Prolonged syncope with multifactorial pulmonary oedema related to dry apnoea training: Safety concerns in unsupervised dry static apnoea

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Abstract

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Many competitive breath-hold divers use dry apnoea routines to improve their tolerance to hypoxia and hypercapnia, varying the amount of prior hyperventilation and lung volume. When hyperventilating and exhaling to residual volume prior to starting a breath-hold, hypoxia is reached quickly and without too much discomfort from respiratory drive. Cerebral hypoxia with loss of consciousness (LOC) can easily result. Here, we report on a case where an unsupervised diver used a nose clip that is thought to have interfered with his resumption of breathing after LOC. Consequently, he suffered an extended period of severe hypoxia, with poor ventilation and recovery. He also held his breath on empty lungs; thus, trying to inhale created an intrathoracic sub-atmospheric pressure. Upon imaging at the hospital, severe intralobular pulmonary oedema was noted, with similarities to images presented in divers suffering from pulmonary barotrauma of descent (squeeze, immersion pulmonary oedema). Describing the physiological phenomena observed in this case highlights the risks associated with unsupervised exhalatory breath-holding after hyperventilation as a training practice in competitive freediving.

Introduction

Freediving-related cerebral hypoxia is well documented.[1,2] In-water activities bear a risk of cerebral hypoxia manifesting as loss of motor control, and loss of consciousness (LOC).[2,3] Dry apnoea exercises (performed on land), which are commonly performed by competitive freedivers, also carry this risk albeit without risk of drowning. These apnoea exercises improve breath-hold ability; their benefits are attributed to increased tolerance to hypercapnia, hypoxia, and onset and strength of the diving response.[4–6] They also familiarise the diver to the uncomfortable sensation of dyspnoea/asphyxia. When performing dry training, hyperventilation can be used to prolong apnoea time with the absence of diaphragmatic contractions, creating a state whereby hypoxia may be achieved without hypercapnia-induced dyspnoea.

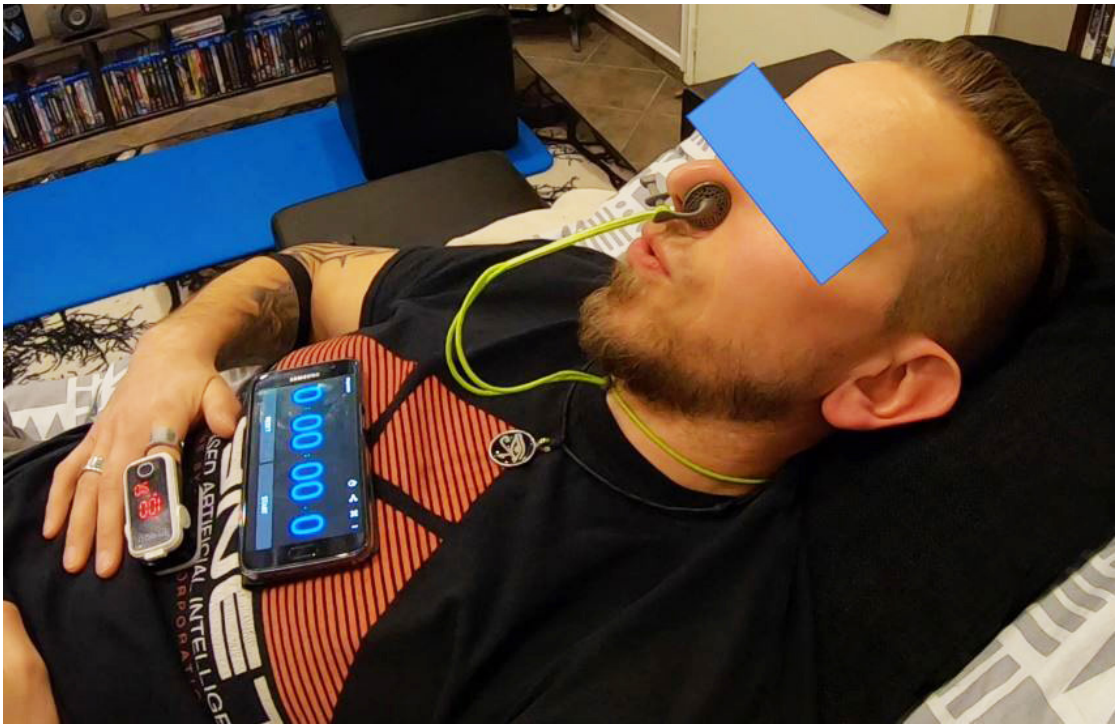
Residual volume (RV) apnoea is the performance of a breath-hold after a fully-controlled forced exhalation, creating inflexion of the diaphragm. In addition, some divers practice glossopharyngeal exsufflation to further reduce pulmonary lung volume to 200–300 ml below RV.[7] This manoeuvre is used to practice flexibility of the ribcage and diaphragm, as well as to increase the rate of induction of hypoxaemia when pulmonary oxygen stores are limited. Some breath-hold divers can hold their breath for 2–3 minutes on empty lungs without loss of consciousness. In addition, a decrease of the intra-alveolar gas pressure down to -90 cm H_2O has been reported.[7] This will decrease the gas diffusion gradient via reduction of P_AO_2 by 1–2 kPa, but will also create a sub-atmospheric intrathoracic pressure that will increase fluid transfer, shifting blood stores centrally and possibly creating pulmonary oedema.

A case is described which demonstrates these physiological phenomena and risk of this practice, hoping to raise awareness of the risks of unsupervised dry apnoea training at pulmonary RV.

Case report

The diver gave written informed consent for publication of this report. A male freediver, 181 cm, 43-years-old,

Figure 1
The diver in a in a semi-reclined (45 degree) position for dry apnoea training with nose clip and pulse oximeter. Picture is a screen shot from the diver's video recording



healthy, non-smoker, no medications, suffered an incident of prolonged unconsciousness and subsequent hospitalisation. He reports never having experienced collapse or syncope before this incident. He had videoed (GoPro Hero 7 Black, China) his training session and was wearing a pulse oximeter (Prego, model PM009, Shenzhen Aeon Technology, Shenzhen, China).

His dry apnoea routine consisted of vigorous hyperventilation (2 s inspiration, 2 s forced exhalation, for approximately 2 min), followed by forced exhalation to RV prior to breath-holding using a nose-clip (Figure 1). The video documented his prolonged unconsciousness (over 3 minutes) with an apneustic breathing pattern and prolonged episodes of recurrent apnoea.

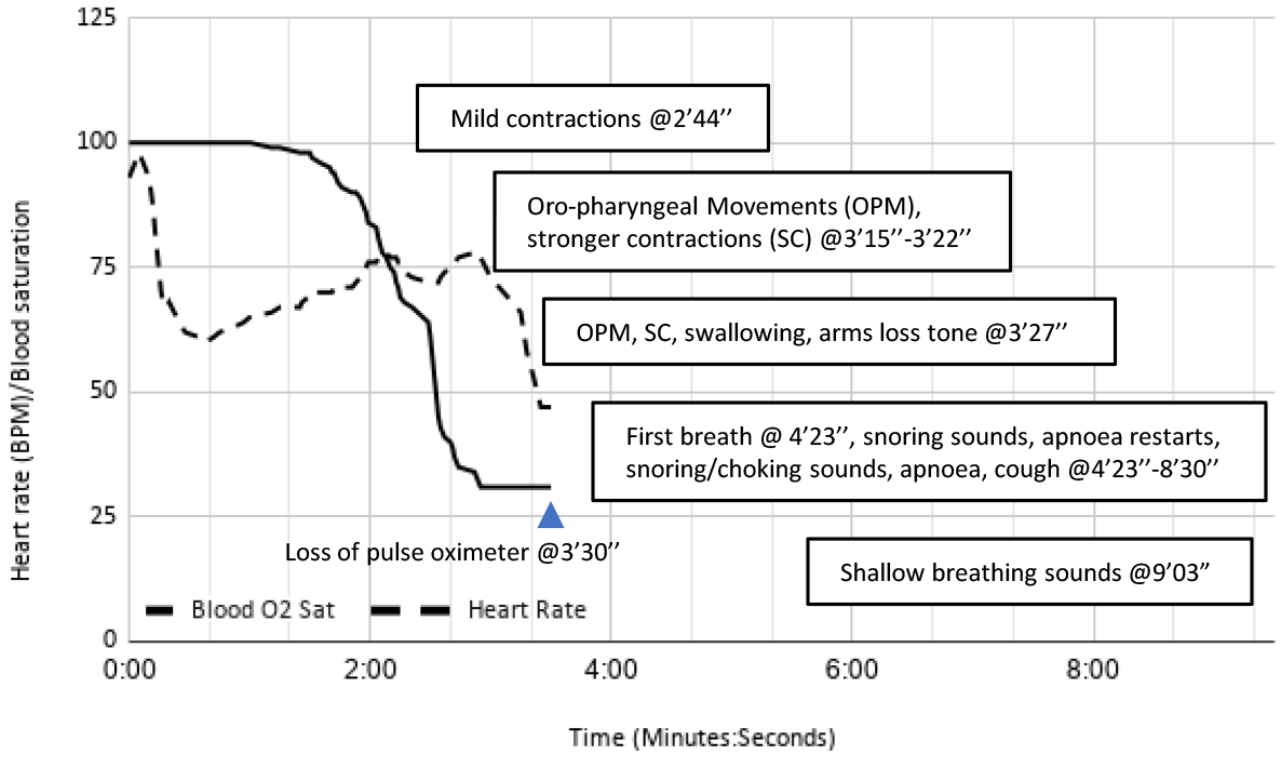
SYNCOPE EPISODE

The course of events is described in Figure 2. Pulse oximetry measurements made from the finger may not represent central hypoxia, and equipment is rarely calibrated below 50%.[8] The diver's saturation declined steadily to 35%, at which point his first mild diaphragmatic contractions were observed, at 2 min 44 s. Thereafter, saturation dropped to 31% and the diver displayed swallowing movements and stronger contractions. An evident change in mental status was seen at 3 min 30 s, with clinical presentation of a right-sided conjugate gaze to the horizon, a subtle loss of motor control of the arms, bilateral upper extremity extension, and right-hand thumb opposition. This caused the pulse oximeter to fall out of sight. At 3 min 43 s there was bilateral upper extremity decerebrate posture in extension, along with sustained head extension, and the gaze continued to be right sided only. At 4 min, the video tilted with only audio remaining; the audio track suggests that breathing was resumed at 4 min 23 s. Thereafter, snoring and choking sounds were evident (resembling possible upper airway obstruction or upper airway muscle discoordination). Coughing and apnoea restarted several times to 8 min 30 s. At 9 min 03 s, shallow breathing could be heard, at close to tidal volume breaths.

Heart rate was 98 beats per minute (bpm) when breath-holding started. It declined to 60 bpm within 30s and thereafter fluctuated between 60–78 bpm. When the contractions started at 3 min, there was a rapid decline to 47 bpm.

The diver estimated that he resumed consciousness 15 min after the episode. He was sweaty and nauseous with a strong headache. He then tried a breathing manoeuvre that creates breathing with positive end expiratory pressure (PEEP), however, oxygen saturation did not rise higher than 90%. After an hour, the diver called an ambulance and was taken to hospital. There he was imaged with a chest X-ray, followed by computed tomography (CT) of the chest, and blood tests. There were no saved reports regarding blood pressure or other abnormal findings on physical examination. He desaturated to 85% during the night. His inflammatory

Figure 2
Chart depicting the timeline for oxygen saturation (%) and heart rate (beats per minute) during the apnoea training. The appearance of various symptoms is shown at the time points noted



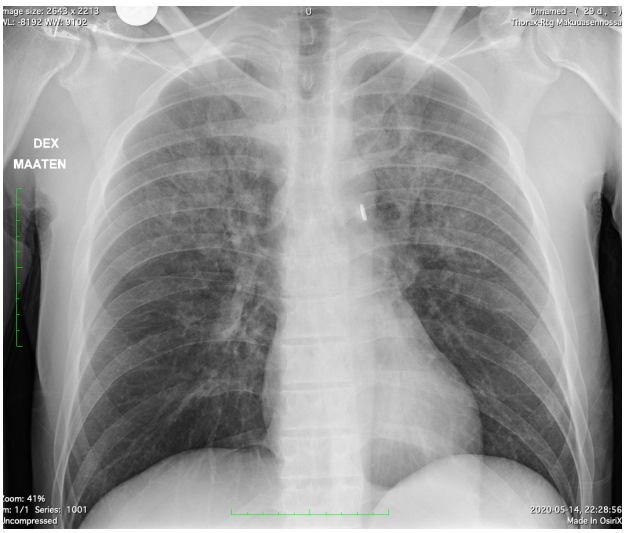
markers were normal, as well as troponin T (TnT), but pro-brain natriuretic peptide (NT pro-BNP) was slightly elevated (158 ng·L^{-1} versus normal value < 84 ng·L^{-1}). He was negative for COVID-19 and cardiac echocardiography was normal. The patient was hospitalised for three days (with supplemental O_2 the first evening and night), and had a checkup five weeks later (including chest CT), with no abnormal findings.

RADIOGRAPHIC FINDINGS

A chest X-ray was taken five hours after the incident (Figure 3). It showed bilateral opacities in the upper half of the lungs, with some subpleural sparing. The opacities had a nodular (or airspace) distribution with very minimal interstitial oedema. Below the hilar regions there were no findings.

The CT pulmonary angiography was performed the following day (Figures 4–6). It showed bilateral ground glass opacities in a pattern following the boundaries of the secondary lobule.[9] Opacities were mainly distributed in the upper lobes, with some in the upper parts of the lower lobes and intralobular gradient as evidence of settling of fluid in a gravity-dependent manner.[10] There were also some peribronchial opacities in the right lower lobes (upper medial part), (paraspinal) suggesting oedema or bleeding. The bronchi were patent, with no findings suggestive of aspiration.

Figure 3
Chest X-ray showing bilateral opacities as pulmonary oedema with a clear distribution in the upper parts of the lungs (the upper half of the lungs are whiter than the lower parts, which is an abnormal finding)



Discussion

To our knowledge this is the first report of dry apnoea training causing pulmonary oedema requiring hospitalisation. As this training method is used commonly among freedivers, raising awareness of its potential adverse effects would be of benefit.

Figure 4
Axial image of upper part of the lungs showing oedema in a distribution following the secondary lobule; Four white arrows point to examples of the 1–2 cm structure that is opacified by oedema

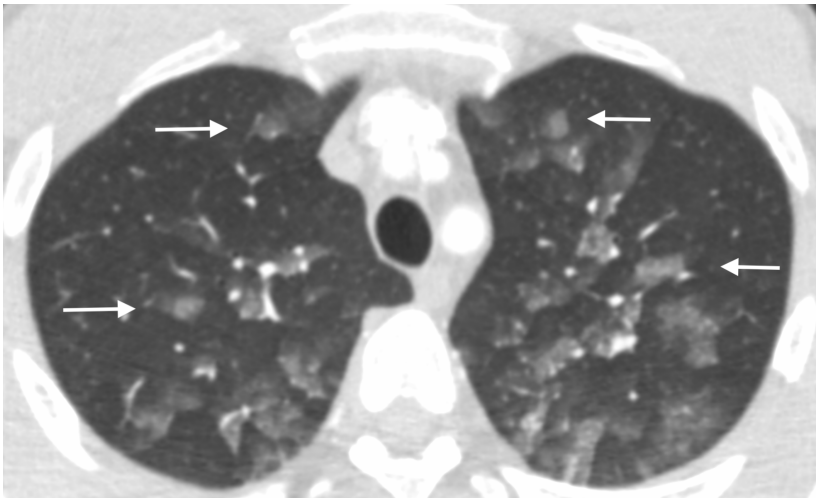
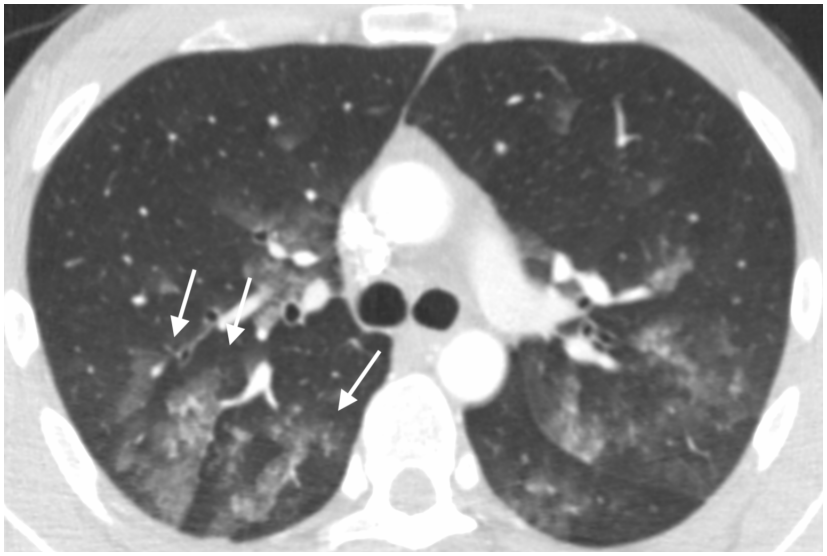


Figure 5
Axial image of middle part of the lungs with three white arrows pointing in the direction of the radial distribution or streak appearance of the edematous lung suggesting some parts where spared, possibly due to partial lung collapses

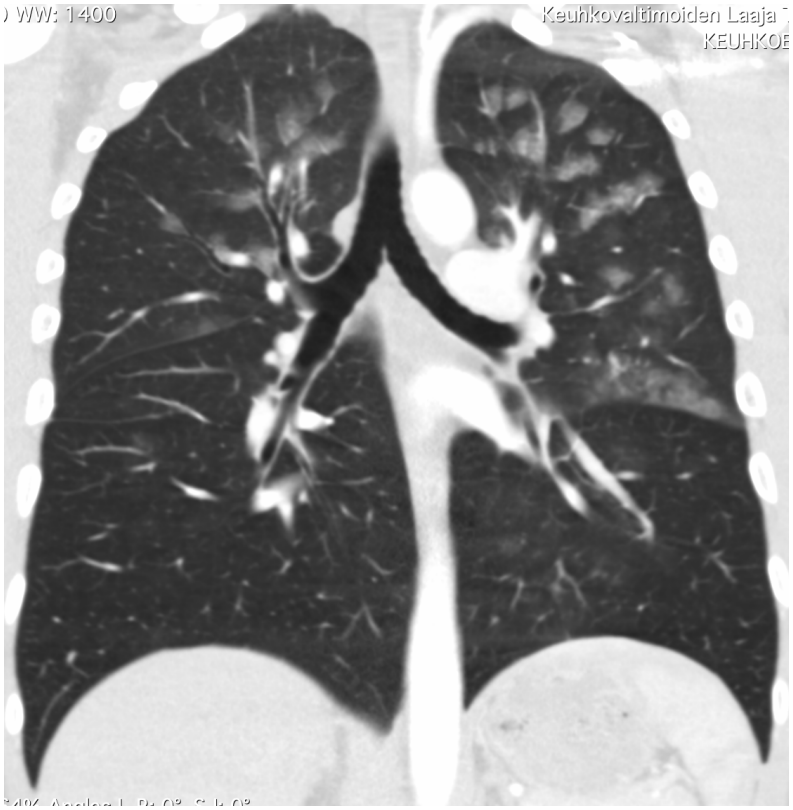


Several practices for apnoea training involve total lung capacity ('full lungs'), RV ('empty lungs or forced exhale'), or a degree in between (referred to as functional residual capacity or passive exhale). Some practices carry higher risk for cerebral hypoxia than others. As the lungs are the major oxygen store, it would be expected that the duration of breath-holding needed to reach hypoxia can be modulated by the amount of inspired air. Hyperventilation is used to delay dyspnoea caused by hypercapnia. This diver used a low lung volume to limit oxygen stores, decrease the alveolar-capillary pressure gradient (negative pressure), and the Bohr effect (hypocapnia) to attain hypoxia.

The practice of RV dry apnoea made during a stretching routine, familiarises the practitioner with the sensation of collapsed lungs and contractions when the diaphragm is introflexed, and simulates some of the sensations perceived at depth when lung volume is compressed. It is also practiced with the notion that it increases the flexibility of the diaphragm and thus the ability to 'collapse' the thoracic cage.

This diver presented with hypoxaemia, and imaging revealed alveolar pulmonary oedema, which could be multifactorial but sub-atmospheric alveolar pressure should be considered primarily. A residual volume breath-hold, with contractions of the diaphragm against a closed mouth (and/or glottis)

Figure 6
Coronal image of the middle part of the lungs showing gravity dependent consolidation within each opacity and the upper lung distribution



will create a relative negative pressure within the alveoli. Many humans also react to dry breath-holding with central hypertension (systemic vasoconstriction as part of the diving response), increasing pulmonary blood volume ('blood shift') and pressure gradient for capillary-alveolar transudation.[11] This would be analogous to the mechanism for a 'negative pressure oedema' observed, for example, in tracheal obstruction.[12]

The prolonged syncope also offers the possibility that severe hypoxia (for minutes) would limit the left ventricular cardiac output causing heart failure, and thus pulmonary venous stasis might cause high pulmonary capillary pressure. Pulmonary oedema is not uncommon in myocardial dysfunction. In this case, the diver made an attempt to breathe but may have been impeded by: upper airway dysfunction related to cerebral hypoxia; upper airway obstruction related to the use of nose clip; and the high muscle effort needed to ventilate from a starting point of RV with partial atelectasis. The prolonged state of cerebral hypoxia was also a consequence of his inefficient recovery breathing.

Prolonged hypoxia could also cause hypoxic pulmonary vasoconstriction in the lungs similar to high altitude pulmonary oedema, although this mechanism seems less likely given the specific distribution of the oedema.

The diver had a predominantly upper lung oedema distribution in comparison to the more common butterfly-wing shape in cardiogenic oedema. This was interpreted as an effect of the diver holding his breath at residual volume with the lower lobes collapsed in partial atelectasis,[13] thus protecting the tissue from transudation (less capillary-alveolar pressure difference if the alveoli are collapsed). It is noteworthy that the oedema showed streaks in the upper lobes, possibly due to radial or subsegmental atelectasis. It is highly likely that the nose clip worsened the diver's ability to resume breathing after syncope. Spasms are a common symptom and the muscles closing the mouth (e.g., masseter) would be stronger than the opposing ones.

In conclusion, we would advise against self-practice/unsupervised dry breath-holds for hypoxic conditioning with a nose clip. It cannot be said for certain that the diver would have resumed spontaneous breathing without the nose clip, so preferably all such dry exercises should be observed or supervised.

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