

CNS Oxygen Toxicity in Closed-Circuit Diving: Signs and Symptoms Before Loss of Consciousness

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Introduction: There is a dearth of information regarding CNS oxygen toxicity accidents in closed-circuit oxygen diving. The aims of the present study were to report the sensations and symptoms that accompany CNS oxygen toxicity accidents, and to evaluate whether loss of consciousness can occur without any warning signs. **Methods:** We documented 36 CNS oxygen toxicity accidents in closed-circuit oxygen diving. The full accident inquiry included the first report from the diving unit, an interview of the victim and his buddy by the researchers, and an examination of the diving equipment. **Results:** The symptoms that appeared before termination of a dive, as reported by the victim or his buddy, were as follows (in descending order of frequency): limb convulsions; hyperventilation; difficulty maintaining a steady depth; headache; and visual disturbances. The symptoms that appeared after detachment from the mouthpiece were, in descending order of frequency: headache; loss of consciousness; confusion; weakness; dizziness; and facial muscle twitching and limb convulsions. A high inspired CO₂ [mean 4.2 kPa (29.9 mmHg)] was connected with loss of consciousness. No dive was terminated before at least two symptoms (mean 3.4) had been noted a minimum of 5 min before termination. **Discussion:** Symptoms that are accepted as being related to CNS oxygen toxicity, as well as others such as headache, difficulty maintaining a steady depth, hyperventilation, weakness, and a choking sensation, were more frequent among the O₂ accident victims compared with divers who did not interrupt their dives. **Conclusion:** Awareness of any unusual sensation can prevent a potentially dangerous situation from arising.

Keywords: hyperbaric oxygen, diving accidents, symptoms, risk.

CLOSED-CIRCUIT OXYGEN diving is common practice in many of the world's navies, and the use of enriched oxygen mixtures is becoming increasingly widespread in professional and sport diving. With this expansion of diving techniques, the risk of central nervous system (CNS) oxygen toxicity has now become a prominent issue. Various aspects of oxygen diving have been studied in the dry or wet hyperbaric chamber (3,5,12), and these were summarized together with other reports by Harabin (7). However, the complexity of in-water diving is much greater than a dry or wet "dive" in a controlled hyperbaric experiment. Some of the factors involved are the cold, being totally submerged, various levels of exercise, limited vision, tension, and the accumulation of carbon dioxide. Some authors suggest that convulsions and loss of consciousness can occur without premonitory symptoms. In the U.S. Navy Diving Manual (13), the claim is made that "There may be no warning of an impending convulsion

to provide the diver the opportunity to return to the surface."

There is a dearth of information regarding CNS oxygen toxicity accidents in closed-circuit oxygen diving. Childs (4) analyzed 114 diving incidents (excluding oxygen dives), 42 of which involved unexplained loss of consciousness. In about half of these no premonitory symptoms were reported, although the author suggests the divers in question may have been unable to recall the events preceding loss of consciousness. Leitch (9) analyzed 46 unusual incidents, where no mechanical or clear-cut cause was evident, in a well-documented series of 1303 dives (air, trimix, and heliox diving) at the Royal Navy Deep Trials Unit. He demonstrated that incidents occurred when the PO₂ during the dive was greater than 120 kPa (1.2 ATA), but only in air breathing did the incident rate increase with increasing PO₂ above 120 kPa (1.2 ATA). It was impossible to separate the effect of oxygen toxicity from other effects of elevated pressure, the inert gas, and nitrogen narcosis. Likewise, Morrison et al. (10) analyzed two cases of loss of consciousness underwater, suggesting involvement of CO₂ retention and nitrogen narcosis. The oxygen pressure in the above cases was 170 kPa (1.7 ATA), and the circumstances, therefore, agree with our finding (6) of a close connection between reduced sensitivity to CO₂ and increased risk of CNS oxygen toxicity. Thus CNS oxygen toxicity may have been the reason for loss of consciousness in Morrison et al.'s report (10). It has been suggested that other fatalities in diving might also be related to CNS oxygen toxicity (11). However, to the best of our knowledge, there is no published information on CNS oxygen toxicity accidents due to in-water ocean diving using closed-circuit oxygen apparatus at depths shallower than 7 msw. The present report on closed-circuit oxygen diving has as its purpose: 1) to present the sensations and symptoms that accompany oxygen toxicity accidents; and 2) to eval-

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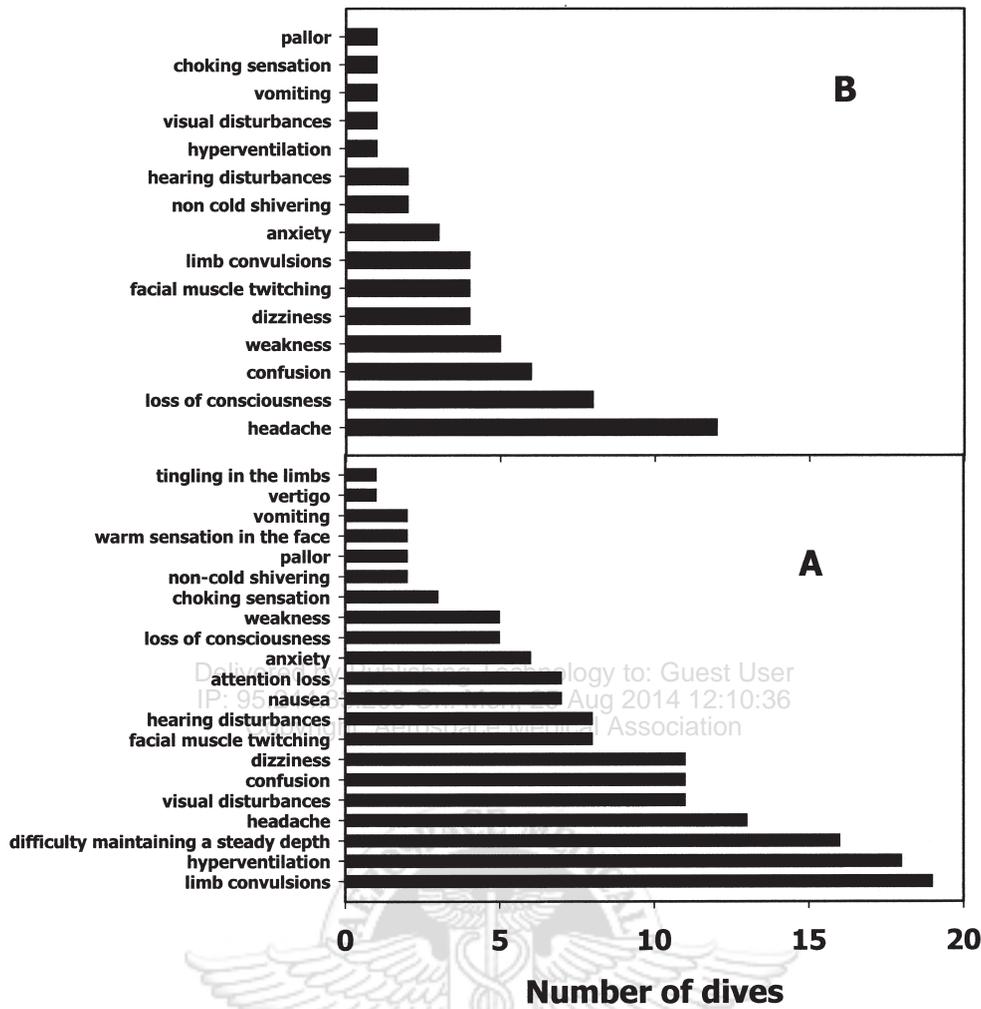


Fig. 1. Frequency of the symptoms appearing A) before termination of a dive, and B) after termination of a dive. Number of dives (x-axis) for which a symptom on the y-axis was reported. Total number of O₂ accidents was 36.

uate whether loss of consciousness can occur without any warning signs.

METHODS

For the past 25 yr, we have documented oxygen toxicity accidents due to closed-circuit oxygen diving in the Israel Defense Forces Navy. Diving depth was shallower than 7 msw, with a depth distribution similar to that reported in an earlier paper (2). An accident is defined as a dive that was terminated because of CNS oxygen toxicity based on the summary report drawn up after an inquiry into the incident. The full inquiry includes the first report from the diving unit, the equipment examination report from their expert in the machine shop, and a thorough debriefing of the victim and his buddy by a physiologist and physician at the Israel Naval Medical Institute. The interview and test are usually completed on the day of the accident, or a day later at most. The sequence and chronology of events are recorded from the account given by the victim and his buddy, who both glance frequently at their watches during the dive. If the victim's diving apparatus is available, and has remained closed and intact, a test is conducted to check

for possible failure of the carbon dioxide absorbent. The diver breathes from his apparatus for 10 min while exercising on a pedal ergometer in a ventilated room at a workload of approximately 1 L of oxygen per min, and inspired O₂ and CO₂ are monitored by O₂ and CO₂ analyzers (S-3A/I and CD-3A, Ametek, Applied Electrochemistry, Pittsburgh, PA). The rate of gas exchange is determined from the recorded ventilation and mixed-box gas concentration while adjusting the workload. A dive is defined as a high CO₂ dive if at the end of this test the inspired CO₂ is above 1 kPa (7.1 mmHg), and as a low CO₂ dive when it is below 1 kPa. We selected 1 kPa CO₂ as the borderline because we had previously shown that values above 1 kPa CO₂ enhanced CNS oxygen toxicity in rats (1). This also agrees with our experience in closed-circuit oxygen diving, where in a number of cases the level of CO₂ in the inhalation hose of a diver who suffered CNS oxygen toxicity was above 1 kPa, whereas a much lower level was detected in his unaffected buddy's inhalation hose. In some of the accidents there was not a full inquiry. For example, the buddy was interviewed in only 22 of the 36 accidents.

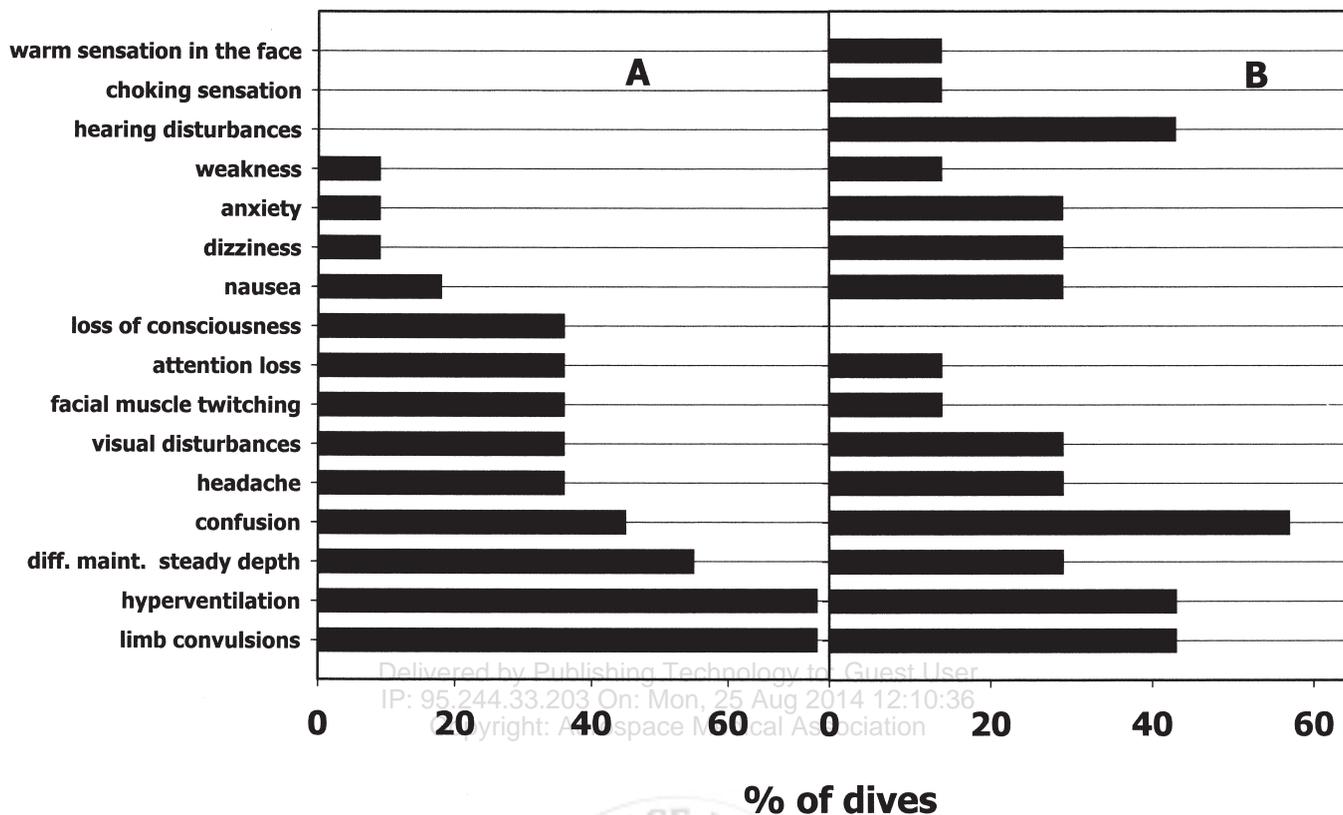


Fig. 2. Frequency of the symptoms appearing before termination of a dive as related to CO₂ level in the inspired gas in the 10-min laboratory test conducted following the accident: A) high CO₂ (2.5–8.0%, n = 11) dives vs. B) low CO₂ (0–0.7%, n = 7) dives.

RESULTS

The frequency with which the symptoms appeared before termination of a dive is shown in the lower panel of Fig. 1. In a large number of dives there were limb convulsions (uncontrollable tremor), hyperventilation, difficulty maintaining a steady depth, and headache. In about one-third of the dives there were visual disturbances, confusion, and dizziness. In about one-quarter of the dives there was facial muscle twitching, nausea, hearing disturbances, attention loss, anxiety, weakness, and loss of consciousness. In a smaller number of dives other symptoms were reported: a choking sensation; non-cold shivering; pallor; a warm sensation in the face; vomiting; vertigo; and tingling in the limbs. A number of symptoms were experienced immediately after detachment from the mouthpiece and breathing atmospheric air. These are presented in the upper panel of Fig. 1. It is clear from Fig. 1 that loss of consciousness occurred more often after termination of a dive. The most frequent symptoms after termination of a dive were headache, confusion, weakness, and facial muscle twitching and limb convulsions.

Carbon dioxide was measured in the exercising diver for 18 of the 36 dives. These were divided into a low CO₂ group [mean 0.2, range 0–0.7 kPa (mean 1.4, range 0–5.0 mmHg)] and a high CO₂ group [mean 4.2, range 2.5–8.0 kPa (mean 29.9, range 17.8–57.0 mmHg)] according to the inspired CO₂ at the end of the 10-min test. The main cause of increased CO₂ was failure of the absorbent: incorrect filling and packing; a prolonged

period of strenuous activity with high CO₂ production; and water leaking into the canister. The frequency with which symptoms appeared before termination of a dive in these two groups is presented in Fig. 2. Loss of consciousness occurred in 40% of the dives in the high CO₂ group, and not at all in the low CO₂ dives. It can be seen that hyperventilation was frequent in the low CO₂ group as well.

The dives are presented in Fig. 3 as a function of the number of symptoms per dive before termination (loss of consciousness not included). In the majority of the accidents, three or more symptoms appeared before termination of a dive. Only four dives were terminated after the appearance of two symptoms, and there was no case in which a dive was terminated after the appearance of only one symptom or when there were none at all. When we reviewed the dives with a small number of reported symptoms, it became clear that there had not been a full inquiry into these incidents. A full inquiry was conducted following only 40% of the dives for which two to three symptoms were reported, compared with 70% of the dives for which four to five symptoms were reported. Two to four symptoms (mean 3.4) appeared before termination in the five dives in which loss of consciousness occurred before dive termination. The frequency of these symptoms was: limb convulsions in four dives; hyperventilation in four dives; difficulty maintaining a steady depth in two dives; and nausea, dizziness, visual disturbances, facial

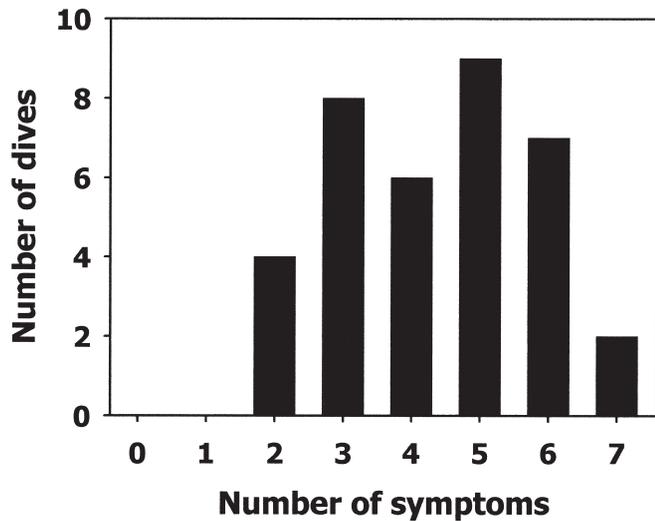


Fig. 3. The number of dives (y-axis) from among the 36 O₂ accidents in which a specific number of symptoms (x-axis) appeared before termination of the dive (loss of consciousness not included).

muscle twitching, confusion, weakness, and headache each reported in one dive.

From 23 of the 36 accident inquiries, it was possible to derive the time of the first symptom and to compare this with the time the dive was terminated. The chronology of these events is depicted in Fig. 4. The shortest time between the first symptom and dive termination was 5 min.

DISCUSSION

Leitch (9) found that incidents occurred in trimix, heliox, and air diving when the oxygen pressure was greater than 120 kPa. His summary of the problems reported before loss of consciousness was (in descending order): unsteadiness/weakness; equipment; neurological; thermal; effect on consciousness; and general distress. These symptoms were reported to occur before loss of consciousness with a similar frequency by Childs (4). This is somewhat different from the list of symptoms presented in Fig. 1, supporting the suggestion by Leitch (9) that factors other than oxygen toxicity (such as nitrogen narcosis, high pressure, and thermal effects) also affected the diving incidents in his study. A comparison with past reports of diving accidents stresses the importance of the present report as the first published analysis of CNS oxygen toxicity accidents due to shallow, closed-circuit oxygen diving.

A large number of the symptoms that preceded the unscheduled termination of a dive were reported less frequently in dives that continued according to plan until completion. This can be seen by comparing the present data with those from 2522 uninterrupted closed-circuit oxygen dives reported by Arieli et al. (2). In Table I, the symptoms from both pools of data are presented in the descending order of frequency for those from the O₂ accident data pool. Symptoms accepted as being related to CNS oxygen toxicity (2,8) are more frequent in the O₂ accident data pool. However, other symptoms which have not previously been as-

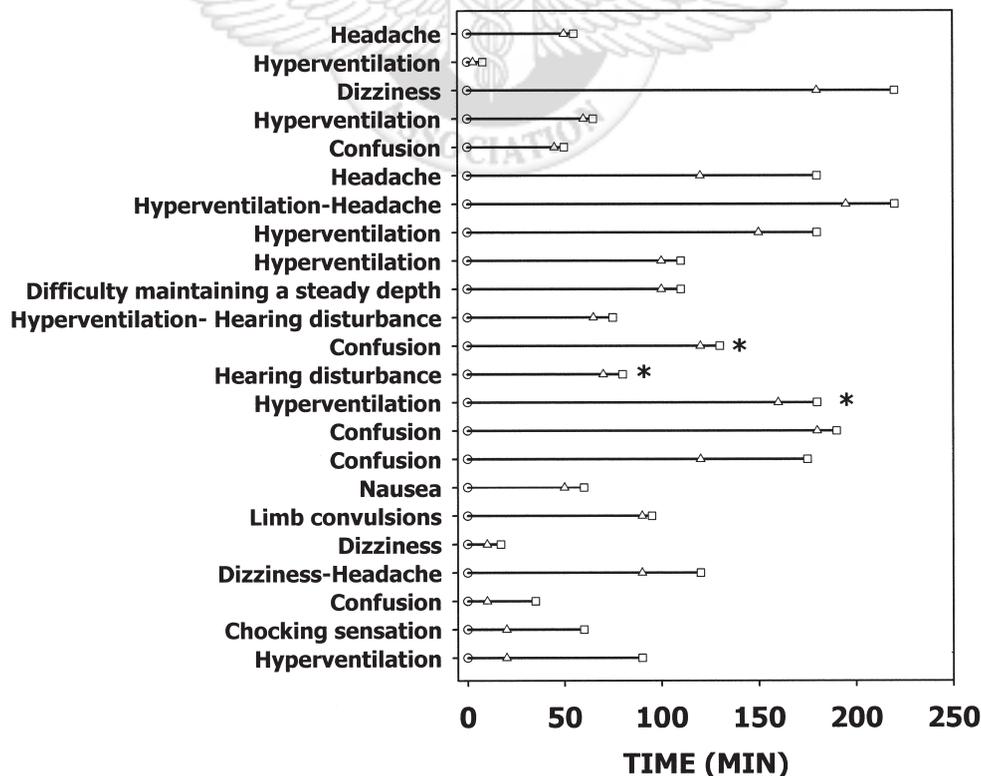


Fig. 4. The time of the first reported symptom compared with the time at which the dive was terminated. A circle represents the beginning of a dive, a triangle the first symptom, and a square the time at which the dive was terminated. Each line represents one of 23 dives. The first symptom is marked on the y-axis. An asterisk (*) denotes underwater loss of consciousness.

TABLE I. COMPARISON OF THE FREQUENCY OF SYMPTOMS IN 2522 UNINTERRUPTED DIVES (REF. 2) AND 36 O₂ ACCIDENTS.

Symptom	Frequency (%)	
	In 2522 Uninterrupted Dives	In 36 O ₂ Accidents
Limb convulsions	0	51
Hyperventilation	2.6	49
Difficulty maintaining a steady depth	0.9	43
Headache	4.5	35
Visual disturbances	0.28	30
Confusion	0.1	30
Dizziness	1.6	30
Facial muscle twitching	0.2	22
Hearing disturbances	0.4	22
Nausea	2.6	19
Attention loss	0	19
Anxiety	0.2	16
Weakness	0	14
Choking sensation	0.4	8
Non-cold shivering	0.1	5.4
Pallor	0	5.4
A warm sensation in the face	0	5.4
Vomiting	0.2	5.4

signed to CNS oxygen toxicity, such as headache difficulty maintaining a steady depth, hyperventilation, weakness, and a choking sensation, are also more frequent in this data pool. All of these symptoms can serve as a warning sign in diving that CNS oxygen toxicity may be expected.

In no case did loss of consciousness occur without some premonitory symptom, and there was at least 5 min between the first sign and the subsequent termination of the dive. Because the present review of accidents was not designed to answer a specific question, a differently designed review might well reveal even earlier symptoms. Awareness of any unusual sensation can prevent a potentially dangerous situation from arising when diving at shallow depths. In deep diving, the time span may not be sufficient to permit a safe escape. The crucial role of CO₂ in CNS oxygen toxicity calls for special care. Carbon dioxide should not be allowed to rise in the breathing cycle, and this stresses the importance of developing a CO₂ sensor to be incorporated into closed-circuit diving apparatus.

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