Respiratory physiology: adaptations to high-level exercise

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ABSTRACT

Most exercise scientists would agree that the physiological determinants of peak endurance performance include the capacity to transport oxygen to the working muscle, diffusion from the muscle to the mitochondria, energy production and force generation, all influenced by signals from the central nervous system. In general, the capacity of the pulmonary system far exceeds the demands required for ventilation and gas exchange during exercise. Endurance training induces large and significant adaptations within the cardiovascular, musculoskeletal and haematological systems. However, the structural and functional properties of the lung and airways do not change in response to repetitive physical activity and, in elite athletes, the pulmonary system may become a limiting factor to exercise at sea level and altitude. As a consequence to this respiratory paradox, highly trained athletes may develop intrathoracic and extrathoracic obstruction, expiratory flow limitation, respiratory muscle fatigue and exercise-induced hypoxaemia. All of these maladaptations may influence performance.

INTRODUCTION

There is general consensus that the capacity of the respiratory system is overbuilt for the demands placed on ventilation and gas exchange by high-intensity exercise.[1] For all but the highly trained, the limiting factor to exercise performance at sea level is the capacity for maximal oxygen transport to the working muscle. With training, structural and functional adaptations occur in the cardiovascular and musculoskeletal systems. The stresses placed on the chest wall, airways and gas exchange mechanisms by heavy exercise challenge the limits to provide adequate minute ventilation and gas exchange in an environment where the demand exceeds supply. In these instances, the respiratory system is the limiting factor – arterial hypoxaemia ensues and VO_{2max} and performance are affected.

PHYSIOLOGICAL DETERMINANTS TO PERFORMANCE

Maximal aerobic capacity (VO_{2max}) is an important indicator of cardiorespiratory fitness and a significant contributor to endurance performance.[2] The transport of oxygen to the working muscle is the most significant contributing factor to VO_{2max}. Extraction of oxygen by the muscle in the periphery also plays a role in determining aerobic capacity. It is clear, however, that at sea level, the oxidative capacity of the muscle exceeds the ability to transport oxygen to the working muscle. If the oxygen content of arterial blood is increased, the additional oxygen will lead to parallel changes in VO_{2max} and endurance performance.[3] The central nervous system plays a supporting role in the regulation of central and peripheral fatigue and perception of effort related to performance through feedback from the muscle to the motor control areas of the brain.[4]

RESPIRATION DURING EXERCISE

Avoiding an increase in airway resistance during exercise is critical, particularly for elite athletes because any change in airway resistance is followed by an increase in the work of breathing, which may affect exercise performance. To maintain airway resistance near resting level, a series of events occur during exercise.[5] The upper airway dilator muscles activate just before the muscles of inspiration to open the glottis; bronchial smooth muscles relax due to withdrawal of parasympathetic tone and expiratory muscles are systematically recruited during progressive exercise to cause a reduction in end-expiratory lung volume. Flow rates increase, but airway resistance is maintained, allowing a rise in tidal volume with minimal change in elastic work by the respiratory muscles (RMs); the distribution of ventilation is close to uniform, and the increases in alveolar ventilation match those of metabolic demand in an effort to maintain oxygen and carbon dioxide concentrations. These changes within the pulmonary system optimise the ability to transport oxygen to the locomotor muscles yet minimise the work of breathing necessary to support this function.

ADAPTATION TO TRAINING

The physiological adaptations to repetitive, intense exercise have been extensively evaluated in order to gain insight into the factors that are responsible for fatigue and limit exercise. The contribution of central and peripheral factors to endurance performance has been a source of vigorous debate and innovative research. Physical performance is dependent upon a complex, integrated series of physiological events involving the heart, lungs and skeletal muscle. The transport of oxygen and energy production requires the coupling of ventilation and blood flow to muscle metabolism. Repetitive exercise applied in a systematic fashion results in predictable changes that, collectively, improve exercise performance. It is beyond the scope of this study to review in detail all the changes that occur as a result of physical training, but the key adaptations to the cardiovascular,[6] haematological[6] and muscular[7] systems are summarised in table 1.

Table 1 Adaptations to training

	Structural	Functional
Cardiovascular	↑ Enlarged LV cavity size	↑ Cardiac contractility
	↑ Enlarged LV wall thickness	↑ Stroke volume; ↑ Cardiac output
	↑ Heart mass	↑ Diastolic function
	↑ Left ventricular compliance	↓ Total peripheral resistance
	↑ Left atrial dimensions	↑ Venous return
	? Pericardial remodelling	↓ Pericardial constraint
Haematological	↑ Red cell mass	↑ Total blood volume
	↑ Plasma volume	↑ O_2-carrying capacity
Muscle	↑ Mitochondrial density	↑ Rate of energy production
	↑ Capillary/fibre ratio	↑ Rate of force production
	↑ Type 1 fibres	
	↑ Mitochondrial oxidative enzymes	
	↑ Neural recruitment	
Respiratory	None	↑ Respiratory muscle endurance
		↑ Respiratory muscle strength
		↑ Ve/VO_2

LV, left ventricular.

RESPIRATORY ADAPTATIONS

With few exceptions, there are no measurable structural changes in the lung parenchyma, airways or chest wall that accompany a physical training programme.[8] Swimmers have larger static lung volumes and an increased pulmonary diffusion capacity when compared with athletic controls.[9] As strength and endurance of the RMs improve with training, it is possible that these muscles adapt in a similar fashion to the skeletal muscle, but the cellular changes in humans have not been documented. Perhaps, the lack of training adaptation should not be a surprise as the response to exercise by the respiratory system has a much greater scope than the cardiovascular or peripheral muscle systems. Minute ventilation can increase 20-fold in comparison with resting values implying that the lung has adequate buffer to deal with the demands of heavy work. Nevertheless, there are several physiological and clinical conditions that are unique to the highly trained athlete where the demands of intense exercise expose the respiratory system as the 'weak link' in terms of performance.[6]

RESPIRATORY LIMITS TO PERFORMANCE

Exercise-induced arterial hypoxaemia

Arterial blood gases taken during maximal exercise at sea level in highly trained athletes can show PaO_2<9.0 kPa, pH<6.8 and percentage saturation of haemoglobin <90%. These measurements were made in healthy, normal athletes without pulmonary disease. The mechanisms responsible for this desaturation have been a source of investigation for over 30 years; relative hypoventilation, V/Q mismatch, diffusion limitation secondary to rapid red cell transit time or transient pulmonary oedema and possible right-to-left or intrapulmonary shunt have all been proposed as explanations for exercise-induced arterial hypoxaemia (EIAH).[1 8] EIAH only occurs in ~50% of highly trained male athletes, and there appears to be a higher prevalence in women with EIAH occurring at a lower exercise intensity.[10 11]

A reduction in exercise performance is linked to the development of EIAH. In highly trained subjects who were exposed to mild and moderate hypoxaemia, there was a significant decrease in cycle performance tests and a linear relationship was reported between the change in the saturation of oxygen on haemoglobin and the decrease in work capacity.[12]

Vocal cord dysfunction

Upper airway extrathoracic obstruction can occur in athletes during high-intensity exercise when the flow rates are high. Paradoxical narrowing of the glottic aperture develops suddenly and immediately affects flow rates and minute ventilation.[5] This results in dyspnoea, an audible inspiratory stridor, hypoxaemia, CO_2 retention and often affects performance. Many of these athletes receive the diagnosis of exercise-induced bronchoconstriction, merely on the basis of dyspnoea during exercise. They are often treated with inhaled asthma medications, which do not benefit this condition.

These athletes deserve to be seen by an ear, nose and throat specialist to rule out other causes of upper airway obstruction. To induce this condition in an exercise laboratory, it is necessary to use high-intensity exercise that requires high-flow rates. With the onset of vocal cord dysfunction, flow rates, minute ventilation and SaO_2 drop quickly, and there is a rapid rise in end-tidal CO_2. Examination of the breath-by-breath flow-volume curves during exercise will show a decrease and blunting of the inspiratory loop.[5] The condition is self-limiting and recovery is rapid once the exercise is stopped and the flow rates decrease. Regretfully, this is a difficult clinical condition to treat effectively and a percentage of these athletes will also have exercise-induced bronchoconstriction.[13]

Expiratory flow limitation

Figure 1 shows the flow-volume curves from an untrained and trained athlete. Many endurance athletes show significant expiratory flow limitation (EFL) during heavy exercise. This results in hyperinflation of their end-expiratory lung volume which leads to reduced dynamic lung compliance and an increased work of breathing.[5 14 15] The normal hyperventilatory response to heavy work is limited, which results in dyspnoea, EIAH and consequentially poor performance. Positive expiratory pleural pressures, which can exceed the critical closing pressure of the airway, lead to increased afterload on the left ventricle. This results in compromised stroke volume and cardiac output, further influencing performance.[14] Tidal volume plateaus at a lower minute ventilation and increases breathing frequency. Finally, with EFL, inspiratory muscle fatigue ensues because the muscles are working at shorter than their optimal length and with a greater velocity of shortening.[5]

Relative to men, women have smaller lung volumes and narrower airway diameters for a given lung volume.[16] This makes them more prone to EFL and its sequelae. The masters athlete, capable of heavy exercise, is also compromised, as with normal ageing there is a loss of elastic recoil leading to a reduction in the maximal flow-volume loop and higher proportions of dead space ventilation at rest and during exercise.[5]

Respiratory muscle fatigue/training

It is clear from the animal and human literature that RM will respond to an appropriate training stimulus. The average man has about 4 kg of skeletal muscle that can be recruited for respiration in heavy work. While the diaphragm has its own metabolic features and is resistant to fatigue, the remaining skeletal muscle

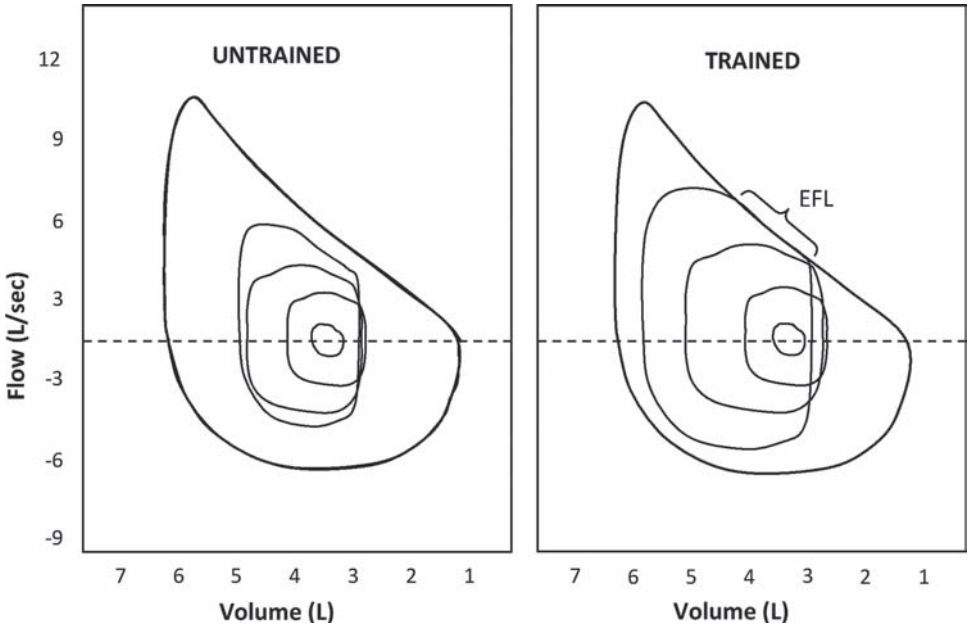

Figure 1 On the left is the flow-volume (V-V) envelope of an untrained subject with a large ventilatory reserve given the size of the maximal tidal breath relative to this maximum flow-volume loop (MFVL). The graph on the right depicts the V-V loop of an endurance-trained athlete. The MFVL is identical but there are much higher ventilatory requirements to meet the increased metabolic demands. The figure shows the consequences of training. The inspiratory reserve volume is much smaller, the athlete is expiratory flow limited and end-expiratory lung volume (EELV) is creeping back towards resting values.

can be trained either by 'volume', via voluntary isocapnic hyperpnoea or 'resistance', using breathing against a resistive load.[17] With volume loading, there are significant increases in maximal sustainable voluntary capacity and maximum voluntary ventilation.[18] Studies using resistive training have reported increases in maximum inspiratory pressure in the range of 8% to 45%.[19]

To the competitive athlete, whether RM training will improve performance remains a controversial issue. Some sport scientists report an improvement in time to exhaustion at submaximal workloads following RM training.[20 21] However, most report no response to RM training in the parameters, assessed during maximal exercise, that would most likely be associated with a change in performance – for example, maximal minute ventilation, maximal aerobic capacity, heart rate, stroke volume, blood gas concentrations or % saturation of haemoglobin with oxygen.[18 21]

The RMs can fatigue with sustained exercise, and there is experimental evidence that RM resistive loading or RM unloading will significantly influence performance time in highly trained cyclists.[22] There are improvements in ventilatory efficiency following RM training, and a reduction in minute ventilation for a given workload would influence the oxygen consumed by the RM, perhaps changing the blood flow distribution during exercise. Training these muscles to delay the onset of fatigue and 'steal' blood flow from the trained RM to locomotor muscles to improve performance is an attractive hypothesis that requires experimental evidence.[6 23]

SUMMARY

The respiratory system, with the capacity of the lung parenchyma, RM recruitment and multilevel neural/hormonal control of breathing, is built for exercise. It is unique; however, in that there is no intrinsic capacity for adaptation to endurance training. In the fit, active individual strenuous exercise is limited by the capacity to transport oxygen to the locomotor muscles. Following endurance training, the cardiac, haematological and muscular systems evolve to the extent that the demand for oxygen transport and usage exceeds that of the lung and chest wall to deliver oxygen to the working muscle. The respiratory system becomes the 'weak link' and is responsible for compromised performance, possibly on the basis of exercise-induced hypoxaemia, EFL, RM fatigue with or without changes in extrathoracic airway resistance.

Competing interests None.

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