Chapter 21

OXYGEN TOXICITY

Oxygen (O_2) is toxic when breathed at a partial pressure in excess of 0.4 ATA (40% O_2 at atmospheric pressure) for sufficient time. The greater the concentration and pressure, the shorter the time. The two common forms of O_2 toxicity affect the lungs and the brain.

When O_2 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 O_2 increases.

MECHANISM

The exact cause of O_2 toxicity is unknown. It is generally considered that hyperbaric O_2 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, accumulation of fluid and difficulty with breathing.

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 O_2 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 O_2 TOXICITY

To calculate the inspired partial pressure of O_2, multiply the percentage of inspired O_2 by the ambient pressure in atmospheres absolute and divide by 100.

e.g. the partial pressure of O_2 in room air is :

$$21\% \times 1\text{ATA} \div 100 = 0.21\ \text{ATA}.$$

The risks of O_2 toxicity increase with increasing partial pressure. In general it is usually possible to breath 100% O_2 (1ATA) for 12–24 hours without developing significant pulmonary O_2 toxicity. This duration reduces as the O_2 pressure increases. If therapeutic recompression is contemplated, a maximum period of only 6-12 hours breathing 100% O_2 may be acceptable since the subsequent therapeutic recompression will generally involve the use of hyperbaric O_2, and this will summate with existing O_2 toxicity. The amount of pre-treatment of diving casualties with O_2 will preferably be discussed with the diving physician responsible for the therapeutic recompression.

Oxygen toxicity results from a combination of O_2 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 O_2 toxicity.

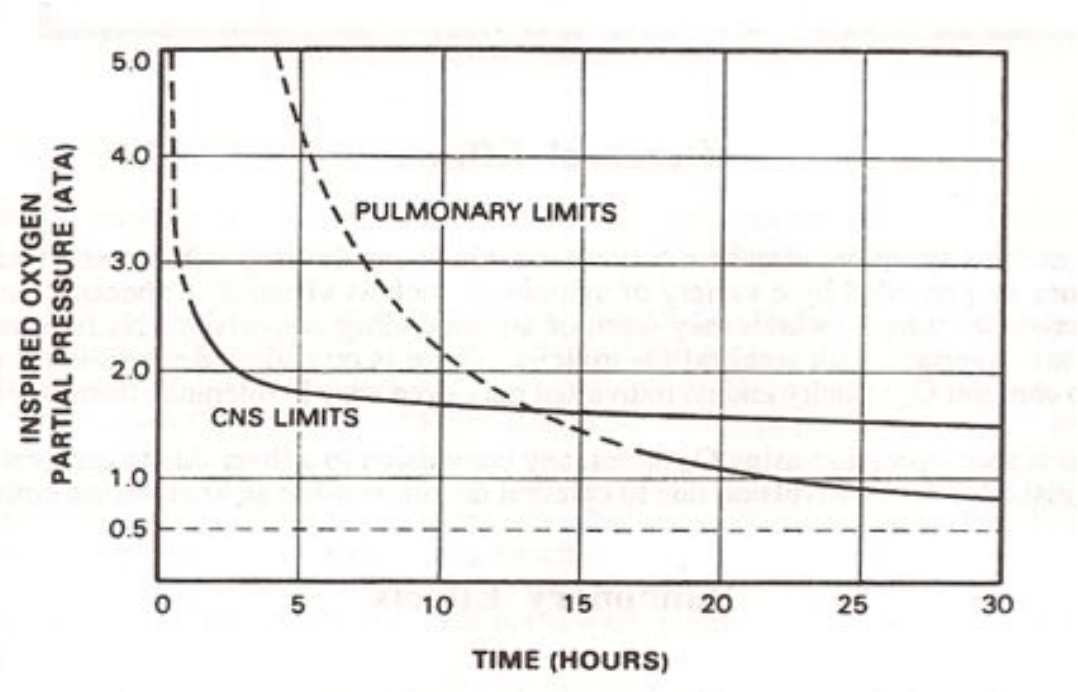

Fig. 21.1

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 O_2 TOXICITY

For **resuscitation, 100% O_2** should be used for hypoxic diving casualties without any fear of O_2 toxicity. As mentioned above, the treatment of decompression sickness and air embolism cases includes 100% O_2, even before consultation with the diving physician regarding any potential negative 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. O_2 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/O_2 (**nitrox**) mixtures are limited to depths which produce an inspired O_2 partial pressure of no more than 1.6 atmospheres, and often less.

In **deep diving** operations, gas mixtures of helium, nitrogen and O_2 should have the composition adjusted so that the inspired partial pressure of O_2 never reaches the toxic range.

Therapeutic recompression using O_2 tables often involves the compression of the diver to 2.8 atmospheres while breathing 100% O_2. There is a significant risk of both pulmonary and cerebral O_2 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. It may sometimes be preceded by a variety of features such as facial pallor, visual or auditory disturbances, tunnel vision, faintness, or facial twitching – which are often not evident underwater. Nausea, retching and even vomiting are common with cerebral O_2 toxicity, as are anxiety and palpitations. There is considerable individual variation in susceptibility to cerebral O_2 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 O_2 tables, any convulsion in a diver due to cerebral O_2 toxicity must be distinguished from a convulsion due to cerebral decompression sickness or air embolism. Sometimes the convulsions occur soon after the O_2 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 O_2 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 O_2 administration is ceased as soon as symptoms develop. If it is essential to continue O_2 therapy however, a reduction in the partial pressure of O_2 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 O_2 therapy.

CONCLUSIONS

1. Recreational divers should not use O_2 enriched diving equipment. Technical divers should not expose themselves to O_2 pressures greater than 1.6 ATA or durations that could cause respiratory manifestations.

2. Resuscitation training with O_2 equipment is of great value to divers and dive boat operators. In diving accidents, the delayed risks of O_2 toxicity are outweighed by the benefits of treating the hypoxic diving casualty.

3. The use of O_2 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 O_2 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 O_2, the use of short air or Heliox breaks during the treatment reduces cerebral and pulmonary O_2 toxicity.

5. There are other logistical problems with the use of oxygen, and some of these are discussed in Chapter 40.