Scuba divers' pulmonary oedema. A review

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Key words
Immersion, scuba diving, pulmonary oedema, review article

Abstract

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The literature on scuba divers' pulmonary oedema (SDPE) is reviewed, especially in its relationship to other immersion-induced pulmonary oedemas. It is concluded that although the three forms induced by swimming, freediving and scuba diving have some features in common, there are significant differences in their demographics, causation and clinical management. The swimming-induced cases tend to be young and fit, but exposed to excessive exertion. The freedivers experience extreme breath-holding and barotraumatic influences. The scuba divers are an older group and may have pre-existing or occult cardiovascular disease. Although the first-aid treatments may be similar, subsequent investigations and preventative measures will differ considerably

Introduction

Scuba divers' pulmonary oedema (SDPE) was first reported in 1981.[1] It is usually described as an uncommon disorder, only some dozens of cases being documented. Comprehensive reviews have been prepared by Lundgren and Miller, Slade et al and more recently by Koehle et al.[2–4] SDPE presents clinically with fast and shallow respirations, dyspnoea, fatigue, cough, sometimes with blood-stained expectoration, and auscultatory signs of pulmonary oedema; cyanosis may be present. Investigations reveal impaired spirometry and reduced pulmonary compliance, hypoxaemia and characteristic radiological (plain X-ray or CT scan) abnormalities.

There are both similarities and differences between terrestrial and immersion pulmonary oedemas. With differing causes and natural histories, extrapolation from the terrestrial to the diving situation may not be appropriate. Several, possibly distinct, forms of immersion pulmonary oedema (IPE) have been described: in swimmers, free (breath-hold) divers and scuba divers. This review attempts to distinguish between these but focuses mainly on SDPE and refers to the others where an association has been claimed.

Terrestrial pulmonary oedema

Pulmonary oedema is the accumulation of fluid in the lungs. It may be either a transudate (from high capillary-to-alveolar pressure gradients), an inflammatory exudate with protein, red cells, etc (capillary damage), or lymph accumulation.[5] It is well described in a variety of disorders in the general medical literature and may develop from both cardiogenic and non-cardiogenic causes. Cardiogenic pulmonary oedema is seen in myocardial infarction, cardiomyopathies, myocarditis, arrhythmias, hypertension, cardiac tamponade and acute fluid overload, etc. Non-cardiogenic pulmonary oedema may develop from a direct injury to the lung parenchyma as in thoracic surgical procedures, infections, allergies, toxic inhalants, trauma and aspiration, or be secondary to acute airway obstruction and other causes of negative inspiratory pressures – negative-pressure pulmonary oedema (NPPE) – or to neurogenic mechanisms.[5,6] Exertional pulmonary oedema has been reported both in athletes (long-distance running, rugby players) and race horses, whilst high-altitude pulmonary oedema occurs in climbers.[7,8] There is evidence that increased expiratory pressures (e.g., continuous positive airway pressure, CPAP) may ameliorate the effects of pulmonary oedema.[9]

Immersion pulmonary oedema

IPE was reviewed recently in 60 cases from the literature.[4] In this review, there were 34 scuba divers, 18 swimmers and eight freedivers. A prior or subsequent history of this disorder was present in at least 13 cases. The symptoms included cough in 82%, dyspnoea in 80% and haemoptysis in 62%. Less common were weakness and confusion; chest pain was not a feature. Although physical examination was not well described, crackles (rales) and wheezing were noted in 25% and 10% respectively. Most had the diagnosis verified radiologically. The mean oxygen partial pressure was 66.2 (SD 17.4) mmHg (8.82 +/- 2.32 kPa) with a mean arterial oxygen saturation (S_aO_2) of 88.8% (SD 7.3). In the majority, symptoms resolved within five minutes to 24 hours, but two cases were fatal. In the swimmers (Special Forces combat swimmers), heavy exertion was incriminated. A relationship between pulmonary oedema and 'thoracic squeeze' (pulmonary barotrauma of descent) was noted in the freedivers. Most of the swimmers and freedivers affected were otherwise healthy. Increased age was observed in the scuba divers with pulmonary oedema. As there are considerable differences in the epidemiology, aquatic behaviour and physiological stressors in each of these three groups, they are now considered separately.

Swimming-induced pulmonary oedema (SIPE)

Reports of dyspnoea and pulmonary congestion during surface swimming have often been associated with extreme exertion in both cold and warm (>20^{O}C) waters.[4,10–13] In a strenuous swimming time trial, eight of 30 young men were affected within the first 45 minutes, with a water temperature of 23^{O}C.[13] Over-hydration may have contributed, as they had all consumed 5 L of water prior to the swim to counter anticipated dehydration. The swimmers wore only bathing suits and fins and two had repeated episodes without the provocation of such extreme exercise and over-hydration.

In an Israeli military swimming fitness programme in water temperature of 19.6 (SD 3.2)^{O}C, 70 cases of SIPE were documented, all with dyspnoea combined with, in the majority, a productive cough, haemoptysis and 'inspiratory crackles'.[12] Chest pain or wheezing was noted in less than 9%. The mean S_aO_2 on air was 88.4% (SD 6.8) whilst spirometry demonstrated a temporary restrictive pattern. The chest radiography was normal after 12–18 hours. SIPE recurred in about a quarter of the swimmers. Over-hydration was not noted in this group.

An investigation of the S_aO_2 and spirometry findings in 29 incidents from 21 of 35 young men exposed to strenuous swimming over a two-month period, revealed similar changes, with a fall in S_aO_2 from 99% to 91%.[14] Forced expiratory volume in 1 sec (FEV_1), forced vital capacity (FVC) and the FEV_1/FVC ratio were all significantly lower in the SIPE group. Interestingly the pre-incident FVC and mid-expiratory flows (FEF_{25-75}) were lower in the swimmers who developed SIPE, and thus may be predictive of this disorder.

Explanations for SIPE include:
- increased cardiac output due to physical exertion;
- pulmonary vascular blood pooling due to immersion;
- increased pulmonary vascular resistance due to cold exposure;
- hydrostatic pressure effects;
- increased perfusion in the dependent lung with side-stroke swimming.[4,12,14,15]

The incidence of SIPE in Israeli combat swimmers, swimming vigorously, was 20 per year, up until 2004.[16] A comprehensive pulmonary investigation, including broncho-alveolar lavage, was undertaken in five such SIPE cases, and the results indicated that the pathology was capillary stress failure, with no evidence of inflammation. FVC and FEV_1 were reduced in two of these cases, but information on the delay between the incident and the investigations was lacking.

The common feature, pathophysiologically, was thought to be capillary stress and failure resulting from exertion and an increased inspiratory load, with the hydrostatic effects of immersion superimposed. The lateral decubitus swimming position (sidestroke) aggravated these effects in the dependent lung. Not all cases of SIPE exercised excessively. Some, especially the older subjects, may have had a cardiac basis, as with SDPE (see below).

Freediving, pulmonary oedema and pulmonary barotrauma of descent

Breath-hold dives to a depth of over 200 metres have been achieved in recent times, through a combination of physiological and anatomical factors and responses and of modern diving techniques. Pulmonary oedema in breath-hold diving has been reported and is believed to be largely a manifestation of pulmonary barotrauma of descent, 'lung squeeze', due to the reduction of lung volume below the residual volume developing according to Boyle's Law.[2,4,17,18] The postulated explanations for pulmonary oedema with freediving include:
- negative intra-alveolar pressure gradients due to descent
- pulmonary vascular blood pooling due to immersion
- increased pulmonary vascular pressure due to cold exposure
- increased cardiac output due to physical exertion
- pulmonary trauma due to 'lung packing'.

Clinical features of this pulmonary barotrauma are poorly documented but may include chest pain and haemoptysis with haemorrhagic pulmonary oedema. Treatment is based on the general principles of resuscitation, with 100% oxygen, treatment of shock, fluid replacement and CPAP. Two breath-hold fatalities attributed to pulmonary oedema have been reported.[4]

Liner and Andersson investigated 19 deep breath-hold divers during an international competition, to elicit signs of pulmonary oedema that were not evident following shallow dives.[18] After diving to 25–75 msw, 12 divers had such signs. The mean reductions in FVC and FEV_1 were -9% and -12% respectively, and -4% for S_aO_2. Six of the divers had respiratory symptoms (dyspnoea, cough, fatigue, retrosternal chest pain or discomfort and haemoptysis) and, in these, the falls in FVC, FEV_1 and S_aO_2 were greater: -16%, -27% and -11% respectively.

Scuba divers' pulmonary oedema (SDPE)

SDPE is usually described as an uncommon disorder, often in apparently healthy individuals with only a few dozen cases being documented.[4,11,15,19] In a survey of 1,250 divers, of the 460 responders, five (1.1%) had a history suggestive of pulmonary oedema.[11] The actual incidence is unknown, but SDPE is probably under diagnosed.[3,19,20] It differs from the other IPEs in being more frequent in older divers (see below). Exertion is not often recorded and is sometimes specifically denied.[15,20–22]

Symptoms usually resolve rapidly (minutes or hours) after the immersion. The hypoxaemia, respiratory function and radiology (chest X-ray or CT scan) also resolve rapidly in most cases.[11,19,23,24] Two deaths have been reported: one in a diver (who had had a previous episode) with hypertension, dyslipidaemia and arteriopathy, the other having no cardiac abnormality.[19] However, other deaths could have occurred and be attributed to drowning. Treatment includes oxygen supplementation, positive pressure respiration and possibly diuretics.

An individual predisposition for pulmonary oedema is a likely factor since a diver or swimmer with pulmonary oedema may have other episodes of IPE, previously or subsequently.[1,3,4,10,11,19–21,25] Yet when diving under similar conditions, the diver may have been spared. Whether the variation in presentation relates to the individual diver, the dive profile, environmental conditions or the dive equipment is unknown.

Discussion

Various conjectures on the aetiology of SDPE have been put forward.

COLD-INDUCED HYPERTENSIVE PULMONARY OEDEMA

Wilmshurst et al first described SDPE and attributed it mainly to the effects of cold, inducing hypertensive pulmonary oedema.[1] In their series, cardiovascular abnormalities were present in those who developed this condition on one or more occasions, compared to divers who never had pulmonary oedema. In a further report comparing divers and swimmers who had IPE to controls, it was hypothesized that 'labile hypertensives' with an exaggerated vasoconstrictor response to cold and/or raised oxygen pressure would be particularly prone to develop pulmonary oedema as a result of an increase in after-load because of systemic vasoconstriction, and a pre-load stress from the pulmonary vascular blood volume increase that occurs with immersion.[10] The divers in the pulmonary oedema group were followed up for an average of eight years, at which time seven had become hypertensive. All the cases occurred in waters below 12^{O}C. Thus, Wilmshurst's hypothesis incriminated a vascular hyper-reactivity to a cold stimulus.

This explanation is supported in some reports,[1,10,19,25] but not others, in which SDPE has been reported in relatively warm or tropical waters.[3,11,20–23] Hampson and Dunford concluded that cold may not have been an important factor in some of their other cases because the divers were protected by insulating drysuits, although they were still presumably exposed to cold-air inhalation.[15] They suggested that individuals with SDPE should be advised to forgo scuba diving. Pons et al reported results which did not support Wilmshurst's observations, finding no differences in forearm vascular resistance, vasoactive hormone levels, and left ventricular function between SDPE subjects and healthy controls.[11]

IMMERSION-INDUCED INTRATHORACIC BLOOD POOLING

Intrathoracic blood pooling can be induced when the body is submerged.[2,3,10,24,26] Some report an increase of up to 700 ml of pulmonary blood in water of 33–35^{O}C, with a 13–21 mmHg increase in pulmonary arteriole transmural pressure and a reduction in vital capacity of 5–10% in warm and cold waters respectively.[24,26] Others have observed smaller volumes of blood pooling, of up to 221 ml.[26,27] The thoracic blood pooling and the raised pulmonary artery pressure are postulated to cause increased capillary permeability, leading to pulmonary oedema.[4,10] Some feel that this is not a likely explanation for the development of this form of oedema.[11] An argument given is that the symptoms typically resolve rapidly once the diver is out of the water. However, many case histories include expectoration of bloody froth, indicating pulmonary capillary damage.[3,11,19,20]

AGE

Advanced age is a predisposing factor according to most authors.[4,10,19,20,23,24] Referencing the literature, Hampson and Dunford cite the age of 20 divers with SDPE from three studies to have been, on average, 42.7 (SD 2.7) years, while a large group of divers with other diving injuries averaged 35.5 (SD 4.0) years.[15] Cochard et al reviewed 37 cases and calculated the mean age of SDPE to be 50.3 (SD 7.5) years, compared to a mean age of other diving-related injuries of 34.0 (SD 9.2) years.[19] The detrimental effects of age could be enhanced by its correlation with hypertension, ischaemic or other heart diseases and impaired respiratory function. Koehle et al compared the demographics of scuba divers in Australia with their SDPE cases. The male/female ratios were almost the same but there were few divers over 45 years old in the diving population, whereas over half the SDPE cases were over this age.[4] This contrasted with the other IPEs.

NEGATIVE-PRESSURE PULMONARY OEDEMA

Negative inspiratory pressure has been postulated as a cause of SDPE by most reviewers.[2,3,11,19] In the scuba-diving environment, negative intrathoracic pressures during inspiration could occur from:

- immersion per se, especially with a head-up/vertical or head-out position;
- inspiratory breathing resistance from diving equipment;
- reduced gas supply/pressure;
- excessive gas density with depth;
- increased ventilation, as occurs with high workloads and anxiety;

- the use of a rebreather device with the counter-lung positioned at a shallower depth than the lung centroid.

Pulmonary oedema has not usually been reported in the numerous 'head-out' immersion experiments in the literature. There were some effects on lung function from head-out immersion in young men, when this was combined with a mild negative inspiratory pressure of 9 cmH_2O.[26] In the absence of clinical symptoms, and with the failure to reduce either FVC or maximal expiratory flows, this contribution to pulmonary oedema is unconvincing. In an ongoing study, this disorder was not provoked when divers were subjected to considerable negative-pressure inspiration, even when the negative pressures induced were extreme and close to intolerable over a one-hour period (Shields S, personal communication, 2009).

Thorsen et al demonstrated that increasing the inspiratory resistive load in divers and subjecting them to head-out immersion, reduced the diffusing capacity of the lung.[26] This may have indicated a subclinical pulmonary oedema. No changes occurred in pulmonary function with either of the conditions separately. There were no changes in FVC or maximal expiratory flows. Pulmonary oedema was not noted in experimental and actual head-out immersion experiences recorded in the literature.

The maximum negative inspiratory pressure likely to be encountered from a scuba-air breathing apparatus is 25–32 cmH_2O.[2] In the diving literature, 15–20 cmH_2O inspiratory resistance is considered moderate and 20–25 cmH_2O high.[27] The maximum sustained inspiratory load that can be tolerated is about 75 cmH_2O.[28] As quoted by Lundgren, even short exposures to 100 cmH_2O (as in attempting to snorkel at 1 metre depth) resulted in ventricular extrasystoles in three out of five subjects.[2] Acute cardiac dilatation results from greater exposures. A temporary increase in heart size was observed by Risch et al during submersion and negative-pressure breathing.[29]

PRE-EXISTING CARDIAC DISORDER

Magder et al compared the different clinical manifestations of myocardial ischaemia induced by exercise in the terrestrial and aquatic environments.[30] In these experiments, middle-aged males with cardiac ischaemia were exercised in both environments, with electrocardiographic monitoring to detect ST depression. Clinically the cardiac ischaemia presented with dyspnoea in the water (both 18^{O}C and 25.5^{O}C), and with angina pectoris on land. This may well have been the first description of mild pulmonary oedema when swimming, and whilst under rigorous scientific observation. The recognition of dyspnoea as a manifestation of ischaemic heart disease while immersed is, thus, understandable, as is the alleviation of this symptom following successful coronary artery surgery.[21,23,31]

Cochard et al described six episodes of SDPE amongst five experienced divers, aged 37–56, three of whom had hypertension, one had cardiac ischaemia with ventricular dysfunction and one died after a cardiac arrest.[19] Garcia et al described 10 cases, aged 46–74 years old, who developed pulmonary oedema, all of whom had cardiovascular disease.[32] SDPE developed in five prior to surfacing. Eight divers were taking beta-blockers, and this association has been noted in other case histories, as has the relationship with hypertension.[22,23,,32,33] Other cardiac pathologies, such as cardiomyopathy, have been reported in association with SDPE.[20]

The association of SDPE with hypertension is confused by other factors such as age, beta-blocker medication and ischaemic heart disease, complicating understanding of the relative significance of each of these factors.[4,32,33] Thus, SDPE, especially in older divers, should be an indication for comprehensive cardiac investigation, not only for possible cardiovascular therapy but also to avoid further SDPE episodes. It seems reasonable that unless the cause can be identified, verified and corrected, divers with SDPE should be advised of the possible risks of continuing with the activity that provoked it, and against further diving or energetic swimming.

DIVING-RELATED DISEASES

Pulmonary oedema may develop in diving, as it does in the terrestrial environment, from a variety of disorders and these complicate the diagnosis of SDPE. Some of these may be related to the diving activity and are more fully described in the diving medical texts.[34] Near drowning is recognised as a common cause of pulmonary oedema. A 'salt-water aspiration syndrome' (SWAS), secondary to inhalation of a fine spray of seawater through the diving regulator, was first described in 1970.[35] The clinical manifestations, time course and underlying mechanism(s) of SWAS, as verified experimentally, are different to those of SDPE.[35] Similar but more gross effects are observed in near-drowning cases.[36] Other pulmonary diseases to which divers are exposed may produce pulmonary oedema, or dyspnoea that could be attributed to pulmonary oedema.[34] These may include respiratory oxygen toxicity, gas contaminations, cold urticaria and asthma. Pulmonary decompression sickness, pulmonary barotrauma and the so-called 'deep diving dyspnoea' are diving disorders that may cause diagnostic confusion with uncomplicated SDPE.

To date, no association has been demonstrated between SDPE and decompression effects, although symptoms of both usually occur on or soon after ascent. Some SDPEs develop in such shallow exposures that intravascular bubble formation is unlikely.[20,22,33] Pulmonary filtration of bubbles during decompression in deeper scuba diving may increase pulmonary hypertension and damage capillary integrity, thereby increasing the likelihood of pulmonary oedema.

EXERCISE

The relationship between severe exertion and pulmonary oedema has been demonstrated in rugby players, cyclists, marathon runners and racehorses. West states "*Pulmonary capillaries have a dilemma. Their walls must be extremely thin for efficient gas exchange, but be immensely strong to resist the mechanical stresses that develop during heavy exercise. Elite human athletes at maximal exercise develop changes in the structure of the capillary wall as evidenced by red blood cells (and protein) in their alveoli. Racehorses routinely break their pulmonary capillaries while galloping.*"[8] Wagner et al indicated that the high cardiac output associated with high-intensity exercise elevates the pulmonary vascular pressure to such a degree that transudation of fluid across the capillary endothelium into the interstitial tissue increases markedly.[37,38]

Zavorsky, in 2007, reviewed the general medical literature, and supported the observation that exercise provoked pulmonary oedema.[39] His studies involved 137 exercising subjects and he grouped these into three: a short exercise stress that reached maximal oxygen consumption for only a couple of minutes; a prolonged (15–120 min) submaximal exercise and a maximum or near-maximum strenuous, sustained effort. Evidence of pulmonary oedema was found in 0%, 16% and 65% of subjects respectively. The likelihood seemed independent of lung size, sex or aerobic fitness. Thus, he supported a simple dose-response relationship between strenuous exercise and pulmonary oedema.

It is possible that negative inspiratory pressure and increased resistance to inspiration during maximal ventilation may occur with strenuous swimming. The association between extreme exercise and SIPE has been well documented, but a relationship with SDPE has not. Most cases of SDPE were not exerting themselves, given the exercise required for normal scuba activities. Because of the increased age and possibly dubious cardiac status in SDPE cases, it is feasible that a lesser degree of exercise may have more pronounced effects. For this reason, the influence of exercise on SDPE awaits further clarification.

MULTIFACTORIAL

Most would agree with Cochard that the explanation for SDPE is most likely in the combination of immersion, cold and compressed-air breathing stresses imposed on the cardiovascular and respiratory systems.[19] However, explanation of SDPE by applying the known physiological stressors from the diving environment is based on assumptions that need validation. For example, a deleterious influence of tight wetsuits has been mentioned, but was not confirmed in one experiment.[40] Such a restriction to respiration may be more likely to increase the work of breathing, thus possibly increasing dyspnoea in cases of SDPE, rather than contributing to the disease itself. Most other causation hypotheses have yet to be validated.

Conclusions

It is proposed that there are three major forms of IPE that occur in surface swimmers, freedivers and scuba divers. It is important to distinguish between these, even though they appear to share provoking causes to very varying degrees. In some instances, observations from the general medical literature enhance our understanding of the possible aetiologies, but often IPE has specific stressors.

The mechanisms of SIPE appear to include severe exertion and immersion, including thoracic blood pooling and the hydrostatic effects on the pulmonary circulation.

Pulmonary oedema in freediving is explained by the barotraumatic effects of descent. Both exercise and pulmonary volutrauma may contribute in some cases.

Existing cardiovascular and diving-induced diseases may cause or contribute to SDPE. The effects of immersion, including the increase in pulmonary vascular blood volume, the vascular response to hypothermia, the negative hydrostatic pressures of head-out and vertical positioning and negative intra-pleural pressures generated from exercise, gas density and inadequate diving equipment all appear to be contributory to SDPE. A full understanding of the mechanisms whereby these interact in individual divers to precipitate SDPE awaits clarification.

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Editor's note:
The following brief case report of a scuba diver with acute-onset dyspnoea at the end of a dive is typical of some of the presentations of SDPE. Here, a well-informed diver self-diagnoses SDPE and takes preventative action. Whether his diagnosis was correct or not is left to the reader to decide.