C. Pressure

Dysbarisum (Decompression sickness)

Dysbarisum is the collective term used to describe the pathogenic changes that occur when the human body is exposed to environmental pressure changes (alternobaric exposure). Those altered pressures are translated into un physiologic behavior of gases in organs and tissues. Failure to adequately or timely adapt to those changes, can generate (depending on a number of exposure and individual factors) the different clinical syndromes of dysbarisum. Alternobaric exposure is a concern in a number of occupational and recreational activities, such diving, compressed air work (as in tunnel constriction and Caisson), as well as in aviation, mountain climbing, and high-altitude flying.

Exposure to altered environmental pressures followed by a return to atmospheric pressure occurs in a number of settings. When the rapidity of pressure changes exceeds that of the compensatory and adaptive mechanisms of the human body, dysbaric disorders can result, depending in part on interindividual differences in responses and susceptibility.

Decompression sickness can occur in three groups of persons:-

- 1. Those working in compressed air who are too rapidly decompressed (Caisson disease).
- 2. Divers who surface too rapidly from depths greater than about 33 feet (10 meters).
- 3. Crew or paratroopers in aircraft who ascend too rapidly from sea level to height of greater than 10,000 feet (5,487).

Caisson disease

Compressed air work is carried out during tunnel construction and caisson work. A caisson is a watertight chamber used in construction work to construct bridge and tunnel foundation under water. The caisson is an iron or concrete tube open at the bottom, which is weighted and driven into the mad or sand below water. The chamber is placed over the site of the proposed underwater foundation, and air is pumped in at a pressure sufficient to displace the water and allow work to be performed under dry condition. In the United State, working pressures for compressed air have varied from 3.1 to as high as 6.1 atmospheric absolute (ATA). Part of this work can now be done with mechanical devices.

The manifestation of decompression sickness are due to the formation of nitrogen bubbles in the body fluids and in the tissues. Bubbles of nitrogen large enough to cause symptoms when the partial pressure of nitrogen in the tissue rapidly becomes twice as great as its partial pressure in the atmosphere.

The symptoms produced depend upon the site in which the bubbles are formed whkist the size and rate of growth of the bubbles determines the severity of the symptoms.

Symptoms

The first symptoms is usually a dull throbbing pain in the joints, most commonly the kness and shoulders, sometimes preceded by numbness. The effected part is held in a semi-flexed position from which it is difficult to move, hence these symptoms are known amongst divers as the bends. These symptoms occur usually within an hour of decompression. Vertigo is common and may be accompanied y nausea and, vomiting and nystagmus. Colloquially these effects are referred to as the staggers. If the skin is chilled during decompression then erythema and pruritus (the itch or the prickles) are regularly noted. More serious symptoms are caused by bubbles of nitrogen in the pulmonary vasculature and are known as the chokes. These symptoms occur then the bends, sometimes following several hours of complete well-being after decompression. The patient experiences a sensation of burning retrosternal, distress with cough. The symptoms are first relieved by shallow breathing but they progress in severity until coughing becomes paroxysmal and uncontrollable. The patient becomes dyspnoeic and his skin cyanotic, cold, and clammy and the pulse thready. He may pass into coma and die.

Massive air embolism may occur due to the blockage of a bronchus with formation of a lung cyst which ruptures into a bronchial vessel.

Almost any form of neurological complication may be preceded by these symptoms and at one hemiplegia was common as to be known as diver's palsy. The lower segment of the cord are most often affected in divers and although most signs are transient, permanent, sequelae do occur. Spontaneous pneumothorax is a rare complication of too rapid decompression. Haemoconcentration is a characteristic finding, presumably resulting from widespread vascular damage with a resultant loss of plasma into the tissues.

Aseptic necrosis of bone is a late of repeated attacks of decompression sickness. Symptoms are insidious and take up to a year to develop and are due to infarcts in the bone brought about by nitrogen emboli in the nutrient arteries. The bone of the lower limbs are commonly affected as are the knee, hip and shoulder joints. The joint lesions may lead to persistent pain and the development of osteoarthrosis.

Treatment

The symptoms of decompression sickness respond rapidly to quick, adequate recompression. They can be avoided by proper attention to preventive measures. The symptoms never occur in those working at a compression 18 psi (pound per square inch) (40 feet or 12 meters of water) and work at this pressure may be prolonged and decompression rapid. As the pressure is increased the shift must be shortened. Thus, the shift should not be longer than six to eight hour at 36 psi, two to three hours at 45 psi and one hour at 50 psi.

Decompression symptoms do not occur if decompression is gradual. The pressure can be reduced by a half with no ill effect so that in a Caisson the pressure in the first airlock is reduced to half that at the working face and then gradually reduced to normal in the other airlocks. It is a slow, tedious process and men are sometimes inclined to hurry the decompression routine in order to get home quickly.

In the case of a diver, decompression is achieved by raising him in stages. Keeping him at the different levels for longer periods as he approaches the surface. The time for each stage is determined by consulting the various decompression tables and varies according to depth and the duration of the dive.

For a dive of 125 feet or 38 meters lasting half an hour, for example, the diver would be brought to 30 feet (9.5 meters) for five minutes and then to 10 feet (3 meters) for twenty minutes so that he would have a total decompression of twenty-five minutes.

Given the pressurized air craft in use in commercial aviation and the elaborate life-support systems used by military pilots, decompression sickness as the result of high flying is unlikely to be a problem. Should symptoms occur, decompression is brought about by returning to a lower altitude.

Decompression sickness is a true medical emergency, especially when neurologic manifestation occur. The primary treatment for decompression sickness (as well as barotrauma) is to administer hyperbaric therapy accompanied by 100% oxygen. The recompression pressure causes bubbles to become smaller, and breathing pure oxygen produces a gradient by which inert gas in bubbles and tissues can diffuse out of the body.

Barotrauma

Barotrauma is the second leading cause of death in scuba divers. Barotrauma may occur during descent or ascent, whenever a gas-filled space, such as pulmonary alveoli, middle ear, paranasal sinuses, stomach, or dental fillings, fails to equalize its internal pressure relative to changes in ambient pressure. The most dramatic changes in pressure and volume occur under hyperbaric conditions, but the hypobaric environment of aviation and space flight can also predispose the aviator to complications of barotrauma. Manifestation of barotrauma of descent are usually referred to as "squeeze" and those of barotrauma of ascent as "reverse squeeze".

Clinical forms of barotraumas.

- Barotrauma of descent
 - Middle ear descent
 - Para nasal and sinus squeeze
 - Inner ear squeeze
 - Dental filling squeeze
 - Face squeeze
- Bartrauma of ascent
 - Gastric rupture
 - Pneumothorax
 - Pneumomediastinum

- Subcutaneous emphysema
- Pneumopericardium
- Arterial embolism