Understanding the Basics of Cardiopulmonary Exercise Testing

RICHARD V. MILANI, MD; CARL J. LAVIE, MD; MANDEEP R. MEHRA, MD; AND HECTOR O. VENTURA, MD

Cardiopulmonary exercise testing adds important additional information to that provided by the standard exercise test. In particular, cardiopulmonary exercise testing provides precise determination of aerobic capacity, the causes of dyspnea with exertion, and prognosis in patients with systolic heart failure. This review provides basic, practical information about cardiopulmonary exercise testing for the clinician.


HOW IS GAS EXCHANGE MEASURED?

Exercise stress testing is commonly used to evaluate the presence and severity of coronary ischemia, as well as exertional symptoms, heart rate (HR) and blood pressure responses, and estimated aerobic capacity.1,2 Additional important clinical information may be obtained by direct measurement of exercise respiratory gas exchange, referred to as cardiopulmonary exercise testing.3 This review updates the clinician on the indications and interpretation of cardiopulmonary exercise testing in modern medicine.

WHY MEASURE GAS EXCHANGE?

An important prognostic component of exercise stress testing is the evaluation and quantification of work performed, also known as functional capacity.3 Because energy consumption, generally quantified as caloric expenditure, is difficult to measure during exercise, a more readily measurable metabolic equivalent (MET) was sought to quantify the work performed at various stages of exercise. In humans, total oxygen consumption approximates caloric expenditure, with both parameters increasing during exercise in a similar fashion. Thus, 1 MET of caloric expenditure was set at the resting level of oxygen consumption of a healthy man weighing 70 kg or 3.5 mL/kg per minute. Therefore, any physical activity can be viewed as a multiple of this unit measure, with threshold requirements identified for individuals who want to perform unique activities. Typically, simple household activities and light work activities require an energy expenditure of approximately 1.5 to 4 METs, moderate work and typical sexual activities require an energy expenditure of approximately 3 to 6 METs, and heavy work or high-level sports activities require an energy expenditure of 5 to 15 METs.3 Direct assessment of METs has become an integral component in occupational development and disability determination, and cardiopulmonary exercise testing remains the sole modality for its determination.

Like the lungs, the primary function of the cardiovascular system is gas exchange, supplying oxygen and other fuels to working muscles and removing carbon dioxide and other metabolites. The heart, lungs, and pulmonary and systemic circulations form a single circuit for exchange of respiratory gases between the environment and the cells of the body.6,7 Under steady-state conditions, oxygen consumption per unit time (\(\dot{V}O_2\)) and carbon dioxide output (\(\dot{V}CO_2\)) measured at the mouth are equivalent to oxygen utilization and carbon dioxide production occurring in the cell; thus, external respiration equals internal respiration (Figure 1).7 Cardiopulmonary exercise testing measures fractions of oxygen and carbon dioxide in expired gas, expired air volume, or flow and calculates \(\dot{V}O_2\), \(\dot{V}CO_2\), and minute ventilation (\(V_e\)) with a nonrebreathing valve connected to a metabolic cart (Figure 2). Samples of expired air are typically assessed every 15 seconds (some systems use 30- to 45-second increments), and real-time data are expressed in both a tabular and a graphic format. Additionally, oxygen saturation using finger, ear lobe, or forehead oximetry is monitored and recorded. From these data, numerous clinically relevant metabolic parameters can be derived (Table 1).

To obtain reliable metabolic data that reflect steady-state conditions, it is imperative to avoid high increments in workload, which are often present when using a Bruce exercise protocol, and extremes of test duration. Many laboratories use ramping treadmill and/or bicycle exercise protocols, which are tailored to the patient’s activity his-
CARDIOPULMONARY EXERCISE TESTING

FIGURE 1. Gas transport mechanisms coupling cellular (internal) respiration to pulmonary (external) respiration. Circ = circulation; CO₂ = carbon dioxide; Consum = consumption; Creat = creatine; Lac = lactate; HR = heart rate; Mito = mitochondria; PO₄ = phosphate; O₂ = oxygen; Periph = peripheral; Prod = production; Pulm = pulmonary; Pyr = pyruvate; QCO₂ = carbon dioxide production; VO₂ = oxygen utilization; SV = stroke volume; VA = minute alveolar ventilation; VD = minute dead space ventilation; VE = minute ventilation; f = breathing frequency; VT = tidal volume; VCO₂ = carbon dioxide output; VO₂ = oxygen uptake. From Principles of Exercise Testing and Interpretation, 3rd ed, with permission from Lippincott Williams & Wilkins.

FIGURE 2. A patient undergoing a cardiopulmonary stress test. The head harness worn by the patient holds the mouthpiece and nonrebreathing valve that is connected to a metabolic cart to the immediate left of the patient.
When compared with other exercise protocols, including the Bruce protocol. The Balke and Naughton protocols can also be considered because they involve only modest increases in treadmill elevations at a constant speed.\(^4\)

With standard exercise testing, METs can be estimated by regression formulas, standard accessories on most commercially available treadmills. Several studies have demonstrated that these formulas are often inaccurate and typically overestimate METs when using fixed work-rate exercise protocols such as the Bruce treadmill protocol, which uses large or unequal increments in work.\(^8\) Ramping exercise protocols permit increases in external work to occur in a constant or continuous fashion such that increases in workload can be individualized throughout a wide range of patient capabilities.\(^11\)

**CARDIOPULMONARY RESPONSES TO INCREMENTAL EXERCISE**

In healthy people, predictable physiologic changes occur during exercise. These changes include a reduction in systemic vascular resistance, increases in oxygen extraction, and augmentation of stroke volume (SV) and HR, resulting in an increase in cardiac output (CO). The coupling between oxygen transport and circulatory function is revealed in the Fick equation, which states \(\dot{V}_O_2 = CO \times (CaO_2 - CvO_2)\), in which \(CaO_2\) is the arterial oxygen concentration and \(CvO_2\) is the mixed venous oxygen content. This formula can be rewritten as \(\dot{V}_O_2 = (HR \times SV) \times (CaO_2 - CvO_2)\). The Fick equation can be modified to reflect the changes in each variable at peak exercise, which contribute to the maximum oxygen consumption per unit time (\(V_0_{2\text{max}}\)) as \(V_0_{2\text{max}} = (HR_{\text{max}} \times SV_{\text{max}}) \times (CaO_2_{\text{max}} - CvO_2_{\text{min}})\), with max indicating maximum and min indicating minimum.

**PHYSIOLOGIC RESERVE**

Although the lungs and the heart are coupled in gas exchange, they differ with regard to physiologic reserve during maximal exercise. By definition, under maximal stress, the HR reserve is zero. Defining the maximal predicted HR as 220 – age, stress tests are considered less than adequate (reduced sensitivity) when HR does not exceed 85% of predicted, thus leaving a reserve of greater than 15%. However, this formula is not accurate in predicting maximal HR for all patients (particularly those taking \(\beta\)-blockers and those with diabetes). A desirable HR reserve under maximal stress is less than 15% and is ideally close to zero. In contrast, the ventilatory reserve or breathing reserve (BR) in healthy controls cannot be less than 20% and is typically between 30% and 50%. Baseline spirometry, including maximum voluntary ventilation (MVV), is measured as part of the testing procedure before exercise. The BR is calculated as follows: \(BR = 1 - V_{\text{max}}/MVV\). Thus, lung mechanics are not limiting in healthy individuals but can become compromised by obstruction of airflow, restriction of lung volumes, or ventilation-perfusion mismatch. These conditions may additionally result in arterial oxygen desaturation at peak exercise, a situation that does not occur in patients with coronary artery disease or cardiomyopathy but may occur in patients with congenital heart disease and/ or pulmonary hypertension.\(^12\)

**ANAEROBIC THRESHOLD**

When the metabolic demands of exercise begin to exceed oxygen delivery to working muscles, anaerobic metabolism ensues.\(^3,7,12\) However, even with low-intensity exercise, anaerobic energy production may make a small contribution that markedly increases at exercise intensities greater than the anaerobic threshold (AT). This is evidenced by rising blood lactate levels and lactate-pyruvate ratios. Lactic acid is then buffered by bicarbonate, producing excess nonmetabolic carbon dioxide via the carbonic anhydrase reaction: \(H^+ + HCO_3^- \Leftrightarrow H_2CO_3 \Leftrightarrow CO_2 + H_2O\). The resultant increase in carbon dioxide output is detected by chemoreceptors in the carotid bodies, which mediate an increase in \(V_{\text{E}}\). The AT, also known as the ventilatory threshold, is defined as the highest \(V_0_2\) attained without a sustained increase in blood lactate concentration and lactate-pyruvate ratio. It is detected metabolically as the point of inflection at which \(V_{CO_2}\) and \(V_{E}\) increase relative to \(V_0_2\). The most common means for identifying the AT is the \(V_\text{slope}\) method, which uses regression analysis to present the inflection point on a plot of \(V_{CO_2}\) vs \(V_0_2\).\(^13,15\) The AT occurs typically between 47% and 64% of the \(V_0_2\)max in healthy untrained individuals and generally at a higher percentage of \(V_0_2\)max in endurance-trained individuals.\(^3,7,12,14\)

For the purposes of exercise prescription, knowledge of the AT can be useful. At workloads below the AT, blood lactate levels remain low, slow-twitch (type I) fibers with high oxidative capacity are used, and exercise can be sustained for prolonged periods, limited only by substrate

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**TABLE 1. Metabolic Parameters Derived From Cardiopulmonary Stress Testing**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Definition</th>
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<tbody>
<tr>
<td>Peak oxygen consumption per unit time</td>
<td>(\dot{V}_O_2)</td>
</tr>
<tr>
<td>Breathing reserve*</td>
<td>(1 - V_{\text{max}}/MVV)</td>
</tr>
<tr>
<td>Oxygen saturation</td>
<td>(\frac{V_{CO_2}}{\dot{V}_O_2})</td>
</tr>
<tr>
<td>Anaerobic threshold</td>
<td>(V_{E}) increase relative to (V_0_2)</td>
</tr>
<tr>
<td>Respiratory exchange ratio</td>
<td>(V_{E}/\dot{V}_O_2)</td>
</tr>
<tr>
<td>Oxygen pulse</td>
<td>(\frac{\dot{V}_O_2}{MVV})</td>
</tr>
<tr>
<td>Ventilatory equivalent for carbon dioxide</td>
<td>(\frac{V_{E}}{V_{CO_2}})</td>
</tr>
<tr>
<td>Peak oxygen consumption per unit time adjusted for lean body mass†</td>
<td>(\dot{V}_O_2) adjusted for lean body mass</td>
</tr>
<tr>
<td>Oxygen pulse adjusted for lean body mass†</td>
<td>(\frac{\dot{V}_O_2}{MVV})</td>
</tr>
</tbody>
</table>

*Requires pretest spirometry with maximum minute ventilation.†Requires body composition assessment.
availability and musculoskeletal trauma. At workloads above the AT, blood lactate levels increase, fast-twitch (type II) fibers with low oxidative capacity are used, and exercise duration shortens progressively with increasing load.\textsuperscript{3,7,12} The AT affects activities of daily living and can be extended in health by exercise training and reduced through deconditioning. In addition, an AT below 11 mL/kg per minute and/or ischemia on the electrocardiographic portion of the cardiopulmonary exercise test has been used to identify a high risk of perioperative death in elderly patients undergoing major surgical procedures.\textsuperscript{16}

**Respiratory Exchange Ratio**

The respiratory exchange ratio (RER) is related but not equivalent to its cellular counterpart the respiratory quotient and is defined as the ratio of V\textsubscript{CO\textsubscript{2}} to V\textsubscript{O\textsubscript{2}}: \textit{RER} = \text{V\textsubscript{CO\textsubscript{2}}} / \text{V\textsubscript{O\textsubscript{2}}}. The RER represents the metabolic exchange of gases in the body's tissues and is dependent in part on the predominant fuel (carbohydrate vs fat) used for cellular metabolism.\textsuperscript{17} At rest and with early exercise, the V\textsubscript{CO\textsubscript{2}} curve runs slightly below the V\textsubscript{O\textsubscript{2}} curve. Once the AT is passed, additional nonmetabolic carbon dioxide is produced, resulting in a steep rise in V\textsubscript{CO\textsubscript{2}} and an accompanying rise in RER to values ultimately exceeding 1.0. As lactate builds in exercising muscle, exercise becomes progressively uncomfortable. Because the level of RER is directly related to muscle lactate accumulation, it can be used as an objective means of quantifying effort. Respiratory exchange ratio values less than 1.0 generally indicate poor effort, 1.0 to 1.1 indicate fair effort, 1.1 to 1.2 indicate good effort, and values that exceed 1.2 indicate an excellent effort. Exceptions to this rule include neuromuscular limitations that prevent sustained exercise, low threshold peripheral or myocardial ischemia, severe ventilatory impairment, failure of testing personnel to encourage good patient effort, or test stoppage due to worrisome symptoms or signs.

**Peak VO\textsubscript{2}**

The peak VO\textsubscript{2} (PkVO\textsubscript{2}) is the highest VO\textsubscript{2} achieved during the cardiopulmonary exercise testing and generally occurs at or near peak exercise. Moreover, if the VO\textsubscript{2} curve demonstrates a plateau, such that VO\textsubscript{2} no longer increases despite progressive increments in workload, then the PkVO\textsubscript{2} can be labeled the VO\textsubscript{2} max.\textsuperscript{15} The VO\textsubscript{2} max is the best and most reproducible index of cardiopulmonary fitness or disability.\textsuperscript{3,35} The PkVO\textsubscript{2}, VO\textsubscript{2} max, and AT are reported as a weight-adjusted value in milliliters of VO\textsubscript{2} per kilogram of body weight per minute to facilitate intersubject comparisons. To convert PkVO\textsubscript{2} or VO\textsubscript{2} max to METs, divide by 3.5 mL/kg per minute.

Normally, VO\textsubscript{2} max decreases with age, declining 8% to 10% per decade in nonathletic individuals and approximately 5% per decade in trained individuals who continue to exercise vigorously.\textsuperscript{4} At any age, VO\textsubscript{2} max is 10% to 20% higher in men than women, in part because of a higher hemoglobin concentration, a larger muscle mass, and a greater SV in men.\textsuperscript{5,19,20} These age and sex differences in VO\textsubscript{2} max must be considered when interpreting cardiopulmonary exercise testing. As a result, the measured VO\textsubscript{2} max will be compared with a predicted value generated from empirically derived formulas based on age, sex, and height.\textsuperscript{7,12,21,22} The VO\textsubscript{2} max would be considered decreased if the measured value is less than 85% of predicted.

**Oxygen Pulse**

The amount of oxygen consumed from the volume of blood delivered to tissues by each heart beat is termed the oxygen pulse and depends on the size of the SV and the arteriovenous oxygen difference and may be more dependent than PkVO\textsubscript{2} on cardiac pump function reserve (because it does not incorporate HR reserve). The oxygen pulse is calculated as VO\textsubscript{2} divided by HR.

Thus, by altering the Fick equation as follows, the oxygen pulse can provide an index of the SV and arteriovenous oxygen difference:

\[
\text{VO}_2 = (HR \times \text{SV}) \times (\text{CaO}_2 - \text{CvO}_2)
\]

\[
\text{VO}_2/HR = \text{SV} \times (\text{CaO}_2 - \text{CvO}_2)
\]

Since oxygen extraction is maximal and relatively constant at peak exercise, the oxygen pulse becomes a reasonable surrogate of SV and is diminished in patients with severe left ventricular dysfunction and/or valvular heart disease.

**Ventilation–Carbon Dioxide Production Ratio**

The major link between the circulatory and ventilatory responses to exercise is carbon dioxide production. Ventilatory efficiency is defined by linking ventilation relative to carbon dioxide production, resulting in the matching of ventilation and perfusion.\textsuperscript{23,24} The breathing requirements of exercise can be described from the equation \( V'_{\text{E}} = V_{\text{CO}_2} \times V_{\text{E}}/V_{\text{CO}_2} \), in which \( V'_{\text{E}}/V_{\text{CO}_2} \) is the ventilation–carbon dioxide production ratio, also known as the ventilatory equivalent for carbon dioxide. \( V'_{\text{E}}/V_{\text{CO}_2} \) is a respiratory control function that reflects chemoreceptor sensitivity, acid-base balance, and ventilatory efficiency at the alveolar-capillary interface.\textsuperscript{12,25,26} At the onset of exercise, \( V'_{\text{E}}/V_{\text{CO}_2} \) typically decreases because of improved ventilation and perfusion matching. As exercise progresses, \( V'_{\text{E}}/V_{\text{CO}_2} \) remains fairly constant because \( V'_{\text{E}} \) tracks \( V_{\text{CO}_2} \) linearly. Finally, as exercise peaks, the development of metabolic acidosis results in an additional ventilatory stimulus, resulting in a small increase in \( V'_{\text{E}}/V_{\text{CO}_2} \). Increased \( V'_{\text{E}}/V_{\text{CO}_2} \) is commonly observed in patients with pulmonary disease and heart failure (HF).
CARDIOPULMONARY EXERCISE TESTING

WHAT WILL THE TEST RESULTS TELL ME?

Besides precise assessment of peak aerobic capacity (ie, measured METs as opposed to estimating METs), cardiopulmonary exercise testing is invaluable in determining the cause of dyspnea on exertion and determining normality of cardiac and pulmonary responses to exercise. The most common indications for cardiopulmonary exercise testing are outlined in Table 2.

Metabolic derangements can occur at multiple sites within the circuitry of gas exchange, including the consumers at the muscle mitochondria, the transporters within the circulatory system, and the exchangers at the site of ventilation (Figure 1). Knowledge of site and extent of metabolic dysfunction through cardiopulmonary exercise testing can have a wide application in clinical medicine.

Cardiopulmonary exercise testing provides an ideal modality for the evaluation of patients who present with exertional dyspnea and fatigue, when the clinician is faced with a breadth of differential diagnoses ranging from circulatory impairment to deconditioning. Standard diagnostic studies may not identify the true cause because circulatory and ventilatory reserves cannot be fully assessed from indices of resting cardiac and pulmonary function. By virtue of obtaining gas exchange data under the provocation of exercise, cardiopulmonary exercise testing can identify potential deficiencies within these systems.

Using the algorithm provided in Figure 3, a PkVo₂ less than 85% of age- and sex-predicted values is considered low, and, as discussed previously, a normal AT is generally closer to 60% of the predicted PkVo₂. However, for purposes of classification an AT less than 40% of the predicted PkVo₂ is considered pathologically reduced and indicative of circulatory insufficiency. A BR less than 30% would indicate ventilatory impairment, especially when accompanied by oxygen desaturation with exercise, although a BR of 20% to 30% is deemed a borderline value. Cardiopulmonary exercise testing is useful in dyspneic patients with combined cardiac and pulmonary diseases who may have a reduction in AT and/or BR, the more dominant of which may indicate the primary cause of the patient’s functional

**TABLE 2. Potential Indications for Cardiopulmonary Stress Testing**

<table>
<thead>
<tr>
<th>Indication</th>
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<tbody>
<tr>
<td>Evaluation for exertional dyspnea</td>
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<tr>
<td>Development of an exercise prescription</td>
</tr>
<tr>
<td>Direct measurement of peak oxygen consumption per unit time (functional capacity)</td>
</tr>
<tr>
<td>Risk stratification and prognosis in heart failure</td>
</tr>
<tr>
<td>Optimization of rate-adaptive or biventricular pacemaker</td>
</tr>
<tr>
<td>Congenital heart disease: determination of need for surgical repair and response to treatment</td>
</tr>
<tr>
<td>Disability determination; work-site readiness</td>
</tr>
<tr>
<td>Assess functional significance of regurgitant valvular heart disease</td>
</tr>
<tr>
<td>Assess the results of medical and surgical therapies</td>
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</tbody>
</table>

**FIGURE 3. Flowchart for the differential diagnosis of exertional dyspnea and fatigue.** Vo₂ = oxygen consumption per unit time. From Arch Intern Med with permission. Copyright 1988 American Medical Association. All rights reserved.
limitation. An RER of less than 1.1 (particularly <1.0) in
the absence of other metabolic abnormalities suggests poor
effort, anxiety, or mild disease.

CARDIOPULMONARY EXERCISE TESTING IN HF

From a clinical standpoint, cardiopulmonary exercise test-
ing has gained widespread use in the evaluation of patients
with advanced systolic HF. Although left ventricular
ejection fraction does not predict PkVO₂, numerous studies
have confirmed the prognostic utility of measuring gas
exchange in patients with systolic HF. In the Veterans
Administration Heart Failure Trial, mortality of patients
with a VO₂ max of 14.5 mL/kg per minute or less was double
that of patients whose VO₂ max exceeded this value. In a
separate investigation of patients with HF referred for car-
diac transplantation, Mancini et al found that PkVO₂ was
the single best predictor of survival. Moreover, transplantation
could be safely deferred in patients whose PkVO₂ values
were greater than 14 mL/kg per minute, in which their
survival exceeded that of patients undergoing heart trans-
plantation. As a result of these seminal studies, cardiopulmo-
nary exercise testing remains a pivotal modality in the initial
evaluation of patients with advanced HF, especially those
who are considered for heart transplantation.

Although the PkVO₂ cutoff of 14 mL/kg per minute
remains an important prognostic discriminator in patients
with HF, disparities in its prognostic utility may occur
when evaluating special populations, including women and
obese people. A fundamental understanding of oxygen
consumption may explain these disparate observations.
Although PkVO₂ is usually corrected for total body weight,
body fat is metabolically inert, consuming essentially no
oxygen, and can represent a significant portion of total
weight. Moreover, considerable variability in body composi-
tion is present across populations, including those with HF.
Correcting PkVO₂ for lean body mass provides a more re-
ned discriminator of outcome than traditionally reported
total weight-adjusted values. In patients with HF, a PkVO₂
corrected for lean body mass cutoff of 19 mL/kg per minute
provides a more robust discriminator of major cardiac events
(death and/or urgent heart transplantation) than the total
weight-adjusted figure of 14 mL/kg per minute (Figure 4).

As such, routine assessment of body fat using the 3-site
skinfold method before each cardiopulmonary exercise
study can be used to calculate lean body mass. From a
practical standpoint, this adds only 3 to 4 minutes to the time
required to perform cardiopulmonary exercise testing. Using
the lean body mass–adjusted PkVO₂ may eliminate previ-
ously observed disparities between the sexes, as well as in
obese individuals, in predicting outcome in patients with HF.

Recently, oxygen pulse has been studied to assess prog-
nosis in patients with HF. We recently reported that an
uncorrected peak oxygen pulse of 10 mL/beat separated
those with clinical events compared with event-free survi-
vors, whereas a lean body mass–adjusted value of 14 mL/
beats for peak oxygen pulse provided an even better prognostic ability for HF event-free survival (Figure 5). In fact, in multivariate models that contained clinical and cardiopulmonary variables, a higher lean body mass–adjusted oxygen pulse was the strongest independent predictor of event-free survival, and this was particularly the case in patients with class III HF.

Several studies have suggested that additional cardiopulmonary variables, including indexed PKVO\(_2\),\(^{37,38}\) percent predicted PKVO\(_2\),\(^{39}\) V\(_{E}/V\text{CO}_2\) or the slope of V\(_{E}/V\text{CO}_2\),\(^{25,38,40,41}\) V\(_{O_2}\) recovery (defined as the time needed for V\(_{O_2}\) to decrease by 50% from its peak value), and cardiac or peak circulatory power (defined as the product of PKVO\(_2\) and systolic arterial pressure),\(^{36}\) may contribute additional prognostic information in the evaluation of patients with HF. Recently, the applicability and prognostic role of cardiopulmonary exercise testing in patients with chronic HF who are maximally treated with an up-to-date pharmacological armamentarium, including β-blocking agents, which result in lower HRs during exercise, have been questioned, with several studies suggesting that PKVO\(_2\) loses some prognostic ability.\(^{27,32,41-44}\) In patients taking β-blockers, a PKVO\(_2\) of less than 14 mL/kg per minute has shown reduced prognostic stratification, whereas variables less affected by HR reserve, such as peak oxygen pulse or peak lean body mass–adjusted oxygen pulse, may be superior. A lean body mass–adjusted peak oxygen pulse (cutoff <14 mL/beat) was superior in risk stratification (7% vs 22% events) compared with PKVO\(_2\) (cutoff <14 mL/kg per minute) (10% vs 17% events).\(^{32}\)

Less commonly, patients with HF may have limited exercise tolerance because of low-threshold angina or severe ventricular arrhythmias, in which an early exercise surrogate of PKVO\(_2\) would be needed for risk stratification. Our laboratory and others have successfully used the pattern of V\(_{E}/V\text{CO}_2\) change during early exercise to predict PKVO\(_2\) and subsequent outcome in such patients.\(^{25,43,46}\) A decrease in V\(_{E}/V\text{CO}_2\) of less than 10% early in exercise predicts a PKVO\(_2\) of less than 14 mL/kg per minute and poor outcome in patients with HF.\(^{25}\)

Currently, PKVO\(_2\) is a commonly used end point in various clinical investigations, particularly HF trials.\(^{47,49}\) In addition to the prognostic importance of baseline PKVO\(_2\), changes in PKVO\(_2\) may have prognostic and therapeutic
Cardiopulmonary exercise testing remains a relevant modality in the clinician’s diagnostic armamentarium for evaluation and treatment of many commonly encountered clinical problems. Measurement of exercise gas exchange provides objective and reproducible indices of functional capacity, generates invaluable information in determining the origin of dyspnea on exertion, and provides unique prognostic capabilities in the assessment of patients with systolic HF. Physicians should be comfortable with the basics of gas exchange presented herein and therefore better able to use cardiopulmonary exercise testing in the appropriate clinical settings.

REFERENCES


