Cardiopulmonary Stress Testing
A Review of Noninvasive Approaches

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With readily available techniques, cardiopulmonary exercise testing permits noninvasive measurement of such parameters as heart rate, cardiac output, oxygen saturation, ventilation, and gas exchange to bring out abnormalities which are either underestimated or not detectable at rest. These parameters may be used to characterize a patient's primary limitation of exercise tolerance as either cardiac or pulmonary in origin. They can also provide precise data to assess response to treatment. Pulmonary gas exchange is evaluated primarily by measurement of oxygen consumption, carbon dioxide production, and ventilation over time. The relationship of these parameters to one another changes throughout the course of incremental exercise testing. By appreciating these basic relationships, the more complex abnormalities found in disease states can be understood.

Cardiac and pulmonary disease frequently coexist in the dyspneic patient. However, evaluation of these patients may frustrate physicians who are faced with vague clinical clues and laboratory tests which fail to delineate the cause of the exercise limitation. In this situation, cardiopulmonary stress testing has two functions, to characterize the exercise limitation and to assess response to treatment. Abnormalities which were underestimated or not clinically appreciated may be demonstrated by monitoring the response to exercise of such parameters as heart rate, cardiac output, ventilation, and gas exchange. These parameters are used to characterize the limitation as primarily cardiac, pulmonary or both. Furthermore, repeat testing provides the physician with quantitative data for assessing effectiveness of therapy. Repeat exercise evaluation may be particularly valuable in combined cardiopulmonary disorders since the response to treatment of one disease may unmask exercise-limiting symptoms from another. For example, treatment of moderately severe COPD may allow exercise tolerance to increase until limitation by chest pain from coronary artery disease occurs. Conversely, progression of lung disease in a patient who also has angina may cause exercise to cease from ventilatory limits before chest pain from myocardial ischemia occurs.

In recent years, the availability of computerized laboratory equipment has made measurement of pulmonary gas exchange during exercise available to most cardiologists and pulmonologists. Similarly, the development of radionuclide computer-assisted cardiac imaging and echocardiography has made widespread use of noninvasive cardiac function studies possible. The combined use of these techniques offers the clinician a powerful tool for the noninvasive evaluation of patients with exercise limitation. In order to interpret the results of exercise testing in combined cardiac and pulmonary disease, one must understand the characteristic responses seen with relatively pure disease of each system. The purpose of this review is to present an overview of the practical aspects of noninvasive incremental exercise testing in cardiac and pulmonary disease.

Measurement of Pulmonary Gas Exchange

Measurement of pulmonary gas exchange provides data points which, when analyzed in concert, allow characterization of the physiologic response to exercise. The primary measurements are the rate of oxygen consumption (\(\text{VO}_2\)), the minute ventilation (\(\text{Ve}\)), and the rate of carbon dioxide production (\(\text{VCO}_2\)). These values are compared to the predicted normal values, and related to one another to produce the characteristic patterns found in normal and diseased states. All three measurements are used to determine the anaerobic threshold (AT) which is important in evaluating the cardiovascular response to incremental exercise.

Oxygen Consumption (\(\text{VO}_2\))

As the body begins exercise, the muscles require increasing amounts of oxygen to do work. The delivery of oxygen depends upon increasing cardiac output and vasodilatation to enhance perfusion of the working muscles by oxygenated blood. In a progressive maximal exercise test the \(\text{VO}_2\) linearly parallels the increase in cardiac output and work performed until a plateau is reached at the maximal cardiac output.\(^{15}\) In normal individuals, the limits of exercise are determined by the cardiovascular system's ability to continue to provide oxygen to the tissues, since the pulmonary

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vascular bed expands and ventilation increases to maintain adequate gas exchange even under strenuous
exercise. Therefore, measurement of the \( \dot{V}_{O_2} \) gives a rough estimate of the power output of the exercising cardiovascular system and is used to assess its performance. The \( \dot{V}_{O_2} \) may be expressed in L/min or related to the lean body weight, ml/min/kg. The maximum oxygen uptake (\( \dot{V}_{O_2}\)max), or aerobic capacity, occurs when increasing the work rate results in no further increase in oxygen consumption.\(^4\) An acceptable definition is an increase of less than 1 ml/min/kg for 60 seconds over the previous load.\(^5\) If exercise stops prior to reaching maximal cardiac output, \( \dot{V}_{O_2}\)max is not reached, but the term is sometimes confusingly applied to the highest achieved \( \dot{V}_{O_2} \). In this article, the term \( \dot{V}_{O_2}\)max will apply to the definition given here, and other references are to the maximal attained \( \dot{V}_{O_2} \).

Estimations of the \( \dot{V}_{O_2}\)max based on treadmill time or ergometer workload may be subject to error in patients with disease, so measurement of this value is preferable.\(^6,7\) Normal subjects are expected to exceed 90 percent of the predicted \( \dot{V}_{O_2}\)max for their age.\(^8\) A decrease in the maximal attained \( \dot{V}_{O_2} \) from predicted will be found in significant exercise limitation of any cause. However, assessment of the maximal attained \( \dot{V}_{O_2} \) in relationship to the minute ventilation (\( V_{E}\)max), heart rate, \( CO_2 \) production and body weight can help define the limitation as predominantly cardiovascular or pulmonary in origin.

As stated above, the \( \dot{V}_{O_2}\)max is related to the limits reached by the cardiac output. Expressed mathematically, the cardiac output is defined by the Fick equation as:

\[ Q = \dot{V}_{O_2}/(CaO_2 - CvO_2) \]

where \( Q \) = cardiac output, and \( (CaO_2 - CvO_2) \) = arterial and mixed venous oxygen content difference. Cardiac output is also represented as the product of the stroke volume (SV) and the heart rate (f):

\[ Q = SV \times f \]

We can combine these formulas to yield:

\[ \dot{V}_{O_2}/f = SV (CaO_2 - CvO_2) \]

Since the \( (CaO_2 - CvO_2) \) remains fairly constant, \( \dot{V}_{O_2}/f \) is approximately proportional to the stroke volume during exercise.\(^1\) This value is known as the \( O_2 \) pulse. It actually indicates the amount of oxygen consumed per heart beat and is expressed as ml/beat. The normal predicted values are obtained by dividing the predicted maximal \( \dot{V}_{O_2} \) by the predicted maximal heart rate.\(^1\)

Most cardiac disorders limiting exercise cause a decreased \( O_2 \) pulse, since the SV is often low and cardiac output is maintained by an increase in heart rate. However, any condition that causes exercise to cease at a low \( \dot{V}_{O_2} \) with a higher than predicted heart rate will produce a low \( O_2 \) pulse. Similarly, the heart rate for a particular \( \dot{V}_{O_2} \) may be low due to disease, pacemaker, or medications such as beta blockers, and produce a falsely normal \( O_2 \) pulse. Therefore, it is best to examine the value of the \( \dot{V}_{O_2} \) before the \( O_2 \) pulse.

Many patients with mild coronary artery disease (CAD) have a decreased maximum attained \( \dot{V}_{O_2} \) due to cessation of exercise because of chest pain without evidence of a decrease in cardiac output.\(^4\) Mild CAD and mild cardiac valvular diseases are associated with normal responses to exercise of \( \dot{V}_{O_2} \), cardiac output and SV.\(^1\) Advanced CAD will produce a more significantly decreased \( \dot{V}_{O_2}\)max but is indistinguishable from other causes of heart failure. Similarly, cardiovascular deconditioning may produce a low \( \dot{V}_{O_2} \) and \( O_2 \) pulse which cannot be distinguished from mild heart failure.\(^10\) Patients with significant cardiac dysfunction will cease exercise at a lower than predicted \( \dot{V}_{O_2}\)max due to a decreased cardiac output. The SV fails to increase normally due to decreased contractility of the myocardium while the cardiac output is initially maintained by an increase in heart rate.\(^2,3,11\) With more significant impairment of cardiac function, the maximum attained \( \dot{V}_{O_2} \) is further decreased and accompanied by evidence of increased muscle anaerobic metabolism due to the sustained low cardiac output at relatively low workloads. Hence, changes in function through time can be quantitatively assessed by repeat exercise testing which may be used to guide response to therapy or assess need for further intervention. In patients with known heart failure, Weber et al\(^\text{11}\) have shown that the \( \dot{V}_{O_2}\)max has been found to reliably classify the severity of the disease when combined with the measurement of the anaerobic threshold (Table 1).

The \( \dot{V}_{O_2} \) at maximal exercise in pulmonary disease varies widely, since the primary limitation to exercise is usually impaired ventilation or gas exchange. Although patients with mild COPD may have no significant decrease in \( \dot{V}_{O_2} \), patients with more severe disease will cease exercise at a low \( \dot{V}_{O_2} \). However, they will demonstrate other coexistent abnormalities such as an inappropriately high \( V_{E} \) or dead space to tidal volume ratio (\( V_{D}/V_{T} \)). Patients with severe COPD usually have

<table>
<thead>
<tr>
<th>Severity</th>
<th>Class</th>
<th>( \dot{V}_{O_2} ) max (ml/min/kg)</th>
<th>Anaerobic Threshold (ml/min/kg)</th>
<th>Maximum Cardiac Output (L/min/m²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>None to mild</td>
<td>A</td>
<td>&gt;20</td>
<td>&gt;14</td>
<td>&gt;8</td>
</tr>
<tr>
<td>Mild to moderate</td>
<td>B</td>
<td>16-20</td>
<td>11-14</td>
<td>6-8</td>
</tr>
<tr>
<td>Moderate to severe</td>
<td>C</td>
<td>10-15</td>
<td>8-11</td>
<td>4-6</td>
</tr>
<tr>
<td>Severe</td>
<td>D</td>
<td>6-9</td>
<td>5-8</td>
<td>2-4</td>
</tr>
<tr>
<td>Very severe</td>
<td>E</td>
<td>&lt;5</td>
<td>3-4</td>
<td>≤2</td>
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(from reference 5, with permission)
a normal cardiac output for the level of $\dot{V}O_2$ which differs from the patient with cardiac disease. However, the $O_2$ pulse is often low in COPD patients due to accelerated heart rates from medications, associated cardiac deconditioning, or right ventricular dysfunction.\textsuperscript{10,12,13} Similarly, patients with mild interstitial lung disease will have a normal $\dot{V}O_2$ and $O_2$ pulse, but as the severity of the disease progresses, exercise will cease at a lower $\dot{V}O_2$ due to ventilatory limitations or hypoxemia.\textsuperscript{13}

Comparing responses in cardiac and pulmonary disease, Nery et al\textsuperscript{14} found the oxygen pulse in patients with mitral valve disease (MVD) to be significantly lower than both normal subjects and patients with COPD. However, the aerobic capacity expressed as the $\dot{V}O_2/kg$ at maximal exercise was lower than normal for patients with COPD and MVD, but there was no significant difference between the two groups. This suggests that the $\dot{V}O_2$ alone cannot be used to distinguish between cardiac and pulmonary disease. This may be particularly important with coexistent COPD and coronary artery disease. In such cases, careful observation of the response to exercise of ventilatory mechanics, gas exchange and anaerobic threshold should help to delineate which one is causing the limitation. Examination of the maximal $\dot{V}O_2$ and $O_2$ pulse is not sufficient.

Figure 1 shows a graphic representation of pulmonary gas exchange data from three typical subjects studied in our laboratory. One subject is normal while the other two have severe COPD and CHF respectively. The $\dot{V}O_2$ is represented on the top row of graphs. The normal individual reached the predicted value of $\dot{V}O_2max$ for his age, while the highest attained $\dot{V}O_2$ for the subjects with cardiac and pulmonary disease was considerably lower. The second row of graphs in Figure 1 shows the $O_2$ pulse during the same studies. Again, the low $O_2$ pulse distinguishes the patients with disease from normal, but not from one another. The remaining data in Figure 1 will be discussed below.

![Figure 1](Downloaded From: http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21542/ on 06/24/2017)
Anaerobic Threshold

At some point during incremental exercise, the O₂ demand of exercising muscle exceeds oxygen delivery, and lactic acid begins to accumulate in the blood with the shift to anaerobic glycolysis by the muscle.\(^\text{15}\) The highest oxygen uptake that can be reached during exercise before an increase in blood lactate concentration occurs is termed the anaerobic threshold,\(^\text{3}\) and is expressed in relation to the maximal \(\text{VO}_2\). This point in incremental exercise testing can be indirectly estimated from pulmonary gas exchange measured at the mouth based on the concept that the increasing lactic acid in the blood causes an increase in \(\text{CO}_2\) and H\(^+\), which in turn stimulate ventilation.\(^\text{16}\) Before AT is reached, the \(\text{VE}\) increases proportionately to the increase in \(\text{VO}_2\), work rate, and \(\text{VCO}_2\). After AT, the slope of the \(\text{VE}\) increases, but remains proportional to the increasing \(\text{VCO}_2\) rather than the \(\text{VO}_2\). Therefore, the AT is detected when the ratio of \(\text{VE}/\text{VO}_2\) (the ventilatory equivalent for oxygen) and end-tidal partial pressure for oxygen (Pet\(\text{O}_2\), the partial pressure of oxygen measured at the mouth at the end of exhalation) begin to systematically increase without an immediate increase in the \(\text{VE}/\text{VCO}_2\) (the ventilatory equivalent for \(\text{CO}_2\)). This method has been found to correlate the best with blood lactate levels in normal individuals.\(^\text{17}\) The determination of AT by indirect means assumes the muscles have a normal ability to produce lactate and the minute ventilation responds appropriately to increases in \(\text{PCO}_2\) and H\(^+\).

There is a brief period immediately after AT when the end-tidal \(\text{PCO}_2\) and \(\text{VE}/\text{VCO}_2\) do not change significantly as the accumulating lactic acid is buffered in the blood. As work continues, lactic acid accumulates, the pH falls, the \(\text{VE}\) increases further due to added stimulation of the carotid bodies, and the \(\text{VE}/\text{VCO}_2\) increases. The period between the increase in the \(\text{VE}/\text{VO}_2\) and the increase in the \(\text{VE}/\text{VCO}_2\) is known as isocapnic buffering. However, it may not be seen easily. Conditions that also cause an abrupt increase in the \(\text{VE}\), such as hyperventilation, hypoxemia or anxiety, may produce changes in the ventilatory equivalents for oxygen and carbon dioxide which may be difficult to distinguish from reaching the AT. However, these conditions are usually associated with a simultaneous fall in the end-tidal \(\text{PCO}_2\) and increase \(\text{VE}/\text{VCO}_2\).\(^\text{18}\) In some instances, anaerobic threshold may be reached, but not be detected by measurement of pulmonary gas exchange.

To facilitate comparison of the AT between different studies, it is commonly expressed as the \(\text{VO}_2\) at AT in relation to the predicted \(\text{VO}_2\)max. In normal sedentary individuals the AT occurs at 50 to 60 percent of the \(\text{VO}_2\)max, with a low normal accepted at 40 percent for older individuals.\(^\text{16,18}\) While endurance-trained athletes may have the AT occur at levels as high as 70-80 percent \(\text{VO}_2\)max.\(^\text{19}\) The determination of the AT in relationship to the \(\text{VO}_2\)max significantly helps to characterize the exercise limitation when evaluating for dyspnea (Fig 2).

Patients who are able to attain a normal \(\text{VO}_2\)max usually reach AT at a normal level. One must keep in mind that this group may include some patients who also have mild cardiac or pulmonary disease. On the other hand, some patients who have exercise limitations with a low maximal attained \(\text{VO}_2\) may have a normal AT. These patients are most likely to be those with poor effort, deconditioning, pulmonary disease or angina.\(^\text{3,14}\) Many patients with advanced pulmonary diseases, particularly COPD, reach their ventilatory limits and cease exercise before reaching AT. Yet, any condition that causes a decreased delivery of oxygen to the exercising muscles to the extent that anaerobic metabolism occurs early in exercise will result in a low AT. For example, patients with significant heart disease or disease of the pulmonary vascular bed will have AT reached at low levels of \(\text{VO}_2\) due to the low cardiac output in relationship to the \(\text{VO}_2\). Other causes of low AT are peripheral vascular disease, anemia, and thy-

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**Figure 2.** Use of the anaerobic threshold (AT) for decision making in the differential diagnosis of exertional dyspnea. The disorders in which the AT is expected to be reduced are heart disease, pulmonary vascular disease, peripheral vascular disease and anemia. Reduced exercise performance in which the AT might be expected to be normal occurs in patients making poor effort, lung disease without significant pulmonary vascular disease, and coronary artery disease in which cardiac output is not significantly impaired at moderate rates. (Reproduced from reference 16, with permission.)

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Figure 3. Changes of minute ventilation to oxygen uptake ratio, ($\dot{V}e/\dot{V}o_2$) and minute ventilation to carbon dioxide production ratio, ($\dot{V}e/\dot{V}co_2$) against increasing oxygen consumption in three subjects studied in our laboratory. Anaerobic threshold is marked by the vertical dashed lines. End-tidal measurements of $Po_2$ and $Pco_2$ were also used for determining anaerobic threshold, but are not depicted here.

rotoxicosis. The figure shows the changes in the AT for the same subjects shown in Figure 1. The AT can be detected as the point where $\dot{V}e/\dot{V}o_2$ begins to increase, and the proportional increase in $\dot{V}e/\dot{V}co_2$ lags slightly behind. The AT is represented as a vertical dashed line. The patient with COPD did not reach AT while the patient with CHF reached AT at 28 percent of his predicted $\dot{V}o_2max$, which was confirmed by blood lactate levels. The normal individual reached AT at 64 percent of $\dot{V}o_2max$. Changes in the AT with repeated testing are used to assess progression of disease, response to medical treatment, or improvement in cardiovascular fitness with training. Expressed as a percentage of $\dot{V}o_2max$, an increase in the AT would indicate an improvement in the circulatory system's ability to deliver oxygen, and a significant decrease in this level would indicate the opposite. This trend should be interpreted in the context of the patient's clinical condition and value of the $\dot{V}o_2max$ or level of $\dot{V}o_2$ at maximal exercise. For example, treatment of pulmonary disease may significantly increase the exercise tolerance and the level of $\dot{V}o_2$ at maximal exercise without causing a change in AT. As the patient is encouraged to exercise more at home, the AT may also rise due to improvement in cardiovascular fitness. If the patient were then to sustain a myocardial infarction with significant impairment of function, subsequent testing may show a fall in the AT and $\dot{V}o_2$ at peak exercise.

A more complex situation would exist with a low AT due to pulmonary vascular disease from an interstitial process such as sarcoidosis. Severe abnormalities of ventilatory mechanics or $\dot{V}d/\dot{V}t$ would indicate that the circulatory problem was not likely to be cardiac in origin. However, since sarcoidosis may also involve the heart, additional testing of myocardial function might be necessary to further delineate the abnormality.

**Carbon Dioxide**

Measurement of the expired CO$_2$ is used to determine the anaerobic threshold as described above, calculate the $\dot{V}d/\dot{V}t$, and may be used to calculate cardiac output based on rebreathing techniques and the Fick equation. This technique for calculating cardiac output is accurate in subjects with normal lungs, and may be valid for some patients with COPD. $\dot{V}d/\dot{V}t$ measurement is discussed later in this review.

**Ventilation**

In progressive incremental exercise testing the minute ventilation ($\dot{V}e$) increases linearly with $\dot{V}o_2$ until anaerobic threshold is reached, after which it increases in relationship to the $\dot{V}co_2$. The value of the maximum attained $\dot{V}e$ divided by the maximum voluntary ventilation measured at rest (MVV) is sometimes called the dyspnea index and is frequently high in patients limited by pulmonary diseases. Inspection of the components of $\dot{V}e$, the tidal volume and the respiratory rate, also provides useful diagnostic information on the type of ventilatory impairment present. For example, a fixed low tidal volume with a rapid respiratory rate is seen in restrictive diseases such as interstitial fibrosis and can sometimes be seen with advanced heart failure.

In normal individuals the functional limits of the ventilatory system are predicted by the MVV. However, Dillard et al recently demonstrated that the $\dot{V}e$
is most accurately predicted in patients with advanced COPD using a formula based on the resting FEV₁ and peak inspiratory flow rate. Exercise in normal individuals is limited by cardiac output and not ventilation, so the maximum $\dot{V}E$ is usually not approached at peak exercise. Most normal individuals have significant ventilatory reserve, reaching peak exercise somewhere below 70 percent of their predicted maximum $\dot{V}E$. Patients with disease of the circulatory system also characteristically have ample ventilatory reserve, usually reaching peak exercise below 50 percent $\dot{V}E_{\text{max}}$, while patients with pulmonary disease show little or no room to increase ventilation. On the other hand, highly trained athletes may actually reach or exceed their predicted maximum capacity at peak exercise. These high levels of ventilation cannot be sustained for long, however, due to the high energy requirements of respiratory muscles at these rates. Exercise in advanced COPD patients is commonly limited by reaching the maximal $\dot{V}E$, and may be accompanied by arterial hypoxemia.

Figure 1 demonstrates the $\dot{V}E$ responses in the three subjects. Note the increase in the slope of the $\dot{V}E$ at AT in the normal individual, with adequate ventilatory reserve in the normal and CHF subjects, and the rapid rise in $\dot{V}E$ to near maximal value early in exercise with COPD.

Inspiratory and expiratory flow rates are also measured during exercise in some laboratories, and help classify the type of ventilatory impairment. In COPD, the high $\dot{V}E$ is associated with flow rates approaching the maximal resting values, in contrast to individuals with normal lungs who have a lower ratio of peak exercise flow rates to resting values. Figure 4 demonstrates the flow rate response to exercise in patients with airflow obstruction compared to normal subjects.

By measuring the mixed expired $\text{PCO}_2$ and arterial $\text{PCO}_2$, the physiologic dead space to tidal volume ratio ($\text{VD}/\text{VT}$) can be calculated using the Bohr equation. This value usually falls with exercise to less than .25 in normal individuals as the tidal volume increases and regional perfusion to alveolar units increases. Failure of this value to fall appropriately with exercise is found in several disease states, such as emphysema and interstitial fibrosis. These high values are due to high ventilation:perfusion ratios, low tidal volumes or both.

Disease of the pulmonary vascular bed decreases perfusion to areas of the lung and results in very high $\text{VD}/\text{VT}$ values. Although high $\text{VD}/\text{VT}$ values are usually associated with pulmonary disease, they may be seen in patients with advanced heart failure. In normal lungs, the end-tidal $\text{PCO}_2$ approximates the arterial $\text{PCO}_2$ with exercise and can be used to estimate the anatomic $\text{VD}/\text{VT}$.

The values of $\text{VD}/\text{VT}$ in normal, COPD, and CHF patients are given in Figure 1. Both the normal subject and the CHF patient had a normal decline in the $\text{VD}/\text{VT}$, but the COPD patient had a sustained elevated ratio typical of this disease.

Other Parameters

The diffusing capacity of carbon monoxide (DCO) can be measured in exercising subjects. It is usually performed at steady state, although single breath DCO has been determined with submaximal exercise. Since the procedure requires additional equipment, it is not commonly used in stress testing laboratories. However, Owens et al. have shown that the resting single breath DCO correlates with exercise-induced arterial hypoxemia in COPD patients when the value is below 55 percent predicted. It has also been shown to correlate with development of arterial hypoxemia during exercise in patients with interstitial fibrosis.

The degree of arterial hypoxemia with exercise has also been determined noninvasively with the use of ear oximetry, which is frequently used as an adjunct to progressive multistage stress testing. Oxygen saturation does not change significantly in normal subjects during exercise testing until late in the study when accumulation of lactic acid may cause a drop in pH and uncoupling of oxygen from hemoglobin causing slight desaturation. Arterial desaturation from hypoxemia frequently occurs in patients with significant COPD, and may be associated with an increase in the arterial $\text{PCO}_2$. Increases in the $\text{PO}_2$, and presumably saturation, have been found in some patients with COPD of the bronchitis type, even when moderately severe disease exists.

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**Figure 4.** Spontaneous peak expiratory flow during exercise as a fraction of resting maximal expiratory peak flow. The shaded area encompasses the range of values determined in normal subjects. The data points were obtained during an incremental exercise test from patients with bronchial asthma with the highest value the limit of tolerable work. The numbers in parentheses are the maximal mid-expiratory flow measured at rest. (From reference 3, with permission.)
The clinical evaluation of the patient before and after the exercise test is just as important as the measurement of other parameters and should not be overlooked. The diagnostic importance of findings of wheezing, S3 gallop, or basilar rales postexercise is readily apparent. Measurement of pulmonary function postexercise is essential to the diagnosis of exercise-induced asthma, in which flow rates may fall several minutes post-exercise.31

In summary, cardiopulmonary exercise testing is used to induce or amplify abnormalities which are not readily evident at rest. An exercise study which examines the responses of both the cardiac and pulmonary systems may clarify whether an exercise limitation results from cardiac or from pulmonary disease. It must be remembered, however, that the results should be interpreted in light of the clinical information available and not relied upon to make the clinical diagnosis. Since patients often have impairment of both cardiac and pulmonary systems, the characteristic abnormalities found with disease of either system may overlap. It is prudent to assume that the most prominent abnormality demonstrated is likely to be the primary source of the exercise limitation at the time of testing. Moreover, measurement of pulmonary gas exchange with exercise can be used to assess the severity of the illness, and repeated studies can be performed to assess progression of disease, or response to treatment.

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