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.

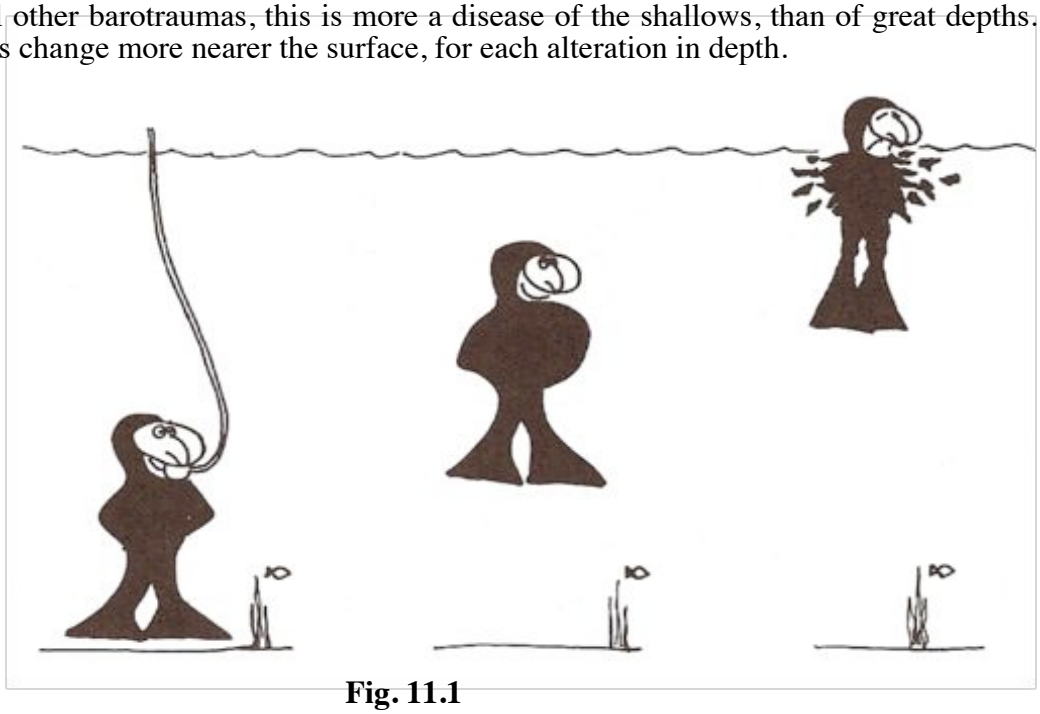


Fig. 11.1

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)**

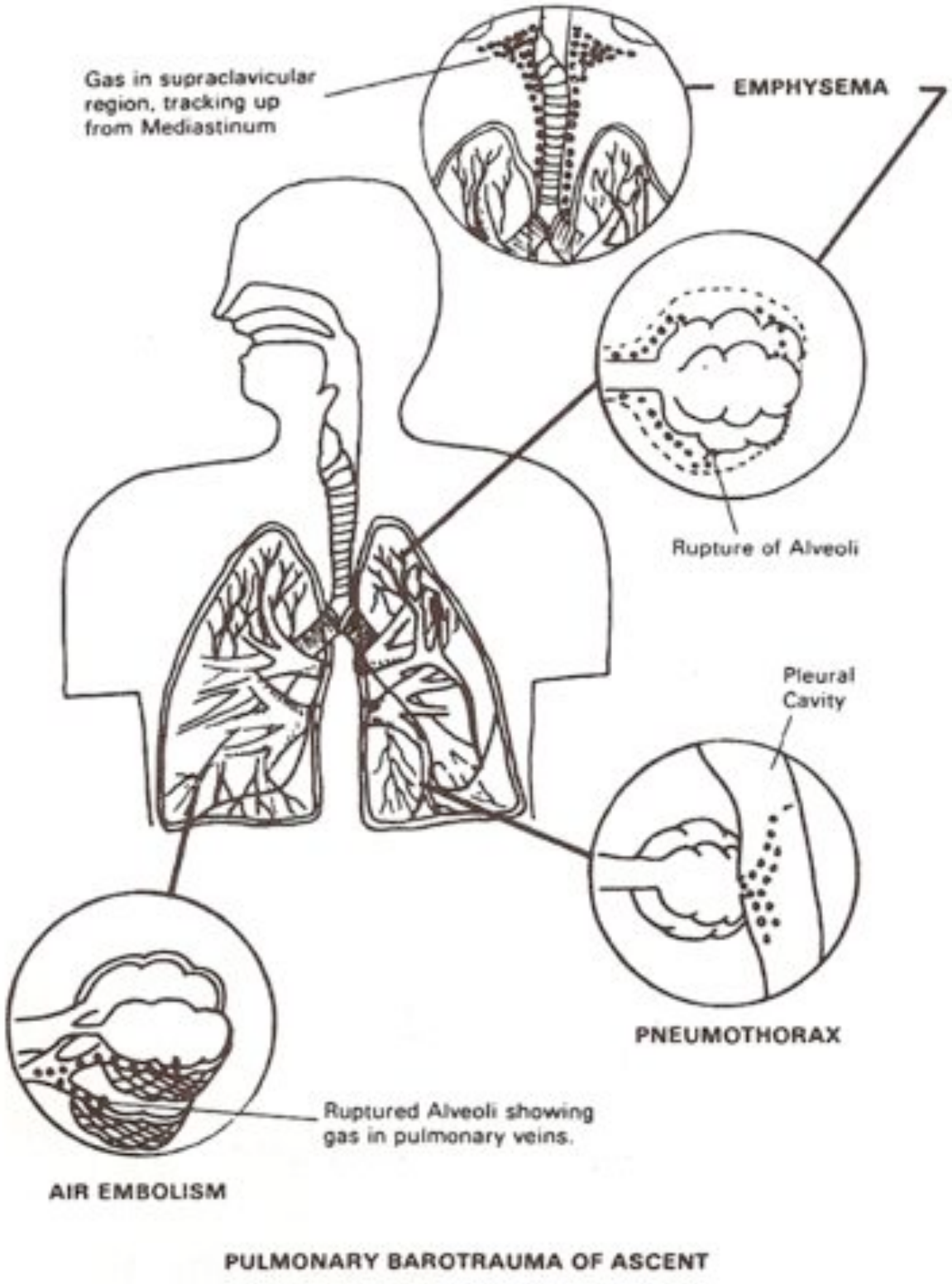


Fig. 11.2

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.**

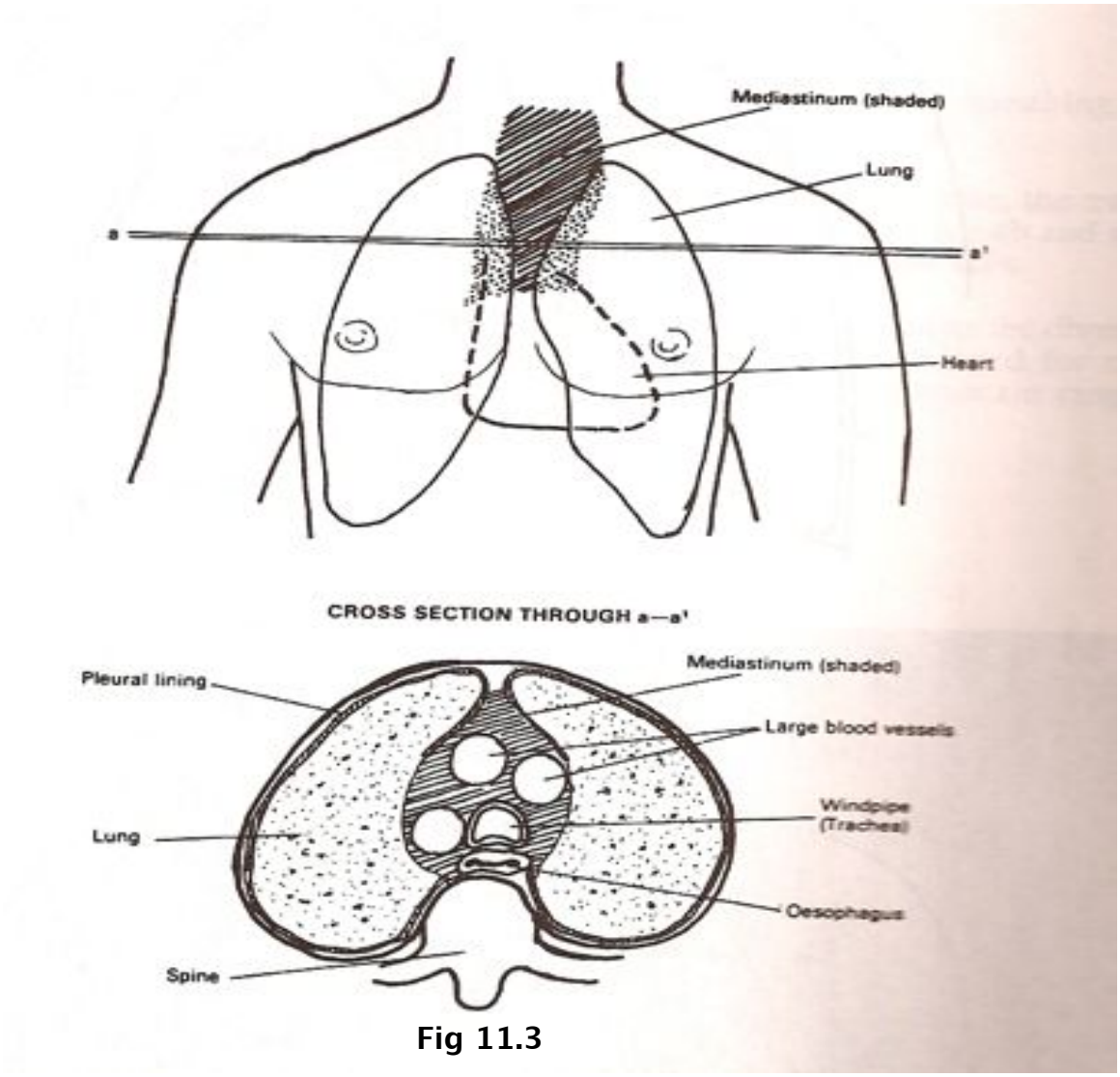


Fig 11.3

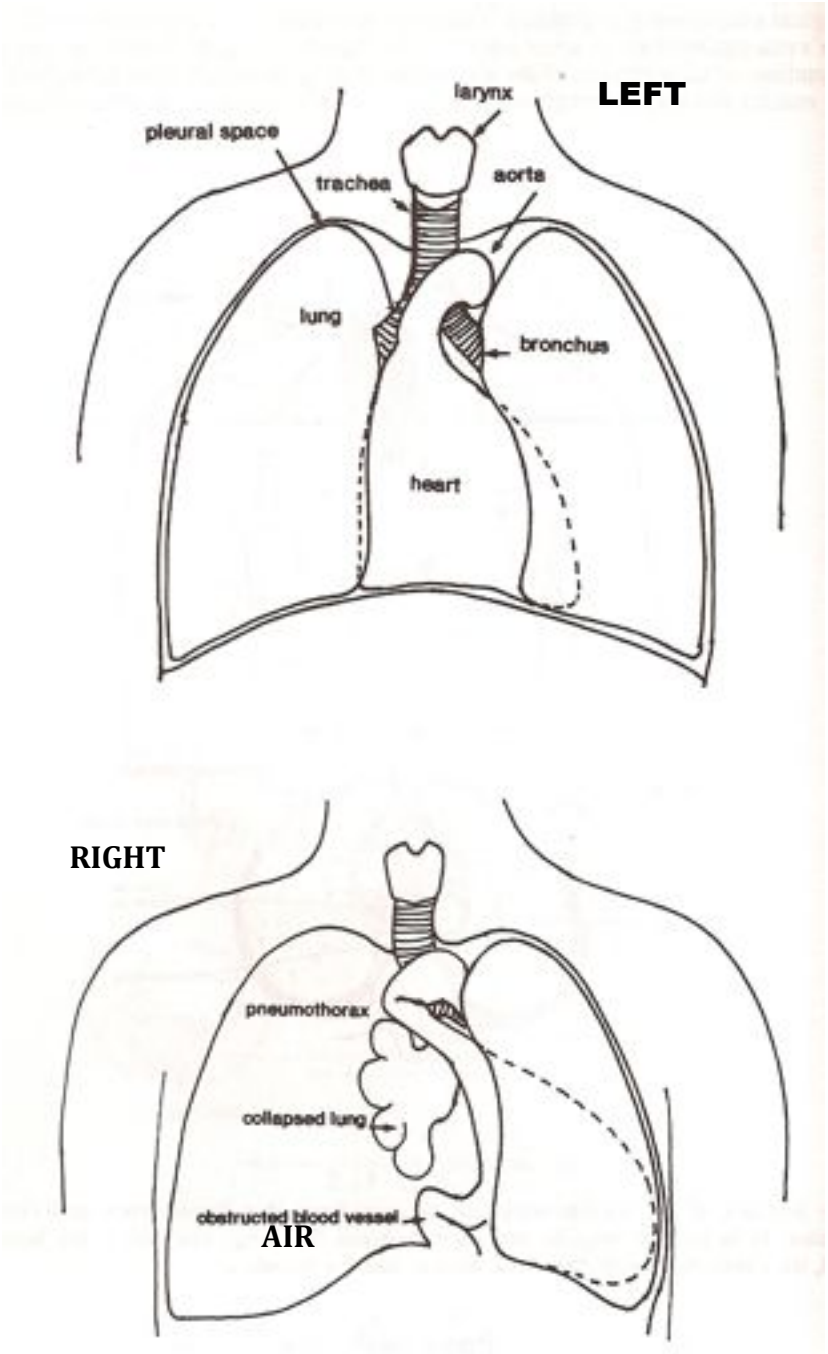
The location of the mediastinum can be seen from this frontal view and cross section. It is located deep in the chest between the lungs and above the heart, and its connection with the neck tissues can be noted.

❑ **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).

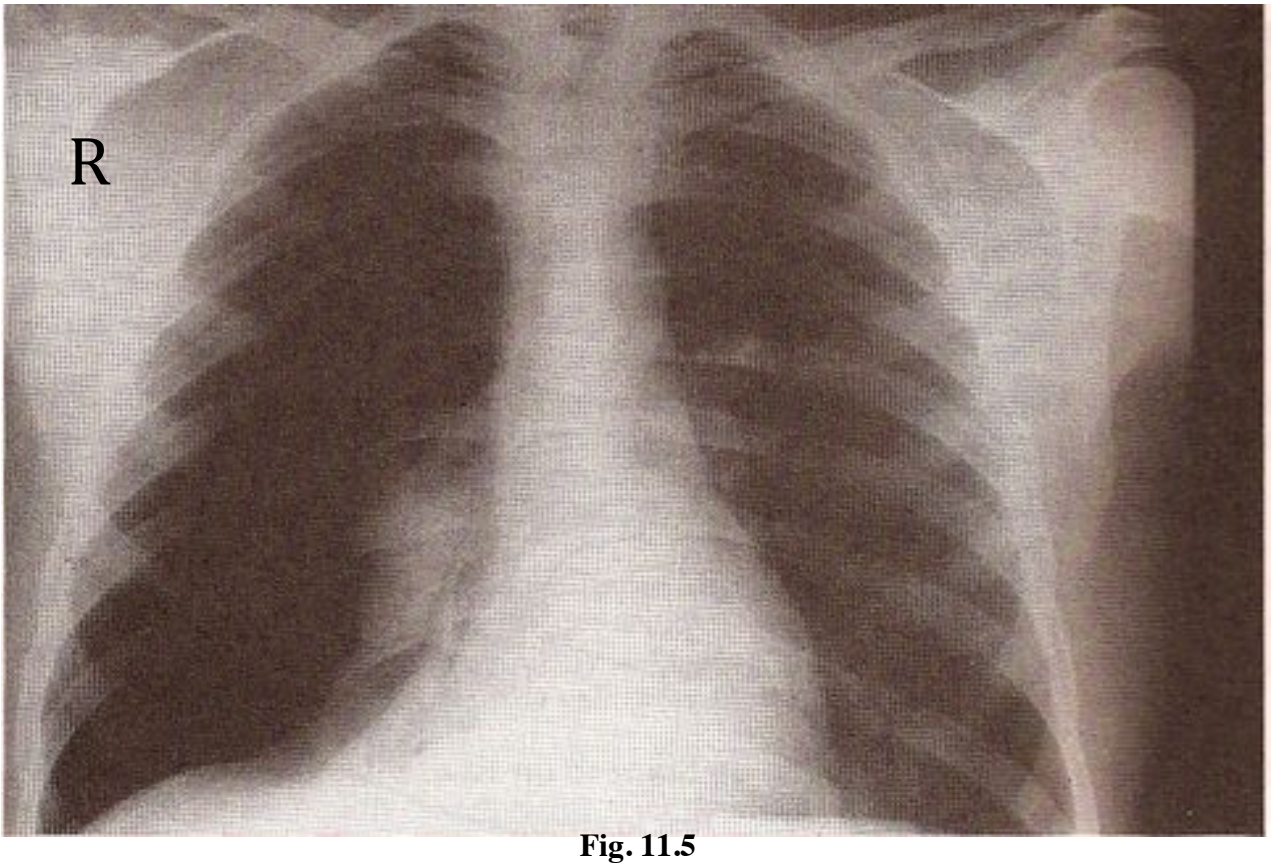


Fig. 11.5

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.

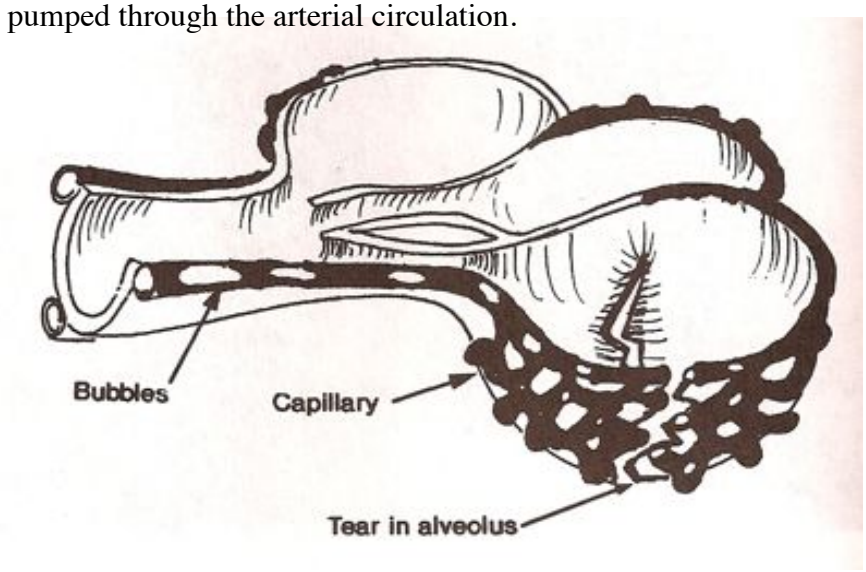


Fig. 11.6

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 to 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 basic life support (**BSL**) priciples take precedence and should 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

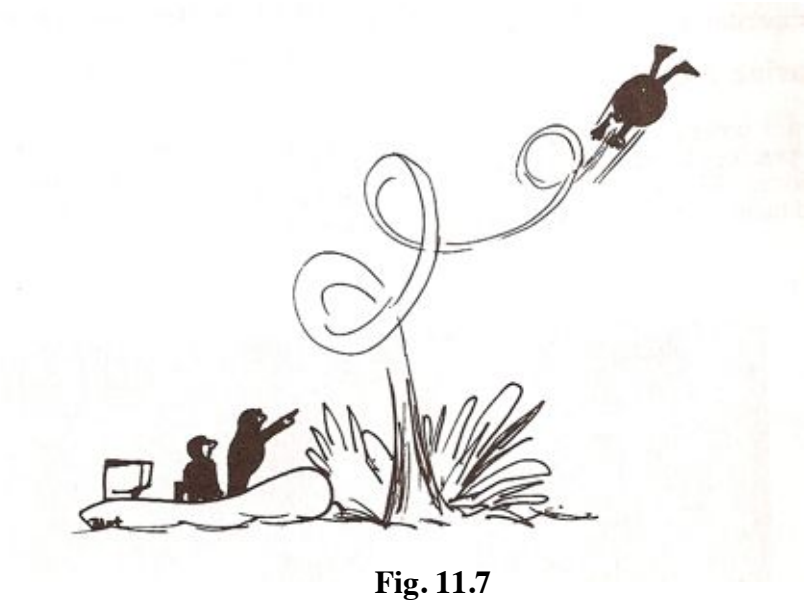


Fig. 11.7

❑ **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 <u>OOA diver's</u> 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 consistant 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.

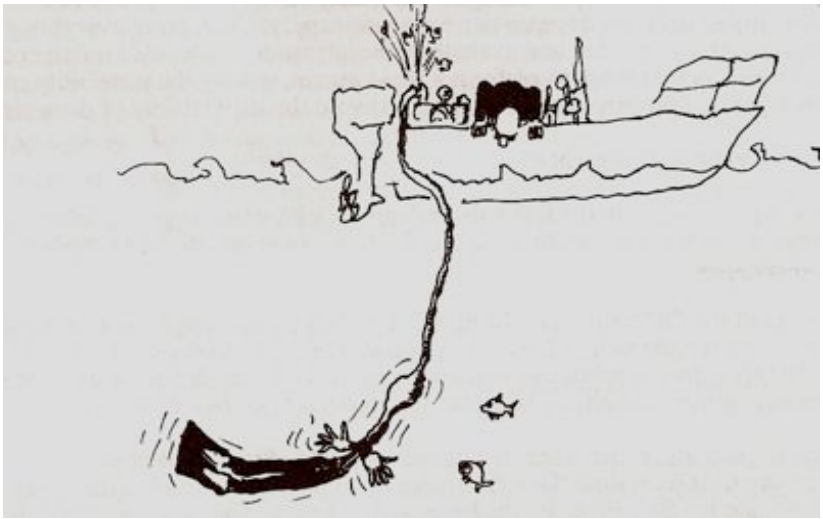


Fig 11.9

SSBA with no non-return valve, when the compressor stops