

## Chapter 22

# CARBON DIOXIDE PROBLEMS

Carbon dioxide (CO<sub>2</sub>) 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 CO<sub>2</sub>, mainly through buffering systems in the blood and exhalation from the lungs.

### CARBON DIOXIDE INSUFFICIENCY (HYPOCAPNOEA OR HYPOCAPNIA)

Hypocapnea refers to a blood carbon dioxide (CO<sub>2</sub>) level below normal. The CO<sub>2</sub> 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 CO<sub>2</sub> 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 CO<sub>2</sub> 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.

With most scuba equipment there is an increased breathing resistance, and this tends to diminish the likelihood of hyperventilation and hypocapnoea, increasing depth also increases this resistance to breathing. Some more sophisticated equipment allows for assisted or pressure supplemented respiration, and this will increase the likelihood of hypocapnoea.

## **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 CO<sub>2</sub> 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 the symptoms of hypocapnoea. These illnesses can in themselves, also cause apparent hyperventilation and can mimic anxiety states.

# **CARBON DIOXIDE TOXICITY (HYPERCAPNOEA)**

CO<sub>2</sub> toxicity is due to accumulation of CO<sub>2</sub> 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 CO<sub>2</sub> 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 CO<sub>2</sub> 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 CO<sub>2</sub> is the most common cause of CO<sub>2</sub> toxicity in divers. Hence, CO<sub>2</sub> 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 CO<sub>2</sub> Toxicity

### ❑ Rebreathing equipment.

Some types of military and technical diving equipment conserve gas and reduce exhaust bubble formation by allowing the diver to rebreathe his exhaled gas (exhaust bubbles can be detected by the enemy!). A canister of CO<sub>2</sub> absorbent (soda lime) is included in the circuit to remove the CO<sub>2</sub> which the diver exhales (see Chapters 5 & 43).

This mechanism can fail due to exhaustion of absorbent material, extended dive duration, salt water contamination, improper packing, excessive CO<sub>2</sub> 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 CO<sub>2</sub>.

### ❑ Chambers and habitats.

CO<sub>2</sub> 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 CO<sub>2</sub> absorbent (scrubber). If either of these mechanisms is inadequate, the occupants can develop CO<sub>2</sub> toxicity by rebreathing their own exhaled CO<sub>2</sub>.

### ❑ Scuba.

Since rebreathing is not possible with scuba equipment, CO<sub>2</sub> toxicity is not generally a problem for scuba divers unless there is excessive resistance to breathing (regulator resistance, increased gas density at depth) or a reduced respiratory response of the diver to CO<sub>2</sub> (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 CO<sub>2</sub>.

A rapid accumulation of CO<sub>2</sub> 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 CO<sub>2</sub> levels have been corrected. It often lasts for hours.
- **death**.

CO<sub>2</sub> 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 CO<sub>2</sub> toxicity are temporarily worsened when a diver suddenly resumes breathing normal gases after partially adapting to a high CO<sub>2</sub> pressure.

## Treatment

Any diver, diving with rebreathing equipment, who experiences symptoms of CO<sub>2</sub> 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 CO<sub>2</sub> toxicity should isolate him from the source of CO<sub>2</sub> rebreathing, give **100% oxygen** by mask, and administer **basic life support** (see Chapter 42) including 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 CO<sub>2</sub> toxicity should be treated with a simple analgesic such as paracetamol (acetaminophen).