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Are obese children truly unfit? Minimizing the confounding effect of body size on the exercise response

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To test the hypothesis that obese children are unfit (i.e., have abnormal responses to exercise testing consistent with reduced levels of habitual physical activity), we used new analytic strategies in studies of 18 obese children performing cycle ergometry. The subjects' weight (mean ± SD) was 168 ± 24% that predicted by height, and the age range was 9 to 17 years. Size-independent measures of exercise (e.g., the ratio of oxygen uptake ($\dot{V}O_2$) to work rate during progressive exercise and the temporal response of $\dot{V}O_2$, carbon dioxide output ($\dot{V}CO_2$), and minute ventilation ($\dot{V}E$) at the onset of exercise) were used. The ability to perform external mechanical work was corrected for $\dot{V}O_2$ at unloaded pedaling (change in maximum oxygen uptake ($\Delta\dot{V}O_2$max) and in anaerobic threshold (ΔAT). On average, obese children's responses were in the normal range: $\Delta\dot{V}O_2$max, 104 ± 41% (±SD) predicted (by age); ΔAT, 85 ± 51%; ratio of change in $\dot{V}E$ to change in $\dot{V}CO_2$, 111 ± 21% and ratio of change in $\dot{V}O_2$ to change in work rate, 100 ± 24%, but six of the obese children had values of $\Delta\dot{V}O_2$max or ΔAT that were more than 2 SD below normal. In addition, obese children did not have increased $\Delta\dot{V}O_2$max or ΔAT with age as observed in nonobese children. Although the response time of $\dot{V}O_2$ was normal (99 ± 32% of predicted), those for both $\dot{V}CO_2$ and $\dot{V}E$ were prolonged. We conclude that the finding of obesity in a child is not a reliable indicator of poor fitness but that testing cardiorespiratory responses to exercise can be used to identify subjects with serious impairment and to individualize therapy. (J PEDIATR 1990;116:223-30)

Understanding the relationship between body mass and metabolic function during physical activity is fundamental for the management of childhood obesity, but an ideal way to account for the metabolic effect of increased adipose tissue when gauging cardiorespiratory responses has not been

found. The question, "Should an obese 6-year-old child who weighs as much as a nonobese 12-year-old be expected to have the same physiologic capacity for exercise as the older child?" becomes clinically important in developing an optimal plan for therapy. If it can be determined that an obese child is "unfit" (that is, has a below-normal level of habitual physical activity manifested as abnormal responses to standard exercise testing), a program combining exercise and diet may be beneficial.[1-4] If the child has "normal" fitness and activity, however, attempting to impose additional exercise may be ineffectual. The problem of defining normal values for obese subjects has traditionally been solved by correcting for the person's body weight, and different conclusions about cardiorespiratory responses have been reached depending on the adjustment used.[5-7]

AT	Anaerobic threshold
ΔAT	Change in anaerobic threshold
$\Delta\dot{V}CO_2$	Change in carbon dioxide output
$\Delta\dot{V}E$	Change in minute ventilation
$\Delta\dot{V}O_2$	Change in oxygen uptake
$\Delta\dot{V}O_2max$	Change in maximum oxygen uptake
ΔWR	Change in work rate
τ	Time constant
$\tau\dot{V}CO_2$	Time constant of carbon dioxide output
$\tau\dot{V}E$	Minute ventilation time constant
$\tau\dot{V}O_2$	Time constant of oxygen output
$\dot{V}CO_2$	Carbon dioxide output (liters per minute, standard temperature and pressure, dry)
$\dot{V}E$	Minute ventilation (liters per minute, body temperature and pressure, saturated)
$\dot{V}O_2$	Oxygen uptake (liters per minute, standard temperature and pressure, dry)

Our goal was to test the hypothesis that obese children are unfit by using exercise protocols and analytic strategies that were designed to minimize the effect of body mass on determining the normality of the cardiorespiratory response to exercise. To accomplish this goal, we used gas exchange responses to exercise, which are known to be relatively independent of body size. In addition, to account for the functional effect of increased body weight on cycle ergometer exercise, we measured the gas exchange response to exercise during unloaded (zero-watt) pedaling. We then could compare the ability of the obese and nonobese subjects to increase metabolic rate above the unloaded steady state in response to an increasing external work rate.

METHODS

Population. The study sample consisted of 18 obese children and teenagers (13 boys) ranging in age from 9 to 17 years (mean $\pm$ SD 13.5 $\pm$ 2.5 years). Obesity was defined as a body weight of >120% predicted by height,[8] and subjects' weight was 142% to 205% predicted (168 $\pm$ 24%). No child had a known cause for the obesity or was taking medications for chronic disease at the time of study.

Progressive exercise protocol. The protocol consisted of a continuously increasing (ramp) work rate pattern[9] in which an electromagnetically braked cycle ergometer (modified Cateye ergometer, Tsuyama Manufacturing Co., Osaka, Japan) was used. Subjects began with 4 minutes of cycling at a zero-watt (unloaded) work rate. The mean oxygen uptake during the 4-minute period was taken as the unloaded cycling $\dot{V}O_2$. Calibration of our ergometer indicated that the actual work at the zero-watt reading was approximately 7 to 10 watts; in the figures we refer to unloaded cycling as "0 watt." The work rate was then continuously increased at a constant rate. The increase in work rate per minute was selected so that the total exercise duration would be more than 6 minutes and less than 14 minutes. The

mean time for the test (not counting the warm-up) was 9 minutes. The children were instructed to raise a hand when they could not continue, and on this signal the work rate was reduced to zero watt. The investigators encouraged the subjects to perform at maximal effort.

The progressive exercise test was used to measure a number of indices of the cardiorespiratory response to exercise. If size-dependent gas exchange measures such as $\dot{V}O_2max$ are normalized to body weight (i.e., $\dot{V}O_2max$ per kilogram body weight), obese children appear to be less fit than normal subjects.[7] This is due to the confounding effect of the larger ratio of fat to muscle tissue, which invariably results in lower $\dot{V}O_2max$ per kilogram even in a child with a normal exercise capacity for his developmental stage. An adipose-independent measure of growth, such as body height, can be used for normal values, but the finding of a normal value for maximum oxygen uptake or anaerobic threshold could be misleading because the metabolic and mechanical cost of increased adipose tissue should result in greater than expected values. To account ideally for the effect of increased fat tissue, the metabolic rate should be related to the mass of the muscle tissue that is responsible for the increasing $\dot{V}O_2$ during exercise. No easily accessible procedures exist to quantify muscle mass in vivo. Lean body mass is used as an estimate of muscle tissue mass in a variety of techniques, but each has its own set of assumptions, errors, and limitations.[10]

An alternative strategy, based on actual measurements of metabolic function rather than attempts to somehow correct for the obese subject's body weight, seemed feasible if data collected during exercise testing were used. We chose to identify the functional effect of the increased weight on cycle ergometry exercise. This effect would be measured as the additional $\dot{V}O_2$ at zero-watt (unloaded) pedaling. Once the legs are moving at a constant rate, then, during a continuously increasing exercise protocol, any increase in metabolic rate is solely the function of the external work being done. On the cycle ergometer the external work is known precisely. Thus we defined the following measures of the gas exchange response to progressive exercise:

1. The change in maximum oxygen uptake was calculated as the difference between $\dot{V}O_2max$ and $\dot{V}O_2$ (unloaded pedaling).

2. As carbon dioxide is liberated by the bicarbonate buffering of lactic acid, minute ventilation and carbon dioxide output increase out of proportion to the increase in $\dot{V}O_2$, thereby allowing the noninvasive determination of the anaerobic threshold.[11] In each subject we measured the AT by finding the $\dot{V}O_2$ above which $\dot{V}E/\dot{V}CO_2$ and end-tidal partial pressure of oxygen increased without an increase in $\dot{V}E/\dot{V}CO_2$ or a decrease in end-tidal partial pressure of carbon dioxide as previously described.[9] The change in anaer-
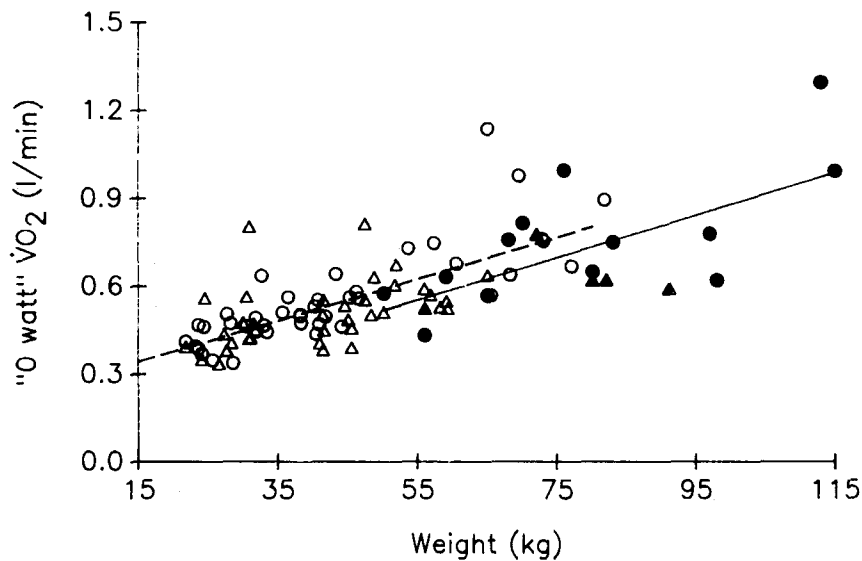
Fig. 1. Effect of body weight on unloaded cycle ergometry in obese and nonobese children. The x axis represents body weight in kilograms, and the y axis represents oxygen uptake ($\dot{V}O_2$ in L × min^{-1}) during zero-watt, or unloaded, cycling. *Clear triangles* and *circles* represent healthy nonobese girls and boys, respectively. *Black triangles* and *circles* represent obese girls and boys, respectively. Best-fit lines for obese and nonobese children are shown as *closed* and *dashed lines*, respectively. $\dot{V}O_2$ during unloaded pedaling increased significantly with body weight in both obese and nonobese subjects. There was no difference of slope or intercepts of linear regression between obese and nonobese subjects.

obic threshold was calculated as the difference between the AT and $\dot{V}O_2$ (unloaded pedaling).

3. The oxygen cost of exercise (change in oxygen uptake/ change in work rate),[9] the increase in $\dot{V}O_2$ that accompanies a given increase in work rate, was determined from the slope of the relationship between $\dot{V}O_2$ and work rate during the progressive test. The oxygen cost of exercise is independent of weight and age in children.

4. The $\dot{V}E$-$\dot{V}CO_2$ slope, the increase in $\dot{V}E$ for a given increase in $\dot{V}CO_2$ during the progressive exercise test (change in minute ventilation/change in carbon dioxide output), was determined from the slope of the relationship between $\dot{V}E$ and $\dot{V}CO_2$.[2] The $\dot{V}E$-$\dot{V}CO_2$ slope decreases to a small but significant degree with increasing age, height, or weight in children.

The normal values for $\Delta\dot{V}O_2$max and ΔAT were obtained from healthy nonobese subjects (44 girls and 42 boys ranging in age from 6 to 17 years) previously tested in our laboratory. There were no significant differences in height between the control subjects and the obese subjects.

Constant work rate protocols. Constant work rate protocols were used to measure the dynamic gas exchange and heart rate responses to exercise at the onset of exercise. The work rate chosen was 75% of the work rate corresponding to the subject's AT. Each subject performed a minimum of six transitions from rest to constant work rate. Exercise periods were 6 minutes each, and heart rate, $\dot{V}O_2$, $\dot{V}E$, and

$\dot{V}CO_2$ returned to the preexercise resting values before a repetition was performed. The subjects began exercise with the activation of a green light signal at the end of exhalation; there was no voice command. So that no energy expenditure would be required to overcome the inertia of the flywheel at the start of exercise, the ergometer flywheel was motorized and maintained at a rate of 60 rpm until the onset of pedaling. When the subject started to pedal, the motor maintaining the flywheel was turned off.

Normal values for the dynamic responses of gas exchange and heart rate at the onset of exercise were obtained from 29 children and teenagers ranging in age from 6 to 18 years. Since we have found no significant gender-related differences for the time constant of $\dot{V}E$, $\dot{V}CO_2$, or $\dot{V}O_2$, the obese subjects were compared as a group to the normal subjects.

Breath-by-breath measurement of gas exchange. Subjects breathed through a low-impedance, turbine, volume transducer for measurement of inspiratory and expiratory volumes. The dead space of the mouthpiece and turbine device was 90 ml. Respired partial pressures of oxygen and carbon dioxide were determined by mass spectrometry from a sample drawn continuously from the mouthpiece at 1 ml/ sec^{-1}. The electrical signals from these devices undergo analog-to-digital conversion for the on-line breath-by-breath computation of oxygen uptake ($\dot{V}O_2$, standard temperature and pressure, dry), carbon dioxide output ($\dot{V}CO_2$, standard
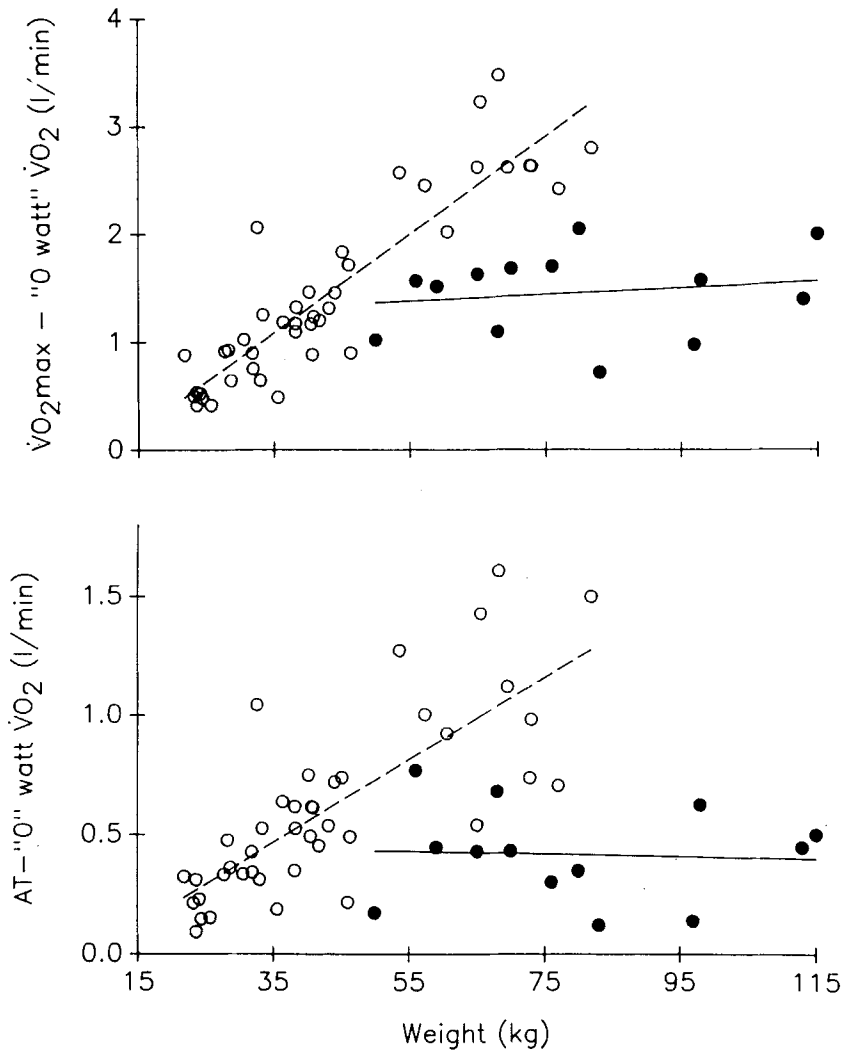
Fig. 2. Effect of body weight on $\Delta\dot{V}O_2$max *(upper panel)* and ΔAT *(lower panel)* in obese *(black circles)* and nonobese *(clear circles)* boys. In nonobese children both $\Delta\dot{V}O_2$max and ΔAT increased significantly with weight (see text). By contrast, normal relationship between these exercise responses and body weight was distorted in obese children.

temperature and pressure, dry), and expired ventilation ($\dot{V}E$, body temperature and pressure, saturated).

The system was calibrated daily. The cycle ergometer used in the experiments was computer driven with a servomechanism to correct for changes in a subject's pedaling rate; in this manner the work rate was kept within 1% when the subject pedaled between 50 and 70 rpm. The ergometer was also calibrated on a regular basis. In our laboratory a separate ergometer has been built to fit the small limb and arm lengths of children.

Data analysis. Data from the six work rate transitions in each subject were superimposed and averaged in an effort to reduce the naturally occurring respiratory "noise."[13] We studied kinetics of gas exchange responses by determining

τ in the following equation[14]: $\Delta\dot{V}(t) = \Delta\dot{V}ss \times (1 - e^{-t/\tau})$ where $\Delta\dot{V}(t)$ is the increase in $\dot{V}O_2$, $\dot{V}CO_2$, or $\dot{V}E$ above the prior control values at any exercise time (t); $\Delta\dot{V}ss$ is the difference between rest and steady-state exercise $\dot{V}$; e is the base of the system of natural logarithms; and τ is the time required to reach 63% [(1 $\times$ 1/e) $\times$ 100%] of $\Delta\dot{V}ss$. Data at the start of exercise were fitted by curves with a fitting window between 20 to 120 sec after the start of exercise. The best-fit exponential was found by iterative techniques. The model calculated a delay representing the difference in time between the start of exercise and the extrapolated start of the best-fit exponential.

Statistical analysis. Standard techniques of linear and exponential regression were used. Comparisons between
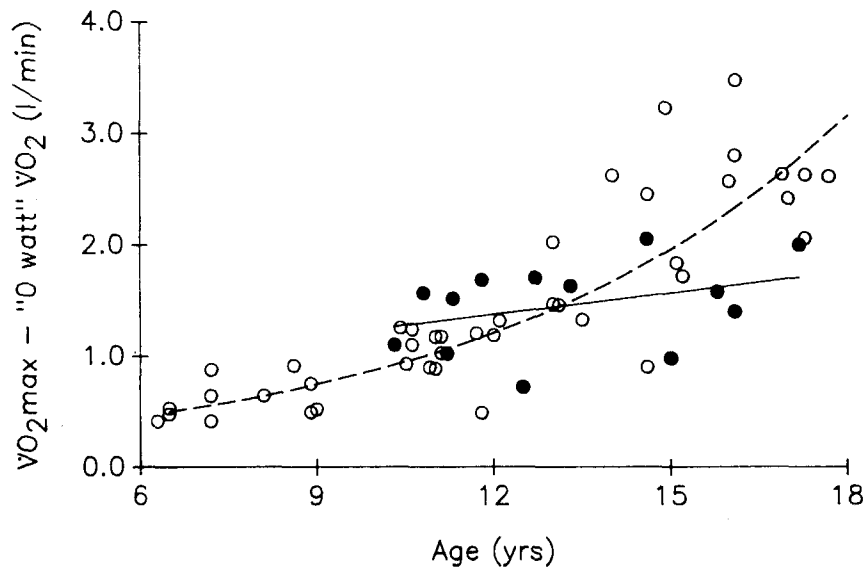
Fig. 3. Relationship between age and $\Delta\dot{V}O_2$max in obese *(black circles)* and nonobese *(clear circles)* boys. *Dashed line* indicates best-fit exponential equation for nonobese subjects. Although obese children were, by and large, well within normal range, they had less marked increase in $\Delta\dot{V}O_2$max with age than did nonobese subjects.

normal control subjects and obese subjects were made with independent t tests. Values are represented as mean ± SD.

RESULTS

Unloaded cycle ergometer. As can be seen in Fig. 1, $\dot{V}O_2$ increased significantly with body weight in both nonobese and obese children. For nonobese children, linear regression analysis resulted in the following relationship: zero-watt $\dot{V}O_2(L \times min^{-1}) = 0.0071 \times weight$ (kg) $+ 0.236$; $r = 0.81$, $p < 0.001$. For obese children the relationship was as follows: zero-watt $\dot{V}O_2 (L \times min^{-1}) = 0.0072 \times weight$ (kg) $+ 0.158$; $r = 0.71$, $p < 0.003$. There was no statistically discernible difference between these equations. An allometric analysis was performed to determine the mass scaling factor for the relationship between body mass and zero-watt cycling. For the obese and nonobese children considered as a group we found that zero-watt $\dot{V}O_2$ (L $\times min^{-1}$) = 0.079 $\times$ [weight (kg)]$^{0.51}$; $r = 0.77$, $p < 0.005$.

Progressive exercise protocols. In obese children the relationship between body weight and either the $\Delta\dot{V}O_2$max or ΔAT was grossly distorted (Fig. 2). In nonobese boys $\Delta\dot{V}O_2$max increased in a highly linear manner with body weight: $\Delta\dot{V}O_2$max (L $\times min^{-1}$) = 0.046 $\times$ weight (kg) $-$ 0.513; $r = 0.90$, $p < 0.001$. For obese boys, however, the slope of the linear regression did not differ from zero. For ΔAT similar results were obtained. In nonobese boys. ΔAT (L $\times min^{-1}$) = 0.017 $\times$ weight (kg) $-$ 0.141; $r = 0.77$, $p < 0.001$. For obese boys the slope of the linear regression did not differ from zero. Qualitatively similar results were obtained in obese and nonobese girls.

When $\Delta\dot{V}O_2$max and ΔAT were examined as a function of age (Fig. 3) or height, both obesity-independent variables, a very different picture emerged. Most boys and girls had values well within the normal range (Table), but six of the obese children had values for either $\Delta\dot{V}O_2$max or ΔAT that were more than 2 SD below the predicted values (i.e., <40%). There did appear to be differences in the pattern of increase of $\Delta\dot{V}O_2$max or ΔAT as a function of age between obese and nonobese subjects. In nonobese boys the relationships between age and $\Delta\dot{V}O_2$max or ΔAT were best described by two exponential equations:

$$\Delta\dot{V}O_2max(L \times min^{-1}) = 0.175 \times e^{0.161 \text{ age}}$$

$$\Delta AT(L \times min^{-1}) = 0.084 \times e^{0.148 \text{ age}}$$

The r values for these equations were significant at the $p < 0.001$ level. The obese boys showed no significant increase in either $\Delta\dot{V}O_2$max or ΔAT with age.

The relationship between $\dot{V}E$ and $\dot{V}CO_2$ in the obese children was, on average, well within the normal range.[12] When height for predicted value was studied, the obese children's slopes were 111 ± 21% predicted (mean slope of 24 ± 1%). Similarly, the relationship between $\dot{V}O_2$ and work rate (calculated as $\Delta\dot{V}O_2/\Delta$WR) was 9.9 ml oxygen $\times$ min^{-1} $\times$ watt^{21} ± 2.3 in the obese children; this value was well within the normal range.[9]

Constant work rate protcols. The mean time of oxygen uptake in obese subjects (29 ± 9 sec) did not differ from normal control values (28 ± 6 sec) (Fig. 4). However, as shown in the Table, three of the obese subjects had significantly prolonged response kinetics. Obese children had, on
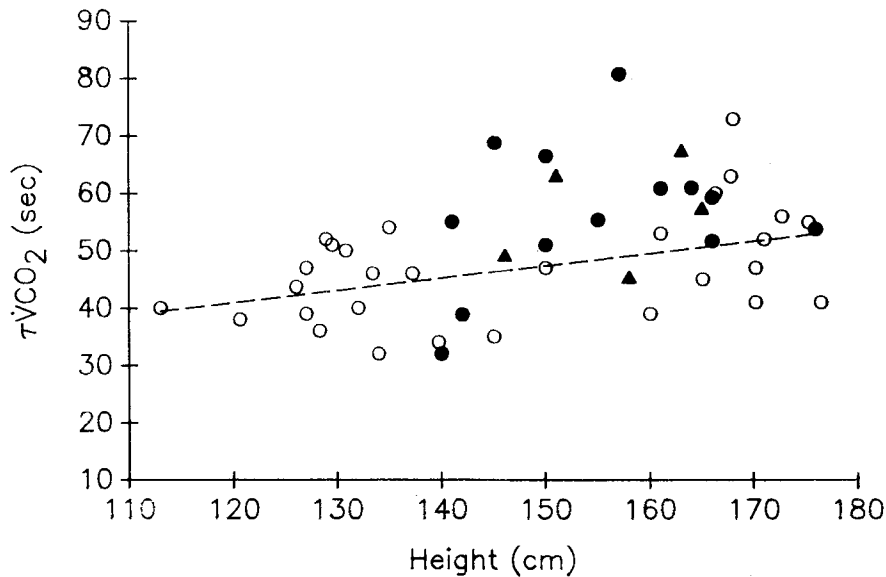
Fig. 4. Time constant for $\dot{V}CO_2$ ($\tau\dot{V}CO_2$) in obese boys *(black circles)*, obese girls *(black triangles)*, and nonobese children *(clear circles)*. Obese children had, on average, significantly longer $\tau\dot{V}CO_2$ than did nonobese subjects.

Table. Predicted results for a variety of cardiorespiratory responses to exercise in obese children

Subject No.	$\Delta\dot{V}E/\Delta\dot{V}CO_2$*	$\Delta\dot{V}O_2/\Delta WR$†	$\Delta\dot{V}O_2max$‡	ΔAT‡	$\tau\dot{V}O_2$†	$\tau\dot{V}CO_2$*	$\tau\dot{V}E$*
1	109	95	172	62	108	92	97
2	99	119	166	96	107	106	94
3	98	98	94	138	130	133	140
4	131	79	92	89	82	135	116
5	122	71	37	39	113	113	117
6	103	111	141	92	95	149	139
7	91	118	109	121	73	108	96
8	95	109	144	36	106	114	134
9	166	94	71	94	103	102	119
10	107	96	126	29	54	122	132
11	98	89	72	111	89	117	121
12	134	79	96	29	71	71	40
13	103	171	120	140	68	121	137
14	100	70	55	—	168	140	155
15	129	111	60	—	171	165	157
16	139	98	112	32	70	102	86
17	90	81	50	31	89	121	140
18	83	116	157	197	88	85	109
Mean	111	100	104	84	99	116	118
SD	21	24	41	51	32	23	29

*Predicted values based on height.[12]
†Size independent.[9]
‡Predicted values based on age from presented control subjects.

average, a prolonged response time for both $\dot{V}CO_2$ (Fig. 4) and $\dot{V}E$. Mean time of carbon dioxide output in the obese children was 57 ± 11 sec compared with 47 ± 9 sec ($p < 0.003$) in nonobese subjects, and mean minute ventilation time was 64 ± 16 sec compared with 50 ± 14 sec ($p < 0.005$) in nonobese subjects. Both $\tau\dot{V}E$ and $\tau\dot{V}CO_2$ are

known to increase with age in healthy children,[12, 15] so the predicted values for obese subjects were derived from regression analysis in which height was used as an adipose-independent measure of growth. The mean $\tau\dot{V}E$ in the obese subjects was 118% predicted, and the mean $\equiv\dot{V}CO_2$ was 116% predicted. End-tidal carbon dioxide partial pressure

in obese children was 39 ± 3 mm Hg at rest and 42 ± 3 mm Hg during steady-state constant work rate exercise. These values did not differ from values in healthy, nonobese children studied in our laboratory.[15]

DISCUSSION

Our data demonstrate that the finding of obesity in an otherwise healthy child is not a reliable indicator of abnormal fitness or impaired cardiorespiratory response to exercise. When appropriate corrections are made for body size, and when size-independent measures of cardiorespiratory response are assessed, most of the exercise responses in the 18 children studied were well within the normal range (Table). This is not to say, however, that the exercise response in obese children was indistinguishable from that of nonobese children. For example, the obese children did not increase $\Delta\dot{V}O_2$max or ΔAT with age to the same extent as nonobese children. Moveover, some of the obese subjects had markedly abnormal responses on both size-dependent and size-independent measures (Table). In fact, one subject, whose obesity was thought to be partly a manifestation of a difficult family situation, continued to gain weight despite intervention and subsequently died of respiratory failure.

The effect of excess body mass on cycle ergometer $\dot{V}O_2$ is complex. For example, a doubling of body weight did not result in a doubling of $\dot{V}O_2$ for unloaded cycling, as would have occurred if the work rate had been doubled. The scaling factor relating body mass to zero watt $\dot{V}O_2$ was not 1 but rather 0.51. The mechanics of cycle ergometry in obese children suggests that the increased $\dot{V}O_2$ results from frictional or perhaps inertial losses during pedaling, as well as from the greater resting metabolic requirement of an increased body mass. The effect of increased body weight on unloaded pedaling was the same in obese and nonobese subjects and appears to be qualitatively similar in adults and in children.[16] Thus a 6-year-old child who weighs as much as a nonobese 12-year-old child has virtually the same metabolic requirement as does the older child in moving his or her legs at low work rates. During progressive exercise, however, this similarity disappears, and the relationship between $\Delta\dot{V}O_2$max or ΔAT and body weight is markedly distorted in the obese children.

Thus the obese child begins exercise, such as pedaling on a cycle ergometer, with a significantly increased metabolic requirement. Our data show that obese children generally have adapted to their increased body weight so that they have the same ability to perform cycle ergometer work as do nonobese children. However, the data also suggest that the adaptation is more successful in younger subjects. As children grow older and perhaps the social stigma of obesity becomes more important, decreased activity and a con-

sequent slower growth of the ΔAT and $\Delta\dot{V}O_2$max become more evident, suggesting a relative "detraining" effect with age, particularly in obese subjects with a low ΔAT. Work done above a subject's anaerobic threshold cannot be sustained for long periods because lactic acid accumulates in the blood and both the ventilatory and buffering capabilities are challenged. We speculate that in the high-intensity exercise range (i.e., above the child's AT) an obese child has the greatest impairment in exercise adjustment. Finally, the concept of "fitness" must ultimately be related to the task at hand. For example, the effect of increased body weight on stair climbing is much greater than on bicycling.[17]

The obese children had the interesting findings of a normal $\tau\dot{V}O_2$ with a prolonged $\tau\dot{V}CO_2$. The temporal response of $\dot{V}O_2$ in the first several minutes of exercise is independent of size and age in children, teenagers, and adults for work rates below the subject's anaerobic threshold.[13] The response characteristics of $\dot{V}O_2$ reflect the initial increases in pulmonary blood flow (cardiac output) in response to the sudden increase in metabolic demand and are known to be abnormally slow in patients with cyanotic congenital heart disease or chronic respiratory disease.[18, 19] We have also shown that, although the dynamics of $\dot{V}E$ and $\dot{V}CO_2$ at the onset of exercise are size dependent, the size effect is far smaller than for the AT or $\dot{V}O_2$max.

The difference between $\tau\dot{V}CO$ and $\tau\dot{V}O_2$ can be used to represent the amount of carbon dioxide stored in the body.[15] The body stores very little oxygen, so at the onset of exercise $\dot{V}O_2$ measured at the mouth reflects events occurring at the exercising muscle tissue.[20, 21] In contrast, carbon dioxide produced by muscle cells is highly soluble in tissue and blood. As a consequence, the time course of the increase in carbon dioxide production detected at the mouth at the onset of exercise is prolonged relative to oxygen uptake even in nonobese subjects.[12, 15, 22] We speculate that increased storage of carbon dioxide in adipose tissue in close proximity to metabolically active muscle tissue may result in the prolonged $\tau\dot{V}CO_2$ observed in some obese children.

The dynamic response of $\dot{V}E$ is, characteristically, more directly related to carbon dioxide production than to oxygen consumption.[11] This phenomenon was observed in the obese children as the prolonged $\tau\dot{V}E$. The sluggish $\dot{V}E$ response despite normal $\dot{V}O_2$ response in some obese children may result in significant impairment in the adjustment to exercise; the arterial oxygen partial pressure falls if ventilation does not maintain normal alveolar partial pressure of oxygen. In some children this hypoxia might account for the sense of breathlessness at the beginning of exercise often reported by very obese subjects.

The clinical management of obese children has proved to be difficult. The idea that these children are usually unfit[7] has resulted in attempts to impose exercise as part of the

therapeutic regimen. Even when the results are good, the improvement associated with exercise is small and difficult to measure.[3] We believe that one reason for the unimpressive results is that many if not most obese children have normal fitness for their developmental stage. Imposing a structured program of physical activity on such subjects may reduce their spontaneous activity.

Our data also show that exercise testing may prove useful in identifying obese children in whom reduced activity or more serious cardiac or pulmonary impairment is not readily apparent. The finding of prolonged $\tau\dot{V}O_2$ and a low AT, for example, suggests an inability to increase cardiac output in a normal manner at the onset of exercise. Similarly, an elevated end-tidal partial pressure of carbon dioxide and abnormal slope of the $\dot{V}E\text{-}\dot{V}CO_2$ relationship during exercise could suggest pulmonary disease. Thus exercise testing can alert the clinician to children whose obesity may lead to serious cardiopulmonary morbidity and require more vigorous intervention.

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