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Swimming-Induced Pulmonary Edema*

Clinical Presentation and Serial Lung Function

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Background: Acute pulmonary edema has been noted in swimmers and divers, and has been termed *swimming-induced pulmonary edema* (SIPE). The mechanisms and consequences of SIPE are unknown, and there are currently no series of carefully evaluated patients with this condition. Herein we report the clinical presentation, incidence of recurrence, findings on physical examination, chest radiography, and oxygen saturation in 70 trainees with a diagnosis of SIPE. We also report the results of forced spirometry in a subgroup of 37 swimmers.
Methods: SIPE was diagnosed when severe shortness of breath and cough were reported during or after swimming, and were associated with evidence of pulmonary edema. During the years from 1998 to 2001, 70 cases of SIPE were documented in young healthy male subjects participating in a fitness-training program. Physical examination and pulse oximetry were performed immediately. Chest radiographs were obtained in all cases 12 to 18 h following onset of symptoms. In 37 swimmers, spirometry was performed at the time of chest radiography and again after 7 days.
Results: All subjects complained of severe shortness of breath. Sixty-seven of the 70 subjects (95.7%) had a prominent cough; in 63 subjects (90%), there was significant sputum production. Hemoptysis was observed in 39 subjects (55.7%). Mean arterial oxygen saturation after swimming was 88.4 ± 6.6% breathing air, compared with 98 ± 1.7% breathing air at rest before the start of the swimming trial (mean ± SD) [p < 0.001]. Chest radiographs obtained 12 to 18 h after swimming were normal in all cases. Sixteen trainees (22.9%) had a recurrence of SIPE. Spirometry demonstrated restrictive lung function, which persisted for a week.
Conclusions: In our trainee population, SIPE is a not uncommon, often recurrent phenomenon that significantly influences performance. It is not clear what predisposes to its occurrence or recurrence and what, if any, are its long-term effects.   *(CHEST 2004; 126:394–399)*

Key words: diving; exercise; pulmonary edema; swimming

Abbreviations: BGB = blood-gas barrier; HAPE = high-altitude pulmonary edema; SIPE = swimming-induced pulmonary edema

To fulfil its function, the blood-gas barrier (BGB) in the mammalian lung has to satisfy two conflicting requirements. It has to be very thin, to allow adequate gas exchange by passive diffusion, and must at the same time be strong enough to withstand high capillary pressures during exercise to avoid stress-induced mechanical failure.[1] During maximal exercise in normal subjects, the capillary pressure at the base of the lung has been estimated to exceed 25 mm Hg, a level that exerts great stress on the capillary endothelium.[1–4] Indeed, it is well known that some thoroughbred racehorses may have alveolar bleeding during maximal exercise. This bleeding has been attributed to a mean pulmonary artery pressure and pulmonary capillary pressures of up to 120 mm Hg and 100 mm Hg, respectively.[5–7] Animal studies[8–10] have shown that the elevated capillary pressure results in structural damage to the BGB, including disruption of areas of the capillary endothelium, basement membrane, and epithelium. These changes diminish alveolar septal barrier function and allow for the development of high-permeability pulmonary edema.

The prevalence of exercise-induced pulmonary

edema in humans is unknown and is probably underreported. Hopkins et al[11] demonstrated in elite human athletes that brief intense exercise results in alteration of the BGB, with increased RBC and protein concentrations in BAL fluid.

There are a number of reports[12–16] describing the occurrence of acute pulmonary edema and hemoptysis in swimmers and divers. We previously reported a series of eight swimmers with pulmonary edema and hemoptysis during a single swimming time trial.[12] In that particular event, overhydration was thought to be an important contributing factor. However, recurrent episodes in two of the swimmers and other new cases associated with strenuous swimming occurred without antecedent fluid loading. During the years from 1998 to 2001, we diagnosed a further 70 cases of swimming-induced pulmonary edema (SIPE) in young, healthy male subjects participating in a fitness-training program. We now report on the clinical presentation, incidence of recurrence, findings on physical examination, chest radiography, and oxygen saturation in those cases. We also report the results of serial lung function (forced spirometry) in a subgroup of 37 of the 70 subjects.

Materials and Methods

We prospectively examined participants in a swimming fitness-training program. The subjects were healthy, nonsmoking men aged 18 to 19 years. All were in good physical condition, and periodically took part in a swimming time trial over 2.4 to 3.6 kilometers in the open sea. The swimming trials were conducted while the sea was calm, and the average duration of the trial was usually between 30 min and 45 min. Subjects swam in the supine position wearing only a bathing suit, and using swim fins in a semi-reclining or half-sitting posture, as required for this type of activity. The average water temperature was $19.6 \pm 3.2°C$. The trainees were examined before the swimming trial, and were found to be in a good state of health with a normal physical examination. Arterial oxygen saturation measured by transcutaneous pulse oximetry while breathing room air and at rest was normal ($98 \pm 1.7\%$) in all trainees (Propaq model 104EL; Protocol Systems; Beaverton, OR).

SIPE was diagnosed when (1) severe shortness of breath and coughing were reported during or after swimming in the absence of seawater aspiration, and (2) evidence of pulmonary edema was found on physical examination (bilateral inspiratory crackles that did not clear with deep inspiration). Immediately on diagnosis, a complete physical examination was conducted with special emphasis on the respiratory system, and arterial oxygen saturation was measured again. A chest radiograph was obtained 12 to 18 h after the onset of symptoms. A comprehensive interview of each subject by the examining physician included a direct question as to whether there was subjective evidence of seawater aspiration.

In a subgroup of 37 trainees, spirometry (ST-250; Fukuda Sangyo; Chiba, Japan) was performed 12 to 18 h after SIPE was diagnosed, and again 7 days later. Before testing, the spirometer was calibrated according to the recommendations of the American Thoracic Society.[17] FVC and FEV_1 were measured, and the FEV_1/FVC ratio was calculated. The best result of at least three tests was recorded. Forced spirometry data obtained for all subjects in a similar fashion 12 months previously, at the time of selection for the program, were available for comparison. Arterial oxygen saturation was measured at rest before the swim and immediately after diagnosis. Comparison among groups was done by analysis of variance using SAS 6.03 (SAS Institute; Cary, NC).

Results

Seventy cases of SIPE were diagnosed during a 3-year period (Table 1), an incidence of 1.8% for all the swimming trials performed by the trainees. All 70 subjects complained of severe shortness of breath, 67 subjects (95.7%) had a prominent cough, and 63 subjects (90%) had sputum production. Pink froth or hemoptysis was observed in 39 subjects (55.7%). Six subjects (8.6%) complained of substernal chest pain. Although 13 subjects (18.8%) reported a considerable amount of seawater in their mouths during swimming, when directly questioned they all denied overt aspiration. Sixty-four subjects (91.4%) had basal inspiratory crackles on chest auscultation, and 6 subjects (8.6%) had expiratory wheezing (Table 1). The mean oxygen saturation was low after the swimming exercise, averaging $88.4 \pm 6.6\%$ breathing air compared with $98 \pm 1.7\%$ before the start of the swimming trial (p < 0.001) [Fig 1]. Findings of chest radiographs obtained 12 to 18 h after the onset of symptoms were normal in all cases. The trainees were treated by oxygen via a breathing mask with a reservoir, and some were treated with IV furosemide. All symptoms and evidence of pulmonary edema, including hypoxemia, resolved within 24 h. During the 3 years of the study, 16 trainees (22.9%) had a recurrence of SIPE. In all cases, this took place at least 3 months after the first incident.

In 37 randomly selected trainees with SIPE, we were able to perform spirometry as they left the water. We found absolute reductions in the mean values of FVC and FEV_1 of 360 mL and 430 mL, respectively. These changes represent a reduction of 7.1% and 8.8% from pre-exercise levels, which were on record from routine testing, suggestive of an acute restrictive ventilatory process. This pattern of

Table 1—Symptoms, Signs, and Clinical Findings in 70 Trainees With SIPE

Symptoms and Signs	Subjects, No. (%)
Dyspnea	70 (100)
Cough	67 (95.7)
Hemoptysis	39 (55.7)
Sputum production	63 (90)
Chest pain	6 (8.6)
Basal inspiratory crackles	64 (91)
Wheezing	6 (8.6)
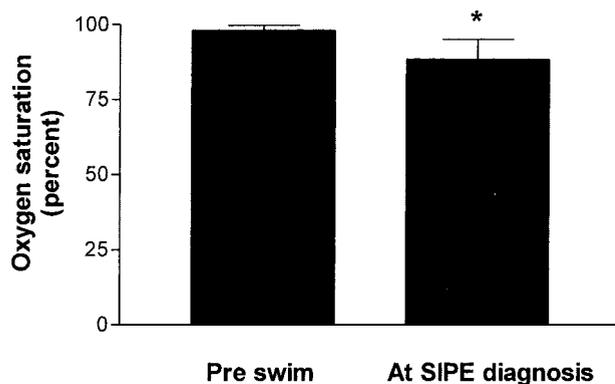
FIGURE 1. Mean oxygen saturation for the 70 trainees at rest before the swim (Pre), and immediately on diagnosis of SIPE breathing air. All data are mean ± SD. *, p < 0.001.

restrictive lung function persisted for a week (Table 2). Echocardiographic findings were normal 7 days after SIPE was diagnosed in 20 of the 37 trainees in whom it was performed. The arterial oxygen saturation by pulse oximetry in this subgroup of 37 trainees was significantly reduced from pre-exercise levels, averaging 76 ± 7.8% breathing air. The oxygenation improved to 96.2 ± 5.4% while breathing 100% oxygen via a nonrebreathing mask (p < 0.01).

Discussion

The occurrence of exercise-induced pulmonary hemorrhage in galloping thoroughbred racehorses is a well-known phenomenon[18] that has been attributed to extremely high pulmonary vascular pressures. Alveolar hemorrhage in this setting is most likely due to stress failure of the pulmonary capillaries.[5–10]

In contrast with the findings in animal studies, such events are very rarely reported in athletes during or after intense exercise. Previous reports[19,20] described acute pulmonary edema or recurrent hemoptysis in a rugby player and two marathon runners following extreme physical exertion. Another study[11]

Table 2—Results of Spirometry Performed in 37 Trainees 6 to 12 Hours After the Diagnosis of SIPE and 1 Week Later, and the Comparison With Baseline Spirometry Data*

Spirometry	Baseline	6 to 12 h	1 wk
FVC, L	5.08 ± 0.556	4.72 ± 0.472†	4.89 ± 0.467
FEV$_1$, L	4.43 ± 0.576	4.04 ± 0.444‡	4.14 ± 0.410‡
FEV$_1$/FVC, %	87.7 ± 5.92	86.4 ± 6.47	85.1 ± 5.40

*Data are presented as mean ± SD.
†p < 0.01.
‡p < 0.003.

has shown increased concentrations of RBCs and protein in BAL fluid obtained 1 h after a simulated cycling race, suggesting that intense exercise reduces alveolar barrier function. No activation of proinflammatory pathways was found in the airspaces, supporting the hypothesis that capillary stress failure is responsible for these changes. In a follow-up study from this same group, it was demonstrated that short-term maximal physiologic stress is needed to alter the integrity of the BGB, because sustained submaximal exercise did not result in stress failure of the pulmonary capillaries.[21]

Caillaud et al[22] demonstrated exercise-induced hypoxemia, widening of the alveolar-arterial oxygen pressure difference, and reduced diffusion capacity for carbon monoxide in highly trained athletes after a triathlon. Increases in lung density and in the number of opacities, suggesting an increase in pulmonary extravascular fluid, were diagnosed by CT of the chest in these athletes.

The relatively frequent occurrence of swimming-induced or immersion-related pulmonary congestion in healthy individuals, in comparison with the apparent rarity of the phenomenon following nonimmersion exercise, is intriguing. There are several important physiologic effects of immersion, which might explain this difference. Immersion increases cardiac preload and pulmonary arterial pressure due to central blood pooling. It has been shown that during immersion in 33 to 35°C water, central blood volume increases by approximately 700 mL and pulmonary arterial transmural pressure by 13 to 21 mm Hg.[23,24] Furthermore, immersion in moderately cold water (17 to 18°C) is associated with a decrease in body core temperature resulting in redistribution of blood from peripheral to thoracic vessels. Hypothermia-induced arterial and venous constriction leads to an increase in both cardiac preload and afterload.[25–27] Previous reports[13,28–31] have noted pulmonary edema in cold water divers. The subjects examined in these studies had abnormal cold pressor tests, a finding which was not corroborated by other investigators. In our subjects, the average water temperature was 19.6 ± 3.2°C, which can cause peripheral vasoconstriction, a rise in central blood volume, and increased cardiac preload and afterload.

A reduction in functional residual capacity due to blood displacement into the lungs, combined with an immersion-induced increment in the closing volume, results in ventilation-perfusion inequality, hypoxemia, and pulmonary vasoconstriction.[32] Under these conditions, because pulmonary vasoconstriction is not uniform throughout the vascular bed, those capillaries not protected by arterial constriction are exposed to the high pulmonary pressures that develop during strenuous swimming. This,

according to the model of West and Mathieu-Costello,[33] facilitates pulmonary capillary stress failure.

Apart from the circumferential tension caused by the capillary transmural pressure, other forces also act on the BGB, such as the longitudinal tension in the alveolar wall elements associated with lung inflation. Immersion increases the pulmonary closing volume and reduces lung compliance, thus leading to air trapping.[34,35]

During head-out immersion, negative pressure respiration due to the hydrostatic pressure differences between the upper and lower airways produces transmural pulmonary hydrostatic forces that favor a fluid shift from the pulmonary vasculature to the alveoli.[36] The more the chest sinks under water and the steeper its vertical tilt, the greater the effect. High lung volumes and peak airway pressures, both present during extreme effort, were found to cause an increase in capillary permeability and the development of microvascular injury.[37,38] This effect is definitely present when swimming in the semi-reclining or half-sitting position, as practiced by the study participants. Excessive negative intrathoracic pressure generated by arduous exertion, causing increased tension on the alveolar walls, is another contributing factor to mechanical failure of the BGB.

We postulate that in our subjects, who were engaged in strenuous swimming, all of these physiologic changes combined to produce damage to the alveolar-capillary membrane, resulting in clinically significant pulmonary edema. Cough with prominent sputum production was noted in most of our subjects, and hemoptysis was present in approximately half of them. These clinical manifestations, in parallel with a significant reduction in oxygen saturation and physical findings on lung auscultation, support the diagnosis of pulmonary edema. Clinical recovery was rapid, with an excellent response to diuretics and oxygen. The chest radiograph reverted to normal as soon as 12 h after the incident.

The clinical manifestations we describe may be found in seawater aspiration, apart from hemoptysis, which is usually seen in fresh water drowning. The presence of hemoptysis supports our preference for stress failure of the pulmonary capillaries as the etiology of the syndrome we describe. Furthermore, we found no reference in the English-language literature to clinically significant aspiration of seawater in professional swimmers in a nondrowning situation. The possibility of our swimmers inadvertently aspirating seawater, an event which induces immediate and violent coughing, without being aware of this, is highly unlikely, whereas the respiratory distress they experienced, cough, shortness of breath, and effort intolerance, developed gradually during the swim.

The rapid clinical improvement with resolution of the pulmonary edema is consistent with experimental animal studies,[8] which demonstrate that the disruptions of the BGB are rapidly reversible with reduction of the capillary pressures. These findings are borne out by human case studies.[39,40] A recent study[41] in elite athletes investigated the effect of maximal exercise on the integrity of the alveolar epithelial membrane, using the clearance rate of aerosolized ^{99m}Tc diethylenetriamine penta-acetic acid as an index for the permeability of the lung BGB. The authors demonstrated an acute increase in pulmonary clearance after maximal exercise, which returned to resting levels within 2 h.

The fact that SIPE is not seen or has not been reported in Olympic or other competitive swimmers requires an explanation. It is possible to discern a number of differences between competitive swimming and the exercise performed by our trainees. Swimming with fins exercises the legs only, compared with other competitive swimming styles, in which cardiac output supports both arm and leg movement. Venous return must be greatly augmented by finning compared with other swimming styles, and by the effects of immersion in the supine, semi-reclining position, as described in detail above. The temperature of the water in which these trainees swam was also significantly lower than the thermo-regulated conditions in competitive swimming, inducing vasoconstriction and an increase in pulmonary vascular pressures. This combination of swimming style and a distinctive posture in cold water to produce an increase in venous return and pulmonary artery pressure could provide a partial explanation for the frequency with which we observed SIPE.

As noted above, the high incidence of hemoptysis in our trainees, which is not a characteristic clinical sign of seawater aspiration, supports our notion that mechanical failure of the BGB was the main contributing factor to the occurrence of pulmonary edema. Moreover, a recent report[39] described unilateral right-sided pulmonary edema in three combat divers who swam right-side down. The increase in pulmonary capillary pressures due both to increased regional pulmonary blood flow with high exertion, exacerbated by the gravitational effect, and the hydrostatic pressure differences between the upper airways and the submerged right lung, led to capillary stress failure and unilateral pulmonary edema.

High-altitude pulmonary edema (HAPE) is another form of noncardiogenic pulmonary edema, which shares a certain similarity with SIPE. In both

cases, there is increased pulmonary artery pressure and increased permeability of the BGB.[16,42] As is the case with SIPE, severe HAPE is infrequent. People with higher pulmonary artery pressure, smaller lungs, and higher pulmonary artery wedge pressure during exercise at sea level are prone to acquire HAPE.[42] Unfortunately, we were unable to elicit any physical findings or behavioral characteristics that might help us predict which of our trainees are at risk of acquiring SIPE.

Lung function tests during immersion reveal a reduction in vital capacity, functional residual capacity and lung compliance, and an increase in the closing volume. Studies[43] on the effects of acute pulmonary edema on lung function tests describe restrictive and obstructive dysfunction, with a decrease in FVC and FEV_1, and a low FEV_1 to FVC ratio.

In our subgroup of 37 trainees, who performed pulmonary function tests, significant restrictive changes were found 6 to 12 h after the acute event, with a persistent reduction in FEV_1 and a normal FEV_1/FVC ratio 1 week later. These restrictive changes support the clinical diagnosis of pulmonary edema. Their persistence for at least a week, despite complete clinical recovery and normal chest radiographic findings within 24 h, suggests that notwithstanding the experimental data indicating rapid repair of the breaks in the BGB, there is still some degree of pulmonary congestion and perhaps also a loss of capillary integrity. In 20 subjects, no cardiac abnormality was found on echocardiography, supporting the concept that the pulmonary edema results from capillary stress failure and is not cardiogenic in origin.

In summary, shortness of breath, coughing, and hemoptysis during strenuous swimming are related to hypoxemia and pulmonary edema, most probably due to mechanical failure of the BGB as a result of high transmural capillary pressure. We speculate that the physiologic effects of immersion, swimming in the semi-supine position with the head and shoulders above water, thus increasing the pressure difference between the lower extremities and the thorax, and the relatively cold water temperature, all serve to increase the risk of exercise-induced pulmonary edema. In our trainee population, SIPE is a not uncommon, often recurrent phenomenon that significantly influences performance. It is not clear what predisposes to its occurrence or recurrence and what, if any, are its long-term effects.

<div align="center">REFERENCES</div>

1 West JB, Tsukimoto K, Mathieu-Costello O, et al. Stress failure in pulmonary capillaries. J Appl Physiol 1991; 70: 1731–1742

2 Reeves JT, Groves BM, Cymerman A, et al. Operation Everest II: cardiac filling pressures during cycle exercise at sea level. Respir Physiol 1990; 80:147–154

3 Wagner PD, Gale GE, Moon RE, et al. Pulmonary gas exchange in humans exercising at sea level and simulated altitude. J Appl Physiol 1986; 61:260–270

4 West JB, Mathieu-Costello O. Strength of the pulmonary blood-gas barrier. Respir Physiol 1992; 88:141–148

5 Groves BM, Reeves JT, Sutton JR, et al. Operation Everest II: elevated high-altitude pulmonary resistance unresponsive to oxygen. J Appl Physiol 1987; 63:521–530

6 Erickson BK, Erickson HH, Coffman JR. Pulmonary artery, aortic and oesophageal pressure changes during high intensity treadmill exercise in the horse: a possible relation to exercise-induced pulmonary haemorrhage. Equine Vet J 1990; 9(suppl):47–52

7 Manohar M. Pulmonary artery wedge pressure increases with high-intensity exercise in horses. Am J Vet Res 1993; 54:142–146

8 Elliott AR, Fu Z, Tsukimoto K, et al. Short-term reversibility of ultrastructural changes in pulmonary capillaries caused by stress failure. J Appl Physiol 1992; 73:1150–1158

9 Shupak A, Weiler-Ravell D, Adir Y, et al. Pulmonary oedema induced by strenuous swimming: a field study. Respir Physiol 2000; 121:25–31

10 Tsukimoto K, Mathieu-Costello O, Prediletto R, et al. Ultrastructural appearances of pulmonary capillaries at high transmural pressures. J Appl Physiol 1991; 71:573–582

11 Hopkins SR, Schoene RB, Henderson WR, et al. Intense exercise impairs the integrity of the pulmonary blood-gas barrier in elite athletes. Am J Respir Crit Care Med 1997; 155:1090–1094

12 Weiler-Ravell D, Shupak A, Goldenberg I, et al. Pulmonary oedema and haemoptysis induced by strenuous swimming. BMJ 1995; 311:361–362

13 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:62–65

14 Pons M, Blickenstorfer D, Oechslin E, et al. Pulmonary oedema in healthy persons during scuba-diving and swimming. Eur Respir J 1995; 8:762–767

15 Hampson NB, Dunford RG. Pulmonary edema of scuba divers. Undersea Hyperb Med 1997; 24:29–33

16 Slade JB Jr, Hattori T, Ray CS, et al. Pulmonary edema associated with scuba diving: case reports and review. Chest 2001; 120:1686–1694

17 American Thoracic Society. Standardization of spirometry, 1994 update. Am J Respir Crit Care Med 1995; 152:1107–1136

18 West JB, Mathieu-Costello O, Jones JH, et al. Stress failure of pulmonary capillaries in racehorses with exercise-induced pulmonary hemorrhage. J Appl Physiol 1993; 75:1097–1109

19 McKechnie JK, Leary WP, Noakes TD, et al. Acute pulmonary oedema in two athletes during a 90-km running race. S Afr Med J 1979; 56:261–265

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

21 Hopkins SR, Schoene RB, Henderson WR, et al. Sustained submaximal exercise does not alter the integrity of the lung blood-gas barrier in elite athletes. J Appl Physiol 1998; 84:1185–1189

22 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; 79:1226–1232

23 Arborelius M Jr, Balldin UI, Lilja B, et al. Hemodynamic

changes in man during immersion with the head above water. Aerosp Med 1972; 43:592–598

24 Moon RE, Baek PS, Lanzinger MJ, et al. Pulmonary hemodynamics and gas exchange at 40 m [abstract]. Undersea Hyperb Med 1998; 25(suppl):26

25 Kurss DI, Lundgren CEG, Pasche AJ. Effect of water temperature and vital capacity in head-out immersion. In: Bachrach AJ, Matzen MM, eds. Underwater physiology VII: Proceedings of the Seventh Symposium on Underwater Physiology. Bethesda, MD: Undersea Medical Society, 1981; 297–301

26 Wolff AH, Coleshaw SRK, Newstead CG, et al. Heat exchanges in wet suits. J Appl Physiol 1985; 58:770–777

27 Arieli R, Kerem D, Gonen A, et al. Thermal status of wet-suited divers using closed circuit O₂ apparatus in sea water of 17–18.5°C. Eur J Appl Physiol 1997; 76:69–74

28 Roeggla M, Roeggla G, Seidler D, et al. Self-limiting pulmonary edema with alveolar hemorrhage during diving in cold water [letter]. Am J Emerg Med 1996; 14:333

29 Lundgren CEG, Pasche AJ. Physiology of diving: immersion effects. In: Shilling CW, Carlston CB, Mathias RA, eds. The physician's guide to diving medicine. New York, NY: Plenum Press, 1984; 86–99

30 Mebane GY. Hypothermia. In: Bove AA, ed. Bove and Davis' diving medicine. 3rd ed. Philadelphia, PA: W.B. Saunders Company, 1997; 207–215

31 O'Keeffe KM. Non-cardiogenic pulmonary edema from accidental hypothermia: a case report. Colo Med 1980; 77:106–107

32 Cotes JE, Reed JW, Elliott C. Breathing and exercise requirements of the work place. In: Whipp BJ, Wasserman K, eds. Exercise: pulmonary physiology and pathophysiology. Lung biology in health and disease (vol 52). New York, NY: Marcel Dekker, 1991; 495–548

33 West JB, Mathieu-Costello O. Stress failure of pulmonary capillaries as a limiting factor for maximal exercise. Eur J Appl Physiol 1995; 70:99–108

34 Bondi KR, Young JM, Bennett RM, et al. Closing volumes in man immersed to the neck in water. J Appl Physiol 1976; 40:736–740

35 Dahlback GO, Jonsson E, Liner MH. Influence of hydrostatic compression of the chest and intrathoracic blood pooling on static lung mechanics during head-out immersion. Undersea Biomed Res 1978; 5:71–85

36 Nuckols ML, Tucker WC, Sarich AJ. Life support systems design: diving and hyperbaric applications. Needham Heights, MA: Simon & Schuster Custom Publishing, 1996

37 Parker JC, Townsley MI, Rippe B, et al. Increased microvascular permeability in dog lungs due to high peak airway pressures. J Appl Physiol 1984; 57:1809–1816

38 Fu Z, Costello ML, Tsukimoto K, et al. High lung volume increases stress failure in pulmonary capillaries. J Appl Physiol 1992; 73:123–133

39 Lund KL, Mahon RT, Tanen DA, et al. Swimming-induced pulmonary edema. Ann Emerg Med 2003; 41:251–256

40 Shupak A, Guralnik L, Keynan Y, et al. Pulmonary edema following closed-circuit oxygen diving and strenuous swimming. Aviat Space Environ Med 2003; 74:1201–1204

41 Hanel B, Law I, Mortensen J. Maximal rowing has an acute effect on the blood-gas barrier in elite athletes. J Appl Physiol 2003; 95:1076–1082

42 Cremona G, Asnaghi R, Baderna P, et al. Pulmonary extravascular fluid accumulation in recreational climbers: a prospective study. Lancet 2002; 359:303–309

43 Khan KA, Jalal S, Jan VM, et al. Pulmonary function profile in chronic congestive heart failure and the effect of ipratropium bromide. Indian Heart J 2000; 52:297–300

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