Determination of anaerobic threshold for assessment of functional state in patients with chronic heart failure

NAOYA MATSUMURA, M.D., HIROTAKA NISHIJIMA, M.D., SHUNICHI KOJIMA, M.D., FUMINORI HASHIMOTO, M.D., MASARU MINAMI, M.D., AND HISAKAZU YASUDA, M.D.

ABSTRACT The use of anaerobic threshold in assessment of aerobic capacity was evaluated in 34 normal subjects and 47 patients with various kinds of chronic heart disease. Anaerobic threshold was determined as the oxygen consumption ($VO_2$) at which a linear relationship between pulmonary ventilation ($VE$) and $VO_2$ was lost during progressive treadmill exercise. Anaerobic threshold determined in this manner was validated with that determined by blood lactate measurements in eight normal subjects and nine cardiac patients ($r = .962, p < .001$). Thereafter, anaerobic threshold was determined only by respiratory measurements. In symptom-limited, maximal exercise, anaerobic threshold was reached well before maximal effort and corresponded to 70% of maximal $VO_2$ both in normal subjects and cardiac patients. Anaerobic threshold decreased as age progressed in normal subjects ($r = -.70, p < .001$). Anaerobic threshold in cardiac patients was lower than that in the normal subjects and decreased progressively as New York Heart Association functional classification advanced (normal, 32.95 ± 6.17 ml/min/kg; class I, 22.78 ± 3.74; class II, 16.99 ± 3.66; class III, 12.97 ± 2.76; $p < .01$ between each group other than between class II and class III). Anaerobic threshold in cardiac patients correlated poorly with other objective indices, e.g., cardiomegaly ($r = -.54, p < .001$) and rise in pulmonary wedge pressure ($r = -.64, p < .001$). At anaerobic threshold, cardiac patients subjectively graded the work load as light (13%), light-to-moderate (27%), moderate (30%), and moderate-to-heavy (28%). Thus determination of anaerobic threshold by respiratory measurements is a safe, accurate, and objective method to measure aerobic capacity in cardiac patients and in normal subjects.


EXERCISE TESTING has been widely used to evoke myocardial ischemia for diagnostic and follow-up evaluations in patients with effort angina. Recently, functional assessment of the impairment of cardiac reserve in patients with various cardiac lesions has also been performed by exercise testing.

Although "maximal exercise testing" is considered to be the best objective method for this purpose, it includes a certain subjective bias because the end point can be determined by the patient. Furthermore, the maximal exercise test may be impractical in the severely diseased patient.

Determination of anaerobic threshold, which is the workload associated with the onset of anaerobic metabolism during exercise, has been proposed as an objective means to assess aerobic capacity. However, its clinical significance in cardiac patients remains obscure.

The purpose of our study was to evaluate the accuracy of anaerobic threshold determined noninvasively by respiratory measurements as an assessment of aerobic capacity of cardiac patients and to clarify its value.

Materials and methods

Subjects. Forty-seven patients with chronic heart disease were studied. The etiology of the cardiac lesion was rheumatic valvular heart disease in 28, ischemic heart disease in two, idiopathic cardiomyopathy in 11, and congenital heart disease in six.

The orthodox functional classification according to the criteria of New York Heart Association (NYHA) was determined before exercise testing by physicians familiar with the medical histories of the patients. Eleven patients were judged as having class I disease, 23 as having class II disease, and 13 as having
class III disease. None had symptoms of congestive heart failure at rest.

Thirty-four normal subjects were recruited from a medical screening clinic and were determined to be free of any significant disease on the basis of history, physical examination, chest x-ray, 12-lead electrocardiogram (ECG) recording, and biochemical profile. The mean age of the normal subjects was 38 years (range 19 to 59) and that of the cardiac patients was 42 years (range 23 to 66).

**Exercise test.** Each subject underwent progressive treadmill exercise. Normal subjects performed a standardized multistage test according to the protocol of Bruce et al. However, cardiac patients performed a less intense, slowly progressive test that had been designed and utilized by us for patients with reduced cardiac reserve (the modified protocol).9 Treadmill speed, grade, and energy requirement of each stage were as follows: stage 0, 1.1 mph, 0%, 2.5 mets; stage 1, 1.1 mph, 10%, 3.5 mets; stage 2, 1.6 mph, 12%, 4.8 mets; stage 3, 2.2 mph, 14%, 6.3 mets; stage 4, 2.8 mph, 16%, 9 mets; stage 5, 3.2 mph, 18%, 11 mets; stage 6, 3.6 mph, 20%, 13 mets. Each stage was 3 min in duration.

The cardiac patients exercised at least two times before the maximal exercise test. Subjects were encouraged to exercise until they felt unable to continue, and this was reconfirmed by questioning after the tests. Symptom-limited maximal exercise was reached only when the subject indicated that he had achieved maximal exercise. Because of the face mask, clear verbal communication was difficult during exercise testing. Each subject was therefore asked to indicate with his fingers the grade of the workload as follows: 1, light; 2, light-to-moderate; 3, moderate; 4, moderate-to-intense; 5, intense and almost intolerable (subjective grade). When the subject thought the workload intolerable, he was asked to raise and wave the hand for the doctors attending to discontinue the exercise test.

**Measurement.** Each subject sat on a chair and wore the respirometer mask. After 10 min the resting values were obtained and the exercise was begun. Heart rate and arrhythmia were monitored from the ECG, and blood pressure was measured by the cuff method at 1 min intervals during the test.

Pulmonary minute ventilation (VE) and oxygen consumption per minute (VO₂) were measured by OXYLOG10 (see OXYLOG instruction book, P. K. Morgan, 1978). This apparatus consists of a face mask with appropriate valves, which is fitted with a turbine flow meter (at the air inlet) and an expired-air hose. Expired air passes through a specially designed mixing and bypass unit from which a miniature piston pump draws a sample for analysis in a polarographic oxygen sensor. A second sensor measures the partial pressure of oxygen (PO₂) in ambient air. The PO₂ difference between the inspired and expired air is measured in the instrument, and the volume of oxygen extracted from the air is calculated and displayed every minute as a minute volume (a total VO₂ of 1 min period). The basic formula is VO₂ (standard temperature and pressure, dry STPD) = (PO₂ inspired air – PO₂ expired air) × inspired air volume/760. This apparatus does not measure the amount of CO₂ produced. A respiratory quotient (RQ) of 1 is assumed. The error can be calculated from the basic formula and the equation VE = VI – (1 – RQ) VO₂. Percent error is calculated as (measured – true) × 100/true equals – PEO₂ (1 – RQ) × 100/P, where VI = inspired air volume, PEO₂ = expired air PO₂, and P = total atmospheric pressure. The percent error is within 5% at RQ 0.7 to 1.1 in the physiologic range of PEO₂ during exercise. The apparatus was calibrated by means of a standard method with a Tissot respirometer for minute air volume and the micro Schohlander method for oxygen and carbon dioxide determination. The correlation coefficient was .990 for VO₂ and .996 for VE.

The calibration by the standard method was done periodically. Calibration for the oxygen sensor using room air was done each time before and after the individual measurement. The normal values of VO₂ during standard treadmill exercise by Bruce’s protocol were determined in five healthy sedentary men. The regression equation was 
\[ y = 3.236x + 6.453 \]
where \( y = \text{VO}_2 \text{ml/kg} \) and \( x = \text{exercise duration in minutes} \). This was in close agreement with the results of Bruce et al.11: 
\[ y = 3.288x + 4.07 \]

Response time of the whole system was determined in the following way in seven normal subjects. First, all subjects underwent two 6 min exercise sessions by the Bruce protocol; stages I and III and the steady state in VO₂ were confirmed after 3 min. On a separate day, subjects underwent the same amount of exercise except that this time they exercised for the first 3 min with a mask on but without the connecting tube to the sensor box (OXYLOG); for the next 3 min (4 to 6 min) the tube was connected and VO₂ was measured. The 6 min value was taken as 100%, and the percentage attainment of the 4 min and 5 min values was calculated. For Bruce stage I, the 4 min and 5 min values were 90.6% and 100%, respectively; for Bruce stage III these values were 92.7% (p < .01 vs 6 min) and 100.4%, respectively. VE was expressed in body temperature and pressure, saturated with water vapor (BTPS) and VO₂ in STPD.

In addition, as a biological check for OXYLOG, one of the authors who has been healthy with stable body weight and who is accustomed to treadmill walking, underwent the same amount of graded treadmill exercise by the Bruce protocol a total of six times in the 2 years during which the current study was done. The coefficients of variation for Bruce stages I, II, III, IV, and V (3 min value) were 2.4%, 3.2%, 4.9%, 3.7%, 2.4%, respectively.

Venous blood samples were drawn from the forearm at rest and within the last minute of each stage of the exercise. Blood lactate was measured by an electrochemical enzymatic method12 with the Lactate Analyzer 640 (Roche Bioelectronics).

**Determination of anaerobic threshold.** Anaerobic threshold was defined as the VO₂ during exercise at which the blood lactate level began to rise (anaerobic threshold by lactate measurements). Anaerobic threshold was also determined by respiratory measurements as the VO₂ at which a linear relationship of VE–VO₂ was lost as the work load increased (anaerobic threshold by respiratory measurements). Actual determination of anaerobic threshold was made by visual inspection after the data of VE/VO₂ or lactate/VO₂ were plotted on an x-y axis by the computer. The point of departure from linearity (the first intersection point) was taken as anaerobic threshold. To circumvent the somewhat subjective nature of the inspection method, an independent objective method was evaluated in 10 normal subjects and nine cardiac patients in whom simultaneous lactate and respiratory measurements were done. Anaerobic threshold was determined objectively in the following way. First, from the peak VO₂ 50% point of VO₂ was determined. A regression equation was calculated from data points up to this point and the 95% confidence limit was calculated. The first value above this limit (beyond 50% peak VO₂) was then found. A midpoint between this value and the one just before it was taken as anaerobic threshold. There was a 1 min interval between data points for respiratory anaerobic threshold excluding the rest value, and a 3 min interval for lactate anaerobic threshold including the rest value. Departure from the regression line, instead of a fixed value of lactate, was used for lactate anaerobic threshold determination, since a gradual rise in lactate was seen in some normal subjects and cardiac patients soon after exercise (well before 50% of peak VO₂ and mostly at the lactate level below 2 mM/l lactate) before the sharp departure from the linearity beyond the 50% point in the symptom-limited maximal
exercise. This objective method was used as a check thereafter. All the anaerobic threshold values reported in this investigation after the basic methodologic assessment were obtained by visual inspection. An example of the simultaneous determination of anaerobic threshold by both methods is shown in figure 1. Thereafter, anaerobic threshold was determined only by respiratory measurements.

**Statistical analysis.** Overall comparisons were assessed with one-way analysis of variance, followed by pairwise modified t tests with Bonferroni’s procedure. Overall differences of determination for anaerobic threshold between methods and observers were evaluated by repeated measurement analysis of variance. Regression analyses were also performed by standard methods to calculate correlation coefficient, slope, and intercept if appropriate. Differences were considered to be significant at p < .05. Values are expressed as mean ± SD.

**Results**

**Methodologic assessment in determination of anaerobic threshold.** This was evaluated in 10 normal subjects and nine cardiac patients in whom simultaneous lactate and respiratory measurements were performed during exercise. Two investigators (observers 1 and 2) independently determined the anaerobic threshold of each patient from respiratory and lactate measurements. Among respiratory plots of 10 patients, two were deleted because one or both investigators judged that the anaerobic threshold point could not be determined by inspection, although anaerobic threshold determina-

![Figure 1](http://circ.ahajournals.org/)

**FIGURE 1.** An example of the simultaneous determination of anaerobic threshold (AT) both by blood lactate and respiratory measurements in a cardiac patient. Dashed line, the linear extension of the relationship between pulmonary ventilation and oxygen consumption.

**TABLE 1**

<table>
<thead>
<tr>
<th></th>
<th>Resp. 1</th>
<th>Resp. 2</th>
<th>Resp. obj</th>
<th>Lactate 1</th>
<th>Lactate 2</th>
</tr>
</thead>
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<td>.956</td>
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<td></td>
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<tr>
<td>Lactate obj</td>
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<td>.958</td>
<td>.944</td>
<td>.968</td>
<td>.983</td>
</tr>
</tbody>
</table>

Resp. = anaerobic threshold determined by respiratory measurements; Lactate = anaerobic threshold determined by lactate measurements; 1, 2 = visual inspections by observer 1 or 2; obj. = objective method.

*Determined by visual inspection (two observers) and by the objective method from respiratory and lactate measurements. All correlations are statistically significant.

The following variables were considered in the analysis: (1) intraobserver variation, (2) interobserver variation (observer 1 vs 2), (3) comparison between the visual inspection and objective determination (observer 1 vs 2), and (4) comparison between anaerobic threshold values determined by the respiratory and lactate methods (observer 1 vs 2).

One of the observers (observer 1) determined AT twice from both respiratory and lactate measurements on separate days. The correlation coefficient between the two determinations was .989 from respiratory measurements and .959 from lactate measurements.

There was no overall statistical difference among the different procedures to determine anaerobic threshold (table 1). A high degree of correlation was obtained in most of the pairs. Correlation coefficients tended to be greater in cardiac patients than in normal subjects when the comparison was made in each category. Figure 2 shows the correlation between anaerobic threshold values determined by the lactate respiratory measurement (visual inspection). Both methods correlated well (r = .962, p < .001). Therefore the respiratory measurements were able to correctly predict the onset of anaerobic metabolism during exercise.

**Reproducibility of anaerobic threshold.** Seven normal subjects and eight cardiac patients underwent exercise tests within a week. The mean difference of anaerobic threshold between the two tests, which was calculated as (the first anaerobic threshold minus the second anaerobic threshold)/the first anaerobic threshold × 100%, was −1.6 ± 6.6% for the normal subjects and 3.6 ± 10.5% for the cardiac patients (statistically not significant).

**Influence of age on anaerobic threshold in normal subjects.** Figure 3 illustrates the relationship between age.
Anaerobic threshold determination in cardiac patients

Exercise test. Age, body weight, and resting cardiopulmonary values (sitting) for the four groups are shown in Table 2. Thirty-eight cardiac patients and 26 normal subjects accomplished their maximal work (subjective) at the exercise test. The limiting symptoms were breathlessness or leg dullness and fatigue in most of the patients. Chest discomfort, which was not typically anginal in nature, was the limiting symptom in only five patients. Table 3 shows the values at the peak exercise in the subjects who attained their maximal effort. Heart rate in cardiac patients was represented only in those with sinus rhythm. Peak oxygen consumption and heart rate decreased as NYHA class advanced. Systolic blood pressure did not differ significantly between groups.

Anaerobic threshold in cardiac patients. Anaerobic threshold was determined by respiratory measurements in each subject. The mean value of anaerobic threshold was 32.95 ± 6.17 ml/min/kg in normal subjects and 17.22 ± 4.86 ml/min/kg in cardiac patients. Table 4 shows the value of anaerobic threshold in normal subjects and cardiac patients graded by NYHA classification. Pulmonary ventilation, ventilatory equivalent (VE/VO2), systolic blood pressure, heart rate, and subjective grade at the time corresponding to anaerobic threshold are also shown.

Figure 4 illustrates the relationship between anaerobic threshold and NYHA functional classification. Significant decrease was noted in accordance with the severity of the disease as determined by NYHA classification.

Both systolic blood pressure and heart rate at the time corresponding to anaerobic threshold were significantly lower in cardiac patients. Subjective grade for the workload corresponding to anaerobic threshold was "moderate" (grade 3) in most normal subjects but varied in cardiac patients. Thirteen percent of cardiac patients graded it as 1, 27% as 2, 30% as 3, 28% as 4, and only one patient graded it as 5. There was gradual decrease in the subjective grade for anaerobic threshold as functional class became advanced. Blood lactate at anaerobic threshold was 1.46 ± 0.44 mM/l in normal subjects and 1.49 ± 0.49 mM/l in cardiac patients.

Peak oxygen consumption and relative anaerobic threshold.

Figure 5 shows the relationship between peak oxygen consumption and anaerobic threshold in subjects who attained maximal effort (r = .946, p < .001). The ratio of anaerobic threshold to peak oxygen consumption (relative anaerobic threshold) was constant in this group of subjects, although the subjective grade of the workload at anaerobic threshold differed among subjects. Relative anaerobic threshold was 68.4 ± 7.2%, 70.3 ± 9.7%, 70.4 ± 8.2%, and 71.0 ± 8.8% in normal subjects and cardiac patients of NYHA class I, II, and III, respectively. No significant differences were observed between groups.

Maximal blood lactate concentration attained in exercise was 5.8 ± 2.0 mM/l in normal subjects and 4.6 ± 2.4 mM/l in cardiac patients.
TABLE 2
Age, body weight, and resting cardiorespiratory values (mean ± SD)

<table>
<thead>
<tr>
<th>Group</th>
<th>Age (yr)</th>
<th>Body weight (kg)</th>
<th>SBP (mm Hg)</th>
<th>HR (bpm)</th>
<th>VO₂ (ml/min/kg)</th>
<th>VE (× 10 ml/min/kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>38 ± 11</td>
<td>63.7 ± 7.1</td>
<td>110.0 ± 9.7</td>
<td>69.2 ± 11.2</td>
<td>4.56 ± 0.64</td>
<td>13.9 ± 3.2</td>
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<tr>
<td>(n = 34)</td>
<td></td>
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<tr>
<td>NYHA I</td>
<td>34 ± 10</td>
<td>56.0 ± 8.2</td>
<td>108.2 ± 9.6</td>
<td>79.7 ± 11.7</td>
<td>4.68 ± 0.94</td>
<td>16.3 ± 4.9</td>
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<td>(n = 11)</td>
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<tr>
<td>NYHA II</td>
<td>45 ± 13</td>
<td>58.0 ± 7.7</td>
<td>115.4 ± 17.9</td>
<td>81.4 ± 16.1</td>
<td>4.18 ± 0.68</td>
<td>14.4 ± 3.1</td>
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<tr>
<td>(n = 23)</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NYHA III</td>
<td>44 ± 11</td>
<td>52.1 ± 10.1</td>
<td>108.6 ± 16.9</td>
<td>78.8 ± 14.1</td>
<td>4.27 ± 0.74</td>
<td>16.3 ± 3.9</td>
</tr>
<tr>
<td>(n = 13)</td>
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</tbody>
</table>

SBP = systolic blood pressure; HR = heart rate.

No significant difference in relative anaerobic threshold was observed in cardiac patients when they were subgrouped by their limiting symptoms (68.3% in patients with breathlessness and 71.6% in those with leg dullness or fatigue).

Relationship of anaerobic threshold to cardiomegaly and resting hemodynamics. Chest plane film was obtained within 2 weeks of the exercise testing in 34 patients. A significant negative correlation was observed between cardiomegaly and anaerobic threshold (r = −.536, p < .001). In 21 patients who underwent diagnostic right heart catheterization within 2 weeks of the exercise testing, a negative correlation was also observed between pulmonary capillary wedge pressure and anaerobic threshold (n = 21, r = −.642, p < .001).

Discussion

The results of our study indicate that it is possible to quantify the exercise capacity and the functional state by the determination of anaerobic threshold, which is independent of the subject's motivation, in normal subjects and cardiac patients.

Despite revision in 1973, the older NYHA functional classification is still used to grade the functional state of cardiac patients in the clinical studies because of its simplicity and familiarity. However, its subjective nature and the possibility for physician's bias can make this system unreliable. Goldman et al. noted the serious defects of the system and pointed out its inadequacy to evaluate a patient's response to therapy or to compare one patient with another. Few studies were performed regarding its correlation with the objective method to evaluate functional capacity, i.e., the symptom-limited maximal exercise test. However, even this could be subjective because the end point is determined by the patient. The motivation may differ from patient to patient, and some patients may discontinue the exercise test well before the true maximal point.

It has been known that the anaerobic metabolism occurs during progressive exercise when the oxygen delivery to the working muscles cannot increase enough to maintain aerobic metabolism. This onset of anaerobic metabolism or anaerobic threshold can be detected noninvasively by expired gas measurements. Wasserman et al. proposed four ways to detect anaerobic threshold: (1) nonlinear increase in pulmonary ventilation, (2) nonlinear increase in carbon dioxide production compared with the increase in workload, (3) increase in end-tidal oxygen fraction without a decrease in that of carbon dioxide, and (4) increase in gas exchange ratio. They found that cardiac patients have lower anaerobic threshold with the gas exchange ratio. However, this method of determining anaerobic

TABLE 3
Results of the maximal treadmill exercise in normal subjects and cardiac patients (mean ± SD)

<table>
<thead>
<tr>
<th>Group</th>
<th>VO₂ (ml/min/kg)</th>
<th>VE (× 10 ml/min/kg)</th>
<th>SBP (mm Hg)</th>
<th>HR (bpm)</th>
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<tbody>
<tr>
<td>Normal (n = 26)</td>
<td>45.17 ± 7.44</td>
<td>108.1 ± 29.1</td>
<td>176.0 ± 18.4</td>
<td>173.1 ± 16.5</td>
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<tr>
<td>NYHA I (n = 10)</td>
<td>33.18 ± 6.50</td>
<td>81.4 ± 20.6</td>
<td>170.0 ± 28.3</td>
<td>157.8 ± 16.3A</td>
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<td>NYHA II (n = 19)</td>
<td>24.43 ± 4.47</td>
<td>67.8 ± 17.2</td>
<td>157.3 ± 21.2</td>
<td>140.6 ± 24.6A</td>
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<tr>
<td>NYHA III (n = 9)</td>
<td>18.86 ± 4.88</td>
<td>60.5 ± 17.9</td>
<td>156.3 ± 36.1</td>
<td>124.6 ± 19.4A</td>
</tr>
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</table>

SBP = systolic blood pressure; HR = heart rate.

Statistical comparisons: A p < .05 vs normal subjects; B p < .05 NYHA I vs NYHA II or III.

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TABLE 4

<table>
<thead>
<tr>
<th>Group</th>
<th>AT (VO₂ ml/min/kg)</th>
<th>VE (VE/VO₂)</th>
<th>VE/VO₂</th>
<th>SBP (mm Hg)</th>
<th>HR (bpm)</th>
<th>Subjective grade (1 to 5)</th>
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<tbody>
<tr>
<td>Normal (n = 34)</td>
<td>32.95 ± 6.17</td>
<td>64.9 ± 15.2</td>
<td>19.7 ± 2.9</td>
<td>157.4 ± 16.5</td>
<td>134.1 ± 18.1</td>
<td>2.94 ± 0.83</td>
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<td>NYHA I (n = 11)</td>
<td>22.78 ± 3.74*</td>
<td>51.2 ± 9.9*</td>
<td>22.6 ± 2.1</td>
<td>142.2 ± 17.7</td>
<td>126.8 ± 19.3</td>
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<tr>
<td>NYHA II (n = 23)</td>
<td>16.99 ± 3.66*</td>
<td>41.9 ± 12.3*</td>
<td>24.9 ± 6.7*</td>
<td>144.9 ± 21.7</td>
<td>114.7 ± 16.4</td>
<td>2.70 ± 1.04</td>
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<tr>
<td>NYHA III (n = 13)</td>
<td>12.97 ± 2.76*</td>
<td>37.8 ± 8.2*</td>
<td>29.7 ± 6.6*</td>
<td>131.4 ± 27.2</td>
<td>106.3 ± 22.5</td>
<td>2.35 ± 1.03*</td>
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</table>

AT = anaerobic threshold; VE/VO₂ = ventilatory equivalent; SBP = systolic blood pressure; HR = heart rate.
Statistical comparisons: *p < .05 vs normal subjects; *p < .05 NYHA I vs NYHA II or III.

The anaerobic threshold was found to be rather insensitive in classifying the patients with cardiac conditions of different severities. Moreover, this method was not widely applied in clinical practice. Later, Wasserman et al. suggested that one of the other three methods would be better.

Our findings confirm the previous observation that anaerobic threshold was decreased according to the severity of the heart disease. In addition, our results clearly demonstrate the major difference in anaerobic threshold between sedentary normal subjects and cardiac patients with a wide range of disease severity, since this is the first report in which a relatively large number of normal subjects (ages 20 to 60 years) were studied with the cardiac patients in the same laboratory. The mean anaerobic threshold of our normal subjects in their 20s, 37.81 ml/kg/min, compares favorably with that reported by Davis et al. in 32.84 ml/kg/min (mean age 21). The somewhat higher value in our report probably reflects the major difference in the mean weight (Davis, 74.0 kg; 67.2 kg in this report). It is well known that maximal VO₂/kg is lower in patients with heavier body weight or obesity. Comparison in the other age range in the sedentary subjects without obesity is not possible because of the lack of sufficient published data. The mean anaerobic threshold values obtained in the report by Weber et al. in which functional class was determined on the basis of the maximal VO₂ (Weber functional class A, 20 or more; B, 16 to 20; and C, 10 to 15 VO₂ ml/kg/min) were 17, 12.7,
and 10.4 ml/kg/min, respectively, for each class. Our results, analyzed in a similar way, yielded the following values of the mean anaerobic threshold for each class: A, 19; B, 14; and C, 11 ml/kg/min. There is close agreement between the two sets of values. These results merely emphasize the universal value of the use of anaerobic threshold in assessing aerobic capacity.

The ratio of anaerobic threshold to peak oxygen consumption, referred to as relative anaerobic threshold, was approximately 70% in the cardiac patients. This also agrees well with the results of Weber et al.\(^6\) However, the mean value of 70% was also obtained in the normal subjects in our report. This is rather high compared with the values of 50% to 60% reported by Wasserman et al.\(^2,3\) This is probably due to the underestimated peak \(\dot{V}O_2\) in our normal subjects and to the fact that exercise testing was done by the continuous protocol (3 min per stage) in our study as symptom-limited exercise. Difficulty in reaching the oxygen plateau in this protocol is well known.\(^9\) Very few of our patients reached the definite \(\dot{V}O_2\) plateau.

Since true maximal \(\dot{V}O_2\) (plateau \(\dot{V}O_2\)) is difficult and probably dangerous to obtain in cardiac patients (and therefore not enough data are available in which both true maximal \(\dot{V}O_2\) and anaerobic threshold are measured in cardiac patients), we are not completely certain whether relative anaerobic threshold is the same in both normal subjects and cardiac patients. Nevertheless, currently available data suggest that the relative anaerobic threshold values may be relatively close.

The point at which a linear relationship of \(\dot{V}O_2/VE\) is lost in progressive exercise (anaerobic threshold) was easily discernible in both normal subjects and cardiac patients in plots of these values on the horizontal and vertical axes. Intraobserver and interobserver variation were sufficiently small by visual inspection in normal subjects and cardiac patients. Generally, departure from linearity was sharper for the cardiac patients. Correlation between anaerobic threshold values determined by the lactate and the respiratory measurements was also satisfactory. The objective method could be used as check. When there were wide discrepancies in the anaerobic threshold point between the two, a scatter of the data was usually observed, probably due to technical problems. In this case, we believe the test should be repeated.

The values of anaerobic threshold for normal subjects showed an age-related decrease. This is an expected finding, since maximal \(\dot{V}O_2\), maximal heart rate, and other parameters of aerobic capacity are known to decrease as age advances. This therefore seems to be an indirect validation of anaerobic threshold as a physiologic index of exercise capacity.

The decrease in anaerobic threshold in cardiac patients is probably caused by the earlier onset of anaerobic metabolism due to insufficient blood supply for working muscles, which is a net result of reduced cardiovascular reserve, reduction in level of physical fitness, and/or deconditioning. A combination of these factors may also lead to the relative predominance of fast-switch muscle fibers in cardiac patients, contributing to onset of anaerobic metabolism.\(^20\)

Although there is a definite general trend that the decrease in anaerobic threshold parallels the advancing NYHA classification, the scatter of anaerobic threshold values in any given class is large. Therefore it is clear that the accurate aerobic functional capacity could not be accurately predicted from this subjective index (NYHA). For this reason the exercise test must be performed and objective indices such as anaerobic threshold must be obtained in each patient.

The routine measurement of respiration during exercise testing may still be cumbersome and complicated. However, recent technologic advances have made continuous measurement during exercise relatively easy, obviating the need for the classic Douglas-bag method, which is time consuming. Therefore, in selected patients in whom objective determination of exercise capacity is desirable, addition of respiratory measurement to the routine exercise testing will be amply rewarded.

In conclusion, anaerobic threshold is an accurate and objective index to quantify the severity of an impairment of aerobic capacity in cardiac patients. It corresponded to approximately 70% of the peak oxygen consumption in the symptom-limited maximal exercise. Its detection is simple and noninvasive by respiratory measurements well before the maximal effort.

References

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