LEADING ARTICLE

Sports Med 2005; 35 (3): 183-190
0112-1642/05/0003-0183/$34.95/0

Pulmonary Oedema of Immersion

Michael S. Koehle,[1,2,3] *Michael Lepawsky*[2] and *Donald C. McKenzie*[1,2,3]

1 Allan McGavin Sports Medicine Centre, University of British Columbia, Vancouver, British Columbia, Canada
2 Department of Family Practice, University of British Columbia, Vancouver, British Columbia, Canada
3 School of Human Kinetics, University of British Columbia, Vancouver, British Columbia, Canada

Abstract

Acute pulmonary oedema has been described in individuals participating in three aquatic activities: (i) scuba diving; (ii) breath-hold diving; and (iii) endurance swimming. In this review, 60 published cases have been compiled for comparison. Variables considered included: age; past medical history; activity; water depth, type (salt or fresh) and temperature; clinical presentation; investigations; management; and outcome. From these data, we conclude that a similar phenomenon is occurring among scuba, breath-hold divers and swimmers. The pathophysiology is likely a pulmonary overperfusion mechanism. High pulmonary capillary pressures lead to extravasation of fluid into the interstitium. This overperfusion is caused by the increase in ambient pressure, peripheral vasoconstriction from ambient cold, and increased pulmonary blood flow resulting from exercise. Affected individuals are typically healthy males and females. Older individuals may be at higher risk. The most common symptoms are cough and dyspnoea, with haemoptysis also a frequent occurrence. Chest pain has never been reported. Radiography is the investigation of choice, demonstrating typical findings for pulmonary oedema. Management is supportive, with oxygen the mainstay of treatment. Cases usually resolve within 24 hours. In some cases, diuretics have been used, but there are no data as to their efficacy. Nifedipine has been used to prevent recurrence, but there is only anecdotal evidence to support its use.

Pulmonary oedema following scuba diving in previously healthy individuals is a rare occurrence that has been reported in the literature with increasing frequency in recent years.[1-10] An analogous phenomenon has also been reported in breath-hold divers[5,11-14] and endurance swimmers.[9,15-18] The similarities present between these entities are extensive and bear analysis. In this review, we have compiled the reported cases of idiopathic pulmonary oedema resulting from these activities and profiled them to help improve understanding of this condition.

1. Methods

PubMed/Medline and MDConsult databases were searched by combining the keywords 'scuba', 'diving' or 'swimming' with 'pulmonary oedema'. The entire date ranges of these databases were searched through August 2003. Bibliographies of articles selected were used to find additional pertinent references. Preference was given to primary

references, including surveys, case series and original research. The search was not limited to articles in the English language. A total of 20 articles and abstracts met these criteria.

Case reports were included if, during or following an episode of scuba diving, breath-hold diving or swimming, case subjects developed signs and/or symptoms that were diagnosed as pulmonary oedema. Four cases[13,14] were also included (all in breath-hold divers) that were not given an initial diagnosis of pulmonary oedema, but the divers experienced haemoptysis and cough, and they also demonstrated clinical or pathological findings consistent with pulmonary oedema and/or alveolar haemorrhage.

2. Results

After the above-mentioned search, 60 cases were identified in 56 individuals (some individuals had experienced more than one episode). There was wide variation in the amount of detail provided for each case; therefore, with the exception of sex and activity at the time of the incident, all other variables represent incomplete data. Sixteen (28.6%) of the individuals were females and accounted for 20 (33.3%) of the cases (figure 1). The other 40 cases (66.7%) occurred in 40 males (71.4%). Mean reported age (±SD) was 36.1 years (±14.2) with a range from 18 to 61 years (figure 2). Among scuba divers, the mean age was 47.1 years (±11.0) with a range from 24 to 61 years (figure 3). Only ten of the case subjects had pre-existing medical conditions. Essential hypertension was most commonly reported (in five cases). Two divers reported having asthma, and one had a history of 'childhood asthma'. Other declared conditions included: a prior cerebrovascular accident, obsessive-compulsive disorder, atrial fibrillation, bicuspid aortic valve, arthritis and migraines. None of the reports included a history of aspiration. Of the affected breath-hold divers, three reported performing voluntary diaphragmatic contraction while diving. Three of the breath-hold divers had taken aspirin (acetylsalicylic acid) prior to diving for its supposed ergogenic benefits.

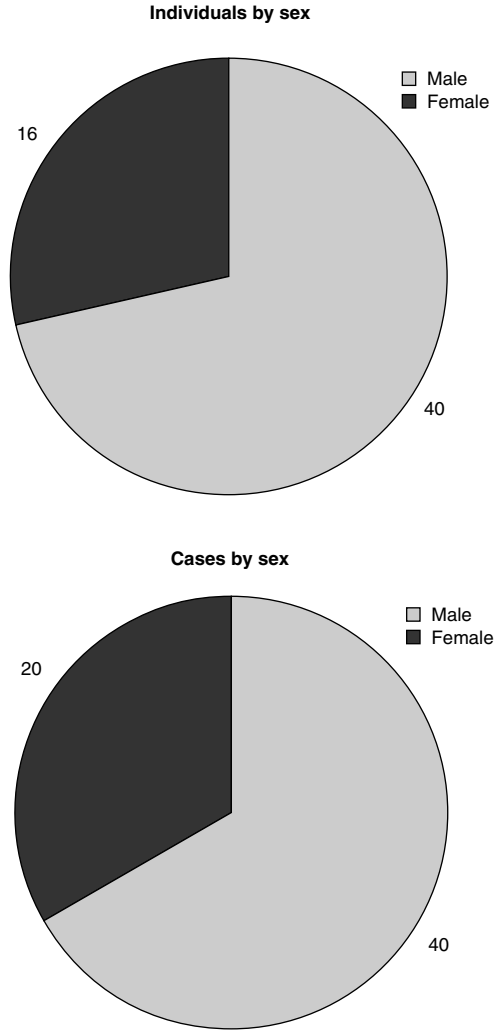

Fig. 1. Distribution of individuals and reported cases by sex.

Scuba diving accounted for the most cases reported at 34 (56.7%). Cases were documented in both commercial and recreational divers in wetsuit and drysuit dives. Swimming and breath-hold diving accounted for 18 (30%) and 8 (13.3%) cases, respectively (figure 4). Patients had a history of a prior or subsequent episode in at least 13 cases. In at least three of the reported individuals, incidents occurred with both scuba diving and swimming.

Incidents occurred in various environmental conditions. Depths ranged from 2.9 to 42m in scuba and

breath-hold divers. Water temperature ranged from 4.7 to 19°C, with a mean of 15.2°C (±7.4). Thirty-six cases reportedly occurred in saltwater, while seven were reported to have occurred in freshwater. On the day of the event, all of the breath-hold divers had performed multiple dives, as had six (17.6%) of the scuba divers.

Cough was the most common presenting symptom, having been reported in 49 cases (81.7%). Dyspnoea was nearly as frequently reported, occurring in 48 cases (80.0%). Haemoptysis was described in 37 cases (61.7%), whereas chest pain was not reported in any of the cases. Less common symptoms were weakness (two cases) and confusion (one case). Physical examination was typically not well described, but crackles (or rales) and wheezes were the most common signs (15 and 6 cases, respectively).

The most frequently performed investigation was chest radiography. In some cases it was unclear at what point in time the radiograph was performed. In cases where the radiograph was performed while the patients were symptomatic, all but one film demonstrated lung infiltrates consistent with oedema. In five cases of endurance swimmers, where the individuals had been swimming in the lateral decubitus position (for military training), the radiographic findings were only present on the dependant side. In 15 cases, blood gases were documented on room air (figure 5). Oxygen partial pressure (pO_2) ranged from 35 to 85mm Hg with a mean of 66.2mm Hg (±17.4) while oxygen saturation ranged from 70% to 98% with a mean of 88.8% (±7.3) [figure 6]. At least 18 of the patients had an ECG performed. In one case, a left bundle-branch block was identified, and in another atrial fibrillation was documented in a patient with a known history of atrial fibrillation. Cardiac enzymes were drawn on two occasions, and were normal. Fifteen echocardiograms were performed (the patients were typically asymptomatic by the time of the study) and only one was abnormal. In this patient, mitral valve prolapse with mild mitral regurgitation was demonstrated. In a series of three breath-hold divers,[12] broncheoalveolar lavage was performed, with findings consistent with intra-alveolar haemorrhage in all three cases. A CT scan was performed acutely in two cases, demonstrating findings consistent with intra-alveolar haemorrhage in both. Follow-up pulmonary function testing was performed in at least 16 cases. All were normal.

The most common acute treatment was the provision of oxygen, typically by mask. A diuretic (usually furosemide) was used in ten cases. β-agonists,

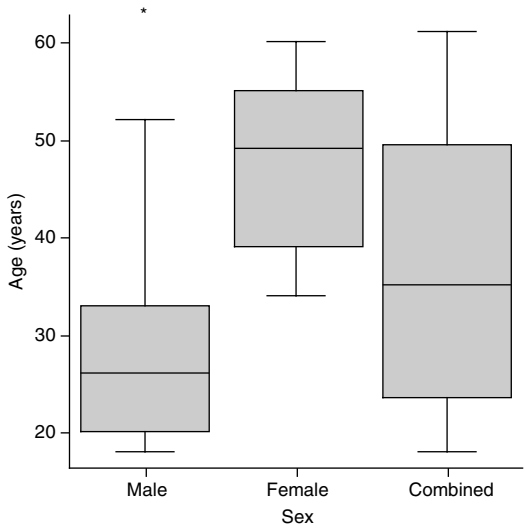

Fig. 2. Age distribution of all reported cases. * indicates outlier value.

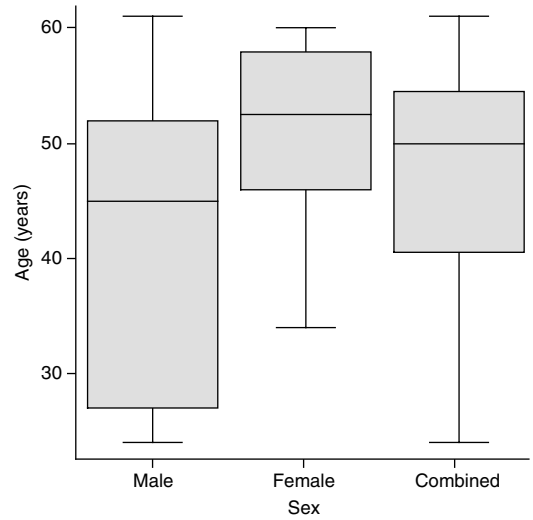

Fig. 3. Age distribution of reported cases involving scuba diving only.

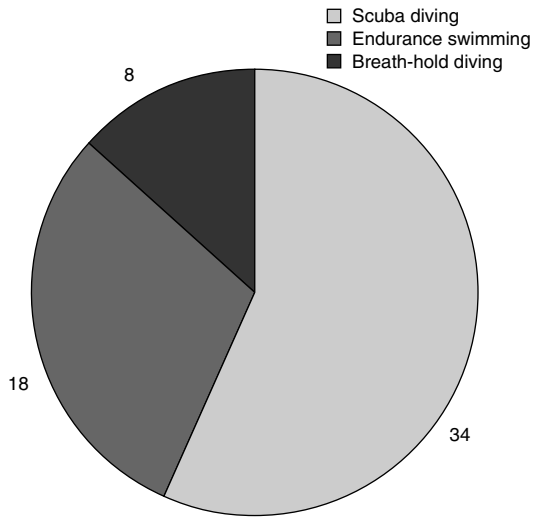

Fig. 4. Distribution of reported cases by activity.

such as salbutamol, were used in six cases. Other treatments involved nitroglycerin, aspirin, heparin, magnesium sulfate, digoxin, ciprofloxacin and hyperbaric oxygen.

In the majority of cases, symptoms resolved between 5 minutes and 24 hours. Two reported cases were fatal.[14,19] These cases were very similar to reports of fatal incidents in breath-hold divers with no medical history. Both occurred at a depth of about 12–15m, while notably they had been previously diving about twice as deep that day. In these two cases, the divers presented unconscious in convulsions with haemoptysis and irregular breathing. In both cases there was a window of partial recovery, followed by deterioration, with death occurring approximately 3 hours after the event. Autopsy was performed in both cases, finding alveolar haemorrhage and interstitial oedema, but no evidence of gas emboli. The findings were not consistent with drowning; the diagnosis in each case was 'thoracic squeeze', which the authors attributed to a large pressure gradient between the capillaries and the alveoli. These cases were included in this series because they were similar enough in inciting activity, interstitial oedema and haemoptysis, to the rest of the cases, that they may have been severe cases of oedema of immersion.

3. Discussion

Initially, oedema of immersion was thought to be caused by scuba diving in cold water.[8] This case series demonstrates that this condition can occur under a wider variety of conditions. It can occur in divers at depths as shallow as 2.9m, as well as endurance surface swimmers. Although incidents

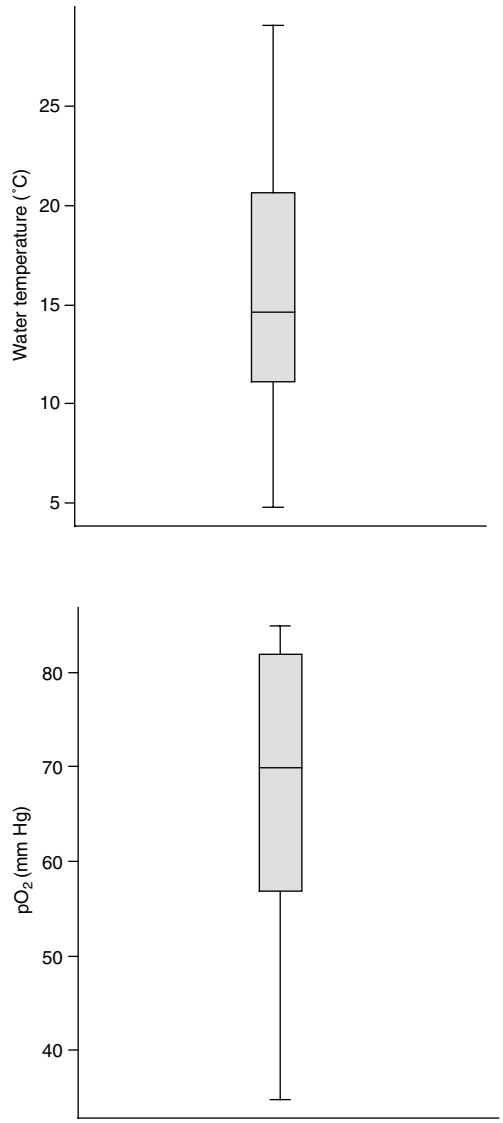

Fig. 5. Temperature distribution of all reported cases and arterial oxygen pressure on room air at presentation. **pO_2** = oxygen partial pressure.

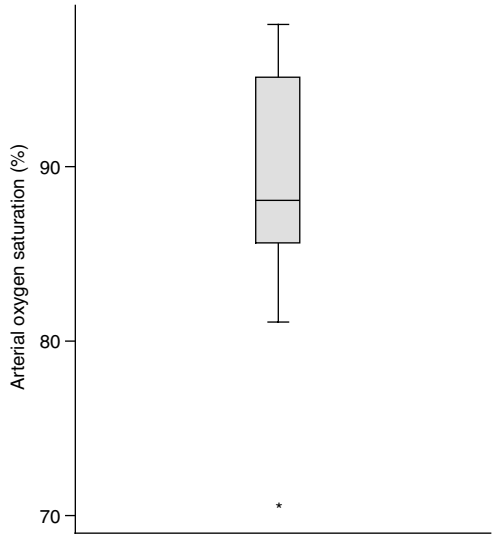

Fig. 6. Oxygen saturation on room air at presentation. * indicates outlier value.

have occurred in water as warm as 29°C, there seems to be a predisposition towards colder water (figure 5).

The proposed mechanism for pulmonary oedema of immersion is interstitial oedema secondary to transudation from the pulmonary circulation.[2,4,9,15,16] Increased hydrostatic pressure during immersion causes a central redistribution and pooling of blood. The increase in intrathoracic blood volume has been estimated to be approximately 700mL; this increase has been associated with an increase in pulmonary artery pressure of 12mm Hg.[20] This pressure would be further increased in colder water due to peripheral vasoconstriction. In a study of exposure to ice-cold showers, both preload and afterload were increased.[21] West et al.[22] examined the mechanism of capillary stress failure resulting from increased pulmonary capillary pressure. In an animal model, they observed that increased capillary pressure led to distension of the capillaries, followed by separation of the capillary endothelial and alveolar epithelial cell layers. In some cases they observed disruption of the basement membrane. Based on this mechanism, we could expect to see an interstitial oedema forming at lower pressures, progressing to an alveolar oedema with progressive breakdown of the capillary-alveolar barrier.

Endurance surface swimmers would experience less ambient pressure than divers at depth. However, other factors coexist that may increase pulmonary blood flow and potentially lead to oedema in these individuals. Heavy exertion has long been demonstrated to cause pulmonary oedema in animals such as horses.[22] There is also some evidence that a similar phenomenon occurs in humans.[23,24] Caillaud et al.[24] examined triathletes, pre- and post-race in terms of diffusing capacity and CT. They demonstrated a post-race decrease in diffusing capacity with a concomitant increase in the mean lung density and the number of opacities on imaging. Marshall et al.[23] found that there was an increase in lung water in the supine position over the upright position, and a further increase with exercise. Thus, athletes swimming horizontally on the surface in cold water at a high intensity could still develop capillary stress failure resulting in oedema in a similar fashion to the divers.

In the reports of Lund et al.[15] and Mahon et al.,[16] special forces combat swimmers developed unilateral pulmonary oedema when swimming in the lateral decubitus position. The infiltrates occurred in the dependent lung only. Presumably, when swimming in the lateral decubitus position, perfusion would be greater on the dependent side, thus leading to an increase in pulmonary capillary pressure and more stress failure. This finding provides compelling evidence for a haemodynamic role in the aetiology of this immersion-related pulmonary oedema. As there have been cases in which the same individual experienced pulmonary oedema with diving and with endurance swimming (at different times), we can be reasonably confident that an analogous pathophysiology may be proposed in both activities. Predisposing factors should be similar and thoracic squeeze may be considered toward the severe to fatal end of a clinical spectrum.

Thorsen et al.[25] suggested that the inspiratory resistive load experienced during scuba diving could further increase the transmural capillary pressure gradient, contributing to the oedema in scuba divers.

They compared lung diffusing capacity following immersion with and without a resistive inspiratory load. A significant decrease in diffusing capacity was observed following the combination of immersion and inspiratory resistive load compared with control. This altered diffusing capacity normalised within 24 hours. Impaired diffusing capacity may represent evidence of a subclinical interstitial pulmonary oedema that acts as a barrier to slow gas diffusion between the alveoli and the pulmonary capillaries. Thus, in addition to the increased ambient pressure and the cold-induced peripheral vasoconstriction experienced by scuba divers, there is an increase in inspiratory load from their breathing equipment that can further amplify the transmural pressure gradient in the lung contributing to pulmonary oedema.

To determine whether sex or age were possible risk factors, the case series data were compared with a survey of diver demographics in Australia.[26] In the survey, 65.4% of divers were male. This number is quite close to the proportion of reported cases in male divers (66.7%). From these very limited data, there is no evidence for a sex predisposition for oedema of immersion. In the same survey of Australian divers, 52% of divers were in the 25- to 34-year age group. Approximately one-fifth of divers were in each of the 18- to 24-year and 35- to 44-year age groups, with 'very few' divers aged >45 years. In contrast, more than half of the oedema cases in scuba divers occurred in individuals aged >45 years. Therefore, the reported cases (when only diving cases are considered [figure 3]) show that oedema of immersion may occur more commonly in older scuba divers. More complete data would be required to confidently estimate the role of age in oedema of immersion.

Wilmshurst et al.[9] proposed that there may be an interaction between hypertension, endothelial function and oedema of immersion. They conducted a study comparing vascular response in 11 subjects with a past history of pulmonary oedema during diving (experimental group) with response in ten control divers. Blood pressure and forearm blood flow were measured (using a strain gauge and plethysmography) at rest and after exercise, hyperoxia, cold, or a combination of cold and hyperoxia. Compared with the control group, the experimental group had a significantly higher blood pressure and forearm vascular resistance following each intervention. The experimental group was also followed up for an average of 8 years after presentation. Seven of the 11 divers went on to develop essential hypertension. From these results the investigators proposed that altered vascular reactivity predisposed the divers to both pulmonary oedema of immersion and essential hypertension. In the current case series, only five individuals had a previous history of hypertension. With the exception of the Wilmshurst study there were no other reports of hypertension on follow-up. Therefore, there does not appear to be much evidence of a link between essential hypertension and pulmonary oedema of immersion. Although it presents a compelling mechanism for why some individuals develop oedema, no other studies have looked at vascular reactivity in this population.

With only two individuals with asthma in the case series (and one case of possible childhood asthma) it does not appear to be a significant risk factor. Approximately 5% of divers admit to being asthmatic,[27] which is similar to the three possible cases out of a total of 60 (5.0%). No other medical conditions occurred in more than one case.

A series of three cases in breath-hold divers reported that each of them ingested aspirin prior to diving.[12] According to the authors, aspirin is commonly taken among European breath-hold divers as an analgesic and an ergogenic aid. Because of its anti-platelet activities, the authors postulated that it might have contributed to the observed haemoptysis. They go on to suggest that all breath-hold divers refrain from using aspirin for this reason. No other reports in this series mention the use of aspirin.

In terms of clinical presentation, much can be learned from this case series. Cough and dyspnoea occur in >80% of cases and are, therefore, the most indicative symptoms. Haemoptysis occurred in at least 61% of cases. None of the patients complained of chest pain in this series. Therefore, if a patient presents with chest pain, alternative diagnoses

should be considered (such as acute coronary syndrome, pulmonary embolism or pulmonary decompression sickness). Other non-specific symptoms included fatigue, confusion and patient reports of 'wheezing'. On physical examination, rales and wheezes were commonly present. A simple chest radiograph was diagnostic in most cases. Blood gases showed a wide range of pO_2 and oxygen saturation on presentation, but they can be useful in quantifying the level of hypoxia. In the two cases where CT was performed acutely, it was also diagnostic of pulmonary oedema. CT would, therefore, be a good adjunct modality for cases when radiographs are non-diagnostic.

The pathophysiology of oedema of immersion and 'thoracic squeeze' may be one in the same. Both reports of 'thoracic squeeze'[14,19] propose a large pressure differential between the capillaries and the alveoli as the cause. They suggest that descent causes the lung volume to decrease (as per Boyle's law) but that once the lung volume reaches residual volume, it cannot compress more, and the capillary-alveolar pressure gradient increases to a level high enough to cause stress failure. This may not be a singular possibility. Both cases occurred at depths of 12–15m, approximately half the depth that these experienced divers had already been diving that same day. Such a depth would provide a pressure of at most 2.5 atmospheres, which maximally would reduce lung volume slightly >60%; not enough to reduce it below residual volume causing collapse. Therefore, we suggest that these two cases may represent fatal pulmonary oedema of immersion, in which the pulmonary pressure changes caused by a depth-induced increase in ambient pressure were potentiated by the physical exertion of breath-hold diving. The term 'thoracic squeeze' should only be used to signify the severe endpoint of this pathophysiology with resultant parenchymal collapse.

Other investigations such as electrocardiography, spirometry and echocardiography were not helpful in making the diagnosis of pulmonary oedema of immersion, but may have a role in excluding other diagnoses.

Most cases of pulmonary oedema appeared to have resolved quickly, many before treatment was instituted. Oxygen has been the most common acute treatment, with diuretics being the second most common. Other modalities that have been used, such as aspirin, heparin and recompression might reflect diagnostic uncertainty on the part of the treating physician. Each of these treatments has known possible adverse consequences, thus underlining the importance of prompt and accurate diagnosis. With the documented good outcomes in 58 cases, supportive care (and/or intensive care, if necessary) with or without diuretics seems to be the most appropriate management. This by no means minimises the risk of withholding a therapy that itself may lessen morbidity and/or mortality.

No work has been done to find a way to prevent pulmonary oedema cases. Wilmshurst in his clinical practice advises divers with a history of pulmonary oedema to refrain from diving.[7] Those that have not heeded his advice have been prescribed 5mg of nifedipine before diving.[7] There have not been enough cases for a trial to be done, but none of the nifedipine-prescribed cases have recurred. Nifedipine has long been used in the treatment of high-altitude pulmonary oedema. The presumed mechanism of action is reduction of the pulmonary hypertension that is caused by the hypoxia of altitude. In oedema of immersion, it may have a similar action. By reducing pressure in the pulmonary vasculature, less capillary stress failure (and less oedema) should develop. More research is required to determine whether nifedipine is safe and effective in preventing this condition.

4. Conclusion

Pulmonary oedema of immersion can occur during scuba diving, breath-hold diving or endurance swimming. It occurs in salt or fresh water, with a predisposition for colder temperatures. It does not appear to be affected by sex, but may be more common in an older population. Affected individuals are typically healthy. Patients present with a combination of cough, dyspnoea and/or haemoptysis. Chest pain is not a feature. Physical and radio-

logical examinations are consistent with pulmonary oedema. Most cases resolve quickly with supportive care. In a significant proportion of individuals, it can become a recurrent phenomenon.

The likely mechanism of action is capillary stress failure resulting from pulmonary vascular hypertension, which is aggravated by cold temperatures, exertion and an increased inspiratory load. 'Thoracic squeeze' may be a severe variant of pulmonary oedema of immersion. Individuals with higher vascular reactivity may be more at risk. Nifedipine has been proposed as a possible preventative agent in individuals who wish to return to diving, but requires research before any recommendation can be made.

Acknowledgements

Dr Koehle was supported by the National Sciences and Engineering Research Council and the British Columbia Sports Medicine Research Foundation. No potential conflicts of interest exist.

References

1. Cosgrove H, Guly H. Acute shortness of breath: an unusual cause. J Accid Emerg Med 1996; 13 (5): 356-7
2. Gnadinger CA, Colwell CB, Knaut AL. Scuba diving-induced pulmonary edema in a swimming pool. J Emerg Med 2001; 21 (4): 419-21
3. Halpern P, Gefen A, Sorkine P, et al. Pulmonary oedema in SCUBA divers: pathophysiology and computed risk analysis. Eur J Emerg Med 2003; 10 (1): 35-41
4. Hampson NB, Dunford RG. Pulmonary edema of scuba divers. Undersea Hyperb Med 1997; 24 (1): 29-33
5. Pons M, Blickenstorfer D, Oechslin E, et al. Pulmonary oedema in healthy persons during scuba-diving and swimming. Eur Respir J 1995; 8 (5): 762-7
6. Slade Jr JB, Hattori T, Ray CS, et al. Pulmonary edema associated with scuba diving: case reports and review. Chest 2001; 120 (5): 1686-94
7. Wilmshurst P. Cardiovascular problems in divers. Heart 1998; 80 (6): 537-8
8. Wilmshurst P, Nuri M, Crowther A, et al. Forearm vascular responses in subjects who develop recurrent pulmonary oedema when scuba diving: a new syndrome [abstract]. Br Heart J 1981; 45: 349
9. Wilmshurst PT, Nuri M, Crowther A, et al. Cold-induced pulmonary oedema in scuba divers and swimmers and subsequent development of hypertension. Lancet 1989; I (8629): 62-5
10. Wilmshurst P. Analysis of decompression accidents in amateur divers. Prog Underwater Sci 1990; 15: 31-7
11. Boussuges A, Succo E, Bergmann E, et al. Intra-alveolar hemorrhage: an uncommon accident in a breath holding diver [in French]. Presse Med 1995; 24 (25): 1169-70
12. Boussuges A, Pinet C, Thomas P, et al. Haemoptysis after breath-hold diving. Eur Respir J 1999; 13 (3): 697-9
13. Kiyan E, Aktas S, Toklu AS. Hemoptysis provoked by voluntary diaphragmatic contractions in breath-hold divers. Chest 2001; 120 (6): 2098-100
14. Strauss MB, Wright PW. Thoracic squeeze diving casualty. Aerosp Med 1971; 42 (6): 673-5
15. Lund KL, Mahon RT, Tanen DA, et al. Swimming-induced pulmonary edema. Ann Emerg Med 2003; 41 (2): 251-6
16. Mahon RT, Kerr S, Amundson D, et al. Immersion pulmonary edema in special forces combat swimmers. Chest 2002; 122 (1): 383-4
17. Shupak A, Weiler-Ravell D, Adir Y, et al. Pulmonary oedema induced by strenuous swimming: a field study. Respir Physiol 2000; 121 (1): 25-31
18. Weiler-Ravell D, Shupak A, Goldenberg I, et al. Pulmonary oedema and haemoptysis induced by strenuous swimming. BMJ 1995; 311 (7001): 361-2
19. Khan MY. Fatal thoracic squeeze. J Indian Med Assoc 1979; 73 (2): 38-9
20. Arborelius Jr JM, Balldin UI, Lilja B, et al. Hemodynamic changes in man during immersion with head above water. Aerosp Med 1972; 43 (7): 592-8
21. Keatinge WR, McIlroy MB, Goldfien A. Cardiovascular responses to ice-cold showers. J Appl Physiol 1964; 19 (6): 1145-50
22. West JB, Tsukimoto K, Mathieu-Costello O, et al. Stress failure in pulmonary capillaries. J Appl Physiol 1991; 70 (4): 1731-42
23. Marshall BE, Teichner RL, Kallos T, et al. Effects of posture and exercise on the pulmonary extravascular water volume in man. J Appl Physiol 1971; 31 (3): 375-9
24. Caillaud C, Serre-Cousine O, Anselme F, et al. Computerized tomography and pulmonary diffusing capacity in highly trained athletes after performing a triathlon. J Appl Physiol 1995; 76 (4): 1226-32
25. Thorsen E, Skogstad M, Reed JW. Subacute effects of inspiratory resistive loading and head-out water immersion on pulmonary function. Undersea Hyperb Med 1999; 26 (3): 137-41
26. Australian Sports Commission. Active Australia: the numbers game [online]. Available from URL: http: //www.activeaustralia.org/facts/scubadiving.pdf [Accessed 2003 Aug 20]
27. Koehle M, Lloyd-Smith R, McKenzie D, et al. Asthma and recreational SCUBA diving: a systematic review. Sports Med 2003; 33 (2): 109-16

Correspondence and offprints: Dr *Michael S. Koehle*, c/o Allan McGavin Sports Medicine Centre, 3055 Wesbrook Mall, Vancouver, BC V6T 1Z3, Canada.
E-mail: mskoehle@interchange.ubc.ca