A neoprene vest hastens dyspnoea and leg fatigue during exercise testing: entangled breathing and cardiac hindrance?

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Abstract

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Symptoms and contributing factors of immersion pulmonary oedema (IPO) are not observed during non-immersed heart and lung function assessments. We report a case in which intense snorkelling led to IPO, which was subsequently investigated by duplicating cardiopulmonary exercise testing with (neoprene vest test – NVT) and without (standard test – ST) the wearing of a neoprene vest. The two trials utilised the same incremental cycling exercise protocol. The vest hastened the occurrence and intensity of dyspnoea and leg fatigue (Borg scales) and led to an earlier interruption of effort. Minute ventilation and breathing frequency rose faster in the NVT, while systolic blood pressure and pulse pressure were lower than in the ST. These observations suggest that restrictive loading of inspiratory work caused a faster rise of intensity and unpleasant sensations while possibly promoting pulmonary congestion, heart filling impairment and lowering blood flow to the exercising muscles. The subject reported sensations close to those of the immersed event in the NVT. These observations may indicate that increased external inspiratory loading imposed by a tight vest during immersion could contribute to pathophysiological events.

Introduction

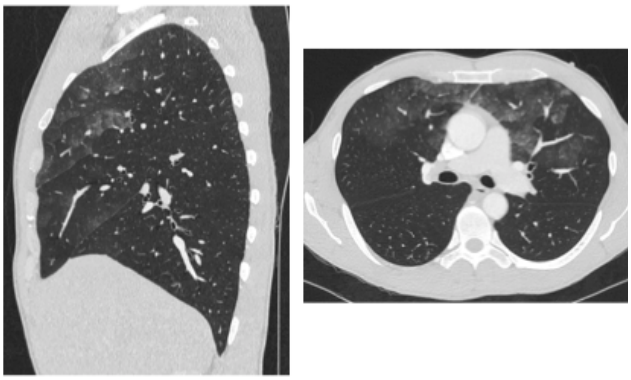
Immersion pulmonary oedema (IPO) can be life-threatening.[1] It is more likely if cardiovascular function is impaired,[2,3,4] but it can occur in fit subjects during immersed exercise.[5–7] However, to date no non-immersed investigation reproduces symptoms and physiological features of an immersed event. Yet recognising individual thresholds of functional tolerance should provide clues to tailor safer patterns of immersed activity. A contribution of inspiratory effort to IPO was evidenced either during exercise or when coping with the breathing loads coming with immersed activities.[8,9] Here we report how subjective sensations and potential physiological features of a strenuous swimming-induced IPO event were elicited during tailored cardiopulmonary testing.

Case report

A 44-year-old fit male fireman was referred for maximal exercise testing after enduring a swimming-induced episode of IPO. The subject was a regularly trained triathlon competitor, with a two pack-year current smoking history and no known cardiovascular disease. The event occurred during rescue training (12°C lake water) with snorkel-swimming over 1 km, followed by 50 m swimming and 3 m depth diving to rescue a dummy 'victim'. The subject used a 5 mm whole body neoprene suit, a mask and a 20 mm lumen snorkel. The 1 km distance was accomplished in 13 min 45 s (< 20 min expected) and after 5 min rest the subject swam to the dummy but was unable to dive and grab it, feeling out of breath. His legs could not continue finning, and he was taken back to shore by a buddy with severe dyspnoea, cough and bloody sputum.

After 30 min of oxygen mask breathing (12 $L \cdot min^{-1}$) the respiratory symptoms had disappeared and the subject went home. Two hours later he attended the emergency department. At examination no signs of heart failure or leg thrombophlebitis were found. Lung wheezes were heard bilaterally. Vital signs were normal. ECG, transthoracic echocardiography and computed tomography (CT) pulmonary angiography were performed, together with blood analysis of B-type natriuretic peptide, D-dimers and troponin. The CT showed anterior ground glass changes in the upper lobes and fine thickening of interlobular septa reflecting venous congestion (Figure 1).

Figure 1
Sagittal and transverse tomodensitometric computed tomography scans taken two hours after the IPO occurrence



IPO was diagnosed. One week later, chest X-ray and pulmonary function tests were normal (FVC = 5.8 L, FEV1 = 4.4 L, FEF50% = 4.79 $L \cdot s^{-1}$).

Two months later, a standard bicycle ergometer maximal exercise test was performed (standard test – ST). The subject wore shorts and T-shirt, and after a 3 min warm-up at 50 W, workload increased by 25 W every minute. Measurements during the test were as previously described.[10] The maximum oxygen consumption (VO_2max) was 57.1 $ml \cdot min^{-1} \cdot kg^{-1}$ (163 % predicted) and maximum of work rate was 400 W. Resting blood pressure was high (169/109 mm Hg), but the blood pressure profile during maximal exercising was normal. A 24 h ambulatory recording of blood pressure confirmed hypertension.

After one month, the exercise test was repeated with exactly the same protocol but included the wearing of a 5 mm thick neoprene vest (neoprene vest test – NVT). During this trial, both dyspnoea intensity and lower limb fatigue (Borg scales) rose earlier and were significantly higher at any given work rate than during the first test (Figure 2). The NVT ended earlier, at 350 W and 55.2 $ml \cdot min^{-1} \cdot kg^{-1}$ VO_2max, with severe breathlessness. Minute ventilation and breathing frequency rose faster during the NVT than ST (Figure 3), but tidal volume was similar at each level in the two tests (max 3.1 L in ST and 3.2 L in the NVT). The patient appraised his sensations as equivalent to those experienced during the immersed event with a feeling of "*occurring death*" before bloody spitting. No significant dynamic hyperinflation, and no mechanical ventilatory limitation were seen. Heart rate and diastolic blood pressure rose earlier than during the ST. Systolic arterial pressure and pulse pressure plateaued earlier and remained lower during the NVT than the ST (Figure 4). In the ear lobe arterialised capillary blood PaO_2 and SaO_2 were not decreased during maximum exercise in either the ST and NVT. The subject described the intensity and unpleasantness of dyspnoea during the NVT as similar to those during the field event.

Figure 2
Dyspnea and leg fatigue scores (Borg scales) during the two cardiopulmonary exercise tests; NVT – neoprene vest test; ST – standard test; W – workload in watts

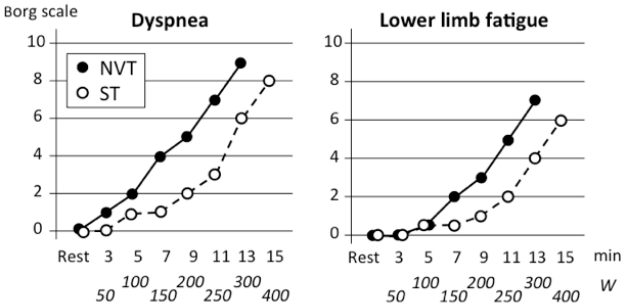


Figure 3
Minute ventilation and breathing frequency during the two cardiopulmonary exercise tests; NVT – neoprene vest test; ST – standard test

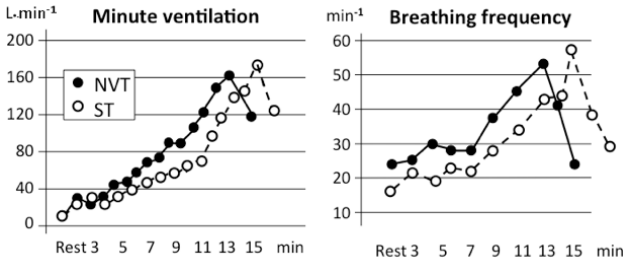
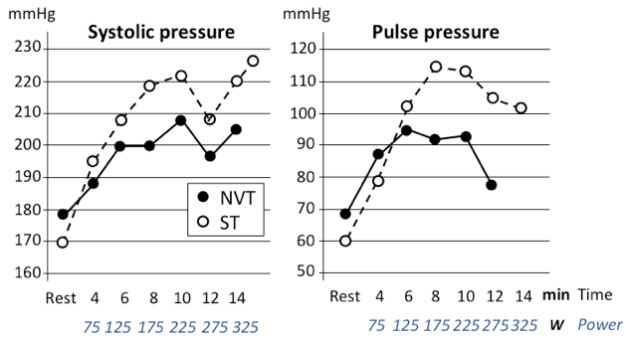


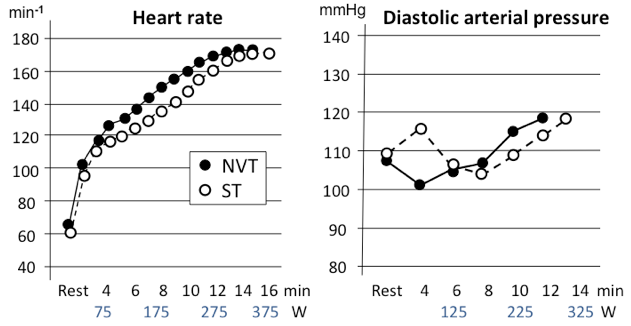
Figure 4
Systolic arterial pressure and pulse pressure during the two cardiopulmonary exercise tests; NVT – neoprene vest test; ST – standard test



Discussion

Completing the 1 km surface swim in less than 14 min i.e., much faster than required, required high-level exercise. The elastic restriction of the neoprene suit reinforced the effects of hydrostatic pressure[11] to reduce both total vascular capacity in the thorax and lung compliance,[12] as venous return, right heart preload and lung blood

Figure 5
Heart rate and diastolic blood pressure during the two cardiopulmonary exercise tests; NVT – neoprene vest test; ST – standard test



volume increased.[8,13] During immersion the neoprene suit also loaded lung mechanics by requiring supplementary inspiratory effort[14,15] and increasing the transpulmonary pressure load.[8] Finally, the snorkel enlarged the anatomical dead space, which also requires an increased ventilatory volume.[16] Together the loads of immersion, elastic suit and snorkel substantially increased inspiratory effort during the 14 min high intensity swim. Adding dead space and chest strapping cause severe dyspnoea and exercise intolerance in healthy subjects cycling on land.[16] The physiological strains and sensations in that study appear similar to those in IPO.

Most of the differences observed between the ST and NVT tests were likely explained by the "abnormal" restrictive effect of the neoprene vest.[17] One study concluded that with "abnormal" restrictive constraints on tidal expansion, the intensity and unpleasantness of dyspnoea reflect the awareness of increased neural respiratory drive needed to achieve any given level of ventilation during exercise.[17] A higher inspiratory power of breathing was required to achieve tidal volume at each exercise level during the NVT than the ST. Minute ventilation rose faster during the NVT in keeping with the previous results.[16] At high exercising levels, ventilatory muscles require increased blood flow, diverted from the working limb muscles.[18] Accordingly, during the NVT the earlier rises of heart rate and diastolic blood pressure at the highest workloads (Figure 5) likely conveyed the higher sympathetic activity instrumental in re-routing part of the cardiac output from the lower limbs towards respiratory muscles, leading to earlier leg fatigue.

The first systolic blood pressure value was 9 mmHg higher in the NVT than the ST (Figure 4). Higher anxiety at beginning of a second maximal exercise test or simple measurement variability[19] might explain this. However, we consider that despite baseline sympathetic possibly being higher during the NVT, the plateauing of systolic arterial pressure and pulse pressure to remain roughly 10 mmHg and 20 mmHg respectively lower than during the ST (Figure 4) could be consistent with restricted stroke volume during this second part of the NVT i.e., at the highest energy expenditure levels.[18] Immersed cumulative inspiratory effort was found to correlate with end-stage right heart preloads and right to left ventricle ratios,[8] thus paving the way for imbalance of ventricular outputs and consequently fluid extravasation into lung interstitium.[20] During the NVT the additionally loaded inspiration due to the restrictive effect of the vest elasticity may have caused lowering of pleural-mediastinal pressure thus increasing both right heart preload and left ventricle afterload while possibly decreasing the left ventricle preload through leftward interventricular septal displacement.[21,22] Such a restricted left ventricular preload would then impede the rise of stroke volume, consistent with reduced systolic and pulse pressures in the NVT. In addition, the larger sympathetic activation during the NVT would have added to left ventricular afterload and further impeded the stroke volume.

Immersion-linked conditions are instrumental in developing IPO.[23] IPO was first described during cold water diving.[24] Immersion in cold water causes peripheral vasoconstriction, lung congestion, increased pulmonary arterial pressure and increased left ventricle afterload.[25] Immersion may also increase airway resistance and breathing work.[26] In the present case the water temperature could have triggered peripheral vasoconstriction and contributed to increase central blood volume and right heart preload.[24,27] However, it is also possible that during the vigorous swimming, skin vasoconstriction was suppressed, given the combination of the large muscular heat production and the suit-hindered heat dispersal into water.[28] The higher work of breathing in water than air is described,[15,29,30] but neither the additional inspiratory load of wet suit, nor its haemodynamic burden have been reported to the best of our knowledge. A small effect of a wet suit on maximal expiratory flow has been described.[31]

The development of haemoptysis and pulmonary oedema during the field event suggest pulmonary capillary stress failure[32,33] suggestive of cardiopulmonary stresses greater than those of the NVT. Our interpretation of the haemodynamic change during the NVT, possibly suggesting increased congestion of right heart and pulmonary circulation with restrained stroke volume would mimic a loss of left ventricular diastolic compliance.[34,35] This also takes place during pulsus paradoxus as it develops during high inspiratory efforts as in acute asthma[36] and whose cardiac features root in normal breathing.[37] Pulsus paradoxus is defined by a more than 10 mmHg inspiratory lowering of pulse pressure linked to right ventricle overdistension and the simultaneously reduced left ventricular filling through parallel biventricular interdependence.[36] This pattern would likely support the feeling of suffocation featured in congestive heart failure at rest[38] and repeatedly reported during IPO. Therefore we submit that the duplicated cardiopulmonary exercise testing sheds some light on symptoms repeatedly reported during the development of IPO. The faster increase

in dyspnoea during the NVT than the ST reflected the required higher inspiratory effort. In turn, this might have prompted a parallel circulatory impairment indirectly evidenced by the restriction of systolic and pulse pressures at the highest levels of exercising power together with higher leg fatigue. In status asthmaticus, pulsus paradoxus and dyspnea are lowered when breathing load is lessened through replacing air by heliox inhalation.[39] Accordingly we hypothesise that, beside the greater inspiratory effort caused by the elastic vest, part of the additional dyspnoeic sensations in the second half of the NVT might have been linked to ventricular imbalance and restriction of left stroke volume.

IPO results from extravasation of plasma into interstitial airway spaces, leading to decreased airway luminal caliber and later to impaired pulmonary gas exchange. Ultimately this causes arterial hypoxaemia and alveolar flooding.[40,41] Arterial blood gases were not altered in either test, and DLCO was not assessed so that no direct evidence of altered lung gas exchange was obtained. The absence of hypoxaemia is nevertheless consistent with the gradual development of right to left ventricular imbalance[17,33] which would eventually result in impaired gas exchange. According to this pathophysiological paradigm various forms of reversible myocardial dysfunction may then follow during immersed activities.[2,3,42] The higher myocardial work and a raised sympathetic stimulation would also pave the way for myocardial ischaemic events.[43]

The occurrence of IPO has been suspected to predict the development of arterial hypertension,[24] and the reverse connection was described.[44] In the present case, the high blood pressures on first test day led to ambulatory monitoring, diagnosing moderate hypertension, and appropriate treatment was started after the second exercise test. Some degree of hypertension-linked left heart diastolic dysfunction and a higher left ventricular afterload bolstered by sympathetic activation at high exercising levels might have enhanced the delay of left ventricular emptying as compared to the right.[45,46]

In conclusion, these observations underscore the potential respiratory burden imposed by an elastic wet suit during substantial swimming effort. We suggest they may also shed some new light on the role of work of breathing in developing IPO through interlocked haemodynamic and respiratory alterations.

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