Facial Nerve Palsy Secondary to Oxygen Toxicity in Closed-circuit diving: A Case Report

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Introduction:

The use of closed-circuit breathing apparatus (CCBA) is common in many navies around the world. Prompt recognition of the symptoms of oxygen toxicity is necessary so that patients can receive timely and appropriate medical care. Oxygen toxicity is well known to affect the ocular, pulmonary and central nervous system (CNS), with CNS complications the leading cause of drowning incidents due to seizures and loss of consciousness underwater.

Case Presentation:

We report a case of a 24-year-old diver of the Singapore Armed Forces (SAF) with an uncommon presentation of oxygen toxicity after CCBA diving using 100% oxygen. The dive time was 89 minutes to a maximum depth of 5 metres.

- No previous medical history
- Underwent CCBA training on 100% oxygen for previous 3 days. Max Depth 6m, Total time 100 min

On surfacing, facial asymmetry was noted by buddy. No ear discomfort or other neurological symptom.

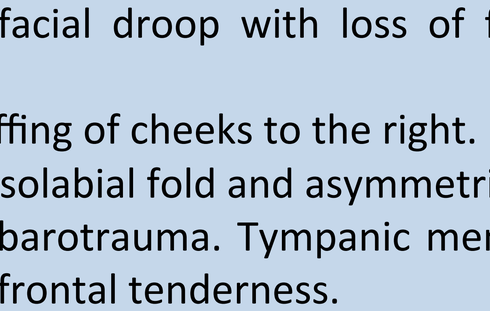
On Examination (10 minutes from presentation):

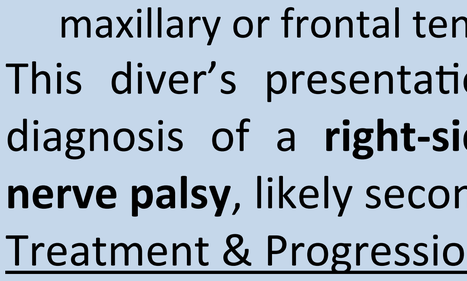
- Stable vital signs, GCS 15
- Right-sided facial droop with loss of frontalis/orbicularis oris action.
- Impaired puffing of cheeks to the right.
- Flattened nasolabial fold and asymmetrical smile.
- No signs of barotrauma. Tympanic membrane normal, no maxillary or frontal tenderness.

This diver's presentation was most consistent with a diagnosis of a **right-sided lower motor neuron facial nerve palsy**, likely secondary to oxygen toxicity.

Treatment & Progression:

- Oral Prednisolone (30mg OM x 5 days) given
- Review at 4 hours from presentation: Most examination signs appear resolved.
- Review at 21 hours from presentation: Full recovery noted.
- Diver was advised to limit further diving with 100% oxygen to a maximum of 1 hour per dive.





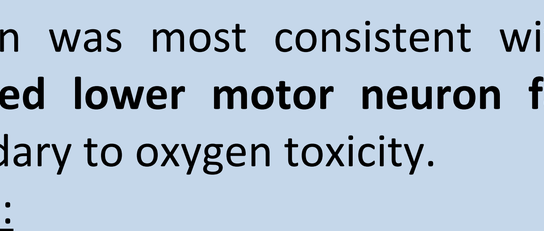
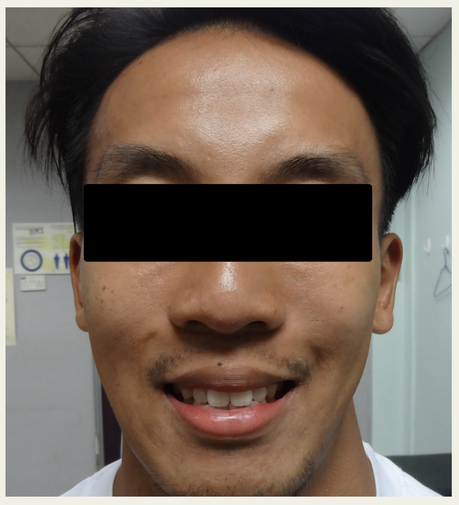


Fig. 1: Initial presentation with asymmetrical smile, and flattened nasolabial fold (top), asymmetrical puffing of cheeks (left), droopy right eyelid and impaired right frontalis (right).



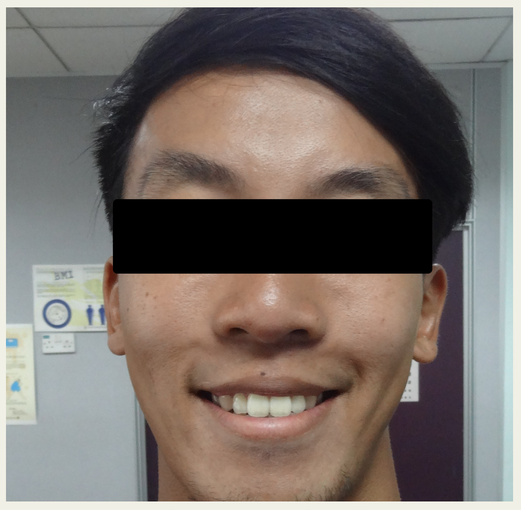


Fig. 2 (left): Review at 4 hours, most signs have resolved.
Fig. 3 (right): Full recovery noted at 21 hours from presentation.

Discussion:

Oxygen toxicity is known to occur during periods of exposure to a higher partial pressure of oxygen, such as during compression treatment, hyperbaric oxygen therapy and CCBA diving. There have also been case reports of oxygen toxicity occurring in the use of Nitrox, Heliox and Trimix gas mixtures at deeper depths. National Oceanic and Atmospheric Administration (NOAA) guidelines suggest a maximum exposure of 45 minutes at an oxygen tension limit of 161 kPa (1.6 ATA). While the dive profile of this case appears to be within safety parameters of oxygen excursion tables following BR 2806, oxygen toxicity has been found to occur at exposures as low as >0.6 ATA.

Recognized presentation of CNS oxygen toxicity include (in descending frequency of occurrence): **limb convulsions, hyperventilation, difficulty maintaining steady depth, headache, visual disturbances, confusion and in rare cases facial muscle twitching**. Symptoms such as confusion and hyperventilation were also associated with loss of consciousness. To the author's knowledge, there is no available literature regarding oxygen toxicity resulting in peripheral nerve lesions. Therefore, this case suggests that the manifestations of oxygen toxicity on our nervous system may be more varied and innocuous than previously known. Divers and diving physicians should maintain a high index of suspicion of oxygen toxicity and consider a possible atypical presentation in patients with a dive history using 100% oxygen to enable timely administration of appropriate medical care.

Possible differential diagnoses include faciobaroparesis and Bell's palsy. However as mentioned, signs or symptoms of middle ear barotrauma, of which faciobaroparesis is commonly associated with, were absent. An acute manifestation of Bell's palsy was difficult to exclude at initial presentation, therefore oral prednisolone was administered to cover this possibility. Rapid resolution of symptoms suggested that oxygen toxicity was the more likely diagnosis, as Bell's palsy may take up to a month to fully recover.

References:

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