Clin Physiol Funct Imaging (2010) **30,** pp181–186 doi: 10.1111/j.1475-097X.2010.00922.x

Pulmonary oedema in healthy SCUBA divers: new physiopathological pathways

M. Coulange[1,2], P. Rossi[2], O. Gargne[2], Y. Gole[2], J. Bessereau[2], J. Regnard[3], Y. Jammes[2], A. Barthélémy[1,2], J. P. Auffray[1,2] and A. Boussuges[2]

[1]*Service de Médecine Hyperbare, Pôle RUSH, Hôpital Sainte Marguerite, Marseille,* [2]*Unité Mixte de Recherche-Ministère de la Défense « Physiologie et Physiopathologie en Condition d'Oxygénation Extrême », Université de la Méditerranée et IRBA, Marseille, and* [3]*Laboratoire de Physiopathologie Cardiovasculaire EA 3920, Université de Franche-Conté, Besançon, France*

Correspondence

Mathieu Coulange, MD, PhD, Service de Médecine Hyperbare, Hôpital Sainte Marguerite, 270 Bd de Sainte Marguerite, 13274 Marseille cedex 09, France

E-mail: mathieu.coulange@ap-hm.fr

Accepted for publication

Received 19 August 2009; accepted 14 December 2009

Key words

cold; exercise; immersion; pulmonary oedema; SCUBA diving

Summary

Introduction: The mechanism of immersion pulmonary oedema occurring in healthy divers is a matter of debate. Among consecutive injured divers admitted to our hyperbaric centre, we analysed prospective data about pulmonary oedema.

Method: A total of 22 divers suffering from immersion pulmonary oedema without cardiac disease were included. The occurrence of events was compared to the diving conditions as assessed by diving-computer. Each patient underwent a clinical examination, laboratory tests, thoracic CT scan and echocardiography.

Results: The median age was 49 years, with a higher proportion of women, in comparison with the data of the French diving federation. The common feature was the occurrence of respiratory symptoms during the ascent after median dive duration of 29 min with strenuous exercise and/or psychological stress. Most of the dives were deep (37 msw–121 fsw) in cool water (15°C-59°F). The average inspired oxygen partial pressure was 0·99 bar. Progression was rapidly favourable, and the medical check-up after clinical recovery was normal.

Conclusion: Immersion, body cooling, hyperoxia, increased hydrostatic pressure and strenuous exercise likely combine to induce pulmonary oedema in patients without cardiac disease. This study underlines new physiopathological tracks related to the frequent occurrence of symptoms noticed in the last part of the ascent and a higher incidence in women.

Introduction

The occurrence of pulmonary oedema during strenuous swimming or SCUBA (self-contained underwater breathing apparatus) diving in cold water was first reported by Wilmshurst *et al.* (Wilmshurst *et al.*, 1989). These authors hypothesized that pulmonary oedema resulted from an increased pulmonary capillary pressure because of peripheral blood being shifted into the thorax as a result of hydrostatic pressure during immersion. This centralization of blood volume would be reinforced in cold water because of the peripheral vasoconstriction. Weiler-Ravell *et al.* reported a pulmonary haemorrhage during the first 45 min of strenuous swimming in temperate Mediterranean waters (23°C–73°F) (Weiler-Ravell *et al.*, 1995). Strenuous exercise markedly increases pulmonary blood flow and may favour pulmonary oedema (West & Mathieu-Costello, 1992). In fact, the mechanism of pulmonary oedema during immersion is a matter of debate as few data are available on the actual diving conditions of occurrence. Furthermore, individual susceptibility to develop this phenomenon remains unclear. Pons *et al.* did not measure any significant change in forearm vascular resistance, left ventricular function and blood level of vasoreactive hormones during a cold exposure test in patients with a history of pulmonary oedema during scuba diving (Pons *et al.*, 1995). These findings were probably attributed to the fact that their cold exposure test was either not intense enough or counteracted by regulatory mechanisms. Indeed, Wilmshurst *et al.* demonstrated a larger increase in forearm vascular resistance during a cold pressure test compared to a control group and reported subsequent development of hypertension (Wilmshurst *et al.*, 1989).

In this work, divers admitted to the hyperbaric centre with suspected immersion pulmonary oedema were prospectively investigated to look over tracks of physiopathological pathways.

Methods

Study setting and design

From 1st January 2003 to 1st July 2009, all injured divers admitted to our hyperbaric centre and suffering from respiratory troubles, were investigated. Investigations included clinical examination with full history, ECG recording, determination of venous blood levels of brain natriuretic peptides (NT pro-BNP or BNP) and troponin, blood gas analyses, standard chest radiography, thoracic tomodensitometry and transthoracic echocardiography. Divers used a diving-computer that recorded diving profiles (length, depth, diving profile, ascent speed and decompression stop time) and allowed the measurement of water temperature.

The diagnosis of immersion pulmonary oedema was supported by history, clinical presentation and results of biological and radiological investigations. We did not include cases in which the clinical history suggested drowning, pulmonary barotraumas, chokes or hypercapnia. BNP above 100 pg ml^{-1} in non-obese patients, NT-proBNP above 450 pg ml^{-1} for patients younger than 50 years old and NT-proBNP above 900 pg ml^{-1} for those 50 years or older, strongly suggested the diagnosis of congestive heart failure in patients with dyspnea (Daniels & Maisel, 2006). A troponin value above 0·35 ng ml^{-1} was considered as abnormal. Chest radiography and thoracic tomodensitometry were analysed by two independent radiologists. The following signs were considered for the determination of the presence of immersion pulmonary oedema: (i) loss of sharp definition of pulmonary vascular markings; (ii) diffuse opacification; (iii) interlobular septal thickening; (iv) peribronchial cuffing; (v) fissural thickening and (vi) pleural effusion (Gluecker *et al.*, 1999). Tomodensitometry filtered out signs of barotraumas (pneumothorax and pneumomediastinum).

Only presumed healthy subjects were enrolled in this study; divers with history of cardiac diseases or abnormal echocardiographic examination were excluded.

All experimental procedures were conducted in accordance with the Declaration of Helsinki and were approved by the local ethics committee. Each method and the potential risks were explained to the participants in detail, and they all gave written informed consent before the experiment.

Statistical analysis

Data are presented as median (range). The sex ratio was compared to that of the whole population with diving accident using Fisher's exact test. Significance was accepted if $P<0·05$.

Results

Among 453 consecutive patients (23% women, average age: 41 ± 9 years) admitted to our hyperbaric centre for a diving accident, 54 patients suffered from dyspnoea and/or cough and were invited to participate in the study. We did not include subjects for whom the accident was attributed to drowning (n = 1), pulmonary barotraumas (n = 16), chokes (n = 2) or hypercapnia (n = 4). Patients with a history of coronary disease (n = 1), hypertension (n = 4), cardiac valvulopathy (n = 1) were excluded from the study. Moreover, echocardiographic examination found left ventricular dysfunction in three divers leading to the exclusion of these subjects.

Population studied

A total of 22 patients suffering from immersion pulmonary oedema without cardiac disease were included in the study. The median age of victims of immersion pulmonary oedema was 49 years (range: 29–66 years) and body mass index 25·2 kg m^{-2} (range: 21·2–28·5 kg m^{-2}). Among the 22 patients, 14 were women. There was statistically higher incidence in the female divers ($P<0·001$) in comparison with diving accident examined over the same period (453 patients, 104 women). Nine were menopausal (natural: seven, surgical: two).

Diving characteristics associated with pulmonary oedema

Diving characteristics are listed in Table 1, and the example of diving profile is illustrated in Fig. 1. The dives were deep with the median depth of 37 msw (121 fsw) (range: 10–63 msw, 33–207 fsw) and median duration of 29 min (range: 15–42 min) in water at a median of 15°C (59°F) (range: 13–27°C, 55–81°F). All divers wore a 5–7 mm thick wetsuit. Median maximal inspired oxygen partial pressure was 0·99 bar (range: 0·42–1·53 bar). Most of them had good diving levels (from medium to experienced recreational divers), but fourteen were training in rescue procedures with iterative ascents. During the dive, none of them experienced any sensation of expiratory blocking.

Symptoms

The symptomatology always began during the ascent, except for patient 12 whose first symptoms occurred after only 15 min of diving at 10 msw (33 fsw). Seven patients reported that their symptoms appeared during the last 10 metres (33 ft) of the ascent. Three divers did not perform their decompression step because of an important dyspnoea. Some divers reported unpleasant cold sensation within the last 15 min of immersion (n = 8), hard effort (n = 13) and/or anxiety (n = 5). As soon as they were out of the water, tachypnea was noticed in all the patients, associated with cough in seventeen and frothy sputum in eight. Nine divers reported haemoptysis. Also three patients said that they had suffered a similar cough during a previous dive, which had ceased spontaneously in <24 h.

Clinical examination

On hospital admission (Table 1), clinical examination evidenced mostly a bilateral pulmonary oedema that was well

Table 1 Diving conditions and clinical observations.

Case	Age, year/sex	Water temperature (°C)	Depth (msw)	P_IO_2 (bar)	Duration (mn)	SaO_2 (%)	PaO_2 (mmHg)	Troponin (ng ml^{-1})	Natriuretic peptides (pg ml^{-1})
1	46/M	19	13	0·48	19	82	65	0·22	nd
2	48/F	15	21	0·65	40	nd	nd	nd	nd
3	50/M	15	20	0·63	20	90	58	0·17	nd
4	50/F	19	36	0·97	25	94	62	0·02	417 (NT pro BNP)
5	59/M	15	63	1·53	20	85	48	0·09	45 (NT pro BNP)
6	43/M	13	25	0·74	32	94	73	0·05	384 (NT pro BNP)
7	37/F	27	40	1·05	30	98	90	0·01	47 (BNP)
8	50/F	25	41	1·07	30	nd	nd	0·02	326 (NT pro BNP)
9	66/M	20	20	0·63	20	96	86	0·04	nd
10	29/F	13	38	1·01	30	97	74	0·01	**134** (BNP)
11	32/F	13	25	0·74	40	94	79	nd	**119** (BNP)
12	56/F	14	40	1·05	33	95	71	0·06	57 (BNP)
13	49/F	13	10	0·42	15	92	65	**1·6**	**708** (BNP)
14	38/F	14	42	1·09	31	96	69	nd	nd
15	49/M	13	40	1·05	26	95	70	0·01	nd
16	63/F	22	38	1·01	42	89	57	**0·6**	**190** (BNP)
17	49/F	18	37	0·99	33	91	53	**1·47**	**200** (BNP)
18	47/F	14	15	0·53	20	92	60	**1**	**700** (BNP)
19	50/M	15	38	1·01	25	88	59	0·16	**131** (BNP)
20	45/F	14	37	0·99	30	96	70	0·02	**175** (BNP)
21	45/M	13	37	0·99	27	94	77	0·01	66 (BNP)
22	43/F	14	40	1·05	15	95	71	**0·7**	**177** (BNP)

BNP, brain natriuretic peptides; M, male; F, female; P_IO_2, inspired oxygen partial pressure; SaO_2, arterial oxygen saturation without oxygenotherapy; PaO_2, arterial oxygen partial pressure without oxygenotherapy; nd, not documented.

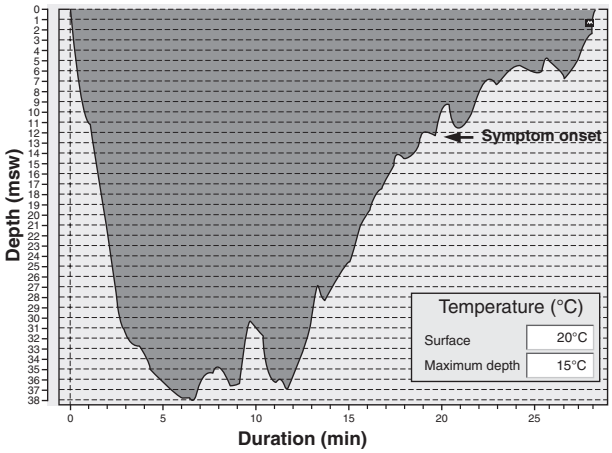

Figure 1 Example of diving profile associated with an occurrence of pulmonary oedema (the arrow indicates the onset of symptoms).

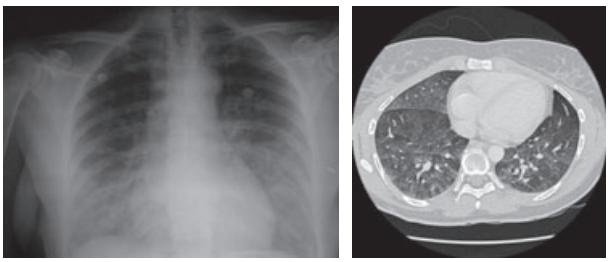

Figure 2 Example of standard radiography and thoracic tomodensitometry carried out less than 6 h after emersion. Imagery evidenced a bilateral alveolar syndrome at the bases.

tolerated. The median mean arterial pressure was 94 mmHg (range: 83–105 mmHg), and median heart rate was 89 beats min^{-1} (range: 60–108 b min^{-1}). At ambient air, the median haemoglobin oxygen saturation was 93% (range: 82–98%), and the median arterial oxygen partial pressure was 69 mmHg (range: 48–90 mmHg). The standard chest radiograph showed a bilateral alveolar damage syndrome mainly localized in the pulmonary bases. CT scan was performed within the first 6 h after emersion. Alveolar-interstitial syndrome was consistent with the diagnosis of pulmonary oedema (Fig. 2). In five patients, an increased troponin plasma level was found within 24 h after emersion, without any ECG abnormality or chest pain. In nine patients, natriuretic peptide levels were considered as reflecting congestive heart failure, but none of them had any abnormal cardiac echocardiogram.

Treatment

The usual treatment included normobaric oxygen therapy. The patients went back home after 24–48 h. None of them required treatment with a diuretic or inotropic agent. As a result of the lack of decompression stop, two patients were treated by hyperbaric oxygen therapy at 2·5 atmospheres absolute during 60 min.

Six months after the accident, clinical status, respiratory function, cardiac echography and exercise tolerance test were normal in all patients.

Discussion

According to the present case series, various stressors such as immersion, cold exposure, hyperoxia and strenuous exercise are probably involved in the physiopathology of immersion pulmonary oedema in healthy subjects. Furthermore, new physiological tracks in relation with the frequent occurrence of symptoms noticed in the last part of the ascent and a higher incidence in women are suggested by this work.

Immersion causes central pooling of blood, which increases venous return and cardiac preload (Arborelius *et al.*, 1972; Hampson & Dunford, 1997). Coldness further enlarges the hemodynamic strains of immersion (Srámek *et al.*, 2000). Indeed, peripheral cold stimulation is common in recreational diving, particularly in deep diving where the elevated ambient pressure limits thermal protection by decreasing the thickness of neoprene clothing. Peripheral body cooling also increases systemic vascular resistance as evidenced by raised arterial diastolic pressures, peripheral resistances and hence left ventricle afterload (Arieli *et al.*, 1997; Mourot *et al.*, 2007). In our study, cold-water immersion was thought to have promoted the occurrence of pulmonary oedema because 15 patients dived in water temperature below 16°C (61°F) and eight complained of the cold despite their wetsuit. Also, the pulmonary capillary pressure further increased during sustained fin swimming (Mahon *et al.*, 2002). Thirteen divers reported the heavy burden of the physical effort they performed before the onset of the respiratory symptoms. Thus, the effects of immersion, body cooling and strenuous exercise probably combined to increase the capillary-alveolar pressure gradient during diving.

Exposure to hyperoxia is usual in divers. In our study, the median dive depth was 37 msw (121 fsw) with a median inspired oxygen partial pressure of 0·99 bar (range: 0·48–1·53 bar). During the inhalation of compressed air at 37 msw depth, the partial pressure of inspired oxygen is equivalent to the surface inhalation of a gas mixture containing 99% of oxygen. In such conditions, heart rate is commonly decreased in parallel with increased parasympathetic activity and a decreased left ventricular contractility (Savitt *et al.*, 1994; Lund *et al.*, 1999). Hyperbaric oxygen conditions also increase systemic vascular resistance through a direct vasoconstrictor effect (Pelaia *et al.*, 1992). Hyperoxic-pulmonary inflammation may also contribute to pulmonary oedema (Shupak *et al.*, 2003). In a recent animal study, hyperoxic breathing at <1·5 atmosphere absolute resulted in diffuse pulmonary damage with an extensive inflammatory response and alveolar-capillary destruction leading to oedema (Demchenko *et al.*, 2007).

In addition, SCUBA divers inhale cold gas because the tank of compressed air is immersed in ambient water, and decompression in the regulator also lowers the stream temperature. Respiratory heat loss in the airway mucosa also increases as a function of the elevated gas density (Burnet *et al.*, 1990). Airway cooling triggers bonchoconstrictor reflexes and mucosal hypersecretion, accentuating the rise in respiratory resistance (Fontanari *et al.*, 1996). The depth-increased ventilatory or panic of certain subjects opposes the regularity of amplified ventilatory movements and may create shear forces in the airway wall (Adir *et al.*, 2004), eliciting mechanical and/or inflammatory injuries of the tracheobronchial and alveolar walls (West & Mathieu-Costello, 1992; Slade *et al.*, 2001). The conspicuous frequency of haemoptysis in our series (41%) as in others (Cochard *et al.*: 59%, Adir *et al.*: 56%) probably reflects a high degree of mechanical strain of capillary-alveolar membrane (Adir *et al.*, 2004; Cochard *et al.*, 2005).

During sustained fin swimming, exercise-induced hyperpnea is superimposed on the mechanical high work of breathing a dense gas mixture and on the obligatory additional load of the SCUBA regulator (Weiler-Ravell *et al.*, 1995; Thorsen *et al.*, 1999). Thus, hyperbaric conditions induce loaded breathing, which requires large airway pressure swings on the adventitial side of the capillary wall (Jammes & Roussos, 1995). As changes in pleural pressure determine airway pressure swings along the breathing cycle, this transmural strain applied to heart walls triggers the release of natriuretic peptides (Nagashima *et al.*, 1995). In healthy trained divers, NT pro-BNP was found increased after one hour of quiet dive, although plasma concentration remained lower than 100 pg ml^{-1} and resumed baseline levels within 24 h (Mourot *et al.*, 2004; Gempp *et al.*, 2005). Conversely, in our series of pathological events, NT pro-BNP were markedly increased beyond the pathological thresholds when the first blood sampling of patients was performed and then returned to normal range within 24–48 h. These results provide strong evidence that high levels of BNP found in divers free of cardiac failure reflected a marked cardiac strain, probably via a combination of sustained exercise with loaded breathing because of diving conditions and an increased ventricular load from both immersion and cooling (Epstein *et al.*, 1989; Ray *et al.*, 1990). In five divers, the cardiac strain resulted in an increase in both troponin and natriuretic peptides. Natriuretic peptides are known to favour plasma fluid extravasation (Curry, 2005). Consequently, plasma levels of natriuretic peptides measured at the end of a scuba dive should provide an overall estimation of the myocardial strains encountered. The quick release of ANP may also participate in the development of oedema, and secondary the later released BNP could extend the process.

The already mentioned factors that might promote pulmonary oedema may be further potentiated by the occurrence of circulatory bubbles, which are always present during decompression. Indeed, venous circulating bubbles are commonly detected after SCUBA dives (Boussuges *et al.*, 2006). Although most of our divers respected decompression procedures, these 'silent circulating bubbles' may be the onset of an inflammatory reaction in vascular endothelium, potentially promoting pulmonary oedema by damaging the alveolocapillar membrane (Zwirewich *et al.*, 1987). Bubbles may have been promoted by

the iterative ascents performed by more than half of our divers. Also, these SCUBA divers underwent a mouth pressure higher than ambient hydrostatic pressure. Whenever the divers are horizontal, the alveolar and capillary pressure gradient should be very similar to normal, and when they are head down, the gradient is reduced. During the ascent, the large venous return persists, while a vertical head up posture imitates head out immersion with its increased capillary pressure relative to alveolar pressure. Superimposed on other factors, this change in pressure regimen may bolster transmural pulmonary hydrostatic forces and favour fluid shift from the pulmonary vasculature into the alveoli during the last metres of the ascent (Lundgren, 1999).

In several studies, about half of the cases of immersion pulmonary oedema occurred in women. Slade *et al.* reported eight cases, five of whom were women with a mean age of 52 years (Slade *et al.*, 2001). In our study, median age is 49 years, and 64% of patients are women. In comparison, the women rate is only 23%, and the mean age is 41 years in the population presenting diving accident. This data can be related to the statistics of the French diving federation (30%, 35 years). Guenette *et al.* observed that breathing work was higher in female than that of male endurance athletes during progressive exercise, because of smaller size of lungs in the former (Guenette *et al.*, 2007). Hopkins *et al.* suggested that smaller lung size could promote interstitial pulmonary oedema (Hopkins *et al.*, 1998). After menopause, loss of the vascular protective effects of the sex hormones may unmask a population of women more prone to hypertension who would incur a higher risk of cardiovascular disease (Harrison-Bernard & Raij, 2000; Reckelhoff, 2001; Khalil, 2005). These hypotheses should be studied in further works.

In summary, cardiopulmonary strain experienced during SCUBA diving is because of common stressors such as water immersion, cold exposure, hyperoxia, increased hydrostatic pressure and strenuous exercise. Furthermore, the present case series underlines new physiological tracks of immersion pulmonary oedema related to the occurrence of symptoms in the last part of the ascent and higher incidence in women. In further studies, careful recordings of subject characteristics as well as detailed circumstances of occurrence should help to better understand the physiopathology of pulmonary oedema in apparently healthy divers.

References

Adir Y, Shupak A, Gil A, Peled N, Keynan Y, Domachevsky L, Weiler-Ravell D. Swimming-induced pulmonary oedema: clinical presentation and serial lung function. *Chest* (2004); **126**: 394–399.

Arborelius M Jr, Ballidin UI, Lilja B, Lundgren CE. Hemodynamic changes in man during immersion with the head above water. *Aerosp Med* (1972); **43**: 592–598.

Arieli R, Kerem D, Gonen A, Goldenberg I, Shoshani O, Daskalovic YI, Shupak A. Thermal status of wet-suited divers using closed circuit O2 apparatus in sea water of 17-18.5 degrees C. *Eur J Appl Physiol* (1997); **76**: 69–74.

Boussuges A, Blanc F, Carturan D. Hemodynamic changes induced by recreational SCUBA diving. *Chest* (2006); **129**: 1337–1343.

Burnet H, Lucciano M, Jammes Y. Respiratory effects of cold gas breathing in humans under hyperbaric environment. *Respir Physiol* (1990); **81**: 413–424.

Cochard G, Arvieux J, Lacour JM, Madouas G, Mongredien H, Arvieux CC. Pulmonary oedema in scuba divers: recurrence and fatal outcome. *Undersea Hyperb Med* (2005); **32**: 39–44.

Curry FE. Atrial natriuretic peptide: an essential physiological regulator of transvascular fluid, protein transport and plasma volume. *J Clin Invest* (2005); **115**: 1458–1461.

Daniels LB, Maisel AS. Natriuretic peptides as diagnostic test: lessons from the first 5 years of clinical application. *Heart Fail Clin* (2006); **2**: 299–309.

Demchenko IT, Welty-Wolf KE, Allen BW, Piantadosi CA. Similar but not the same: normobaric and hyperbaric pulmonary oxygen toxicity, the role of nitric oxide. *Am J Physiol Lung Cell Mol Physiol* (2007); **293**: L229–L238.

Epstein M, Norsk P, Loutzenhiser R. Effects of water immersion on atrial natriuretic peptide release in humans. *Am J Nephrol* (1989); **9**: 1–24.

Fontanari P, Burnet H, Zattara-Hartmann MC, Jammes Y. Changes in airway resistance induced by nasal inhalation of cold dry, dry or moist air in normal individuals. *J Appl Physiol* (1996); **81**: 1749–1753.

Gempp E, Blatteau JE, Louge P, Drouillard I, Galland FM. N-terminal pro brain natriuretic peptide increases after 1-h scuba dives at 10 m depth. *Aviat Space Environ Med* (2005); **76**: 114–116.

Gluecker T, Capasso P, Schnyder P, Gudinchet F, Schaller MD, Revelly JP, Chiolero R, Vock P, Wicky S. Clinical and radiologic features of pulmonary edema. *Radiographics* (1999); **19**: 1507–1531.

Guenette JA, Witt JD, McKenzie DC, Road JD, Sheel AW. Respiratory mechanics during exercise in endurance-trained men and women. *J Physiol* (2007); **581**: 1309–1322.

Hampson NB, Dunford RG. Pulmonary oedema of scuba divers. *Undersea Hyperb Med* (1997); **24**: 29–33.

Harrison-Bernard LM, Raij L. Postmenopausal hypertension. *Curr Hypertens Rep* (2000); **2**: 202–207.

Hopkins SR, Gavin TP, Siafakas NM, Haseler LJ, Olfert IM, Wagner H, Wagner PD. Effect of prolonged, heavy exercise on pulmonary gas exchange in athletes. *J Appl Physiol* (1998); **85**: 1523–1532.

Jammes Y, Roussos Ch. Respiration during diving. In: *The Thorax*, 2nd edn (ed. Roussos, Ch) (1995), pp. 1557–1584. Marcel Dekker, New York.

Khalil RA. Sex hormones as potential modulators of vascular function in hypertension. *Hypertension* (2005); **46**: 249–254.

Lund VE, Kentala E, Scheinin H, Klossner J, Helenius H, Sariola-Heinonen K, Jalonen J. Heart rate variability in healthy volunteers during normobaric and hyperbaric hyperoxia. *Acta Physiol Scand* (1999); **167**: 29–35.

Lundgren CEG. Immersion effects. In: *The Lung at Depth* (eds Lundgren, CEG, Miller, JN) (1999), pp. 91–128. Marcel Dekker, New York.

Mahon RT, Kerr S, Amundson D, Parrish JS. Immersion pulmonary oedema in special forces combat swimmers. *Chest* (2002); **122**: 383–384.

Mourot L, Wolf JP, Galland F, Robinet C, Courtière A, Bouhaddi M, Meliet JL, Regnard J. Short-term vasomotor adjustments to post immersion dehydration are hindered by natriuretic peptides. *Undersea Hyperb Med* (2004); **31**: 203–210.

Mourot L, Bouhaddi M, Gandelin E, Cappelle S, Nguyen NU, Wolf JP, Rouillon JD, Hughson R, Regnard J. Conditions of autonomic reciprocal interplay versus autonomic co-activation: effects on non-linear heart rate dynamics. *Auton Neurosci Basic Clin* (2007); **137**: 27–36.

Nagashima K, Nose H, Yoshida T, Kawabata T, Oda Y, Yorimoto A, Uemura O, Morimoto T. Relationship between atrial natriuretic peptide and plasma volume during graded exercise with water immersion. *J Appl Physiol* (1995); **78**: 217–224.

Pelaia P, Rocco M, Conti G, De Blasi RA, Bufi M, Antonelli M, Bortone C. Hemodynamic changes during hyperbaric oxygen therapy. *J Hyperbaric Med* (1992); **7**: 229–237.

Pons M, Blickenstorfer D, Oechslin E, Hold G, Greminger P, Franzeck UK, Russi EW. Pulmonary oedema in healthy persons during scuba-diving and swimming. *Eur Respir J* (1995); **8**: 762–767.

Ray CA, Delp MD, Hartle DK. Interactive effect of body posture on exercise-induced atrial natriuretic peptide release. *Am J Physiol* (1990); **258**: E775–E779.

Reckelhoff JF. Gender differences in the regulation of blood pressure. *Hypertension* (2001); **37**: 1199–1208.

Savitt MA, Rankin JS, Elberry JR, Owen CH, Camporesi EM. Influence of hyperbaric oxygen on left ventricular contractility, total coronary blood flow and myocardial consumption in the conscious dog. *Undersea Hyperb Med* (1994); **21**: 169–183.

Shupak A, Guralnik L, Keynan Y, Yanir Y, Adir Y. Pulmonary oedema following closed-circuit oxygen diving and strenuous swimming. *Aviat Space Environ Med* (2003); **74**: 1201–1204.

Slade JB, Hattori T, Ray CS, Bove AA, Cianci P. Pulmonary oedema associated with scuba diving: case reports and review. *Chest* (2001); **120**: 1686–1694.

Srámek P, Simecková M, Janský L, Savlíková J, Vybíral S. Human physiological responses to immersion into water of different temperatures. *Eur J Appl Physiol* (2000); **81**: 436–442.

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**: 137–141.

Weiler-Ravell D, Shupak A, Goldenberg I, Halpern P, Shoshani O, Hirschhorn G, Margulis A. Pulmonary oedema and haemoptysis induced by strenuous swimming. *BMJ* (1995); **311**: 361–362.

West JB, Mathieu-Costello O. Stress failure of pulmonary capillaries: role in lung and heart disease. *Lancet* (1992); **340**: 762–767.

Wilmshurst PT, Nuri M, Crowther A, Webb-Peploe MM. Cold-induced pulmonary oedema in scuba divers and swimmers and subsequent development of hypertension. *Lancet* (1989); **1**: 62–65.

Zwirewich CV, Muller NL, Abboud RT, Lepawsky M. Non cardiogenic pulmonary oedema caused by decompression sickness: rapid resolution following hyperbaric therapy. *Radiology* (1987); **163**: 81–82.