Impaired consciousness when scuba diving associated with vasovagal syncope

Peter Wilmshurst[1], Margaret Clamp[2]

[1] *Royal Stoke University Hospital, Stoke-on-Trent, United Kingdom*
[2] *MC Occupational Health, Colwick, Nottingham, United Kingdom*

Corresponding author: *Dr Peter Wilmshurst, Consultant Cardiologist, Royal Stoke University Hospital, Stoke-on-Trent, ST4 6QG, United Kingdom*
peter.wilmshurst@doctors.org.uk

Key words
Vasovagal syncope; Neurocardiogenic syncope; Faints; Micturition syncope; Scuba diving

Abstract
(Wilmshurst P, Clamp M. Impaired consciousness when scuba diving associated with vasovagal syncope. Diving and Hyperbaric Medicine. 2020 December 20;50(4):421–423. doi: 10.28920/dhm50.4.421-423. PMID: 33325026.)
Introduction: Drowning is likely to result from impairment of consciousness when scuba diving. Causes include toxic effects of breathing gas, including nitrogen narcosis and oxygen toxicity, and arterial gas embolism.
Methods: Review of the medical records of scuba divers who had impaired consciousness underwater that could not be attributed to toxic effects of breathing gas or arterial gas embolism.
Results: Four scuba divers had episodes of impaired consciousness when at shallow depths (8–18 m) underwater. The descriptions of the episodes were very similar. Three had histories of recurrent episodes of vasovagal syncope on land.
Conclusions: Absence of other causes for their impaired consciousness underwater leads to the conclusion that the probable cause was vasovagal syncope.

Introduction

A history of blackouts, unless long ago and unlikely to recur, is considered a contraindication to scuba diving because of the risk of drowning if unconsciousness occurs underwater.

Vasovagal syncope (neurocardiogenic syncope) is one of the most frequent causes of loss of consciousness. It is usually benign. It is caused by a combination of a neurally mediated increase of parasympathetic tone to cause bradycardia and a reduction of sympathetic tone to cause peripheral vasodilatation.[1,2] The latter causes blood to pool in the legs and causes a sudden reduction of cardiac filling pressures and hence stroke volume. Vasovagal syncope most commonly occurs when people are standing when the effects of blood pooling on cardiac filling and hence cardiac output are greatest. It rarely occurs when people are lying down, because when recumbent the cardiovascular effects of peripheral blood pooling due to vasodilatation are smaller. However, even when lying down, bradycardia occurs and may be sufficient to cause unconsciousness in susceptible people.

The hydrostatic effects of head-out immersion increase venous return so that cardiac filling pressures are elevated.[3] As a result, theoretically there may be some protection against occurrence of vasovagal syncope when surface swimming and scuba diving.

This article describes four individuals who, whilst scuba diving, had impaired consciousness which was almost certainly the result of vasovagal syncope. No other plausible explanation was found. The description of the episode in each case was very similar. Some of the divers described were seen many years ago. As a result, it was possible to contact only the patient seen most recently to obtain permission to publish the histories. So, only essential patient information is presented to avoid identification of the cases. The patient who was contacted gave written consent for data to be published.

Observations

In the last 20 years, four scuba divers (two male and two female, aged 22 to 55 years) presented to the authors with a history of an episode of impaired consciousness whilst scuba diving that could not be attributed to well known causes of unconsciousness in divers such as hypoxia, toxic gas effects and arterial gas embolism. Both women had more than ten episodes of vasovagal syncope over many years. One man had four episodes of micturition syncope, when he got up at night to pass urine, usually after drinking more alcohol than usual for him the evening before. The other man had no history of blackouts out of the water, but the description of his episode underwater was so similar to that in the other three divers that it is believed likely to have a similar mechanism. None of the divers had other significant medical history and none were taking medication.

One diver had performed about 150 uneventful dives previously and her unresponsive event occurred on a dive to 18 metres (m) depth. The other three divers were trainees, who were diving with an instructor on their fifth, ninth and twelfth dives to depths of 8–12 m. All were breathing air on open-circuit scuba. The episodes of impaired consciousness occurred 8 to 17 minutes into the dives and were not during either descent or ascent.

In each case, the diver's buddy or instructor reported that the diver ceased swimming and had a blank staring expression. Some described the diver as 'not with it'. They did not have convulsions and they retained their regulators in their mouths, but they did not respond to signals from their buddy and did not react to a hand waved in front of their facemask. Their buddies took hold of the divers and performed a controlled lift to the surface. Two divers appeared to come round during the ascent and one came round immediately on surfacing. These three were oriented immediately after surfacing. The fourth, a man of 48, was recovered into a boat and, according to his instructor, he 'was not with it' for about 10 minutes.

One male diver recalled feeling cold on his dive. Both the male divers recalled feeling tired and feeling as if they were falling asleep immediately before their impaired consciousness. The experienced female diver recalled a mild headache before she became unresponsive. The fourth diver had no recollection of anything unusual. None of the divers had neurological findings immediately after the events.

When examined subsequently, one had significant postural hypotension (a 26 mmHg drop in systolic blood when standing from lying). Cardiovascular findings were otherwise normal in all. Their electrocardiograms and 24 hours Holter records were normal. Specifically there was no evidence of Long QT syndrome or other cardiac ion-channelopathies. One had an echocardiogram, which was normal. The three novice divers were advised not to dive again. After much discussion, the experienced diver was advised to dive only with an experienced buddy and that the dives should not require compulsory decompression stops.

Discussion

Scuba divers breathing air can lose consciousness at depth as a result of nitrogen narcosis, oxygen toxicity, carbon monoxide toxicity and other toxic gas effects. The shallow depths of the dives in these cases means that gas toxicity is implausible. In addition, three of the divers regained consciousness within minutes without changing their breathing gas source, which would be difficult to explain if the gas source had toxic impurities.

Arterial gas embolism secondary to pulmonary barotrauma can cause unconsciousness during ascent and usually there are neurological findings after surfacing, if only transiently. The impaired consciousness in these cases was unrelated to ascent and there was no neurological abnormality.

None of the divers had convulsive movements and were rapidly orientated, which makes it unlikely that they had an epileptic fit. Absence attacks due to petit mal epilepsy have some similarities to the events underwater but have much shorter durations. In addition, the attacks out of the water in three of the four divers were not consistent with petit mal epilepsy, but rather were recurrent episodes of vasovagal syncope.

It seems likely that these four divers had vasovagal syncope underwater. Three had some premonition or symptoms before impaired consciousness occurred. Their episodes of impaired consciousness underwater did not result in them losing their regulators. The reason that they did not become more deeply unconscious may have been that the hydrostatic effects of immersion mitigated the effects of vasodilatation.

Vasovagal syncope has been reported in a competitive swimmer during a swimming race.[4] Breath-hold divers can experience impaired consciousness and even syncope as a result of hypoxia, particularly during ascent when the partial pressure of oxygen in the lungs and hence in the arterial blood is decreasing rapidly.[5] In a scuba diver breathing air, the partial pressure of oxygen in inspired air (P_iO_2) decreases during ascent in line with absolute pressure, but the P_iO_2 cannot fall below 0.21 bar. So hypoxia is not an issue in scuba divers breathing air if their equipment is functioning properly and they have no acute cardiorespiratory disease (such as immersion pulmonary oedema).

The diving reflex can cause bradycardia in breath-hold divers, but only a small effect from cold stimulation is observed in scuba divers because they are not breath-holding. Elevated P_iO_2 produces a statistically significant but small dose-dependant reduction in heart rate as a result of parasympathetic stimulation and that might be expected to have a small effect on heart rate at depth.[6] The episodes of impaired consciousness occurred at shallow depths, where the degree of elevation of P_iO_2 would be expected to have only a very small effect on heart rate in most divers.[6] The effect might be larger in individuals who had greater susceptibility to changes in autonomic tone as suggested by the history of vasovagal syncope in three of the four. Immersion in test conditions in a swimming pool was found to be a powerful stimulus for both the sympathetic and parasympathetic nervous system in scuba divers.[7] Head-out immersion and submersion in scuba divers caused changes in all measures of heart rate variability consistent with an increase in parasympathetic activity.[7]

In the divers reported here, three of whom were highly susceptible to vasovagal syncope, the parasympathetic activation caused by immersion and by the elevated partial pressure of oxygen might have been great enough to cause a

clinically important bradycardia such that cerebral perfusion was reduced even though cardiac filling pressures were relatively well maintained by hydrostatic compression of peripheral vessels.

This small series of clinical observations suggests that individuals prone to vasovagal syncope are at risk of impaired consciousness when scuba diving. These observations require confirmation by other diving doctors.

References

1 Lewis T. A lecture on vasovagal syncope and the carotid sinus mechanism. Br Med J. 1932;1:873–6. doi: 10.1136/bmj.1.3723.873. PMID: 20776843. PMCID: PMC2520889.

2 Morillo CA, Eckberg DL, Ellenbogen KA, Beightol LA, Hoag JB, Tahvanainen KU, et al. Vagal and sympathetic mechanisms in patients with orthostatic vasovagal syncope. Circulation. 1997;96:2509–13. doi: 10.1161/01.CIR.96.8.2509. PMID: 9355886.

3 Arborelius M Jr, Balldin UI, Lilja B, Lundgren CE. Hemodynamic changes in man during immersion with the head above water. Aerosp Med 1972;43:592–8. PMID: 5035546.

4 Edenfield KM, Stern AN, Dillon MC, Burkart TA, Clugston JR. A case of vasovagal syncope in a collegiate swimmer during competition. Current Sports Med Rep. 2015;14:86–90. doi: 10.1249/JSR.0000000000000128. PMID: 25757001.

5 Fitz-Clarke JR. Adverse events in competitive breath-hold diving. Undersea Hyperb Med. 2006:33:55–62. PMID: 16602257.

6 Shibata S, Iwasaki K, Ogawa Y, Kato J, Ogawa S. Cardiovascular neuroregulation during acute exposure to 40, 70 and 100% oxygen at sea level. Aviat Space Environ Med. 2005;76:1105–10. PMID: 16370259.

7 Schipke JD, Pelzer M. Effect of immersion, submersion and scuba diving on heart rate variability. Br J Sports Med. 2001;35:174–80. doi: 10.1136/bjsm.35.3.174. PMID: 11375876. PMCID: PMC1724326.

Conflicts of interest and funding: nil

Submitted: 03 April 2020
Accepted after revision: 29 August 2020