CASE REPORT

Carbon monoxide poisoning in a diver

H. ALLEN

Accident and Emergency Department, Morriston Hospital, Swansea

SUMMARY

Carbon monoxide poisoning is a well recognized, but uncommon hazard of sport and inshore diving, which occurs either as a result of a faulty air compressor or from air contamination by the exhaust of nearby petrol engines. The incidence of carbon monoxide poisoning may be under-reported as it may mimic decompression sickness, and respond to the same treatment i.e. hyperbaric oxygen.

CASE REPORT

A 25-year-old diver was working in a dock, using an airline from a surface compressor. As is standard practice, the air inlet was situated upwind of the compressor to prevent air contamination with exhaust fumes.

Having dived to 10 m for 30 min, he completed his task and ascended. On surfacing, he was seen by his colleagues to be confused, with possibly a brief loss of consciousness. He had difficulty returning to the quayside, where he needed help removing his equipment and was noted to be coughing, disorientated and weak. On removal of his helmet, the smell of foul air was noted.

On arrival at the A&E department, he was alert and orientated, but complaining of nausea and headache. Examination was unremarkable except for a tachycardia of 120 min⁻¹ and generalized hyperreflexia. ECG showed a sinus tachycardia. Hb CO levels were 37%. Oxygen was commenced by face mask.

After discussion with the duty staff officer on HMS Vernon, Portsmouth, it was decided to treat him as a case of carbon monoxide poisoning with the possibility of air embolism. He was transferred to the nearest available Decompression Unit where hyperbaric oxygen therapy was instituted. This was achieved uneventfully and a complete recovery made.

Correspondence: H. Allen, Accident and Emergency Department, Morriston Hospital, Morriston, Swansea, West Glamorgan, Wales.
Subsequent examination of the air compressor revealed a worn washer on the piston ring, with carbon particles in the first air filter, indicating leakage, and partial combustion of the lubricating fluid. This would produce carbon monoxide contamination of the delivered air supply.

DISCUSSION

Carbon monoxide is a well recognized, but uncommon hazard of diving. Unusually, in 1990 the British Sub-Aqua Club were aware of five cases (Allen, 1990). The last reported case before this was in 1987. In 1990 the majority of cases were from a single source. Both the Department of Energy and Health & Safety Executive are unaware of further cases. This low number of reported incidents may represent under-recognition of the condition, which may mimic decompression sickness, and responds to the same treatment.

Carbon monoxide poisoning in a diver may occur as a result of a faulty compressor, or from exhaust fumes contaminating air entering the compressor (BSAC Diving Manual, 1982). Consequently, it occurs in sports divers and inshore/inland divers, but is unlikely to occur in deep offshore divers where air is supplied by decanting mixtures of pure oxygen and inert gases. Symptoms of carbon-monoxide poisoning occurring at depth are the same as those at atmospheric pressure, i.e. headaches, agitation, confusion, breathlessness on exertion and loss of consciousness, but as the partial pressure of carbon monoxide increase in direct proportion to the absolute pressure, symptoms may be of rapid onset and severe nature. Hence, levels of contamination that, at atmospheric pressure, are likely to be asymptomatic produce severe symptoms at depth such as loss of consciousness, which is likely to have fatal consequences occurring in such a hazardous environment.

Indications for treatment with hyperbaric oxygen currently recommended by the Diving Disease Research Centre are similar to those generally recommended (Anon, 1988) i.e. loss of consciousness, neurological signs, cardiac signs, including T-wave changes on ECG, but if practical, they would recommend treatment with a Hb CO of 25–30% (Bryson, 1990) rather than the usually recommended 40%. The use of hyperbaric oxygen in this manner has been shown to both enhance the elimination of carbon monoxide, decreasing acute morbidity, but also decreases the otherwise high incidence of late neuropsychiatric sequelae (Meredith & Vale, 1988).

REFERENCES