

Neurological oxygen toxicity

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ABSTRACT

SCUBA diving has several risks associated with it from breathing air under pressure—nitrogen narcosis, barotrauma and decompression sickness (the bends). Trimix SCUBA diving involves regulating mixtures of nitrogen, oxygen and helium in an attempt to overcome the risks of narcosis and decompression sickness during deep dives, but introduces other potential hazards such as hypoxia and oxygen toxicity convulsions. This study reports on a seizure during the ascent phase, its potential causes and management and discusses the hazards posed to the diver and his rescuer by an emergency ascent to the surface.

Ascending from a deep (>100 m) dive using trimix techniques, three divers stopped to decompress at 8 m. One had a grand mal seizure and lost consciousness. The dive leader immediately made an emergency ascent with the casualty, commenced expired-air resuscitation and towed him to shore once he started breathing.

The local air ambulance (paramedic/doctor crew) was despatched and found the patient conscious and receiving 100% oxygen. Seizure due to neurological oxygen toxicity was the presumptive diagnosis,^{1 2} but other causes were looked for and excluded when possible—there was no relevant medical history or medication of note, no cardiac symptoms or signs and neurology was grossly intact. Observations were normal and the Glasgow coma scale score was 15. The case was discussed on scene with the nearest hyperbaric chamber who advocated maintaining high fractional inspired oxygen and transporting the patient by low-level flight for emergency recompression. Maximum flight altitude was 500 ft; the patient underwent recompression and made a full recovery.

DISCUSSION

SCUBA diving is popular worldwide—one organisation alone has over 19 million members.³ Seizures during diving are rare but can be fatal and potentially confused with simple drowning—meaning accurate data are difficult to obtain. Furthermore, accident reporting is entirely voluntary, leading to significant underestimates. The best information is the 2008 diver's alert network (DAN) report—this includes all accidents reported to DAN in 2006 with 138 reported fatalities. Seventy-five were further investigated by DAN. The commonest disabling injury was drowning (48%), arterial gas embolism (33%) and cardiac events (28%). There was one reported death for each of decompression sickness, hypoxia and oxygen toxicity seizure (1.3%).⁴

To maintain lung volume at depth, inspired gases must equal hydrostatic pressure. However, breathing air under pressure is hardly benign:

1. Nitrogen is absorbed into the tissues until saturation is reached. During ascent, supersaturation and subsequent bubble formation can occur causing decompression sickness. These bubbles may form at any point in the tissues or bloodstream.
2. Altering pressure proportionally changes the volume of a fixed mass of gas—trapped gas expands during ascent and may cause barotrauma, for example pneumothorax or gas embolism.
3. Altered pharmacological properties—oxygen becomes toxic. The recommended maximum partial pressure oxygen in inspired gas (PiO₂) is 1.4 bar⁵ (142 kPa)—achieved at only 4 m on 100% oxygen.

To reduce these saturation/toxicity effects during deep dives, insoluble helium can be added to air, creating trimix. This would create a hypoxic mix at 1 atmosphere, but not necessarily at depth. For example, a PiO₂ of 1.4 bar occurs with just 12% oxygen at 100 m. Trimix diving therefore requires detailed knowledge and complex techniques.⁶ Separate premixed cylinders or rebreathers are used to achieve different gas compositions for descent/ascent and bottom time, while 100% oxygen is often used for near-surface decompression. This can mean multiple changes of mouthpiece under cold, dark, stressful conditions. If the wrong mouthpiece is used and either a hypoxic mixture, or over 1.4 bar of oxygen, is inspired then seizures and incapacitation are highly likely.

Considering our diver, the differential diagnoses include neurological oxygen toxicity, hypoxia, arterial gas embolism or pre-existent pathology. We know he had 100% oxygen available and he was decompressing at 8 m. If he inadvertently inspired 100% oxygen at 8 m, this would give him a PiO₂ of 1.8 bar (182 kPa). After such a deep dive, he is likely to be cold, tired and stressed—all of which decrease seizure threshold. The oxygen concentration of their bottom mixture is unknown but anything less than 12% would have been hypoxic at 8 m so, again, through mouthpiece misselection, hypoxia is possible. A seizure from gas embolism without subsequent neurological symptoms is unlikely, but also feasible. Furthermore, he has undergone an emergency ascent during the seizure (with its inherent possibility of a closed glottis) so decompression sickness and/or barotrauma are potential sequelae. For any gas embolus or decompression illness, recompression is one of the first-line treatments. The diver must be kept flat, given 100% oxygen, intravenous fluids, kept warm, monitored for ABCD and transferred at the lowest safe level to

a British Hyperbaric Association category 1 recompression chamber.⁷

CONCLUSION

Seizures underwater are frequently fatal yet present a set of irreconcilable problems. The closed glottis makes ascent extremely dangerous; however, the surface is the only place where help can be given. This case report would suggest, at shallow depth at least, that immediate ascent is probably the best course of action. Initial treatment must include high fractional inspired oxygen and follow standard ABCD algorithms along with referral to the nearest hyperbaric unit. See <http://www.hyperbaric.org.uk/> for hyperbaric unit details.⁸

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