced after 30 minutes in mild cases, or 60 minutes in severe cases, if improvement has occurred. These times may be extended to 60 minutes and 90 minutes respectively if there is no improvement.
3. Ascent is at the rate of 1 metre every 12 minutes.
4. If symptoms recur, remain at depth a further 30 minutes before continuing ascent.
5. If oxygen supply is exhausted, return to the surface, rather than breathe air underwater.
6. After surfacing, the patient should breathe 1 hour on 100% oxygen, one hour off, for a further 12 hours.

Fig. 1 App C. Breathing intermittent O2 after treatment



Fig. 2 App C. An Underwater O2 unit



Fig. 3 App C. A cheap full face mask. *Cressi-Sub*

Appendix D

DIVERS ALERT NETWORK

(DAN)

The Divers Alert Network (DAN) is a group of not-for-profit organisations dedicated to improving diving safety for all divers. Regional DAN organisations have been established throughout the world to provide safety services to divers. These include DAN America, DAN Europe, DAN Asia-Pacific (AP), DAN Europe, DAN Southern Africa and DAN Japan.

As part of its mission, DAN provides or funds 24-hour diving emergency hotlines throughout the world; offers emergency evacuation cover and dive injury insurance to members; provides diving medical information services; offers quality training in accident management including first aid and oxygen provision; collects data and reports on dive accidents; funds research into dive medicine and dive safety; and provides support for recompression chambers in remote locations.

DAN Asia-Pacific

P.O. Box 384 (49A Karnak Rd)
Ashburton, Victoria 3147
AUSTRALIA

Tel +61-3-9886 9166

Fax +61-3-9886 9155

info@danasiapacific.org

www.danasiapacific.org

Diving emergencies:

DES Australia

within Australia 1800-088200

outside Australia +61-8-8212 9242

DES New Zealand 0800-4DES 111

DAN AP Korean Hotline

010-4500 9113

DAN AP Chinese Hotline

+852-3611 7326

DAN America

6 West Colony Place Durham, N.C. 27705 USA

Tel +1-919-684-2948

Fax+1-919-490-6630

dan@DiversAlertNetwork.org

www.diversalertnetwork.org

Diving emergencies:

DAN America

+1-919-684 8111

DAN America-Mexico

+52-5-629 9800 code 9912935

DAN Europe

P.O. Box DAN

Roseto 64026 ITALY

Tel +39-085-893-0333

Fax +39-085-893-0050

mail@daneurope.org

www.daneurope.org

Diving emergencies:

+39-06-4211 8685

DAN Japan

Japan Marine Recreation Association Kowa-Ota-Machi Bldg, 2F,
47 Ota-Machi 4-Chome, Nakaku, Yokohama City, Kagawa 231-0011 JAP

Tel +81-45-228-3066

Fax+81-45-228-3063

E-mail dan@danjapan.gr.jp

www.danjapan.gr.jp

Diving emergencies:

+81-3-3812 4999

DAN Southern Africa

Private Bag X 197 Halfway House 1685 SOUTH AFRICA

Tel +27-11-254 1991

Fax: +27-11-254 1993

E-mail mail@dansa.org

www.dansa.org

Diving emergencies:

inside South Africa:

0800-020111

outside South Africa

+27-10-209 8112