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Mini-Symposium: The Clinical Applications of Exercise Testing in Children

Assessment of physiological capacities of elite athletes & respiratory limitations to exercise performance

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SUMMARY

Physiological assessment of athletes is an important process for the characterization of the athlete, monitoring progress and the trained state or 'level of preparedness' of an athlete, as well as aiding the process of training program design. Interestingly, the majority of physiological assessments performed on athletes can also be performed on children with disease, and therefore clinicians can learn a great deal about physiology and assessment of patient populations through the examination of the physiological responses of elite athletes. This review describes typical physiological responses of elite athletes to tests of aerobic and anaerobic metabolism and provides a specific focus upon respiratory limitations to exercise performance. Typical responses of elite athletes are described to provide the scientist and clinician with a perspective of the upper range of physiological capacities of elite athletes.

INTRODUCTION

Physiological assessment of elite athletes provides a unique opportunity to examine the tremendous adaptations of humans to specific types of exercise and training.[1] These unique and extreme adaptations can be valuable to a clinician, as the acute adaptation to exercise in athletes, for example coping with a transient metabolic acidosis, can mirror the conditions experienced by patients, albeit at a much lower intensity. Further, understanding the effects of long term exercise training on humans can provide a physiological rationale for the implementation of exercise and physical activity recommendations for patients with disease. Young athletes under the age of 18 are regularly achieving world class performances in many sports and respond differently to training than adults[2], thus special opportunities to learn from paediatric athletes are available.

More specifically, testing and monitoring of athletes has a number of specific benefits that are similar to the benefits of exercise testing of patients for the clinician, and these include: allowing for the design of more precise training programs (monitoring the effect of therapies), helping to monitor progress in training (monitoring clinical status), helping athletes understand the objectives of and rationale for training (helping patients and their families understand therapeutic interventions), providing data on athletes' physical characteristics (or a patient's physiological characteristics), and helping to predict performance (or impact on the disease, condition, and quality of life). A typical example of this is the assessment of aerobic capacity, which has predictive effects for athletic performance[3] and for survival in respiratory disease.[4] Physiological tests can provide both direct and indirect measures of metabolic factors across the spectrum of bioenergetic pathways from the immediately and rapidly available systems through to those that contribute extensively to long duration activities and recovery or even resting conditions. In addition, key limitations of the respiratory system that can be readily observed during exercise performance may provide valuable insights for pediatric respiratory clinicians and other interested parties.

PHYSIOLOGICAL ASSESSMENT OF AEROBIC OXIDATIVE CAPACITY

The overall capability of the aerobic oxidative system is generally evaluated by measuring the maximal volume of oxygen

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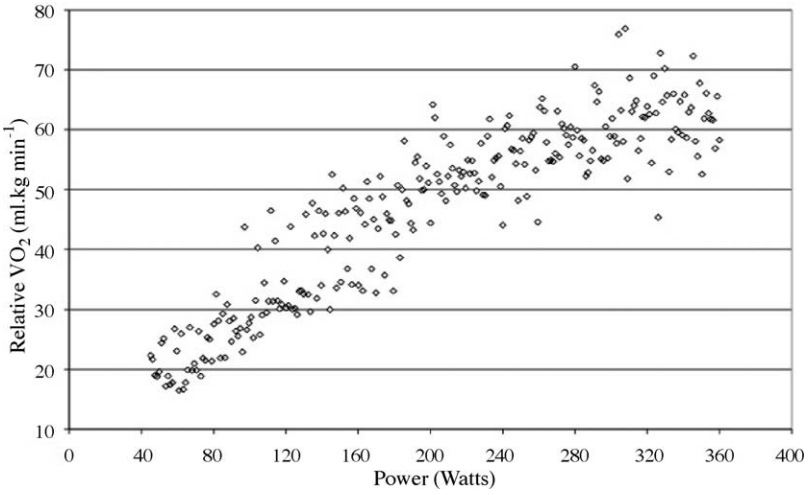

Figure 1. The relative volume of oxygen (VO_2) consumed during an incremental cycling test. Subject is an elite cyclist.

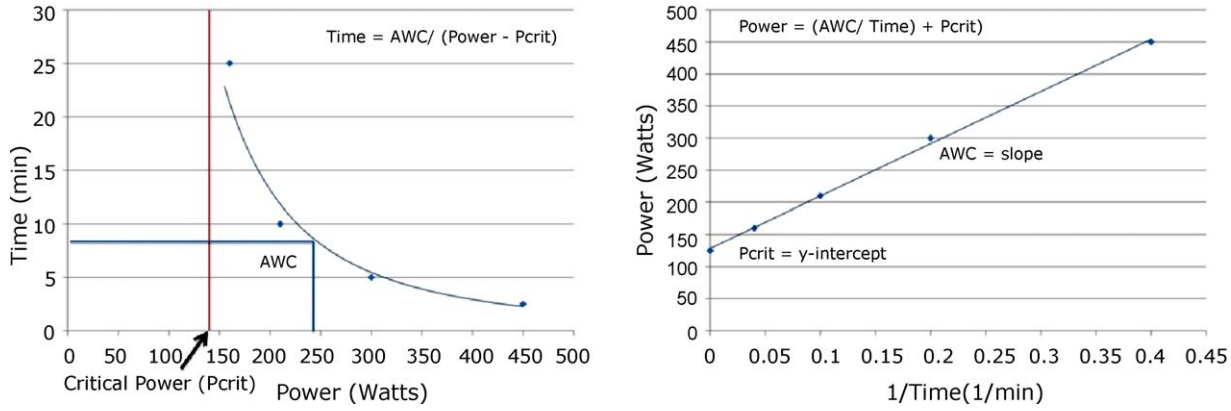

Figure 2. The hyperbolic and linear representations of critical power based on typical data from a cyclist. Abbreviations: Pcrit, critical power; AWC, anaerobic working capacity.

that can be consumed per kilogram of mass in a given amount of time.[5] This measure is called aerobic power or VO_{2max} and is measured in either absolute ($L\,min^{-1}$) or relative units ($ml\,min^{-1}\,kg^{-1}$). The average healthy sedentary adult will have a VO_{2max} of 25–40 $ml{\cdot}min^{-1}\,kg^{-1}$, and highly trained endurance athletes, such as elite cross country skiers, have been measured to have VO_{2max} levels well in excess of 75 $ml\,min^{-1}\,kg^{-1}$.[6] Typical VO_2 results recorded during a maximal incremental exercise test are presented in Fig. 1. Normal values for children have been reported[7], as have values for paediatric athletic populations such as swimmers.[8]

In addition to the maximal aerobic power that is typically assessed by incremental exercise tests, aerobic endurance capacity is also of great importance for many elite athletes and may be important for patients with muscle disease[9], cardiac disease[10] and also respiratory disease[11], although to date the literature on endurance capacity in pediatric disease populations is limited. Aerobic endurance capacity may be assessed using several different approaches such as submaximal predictive protocols or exhaustive exercise tasks as various levels of relative intensity. An interesting and increasingly utilised format for endurance or 'sustainable' exercise evaluation is the critical power (P_{Crit}) concept[12], which describes the hyperbolic relationship between power output and time to exhaustion.[13,14,15] The hyperbolic data can be transformed into a linear relationship between total work performed and time to exhaustion (see Fig. 2).[16,17] Whole body P_{Crit} has been reported for running[18], swimming[19], rowing[20] as well as for specific muscle groups.[21] Researchers have also suggested that P_{Crit} may apply to cardiac and respiratory muscle as well, with both cardiac and respiratory muscle having higher P_{Crit} than skeletal muscle.[17]

In theory, P_{Crit} represents a work intensity that can be performed for a very long time without fatigue[17]. Despite more recent work that suggests that P_{Crit} is not sustainable *ad infinitum*[22,23], P_{Crit} correlates well with endurance performance and associated physiological parameters, and can be considered as an index of an overall aerobic endurance capability.[16,24] In clinical investigations to date, P_{Crit} has been used primarily for the evaluation of chronic obstructive pulmonary disease[25], however it is clear that P_{Crit} as a protocol tool does present an interesting alternative to traditional incremental aerobic exercise tests for the assessment of endurance capability in those patients with muscle, cardiac and respiratory diseases. However, it should be recognised that this form of exercise testing requires several trials of variable duration maximal efforts and, as such, is extremely demanding of the athlete or patient.

Incremental (either at submaximal levels and/or to maximum levels) and critical power endurance testing can provide insight into aerobic oxidative physiology.[26] More specifically the acute adaptation of several physiological factors can be observed during incremental exercise. For example, increases in volume of oxygen uptake (see Fig. 1), ventilation (see Fig. 3), and heart rate (see

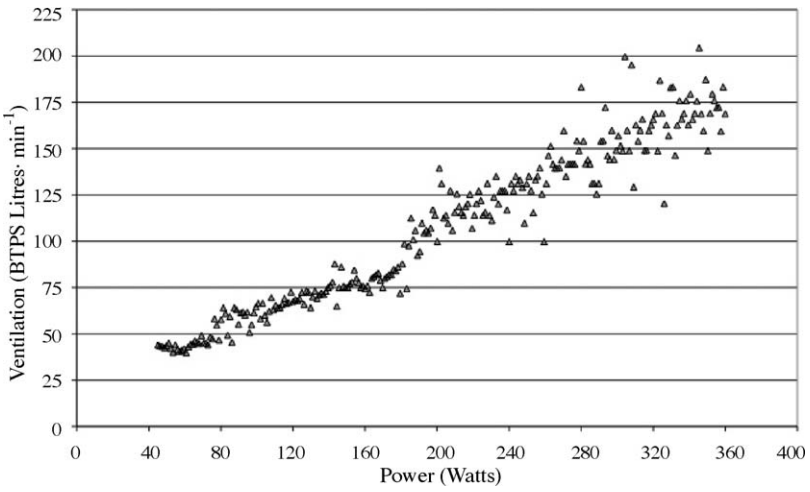

Figure 3. The ventilatory response to incremental exercise in an elite cyclist.

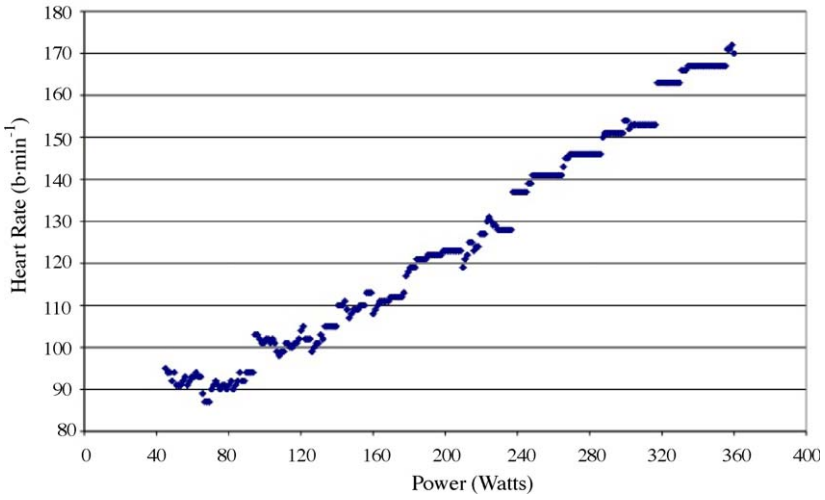

Figure 4. Relationship between heart rate and incremental work load in an elite cyclist.

Fig. 4), are easily observed. Measurement and interpretation of these factors can provide a window to understanding the many contributors to a high aerobic power or capacity including (a) arterial oxygen content, (b) cardiac output (heart rate x stroke volume), and (c) tissue oxygen extraction.[27] Arterial oxygen content depends on adequate ventilation and the oxygen-carrying capacity of the blood, which is determined by haematocrit levels and haemoglobin (Hb) concentration, with the global parameter of total Hb being of prime importance[26] Tissue oxygen extraction depends upon the rate of oxygen diffusion from the capillaries to the muscle cell and on the rate of oxygen utilization, which depend on the level of activity of various enzymes influencing the aerobic pathways, with particular reference to mitochondrial efficiency[28,29,30]. All of these factors combine to allow for the relative (per kilogram) consumption of oxygen to rise up to 60 fold from rest to intense exercise.[27]

Aerobic capacity or endurance testing has excellent applicability in monitoring the progress of athletes in response to training as well as for evaluating the function of the various steps of the oxygen transport pathway in patients with cardiovascular or respiratory disease. It is of importance to note that maximal aerobic power may plateau after years of training[31], and yet performance improvements in endurance events may still be achieved.[32] These subsequent improvements are realized through many adaptations including increases in blood plasma and erythrocyte volume[33], cardiac adaptations[34], type I muscle fibre characteristics[35], mitochondrial volume[26,36], capillarization & aerobic enzyme function.[37,38,39,40] This can be observed in elite athletes as an increase in power production or speed at a given (sub-maximal) VO_2[31], or increases in the lactate threshold (that is, the velocity or power output at which lactate begins to accumulate in the blood and a point that is generally accepted as being associated with the onset of fatigue)[27].

A further test parameter that is gaining increasing recognition as an important evaluative tool in this area is the use of oxygen uptake kinetics.[41] A significant body of work has been accumulated over the last 40 years[42,43] and in particular over the last 5–10 years[44,45] concerning both the theory and use of VO_2 kinetics. Due to the equipment-intensive nature of this area of physiological evaluation, it is best suited to clinical and exercise laboratory situations. As with the previously mentioned avenues of testing, VO_2 kinetics evaluation may provide the clinician and exercise scientist with both individual-specific and training sensitive (intervention) information[41,45]. Jones & Poole[44] provide an excellent overview resource concerning VO_2 kinetics for clinicians and researchers wishing to embark on such analyses with specific populations.

Aerobic oxidative metabolic and endurance exercise capacity change throughout growth and development phases in children and adolescents. Generally, the aerobic oxidative system does not respond well to training prior to the onset of the peak height velocity growth phase around puberty[46], after which the maximal oxygen uptake can increase from 15 through to 25 years of age in athletes[6], but also can be observed to increase cross-sectionally in the non-athletic population from 8–16 years of age.[47] The responsiveness of the aerobic system at puberty may be related to the hormonal changes and growth of the cardiorespiratory and musculoskeletal systems, changes that may be more pronounced in adolescent boys than girls.[2] Therefore, growth and development as well as training status must be considered when interpreting aerobic power and endurance capacity test results from children and adolescents.

PHYSIOLOGICAL ASSESSMENT OF ANAEROBIC GLYCOLYTIC CAPACITY

The anaerobic glycolytic system is the primary energy system involved in shorter activities of high intensity and is critically important for athletes involved in events lasting more than a few seconds through to several minutes (e.g., sprint running, many swimming events, and track cycling sprint events,), as well as for most team sports that require bursts of high intensity activity (e.g., basketball, volleyball, soccer, hockey). Anaerobic glycolytic power and capacity is typically negatively impacted in a number of disease states such as McArdle's disease[48], muscular dystrophies[49], and glycogen storage disorders.[50]

Anaerobic glycolytic metabolism provides ATP for muscular contraction at a rapid rate, and at such high rates of glycolytic flux (as seen in high intensity, short duration exercise) that pyruvate formation exceeds pyruvate oxidation, resulting in lactic acidosis. Lactic acid is known to dissociate quickly to lactate and hydrogen ions, factors known to be associated with various mechanisms for muscular fatigue, as well as the close relationship between blood lactate concentration and 'fatigue' when exercising.[51,52,53] It is due to these factors, as well as the relative ease with which blood lactate can be measured, that lactic acid and lactate have been of considerable interest to exercise scientists for many years.[53]

Typical blood lactate responses to incremental exercise in a sprinter and endurance swimmer of the same gender (female) and age (16 years) are shown in Fig. 5. Two key observations are of note, firstly that the endurance athlete can work at a higher intensity than the sprinter before blood lactate begins to accumulate beyond the 4 mmol L^{-1} level (see note *1 in Fig. 5). This is a level of lactate that is associated with endurance performance capability.[54] Secondly, the sprinter can produce significantly more lactate than the endurance athlete (see note *2 in Fig. 5), possibly indicating a greater anaerobic glycolytic metabolic flux and, therefore, an ability (in this example) for the athlete to swim at a faster peak speed (that is, produce a greater muscular power, all other things being equal).[55] Obviously, this area and indeed the expanded areas of buffering capacity and ammonia production may well be of interest for respiratory clinicians due to the close association between hydrogen ion production via lactic acid and the respiratory system, as well as the impact of buffering capacity and ammonia levels on sustainable performance. An important aspect of overall buffering capacity is the ability of the muscle tissue and blood to store and transport CO_2 produced through metabolism. H^+ combines with HCO_3 to form carbonic acid (H_2CO_3) which then dissociates into CO_2 and water. If CO_2 can be removed through increased ventilation at the lung, the equation can shift and more H^+ can be accepted and therefore buffer the pH of the blood. This provides great insight into the close association between anaerobic metabolism and ventilation during exercise.[56,57]

The efficiency and capacity of the anaerobic system can also be evaluated, by measuring maximal short-term exercise capacity and looking at peak power and the resistance to fatigue during the short exercise interval.[58] The classic "Wingate" test is a reliable and valid test for the anaerobic glycolytic system.[59] Typical results from a 30 second anaerobic cycle test are show in Fig. 6, with both the peak power and end power indicated. A fatigue index is often calculated based on the difference between peak power and end power, also shown in Fig. 6. Some anaerobic test results for children have been published for boys[60,61] and girls[62], however the data on females is limited.

Children demonstrate greater resistance to the muscular fatigue often associated with high-intensity exercise than adults[49], an important consideration for the paediatric clinician or sport scientist. This can be observed functionally as sprint running or cycling, jumping, or agility.[58] Research on anaerobic glycolytic function in paediatric athletes and / or patients is relatively less extensive than aerobic research, however there is some consensus that short-term muscle power increases during childhood and

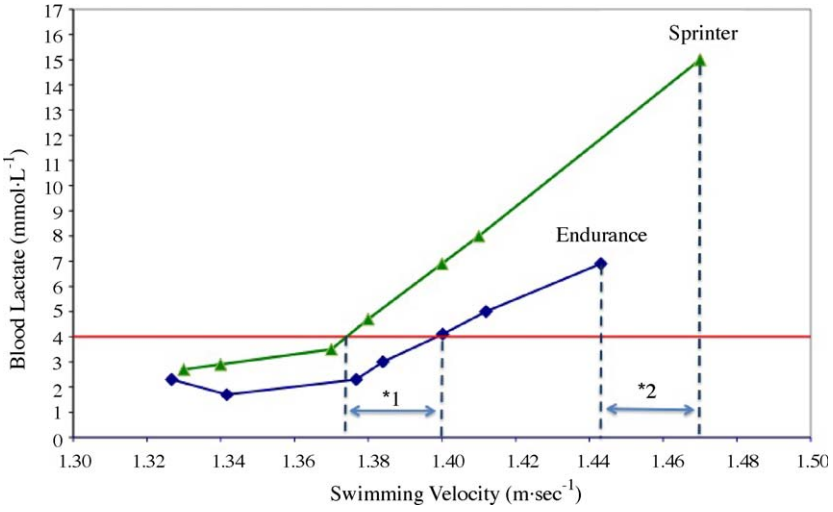

Figure 5. The blood lactate response to an incremental swimming test. Results are presented for a typical sprinter (100 metre freestyle swimmer) and an endurance athlete (1500 m freestyle swimmer). *1 indicates the difference in velocity at the 4 mmol L^{-1} lactic acid level, and *2 indicates the difference in peak lactate and peak swimming speeds for the two athletes.

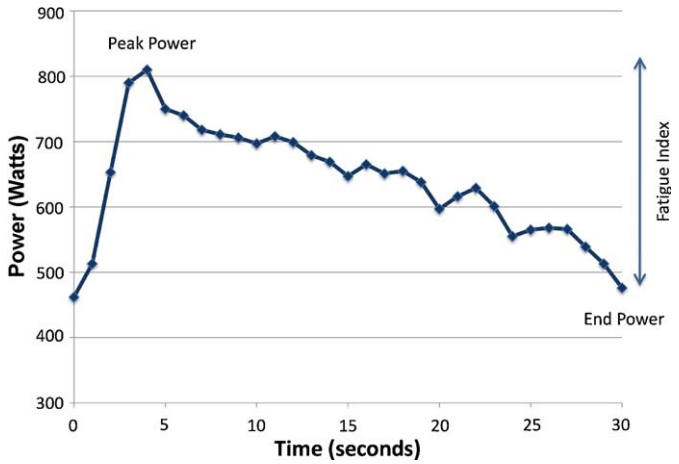

Figure 6. Power measured during a 30 second anaerobic cycle test in a female Olympic hockey player. Peak power is reached quickly, and end power represents the power at the end of the 30 second test. Fatigue index is interpreted as the difference between the peak and end power.

adolescence, and that late puberty is a critical period for positive adaptation in this system.[58] Differences between children and adolescents may be due to hormonal factors, improved motor-co-ordination, and increases in muscle mass.[58]

NOVEL METHODS FOR DIRECT, NON-INVASIVE EXAMINATION OF EXERCISE CAPACITY

Fortunately, advanced physiological assessment equipment is becoming smaller and more portable, meaning that athletes can now be assessed accurately while exercising in the field or while performing their sport. An example is the portable Cosmed K_4B^2 system (www.cosmed.it) that allows for VO_2, VCO_2, heart rate and other physiological data to be collected via telemetry while also collecting GPS data related to speed and location (see Fig. 7). When coupled with modern micro-sample blood lactate equipment, such as the Lactate Pro Analyzer (www.lactate.com), advanced exercise physiology is now available in the laboratory and field (see Fig. 8). This provides both clinicians and exercise physiologists with the ability to evaluate their athletes or patients in more realistic situations with laboratory quality assessments.

Direct, accurate, and non-invasive measurement of muscle metabolism is also now more readily available with new magnetic resonance imaging and spectroscopy techniques. Our research

Figure 7. An example of the use of a portable metabolic assessment apparatus to measure exhaled gases. Note the global positioning unit taped to the kayak and the heart rate monitor on the left quadriceps.

Figure 8. An example of the use of a portable blood lactate testing assessment apparatus in a field testing situation.

group has recently described potential methods for examination of muscle lipid and mitochondrial oxidative metabolism in a clinical population.[63] *In vivo* magnetic resonance imaging (MRI), and magnetic resonance spectroscopy (MRS) are safe, non-invasive techniques that are rapidly becoming a popular approaches for assessing tissue metabolism. Unlike other *in vivo* methodologies, there is no injection of radioactive materials (as with positron emission tomography) and no application of external ionizing radiation (as with computed tomography). Exercise protocols combined with phosphorus MRS (^{31}P-MRS) have become an additional effective, non-invasive technique to detect abnormalities in mitochondrial oxidative and glycolytic / glycogenolytic metabolism. The primary metabolites visible using this approach are ATP (3 distinctive peaks corresponding to *A*, *B* and *Y* phosphates), phosphocreatine (PCr), inorganic phosphate (Pi), and phosphate mono- and di-esters. In addition, intracellular pH and magnesium ions (Mg^{2+}) can be calculated based on the chemical shift of Pi and *B*-ATP, respectively. Examination of the changes in the metabolites pre and post exercise can provide an indication of bio-energetic activity (see Fig. 9). Further, Prompers and colleagues[64] have shown that the recovery rate of phosphocreatine after exercise is related to mitochondrial oxidative function, and our research team has recently observed differences in this variable between athletes, healthy controls and patients with obesity as shown in Fig. 10.

RESPIRATORY LIMITATIONS TO MAXIMAL EXERCISE PERFORMANCE

The ventilatory system has not been traditionally viewed as a limiting factor during exercise.[65] However, recent research has questioned this notion.[66] Maximal breathing frequencies during exercise have been reported to be between 40-60 breaths per minute and maximal tidal volumes can range from 1.5-4.0 litres per minute[67]. Thus, ventilation has the potential to exceed 200 litres per minute during maximal exercise (see Fig. 2); an amazing feat given the size of most people's lungs! More recent research does suggest that high intensity or sustained duration exercise encroaches on the capacity of the respiratory system and that this impacts whole body exercise performance.[68] The specific mechanisms where the respiratory system is likely to limit exercise performance include (1) increased work of breathing, (2) exercise-induced arterial hypoxemia, (3) respiratory muscle fatigue, and (4) dyspnoea. The mechanistic physiology and impact of these four factors on whole body exercise performance is reviewed in this section.

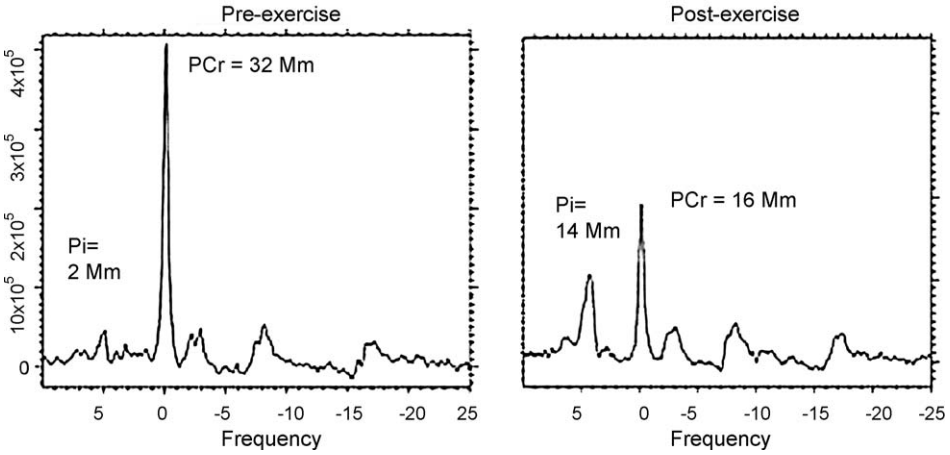

Figure 9. Typical spectra acquired during ^{31}phosphorus magnetic resonance spectroscopy scanning before and after 60 seconds of intense exercise. Abbreviations: Pi, inorganic phosphate; PCr, phosphocreatine.

THE WORK OF BREATHING

The work of breathing may have an impact on exercise performance because the relationship between ventilation and energy requirement is represented not by a straight line, but by a curve of increasing slope. As exercise intensity increases, greater and greater proportions of the increase in total body oxygen consumption are made up of increased oxygen requirements of the respiratory muscles.[69] During maximal exercise, the blood flow to the diaphragm and to the inspiratory and expiratory muscles of the rib cage and abdominal wall represents up to 15% of the total oxygen cost of exercise.[70] Increasing ventilation increases the total metabolic cost of the activity and creates competition for blood flow between the respiratory and locomotor muscles that may well result in decreased exercise performance[71–73] via a vasoconstrictive mechanism.[74] St. Croix and colleagues[74] demonstrated that inspiratory muscle work causes an increased muscle sympathetic nerve activity and that a significant inverse relationship exists between work of the breathing and blood flow to the legs. This suggests that the work of breathing normally incurred during maximal exercise causes significant vasoconstriction in the locomotor muscles via activation of the sympathetic nervous system, thus compromising locomotor muscle perfusion and, ultimately, VO_2. More recent work[75] has extended this observation and has shown that unloading the respiratory muscles during exercise resulted in increased hyperpnoea, decreased perceptions of dyspnoea and limb discomfort, and an increase in the time to exhaustion during an exercise test.

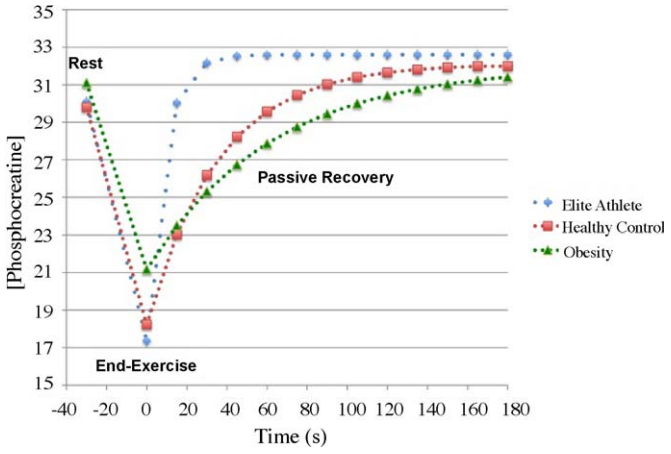

Figure 10. Typical results for phosphocreatine concentration in muscle before and after 90 seconds of exhaustive exercise in elite athletes, healthy controls, and patients with obesity. Phosphocreatine concentrations were measured in the quadriceps muscle using ^{31}phosphorus magnetic resonance spectroscopy techniques.

EXERCISE-INDUCED COMPROMISE OF OXYGEN UPTAKE AT THE LUNG

Arterial blood oxygenation is indexed by the partial pressure of oxygen in arterial blood, PaO_2, and the saturation of oxygen in haemoglobin, Hb SaO_2. Exercise-induced compromised oxygen uptake at the lung consists of a decrease in arterial blood oxygenation of more than 10% due to an inadequate alveolar hyperventilation and/or inadequate O_2 exchange between the alveolar gas and the arterial blood, or possibly due to a temperature and pH induced rightwards shift in the oxygen saturation curve.[77] Regarding the latter, Dempsey has suggested that the lung may not be capable of maintaining blood pH and acid-base balance during intense exercise.[76] It has now been documented that exercise-induced arterial hypoxemia during intense exercise occurs in a significant number of normal healthy subjects[77], and in many highly trained endurance athletes.[78,79] Notably, the trend towards an exercise-induced arterial hypoxemia occurs even during moderate exercise in some highly trained athletes.[79] The impact of the hypoxemia is that the fall in SaO_2 results in a decreased oxygen transport, which may limit VO_{2max}, peripheral muscle perfusion, and exercise performance.

Unfortunately, exercise training may actually cause exercise-induced arterial hypoxemia. Recently, Durand and colleagues have postulated that, among athletes with similar VO_{2max} values, an increased training volume is correlated with the incidence of exercise-induced arterial hypoxemia.[79] Further, highly trained endurance athletes exhibit a reduced ventilatory response to exercise[80,81], perhaps as a result of blunted chemoreflexes.[82] This reduced "drive" to breathe during exercise may result in inadequate ventilation that has been suggested as a cause of exercise-induced arterial hypoxemia.[83] An alternative explanation for the link between exercise training and arterial hypoxemia may be that the individual who experiences exercise-induced arterial hypoxemia has undergone compensatory adaptation in the cardiovascular and metabolic aspects of body function, but the training has not had an effect on the chest wall, lungs, and airways.[76] A combination of these factors is likely to explain the

observation of exercise-induced arterial hypoxemia in athletes during exercise.

RESPIRATORY MUSCLE FATIGUE

Similar to other skeletal muscles, the respiratory muscles are subject to fatigue.[84,85] Nickerson and Keens[86] reported that high-intensity exercise (>85%VO_{2max}) results in respiratory muscle fatigue. Perret and colleagues[87] showed that respiratory muscle performance is reduced after endurance exercise, regardless of the preceding exercise intensity. Further, respiratory muscle fatigue may limit exercise performance[88], by at least four mechanisms: (1) an inadequate hyperventilatory response to exercise, (2) tachypnoea, (3) increased sympathetic vasoconstrictor outflow to limb skeletal muscle, and (4) an increased sensation of dyspnoea. Inadequate hyperventilation may arise because the respiratory muscles are unable to produce the required pressures.[85] Respiratory muscle fatigue may also result in a pattern of rapid shallow breathing termed tachypnoea[89], although elite-level athletes do not seem to exhibit tachypnoea.[90] As described in the work of breathing section of this paper, St. Croix and colleagues[74] have demonstrated that respiratory muscle work and fatigue result in an increased muscle sympathetic nerve activity at exhaustion, which compromised blood flow to the locomotor muscles.

DYSPNOEA

Dyspnoea can be described as the conscious sensation of breathlessness produced by changes in thoracic displacement or respiratory muscle force during breathing that allows individuals to assess the depth of a breath and the level of ventilation[91], or, more simply, as an uncomfortable sensation or awareness of breathing.[92] It may be a function of the intensity of the motor command signals arising from medullary respiratory neurones and the resulting neural traffic to the respiratory muscles[91], or may arise from afferent feedback primarily from receptors in the respiratory muscles.[93] The latter possibility has been called the length–tension inappropriateness theory of respiratory load sensation. In addition to mechanoreceptor input, feedback arising from chemoreceptors may also contribute to sensations of dyspnoea.[91] The physiological purpose of dyspnoea may be to protect and limit strain on the respiratory muscles and to prevent the development of respiratory muscle fatigue.[91]

Exercise training may reduce sensations of dyspnoea via several mechanisms. Any depression of the chemoreceptors' activity would be expected to reduce the level of peripheral chemoreflex response and, therefore, decrease the sensation of dyspnoea.[82] Specific respiratory muscle training has been shown to increase the maximal strength and pressure producing capacity of the respiratory muscles.[66] Therefore, at a given exercise intensity, relative – inspiratory pressures that are required to produce the appropriate ventilation would be reduced, and would result in a decreased sensation of respiratory discomfort or in a reduction in the nervous activity required to generate a given respiratory pressure.

SUMMARY

This review describes typical physiological responses of elite athletes to tests of aerobic and anaerobic metabolism and details respiratory limitations to exercise performance. Aerobic and anaerobic test results for elite athletes are presented and provide insight into the positive adaptations in human physiology that are possible with exercise training. Growth and training both have an effect on metabolic parameters related to exercise performance and these must be considered in the interpretation of test results. Novel methods such as advanced field testing and non-invasive magnetic resonance imaging and spectroscopy hold great potential for future physiological investigations.

Acknowledgements

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KEY POINTS

1. Respiratory limitations to maximal exercise performance may be similar to conditions faced by patients with respiratory disease at rest or in light exercise.
2. Physiological assessments that have been developed for athletes can be used to help elucidate the pathophysiology of respiratory diseases.
3. Interpretation of the results of such tests in comparison to both normal and athletic populations can provide unique insights into the physiology of patients with respiratory disease.
4. Exercise can be used as therapy for patients with respiratory disease, and exercise programs can be tailored to the individual based on the results of physiological assessments.

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