Immersion pulmonary oedema in a healthy diver not exposed to cold or strenuous exercise

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Abstract

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In healthy divers, the occurrence of immersion pulmonary oedema (IPE) is commonly caused by contributory factors including strenuous exercise, cold water and negative-pressure breathing. Contrary to this established paradigm, this case reports on a 26-year-old, well-trained combat swimmer who succumbed to acute IPE during static immersion in temperate (21°C) water, while using a front-mounted counterlung rebreather. The incident occurred during repeated depth-controlled ascent practice at the French military diving school. It was discovered that the diver had attempted to stop any gas leakage into the system by over-tightening the automatic diluent valve (ADV) (25th notch of 27) during the dive, thus causing a high resistance to inspiratory flow. The ventilatory constraints imposed by this ADV setting were assessed as a 3.2 Joules·L^{-1} inspiratory work of breathing and -5 kPa (-50 mbar) transpulmonary pressure. This report confirms the key role of negative pressure breathing in the development of interstitial pulmonary oedema. Such a breathing pattern can cause a lowering of thoracic, airway and interstitial lung pressure, leading to high capillary pressure during each inspiration. Repetition of the diving drills resulted in an accumulation of interstitial lung water extravasation, causing pathological decompensation and proven symptoms.

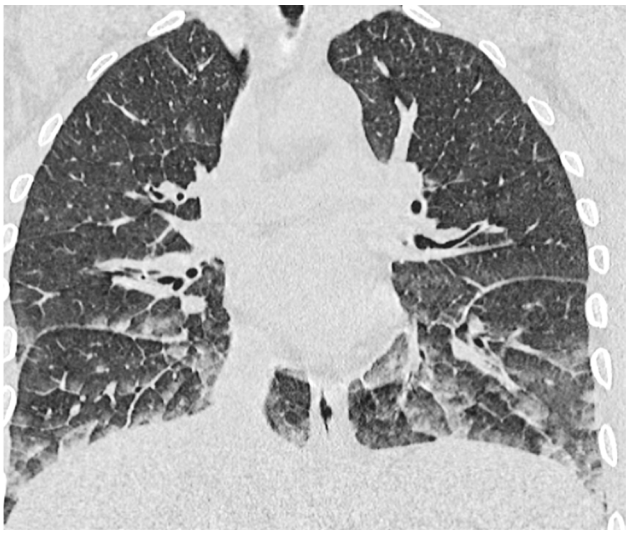
Introduction

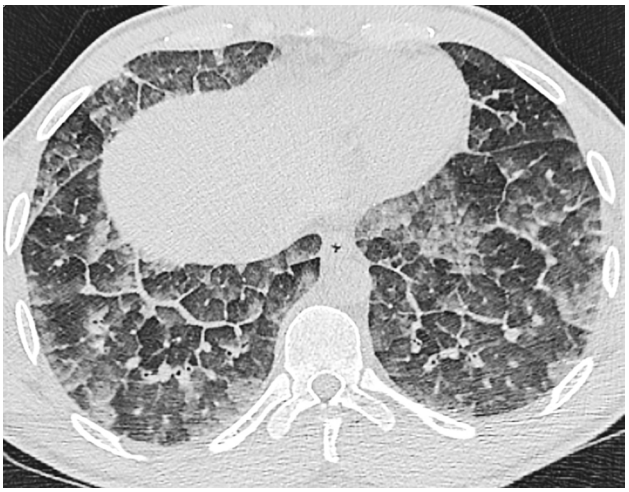
Immersion pulmonary oedema (IPE) is reported during sustained swimming, for example during triathlon competitions, and also during dives that involve exercise, particularly in cold water.[1,2] For example, we have observed that thirty minutes of moderate scuba exercise could lead to extravascular lung water accumulation, which correlated with increases in inferior vena cava diameter, systolic pulmonary artery pressure and an increased right/left ventricle ratio.[2] We have also observed the key role of negative-pressure breathing (using back-mounted counterlung rebreather equipment) as a contributory factor to IPE.[3,4] Contrary to this well documented paradigm, a young, well-trained military diver developed severe IPE during static immersion, while using a front-mounted counterlung rebreather in temperate (21°C) shallow water (maximum depth 7 metres' sea water (msw).

Case report

A physically fit, well-trained, 26-year-old male (height 1.89 m, weight 85 kg, and 4.02 L·min^{-1} maximum oxygen uptake) undergoing combat swimmer training was diving using a front-mounted counterlung closed circuit rebreather (FROGS, Aqualung™, Carros, France) to practice depth-controlled ascent at sea. The drill entailed a return from 7 msw to surface within 10 min, without releasing any gas in the water (no bubbles). This trial had to be repeated five times within one hour. At the end of the fourth attempt, the subject experienced breathing discomfort and coughing, but persisted to the end of that repetition. As soon as the counterlung bag was refilled and he resumed the prone position, the pulmonary symptoms vanished. At the beginning of the fifth repeat, he started to cough uncontrollably and experienced pronounced dyspnea. On admission to the medical department of the diving school, he presented with dyspnoea, was coughing frothy sputum, had chest tightness and bilateral rales. Pulse oximetry

Figure 1
Pulmonary CT scan performed one hour after emersion; left – coronal plane, right – axial plane; typical patchy "*frosted glass*" zones are observed, often adjacent to highly contrasted interlobular walls and peribronchial bundles and predominantly in the gravity-dependent basal regions of the lungs





was 93% breathing ambient air. He was transferred to the hyperbaric department of the military hospital while breathing high-flow 100% oxygen (non-rebreathing mask, 15 L·min^{-1}). Ultrasound lung comet tails were observed in basal lung regions. A chest CT scan performed one hour after emersion showed frosted-glass areas adjacent to highly contrasted interlobular walls, predominantly in the basal areas (Figure 1). Two hours after his arrival in hospital, all clinical pulmonary symptoms had disappeared, but bilateral rales remained for five hours. The chest X-ray examination was not repeated.

Technical aspects of the dive drill

The counterlung bag of the rebreather device was worn ventrally while lying prone, i.e., establishing a slight transpulmonary positive pressure, in line with guidelines and therefore helping to prevent the development of IPE.[3,4,5] The return from 7 msw to surface was performed in the upright position. A significant increase in buoyancy due to gas dilatation in the counterlung bag would require a compensatory expiratory gas release. To achieve the controlled ascent without releasing any gas bubbles, the divers reduce their ascent speed, which in turn allows time for further consumption of oxygen from the bag. They simultaneously reduce their tidal volume to abate buoyancy. Further, to facilitate the drills, some trainees tighten the automatic diluent valve (ADV) to reduce the chance of diluent gas leaking into the system. An over-tightening of the ADV can cause the counterlung bag to empty, thus requiring a very pronounced inspiratory effort. The diver can refill the breathing bag (according to Boyle's Law) by ascending 20–40 cm in the water column, or thereabouts.

Analysis of the breathing load faced by the dive

Three days after the incident and once the diver was recovered, the diver agreed to be involved in an evaluation study. All experimental procedures were conducted in line with the Declaration of Helsinki and the study protocol was included in a larger pool of studies approved by the local Ethics Committee (Comité de Protection des Personnes-CPP Sud Méditerranée V, ref 16.077). Informed consent was obtained from the diver before submission.

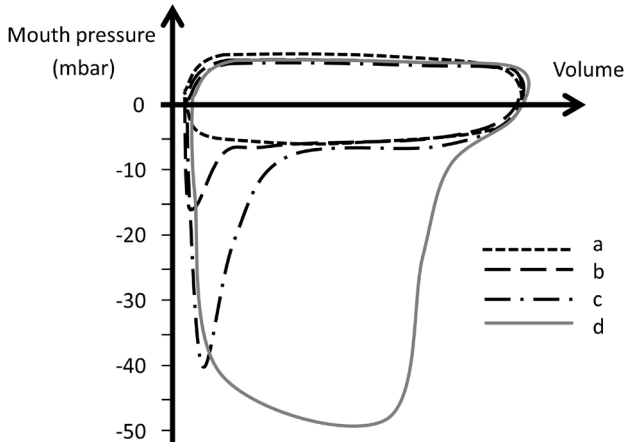
The diver was asked to reproduce the pattern of practice drills in the laboratory test pool. These trials were performed in 21°C water while wearing the same neoprene wetsuit as during the test at sea. It was observed that the subject tightened the ADV to a high degree (25th notch of 27). We then used a bespoke electronic pneumo-baro-tachograph to assess the breathing pressure at the mouth and the breathing flow across tidal cycles.[4] Assessing the tidal pressure cycle at the mouth allows calculation of the work of breathing (WOB, joules) from the area of the pressure vs tidal volume ($V_{t)}$ loop. The WOB/V_t defines the pressure required to perform one unit (L) tidal volume as suggested by Warkander et al.[5]

ADV ASSESSMENT

The setting of the ADV was assessed first, according to four conditions (Figure 2):

- breathing from a bag full enough so that the ADV was not activated;
- bag empty at the end of inspiration and the ADV at its loosest setting;
- bag empty at the end of inspiration and the ADV at its tightest setting;
- performing a deep inspiration with the ADV at its tightest.

Figure 2
Schematic representation of the 'pressure-volume' loops measured in four conditions whilst a prone diver used a front-mounted counterlung closed circuit rebreather at 1 m depth: a. breathing from a bag full enough so that the ADV was not activated; b. bag empty at the end of inspiration and the ADV at its loosest setting; c. bag empty at the end of inspiration and the ADV at its tightest setting; d. performing a deep inspiration with the ADV at its tightest



When inspiring from a full bag (a) there was no peak inspiratory pressure. The inspiratory pressure amounted to -0.5 kPa (-5 mbar) and WOB to 0.5 $J \cdot L^{-1}$.
When the bag was empty and the valve was set at its loosest (b), the pressure achieved to open the regulator was -1.5 kPa (-15 mbar) and the WOB was 0.72 $J \cdot L^{-1}$.
When the bag was empty and the valve was set at its tightest (c), the pressure achieved to open the regulator was -4.5 kPa (-45 mbar) and the WOB was 1.04 $J \cdot L^{-1}$.
When the inspired volume was high and the valve at its tightest (d), the low pressure lasted throughout the inspiration, at its peak reaching -5 kPa (-50 mbar) while the WOB was 3.2 $J \cdot L^{-1}$.

BODY POSITION

Secondly, the effect of body position on breathing load, i.e., the hydrostatic transpulmonary imbalance, was assessed. The hydrostatic transpulmonary pressure difference is given by the vertical distance between the counterlung bag centroid and the diver's airways centroid. As the FROGS device is worn ventrally, the bag centroid is lower than lungs during prone finning, resulting in a higher hydrostatic pressure surrounding the bag than the airways, hence a positive-pressure breathing condition. Conversely, when the diver stands upright (as during ascent towards thesurface), the bag centroid is somewhat higher than the airways centroid, which gives a negative-pressure breathing setting (Figure 3).

In a prone position, the 0.6 kPa (6 mbar) higher pressure in the counterlung bag as opposed to that in the diver's airways, provides a 0.6 kPa (+6 mbar) inspiratory aid and the breathing effort takes place during expiration. Conversely,

Figure 3
Schematic representation of a diver using a front-mounted counterlung closed circuit rebreather in two positions: left – upright position during a depth-controlled ascent, static lung load -8 mbar (-0.8 kPa); right – prone position during fin swimming, static lung load +6 mbar (-0.6 kPa)

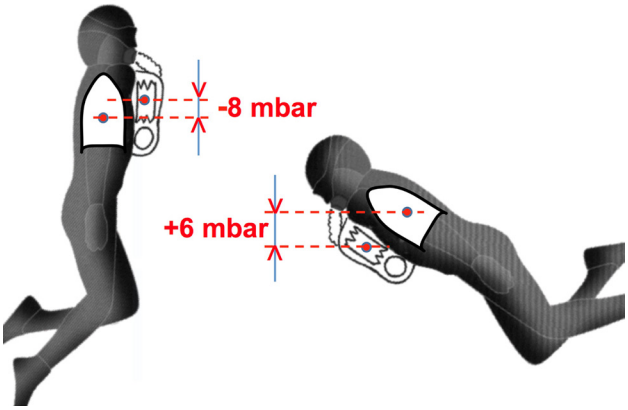
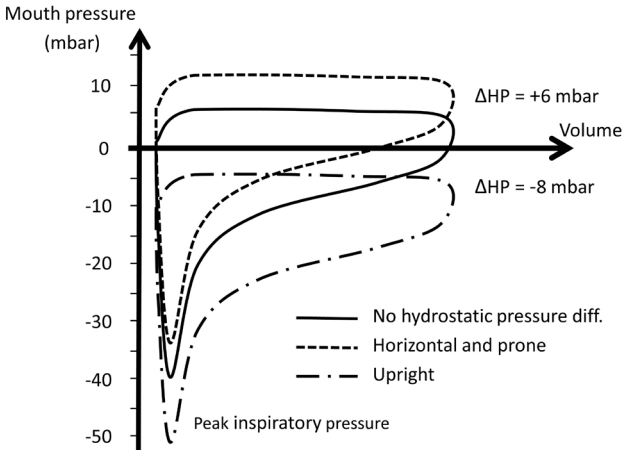


Figure 4
Schematic representation of the 'pressure-volume' loops through tidal recordings with three different transthoracic pressure conditions (mouth pressure and flow assessed with the diver used a front-mounted counterlung closed circuit rebreather at 1 m depth with the automatic diluent valve (ADV) tightened): without any hydrostatic transpulmonary imbalance (HI) (solid line); while prone (fin swimming position) i.e., positive imbalance (dotted line); while upright i.e., negative imbalance (dot and dash line)



the upright posture assumed during ascent towards the surface switches a hydrostatic transpulmonary pressure imbalance to 0.8 kPa (-8 mbar), which alleviates the work of expiration, but prompts an increased inspiratory effort (negative-pressure breathing).

If the diver breathes without over-tightening the ADV, whatever the posture, the hydrostatic pressure difference between the bag and the airways remains low, as does the

breathing load. However, upon tightening the ADV, even to a low level, the inspiratory pressure rapidly becomes more negative (Figure 4). Thus, the hydrostatic pressure difference (HD) adds its own load to the tightening of the ADV (Figure 3): when HD is null the peak inspiratory pressure is about -4 kPa (-40 mbar); when the diver is prone, peak inspiratory pressure is somewhat alleviated to -4 + 0.6 = -3.4 kPa (-34 mbar); when the diver is upright, HD amounts to -0.8 kPa (-8 mbar) and peak inspiratory pressure reaches -4.8 kPa (-48 mbar). In other words, a -1.4 kPa (-14 mbar) lower intrathoracic pressure is required whilst upright to open the ADV (hence a higher inspiratory effort).

To summarise, with this front-mounted counterlung rebreather, the breathing work during inspiration is minimal when the diver lies prone and the counterlung bag is full, thus the diver does not need to activate the ADV. The inspiratory breathing load is increased:
i) in the upright posture;
ii) when the ADV has to be activated, and
iii) even more so when this valve has been tightened.

General discussion

Contrary to most descriptions to date, this case of IPE did not involve substantial physical effort, but rather a prolonged struggle (repetitive over several 10-minute ascents) against a high resistance to inspiratory flow. The condition occurred even though other known IPE contributory factors were not present: the sea water was temperate at 21°C; the diver wore a well-fitting 7 mm neoprene wetsuit with boots and gloves; there was no substantial finning effort and the front-mounted counterlung rebreather offered slight positive pressure breathing in the prone position, so lowering inspiratory effort.

However, when the subject decided to tighten his ADV to avoid positive buoyancy, he created a major inspiratory load. Each tidal inspiration then required a markedly larger effort, achieved through substantial lowering of thoracic, airway and mouth pressure. Upon ascent, assumption of the upright position created a further slight inspiratory load in addition to the problem of the over-tightened ADV. Preservation of such a breathing pattern for several minutes during repeated ascents from 7 msw likely led to substantial blood accumulation in the lungs and to the development of high lung capillary pressure, while thoracic (i.e., airway and interstitial lung) pressures were considerably lowered during each inspiration. The resulting interstitial lung water extravasation, accumulated over five repetitions of the diving drill, then led to the development of pathological decompensation and onset of symptoms. Indeed, the CT-scan showed a frosted-glass appearance, reflecting alveolar flooding. They also displayed highly contrasted interlobular and peribronchial areas, revealing the substantial fluid accumulation in the interstitial bronchial bundles and junctional spaces that is consistent with the established sequence of lung fluid accumulation.[6] The predominance of interstitial oedema in the basal (gravity-dependent) regions of the lungs was also in line with the upright posture during which the inspiratory efforts were completed.

Achieving markedly negative intrathoracic pressure (NIP) has been recognized to rapidly promote non-cardiogenic pulmonary oedema on land under normobaric conditions.[7] Healthy subjects can produce a large NIP that results in reduced left ventricular stroke volume concomitant with substantially increased blood flow in the superior vena cava, while the increased abdominal pressure likely shifts blood from the splanchnic veins into the inferior vena cava.[8,9] Thus, during these high inspiratory efforts, the right heart preload increases while left heart ejection is hindered. The resulting right to left heart imbalance leads to rapid accumulation of blood volume in the lung vessels and a concomitant rise in pulmonary capillary pressure; this combined with a high NIP creates a high transcapillary hydrostatic pressure that promotes extravascular lung water accumulation.[10] Immersion bolsters this chain of events, via an inescapable augmented right heart preload and congestion of the pulmonary circulation, even while at rest.[4,11,12] Finally, it should be noted that a neoprene wetsuit will also add its own weight to all inspiratory efforts, as it restricts chest wall and abdominal expansion over and above the hydrostatic pressure.[13,14]

It has been surmised that intense diaphragmatic contractions contribute to the observation of lung comet tails after dynamic surface apnea. Indeed, involuntary diaphragmatic spasms develop during the 'struggle phase' of breath holding.[15] Besides the increased density of gases at depth, the hydrostatic imbalance due to the relative positions of the breathing device and the airways has been considered an important determinant of breath loading of the WOB, especially if the diver must cope with the imbalance over a prolonged period.[3,5,11,12] However, sustained physical effort will be more important than position in the development of IPE, and will exacerbate the effect imparted by the latter.[4] In the present case, all of these factors were made redundant by the action of over-tightening the gas ADV, thus increasing the inspiratory load so significantly that IPE was induced.

Conclusion

Cold exposure and sustained effort were first identified as contributory factors to the occurrence of immersion pulmonary oedema.[16–19] Hydrostatic lung load was linked to the WOB during a dive. In this young, healthy, highly trained diver, the inspiratory WOB was a major trigger of IPE, aside from any upright hydrostatic lung loading and in the absence of an enlarged ventilatory requirement during exercising, as the case developed following static immersion. It is essential when considering IPE that every possible cause of inspiratory effort should be examined and guarded for in order to prevent its occurrence.

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