PULMONARY ASPECTS OF EXERCISE AND SPORTS

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Abstract

Although the lungs are a critical component of exercise performance, their response to exercise and other environmental stresses is often overlooked when evaluating pulmonary performance during high workloads. Exercise can produce capillary leakage, particularly when left atrial pressure increases related to left ventricular (LV) systolic or diastolic failure. Diastolic LV dysfunction that results in elevated left atrial pressure during exercise is particularly likely to result in pulmonary edema and capillary hemorrhage. Data from race horses, endurance athletes, and triathletes support the concept that the lungs can react to exercise and immersion stress with pulmonary edema and pulmonary hemorrhage. Immersion in water by swimmers and divers can also increase stress on pulmonary capillaries and result in pulmonary edema. Swimming-induced pulmonary edema and immersion pulmonary edema in scuba divers are well-documented events caused by the fluid shifts that occur with immersion, elevated pulmonary venous pressure during extreme exercise, and negative alveolar pressure due to inhalation resistance. Prevention strategies include avoiding extreme exercise, avoiding over hydration, and assuring that inspiratory resistance is minimized.

Introduction

All forms of exercise involve an increase in whole body oxygen consumption, therefore, the cardiopulmonary system becomes a major focus for functional changes during exercise. Numerous studies on the physiology of exercise have demonstrated a clear relationship between oxygen consumption (VO_2) heart rate and cardiac output, ventilatory rate and minute ventilation, energy metabolism, and lactate production.[1,2] These relationships have been well defined for several decades. Recently, however, interesting effects of exercise involving swimming and water immersion and extreme aerobic exercise have provided new insights into lung function in various environments and exercise exposures.

Circulatory and Respiratory Changes with Exercise

Ventilatory responses to exercise are well defined.[2] Minute ventilation increases linearly with VO_2 until it approaches the anaerobic threshold, when ventilation rate increases to compensate for the metabolic acidosis created by excess lactate production.[3] Cardiac output also increases linearly with VO_2, but heart rate response, while linearly related to VO_2 at submaximal exercise, increases disproportionately at high workloads as stroke volume falls. Recent data indicate that an important limiting factor to aerobic exercise is a rising pulmonary venous pressure at high exercise loads that causes interstitial fluid accumulation and reduced oxygen transport (Figure 1). The elevated pulmonary venous pressure is directly related to left ventricular (LV) end diastolic pressure that increases at high levels of exercise as the ventricle fails to relax adequately during diastole.[4] The relationship of LV diastolic function to exercise-induced pulmonary congestion has been elucidated in several articles which show that diastolic relaxation is greater in high-capacity aerobic athletes and enhances their ability to withstand very high cardiac outputs during maximal exercise while preserving a low pulmonary venous pressure.[4,5] Recent studies demonstrate a training effect on LV diastolic relaxation, indicating that high levels of aerobic exercise are possible because of improved diastolic relaxation in well-trained athletes.[6,7]

Exercise-Induced Pulmonary Hemorrhage

Veterinary observations in high-performing race horses documented pulmonary hemorrhage and pulmonary edema in some horses racing at very high speeds. West et al. examined one horse prone to exercise-induced pulmonary hemorrhage (EIPH) with measures of pulmonary pressure during exercise on a treadmill (Figure 2) and postmortem microscopy to determine the source of lung edema and hemorrhage.[8,9] They found clear evidence

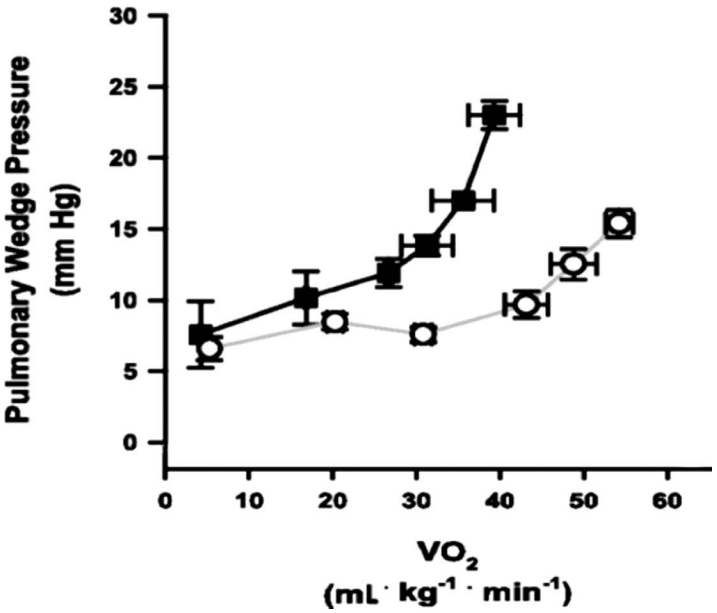

Figure 1. Pulmonary capillary wedge pressure (PCWP) measured during increasing exercise intensity, determined by oxygen consumption (VO_2), in well-trained (circles) and average-trained (squares) men with a mean age of 29.6. Note that the well-trained subjects preserved normal levels of PCWP until they reached high levels of exercise, while the average-trained men demonstrated increases in PCWP at lower levels of exercise intensity. Adapted from Stickland et al.[4]

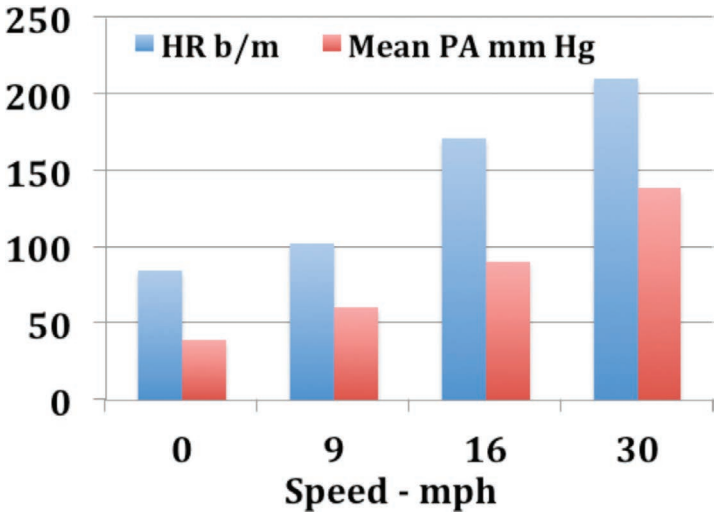

Figure 2. Horse with exercise-induced pulmonary hemorrhage. Heart rate (HR) and mean pulmonary pressure (PA mm Hg) from a horse with exercise-induced pulmonary hemorrhage running on a treadmill.[8]

of interstitial hemorrhage and fluid accumulation following a high workload in one horse that developed EIPH. West et al.[9] suggested that two factors contributed to this problem in horses: (1) They were likely to have a congenital weakness of pulmonary capillaries that resulted in capillary rupture at high pulmonary venous and capillary pressure, and (2) they developed high pulmonary venous pressure due to failure of the left ventricle with subsequent elevation of LV end diastolic pressure during high workloads.

Human studies of lung responses to high workloads[10] corroborate the studies of West et al. to some extent. Zavorsksy et al.[11] studied individuals under several different workloads and performed lung imaging to document the presence or absence of lung edema. Radiographic image readers were blinded to the exposures and reported visual evidence of lung fluid. In individuals undergoing a diagnostic graded exercise test, no evidence of lung edema was noted. However, 15% of individuals who ran on a treadmill at 70% of maximum capacity for 2 hours demonstrated evidence of pulmonary edema, as did 65% of those who ran at maximum capacity for 7 minutes. Similar findings were noted in female athletes.[12] Pingitore et al. examined 48 athletes before and after completing an iron man triathlon. They used ultrasound to detect lung edema and reported the incidence of ultrasound lung comets.[13] None of the athletes had evidence of lung edema before the event, while 75% showed evidence of pulmonary edema immediately post-race, and 42% had persistent findings of pulmonary edema 12 hours post-race. Their data and several case reports[14-16] have demonstrated that extreme exercise can result in pulmonary edema and support the findings of West et al.[9]

Swimming-Induced Pulmonary Edema

Development of pulmonary edema while participating in competitive swimming events has increased in frequency over the past decade due, in part, to increased participation in triathlon events.[17] Shupak et al. described the development of acute pulmonary edema in healthy, highly trained military divers who were swimming in a long-distance competitive swim.[18] All were exercising at a high workload when they developed dyspnea and cough associated with production of pink frothy sputum.[19] Subsequent cardiac evaluation demonstrated normal cardiac function. Since that report, numerous other reports of swimmers who developed pulmonary edema while swimming competitively have emerged.[20-22] All of the military swimmers reported by Schupak et al.[18] had hydrated excessively prior to swimming to avoid dehydration during their long exposure. Miller et al. reported on 70 subjects seen in their emergency department for treatment of swimming-induced pulmonary edema (SIPE).[22] Significant factors found to correlate with SIPE included a history of hypertension, use of a wet suit for thermal protection, and swimming longer than one mile. Age greater than 50 years old and female gender trended toward significance in relating to SIPE. Of interest in their study was the high number of patients who were ingesting fish oil.[22]

Wilmshurst et al.[23] described a group of swimmers and divers who developed pulmonary edema while immersed (IPE) at rest or with very low exercise activity. Symptoms included sudden onset of dyspnea and cough associated with pink frothy sputum. They compared the edema-prone individuals to a group of age- and exposure-matched individuals who had never experienced IPE.[23] Individuals prone to IPE demonstrated increased blood pressure reactivity to cold, oxygen breathing, and exercise compared to the matched control group (Figure 3). The group[23] hypothesized that the IPE-prone individuals develop hypertension and elevated LV end diastolic pressure when exposed to cold, increased oxygen intake, and exercise, although more recent studies show that cold exposure is not a prerequisite for developing IPE.[24,25] A unique factor related to immersion in both swimming and diving is the well-documented fluid shift that occurs due to pressure effects on venous blood pooling. Hong et al.[26] demonstrated a 600- to 700-mL shift of blood from the venous system into the central circulation when immersed to the neck, which in turn increases lung vascular volume and likely contributes to the development of pulmonary edema. This fluid shift can aggravate existing chronic conditions that compromise cardiac and pulmonary function. Carter et al.[27] used quantitative measures of lung computed-tomographic density to identify anatomic correlates in individuals who were prone to IPE or high-altitude pulmonary edema (HAPE). They measured lung tissue density, total lung mass, and the thickness

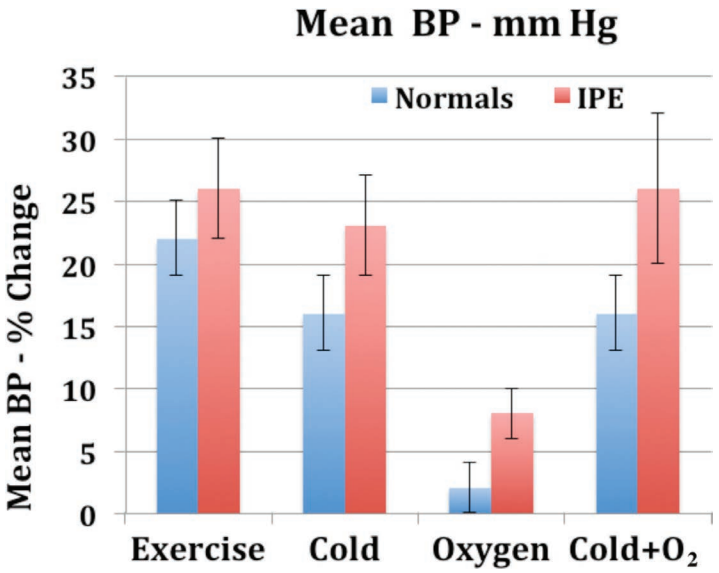

Figure 3. Changes in mean arterial blood pressure after exposure to exercise, cold, and increased inspired oxygen in a group of divers who experienced immersion pulmonary edema (IPE) and a group of similarly exposed divers who did not experience IPE. The IPE-prone subjects demonstrated increased blood pressure responses to all three stimuli compared to the subjects who did not experience IPE with immersion.[23]

Comorbidities in Divers with Immersion Pulmonary Edema
Age
Asthma
Atrial fibrillation
Coronary disease
Cardiomegaly
Cardiomyopathy
Diabetes
Hyperlipidemia
Hypertension
Left ventricular hypertrophy
Obesity
Obstructive sleep apnea
Peripheral arterial disease
Valvular heart disease

Table 1. Comorbidities found in a group of divers who developed Immersion pulmonary edema.[29]

of interlobular septae. Edema-prone individuals demonstrated a lower total lung tissue mass and thinner lung septae. Gempp et al.[28] found that individuals prone to IPE demonstrated a high incidence of hypertension, and Peacher et al.[29] reported a variety of common clinical comorbidities in 70% of individuals who showed evidence of IPE while diving (Table 1).

There have been reports[30-32] describing the development of pulmonary edema when an individual inhales against a closed glottis (negative pressure pulmonary edema). The sharp fall in intrathoracic and intra-alveolar pressure increases the pressure gradient across the pulmonary capillary wall and can result in fluid and blood entering the interstitial and alveolar spaces. This is seen primarily in postanesthesia care where this type of dynamic situation can arise.[31,32] However, a similar pressure change can occur with minor airway restriction that might be found in a mild asthmatic who is exercising at high workload, or in divers who are breathing through a device that adds inspiratory resistance—which, for example, can occur with a faulty diving regulator. Toumpanakis et al.[33] studied rats with restricted airways and found a significant negative intrapulmonary pressure that resulted in interstitial and alveolar edema and evidence of inflammatory injury to lung parenchyma within 2 hours of exposure to the increased inspiratory resistance.

Breath-Hold Diving

The earliest experience of diving involved swimming underwater while breatholding.[34] The "Ama" (or "sea woman") divers of the western Pacific are excellent examples of a centuries-old tradition of commercial diving using single-breath descents to harvest food.[35] While this form of underwater exposure has traditionally been used for working for brief periods of time, the development of breathing support using either self-contained or surface-supplied equipment has greatly extended the duration of time that individuals can work underwater. Breath-hold diving, however, has not disappeared and more recently has become a competitive sport that seeks to extend the depth of exposure on a single breath. To date, depths of more than 700 feet have been achieved on a single breath.[36] However, data obtained from competitive breath-hold divers indicate that they sustain lung injury that involves hemoptysis, lung edema, and cough,[37] and several fatalities have been reported in divers seeking to set depth records while breathholding.[38-40]

Boyles Law Effects on Lung Volume–Pulmonary Barotrauma

A basic physical relationship between pressure and volume of a gas was first stated by Robert Boyle in 1678.[41] He described the relationship of pressure and volume for a fixed mass of an ideal gas as the product of pressure and volume as a constant (P • V = Constant). For a fixed lung volume, a diver submerged underwater with a single breath and fixed lung volume will experience a progressive reduction in lung volume as absolute pressure increases (Figure 4). At 5 atmospheres absolute pressure (a depth of 132 feet in seawater, or FSW), lung volume will be reduced to 20% of surface volume, and if residual volume (RV) is 20% of total lung capacity, lung volume will be reduced to residual volume at 132 FSW underwater. Further depth excursion will reduce lung gas volume below RV and require expansion of the lung vasculature to account for the lung volume reduction.[42] One can note from Figure 4 that at 10 atmospheres absolute pressure (297 FSW), an initial lung gas volume of 5 L would be reduced to 500 mL, and lung vasculature will become engorged to account for the reduced gas space. Liner et al.[43] reported lung injury in competitive breath-hold divers manifested by cough, hemoptysis, and dyspnea with reduced arterial oxygen saturation post dive. Figure 5 shows a theoretical lung volume to depth relationship that includes estimates of blood shifts into the lung vascular space. Note that at record depths for breath-hold diving (> 500 FSW), nearly a liter of blood is shifted into the lung vasculature to account for the reduced gas space. Several fatalities have been reported in extreme breath-hold divers due to massive lung injury and pulmonary hemorrhage.

The same pressure–volume relationship can cause injury to the lung during ascent in divers who breathe compressed gas underwater. In this case, compressed air is supplied to the diver at ambient pressure to allow normal ventilation while underwater. During ascent, the compressed gas will expand according to Boyle's law, and if not properly ventilated, the expanding gas will result in lung rupture, pneumothorax, pneumomediastinum,

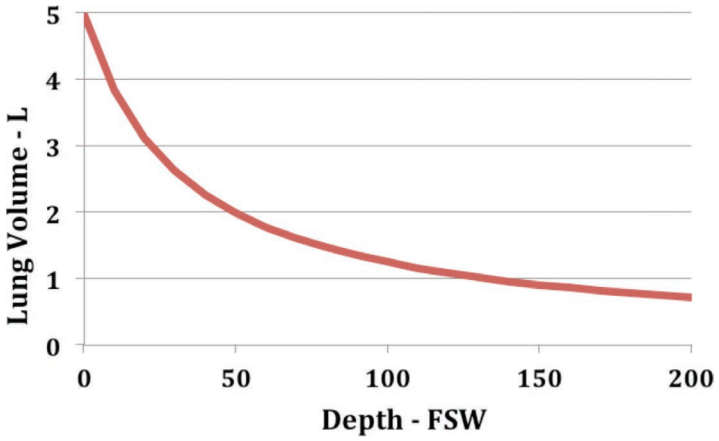

Figure 4. Lung volume vs depth for a breath-hold diver with total lung capacity of 5 L. If residual volume is 20% of total lung volume, at 132 FSW, lung volume will be reduced to residual volume. Further depth changes will result in blood shifting into the pulmonary vascular space to account for reduced gas volume in the lung. Note that the greatest percent volume change occurs in the first 30 feet of depth. FSW: feet seawater.

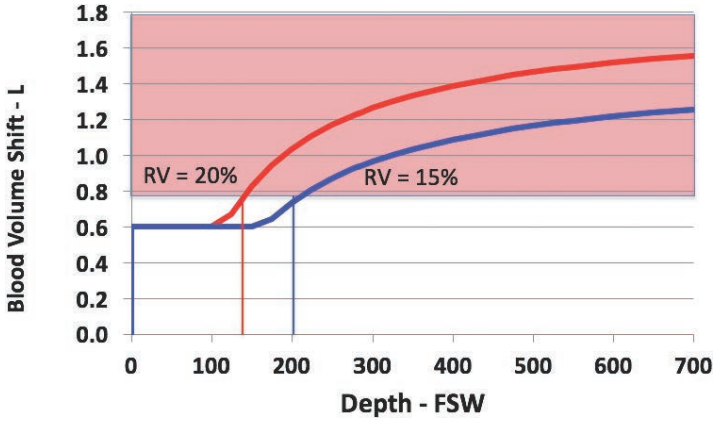

Figure 5. Estimated blood volume shifts vs depth during breath-hold diving. Note that an initial 600 mL of blood shifts from the venous system into the thorax due to pressure effects of immersion alone. Subsequently, based on Boyle's law reductions in lung gas volume, further blood shifts into the pulmonary vasculature will follow lung volume reduction below residual volume. Two lines show changes for a 15% and 20% residual volume fraction of total lung capacity. FSW: feet seawater.

and arterial gas embolism.[44,45] The lung injury and air in the arterial system results in a number of well-defined clinical manifestations (Table 2), the most serious of which is injury to the brain due to cerebral air embolism. This syndrome produces stroke-like manifestations in a diver immediately upon ascent.[46] Gas embolism to the coronary circulation can produce myocardial ischemia, and large amounts of gas filling the central circulation can obstruct blood flow through the heart and produce shock with pulseless electrical activity.[47] Therapy requires treatment in a hyperbaric chamber with increased pressure to compress intra-arterial gas to a smaller, nonobstructive volume and added hyperoxygenation (known as hyperbaric oxygen therapy) to improve tissue viability.[48]

Conclusions

Sports and recreational participation can result in lung injury caused by high pulmonary pressures and increased blood volume that raises intracapillary pressure and results in capillary rupture with subsequent pulmonary edema and hemorrhage. High-intensity exercise can result in accumulation of pulmonary fluid and evidence of pulmonary edema. Competitive swimming can result in both pulmonary edema related to fluid shifts into the thorax from immersion and elevated LV end diastolic pressure related to diastolic dysfunction, particularly in the presence of high-intensity exercise. Underwater diving increases pressure and gas volume changes related to Boyle's law and can result in lung injury during descent in breath-hold diving and with ascent when breathing compressed gas underwater. The most important approach to many of these disorders is prevention. Careful attention to volume loading can reduce the risk for SIPE or IPE, while education regarding Boyle's law in breath-hold diving and compressed air diving will allow the diver to avoid lung injury related to changing gas volume. In addition, avoiding extremes of exercise will reduce the risk of exercise-induced lung injury. It is therefore important when caring for athletes to provide basic education regarding risks for lung injury and means of prevention.

Key Points

1. Left ventricular diastolic relaxation is a key component of aerobic fitness.
2. Extreme high-intensity exercise can cause transient pulmonary edema.
3. Triathletes can develop pulmonary edema during the swimming phase of the event due to fluid shifts related to immersion, high-intensity exercise, and impaired diastolic relaxation.
4. Scuba divers can experience pulmonary edema related to immersion that causes severe dyspnea while underwater.
5. Breath-hold diving to very deep depths can cause severe lung injury.

Arterial Gas Embolism Signs and Symptoms
Unconsciousness
Paralysis
Numbness
Weakness
Extreme fatigue
Paresthesias
Difficulty thinking
Vertigo
Convulsions
Vision abnormalities
Loss of coordination
Nausea and/or vomiting
Hearing abnormalities
Bloody sputum
Dizziness
Personality changes

Table 2. Signs and symptoms of arterial gas embolism.[48]

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Keywords: exercise, pulmonary edema, swimming-induced pulmonary edema, SIPE, immersion pulmonary edema, IPE, diastolic dysfunction

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