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Oxygen cost and oxygen uptake dynamics and recovery with 1 min of exercise in children and adults

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ZANCONATO, STEFANIA, DAN MICHAEL COOPER, AND YAACOV ARMON. *Oxygen cost and oxygen uptake dynamics and recovery with 1 min of exercise in children and adults.* J. Appl. Physiol. 71(3): 993–998, 1991.—To test the hypothesis that $\dot{V}O_2$ dynamics are different in adults and children, we examined the response to and recovery from short bursts of exercise in 10 children (7–11 yr) and 13 adults (26–42 yr). Each subject performed 1 min of cycle ergometer exercise at 50% of the anaerobic threshold (AT), 80% AT, and 50% of the difference between the AT and the maximal $\dot{V}O_2$ ($\dot{V}O_{2\max}$) and 100 and 125% $\dot{V}O_{2\max}$. Gas exchange was measured breath by breath. The cumulative O_2 cost [the integral of $\dot{V}O_2$ (over baseline) through exercise and 10 min of recovery ($ml\ O_2/J$)] was independent of work intensity in both children and adults. In above-AT exercise, O_2 cost was significantly higher in children [0.25 ± 0.05 (SD) ml/J] than in adults ($0.18 \pm 0.02\ ml/J$, $P < 0.01$). Recovery dynamics of $\dot{V}O_2$ in above-AT exercise [measured as the time constant ($\tau\dot{V}O_2$) of the best-fit single exponential] were independent of work intensity in children and adults. Recovery $\tau\dot{V}O_2$ was the same in both groups except at 125% $\dot{V}O_{2\max}$, where $\tau\dot{V}O_2$ was significantly smaller in children (35.5 ± 5.9 s) than in adults (46.3 ± 4 s, $P < 0.001$). $\dot{V}O_2$ responses (i.e., time course, kinetics) to short bursts of exercise are, surprisingly, largely independent of work rate (power output) in both adults and children. In children, certain features of the $\dot{V}O_2$ response to high-intensity exercise are, to a small but significant degree, different from those in adults, indicating an underlying process of physiological maturation.

anaerobic threshold; maturation; oxygen uptake kinetics

THE NATURAL GROWTH and development of the integrated cellular and cardiorespiratory adjustments to exercise are not yet fully understood. Some data suggest that the time course of O_2 uptake ($\dot{V}O_2$) at the onset of low-intensity exercise [work performed below the anaerobic threshold (AT) or lactate threshold] is the same in children and adults (6), suggesting a fully mature ability of children ≥ 7 yr to increase $\dot{V}O_2$ in response to physical activity. Other investigations demonstrated that children compared with adults have faster on-transient $\dot{V}O_2$ dynamics to maximal exercise (14), reach lower levels of blood lactate for similar degrees of heavy exercise (16, 21), and are less able to sustain exercise in the high-intensity range (2). These observations imply that there do exist growth-related differences in cellular O_2 metabolism or transport in response to exercise.

The goal of this study was to test the hypothesis that the dynamics of $\dot{V}O_2$ in response to exercise in children

are different from those in adults. We did this by devising protocols that would, to some extent, mimic patterns of exercise found in the daily life of children and that would also allow a precise analysis of the dynamic $\dot{V}O_2$ response. Thus we used a cross-sectional study design and compared the $\dot{V}O_2$ response with 1-min bursts of exercise at a variety of work intensities in a group of adults and children.

METHODS

Population

Ten healthy children [6 boys and 4 girls, aged 7–11 yr, mean 9.0 ± 1.3 (SD)] and 13 healthy adults (10 males and 3 females, aged 26–42 yr, mean 32.6 ± 4.8) comprised the study population. All were volunteers, had no chronic diseases, and did not smoke or use medication. The study was approved by the Human Subjects Committee of Harbor-UCLA Medical Center. Informed consent was obtained from each subject and guardian when appropriate. In addition, we reanalyzed maximal $\dot{V}O_2$ ($\dot{V}O_{2\max}$) and work rate (WR, also power output) data from 65 randomly selected healthy children (age range 6–18 yr, 36 boys) who participated in a study of cardiorespiratory responses to exercise in this laboratory published previously (7).

Protocol

Progressive exercise test. Each subject performed a ramp-type progressive exercise test on a electromagnetically braked cycle ergometer to determine the $\dot{V}O_{2\max}$ and the AT (also known as the lactate or ventilatory threshold) (18, 19).

Burst-exercise tests. The subjects performed 1-min bursts of varying-intensity exercise: 50% of the AT, 80% of the AT, 50% of the difference between the $\dot{V}O_{2\max}$ and the AT (we refer to this difference as Δ), 100% of the $\dot{V}O_{2\max}$, and 125% of the $\dot{V}O_{2\max}$. Each subject exercised at unloading pedaling (~ 7 – 12 W) for 3–5 min before exercise and for 10 min after exercise. The tests were performed in randomized order and usually required two or three separate sessions. When studies were performed on the same day, a sufficient interval (30–90 min) between studies was allowed so that all gas exchange variables and heart rate had returned to preexercise values.

Breath-by-Breath Measurements of Gas Exchange

Ventilation ($\dot{V}E$) and gas exchange were measured breath by breath. The subjects breathed through a mouthpiece connected to a turbine flowmeter and a low-resistance inspiratory-expiratory valve for continuous measurement of inspired and expired volume. The apparatus dead space was 90 ml. CO_2 and O_2 concentrations were measured by a mass spectrometer that sampled continuously from the mouthpiece at 1 ml/s. $\dot{V}E$ (BTPS), $\dot{V}O_2$ (STPD), CO_2 output (STPD), end-tidal PO_2 , and end-tidal PCO_2 were computed on-line breath by breath as previously described (3). Heart rate was measured beat by beat by a standard lead I electrocardiogram with the use of three electrodes placed on the chest. The data from each test were stored on digital tape for further analysis.

Data Analysis

$\dot{V}O_{2\max}$ and AT. $\dot{V}O_{2\max}$ was taken as the peak $\dot{V}O_2$ achieved by each subject before cessation of exercise. The AT was measured noninvasively from the gas exchange data obtained during the progressive exercise (7). AT was defined as the $\dot{V}O_2$ at which the ventilatory equivalent for O_2 ($\dot{V}E/\dot{V}O_2$) and end-tidal PO_2 increased without an increase in the ventilatory equivalent for CO_2 ($\dot{V}E/CO_2$ output) and end-tidal PCO_2 .

Normalization

To compare the $\dot{V}O_2$ responses from the adults and children, we used several strategies. First, the data were normalized to body weight by using the increase in $\dot{V}O_2$ above baseline (i.e., $\Delta\dot{V}O_2/kg$). Second, the data were normalized to the actual work performed in two ways as described below.

Cumulative O_2 cost per joule. We defined the cumulative O_2 cost of exercise bursts as the integral of $\dot{V}O_2$ over baseline values (i.e., unloaded cycling) from the onset of exercise, through the exercise period, and for 10 min of recovery (Fig. 1). To facilitate comparison of results from adults and children, we divided the cumulative $\dot{V}O_2$ values by the external work done (ml O_2/J). In this manner, differences observed indicated true difference in the relationship between $\dot{V}O_2$ and work performed. To determine whether the 10-min period allowed for complete recovery of the $\dot{V}O_2$, we compared the mean $\dot{V}O_2$ during the last 30 s of the preexercise period with the mean value of the last 30 s of the recovery phase. This analysis was done only for the highest work rates (i.e., 100%max and 125%max).

To gain additional insight into the physiological mechanism of the $\dot{V}O_2$ response, we measured the cumulative O_2 volume that occurred during recovery (Fig. 1). For each subject, we calculated the ratio of these cumulative costs (i.e., recovery to total) at each WR.

End-exercise O_2 cost per watt. In addition to the cumulative O_2 cost, we calculated the end-exercise O_2 cost by dividing the mean $\dot{V}O_2$ (above baseline) over the last 6 s of exercise by the WR (ml $O_2 \cdot \min^{-1} \cdot W^{-1}$).

FIG. 1. O_2 uptake response to 1 min of high-intensity exercise (125% of the maximal work rate) in an 8-yr-old girl. Shown also is best-fit single exponential as described in text. Vertical line indicates end of 60-s exercise. Area under $\dot{V}O_2$ curve from time 0 to end of 10-min recovery period [mean baseline values (---) were subtracted] is used to calculate cumulative O_2 cost of exercise. Area to right of vertical line to end of recovery (again, mean baseline values were subtracted) represents O_2 cost for recovery period.

Kinetics of $\dot{V}O_2$

On-transient. As noted, 60-s periods of data collection are not sufficient to allow the achievement of a steady state for $\dot{V}O_2$. Thus modeling these data to exponential functions is confounded by the lack of knowledge of the asymptotic steady state. To empirically quantify the on-transient response, we calculated the half time ($t_{1/2}$) as the time required to reach one-half the value of the preexercise to peak $\dot{V}O_2$ (the largest $\dot{V}O_2$ achieved by the subject).

Recovery time. $\dot{V}O_2$ recovery time was determined by fitting the recovery data (i.e., after the 1-min WR) from each subject with a single-exponential equation. Nonlinear techniques were used to calculate the parameters of the characteristic equation (12)

$$\dot{V}O_2(t) = Ae^{-kt} + C \quad (1)$$

where $\dot{V}O_2(t)$ is the value of $\dot{V}O_2$ at time t after exercise, A is a parameter, k is the rate constant, and C is the asymptotic baseline value. The time constant ($\tau = 1/k$) was used to quantify the recovery time and indicates the time required to achieve 63.2% of the difference between peak and baseline values.

On the basis of previous analysis of $\dot{V}O_2$ responses to high-intensity exercise (1), where the on-transient data were better fit by models more complex than a single exponential, the recovery data for the highest WR (125%max) were fitted both with a single-exponential (as described above) and a two-exponential model

$$\dot{V}O_2(t) = A_1e^{k_1t} + A_2e^{k_2t} + C \quad (2)$$

Statistical Analysis

Analysis of variance (repeated measures) with subsequent modified t tests (Duncan and Bonferroni method)

TABLE 1. *WR normalized to body weight in children and adults*

	50% AT	80% AT	50%Δ	100%max	125%max
Adults	0.45±0.12	0.85±0.22	2.11±0.34	3.12±0.52	3.88±0.61
Children		0.73±0.19	1.63±0.24*	2.33±0.32*	2.96±0.43*

Values are means ± SD in W/kg. WR, work rate; AT, anaerobic threshold. * $P < 0.01$, difference between values in children and adults.

FIG. 2. Cumulative O_2 cost per joule at different work intensities in adults and children. Values are means ± SD. Cumulative O_2 cost was not affected by increasing work intensity in children and adults. However, cost was significantly higher in children than in adults at 50%Δ (* $P < 0.001$), 100%max, and 125%max (** $P < 0.01$).

was used for the statistical analysis of work-intensity related differences within and between the groups of adults and children. In children, the noise-to-signal ratio for the lowest WR (50% AT) was too high to permit accurate data analysis. A paired t test was used to compare preexercise and recovery values of $\dot{V}O_2$. In the analysis of data obtained in our previous study (7), linear regression was used to assess the correlation between the ratio $\dot{V}O_{2\max}/WR_{\max}$ and both height and age. Statistical significance was taken at $P < 0.05$. Data are expressed as means ± SD.

RESULTS

$\dot{V}O_{2\max}$ and AT

There were no significant differences in the $\dot{V}O_{2\max}$ per kilogram between adults (41.5 ± 8.5 ml $O_2 \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$) and children (41.7 ± 5.8 ml $O_2 \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$). Similarly, no differences were found between the AT per kilogram in children (24.8 ± 5.6 ml $O_2 \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$) and adults (22.6 ± 3.5 ml $O_2 \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$). Despite this, the WR normalized to body weight was significantly higher in adults for all the tests above AT (Table 1).

Cumulative O_2 Cost per Joule

There was no difference between the preexercise and the end-recovery $\dot{V}O_2$ in either group, indicating that recovery for this variable was complete within the 10 min of observation. The cumulative O_2 cost of exercise per joule was independent of the work intensity in children and adults (Fig. 2). For WRs above AT (i.e., 50%Δ, 100%max,

TABLE 2. *End-exercise O_2 cost in children and adults*

	50% AT	80% AT	50%Δ	100%max	125%max
Adults	9.5±2.1*	8.4±0.8†	7.4±1.2	7.1±0.8	6.5±0.7
Children		10.9±2.6	10.9±2.1‡	10.6±3.1‡	9.8±2.8‡

Values are means ± SD in ml $\cdot$ min $^{-1} \cdot$ W $^{-1}$. * Greater than 80% AT, 50%Δ, 100%max, and 125%max, $P < 0.001$. † Greater than 100%max and 125%max, $P < 0.05$. ‡ Greater than adults at comparable work intensity, $P < 0.001$.

FIG. 3. Ratio of $\dot{V}O_{2\max}$ to maximal work rate ($\dot{V}O_{2\max}/WR_{\max}$) as a function of height. Dashed lines enclose mean ± 2 SE of 65 healthy children and teenagers previously studied in our laboratory. ●, Children in present study; ○, adults. Similar to previous data, ratio decreased significantly with height (as well as age, not shown) in present study.

125%max), O_2 cost per joule was significantly higher in children (mean O_2 cost 0.25 ± 0.05 ml/J) than in adults (0.18 ± 0.02 ml/J, $P < 0.01$). In adults the recovery O_2 cost (as percent cumulative O_2 cost) at the highest WR (125%max) was significantly higher ($62.3 \pm 4.2\%$) than at the two WRs below AT ($55.2 \pm 13.4\%$ at 50% AT and $54.3 \pm 4.1\%$ at 80% AT, $P < 0.05$). At 50%Δ and 100%max the ratio was 57.8 ± 2.7 and $59.3 \pm 3.5\%$, respectively. There was no significant effect of WR on the ratio in children ($51 \pm 12\%$ at 80% AT, $57.5 \pm 5.8\%$ at 50%Δ, $56.1 \pm 5.6\%$ at 100%max, $59.7 \pm 4.1\%$ at 125%max). No significant difference was found between the values in children and adults at any of the WRs studied.

End-Exercise O_2 Cost per Watt

In adults, the end-exercise O_2 cost decreased as WR increased (Table 2). By contrast, the values in children did not change as WR increased. Moreover, the children's results were higher than those of adults for all the WRs above AT.

Reanalysis of Previous Data

The higher O_2 cost in children was an unexpected finding. Thus we thought it would be useful to reanalyze previously collected data to corroborate the results. There was a small but significant decrease in the $\dot{V}O_{2\max}/WR_{\max}$ as both height ($r = -0.48$, $P < 0.001$) (Fig. 3) and age ($r = -0.53$, $P < 0.001$) increased in the 65 children previously studied. The correlation between $\dot{V}O_{2\max}/WR_{\max}$ and height in the current subjects was

FIG. 4. Time constant for $\dot{V}O_2$ recovery ($\tau\dot{V}O_2$) in adults and children. Values are means $\pm$ SD. In adults $\tau\dot{V}O_2$ increased significantly from 50 to 80% AT ($P < 0.01$), but we found no effect of work intensity on $\tau\dot{V}O_2$ in children. At 125% max $\tau\dot{V}O_2$ was significantly higher in adults than in children (* $P < 0.001$).

$$\dot{V}O_{2\max}/WR_{\max} (l \cdot \min^{-1} \cdot W^{-1})$$

$$= -0.095 \cdot \text{height (cm)} + 29.11; \quad r = -0.86, P < 0.001$$

Kinetics of $\dot{V}O_2$

On-transient. The $t_{1/2}$ was not affected by WR in either the children or the adults tested. Moreover, there were no differences in the mean values between the groups (23.0 ± 5.3 s, children; 24.8 ± 4.7 s, adults).

Recovery Time Constant

In adults the $\dot{V}O_2$ recovery time constant increased between the two lowest WRs (50 and 80% AT) ($P < 0.01$), but the time constants were independent of WR for the remaining protocols. In the children, the $\dot{V}O_2$ recovery time constants were independent of WR. The recovery time constants were lower in children than in adults, but only at 125% of the $\dot{V}O_{2\max}$ was there a statistically significant difference ($P < 0.001$) (Fig. 4). We observed no differences in the recovery time constants of females compared with males in either the adults or children. We also attempted to fit a two-exponential equation for the 125% max exercise intensity recovery data. In four children and six adults, the two-exponential model resulted in slightly better fits than the single-exponential.

DISCUSSION

The response to and recovery from 1-min bursts of exercise reveal much about the integrated cardiorespiratory adjustment to changes in metabolic rate. It is noteworthy that in both adults and children $>50\%$ of the total O_2 cost occurred during the recovery period. The significantly greater cumulative O_2 cost per joule in children at virtually all WRs was the major difference between the groups. In addition, the $\dot{V}O_2$ recovery time was prolonged in adults at the highest WR. Surprisingly, we found that the $\dot{V}O_2$ recovery times and cumulative O_2 cost per joule were minimally affected by WR in both adults and children.

Previous investigators have examined the dynamics of $\dot{V}O_2$ at the beginning and end of exercise by the use of the

concepts of O_2 debt and deficit (11, 20). These measurements can only be made if the exercise conditions allow for the achievement of a steady state for $\dot{V}O_2$. The short exercise time and high intensities of our protocol did not result in a steady-state $\dot{V}O_2$ in any of the subjects. Nonetheless, certain insights gained from analysis of O_2 debt-deficit relationships are relevant to our data. For example, Whipp et al. (20) demonstrated that the total O_2 cost for constant WR exercise of short duration (i.e., <4 min) was greater than for longer periods. The results of our study are consistent with this observation: the mean cumulative O_2 cost (converted to $ml O_2 \cdot \min^{-1} \cdot W^{-1}$) was 11.5 ± 0.65 in adults compared with O_2 costs of $10.1 ml O_2 \cdot \min^{-1} \cdot W^{-1}$ measured from steady-state tests (10). Similarly in children, the O_2 cost was $15.3 \pm 0.65 ml \cdot \min^{-1} \cdot W^{-1}$ compared with previously reported values of ~ 10.2 (7).

The reanalysis of previously collected data demonstrates a higher $\dot{V}O_2$ per watt at maximal WR in children than in young adults even though the $\dot{V}O_{2\max}/kg$ was the same (7) (Fig. 3). These results are consistent with the findings of the present study in which the cumulative O_2 cost per joule was consistently greater in the children than in the adults (Fig. 2). Our calculations of the cumulative O_2 cost represented the metabolic energy expenditure of the work performed for two reasons: first, because the $\dot{V}O_2$ had returned to baseline values during the observation period (see RESULTS), meaning that we had measured all the increase in $\dot{V}O_2$ resulting from the performance of exercise; and second, because we subtracted the $\dot{V}O_2$ due to unloaded pedaling and, thereby, eliminated energy expenditure related to inertial and frictional costs incurred as the subject moved his or her legs in the performance of cycle ergometry.

There are several possible mechanisms for the higher total O_2 costs per joule in children than in adults. Substrate utilization itself can modify the relationship between $\dot{V}O_2$ and WR because the high-energy phosphate-to- O_2 ratio is 2.82 for fats and 3.00 for glucose (17). If children metabolized solely fat and adults only glucose (which is highly unlikely), then the difference in O_2 cost could be no more than 6%. The difference we observed was, on average, almost 40% greater in children and, therefore, inconsistent with a growth-related difference in substrate utilization.

The larger O_2 cost in children could reflect a greater influence of O_2 -requiring processes such as thermoregulation that accompany muscular work (13). Heat loss during exercise in children is likely to be facilitated by their larger ratio of surface area to body mass, and this would tend to reduce rather than increase any additional O_2 requirement for the maintenance of temperature homeostasis. Other O_2 -dependent processes like cardiac or respiratory work are small in magnitude and unlikely to account for the differences in cumulative O_2 cost that we observed between adults and children (4).

Margaria et al. (15) were among the first to postulate the mechanisms responsible for O_2 kinetics at the onset of exercise. One of the determinants of $\dot{V}O_2$ at the on-transient of exercise was the amount of O_2 stored in the tissues available for aerobic energy metabolism. At the end of exercise, these stores would be replenished. The

stored O_2 (largely venous blood) accounted for a small but measurable amount of the O_2 deficit. It is noteworthy that children have slightly smaller hemoglobin concentrations than do adults, and smaller stores might result in more rapid $\dot{V}O_2$ kinetics. However, we found no apparent growth-related differences in $\dot{V}O_2$ on-transient kinetics in either the present study or in our previous investigation of $\dot{V}O_2$ kinetics during low-intensity exercise (6). Moreover, the cumulative O_2 cost is not likely to be affected by the size of the O_2 stores if they return to their preexercise values in a relatively short period of time. It is important to note that the brief exercise period (i.e., 1 min) virtually precluded the possibility of evaluating all but the simplest $\dot{V}O_2$ kinetics at the onset of exercise. For example, we would not be able to find age-related differences in slower exponential components of the on-transient, even if they existed, using the current protocol and data analysis.

Biomechanical factors (e.g., use of additional muscle groups for limb acceleration) have been used to explain greater energy expenditure during walking and running in children than in adults (5, 8). However, these studies were limited because one cannot measure the precise work done by the use of treadmill exercise. In addition, cycle ergometry is likely to involve comparable muscle groups in adults and children because pedaling on the ergometer constrains limb movement to a greater degree than does walking or running.

The end-exercise O_2 cost per watt did not entirely parallel the values obtained for the cumulative O_2 cost per joule. The end-exercise O_2 cost was consistently higher in children than in adults (Table 2). Although the end-exercise O_2 cost fell in adults as WR increased, it was unaffected by the work intensity in children. These observations may be related to the maturation process associated with anaerobic metabolism.

Lower levels of blood lactate during exercise have been found in children than in adults (16, 21). The metabolism of pyruvate to lactate can, for a limited period of time, act as an O_2 -sparing mechanism during the exercise response by providing a source of ATP rephosphorylation that does not require O_2 . As judged by the fall in the O_2 cost at higher work intensity in adult subjects, a greater proportion of the energy expenditure at the end of the 1-min exercise bout was derived from anaerobic sources. Note, however, that the total O_2 cost per joule did not change (Fig. 2), implying greater $\dot{V}O_2$ during recovery. In fact, at 125%max, the O_2 cost during recovery (as percent cumulative O_2 cost) in adults was significantly greater than for below-AT exercise. Moreover, the finding of generally higher end-exercise O_2 costs per watt in children during high-intensity exercise suggests a greater dependence on O_2 -dependent ATP generation. Ultimately, this issue will be resolved either by measurements of blood lactate during exercise in children (few healthy children or their parents agree to blood sampling during exercise tests) or, alternatively, by using noninvasive estimates of cellular energy metabolism using magnetic resonance spectroscopy. There are, in fact, limited data suggesting that the level of phosphofructokinase, a key muscle glycolytic enzyme, is lower in children than in adults (9). This observation supports the concept that children have less

"anaerobic capacity" than do adults because glycolysis provides the pyruvate necessary for anaerobic ATP production. Whether the greater O_2 dependence in children represents 1) less ability for anaerobic metabolism, 2) less efficient coupling of oxidative metabolism to force generation in the muscle, or 3) a more efficient ability to increase uptake of atmospheric O_2 has yet to be determined.

It was surprising to find that the kinetics of the recovery phase were only minimally WR dependent in adults and virtually independent of work intensity in children (Fig. 4). Previous studies in this laboratory demonstrated that constant WR performed above the subject's AT results in on-transient kinetics that are not described well by single-exponential functions. Accurate modeling of the high-intensity $\dot{V}O_2$ response requires the addition of another slower (i.e., longer time constant) exponential term to adequately fit the data.

The increasing complexity may result from O_2 -dependent processes of lactate metabolism [e.g., lactate oxidation or lactate conversion to glucose (Cori cycle)] once excess lactate has been produced during high-intensity exercise (17). Thus it is reasonable to expect that the metabolic processes associated with high-intensity exercise, once initiated, would prolong the recovery dynamics of $\dot{V}O_2$. However, models more complex than the single exponential did little to improve the fit of the data.

Why did short-term burst exercise lead to relatively simple $\dot{V}O_2$ kinetics during the recovery phase? One possible explanation is that the 1-min period of exercise was too brief to induce the slow metabolic adaptations characteristic of longer bouts of high-intensity exercise. The response dynamics of the slower process (related to the second exponential term in the on-transient) are so slow that there is only a very small effect on the recovery after 1 min of exercise.

Although the time constant for O_2 recovery ($\tau\dot{V}O_2$) tended to be smaller (i.e., faster recovery) in children at all work intensities, $\tau\dot{V}O_2$ was significantly faster than in adults only at 125%max. As noted, a greater anaerobic metabolism may explain the slower $\dot{V}O_2$ responses, thus the slower $\dot{V}O_2$ kinetics at the highest WR in the adults.

Finally, there may be a practical implication of our data for clinical investigations of abnormalities in cardiorespiratory responses to exercise in children. Above-AT exercise protocols are advantageous because the noise-to-signal ratio is often lower than in below-AT exercise, but the metabolic acidosis and stress that accompany high-intensity exercise are not advisable in patients with impaired function. Using short bursts of exercise in individuals with heart or lung disease could mitigate this problem. Moreover, choosing the appropriate WR to ensure a valid comparison with normal children is easier because the on-transient $t_{1/2}$, the cumulative O_2 cost, and the recovery $\tau\dot{V}O_2$ are virtually independent of WR in healthy children.

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