SAMPLE CHAPTER

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CHAPTER 12

Aerobic fitness

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Introduction

Aerobic fitness may be defined as the ability to deliver oxygen to the muscles and to utilize it to generate energy to support muscle activity during exercise. Aerobic fitness therefore depends upon the pulmonary, cardiovascular, and haematological components of oxygen delivery and the oxidative mechanisms of exercising muscles.

Maximal oxygen uptake ($\dot{V}O_2$ max), the highest rate at which oxygen can be consumed by the muscles during an exercise test to exhaustion, is widely recognized as the best single measure of aerobic fitness. However, only a minority of children satisfy the classical $\dot{V}O_2$ plateau criterion for achieving $\dot{V}O_2$ max in a single exercise test, and it has become conventional to use the term peak $\dot{V}O_2$ when discussing young people's aerobic fitness. The distinction between $\dot{V}O_2$ max and peak $\dot{V}O_2$ will be clarified in the Methodological issues section and thereafter the term peak $\dot{V}O_2$ will be adopted when referring to children or adolescents. In the meantime, the conventional term $\dot{V}O_2$ max will be used unless the research cited specifically refers to peak $\dot{V}O_2$.

Maximal $\dot{V}O_2$ limits the capacity to perform aerobic exercise but it does not define all aspects of aerobic fitness. The ability to sustain submaximal exercise is aptly represented by blood lactate accumulation, which also provides a sensitive means of evaluating improvements in muscle oxidative capacity with exercise training, often in the absence of changes in $\dot{V}O_2$ max. However, in everyday life young people's spontaneous play and participation in sport are more concerned with short duration, intermittent exercise, and rapid changes in exercise intensity. Under these conditions $\dot{V}O_2$ max and blood lactate accumulation might be considered variables of investigative convenience rather than factors underpinning exercise behaviour, and it is the kinetics of pulmonary $\dot{V}O_2$ ($p\dot{V}O_2$) which best describe this aspect of aerobic fitness.

To provide an appropriate framework for subsequent discussion of aerobic fitness initially the concepts of $\dot{V}O_2$ max, blood lactate accumulation, and $p\dot{V}O_2$ kinetics will be introduced. Thereafter the focus will be on aerobic fitness in relation to chronological age, body size, biological maturity, and sexual dimorphism. It is recognized that aerobic fitness has a genetic component, with the heritability of $\dot{V}O_2$ max estimated to be ~50%,[1] but genetics are outside the scope of this chapter and the topic is addressed in Chapter 20 and Chapter 31.

The following terms are used throughout the chapter: 'prepubertal children' when prepuberty is confirmed in the research cited; 'children' to represent those 12 years and younger but without proof of pubertal status; 'adolescents' to refer to 13- to 18-year-olds; and 'youth' or 'young people' to describe both children and adolescents.

Measures of aerobic fitness

Maximal oxygen uptake

The seminal work of Hill and Lupton[2] in the 1920s gave rise to the concept of $\dot{V}O_2$ max in humans. They were, of course, constrained by the available technology and for context their experimental protocol is worth describing in their own words,

> In determining the rate of oxygen intake during running at various speeds, the subject ran with a constant measured velocity around a grass track carrying a Douglas bag, and breathing through mouthpiece and valves, the tap being turned to allow the expired air to escape into the atmosphere. After continuing this for a time known to be sufficient for the oxygen intake to attain a steady value, the tap was turned for a measured interval (usually about 1 min) to allow a sample of expired air to be collected in the bag, the running being continued at the same speed. After the interval, the running ceased, and the measurement and analysis of the expired air were carried out in the usual manner. Experiments were made at a variety of speeds and on several subjects (which amply confirm one another).[2(p156)]

Their observations revealed a near-linear relationship and eventual plateau between $p\dot{V}O_2$ and running speed during discontinuous, incremental exercise (but see the section on Methodological issues). Hill and Lupton's findings evolved into the development of a range of laboratory protocols designed to investigate the $p\dot{V}O_2$ response to incremental exercise, based on the concept of $\dot{V}O_2$ max being achieved when a levelling-off or plateau in $p\dot{V}O_2$ emerged (see Figure 12.1). By the late 1930s boys were participating in laboratory determinations of $\dot{V}O_2$ max.

The first laboratory-based investigations of boys' $\dot{V}O_2$ max were carried out by Robinson[3] and Morse *et al.*[4] in the US, on either side of the Second World War. They determined 6- to 18-year-old boys' $\dot{V}O_2$ max using a treadmill protocol involving a 15 min walk at 3.5 miles $\cdot$ h^{-1} up an 8.6% gradient, followed by a 10 min rest, and a run to exhaustion at a speed of 6 or 7 miles $\cdot$ h^{-1} up an 8.6% gradient. Åstrand's[5] doctoral thesis, published in Scandinavia in 1952, was the first study to report the $\dot{V}O_2$ max of both girls and boys, aged 4–18 years. The three studies reported $\dot{V}O_2$ max in ratio with body mass (mL $\cdot$ kg^{-1} $\cdot$ min^{-1}) but Åstrand insightfully expressed reservations about whether this approach was appropriate with children (see the section on Peak oxygen uptake and body size).

Åstrand[5] criticized the Robinson[3] and Morse *et al.*[4] methodology as 'certainly practical from the investigator's point of view but hardly so from that of the subject, especially if he is 6–10 years old'.[5] He commented that on the basis of the exercise protocol and the post-exercise blood lactate accumulation, 'the work in several cases must have been submaximal',[5(p110)], a point conceded

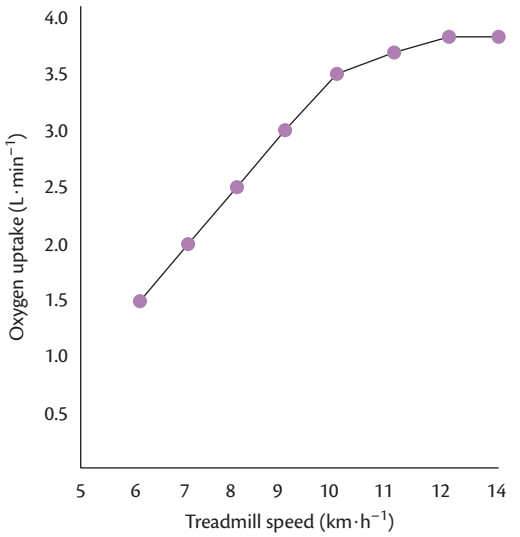

Figure 12.1 Pulmonary oxygen uptake and treadmill speed.
The classical pulmonary oxygen uptake response to incremental treadmill exercise illustrating a near-linear relationship and levelling-off (plateauing) as maximal oxygen uptake approaches.

by Morse *et al.*,[4] who observed that, 'undoubtedly all of the boys did not push themselves to the same state of exhaustion, and some had not reached the limit of their capacity in 5 min of running at 7 m.p.h.'[4(p699)] (see Methodological issues section). In his studies Åstrand used a discontinuous, incremental protocol in which the first session was carried out on a horizontal treadmill running at 7–8 km·h^{-1}. He described subsequent sessions as follows: 'after a couple of days the experiment was repeated with a higher speed of 1–2 km·h^{-1} etc. until the intensity was reached which exhausted the subject in 4–6 min. The determinations for each subject were spread over a period of 3 weeks or more'.[5(p19)] The vast majority of subsequent investigations of young people's $\dot{V}O_2$ max followed Åstrand's lead and adopted either a discontinuous or (more recently) a continuous, incremental exercise protocol to voluntary exhaustion.

Oxygen uptake response to incremental exercise

Incremental exercise to $\dot{V}O_2$ max requires the cardiopulmonary oxygen delivery and muscle oxygen utilization mechanisms to accommodate the rising metabolic demands. Interested readers are invited to peruse Chapter 10 for a comprehensive review of pulmonary function during exercise and Chapter 11 for an insightful analysis of cardiovascular function during exercise, but the main points can be summarized as follows.

Pulmonary ventilation ($\dot{V}_E$) increases in accord with exercise intensity, but it is primarily driven by carbon dioxide (CO_2) production and the need to minimize metabolic acidosis. Pulmonary ventilation is therefore only matched with exercise intensity and $p\dot{V}O_2$ until the ventilatory threshold (T_{VENT}) is reached. The T_{VENT} is defined as the point during incremental exercise at which $\dot{V}_E$ begins to increase out of proportion to the increase in $p\dot{V}O_2$. Beyond the T_{VENT} the bicarbonate buffering of hydrogen ions accompanying lactic acid dissociation to lactate causes CO_2 and therefore $\dot{V}_E$ to rise faster than $p\dot{V}O_2$. As $\dot{V}O_2$ max is approached, a further reduction in blood pH causes $\dot{V}_E$ to compensate (ventilatory compensation point) by increasing at a disproportionately higher rate than carbon dioxide expired ($\dot{V}CO_2$).

The general pattern of the $\dot{V}_E$ response to progressive exercise is similar in children and adults, but there are clear age and maturity differences in the quantitative and relative responses of $\dot{V}_E$. Data on sex differences in the pulmonary response to exercise are equivocal.

Children have a higher ratio of respiratory frequency (f_R) to tidal volume than adults and during maximal exercise a f_R > 60 breaths·min^{-1} is not uncommon compared with ~40 breaths·min^{-1} in adults. Children display a higher $\dot{V}_E$ and therefore a less efficient response to a given metabolic demand than adults, which suggests that there is some maturation of the ventilation control mechanisms during childhood and adolescence. However, gas exchange in the alveoli is determined by alveolar, rather than pulmonary, ventilation and young people's alveolar ventilation is more than adequate to optimize gas exchange. Although at $\dot{V}O_2$ max the ventilatory equivalent ($\dot{V}_E/\dot{V}O_2$) is generally lower in adults than in children, $\dot{V}_E$ at $\dot{V}O_2$ max seldom exceeds ~70% of maximal voluntary ventilation. With healthy children and adolescents, $\dot{V}_E$ does not normally limit $\dot{V}O_2$ max and will therefore not be considered further in this chapter.[6,7]

Oxygen delivery in the blood and subsequent uptake by the muscles is conventionally described by the Fick equation, where $p\dot{V}O_2$ is the product of cardiac output ($\dot{Q}$) and arteriovenous oxygen difference (a-vO_2 diff), where $\dot{Q}$ is the product of heart rate (HR) and stroke volume (SV). Ethical and methodological issues related to the determination of $\dot{Q}$, SV, and a-vO_2 diff during exercise have clouded the interpretation of cardiovascular data, but the introduction of technologies such as Doppler echocardiography, thoracic bioimpedance, and near-infra red spectroscopy (NIRS) has clarified responses to incremental exercise.

Heart rate rises in a near-linear manner before tapering prior to reaching HR max. Maximal HR is independent of age during youth and typical mean values at $\dot{V}O_2$ max on a treadmill and a cycle ergometer are ~200 and ~195 beats·min^{-1}, respectively.[8] In the upright position untrained young people's SV rises progressively with incremental exercise to values ~30–40% higher than resting but at ~50% of $\dot{V}O_2$ max SV plateaus and remains stable to the end of the test.[9] In contrast, trained young people's SV has been reported to increase progressively to exhaustion.[10] (Interested readers are referred to Chapter 34 for more detailed discussion). Stroke volume and $\dot{Q}$ are normally expressed in relation to body surface area, as the stroke or cardiac index respectively. Prepubertal boys' peak cardiac index has been reported to be ~10% higher than that of prepubertal girls, but in both sexes values appear to remain stable from ~10 years of age into young adulthood.[11]

Investigations of a-vO_2 diff during youth are sparse but a-vO_2 diff has been observed to increase with incremental exercise before plateauing at near-maximal exercise in both children and adults, with adults having a greater maximum a-vO_2 diff than children.[12] Data are equivocal, but at least one study has reported prepubertal boys to have a significantly higher maximum a-vO_2 diff than prepubertal girls.[13]

Blood lactate accumulation

At rest, lactate is continuously produced in skeletal muscles, but with the onset of exercise there is an increased production and accumulation of lactate in the muscles. Muscle lactate accumulation is a dynamic process where active muscle fibres produce lactate and adjacent fibres simultaneously consume it as an energy source.

Some of the lactate diffuses into the blood where it can be sampled and assayed to provide an estimate of the anaerobic contribution to exercise and therefore an indication of submaximal aerobic fitness. Lactate is, however, continuously removed from the blood by oxidation in the heart or skeletal muscles or through conversion to glucose in the liver or kidneys. Blood lactate accumulation must therefore be interpreted cautiously as lactate sampled in the blood cannot be assumed to reflect a consistent or direct relationship with muscle lactate production.

Hill and Lupton[2] described the production of lactate in humans in relation to the 'limit of muscular exertion', but much of the subsequent research concerned the interpretation of blood lactate accumulation during submaximal exercise and was initially published in the post-Second World War German literature. The hypothesis of an 'anaerobic threshold' to describe blood lactate accumulation during progressive exercise was popularized in the 1970s, but more recent research has both challenged and defended the threshold hypothesis.[14] Current thought on lactate thresholds in adults can be found in the work of Wassermann and his colleagues.[15]

Blood lactate accumulation during incremental exercise

During an incremental exercise test to exhaustion blood lactate accumulation typically increases, as illustrated in Figure 12.2. The onset of the test stimulates minimal change in blood lactate accumulation, which often does not significantly rise above resting values. It is not unusual for blood lactate accumulation to initially increase and then fall back to near resting values due to the interplay between type I and type II muscle fibre recruitment. However, as the exercise progresses blood lactate accumulation gradually increases until an inflection point is reached where lactate begins to accumulate rapidly. The blood lactate accumulation inflection point is defined as the lactate threshold (T_{LAC}), which serves as a useful estimate of submaximal aerobic fitness.[16]

The highest exercise intensity which can be sustained without incurring a progressive increase in blood lactate accumulation is termed the maximal lactate steady state (MLSS). It corresponds to the highest point at which the diffusion of lactate into the blood and removal from the blood are in equilibrium. Exercise can be sustained for prolonged periods at or below the MLSS, and it therefore has the potential to provide an indicator of aerobic fitness, but for methodological reasons secure data from children and adolescents are not currently available.[17]

To avoid taking multiple blood samples from young people, non-invasive alternatives to blood lactate reference values have become the preferred option in many paediatric exercise science laboratories. Robust methods have been developed to determine and evaluate the T_{VENT}[18] (or V-slope[19]) and the critical power (CPo)[20] of children. The V-slope, which is often the preferred method of estimating a threshold, is determined using linear regression to detect the point at which $\dot{V}CO_2$ begins to rise at a more rapid rate than $p\dot{V}O_2$, and is independent of the $\dot{V}_E$ response. Critical power is defined as the power asymptote of the theoretical hyperbolic relationship between muscle power output and the time to exhaustion. The T_{VENT} (or V-slope) and CPo are often used to replace T_{LAC} and MLSS respectively, for example, in defining exercise domains[21] and monitoring training programmes.[22]

Peak blood lactate accumulation following an exercise test to exhaustion has been used routinely to estimate whether a young person has given a maximal effort.[23] Some authors have advocated the use of specific values of post-exercise blood lactate accumulation (e.g. 6 to 9 $mmol \cdot L^{-1}$) to confirm maximal efforts during tests to determine peak $\dot{V}O_2$.[24] There is, however, considerable variability in young people's blood lactate accumulation. Post-exercise values of blood lactate accumulation at peak $\dot{V}O_2$ of untrained 11- to 13-year-olds have been observed to range from 4 to 13 $mmol \cdot L^{-1}$, using the same exercise protocol, blood sampling, and assay techniques.[25] Post-exercise blood lactate accumulation is dependent on mode of exercise, protocol employed (see variations of blood lactate accumulation with different exercise test protocols in Table 12.1), and timing of the post-exercise blood sample relative to the cessation of the exercise.[26] The recommendation of a specific minimum post-exercise blood lactate accumulation to validate peak $\dot{V}O_2$ as a maximal effort during youth is therefore untenable.

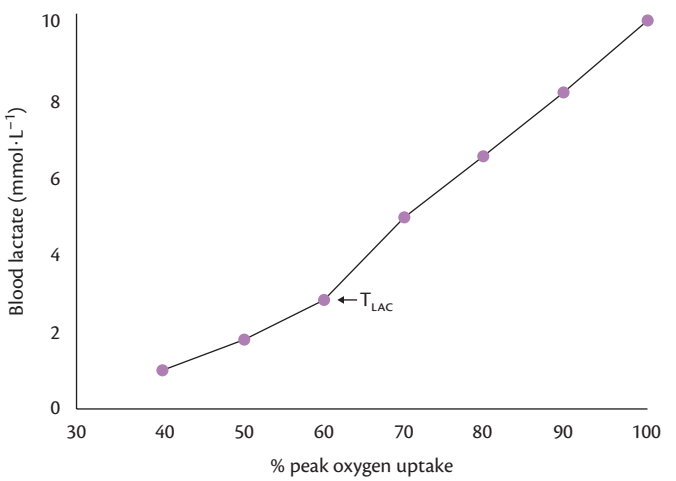

Figure 12.2 Blood lactate accumulation and % peak oxygen uptake.
Blood lactate accumulation in relation to % peak oxygen uptake during incremental exercise. The lactate threshold (T_{LAC}) is illustrated as the point where blood lactate begins to accumulate more rapidly (the inflection point).

Table 12.1 Peak physiological data of 9-year-olds across three maximal exercise tests

Boys	**Test 1**	**Test 2**	**Test 3**
Oxygen uptake ($L \cdot min^{-1}$)	1.93 (0.23)	1.95 (0.24)	1.98 (0.17)
Heart rate ($beats \cdot min^{-1}$)	203 (8)	196 (10)*	196 (7)*
Respiratory exchange ratio	0.99 (0.05)	1.15 (0.07)*	1.18 (0.09)*
Blood lactate ($mmol \cdot L^{-1}$)	5.7 (1.7)	8.4 (2.2)*	9.3 (1.9)*
Girls	**Test 1**	**Test 2**	**Test 3**
Oxygen uptake ($L \cdot min^{-1}$)	1.85 (0.28)	1.90 (0.26)	1.91 (0.35)
Heart rate ($beats \cdot min^{-1}$)	211 (9)	205 (9)*	206 (10)*
Respiratory exchange ratio	1.00 (0.04)	1.13 (0.06)*	1.13 (0.06)*
Blood lactate ($mmol \cdot L^{-1}$)	6.4 (1.3)	8.3 (1.3)*	8.3 (2.1)*

Values are mean (standard deviation).

* Mean significantly different ($p < 0.05$) from test 1.

Source data from Armstrong N, Welsman JR, Winsley RJ, Is peak $\dot{V}O_2$ a maximal index of children's aerobic fitness? Int J Sports Med. 1996; 17: 356–359.

Pulmonary oxygen uptake kinetics

A high $\dot{V}O_2$ max and/or the ability to sustain submaximal exercise are prerequisites of elite performance in some sports, but exercise of the intensity and duration required to elicit $\dot{V}O_2$ max or to sustain performance at the T_{LAC} or MLSS is rarely experienced by most young people.[27] The outcome is that there is no meaningful relationship between daily (or habitual) physical activity during youth and either $\dot{V}O_2$ max[28] or blood lactate indices of aerobic fitness.[29] The vast majority of young people's daily physical activity is intermittent and consists of periods of rest interspersed with physical activity of short duration. Furthermore, in many sports the ability to engage in rapid changes in exercise intensity is at least as important as $\dot{V}O_2$ max or T_{LAC}. Under these circumstances, it is the kinetics of $p\dot{V}O_2$ which best reflect the effective integrated response of the pulmonary, circulatory, and muscular systems.

The introduction of breath-by-breath respiratory gas exchange technology in the late 1960s enabled innovative scientists, including Margaria, Wasserman, and Whipp, to map out the kinetic response of $p\dot{V}O_2$ following the onset of exercise. Critical reviews of the assessment and interpretation of the respiratory gas kinetics of both adults[30] and youth[31] are available elsewhere. The following paragraphs summarize current understanding of the phenomenon; a more detailed analysis is presented in Chapter 13.

Kinetics of the pulmonary oxygen uptake response at exercise onset

The $p\dot{V}O_2$ response to a step change from rest (experimentally usually from unloaded pedalling on a cycle ergometer) to moderate-intensity exercise (i.e. exercise intensity below the T_{LAC} or T_{VENT}) is characterized by three phases as illustrated in Figure 12.3. Phase I (the cardiodynamic phase), which lasts ~15–20 s in young people, is associated with an increase in $\dot{Q}$ which occurs prior to the arrival at the lungs of venous blood from the exercising muscles and is therefore independent of muscle $\dot{V}O_2$ ($m\dot{V}O_2$). Phase I is followed by an exponential increase in $p\dot{V}O_2$ (phase II) that drives $p\dot{V}O_2$ to a steady state (phase III) within ~2 min. Phase II (the primary component) is described by its time constant (τ), which is the time taken to achieve 63% of the change in $p\dot{V}O_2$. The shorter the primary component τ, the smaller the oxygen deficit and the anaerobic contribution to the energy required for the change in exercise intensity.

In contrast to the $p\dot{V}O_2$ response at the onset of moderate-intensity exercise, a step change from rest to heavy-intensity exercise (i.e. exercise intensity above the T_{LAC} or T_{VENT}, but below CPo or the MLSS) elicits a phase III, where the oxygen cost increases over time as a slow component of $p\dot{V}O_2$ is superimposed and the achievement of a steady state is delayed by ~10 min in children[32] and 10–15 min in adults.[30]

Although largely ignored in the physiology literature for over 60 years, initial indications of the presence of a $p\dot{V}O_2$ slow component lie in the 1913 data of Krogh and Lindhard.[33] Ten years later, Hill and Lupton[2] observed what was probably a $p\dot{V}O_2$ slow component in a subject running at constant speed, but reported that, 'The gradual rise in oxygen consumption is probably to be attributed to a painful blister on the foot causing inefficient movement'.[2(p155)] It required the advent of data from more sophisticated breath-by-breath technology before Gaesser and Poole[34] were able to provide an insightful clarification of the sources of the $p\dot{V}O_2$ slow component. The mechanisms still remain speculative, but compelling arguments suggest that ~85% of the $p\dot{V}O_2$ slow component originates from the exercising muscles, perhaps largely

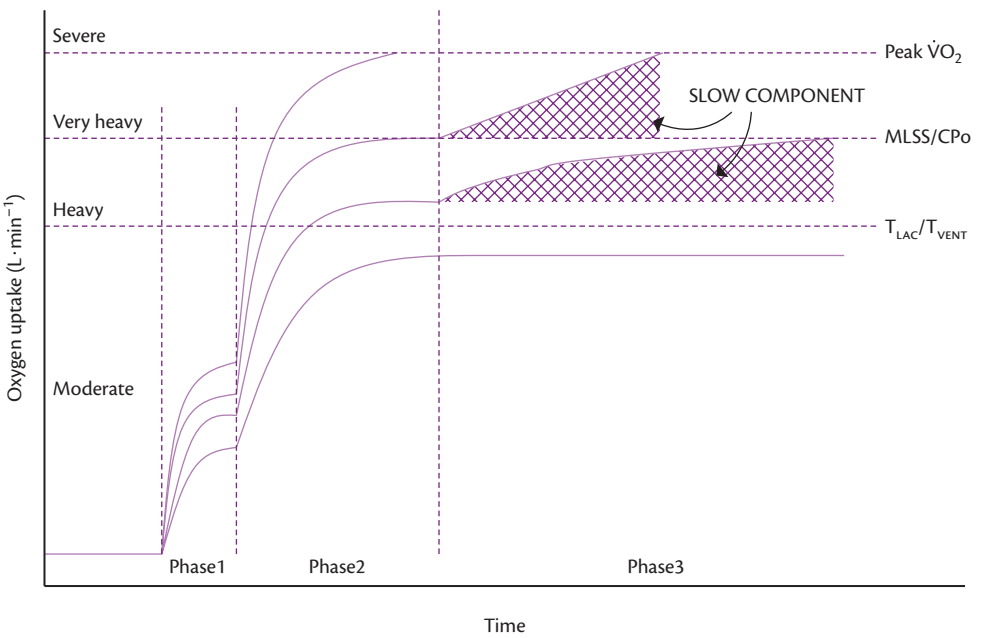

Figure 12.3 Pulmonary oxygen uptake in response to step changes in exercise intensity.
The pulmonary oxygen uptake response at the onset of constant exercise in different exercise intensity domains. The horizontal lines represent the boundaries of the domains where peak $\dot{V}O_2$, MLSS, CPo, T_{LAC}, and T_{VENT} denote peak oxygen uptake, maximal lactate steady state, critical power, lactate threshold, and ventilatory threshold, respectively. The slow component of pulmonary oxygen uptake is represented by the hatched areas.

due to a change in muscle fibre recruitment as exercise progresses. In prepubertal children[32] and adolescents[35] neither the primary component τ nor the $p\dot{V}O_2$ slow component are significantly related to peak $\dot{V}O_2$.

The very heavy-intensity exercise domain encompasses exercise intensities lying between the MLSS (or CPo) and $\dot{V}O_2$ max. In this domain a $p\dot{V}O_2$ steady state is not achieved and in adults the $p\dot{V}O_2$ slow component rises with time and projects to $\dot{V}O_2$ max. The higher the work rate is above CPo the faster the projection of the $p\dot{V}O_2$ slow component to $\dot{V}O_2$ max. This phenomenon has not to date been reported in children where it has been observed that $p\dot{V}O_2$ projects progressively towards peak $\dot{V}O_2$ but stabilizes at ~85–90% of peak $\dot{V}O_2$.[36]

The severe-intensity exercise domain describes step changes in exercise intensity in which the primary component of $p\dot{V}O_2$ is predicted to project to or above $\dot{V}O_2$ max and the maximal rate of $p\dot{V}O_2$ is achieved within 2–3 min of exercise onset. In the severe-intensity exercise domain a $p\dot{V}O_2$ slow component is not discernible from the primary component, but it is unclear whether this is due to the prominence of the primary component of $p\dot{V}O_2$ or insufficient time for a $p\dot{V}O_2$ slow component to be expressed. It has been suggested that as there is a large slow phase in recovery from exercise in this domain it is likely that a $p\dot{V}O_2$ slow component also exists during the onset of exercise.[30]

An alternative classification scheme of $p\dot{V}O_2$ kinetics defines all exercise intensities that achieve $\dot{V}O_2$ max to reside within the severe domain irrespective of whether $p\dot{V}O_2$ projects to $\dot{V}O_2$ max through the primary component or via the $p\dot{V}O_2$ slow component. In this schema there exists a domain termed extreme intensity in which the exercise intensity is so great that fatigue intervenes before $\dot{V}O_2$ max can be attained.[30] As the majority of the paediatric exercise science literature has adopted the terminology illustrated in Figure 12.3 this classification will be adhered to throughout this chapter.

Peak oxygen uptake

Maximal (and later peak) $\dot{V}O_2$ has been the criterion measure of young people's aerobic fitness since the pioneering studies of Robinson,[3] Morse *et al.*,[4] and Åstrand[5], yet the assessment and interpretation of $\dot{V}O_2$ max or (peak $\dot{V}O_2$) during growth and maturation remain shrouded in controversy.[37] This section identifies pertinent methodological issues in the determination of peak $\dot{V}O_2$, clarifies the distinction between $\dot{V}O_2$ max and peak $\dot{V}O_2$, discusses the increase in peak $\dot{V}O_2$ with chronological age, challenges the conventional interpretation of peak $\dot{V}O_2$ in relation to body size; demonstrates the independent contribution of biological maturity to peak $\dot{V}O_2$; and addresses sexual dimorphism in peak $\dot{V}O_2$.

Methodological issues

Maximal or peak oxygen uptake?

Classically, $\dot{V}O_2$ max was determined in a laboratory using a discontinuous, incremental exercise test to exhaustion on a treadmill or cycle ergometer. Typically, the participant exercised at a predetermined, submaximal intensity for about 3–5 min to obtain a steady state $p\dot{V}O_2$ and then rested for ~60 s (in some cases submaximal stages were carried out on different days) before completing a more intense exercise stage. This protocol continued until a stage beyond which a $p\dot{V}O_2$ plateau was reached. The additional energy required to exercise above the point where the $p\dot{V}O_2$ plateau occurred was assumed to be provided exclusively by anaerobic metabolism, resulting in an intracellular accumulation of lactate, acidosis, and eventual termination of exercise. In practice a genuine plateau in $p\dot{V}O_2$ with increasing exercise intensity seldom occurred and less stringent criteria for establishing the existence of a plateau were developed. In order to increase confidence that a true $\dot{V}O_2$ max had been achieved, subsidiary criteria related to HR, respiratory exchange ratio (i.e. $\dot{V}CO_2/p\dot{V}O_2$; R), and blood lactate accumulation at the termination of the $\dot{V}O_2$ max test were introduced.[38]

The $\dot{V}O_2$ plateau concept has retained primacy in the literature as the principal criterion for establishing $\dot{V}O_2$ max, but the validity of the classical model has been a topic of lively debate for several years.[39,40] The practice of reporting submaximal $p\dot{V}O_2$ steady states or describing exercise intensities as % $\dot{V}O_2$ max in the heavy- and very heavy-exercise intensity domains has fallen into disrepute with evidence of a $p\dot{V}O_2$ slow component emerging at exercise intensities above the T_{LAC} in adults,[30] adolescents,[35] and prepubertal children.[32]

Åstrand's[5] studies revealed that a $p\dot{V}O_2$ plateau was found in '70 of 140 running experiments with school children'.[5(p23)] It was subsequently argued by some authors that the failure of some children to elicit a $p\dot{V}O_2$ plateau was related to low motivation or low anaerobic capacity.[41] But others demonstrated that with both prepubertal children[42] and adolescents[43] those who exhibited a $p\dot{V}O_2$ plateau at the termination of an incremental exercise test to voluntary exhaustion were indistinguishable in terms of HR, R, or blood lactate accumulation at test termination from those who did not. This raised the question of whether a $p\dot{V}O_2$ plateau was required to indicate a maximal index of aerobic fitness during youth.

The problem was addressed experimentally by determining the peak $\dot{V}O_2$ of 20 boys and 20 girls, mean age 9.9 years, on three occasions, 1 week apart. On the first occasion, the children completed a discontinuous, incremental protocol on a treadmill with the belt speed held at 1.94 $m \cdot s^{-1}$ but with the gradient increasing every 3 min. The children exercised until voluntary exhaustion. Using a <2 $mL \cdot kg^{-1} \cdot min^{-1}$ increase in $p\dot{V}O_2$ as the criterion, six boys and seven girls exhibited a $p\dot{V}O_2$ plateau. No significant differences in either anthropometrical or peak physiological data were revealed between those who did and did not exhibit $p\dot{V}O_2$ plateaus. The second and third tests were performed at the same belt speed as test one (i.e. 1.94 $m \cdot s^{-1}$) but, following a 3 min warm-up running at 1.67 $m \cdot s^{-1}$, the children ran up gradients which were 2.5% and 5% greater, respectively, than the highest gradient achieved on the first test. The children were strongly motivated and the data were accepted if the child ran for at least 2 min up the higher gradient. Eighteen girls and 17 boys completed all tests and although they exhibited significantly higher post-exercise blood lactate accumulation, peak $\dot{V}_E$, and peak R in tests two and three than in the initial test, as illustrated in Table 12.1, there were no significant differences in peak $\dot{V}O_2$ across the three tests.[44]

These data and those from a similar study[45] imply that with well-motivated children 'true' $\dot{V}O_2$ max values can be achieved in a single, incremental test to exhaustion despite the majority of participants not demonstrating a $p\dot{V}O_2$ plateau. There is, however, no easy solution to the problem of whether an individual child has delivered a maximal effort in an incremental test to exhaustion. Habituation to the laboratory environment, subjective criteria of intense effort (e.g. facial flushing, sweating, hyperpnoea, unsteady gait), and the

paediatric exercise testing experience of the experimenters are vital ingredients in making this decision. Various other physiological indicators of a maximal effort such as HR and R at peak $\dot{V}O_2$ and peak post-exercise blood lactate accumulation have been proposed as subsidiary criteria,[46] but, as illustrated by the data described in Table 12.1, they are all protocol dependent. Furthermore, there is no 'one size fits all' criterion of a maximal effort. For example, at the termination of an incremental treadmill exercise test HR at peak $\dot{V}O_2$ has a mean ± standard deviation of ~200 ± 7 beats · min^{-1}, in the age range 8–16 years.[8] As ~95% of young people's HRs at peak $\dot{V}O_2$ would therefore be expected to fall in the range 186–214 beats · min^{-1} it is futile to interpret a spot HR of, for example, 195 or 200 beats · min^{-1} as is commonly advocated, as reflecting maximal effort. (Interested readers are referred to Chapter 11 for further detail of the cardiovascular response to exercise).

As the term $\dot{V}O_2$ max conventionally requires a $p\dot{V}O_2$ plateau to be exhibited, it has become common practise in paediatric exercise science to define the highest $p\dot{V}O_2$ observed during a progressive exercise test to exhaustion as peak $\dot{V}O_2$ rather than $\dot{V}O_2$ max.

Respiratory gas analysis

The determination of peak $\dot{V}O_2$ depends upon the accurate measurement of inspired and/or expired air per unit of time and the fraction of oxygen and carbon dioxide therein. Automated respiratory gas analysis systems and sophisticated metabolic carts with appropriate calibration facilities are commercially available and commonplace in research laboratories. Paediatric physiologists must, however, be cautious of measuring children's respiratory responses to exercise using apparatus primarily designed for use with adults.

Most respiratory gas analysis systems measure volume using a breathing valve (normally a lightweight turbine or pneumotachograph) connected to the participant via a mouthpiece and nose clip or facemask. With children it is imperative that the mouthpiece and nose clip or facemask is comfortable and appropriately sized to prevent leakage. To prevent the significant inspiration of previously expired air the combined dead space of the mouthpiece/facemask and breathing valve should be minimized, although this must be balanced against the resulting increase in resistance to flow.

To periodically sample respiratory gases metabolic carts normally use either a mixing chamber, which stores expiratory gases over a given interval, or a breath-by-breath system. Large mixing chambers may cause substantial measurement errors as children have smaller exercise tidal volumes than adults. Breath-by-breath systems with rapid gas analysers allow continuous measurement of volume and respiratory gas content and overcome the potential size limitations of mixing chambers. However, breath-by-breath systems are challenged by the large inter-breath variations of exercising children in relation to their $p\dot{V}O_2$ response amplitude (i.e. high noise-to-signal ratio).[47]

In addition, the breath-by-breath gas sampling interval can have a significant impact on the reported $p\dot{V}O_2$. Short sampling intervals increase the variability in measuring $p\dot{V}O_2$ and with their smaller peak $\dot{V}O_2$ this is more marked in children than in adults. However, large sample intervals may 'over-smooth' the data and artificially reduce the 'true' $\dot{V}O_2$ response. A sampling interval of ~15–30 s is optimum for children and adolescents, but whatever the chosen interval it should be recorded and reported to allow cross-study comparisons.[48]

Ergometry

Young people's peak $\dot{V}O_2$ has been determined using a wide range of ergometers, and although it is important to simulate competitive performance when testing and monitoring young athletes, cycles and treadmills remain the ergometers of choice in most paediatric exercise science laboratories.

Cycle ergometry provides a portable, relatively cheap, and more quantifiable mode of exercise than treadmill running and it tends to induce less anxiety in young children. Cycle ergometer crank lengths may need to be modified for young children who sometimes experience difficulty with the need to maintain a fixed pedal rate when cycling on mechanically braked ergometers. Electronically braked cycle ergometers which adjust resistance to pedalling frequency alleviate this difficulty to some extent, but the increase in resistance required to maintain exercise intensity following a reduction in pedal rate may in itself cause problems with young children.

Limited upper body movement during cycle ergometry facilitates the measurement of ancillary variables such as HR, blood pressure, and blood lactate accumulation. However, a disadvantage of cycle ergometry with young children is that a high proportion of the total power output is developed by the quadriceps muscles[49] and the effort required to push the pedals during the later stages of an incremental test may be high in relation to children's muscle strength.[50] This leads to blood flow through the quadriceps being restricted and results in increased anaerobic metabolism and consequent termination of the test through peripheral muscle fatigue.[51]

Treadmill running engages a larger muscle mass than cycling. The increased venous return and reduced peripheral resistance during running enhances $\dot{Q}$, and peak $\dot{V}O_2$ is more likely to be limited by central than peripheral factors. Peak $\dot{V}O_2$ is typically about 8–10% higher during treadmill running than cycle ergometry, although some adolescents have been reported to achieve higher peak $\dot{V}O_2$ on a cycle ergometer. Pearson product-moment correlations between peak $\dot{V}O_2$ rigorously determined on a treadmill and a cycle ergometer are ~0.90.[52]

Exercise protocols

Peak $\dot{V}O_2$ during youth is a robust variable which, on a specific ergometer, is normally independent of exercise protocol[53] with a coefficient of variation in repeated tests of ~5% on both treadmill and cycle ergometer.[52]

Incremental, continuous, or discontinuous protocols on a treadmill have traditionally been the exercise tests of choice in paediatric exercise research laboratories.[54] However, with clear experimental evidence that children[55] and adolescents[35] exhibit a $p\dot{V}O_2$ slow component during exercise above the T_{LAC}, the availability of commercial breath-by-breath metabolic carts, and the development of electromagnetically braked cycle ergometers, ramp protocols have become popular. In many paediatric exercise science laboratories ramp cycle tests, where power output is increased linearly with time, have replaced classical, discontinuous, 'steady state', incremental protocols. Ramp protocols have the advantages of flexibility of rate and magnitude of power output, short test duration (~10 min), and the ability to determine other parameters of cardiopulmonary function (e.g. V-slope) during a single test.

In a single ramp test to exhaustion a $p\dot{V}O_2$ plateau is an infrequent occurrence. However, a study of 10- and 11-year-old

children, across three ramp tests each 1 week apart, reported a typical error in peak $\dot{V}O_2$ of ~4%, which compares favourably with the reliability of adults' $\dot{V}O_2$ max, regardless of protocol.[56] A short duration ramp test coupled with children's ability to recover quickly from exhaustive exercise[57] allows the use of a follow-up supramaximal test to verify whether a maximal effort was elicited in the initial test.

The following protocol has been found to be appropriate for children: After a 3 min period of cycling at 10 W, participants undertake a ramp incremental test to exhaustion with power output increasing by 10 W · min^{-1}. Cycling cadence is maintained at 75 revs · min^{-1} throughout the test and exhaustion is defined as a drop in pedal cadence below 60 revs · min^{-1} for 5 consecutive seconds. Immediately after exhaustion, power output is reduced to 10 W and the child cycles at this intensity for 10 min followed by 5 min of rest. The participant then performs a supramaximal test consisting of 2 min pedalling at 10 W, followed by a step transition to 105% of the peak power achieved during the ramp test. The pedalling cadence is maintained at 75 revs · min^{-1} with the same criterion as in the initial test to define exhaustion. The power output is then returned to 10 W until the HR has recovered to ~120 beats · min^{-1}. With prepubertal children the time to exhaustion in the supramaximal test is ~90 s. On the rare occasions (<5%) that the peak $\dot{V}O_2$ is higher than in the ramp test, the supramaximal test can be repeated at 110% of peak power following full recovery.[58]

Peak oxygen uptake and chronological age

The peak $\dot{V}O_2$ of children and adolescents has been extensively documented with data available from children as young as 3 years of age. The validity of peak $\dot{V}O_2$ determinations in children younger than 8 years has been questioned since the original studies of Robinson.[3] He noted that, 'the youngest boys were unwilling to continue work after it ceased to be fun, whereas all of the boys of 8 years and older could be encouraged to carry on for some time after the first signs of fatigue'.[3(p281)] As very young children typically have short attention spans, poor motivation, and lack sufficient understanding of experimental procedures it is difficult to elicit genuine maximal efforts.[59] Equipment and protocols designed for adults make exercise testing with young children problematic, and the smaller the child, the greater the potential problem. Reports of peak $\dot{V}O_2$ in very young children are often difficult to interpret. Small sample sizes are common and several studies have pooled data from boys and girls. Whether the children exhibited maximal values is unclear in some reports, and there is a strong tendency to report only mass-related data (mL · kg^{-1} · min^{-1}).[46]

One study suggested that it is possible with rigorous techniques to estimate the peak $\dot{V}O_2$ of most young children and reported achieving 'maximal' values in ~84% of 706 6- to 7-year-olds. Boys were noted to have peak $\dot{V}O_2$ values (L · min^{-1}) ~11% higher than girls, confirming the importance of not pooling boys' and girls' values and reporting data in relation to sex, even at a young age.[60] There are, however, few secure data from children aged less than 8 years in the literature and the focus herein will therefore be on the age group 8–18 years.

A comprehensive review of the extant literature generated graphs representing ~10 000 peak $\dot{V}O_2$ determinations of untrained eight- to 16-year-olds. Because of the ergometer dependence of peak $\dot{V}O_2$ data from treadmill and cycle ergometry were graphed separately and the treadmill-determined peak $\dot{V}O_2$ values (n = 4937) are illustrated in Figure 12.4. The data must be interpreted cautiously, as means from a range of studies with varying sample sizes are included. No information is available on randomly selected groups of young people, and since participants are generally volunteers selection bias cannot be ruled out. This type of analysis tends to smooth data, but Figure 12.4 clearly illustrates a near-linear increase in peak $\dot{V}O_2$ in relation to age. Linear regression equations indicate that peak $\dot{V}O_2$ increases by ~80% from 8 to 16 years in girls and by ~150% in boys over the same time period.[46]

Longitudinal studies provide a more granular analysis of peak $\dot{V}O_2$ in relation to age, but few longitudinal studies have reported data from a broad age range and coupled rigorous determination of peak $\dot{V}O_2$ with substantial sample sizes. Data from rigorous longitudinal studies of treadmill-determined peak $\dot{V}O_2$ are illustrated in Figure 12.5 but between studies comparisons should be interpreted with caution.

Longitudinal data from boys are consistent, with a similar trend to that shown in Figure 12.4. The pooled data show an increase in peak $\dot{V}O_2$ of ~150%, from 8 to 18 years, with the largest annual increases occurring between 13–15 years. It has been suggested that the greatest increase in boys' peak $\dot{V}O_2$ accompanies the attainment of peak height velocity (PHV),[66] but others[67] have noted a stable increase in peak $\dot{V}O_2$ from 3 years before to 1 year after PHV. Longitudinal data from girls are sparse and when pooled they indicate an increase in peak $\dot{V}O_2$ of ~98%, from 8 to 17 years. One study observed a growth spurt in peak $\dot{V}O_2$ aligned with PHV,[66] but when data across studies are compared they suggest, on balance, that girls' peak $\dot{V}O_2$ rises progressively from 8 to 13 years and then begins to level off from ~14 years. A trend also noted in some cross-sectional studies.[46]

The most comprehensive longitudinal study reported is the Amsterdam Growth and Health Longitudinal Study (AGHLS) which followed 12- to 14-year-old boys and girls for a period of 25 years.[68] Boys demonstrated a linear increase in peak $\dot{V}O_2$ of

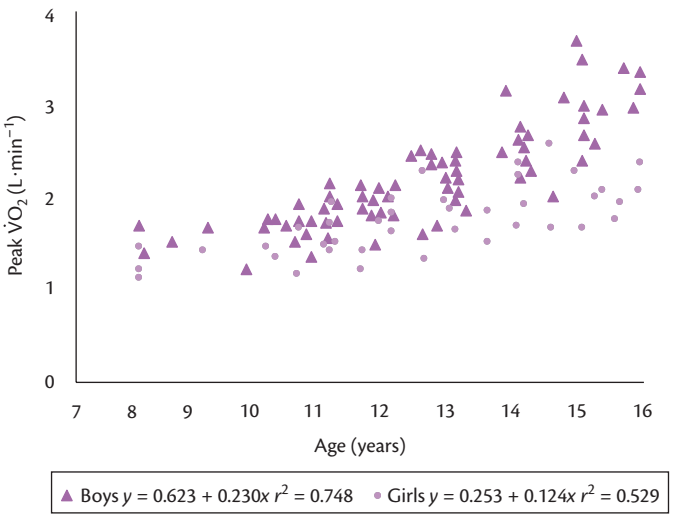

Figure 12.4 Peak oxygen uptake by chronological age and sex.
Treadmill-determined peak oxygen uptake in relation to chronological age and sex in 8- to 16-year-olds. Figure describes peak oxygen uptake data on 3703 boys and 1234 girls.
Source data from Armstrong N, Welsman JR. Assessment and interpretation of aerobic fitness in children and adolescents. Exerc Sport Sci Rev. 1994; 22: 435–476.

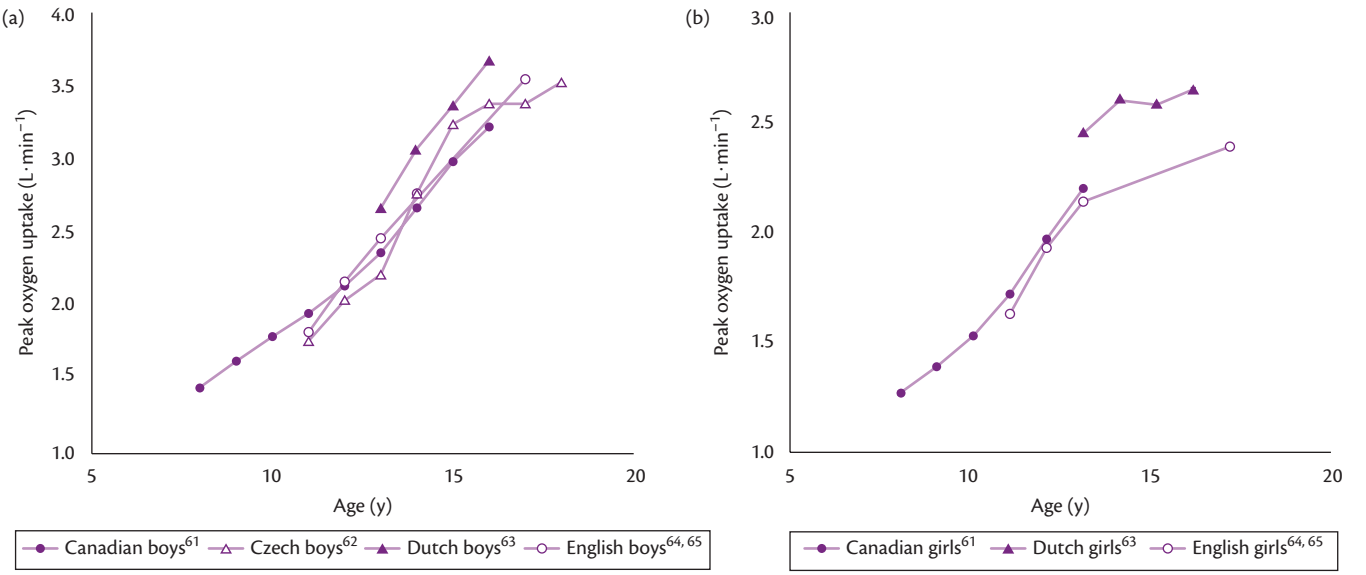

Figure 12.5 (a) Longitudinal studies of boys' peak oxygen uptake and chronological age. (b) Longitudinal studies of girls' peak oxygen uptake and chronological age. Treadmill-determined peak oxygen uptake in relation to age in 8- to 18-year-olds. Figure 12.5a drawn from data in four longitudinal studies[61–65] with 1818 determinations of boys' peak oxygen uptake. Figure 12.5b drawn from data in three longitudinal studies[61,63–65] with 707 determinations of girls' peak oxygen uptake.

~57%, from age 12–17 years. Girls' values increased by ~11% over the same time period, with a marked levelling-off from 14–17 years (~2% change).[69] The Dutch data[63] contrast with a mixed longitudinal study from England, which reported increases in peak $\dot{V}O_2$, from 12–17 years, of ~70% and ~24% for boys and girls respectively.[65] As Dutch values at age 12 years were ~12% and ~22% higher than English values, for boys and girls respectively, the conflicting data may be at least partially explained by the initial high level of aerobic fitness of Dutch youth compared with English youth.

Peak oxygen uptake and body mass

Peak $\dot{V}O_2$ is strongly correlated with body mass and, in particular, with lean body mass (LBM). Much of the age-related increase in peak $\dot{V}O_2$ illustrated in Figure 12.4 reflects the increase in muscle mass during the transition from childhood into young adulthood. Because of the problems in assessing LBM, researchers have conventionally focused on controlling for body mass differences by dividing peak $\dot{V}O_2$ by total body mass and expressing it as the simple ratio $mL \cdot kg^{-1} \cdot min^{-1}$ (ratio scaling).[46]

When peak $\dot{V}O_2$ is expressed in this manner a different picture emerges from that apparent when absolute values ($L \cdot min^{-1}$) are used. Cross-sectional data indicate that boys' mass-related peak $\dot{V}O_2$ decreases slightly or remains unchanged at ~48 $mL \cdot kg^{-1} \cdot min^{-1}$, from 8 to 18 years, while in girls a progressive decline, from ~45–35 $mL \cdot kg^{-1} \cdot min^{-1}$, is apparent. Boys consistently demonstrate higher mass-related peak $\dot{V}O_2$ than girls throughout childhood and adolescence, with the sex difference being reinforced by the greater accumulation of body fat by girls in puberty.[46] The AGHLS data are intriguing in this context as, in conflict with the extant literature, they indicate that from 12–17 years boys' peak $\dot{V}O_2$ decreases from ~59–52 $mL \cdot kg^{-1} \cdot min^{-1}$ and girls' values fall from ~57–45 $mL \cdot kg^{-1} \cdot min^{-1}$.[69]

Although informative in relation to the performance of, for example, track athletes who carry their body mass,[70] the conventional use of ratio values has clouded the physiological understanding of peak $\dot{V}O_2$ during growth. Rather than removing the influence of body mass, ratio scaling 'over scales' and favours light individuals and penalizes heavy individuals. Tanner[71] described the fallacy of ratio scaling in 1949 and Åstrand[5] noted its limitations in relation to expressing children's peak $\dot{V}O_2$ in 1952, but its use has persisted in the paediatric literature. The interpretation of exercise performance data in relation to body size has been critically reviewed elsewhere,[72] but the inadequacy of ratio scaling can be explained simply.

To create a size-free variable in this context requires a product-moment correlation coefficient between peak $\dot{V}O_2$, expressed in $mL \cdot kg^{-1} \cdot min^{-1}$, and body mass in kg, which is not significantly different from zero. Significant negative correlations between ratio scaled peak $\dot{V}O_2$ and body mass have been reported on numerous occasions,[72] but data drawn from the first year of a longitudinal study[64] of 11- to 13-year-olds and summarized in Figure 12.6 clearly illustrate the phenomenon. Figure 12.6a shows significant positive correlations between peak $\dot{V}O_2$ ($L \cdot min^{-1}$) and body mass (kg). Figure 12.6b describes the presence of significant negative correlations between ratio scaled peak $\dot{V}O_2$ ($mL \cdot kg^{-1} \cdot min^{-1}$) and body mass (kg), and confirms the inability of the simple ratio to remove the influence of body mass from peak $\dot{V}O_2$. Figure 12.6c, however, presents the same data and shows that correlations between allometrically scaled peak $\dot{V}O_2$ ($mL \cdot kg^{-0.68} \cdot min^{-1}$) and body mass (kg) are not significantly different from zero. Body mass has therefore been appropriately controlled for using allometric scaling with, in this case, a common mass exponent of 0.68.

Several studies have generated data illustrating how inappropriate ratio scaling has led to misplaced interpretation of physiological variables, whereas studies in which the use of more appropriate means of controlling for body size have provided new insights into peak $\dot{V}O_2$ during growth.[72] For instance, an early exploration of scaling children's peak $\dot{V}O_2$ used a simple linear regression model to investigate changes in peak $\dot{V}O_2$ with chronological age in two groups of boys aged 10 and 15 years. The mean values for peak $\dot{V}O_2$ were 1.73 and 3.12 $L \cdot min^{-1}$ respectively, but when ratio scaled, the two groups had identical mean values of 49 $mL \cdot kg^{-1} \cdot min^{-1}$. However, the regression lines for the relationship between peak $\dot{V}O_2$ and body mass described two clearly different populations (see Figure 12.7). Intuitively this appears

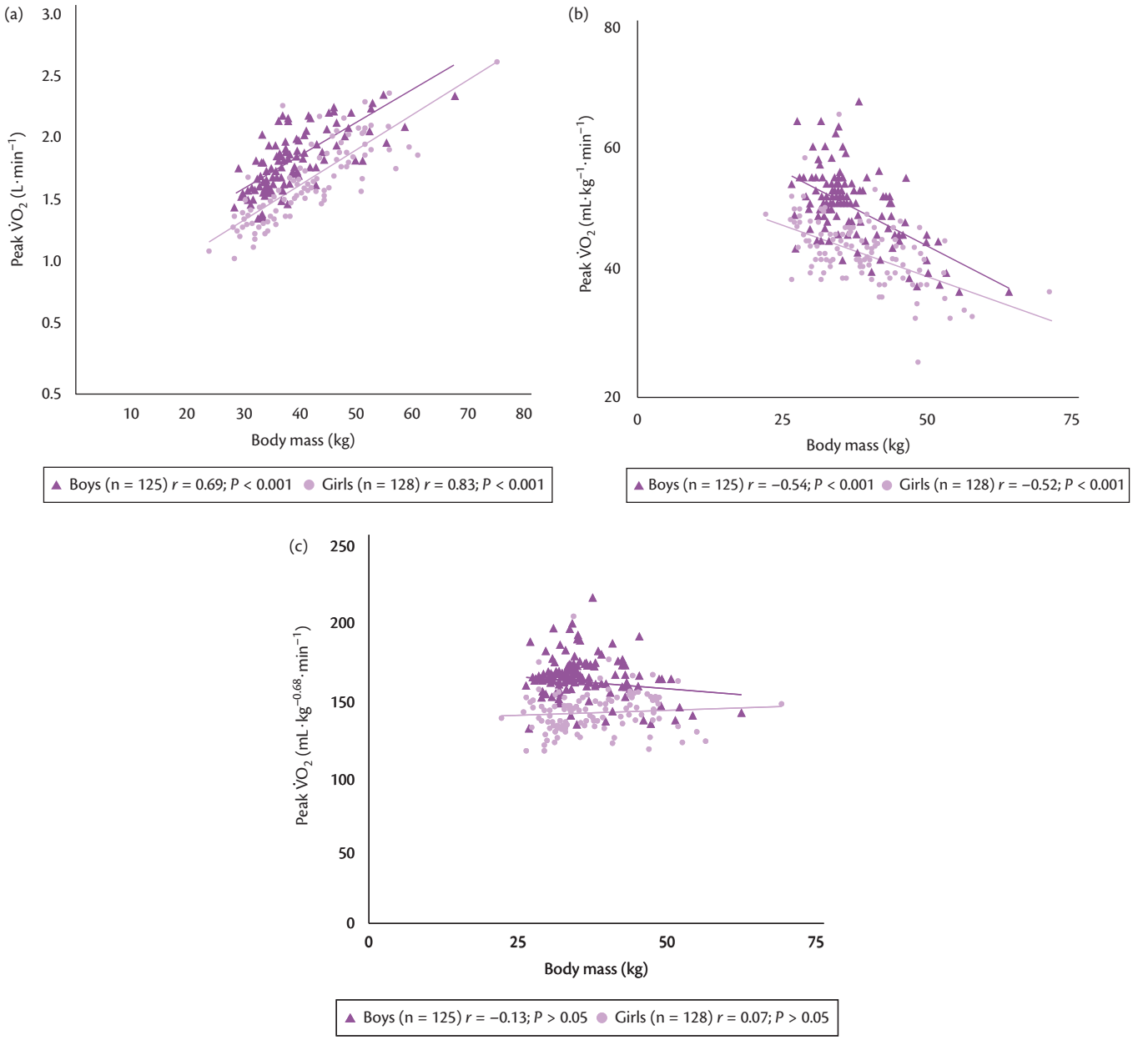

Figure 12.6 (a) Peak oxygen uptake and body mass in 11-year-olds:
This shows significant positive relationships between peak oxygen uptake and body mass in both boys (***r*** = 0.69) and girls (***r*** = 0.83).
(b) Ratio scaled peak oxygen uptake and body mass in 11-year-olds:
This shows significant negative relationships between ratio scaled peak oxygen uptake and body mass in both boys (***r*** = −0.54) and girls (***r*** = −0.52) and illustrates the failure of ratio scaling to deliver a body mass free variable.
(c) Allometrically scaled peak oxygen uptake and body mass in 11-year-olds:
This shows relationships between allometrically scaled peak oxygen uptake and body mass in both boys (***r*** = −0.13) and girls (***r*** = 0.07) are not significantly different from zero and illustrates that body mass has been appropriately controlled for using allometric scaling, with a common mass exponent of 0.68.
Source data from Armstrong N, Welsman JR, Nevill AM, Kirby BJ. Modeling growth and maturation changes in peak oxygen uptake in 11–13 yr olds. J Appl Physiol. 1999; 87: 2230–2236.

appropriate and is in accord with the observed differences in 10- and 15-year-olds' performance in athletic events primarily dependent on aerobic fitness.[73]

A more sophisticated analysis[74] avoiding the limitations of linear regression scaling[75] used both ratio and allometric (log-linear analysis of covariance) scaling to partition size effects from peak $\dot{V}O_2$ data in groups of males and females spanning the age range 11–23 years (see Table 12.2). The results of the ratio analyses conformed to the conventional interpretation with mass-related peak $\dot{V}O_2$ consistent across the three male groups (11, 14, and 23 years). In the females mass-related peak $\dot{V}O_2$ did not change from 11–13 years, but there was a significant decrease in peak $\dot{V}O_2$ from 13–22 years. In direct contrast, allometric scaling revealed significant, progressive increases in peak $\dot{V}O_2$ across male groups demonstrating that, with body size appropriately controlled for, peak $\dot{V}O_2$ is, in fact, increasing during growth rather than remaining static. In females, peak $\dot{V}O_2$ increased significantly from 11–13 years, subsequently remaining constant with no decline into adulthood evident.[74]

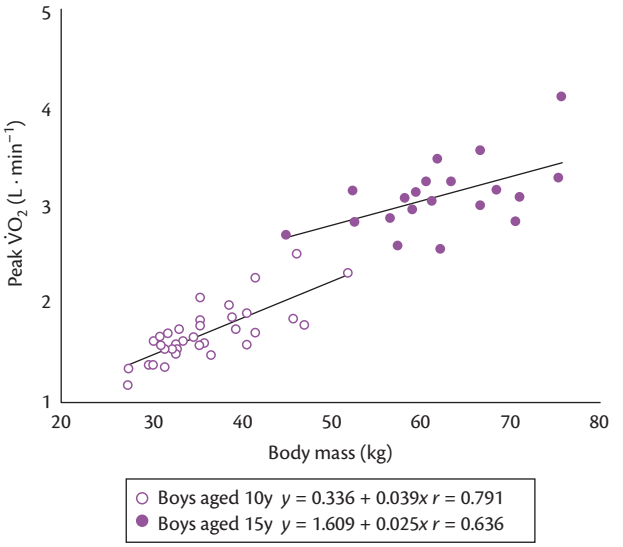

Figure 12.7 Peak oxygen uptake and body mass in 10- and 15-year-old boys. Mean values for peak $\dot{V}O_2$ 1.73 and 3.12 L·min^{-1} for 10- and 15-year-olds respectively. Ratio scaled identical values of 49 mL·kg^{-1}·min^{-1} were revealed; however, the regression lines for the relationship between peak $\dot{V}O_2$ and body mass describe two clearly different populations. Data from Williams JR, Armstrong N, Winter EM, Crichton N. Changes in peak oxygen uptake with age and sexual maturation in boys: Physiological fact or statistical anomaly? In: Coudert J, Van Praagh E, eds. Pediatric work physiology. Paris: Masson; 1992: 35–37.

The application of allometry to longitudinal data is complex, but its use is increasing and evidence to support the cross-sectional analyses is accumulating. Multilevel modelling techniques represent a sensitive and flexible approach to the interpretation of longitudinal exercise data which enable body size, chronological age, and sex effects to be partitioned concurrently within an allometric framework. The interested reader is referred to Welsman and Armstrong,[75] where the theoretical principles of allometry and multilevel modelling are explained and applied to paediatric data sets.

The independent effect of age on peak $\dot{V}O_2$ was clearly demonstrated in a longitudinal study which used multilevel regression modelling to interpret peak $\dot{V}O_2$ in 11–13-year-old boys and girls. The analysis was founded on 590 peak $\dot{V}O_2$ determinations over three annual occasions.[64] A multiplicative, allometric model was adopted based on the model:

$$\text{Peak}\,\dot{V}O_2(Y) = \text{mass}^{k1} \cdot \text{stature}^{k2} \cdot \exp(\alpha_j + b_j \cdot \text{age} + c \cdot \text{age}^2)\,\varepsilon_{ij}$$

In this model all parameters are fixed, with the exception of the constant (α, intercept term) and age parameters, which are allowed to vary randomly at level two (between individuals), and the multiplicative error ratio ε that varies randomly at level one, describing the error variance between occasions. The subscripts i and j denote this random variation at levels one and two respectively. The variable age is centred on the group mean age of 12.0 years.

In order to allow the unknown parameters to be solved using multilevel regression, the model was linearized by logarithmic transformation and multilevel regression analysis on $\log_e$ y used to solve for the unknown parameters. Once transformed, the initial equation became:

$$\text{Log}_e\,\text{peak}\,\dot{V}O_2(\log_e y) = k_1 \cdot \log_e \text{mass} + k_2 \cdot \log_e \text{stature} + \alpha_j + b_j \cdot \text{age} + c \cdot \text{age}^2 + \log_e(\varepsilon_{ij})$$

From this baseline model the additional explanatory variable sex was incorporated as an indicator variable (i.e. sex, boys = 0; sex,

Table 12.2 Peak oxygen uptake in relation to stage of maturation

Females	**Prepubertal girls (Maturation stage 1) (n = 33)**	**Circumpubertal girls (Maturation stage 3/4) (n = 34)**	**Adult women (n = 16)**
Age (years)	10.7 (0.2)*	13.0 (0.2)**	21.7 (2.8)
Body mass (kg)	32.7 (4.6)*	46.5 (9.6)**	60.5 (6.3)
Peak $\dot{V}O_2$(L·min^{-1})	1.48 (0.2)*	2.14 (0.32)**	2.58 (0.26)
Ratio scaled peak $\dot{V}O_2$ (mL·kg^{-1}·min^{-1})	45 (3)	47 (4)***	43 (3)
Allometrically adjusted peak $\dot{V}O_2$ (L·min^{-1})	1.99*	2.19****	2.13
Males	**Prepubertal boys (Maturation stage 1) (n = 29)**	**Circumpubertal boys (Maturation stage 3/4) (n = 26)**	**Adult men (n = 18)**
Age (years)	10.7 (0.2)*	14.1 (0.3)**	22.8 (2.9)
Body mass (kg)	34.9 (5.4)*	49.5 (8.9)**	78.6 (8.7)
Peak $\dot{V}O_2$(L·min^{-1})	1.76 (0.28)*	2.60 (0.47)**	4.18 (0.47)
Ratio scaled peak $\dot{V}O_2$ (mL·kg^{-1}·min^{-1})	50 (4)	53 (4)	53 (3)
Allometrically adjusted peak $\dot{V}O_2$ (L·min^{-1})	2.25*	2.50**	2.80

Data from Welsman JR, Armstrong N, Kirby BJ, Nevill AM, Winter EM. Scaling peak $\dot{V}O_2$ for differences in body size. Med Sci Sports Exerc. 1996; 28: 259–265.

Values are mean (standard deviation).

level of significance $p < 0.05$.

* Significantly different from circumpubertals and adults.

** Significantly different from prepubertal children and adults.

*** Significantly different from adults.

**** Significantly different from prepubertal children.

Table 12.3 Multilevel regression model for peak oxygen uptake in 11–13-year-olds

Parameters	Estimate (SE)
Fixed	
Constant	−1.3903 (0.0970)
Log_e mass	0.5011 (0.0322)
Log_e stature	0.9479 (0.1162)
Age	0.0585 (0.0111)
Sex	−0.1378 (0.0093)
Age·sex	−0.0134 (0.0068)
Random	
Level 2	
Constant	0.0042 (0.0005)
Age	0.0007 (0.0003)
Covariance	NS
Level 1	
Constant	0.0030 (0.0004

N = 590 NS, not significant.

Data from Welsman JR, Armstrong N, Kirby BJ, Nevill AM, Winter EM Scaling peak VO2 for differences in body size. Med Sci Sports Exerc. 1996; 28: 259–265

girls = 1), which sets the boys' constant as the baseline from which the girls' parameter may deviate. The interaction term age by sex was constructed to investigate whether age effects on peak $\dot{V}O_2$ differed for boys and girls. Age was allowed to vary randomly at level one to investigate within individual variation around the individual growth trajectory. The need to allow each individual their own mass exponent was examined by letting body mass vary at level two. The model is presented in Table 12.3.

The multilevel regression model reveals stature and body mass as significant covariates with an additional significant positive effect for age, which is larger for boys than girls as reflected by the significant age by sex interaction term, which is deducted from the age term for girls. With age, stature, and body mass accounted for, the negative term for sex shows girls' peak $\dot{V}O_2$ to be significantly lower than that of boys. The fixed estimates in the model describe the population mean response, while the random parameters show the variance which remains unaccounted for by the fixed part of the model. Table 12.3 illustrates significant random variation at level two for age, reflecting differential individual growth rates in peak $\dot{V}O_2$. Allowing each individual their own mass exponent proved unnecessary, as there was no random variation between individuals around the fixed (mean) parameter.

Taken together, the cross-sectional and longitudinal data clearly challenge the conventional interpretation of peak $\dot{V}O_2$ during growth in both boys and girls, and demonstrate that there is a progressive increase in peak $\dot{V}O_2$ with chronological age in both sexes, independent of the influence of body size.

Peak oxygen uptake and biological maturation

As young people grow they also mature, and the physiological responses of adolescents must be considered in relation to biological maturity as well as chronological age. Some studies indicate an adolescent growth spurt in peak $\dot{V}O_2$ in boys, with the spurt reaching a maximum gain near the time of PHV, but secure data are insufficient to offer any generalization for girls. With stage of maturity classified using secondary sexual characteristics, more mature young people have been reported to have a higher peak $\dot{V}O_2$ in $L \cdot min^{-1}$ than those less mature, but ratio scaled peak $\dot{V}O_2$ ($mL \cdot kg^{-1} \cdot min^{-1}$) has been reported to be unrelated to state of maturity, indicating no additional effect of biological maturity on peak $\dot{V}O_2$ above that due to growth.[76]

Armstrong *et al.*[77] argued that the true relationship between peak $\dot{V}O_2$ and biological maturity may have been obscured through an inappropriate means of controlling for body mass. They determined the peak $\dot{V}O_2$ of 176 12-year-olds and classified them according to the stages of pubic hair development described by Tanner[78]. In accord with the extant literature, mass-related peak $\dot{V}O_2$ ($mL \cdot kg^{-1} \cdot min^{-1}$) was not significantly different across stages of pubic hair development in either boys or girls. However, when body mass was controlled using allometry (log-linear analysis of covariance with mass as the covariate) peak $\dot{V}O_2$ was demonstrated to significantly increase with biological maturity in both sexes. None of the children were classified as in stage 5 for pubic hair development (PH5), but, with body mass controlled for, boys in PH4 exhibited peak $\dot{V}O_2$ values 14% higher than similarly aged boys in PH1. The corresponding difference in girls was 12%, thus demonstrating that in both boys and girls there is a significant independent effect of biological maturity on peak $\dot{V}O_2$ above that attributed to chronological age and body mass.

Armstrong and Welsman[65] introduced the same criterion of biological maturity into their multilevel regression model of 11–17-year-olds and confirmed their earlier findings on 11–13-year-olds[64] by showing incremental effects of stage of maturity on peak $\dot{V}O_2$ independent of chronological age and body mass (see Table 12.4). The positive effect of biological maturity on aerobic fitness was consistent for both boys and girls. When skinfold thicknesses were introduced into the model, the stage of pubic hair development remained a significant covariate in all but stage PH5, but the magnitudes of the effect were reduced, indicating the relationship between stage of biological maturity and body composition. With body mass, skinfold thicknesses, and pubic hair development accounted for, peak $\dot{V}O_2$ was shown to increase throughout the age range studied in both sexes. The girls' data are noteworthy as earlier longitudinal studies using conventional analyses to control for body mass suggested a decline in females' peak $\dot{V}O_2$ from age ~14 years. The authors concluded that LBM was the predominant influence in the increase in peak $\dot{V}O_2$ through adolescence, but that both chronological age and stage of biological maturity were additional explanatory variables, independent of body size and fatness.[65]

Peak oxygen uptake and sex

Boys' peak $\dot{V}O_2$ values are consistently higher than those of girls by late childhood[60] and the sex difference becomes more pronounced as young people progress through adolescence.[46] The data presented in Figure 12.4 indicate that peak $\dot{V}O_2$ is ~12% higher in boys than in girls at age 10 years, increasing to ~25% higher at 12, ~30% higher at 14, and ~35% higher at 16 years of age. Longitudinal data support this trend, although with small sample sizes there is some variation in the magnitude of sex differences, particularly within the age range 12–14 years, which is likely to be due to individual variations in the speed of biological clocks. These sex differences in peak $\dot{V}O_2$ during adolescence have been attributed to a combination of

Table 12.4 Multilevel regression model for peak oxygen uptake in 11–17-year-olds

Fixed	**Estimate (SE)**
Constant	−1.9005 (0.1400)
Log_e mass	0.8752 (0.0432)
Log_e stature	NS
Log_e skinfolds	−0.1656 (0.0174)
Age	0.0470 (0.0094)
Sex	−0.1372 (0.0121)
Age·sex	−0.0214 (0.0053)
Maturity 2	0.0341 (0.0094)
Maturity 3	0.0361 (0.0102)
Maturity 4	0.0537 (0.0116)
Maturity 5	NS
Random	0.0030 (0.0004)
Level 2	
Constant	0.0030 (0.0005)
Age	0.0004 (0.0001)
Level 1	
Constant	0.0032 (0.0004)

N = 388 NS, not significant.

Data from Welsman JR, Armstrong N, Kirby BJ, Nevill AM, Winter EM Scaling peak VO2 for differences in body size. Med Sci Sports Exerc. 1996; 28: 259–265

factors including differences in daily physical activity, body composition, and blood haemoglobin concentration ([Hb]).

Boys are generally more physically active than girls,[27] but reviews of current physical activity patterns demonstrate that both sexes rarely experience the intensity, frequency, and duration of physical activity associated with increases in peak $\dot{V}O_2$.[79] Data are remarkably consistent and demonstrate no meaningful relationship between objectively measured daily physical activity and directly determined peak $\dot{V}O_2$ (see Armstrong *et al.*[80] for a table of relevant studies to date). There is therefore no compelling evidence to suggest that current levels of physical activity are likely to contribute to sexual dimorphism in peak $\dot{V}O_2$.

Muscle mass increases through childhood and adolescence, but although boys generally have more muscle mass than girls, marked sex differences do not become apparent until the adolescent growth spurt. Girls experience a growth spurt in muscle mass but it is less dramatic than that of boys. Between 5 and 16 years of age, boys' relative muscle mass increases from ~42–54% of body mass, whereas in girls muscle mass increases from ~40–45% of body mass between 5 and 13 years of age, and then, in relative terms, it declines due to an increase in fat accumulation during adolescence. Girls have slightly more body fat than boys during childhood, but during the growth spurt, girls' body fat increases to ~25% of body mass while boys decline to ~12–14% of body fat.[59] These dramatic changes in body composition in adolescence contribute to the progressive increase in sex differences in peak $\dot{V}O_2$ over this period. Boys' greater muscle mass not only facilitates the use of oxygen during exercise, but also supplements the venous return to the heart, and therefore augments SV through the peripheral muscle pump.

In adolescence there is a marked increase in [Hb] and hence oxygen-carrying capacity in boys, whereas girls' values plateau in their mid-teens. As [Hb] is significantly correlated with peak $\dot{V}O_2$ during adolescence it would be expected that differences in [Hb] between boys and girls, which are ~11% at 16 years, would be a contributory factor to the observed sex difference in peak $\dot{V}O_2$ during the late teens.[8] However, when [Hb] was investigated longitudinally as an additional explanatory variable to body mass, stature, skinfold thicknesses, age, and pubic hair development (as an indicator of stage of maturity), in a multilevel regression model of peak $\dot{V}O_2$ a non-significant parameter estimate was obtained with 11–17-year-olds.[65]

Prior to the onset of puberty there are only small sex differences in muscle mass and [Hb], but even with body size controlled for, prepubertal boys have consistently been demonstrated to have higher peak $\dot{V}O_2$ than prepubertal girls. For example, in a sample of 164 (53 girls) 11-year-old prepubertal children, boys' peak $\dot{V}O_2$ was observed to be ~22% higher than that of girls. With the removal of the influence of body mass using a log-linear adjustment model, boys' peak $\dot{V}O_2$ remained significantly higher (~16%) than girls' values despite there being no sex difference in either skinfold thickness or [Hb].[42]

Why prepubertal boys have significantly higher values of peak $\dot{V}O_2$ than prepubertal girls is not readily apparent, but the explanation might lie in the Fick equation. (Interested readers are referred to Chapter 11 for further discussion). There is no evidence to indicate sex differences in HR max, but boys have generally been observed to have higher SV max,[81] and therefore higher $\dot{Q}$ max, than girls, although there are conflicting data.[13] The trend for boys to have higher SV max during exercise has been attributed to their greater heart mass (or size) in relation to body mass (or size),[82] but conflicting data indicating no sex differences in relative heart size are available.[83] Exercise SV is, however, not just a function of ventricular size and it is difficult to distinguish between the complex and inter-related effects of ventricular preload, myocardial contractility, and ventricular afterload.

Vinet *et al.*[84] compared the cardiovascular responses of prepubertal boys and prepubertal girls using Doppler echocardiography during maximal cycle exercise. They reported no significant sex differences in a-vO_2 diff or HR at peak $\dot{V}O_2$, but the boys demonstrated significantly higher peak $\dot{V}O_2$ and SV max. They therefore concluded that the only component of peak $\dot{V}O_2$ that distinguished girls from boys was their lower SV max. The data indicated no significant sexual dimorphism in diastolic function indices or shortening or ejection fractions. Vinet and her colleagues[84] concluded that it is unlikely that overall cardiac contractility, relaxation, and compliance properties or loading conditions contribute to the sex difference in SV max, which is therefore due to differences in cardiac size rather than function.

In a similar study, Rowland *et al.*[85] compared prepubertal boys and premenarcheal girls and demonstrated that SV max was the sole cardiac variable responsible for sexual dimorphism in peak $\dot{V}O_2$ These authors noted that a characteristic that distinguished girls from boys was a lower rise in SV at the onset of exercise in girls. They suggested that cardiac functional factors (skeletal muscle pump function, systemic vascular resistance, and adrenergic responses) rather than intrinsic left ventricular size are responsible for the sex differences in SV max during childhood.

There are few secure data on young children's a-vO_2 diff at peak $\dot{V}O_2$, but a study which used thoracic bioelectrical impedance to determine the $\dot{Q}$ at peak $\dot{V}O_2$, of 31 (13 girls) 10-year-olds provided

some interesting insights into prepubertal differences in peak $\dot{V}O_2$. The boys had a significantly higher mean peak $\dot{V}O_2$ than the girls (~19%) but no significant sex differences in stature, body mass, LBM, % body fat, body mass index, body surface area, [Hb], HR at peak $\dot{V}O_2$, R at peak $\dot{V}O_2$, SV at peak $\dot{V}O_2$, or $\dot{Q}$ at peak $\dot{V}O_2$ were observed. Furthermore, heart size variables determined at rest using magnetic resonance imaging (MRI) revealed no significant sex differences in left ventricular muscle mass, left ventricular muscle volume, posterior wall thickness, septal wall thickness, left ventricular end-diastolic chamber volume, or left ventricular end-systolic chamber volume. The only significant sex difference was in a-vO_2 diff at peak $\dot{V}O_2$ where boys' values were ~17% higher than those of girls.[13]

The emergence of non-invasive technology has opened up new avenues of research with, for example, NIRS allowing the non-invasive measurement of microcirculatory changes in deoxygenated haemoglobin and myoglobin ([HHb]). An initial study demonstrated a more rapid rate of change in [HHb] during ramp exercise to peak $\dot{V}O_2$ in prepubertal girls than in prepubertal boys. These intriguing data indicate that a poorer matching of muscle oxygen delivery to muscle oxygen utilization in prepubertal girls might contribute to their lower peak $\dot{V}O_2$, but they require confirmatory evidence from different exercise models.[86]

Blood lactate accumulation

PO Åstrand's[5] experimental studies of physical working capacity popularized the use of blood lactate accumulation as an objective measure of young people's effort, and blood sampling for lactate is a common procedure in many paediatric exercise physiology laboratories. The extant literature is, however, confounded by methodological issues which have contributed to the controversy surrounding the interpretation of young people's blood lactate responses to exercise. This section outlines methodological issues, comments on blood lactate thresholds and reference values of performance during youth, and reviews the data on blood lactate responses to exercise in relation to chronological age, biological maturity, and sexual dimorphism.

Methodological issues

Research with children should not employ blood sampling as a routine procedure and for ethical reasons a strong case should always be made to justify it in relation to the research question. Strict practices must be followed at all times in the sampling and handling of blood with the health and safety of both the child and the investigator paramount. Detailed health and safety issues in haematology are beyond the scope of this chapter, and readers are referred to Maughan *et al.*[87] for further guidance. Similarly, a detailed review of blood lactate assessment techniques during youth appears elsewhere,[26] and only key issues are outlined here.

Muscle lactate produced during leg exercise diffuses into the femoral veins, and then rapidly appears in the arterial circulation. It has been demonstrated that blood sampled from the arm arteries provides a close reflection of the extent of lactate diffusion into the systemic circulation.[88] The ethical, technical, and medical hazards associated with arterial blood sampling preclude its use with healthy young people, but it has been shown that arterial lactate levels are closely reflected by capillary lactate levels during treadmill exercise if a good blood flow is maintained at the sampling site.[89] Most paediatric laboratories therefore sample lactate from the capillaries in the fingertip or earlobe. To facilitate blood flow the site can be warmed and to reduce children's anxiety an anaesthetic cream or spray can be applied.

Once sampled, 'whole blood' can be immediately assayed in an automatic analyser and results reported as blood lactate accumulation. Before making cross-study comparisons of blood lactate accumulation during or following exercise, researchers must, however, confirm the comparability of the lactate assay used and the automatic analyser. Prior to the ready availability of automatic analysers, lactate was routinely assayed in preparations such as lysed blood, protein-free blood, plasma, or serum, and often reported as 'blood lactate'. The significant variation in reporting children's blood lactate accumulation from different assays was clearly illustrated in a study which reported lactate values from the same blood sample as 4 $mmol \cdot L^{-1}$ when assayed as whole blood, 4.4 $mmol \cdot L^{-1}$ when the blood was lysed, and 5.5 $mmol \cdot L^{-1}$ from a plasma preparation.[89]

Young people's blood lactate responses to exercise are influenced by mode of exercise, exercise protocol, and time of sampling. Blood lactate reference values are heavily dependent on definition and measurement technique. As discussed in the Ergometry section, when cycling during part of the pedal revolution there is a potential for restriction in children's blood flow through the quadriceps, which will promote anaerobic metabolism. Children's blood lactate accumulation in relation to $p\dot{V}O_2$ is therefore not directly comparable during cycling and treadmill running. Regardless of ergometer, during an incremental exercise test, increments should be small and each exercise stage must be sustained for at least 3 min to allow adequate diffusion of lactate from muscle to blood. If sampled too soon the blood lactate accumulation will not reflect the intensity of the exercise and will profoundly influence the blood lactate reference value.[25]

Numerous fixed blood lactate values (e.g. 4 $mmol \cdot L^{-1}$) have been recommended as submaximal reference measures of adult performance. However, several of these reference values were originally determined using serum or plasma samples and all are problematic when applied to children. A study of 11–13-year-olds, for example, reported that 34% of boys and 12% of girls did not achieve a whole blood lactate value of 4 $mmol \cdot L^{-1}$ at peak $\dot{V}O_2$. The authors suggested that a criterion reference of 2.5 $mmol \cdot L^{-1}$ from a whole blood assay might be more appropriate for children,[17] but any fixed blood lactate value is likely to be inappropriate throughout childhood and adolescence as studies suggest an age-dependent trend in blood lactate responses to exercise.[90]

The T_{LAC}, which represents the individual's response to increasing exercise-induced metabolic demands, has become recognized as an appropriate blood lactate indicator of young people's submaximal aerobic fitness. The T_{LAC} is defined as the first observable increase in blood lactate accumulation above resting levels. It can be determined from visual inspection of the inflection in blood lactate accumulation, but a clear inflection point is not always discernible and some investigators have used mathematical interpolation or defined the point of inflection as a 1 $mmol \cdot L^{-1}$ increase over baseline.[16]

The MLSS represents the upper point at which the processes of blood lactate accumulation and elimination are in equilibrium and theoretically provides a sensitive measure of submaximal aerobic fitness. However, because of the requirement for multiple blood

samples over several ~20 min stages at the border of heavy and very heavy-intensity exercise, it is difficult to motivate children to participate in this type of test. Furthermore, there is no consensus over the optimum test time or magnitude of acceptable variation in blood lactate accumulation to represent MLSS.[17] Young people's MLSS data should therefore be interpreted cautiously. Exercise just below CPo has been shown to correspond reasonably well with MLSS in adolescents, and this non-invasive variable may be more appropriate for use during youth.[36]

Chronological age, biological maturity, and sex

Data describing the relationship between chronological age and blood lactate responses to submaximal exercise are equivocal primarily as a result of variation in exercise protocols, threshold and reference value definitions, blood sampling and assay techniques, and the predominance of underpowered and single-sex studies. Data from girls are sparse but there is no compelling evidence of sexual dimorphism in blood lactate responses to submaximal exercise during youth.[90]

Investigations have consistently observed an age-related trend of the T_{LAC} occurring at a higher % of peak $\dot{V}O_2$ in children than in adults.[90] These data have been supported by studies reporting an age-related trend in the T_{VENT} in relation to % of peak $\dot{V}O_2$ during youth.[18] In contrast there is no evidence to support a relationship between blood lactate accumulation at MLSS and age, or between MLSS as a % of peak $\dot{V}O_2$ and age.[17] In general support of differentiating between T_{LAC} and MLSS, a study of 149 11–16-year-olds found no significant relationship between age and % peak $\dot{V}O_2$ at a blood lactate of 4 $mmol \cdot L^{-1}$, but a significant correlation was observed between age and % peak $\dot{V}O_2$ at a reference value postulated to be near T_{LAC} (i.e. 2.5 $mmol \cdot L^{-1}$) in the same 11–16-year-olds studied.[91]

In his seminal thesis Eriksson[92] hypothesized a maturation effect on muscle lactate production, as he observed it to be 'almost significantly' correlated with testicular volume. He proposed that boys' blood lactate accumulation would reflect their muscle lactate production. On the basis of proportion of type I muscle fibres, $p\dot{V}O_2$ kinetics, exercise metabolism, exercise endocrinology, and substrate use during exercise, a compelling theoretical case can be made for a maturational effect on the production of muscle lactate and its accumulation in blood.[93] Empirical studies have, however, been consistent in failing to detect an independent effect of maturity on blood lactate accumulation during exercise. For example, an investigation using multiple regression analyses to examine the effect of salivary testosterone upon the blood lactate responses to exercise of 50 12–16-year-old boys observed no significant, independent effect of testosterone on blood lactate accumulation.[94] Similarly, an analysis of 119 11–16-year-old boys and girls classified into the maturity stages described by Tanner[78] observed no effect of stage of maturity on blood lactate accumulation at peak $\dot{V}O_2$.[91]

Pulmonary oxygen uptake kinetics

During a step change in exercise intensity, once the cardiodynamic phase has been deleted, the exponential rise in $p\dot{V}O_2$ has been demonstrated to reflect in adults the kinetics of $m\dot{V}O_2$ and to therefore provide a non-invasive window into metabolic activity in muscle.[95] The work has not been replicated with children, but a close relationship between children's intramuscular phosphocreatine (PCr) kinetics during prone quadriceps exercise in a MR scanner and $p\dot{V}O_2$ kinetics during upright cycling at both the onset and offset of exercise has been demonstrated.[96] This relationship has opened up new avenues of research in developmental exercise metabolism.[97] The kinetics of $p\dot{V}O_2$ and intramusclular PCr (as a surrogate of $m\dot{V}O_2$) at the onset of exercise are complex, and are comprehensively analysed in Chapter 13 and Chapter 6 where the theoretical principles are explained, the underlying mechanisms explored, and the rigorous methodology required to characterize the kinetic responses critiqued. Discussion here is restricted to identifying methodological issues in the determination of young people's $p\dot{V}O_2$ kinetics responses which might influence their interpretation. The focus is on exploring the $p\dot{V}O_2$ kinetics responses to a step change in exercise intensity in relation to exercise domain, chronological age, and sexual dimorphism. The independent influence (if any) of biological maturity on $p\dot{V}O_2$ kinetics remains to be rigorously investigated.

Methodological issues

The clarification of the $p\dot{V}O_2$ kinetics response at the onset of exercise depends upon the ability to rigorously evaluate the speed and the magnitude of the respiratory gas exchange response to a given metabolic demand. This can be achieved by imposing a predetermined square wave exercise stress and then using non-linear regression and iterative fitting procedures with the response data to fit a specified model to return the rate of the exponential rise and the amplitude of the response. Unfortunately, a wide array of models with various degrees of rigour have been employed to evaluate $p\dot{V}O_2$ kinetics, and interested readers are referred to Fawkner and Armstrong,[98] who have critiqued and tabulated chronological models used with children and adolescents. The confounding effect of different modelling techniques on the interpretation of young people's response parameters has been shown empirically by applying several different models to the same dataset in both the moderate[99] and heavy-intensity exercise domains.[100] The use of different models, several with limited physiological rationales, has made understanding the extant paediatric literature problematic.[31]

Even with an appropriate modelling procedure, the rigorous resolution of the $p\dot{V}O_2$ kinetics of children and adolescents is challenging. Children's inherently erratic breathing pattern reduces the signal-to-noise ratio of their pulmonary gas exchange kinetics.[47] Large inter-breath fluctuations reduce the confidence with which $p\dot{V}O_2$ kinetic responses can be estimated, and confidence intervals are likely to be beyond acceptable limits unless sufficient identical transitions are time aligned and averaged to improve the signal to noise ratio.[101] The number of transitions that are required to achieve suitable confidence is directly proportional to the amount of data being fit, the variability of the data, and the magnitude of the signal, and will thus vary from one person to another. With children, as many as ten transitions may be required in the moderate-intensity exercise domain to establish an acceptable confidence interval for the primary component τ.[102] Fewer transitions are required in heavier-intensity exercise domains because the magnitude of the signal is greater.

Young people's lower peak $\dot{V}O_2$, and therefore smaller range of metabolic rates achievable, may compromise the integrity of working within specific exercise domains. Children's T_{VENT} occurs at ~60–70% of peak $\dot{V}O_2$[19] and to ensure that the prescribed exercise is clearly within the moderate domain, the upper border of

exercise intensity is normally set at ~80% of T_{VENT}. With children, the $p\dot{V}O_2$ kinetics responses to exercise intensities above T_{VENT} have rarely been investigated within carefully defined parameters. This is most likely because the assessment of CPo, the upper boundary of heavy-intensity exercise, and the threshold of very heavy-intensity exercise is demanding in terms of both subject effort and testing time.[20] Investigators in adult studies normally use 40–50% of the difference between T_{VENT} and peak $\dot{V}O_2$ (e.g. 40% Δ) as describing exercise within the heavy-intensity domain[30]. It has been demonstrated that CPo occurs at ~70–80% of peak $\dot{V}O_2$ in children, similar to relative values reported for adults, and that exercise at an intensity of 40% Δ is below CPo and falls within the heavy-intensity exercise domain.[103] However, the absolute range of $p\dot{V}O_2$ between T_{VENT} and CPo is small in children and there is considerable individual variation in the relative position of both T_{VENT} and CPo in relation to peak $\dot{V}O_2$. The 40% Δ concept is therefore less secure on an individual basis with children than with adults.

Exercise phases, exercise domains, chronological age, and sex

Macek and Vavra[104] were the first to investigate the half-time of children's transient responses to the onset of exercise, but the initial application of breath-by-breath technology to children's $p\dot{V}O_2$ kinetics was carried out by Dan Cooper and his colleagues.[105] Data from early studies are inconsistent, but recent research using more rigorous methodology, sophisticated mathematical modelling techniques, and emerging technologies has begun to map out young people's transient responses to the onset of a step change in exercise intensity.[31]

Cardiodynamic phase

Following the onset of exercise, $p\dot{V}O_2$ measured at the mouth is dissociated temporarily from $\dot{V}O_2$ at the muscle by the muscle-lung transit delay. The speed of the response is due to the almost instantaneous increase in $\dot{Q}$ which is initiated by vagal withdrawal and the mechanical pumping action of the contracting muscles. The rise in $\dot{V}O_2$ that is evidenced at the mouth during this translational phase is therefore independent from absolute changes in mixed partial pressures arising from the working muscles. If the muscle-lung transit delay is a function of growth, the shorter distance between exercising muscles and the lung in children might suggest an age-related increase in the length of phase I. However, data on phase I in various exercise domains are ambiguous and often confounded by methodological issues. Studies which have attempted to determine the end of phase I visually from response profiles of end-tidal partial pressures of oxygen and carbon dioxide and R must be interpreted cautiously. For the purposes of modelling the primary component the duration of phase I is normally not measured but is assumed to be constant at ~15–20 s.[21,106]

Rigorously determined age- and sex-related data from phase I are sparse. Men have been reported to have a longer duration of phase I than boys in response to the onset of a transition to 50% of peak $\dot{V}O_2$.[107] Longitudinal data show the duration of phase I following the onset of heavy intensity exercise to increase from age 10 to 13 years in both boys (16.7–19.5 s) and girls (20.7–24.3 s).[32] In contrast to adults it has been noted that the duration of phase I in boys is not reduced at higher metabolic rates.[107] Sexual dimorphism has been reported in prepubertal children, with boys noted to have a shorter phase I duration than girls (17.0 vs 19.3 s).[55] This might be indicative of a more rapid increase in SV in boys than in girls, an observation previously noted using Doppler echocardiography.[85] Evidently little is known about age and sex differences and their mechanisms in the duration of phase I. Independent effects of biological maturity on the cardiodynamic phase have not been investigated.

Moderate-intensity exercise

Data from studies of young people's phase II response to the onset of moderate-intensity exercise are equivocal, but several studies are methodologically flawed on the basis of their failure to employ well-defined participant groups, to apply appropriate analytical modelling techniques, to address the low signal-to-noise ratio through adequate exercise transitions, and to report the 95% confidence intervals.[21] These studies and their modelling techniques have been tabulated elsewhere (see Table 13.1), and although often failing to reach statistical significance the strong trend among the studies which used breath-by-breath respiratory gas analysis is for a shorter τ in children and adolescents than in adults.[31]

A carefully designed and modelled study, in which up to ten repeat exercise transitions were completed to ensure the 95% confidence intervals spanning the primary component τ were no more than ± 5 s, addressed the lack of consensus in the extant literature[102]. It was demonstrated unequivocally that the primary component τ was significantly shorter in boys than men (19 vs 28 s) and in girls than women (21 vs 26 s). No sexual dimorphism was observed in the primary component τ despite significant sex differences in peak $\dot{V}O_2$. Peak $\dot{V}O_2$ was not related to the phase II τ, a finding in contrast to earlier data from adults,[22] but later confirmed in a comparison of trained and untrained prepubertal children.[108]

It is not only the speed of the $p\dot{V}O_2$ kinetics responses, but also the magnitude of the response (gain of the primary component or oxygen cost of the response) that provides information on the efficiency of the integrated pulmonary, cardiovascular, and muscle metabolic systems. A substantially higher primary gain in children than in adults has been reported during both treadmill running[109] and cycling.[110] However, in the treadmill running study the gain was expressed in ratio with body mass ($mL \cdot kg^{-1} \cdot km^{-1}$) and, as argued throughout this chapter, in most circumstances this is an inappropriate way to normalize comparative data.[72] Although the balance of evidence supports a higher gain in children,[30] further examination with more rigorous study designs is required to tease out definitive age-related differences in the oxygen cost of the response to moderate intensity exercise.

Children's shorter τ, and therefore greater aerobic contribution to ATP re-synthesis at the onset of exercise, indicates that compared to adults they have an enhanced oxidative capacity, which might be due to greater oxygen delivery or better oxygen utilization in the muscles. Peak $\dot{V}O_2$, which is thought to be primarily dependent on oxygen delivery, is not related to the primary component τ and there is no compelling theoretical hypothesis to indicate that increased delivery of oxygen would speed the rate of healthy young people's $p\dot{V}O_2$ kinetics during moderate-intensity exercise.[30] However, a study utilizing NIRS, HR kinetics, and breath-by-breath technology noted that, compared with men, prepubertal boys presented a shorter primary component τ supported by both a quicker adjustment in [HHb] kinetics and faster local blood flow. It was concluded that faster oxygen extraction and oxygen delivery

might both have a role to play in children's faster $p\dot{V}O_2$ kinetics.[111] More research is required to tease out the mechanisms underpinning age-related differences in τ.

Heavy-intensity exercise

Young people's $p\dot{V}O_2$ kinetics responses to the onset of exercise above T_{VENT} are masked in several studies which employ inadequate mathematical models and poor definitions of the heavy- and very heavy-intensity exercise domains, sometimes by simply using % peak $\dot{V}O_2$ to characterize the exercise domain.[98] Nevertheless, despite often-flawed methodology, the literature is consistent in demonstrating that at the onset of heavy-intensity exercise, children have a faster primary component τ than adults.[110]

Two longitudinal studies determined acceptable confidence intervals for the τ and amplitude of the primary component at the onset of heavy-intensity exercise. The first study demonstrated a faster primary component τ in both prepubertal girls and boys than the same children presented 2 years later (boys, 17 vs 21 s; girls, 22 vs 26 s).[32] In the second study 14-year-old boys presented a shorter phase II τ than they did 2 years later (26 vs 30 s).[35] In children, but not adolescent boys, the primary gain decreased over a 2-year period (12.4 vs 12.0 $mL \cdot min^{-1} \cdot W^{-1}$), thus supporting the age-related effect shown in wider age comparisons of children and adults (11.6 vs 9.9 $mL \cdot min^{-1} \cdot W^{-1}$).[110] The same research group demonstrated that, in contrast with their findings at the onset of moderate-intensity exercise, prepubertal boys presented a faster primary component τ than prepubertal girls (18 vs 22 s). No significant sex difference in the primary gain was observed.[55] In accord with exercise in the moderate-intensity domain, but in conflict with adult data, studies consistently show that peak $\dot{V}O_2$ is not related to the phase II τ in prepubertal children[32] or adolescents[35] during exercise in the heavy-intensity domain.

During exercise above T_{VENT} but below CPo adults' phase III is characterized by the oxygen cost increasing over time as a $p\dot{V}O_2$ slow component is superimposed and the achievement of a steady state is delayed[30] (see Figure 12.3). Initial comparisons of children and adults concluded that during heavy-intensity exercise, children demonstrate a negligible $p\dot{V}O_2$ slow component and their responses could be modelled as a mono-exponential process.[109,110] Contrary to these reports, in a series of studies Fawkner and Armstrong[100] demonstrated empirically that a slow component does exist in children and the data should not be modelled mono-exponentially. They showed that with appropriate modelling prepubertal children exhibit a $p\dot{V}O_2$ slow component, which contributes ~10% of the end-exercise $p\dot{V}O_2$ after 9 min of exercise and increases in magnitude with age.[32] It was noted that, despite an increase in the magnitude of the $p\dot{V}O_2$ slow component, the overall oxygen cost at the end of exercise was equal on test occasions 2 years apart. This suggests that the phosphate turnover required to sustain exercise is independent of age and that younger children achieve a larger proportion of their end-exercise $p\dot{V}O_2$ during phase II. A subsequent study identified significant sexual dimorphism in prepubertal children with the $p\dot{V}O_2$ slow component of boys and girls accounting for ~9% and ~12% respectively of the end-exercise $p\dot{V}O_2$.[55] The same research group also demonstrated the presence of a $p\dot{V}O_2$ slow component, which increased in magnitude with age in adolescent boys.[35] Subsequent research has unequivocally established the presence of a $p\dot{V}O_2$ slow component during heavy-intensity exercise in both prepubertal children[112] and adolescents.[113]

There is symmetry between the rate of PCr breakdown and the $p\dot{V}O_2$ primary component τ at the onset of exercise above T_{VENT},[30] which suggests that the shorter phase II τ in young people might be due to an age-dependent effect on mitochondrial oxidative phosphorylation. There is a paucity of data, but this postulate is supported by children's enhanced aerobic enzymes activities and/or lower glycolytic enzymes activities compared to adults.[57] As ~85% of the $p\dot{V}O_2$ slow component originates from the exercising muscles, the increase in the magnitude of the $p\dot{V}O_2$ slow component with age is likely due to progressive changes in muscle fibre recruitment patterns. The enhanced glycogen depletion of type I fibres and the greater recruitment of type II fibres by adults will promote an increased $p\dot{V}O_2$ slow component. The data are in accord with children having a higher % of type I muscle fibres than adults, and the reported sexual dimorphism in the phase II τ and $p\dot{V}O_2$ slow component are consistent with girls having a lower % of type I fibres than similarly aged boys.[57]

Very heavy-intensity exercise

In the very heavy-exercise intensity domain boys have a shorter phase II τ (21 vs 34 s), a greater primary gain (10.8 vs 8.2 $mL \cdot min^{-1} \cdot W^{-1}$), and a smaller relative $p\dot{V}O_2$ slow component (11 vs 16%) than men.[114,115] As demonstrated in other exercise domains, peak $\dot{V}O_2$ is not related to the phase II τ.[114] The presence of a $p\dot{V}O_2$ slow component in prepubertal children[116] and teenage boys[117] during very heavy exercise is not disputed, but sexual dimorphism has not been addressed in this exercise domain. Exploratory investigations of the contribution of oxygen delivery and oxygen utilization to boys' $p\dot{V}O_2$ kinetics have used priming exercise to elevate $\dot{Q}$ and muscle oxygenation prior to and throughout subsequent very heavy-intensity exercise. Phase II $p\dot{V}O_2$ kinetics (i.e. τ) were reported to be unaltered but the $p\dot{V}O_2$ slow component amplitude was reduced, suggesting that phase II $p\dot{V}O_2$ kinetics are principally limited by intrinsic muscle metabolic factors, and that the $p\dot{V}O_2$ slow component is sensitive to oxygen delivery.[118,119]

In conflict with adult data[30] (see Figure 12.3), young people's $p\dot{V}O_2$ slow component when exercising above CPo has not been demonstrated to project to peak $\dot{V}O_2$ over time but to stabilize at ~85–90% of peak $\dot{V}O_2$.[36,] It has been suggested that this may be due to an early termination of exercise by young people through exhaustion.[120]

Severe-intensity exercise

The resolution of the $p\dot{V}O_2$ kinetics response to a system-limited value (i.e. peak $\dot{V}O_2$) is complex and it is problematic to compare investigations using different data collection and modelling techniques. Few studies have been dedicated to unravelling the $p\dot{V}O_2$ kinetics response to severe exercise during youth and available data must be interpreted with caution.[21]

Early studies, which collected $\dot{V}_E$ in gasometers or Douglas bags over 30 s periods, reported boys to achieve a higher percentage of their peak $\dot{V}O_2$ than men during the first 30 s of exercise, requiring at least 100% peak $\dot{V}O_2$.[3,104] However, the lack of temporal resolution of the respiratory gas exchange measurements and the failure to consider phase I compromises the data, and two more recent studies using breath-by-breath technology have failed to confirm these findings.

Hebestreit *et al.*[107] investigated the $p\dot{V}O_2$ kinetics of 9- to 12-year-old boys and of men at the onset of cycling exercise to 100% and 130% of peak $\dot{V}O_2$. On both occasions, once phase I had been excluded, $p\dot{V}O_2$ kinetics could be described by a mono-exponential function (i.e. no $p\dot{V}O_2$ slow component detected) in which no age-related differences were observed for the primary component τ. This study confirmed earlier work which had shown that, when compared to adults, children have higher end-exercise oxygen cost during severe exercise.[121]

Recovery kinetics

Further insights into the development of aerobic fitness undoubtedly lie in the $p\dot{V}O_2$ kinetics of recovery from exercise in different domains, but the only study to specifically investigate the influence of age on $p\dot{V}O_2$ kinetics at the offset of exercise appears to be that of Zanconato *et al.*[121] They analysed the recovery $p\dot{V}O_2$ kinetics of mixed sex groups of children (7–11 years) and adults (26–42 years) following a single 1 min bout of exercise at 80% T_{VENT}, 50% Δ, 100% peak $\dot{V}O_2$, and 125% peak $\dot{V}O_2$. The only significant child-adult difference reported was children's faster recovery from 125% peak $\dot{V}O_2$.

Rigorously determined breath-by-breath data on $p\dot{V}O_2$ kinetics at the offset of exercise during youth are sparse. Lai *et al.*[120] observed similar time courses for recovery $p\dot{V}O_2$ kinetics of male adolescents (14–17-year-olds) when compared with previously published data on adults. They reported that adolescents' recovery $p\dot{V}O_2$ kinetics from both moderate- and heavy-intensity exercise can be described with a single exponential, but that recovery from very heavy-intensity exercise is characterized by two components, one fast and one slow. Lai *et al.*[120] noted that steady states for the $p\dot{V}O_2$ kinetics off-response were attained in ~5 min for moderate- and heavy-intensity exercise and within ~10 min for very heavy-intensity exercise. In contrast, with adults the recovery $p\dot{V}O_2$ slow component from very heavy exercise has been reported to take >20 min to dissipate.[30]

Comparisons of trained and untrained young athletes have confirmed that the $p\dot{V}O_2$ offset kinetics of both moderate- and heavy-intensity exercise can be described with a single exponential. Intriguingly, although the trained athletes displayed faster $p\dot{V}O_2$ kinetics at the onset of both moderate- and heavy-intensity exercise than their untrained peers, only the recovery $p\dot{V}O_2$ kinetics from heavy-intensity exercise were faster in the trained athletes.[122,123] (Interested readers are invited to read Chapter 34 for more detailed discussion).

Conclusions

The laboratory assessment of young people's peak (or max) $\dot{V}O_2$ dates back to 1938 and it is the most researched variable in paediatric exercise science. Yet, debate over the determination and terminology of peak and/or maximal values of $\dot{V}O_2$ persists. The fallacy of expressing peak $\dot{V}O_2$ in ratio with body mass has been documented for over 65 years, but ratios are still reported. Decisive action and insistence on contextual reporting of peak $\dot{V}O_2$ by academic journal editors is required to disseminate the appropriate interpretation of aerobic fitness during growth and maturation. Nevertheless, analysis of data using sophisticated modelling techniques has enhanced understanding of sexual dimorphism and the independent effects of chronological age, body size, and biological maturity on peak $\dot{V}O_2$. The mechanisms underlying sex differences in peak $\dot{V}O_2$ prior to puberty remain to be elucidated, but the introduction of recent non-invasive technology such as NIRS provides promising avenues for future research.

Despite its ubiquity in the literature, the use of fixed post-exercise values of blood lactate accumulation to verify a maximal effort during an exercise test to elicit peak $\dot{V}O_2$ is untenable. The monitoring of blood lactate accumulation and the determination of blood lactate accumulation thresholds (e.g. T_{LAC}) during exercise provides an indicator of the ability to sustain submaximal exercise and a sensitive means of evaluating improvements in muscle oxidative capacity with exercise training. The relationship between blood lactate accumulation and chronological age is well-documented, but sex differences in blood lactate accumulation during youth remain to be proven. A persuasive theoretical argument can be presented for an independent effect of maturity on blood lactate accumulation, although there is no compelling empirical evidence to support the case and more research with appropriate methodology and power to adequately address the problem is required.

Young people's physical activity patterns and participation in most organized sports are reliant on intermittent exercise and rapid changes in exercise intensity. Under these conditions peak $\dot{V}O_2$ and blood lactate accumulation thresholds are variables of investigative convenience rather than factors underpinning exercise behaviour, and it is the kinetics of $p\dot{V}O_2$ which best describe aerobic fitness. Rigorously determined and appropriately analysed studies of young people's $p\dot{V}O_2$ kinetic responses to step changes in exercise intensity are sparse. The extant data describe intriguing chronological age- and sex-related differences across exercise domains, although independent effects of biological maturity are yet to be revealed. Unique insights into aerobic fitness during youth rest in the transient response to and recovery from a forcing exercise regimen. The challenge is to identify and explain the underlying mechanisms and how they evolve during childhood and adolescence.

No single measure describes fully aerobic fitness and this chapter has focused on arguably the three most important variables in relation to chronological age, body mass, biological maturity, and sex. We conclude that although aerobic fitness is the most researched trait in paediatric exercise physiology, much remains to be learned.[124]

Summary

- Aerobic fitness can be defined as the ability to deliver oxygen to the exercising muscles and to utilize it to generate energy during exercise. No single variable describes fully aerobic fitness.
- Boys' peak $\dot{V}O_2$ expressed in $L \cdot min^{-1}$ increases in a near-linear manner with chronological age. Girls' data demonstrate a similar but less consistent trend with several cross-sectional and longitudinal studies indicating a tendency for peak $\dot{V}O_2$ to plateau from ~14 years of age.
- With body mass appropriately controlled for using allometry, boys' peak $\dot{V}O_2$ increases from childhood through adolescence and into young adulthood. Girls' values increase from prepuberty until mid-teens, then level-off as they approach young adulthood.
- Biological maturation exerts a significant and positive effect on the peak $\dot{V}O_2$ of both sexes independent of that due to chronological aging, body composition, and body mass.

- Prepubertal boys have higher peak $\dot{V}O_2$ values than prepubertal girls.
- There is a progressive divergence in sex differences in peak $\dot{V}O_2$ in puberty largely due to sex-related growth in muscle mass.
- Interpretation of blood lactate accumulation during exercise is clouded by methodological issues related to mode of exercise, exercise protocol, timing of blood sample, site of sampling, and assay technique
- The lactate threshold normally occurs at a higher % of peak $\dot{V}O_2$ in children than in adults, and there is no compelling evidence to suggest sexual dimorphism.
- Empirical studies have consistently failed to detect an independent effect of biological maturation on blood lactate accumulation during exercise.
- The confident estimation of the primary component time constant and appropriate modelling of the amplitude of the slow component is challenging and rigorous studies of $p\dot{V}O_2$ kinetics during youth are sparse.
- The primary component time constant is negatively related to chronological age in both sexes across the moderate, heavy, and very heavy exercise domains.
- During exercise above the ventilatory threshold, boys' primary component time constant is shorter than that of girls, whereas the amplitude of the $p\dot{V}O_2$ slow component is greater in girls. Little is known about $p\dot{V}O_2$ kinetic responses to severe exercise.
- The relative contribution of oxygen delivery and oxygen utilization to the speed of the primary component time constant in different exercise domains remains to be elucidated.
- In contrast to adults, the primary component time constant is not related to peak $\dot{V}O_2$ during youth.
- The amplitude of the $p\dot{V}O_2$ slow component of oxygen uptake is positively related to chronological age during exercise above the ventilatory threshold.
- Data on the $p\dot{V}O_2$ kinetics recovery from exercise in different domains are sparse.
- Whether there is an independent effect of biological maturation on the $p\dot{V}O_2$ kinetics response at the onset and offset of exercise is unknown.

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