Cardiopulmonary exercise testing (CPX) is established as a useful clinical tool for evaluating the severity of heart failure and the limitations of activities of daily life in cardiac patients. Among the several key indices obtained from CPX, a long-accepted gold standard of exercise capacity is the peak oxygen uptake (VO₂), a marker of maximal cardiac output. There have been ongoing attempts, however, to develop an evaluation of exercise capacity without requiring maximal effort. The anaerobic threshold (AT) is the first established index of exercise capacity obtained from submaximal exercise testing.

A classic study from the 1920s proposed that anaerobic me-
metabolism occurred during exercise with lactic acid accumulation when the O2 supply to the working muscles could not be increased to a level sufficient to maintain aerobic metabolism. In 1964, Wasserman and McIlroy proposed that exercise capacity could be reliably assessed by an index called the AT, the threshold VO2 above which a subject was incapable of working without the development of lactic acidosis. During exercise below the AT, CO2 output (VCO2) increases as a linear function of VO2 in response to aerobic metabolism with a slope of 0.95–1.0. The slope of the plot of VCO2 against VO2 steepens above the VO2 at which lactic acidosis develops, as additional CO2 over that produced from aerobic metabolism is generated from lactic acid buffered by HCO3. Based on these phenomena, the AT, an index reflecting the lactate (lactic acidosis) threshold, has been determined as a breakpoint in the VCO2–VO2 plot (V-slope method). The AT, lactate threshold, and lactic acidosis threshold are all part of the same physiological phenomenon (ie, the anaerobic metabolism), and the distinction in terminology simply describes the method of measurement. Although the AT has been primarily watched with interest in the field of sports medicine, it was also taken up by cardiologists in the 1980s as a useful, noninvasive index of the severity of heart disease without requiring maximal effort. The AT has been found to serve as an objective measure of the exercise intensity beyond which exercise begins to impair left ventricular function in cardiac patients. The AT is also used to prescribe optimal exercise intensity in cardiac rehabilitation. However, the drawback of the AT is its somewhat lower detectability in heart failure patients compared with other established CPX indices. The peak VO2, the slope of the increase in ventilation (VE) relative to the increase in VCO2 (VE–VCO2 slope), and the ratio of the increase in VO2 to the increase in work rate (∆VO2/∆WR) are all more easily determined than the AT. Agostoni et al recently reported that the AT was impossible to detect in approximately 10% of patients with heart failure (NYHA I–III). The AT is also difficult to determine in patients with exaggerated oscillatory breathing arising from alternation between hyperpnea and hypopnea, even though the V-slope method is insensitive to breathing irregularities.

The high price of respiratory gas systems has prompted efforts to estimate the AT without respiratory gas analysis, with help from parameters such as the heart rate deflection point and heart rate variability. In 1982, Conconi et al discovered that the heart rate deflection point coincides with the lactate threshold. Michele et al recently reported that the AT can be assessed by analyzing heart rate variability.

In this issue of the Journal, Tanaka et al also focus on the noninvasive detection of the lactate threshold without respiratory gas analysis. They draw attention to the amplitude of the first sound (AHS1) measured from a phonocardiogram. Previously, they reported that the double product (DP), calculated by multiplying the heart rate and systolic blood pressure, increased more steeply during exercise above the lactate threshold in healthy subjects. They proposed that the breakpoint of DP was a valid and useful index of the lactate threshold. Noting that further increases of DP above the lactate threshold appeared to be related to rising catecholamine levels, they hypothesized in the present study that AHS1, a parameter assumed to reflect left ventricular contractility, may be used as a substitute for systemic blood pressure in DP. Tanaka et al found a strong positive correlation between AHS1 and left ventricular dp/dt max in their healthy subjects, as well as correlations of the product of heart rate and AHS1 (DP-AHS1) with adrenaline and blood lactate levels. They also noted that the breakpoint in DP-AHS1 strongly correlated with the AT and peak VO2 in their subjects. Agostoni et al proposed that a failure to detect AT in heart failure patients is associated with a reduced peak VO2, and that an indeterminable AT itself has an independent prognostic role. It would be helpful, however, to detect the anaerobic (or lactate) threshold even in patients with severe heart failure, for the purpose of obtaining a valuable CPX index and prescribing optimal exercise intensity for cardiac rehabilitation. It may be possible to increase the detectability of the lactate threshold in heart failure patients by a method independent of respiratory gas analysis, such as one of the techniques proposed by Tanaka et al. Further investigation may help clarify whether the breakpoint of DP and the breakpoint of DP-AHS1 reflect the lactate threshold not only in healthy subjects, but also in heart failure patients in whom the AT is indeterminable by respiratory gas analysis. It would also be helpful to clarify whether these indices have a prognostic value in cardiac patients similar to the prognostic value of AT determined by respiratory gas analysis.

References