

The Anaerobic Threshold: What it is and How to Estimate it

Susan A. Ward DPhil
University of Leeds
and
Human Bio-Energetics Research Centre
Lymington, United Kingdom

The anaerobic (*lactate; lactic acidosis; ventilatory; gas exchange; ...*) threshold (AT) is considered to be a parameter of aerobic function during exercise that characterises the upper limit for wholly-aerobic energy provision. It thus partitions exercise intensity between the "moderate" and the higher "heavy" or "severe" domains. Moderate-intensity exercise categorizes the range of constant work rates within which there is not a sustained metabolic acidaemia, with arterial blood [lactate] and $[H^+]$ remaining at or close to resting levels. In contrast, heavy-intensity exercise reflects the work-rate range for which arterial blood [lactate] and $[H^+]$ are elevated, but can eventually stabilize (or may even decrease) as the applied work rate is continued. The higher range of work rates for which arterial [lactate] and $[H^+]$ are not only elevated but increase inexorably with time throughout the test are of severe intensity. Moderate exercise may be sustained for long periods, whereas heavy and severe exercise may not - as a result of the increased metabolic, acid-base, ventilatory and perceptual stress.

The AT highly task-specific - for example, occurring at an appreciably lower oxygen uptake ($\dot{V}O_2$) for arm exercise than for leg exercise, and is typically lower for cycle ergometry than for treadmill exercise, reflecting the magnitude of the involved muscle mass. A wide range of techniques have been advocated for anaerobic threshold measurement, both direct from blood analysis and indirect from respired gas analysis.

For the direct method, it is important that an appropriate sampling site is used, ideally arterial. However, sampling venous blood from properly "arterialized" venous blood is also acceptable. For this, a site with low metabolic rate is selected and the blood flow across this region is raised, usually by appropriate external heating, so that the arterio-venous difference for metabolites becomes negligible. The dorsum of the hand has been shown to be such an appropriate site. However, a forearm vein is unacceptable, as the forearm muscles can extract lactate, delaying the appearance of the AT. Puncturing or lancing the earlobe or fingertip provides acceptable single (or possibly a few) samples but is not practicable for the numerous samples required for reliable AT determination.

However, one can forego the necessity for serial blood sampling and, in many cases, enhance the discriminability of the AT by utilizing a particular cluster of ventilatory and pulmonary gas-exchange responses that reflect the consequence of the associated $[H^+]$ increase, in response to a rapid incremental or ramp work-rate protocol. This is based on the demonstration that over 90% of the buffering of the proton produced anaerobically with the lactate anion is accomplished by the bicarbonate system. Each molecule of bicarbonate decrease yields one molecule of CO_2 , which supplements the aerobically-produced CO_2 from the oxidative breakdown of carbohydrate and free fatty acids. This results in an accelerated CO_2 output ($\dot{V}CO_2$) response at the AT. As ventilation ($\dot{V}_E$) during exercise, in the first instance, operates to regulate arterial PCO_2 , there is also an associated accelerated $\dot{V}_E$ response at the AT in proportion to the $\dot{V}CO_2$ response. As a result, the ventilatory equivalent for CO_2 ($\dot{V}_E/\dot{V}CO_2$) remains essentially stable with no change in alveolar end-tidal PCO_2 (i.e. *no hyperventilation relative to CO_2*). However, O_2 uptake ($\dot{V}O_2$) continues to increase essentially linearly with the work rate beyond the AT, the ventilatory equivalent for O_2 ($\dot{V}_E/\dot{V}O_2$) and the alveolar end-tidal PO_2 both therefore start to increase at the AT (i.e. *hyperventilation relative to O_2*).

Of note, it is only some way beyond the AT that respiratory compensation for the metabolic acidemia becomes evident for rapid incremental or ramp work-rate exercise – as an increase in $\dot{V}_E/\dot{V}CO_2$ and a corresponding fall in alveolar end-tidal PCO_2 . This is thought to reflect the kinetics of the carotid chemoreceptors (the primary involved sensors for arterial metabolic H^+ detection) being slow relative to the rate of metabolic rate increase with this test format.

In conclusion, the AT (as estimated from the ventilatory and pulmonary gas exchange indices described above) is currently used for:

- a) assessing the normalcy, or otherwise, of an individual's integrative systemic function;
- b) optimising the intensity of sports training and clinical rehabilitative and prehabilitative protocols;
- c) judging a patient's appropriateness to undergo major thoracic or abdominal surgery;
- d) triaging a patient post-operatively to the ward or to an intensive care facility; and
- d) providing an index of life expectancy in patients with heart disease.

It is therefore important that the threshold determination should be both valid and reliable. Procedures will be discussed to optimise the discrimination and assure its validity.