Physiological Determinants of Maximal Aerobic Power in Healthy 12-Year-Old Boys

Thomas Rowland, Gregory Kline, Donna Goff, Leslie Martel, and Lisa Ferrone

Little is known regarding the physiological determinants of maximal oxygen uptake (VO\textsubscript{2,max}) in children. A group of 39 healthy sixth-grade boys (mean age, 12.2 years) underwent maximal cycle testing with determination of cardiovascular factors using Doppler echocardiography as well as standard gas exchange variables. Maximal stroke index was related to VO\textsubscript{2,max}/kg (r = 0.52, p < .05), but no relationship was observed between VO\textsubscript{2,max}/kg and either maximal heart rate or calculated maximal arteriovenous oxygen difference. Values of maximal stroke index were closely related to those at rest (r = 0.67). These findings suggest that factors influencing resting stroke volume are primarily responsible for inter-individual differences in VO\textsubscript{2,max} per kg in healthy, non-athletically-trained boys.

Maximal oxygen uptake (VO\textsubscript{2,max}), related to body size, serves as the traditional laboratory marker of aerobic fitness. A value of VO\textsubscript{2,max} indicates the integrity of the pulmonary, cardiac, and hematologic chain of oxygen delivery as well as the capacity of exercising muscle to extract and utilize oxygen for metabolic energy. From a functional standpoint, VO\textsubscript{2,max} reflects performance capabilities in endurance events that are dependent on aerobic energy sources.

Given its importance in determining aerobic fitness and physical performance, factors which define the limits of oxygen uptake have come under extensive research scrutiny (16). By the constraints of the Fick equation, VO\textsubscript{2,max} must be established either centrally by one's cardiac reserve capacity (maximal cardiac output, the product of maximal stroke volume and heart rate), or peripherally by maximal arteriovenous oxygen difference. The most compelling research data in adults indicates that differences in VO\textsubscript{2,max} among populations of athletes, non-athletes, and patients with heart disease principally reflect variations in maximal cardiac output (8). Maximal heart rate is independent of level of aerobic fitness. Consequently, differences in maximal stroke volume appears to be the critical physiological factor that separates aerobically "fit" from "unfit" adults.

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The mechanisms that influence individual differences in maximal stroke volume (and thus VO$_2$max) have not been clearly delineated. While myocardial contractility and afterload can affect stroke volume, most research on adults suggests that diastolic filling (ventricular preload) is the most important factor in defining the limits of stroke volume during exercise (1, 20). Evidence exists that this might occur through two possible means. First, a larger maximal stroke volume might simply reflect a greater resting stroke volume, itself a manifestation of factors causing increased preload at rest (greater plasma volume, larger left ventricular diastolic volume) (6).

Alternatively, individuals with higher values for VO$_2$max might demonstrate a greater rise in stroke volume during exercise. Typically, stroke volume increases at the beginning of a progressive upright exercise test and then demonstrates a plateau to the point of exhaustion. Those with higher VO$_2$max may instead show a progressive rise in stroke volume at high exercise intensities (5, 10). This mechanism demands other explanations, such as differences in dynamics of diastolic filling (i.e., left ventricular "suction") and effectiveness of the peripheral skeletal muscle pump.

Despite the increased interest in aerobic fitness in children, very limited data are available regarding the determinants of VO$_2$max in this age group. Similarly, it is unknown whether the factors that establish VO$_2$max in adults are evident in the childhood years. This study used Doppler and two-dimensional echocardiography to examine physiological factors responsible for determining VO$_2$max, expressed relative to body size, in a group of 39 healthy sixth-grade boys. The investigation was specifically designed to include subjects with a wide range of aerobic fitness that would reflect the fitness of the general pediatric population.

**Methods**

Ten sixth-grade boys were recruited from each quartile of finish times in a recent 1-mile run performed as part of routine physical education fitness testing. Of the 40 who agreed to perform laboratory exercise testing, 1 was unable to complete testing. Findings of the remaining 39 boys constitute this report. Thirty-six of these subjects were included in another publication describing the relationship of physiological and anthropometric factors in boys with 1-mile run performance (11).

The mean age of the subjects was 12.2 (SD = 0.5) years. The entire group was Caucasian and attended the same suburban middle school. All were in good health, and none of the boys was taking medications that would affect aerobic fitness. By questionnaire, 14 (36%) had entered early puberty, as indicated by voice change, appearance of pubic hair, or shaving.

Parents were asked to grade their child’s usual level of physical activity on a 5-point scale (1 = very sedentary, 5 = involved in sports, always active). The average rating for the group was 3.5 (1.0). Twenty-seven (69%) had recently participated on a community sports team (most commonly soccer and basketball).

Subjects were instructed to refrain from vigorous physical activity in the 24 hours before testing and to avoid eating 2–3 hours before arriving at the exercise laboratory. In an attempt to assure some uniformity of hydration status, 8 oz. of a commercial sports drink (Gatorade) was consumed by each boy 1 hour before testing.

Following measurement of weight and height, triceps and scapular skinfold measurements were determined on the right side using standard techniques. Values in triplicate were averaged and summed to create a skinfold score. Percent
body fat was estimated from skinfold measurements using the Slaughter equations (17). Lean body mass was calculated as (body mass) – (percent fat \times body mass \times 100).

Prior to exercise, left ventricular dimensions were measured by M-mode echocardiography under two-dimensional guidance (Hewlett Packard Sonos 1000, Andover, MA) with the subject supine. Measurements were obtained in the parasternal long axis view just distal to the tips of the mitral valve leaflets. The distance from the posterior edge of the ventricular septum to the left ventricular endocardial surface coincident with the Q wave on the electrocardiogram was recorded as the left ventricular end-diastolic dimension (EDD). The left ventricular end-systolic dimension (ESD) was determined as the shortest distance from the free wall endocardial echo in systole to the ventricular septum. Left ventricular shortening fraction (SF) was calculated as the quotient \( \frac{EDD - ESD}{EDD} \times 100 \).

Exercise testing was performed in an air-conditioned laboratory at a temperature of 20–21°C. Subjects cycled to exhaustion in the sitting position on a mechanically-braked Monark ergometer. Seat height was adjusted to provide an approximate 15° knee angle at full extension. Initial and incremental workloads were 25 W, with a 3-min stage duration and 50 rpm constant pedaling cadence. Subjects were encouraged verbally by the testing staff to provide an exhaustive effort. The test was terminated when the subject could no longer maintain the appropriate pedaling rate.

Heart rate was measured electrocardiographically. Standard open circuit techniques were utilized to measure gas exchange variables with a mouthpiece, 94-ml dead space Rudolph valve, and Q-Plex Cardio-Pulmonary Exercise System (Quinton, Seattle, WA). Expired air samples were drawn from a 6-L mixing chamber and analyzed for oxygen and carbon dioxide content by zirconia oxide and infrared analyzers, respectively. Minute ventilation was measured by pneumotachometer in the expiratory line. Mean values for \( VO_2, VCO_2, VCO_2/VO_2 \) (RER), and \( V_e \) were calculated over 15-s intervals. The system was calibrated prior to each individual test using standard gases of known oxygen and carbon dioxide concentration.

Peak oxygen uptake was defined as the average of the two greatest values (15-s averages) during the final minute of exercise. Peak \( VO_2 \) was considered equivalent to \( VO_2 \)max if subjects presented subjective evidence of exhaustion and maximal heart rate exceeded 185 bpm with a maximal RER over 1.00.

Cardiac output at rest, during submaximal exercise, and at exhaustion was estimated by standard Doppler echocardiographic techniques (3). A 1.9 Mhz continuous Doppler transducer (Pedof) was directed from the suprasternal notch to measure velocity of blood in the ascending aorta. The integral of velocity over time (velocity-time integral, VTI) for individual beats was measured by tracing the contour of the velocity curve both on-line and off-line. Values for VTI at rest, during the final minute of each workload, and in the final 30 s of exercise were determined by averaging the 3–10 curves with the highest values and most distinct spectral envelopes.

Prior to testing, the maximal diameter of the ascending aorta in systole was measured by two-dimensional echocardiography (parasternal long axis view) with the subject seated on the cycle ergometer. The diameter was measured at the level of the junction of the sinuses of Valsalva with the ascending aorta (sinotubular junction) from inner edge to inner edge. The aortic cross-sectional area was calculated from the average of 5–10 such diameter measurements, assuming the aorta to be circular.
Data describing the reliability and validity of the Doppler technique for estimating maximal stroke volume during exercise have previously been reported from this laboratory (12, 13). The test-retest intraclass correlation coefficient in 13 subjects was $R = 0.90$ with a coefficient of variation of 8.5% (12). Mean values for maximal stroke volume were not significantly different in 8 boys using the Doppler method (60 ± 11 ml) and thoracic bioimpedance technique (68 ± 13 ml) (13).

Resting and exercise stroke volume (SV) was estimated from the product of VTI and aortic cross-sectional area. Cardiac output (Q) was then calculated as heart rate · stroke volume, and arteriovenous oxygen difference (AV $O_2$ diff) was calculated by dividing $VO_2$ by Q.

The contribution of maximal cardiac output, stroke volume, heart rate, and arteriovenous oxygen difference to size-relative $VO_2$max was assessed by Pearson moment correlation. This analysis was performed by two means of relating physiological variables to body size. The first analysis utilized traditional denominators for relating these factors to body dimensions: $VO_2$max per kg, cardiac output and stroke volume indexed to body surface area (BSA), and heart rate and arteriovenous oxygen difference in absolute terms.

In the second analysis, maximal values for oxygen uptake, cardiac output, stroke volume, and arteriovenous oxygen difference were scaled to allometric powers of body mass or surface area derived from this specific subject population. This analysis involved identification of the scaling exponent $b$ in the allometric equation $Y = aX^b$, where $Y$ is the physiological variable, $X$ is the anthropometric scaling variable (mass or body surface area), and $a$ is a constant multiplier. To obtain $b$, log transformation of both physiological outcome and anthropometric scaling variables was performed, and least squares regression identified $b$ in the equation log $(Y) = \log (a) + b \log (X)$.

To examine the potential influence of variations in size-relative $VO_2$max on the pattern of stroke volume response to exercise, changes in stroke volume with increased exercise intensity were compared in the highest and lowest quintile of subjects ranked by $VO_2$max per kg. Physiological and anthropometric differences between these groups were examined by student $t$ test. Statistical significance was defined as $p < .05$. Informed consent and assent were obtained from the parents and children, respectively. This study was reviewed and approved by the Institutional Review Board of the Baystate Medical Center.

**Results**

The mean weight of the subjects was 45.6 (10.1) kg and height was 153 (9) cm—values which are at the 60th and 55th percentiles, respectively, on published population norms for 12-year-old American boys (9). The average sum of scapular and triceps skinfolds was 21.2 (9.1) mm. This corresponds to the 65th percentile in the National Children and Youth Fitness Study (15). Thus, the study population in this investigation reflects near-average values for height, weight, and body fat compared to the general pediatric population in the U.S.

Maximal hemodynamic and gas exchange variables were obtained on all subjects. One subject did not satisfy heart rate and RER criteria for a maximal test. However, based on subjective assessment, it was considered that an exhaustive effort had been provided, and his data are included in these results.
Average maximal heart rate and RER were 198 (10) bpm and 1.06 (.04), respectively. The mean values of maximal oxygen uptake of 47.0 ml · kg⁻¹ · min⁻¹ and maximal cardiac index of 11.98 L · min⁻¹ · m⁻² are typical for those obtained during cycle testing in boys of this age group (9).

Pearson moment correlation coefficient between \( \dot{V}O_2 \text{max} \) expressed per kg body mass and percent body fat was \( r = -0.73 \) (\( p < .01 \)). Coefficients between \( \dot{V}O_2 \text{max} \) per kg and maximal cardiac index and stroke index were \( r = 0.54 \) and \( r = 0.52 \), respectively (\( p < .01 \)). No significant association was observed between \( \dot{V}O_2 \text{max} \) per kg and absolute values of maximal AV \( O_2 \) diff or maximal heart rate (\( r = 0.23 \) and \( r = -0.22 \), respectively, \( p > .05 \)).

In this population of subjects, appropriate scaling factors were \( \dot{V}O_2 \text{max}/M^{0.70} \), \( Q_{\text{max}}/\text{BSA}^{0.51} \), \( SV_{\text{max}}/\text{BSA}^{0.62} \), and \( \text{AV } O_2 \text{,max}/M^{0.43} \). No significant relationship was observed between maximal heart rate and any anthropometric variable. Relationships of the scaled cardiovascular variables with \( \dot{V}O_2 \text{max}/M^{0.70} \) were similar to those observed for the non-scaled associations outlined above. Correlation coefficients for scaled \( Q_{\text{max}} \) and \( SV_{\text{max}} \) were \( r = 0.53 \) and \( r = 0.51 \), respectively (\( p < .05 \)), and \( \text{AV } O_2 \text{, diff} \) max \( r = 0.06 \) (\( p > .05 \)), with scaled \( \dot{V}O_2 \text{max} \).

Comparisons of physiological and anthropometric variables between the high and low fit groups (\( n = 8 \) in each) are outlined in Table 1 (traditional size normalization) and Table 2 (allometric-scaled variables). Mean \( \dot{V}O_2 \text{max} \) values for the high and low fit children were 54.8 (1.2) and 38.8 (2.5) ml · kg⁻¹ · min⁻¹, respectively. The low fit demonstrated a greater percent body fat than the high fit, but the latter still demonstrated a higher \( \dot{V}O_2 \text{max} \) relative to lean body mass. No signifi-

<table>
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<th>Table 1 Physiological and Anthropometric Comparisons of High (( N = 8 )) and Low Fit (( N = 8 )) Boys Using Traditional Means of Normalization for Size</th>
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<td><strong>Variable</strong></td>
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<tr>
<td>Weight (kg)</td>
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<td>Height (cm)</td>
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<td>Body fat (%)</td>
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<td>RERmax</td>
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<td>( \dot{V}O_2 \text{max} ) (ml · kg⁻¹ · min⁻¹)</td>
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<td>( \dot{V}O_2 \text{max} ) (ml · kg LBMP⁻¹ · min⁻¹)</td>
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<td>Cardiac index, maximal (L · min⁻¹ · m⁻²)</td>
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<td>Heart rate, resting (bpm)</td>
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<td>Heart rate, maximal (bpm)</td>
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<td>Stroke index, rest (ml · m⁻²)</td>
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<td>Stroke index, maximal (ml · m⁻²)</td>
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<td>Arteriovenous oxygen difference, maximal (ml · 100 ml⁻¹)</td>
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<td>Peak aortic velocity, maximal (cm · sec⁻¹)</td>
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\* \( p < .05 \).
**Table 2** Physiological Values in High Fit and Low Fit Boys Related to Scaled Anthropometric Variables

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<tr>
<th>Variable</th>
<th>High fit</th>
<th>Low fit</th>
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<tr>
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<td><em>M (SD)</em></td>
<td><em>M (SD)</em></td>
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<tr>
<td>VO₂max (ml · kg⁻⁰.⁷⁰ · min⁻¹)</td>
<td>166 (9)</td>
<td>127 (11*)</td>
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<tr>
<td>Stroke volume, rest (ml · m⁻⁶²)</td>
<td>55 (6)</td>
<td>46 (10*)</td>
</tr>
<tr>
<td>Stroke volume, maximal (ml · m⁻⁶²)</td>
<td>76 (12)</td>
<td>65 (9*)</td>
</tr>
<tr>
<td>Cardiac output, maximal (L · m⁻⁵¹)</td>
<td>15.71 (2.34)</td>
<td>13.02 (1.55*)</td>
</tr>
<tr>
<td>Arteriovenous oxygen difference, maximal</td>
<td>2.63 (0.27)</td>
<td>2.42 (0.36)</td>
</tr>
<tr>
<td>(ml · 100 ml⁻¹ · kg⁻⁴²)</td>
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*p < .05.

Significant differences were observed between groups in maximal heart rate or RER, indicating an equal testing effort.

Consistent with the correlational analysis of the total cohort, the only cardiovascular variables that separated the two groups were resting and maximal stroke volume, and maximal cardiac output. This was apparent regardless of method of size normalization. The pattern of stroke volume response in the two groups was similar (Figure 1), and the ratios of maximal to resting stroke volume were not significantly different in the high and low fit boys [1.38 (.18) and 1.43 (.30), respectively, *p > .05]. These findings indicate that differences in maximal stroke volume in the two groups reflected differences in resting values. Correlation coefficient between resting and maximal stroke volume for the entire study group was *r* = 0.67.

No significant difference was observed in resting echocardiographic left ventricular size between the high and low fit groups. Mean left ventricular EDD related to BSA⁰.⁶¹, the appropriate scaling factor for this cohort, was greater in the high fit [3.60 (.17) cm vs. 3.46 (.21) cm], but the difference was not statistically significant (one-tailed *p* = .10). Similarly, the high fit demonstrated a statistically insignificant greater resting left ventricular SF than the low fit [39.7 (6.5)% vs. 36.9 (4.2)%].

**Discussion**

This study provides evidence that maximal stroke volume is the sole physiological factor influencing individual differences in VO₂max per kg in populations of healthy, non-athletically trained children. Moreover, the findings suggest that factors influencing resting stroke volume are responsible for inter-individual variations in maximal stroke volume (and thus VO₂max). The failure to identify an association between VO₂max per kg and maximal arteriovenous oxygen difference implies that peripheral variables such as muscle aerobic enzyme capacity, mitochondrial density, and muscle capillarization are not limiting factors in maximal oxygen uptake in this population of subjects. Similarly, hemoglobin concentration influences in-
Figure 1 — Pattern of stroke index response in high and low fit boys.

In the present study, the stroke volume response to progressive exercise—an initial small rise and subsequent plateau—was similar in the high and low fit boys. The pattern was "displaced upward" in the high fit, parallel to that of the low fit, with significantly greater resting and maximal values. However, the ratio of maximal to resting stroke index was not different. This finding implies that maximal stroke volume is strongly influenced by values at rest and that the determinant(s) of aerobic fitness, as measured in the laboratory (VO₂ max per kg) in this cohort, lies among the factors that establish resting stroke volume.

Ventricular preload (end-diastolic left ventricular volume), intrinsic myocardial contractility, and ventricular afterload all influence resting stroke volume. Echocardiographic studies in adults have suggested that ventricular preload (left ventricular size) is the principal factor differentiating individuals with high and low VO₂ max (1, 7, 20). If so, greater plasma volume, larger intrinsic left ventricu-
lar chamber size, or resting bradycardia—all determinants of augmented ventricular preload—would be likely candidates as the critical factor responsible for separating VO₂max in the boys in this study.

Unfortunately, the echocardiographic data in this study failed to provide an answer to the question. The high fit group had, on average, a larger size-relative left ventricular EDD (1.4 mm) than the low fit boys, but this difference failed to achieve statistical significance. The difference in size-relative stroke volume between the two groups averaged 9 ml m⁻². Left ventricular EDD approximates a cube root function of end diastolic volume. If the difference in resting stroke volume between the two groups was entirely related to preload (i.e., left ventricular size), the expected average difference in LVED would have been approximately only 2 mm. It appears, then, that given the technical and biological variability affecting reproducibility of this technique, coupled with limits of resolution, echocardiography lacked sufficient sensitivity to identify whether differences in ventricular morphology and function were in fact present.

Turley and Wilmore described the relationships between resting left ventricular mass (by echocardiography) and submaximal stroke volume (by CO₂ rebreathing) in 7–9-year-old boys and girls (19). Correlations between the two were r = 0.32, 0.48, and 0.41 at 20, 40, and 60 W, respectively, on the cycle ergometer. On the treadmill, coefficients were r = 0.50, 0.56, and 0.47, respectively, at 3.0, 4.0, and 5.0 mph. The authors felt that these low correlations reflected the small number of subjects in the study (N = 24).

Such relationships may be influenced by body size as a covariant. Blimkie et al. found significant correlations between left ventricular end diastolic diameter, mass, and stroke volume at rest and VO₂max in a study of 117 boys, ages 10–14 years (2). However, body size accounted for most of the variance in VO₂max. It was concluded that cardiac dimensions were of only minimal importance in determining VO₂max once body size was taken into account.

It should be emphasized that the findings of the present study may be population-specific. In both child and adult endurance athletes, for instance, evidence exists that stroke volume rises rather than plateaus during progressive exercise (5, 10). This would suggest that factors influencing stroke volume during exercise (such as diastolic filling properties of the left ventricle) may be important in determining VO₂max in these subjects. On the other hand, stroke volume may decrease during exercise in pediatric patients with myocardial dysfunction, and factors limiting contractility may be critical to aerobic fitness in this group (14).

Likewise, there are no implications in this study regarding the cardiovascular variables that are responsible for change in aerobic fitness. Nonetheless, Eriksson and Koch demonstrated an average increase in VO₂max from 38.6 to 45.5 ml kg⁻¹ min⁻¹ in 9–11-year-old boys after a 4-month aerobic training program (4). This 17% rise was due almost entirely to an increase in maximal stroke volume from 67 to 80 ml. No significant change was observed in mean maximal arteriovenous oxygen difference (14.2 vs. 14.7 ml 100 ml⁻¹). These findings suggest that mechanisms responsible for improving VO₂max with training may be similar to those distinguishing high from low fit children.

It was not the purpose of this study to evaluate body composition effects on VO₂max. Still, it is important to note the striking influence observed of body fat content on VO₂max expressed per kg body weight, an effect of the inert load of adipose tissue on the denominator. In this cohort, percent body fat was more closely
correlated to VO₂max per kg \((r = -0.73)\) than was cardiac or stroke index \((r = 0.54, 0.52)\). That is, a value for VO₂max per kg in this population of subjects was more likely to provide information regarding a child’s body composition than his or her cardiovascular fitness.

Considerable attention has been focused on the potential errors that may result from relating physiological variables to body mass or surface area (the ratio standard) \((21)\). Possible spurious results may be avoided by the use of allometric analysis, in which such variables are related to scaling exponents of anthropometric measures. In this study, no advantages were observed with use of allometric analysis. As noted in a previous publication \((11)\), the appropriate exponents for cardiovascular variables related to mass or body surface area in this group of children are quite different than when using the ratio standard \((\text{mass}^{1.0}, \text{BSA}^{1.0})\). Nonetheless, in this study examining relationships between cardiovascular variables and VO₂max, no important differences were observed between use of the conventional ratio standard and allometrically derived exponents.

In summary, among a cross-section of healthy sixth-grade boys, maximal stroke volume, related to body size, provided the only physiological explanation for differences in VO₂max per kg. When boys of high and low fitness were compared, differences in maximal stroke volume reflected values at rest. This suggests that factors influencing resting stroke volume are responsible for variations in VO₂max per kg in this group of subjects. Body fat was more closely associated with mass-relative VO₂max than physiological variables, emphasizing the importance of considering body composition when interpreting values of VO₂max per kg.

**References**


**Acknowledgment**

This study was supported by a grant from the Massachusetts Governor’s Committee on Physical Fitness in Sports.