Possible Mechanisms of the Anaerobic Threshold
A Review

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Summary

The anaerobic threshold consists of a lactate threshold and a ventilatory threshold. In some conditions there may actually be 2 ventilatory thresholds.

Much of the work detailing the lactate threshold is strongly based on blood lactate concentration. Since, in most cases, blood lactate concentration does not reflect production in active skeletal muscle, inferences about the metabolic state of contracting muscle will not be valid based only on blood lactate concentration measurements.

Numerous possible mechanisms may be postulated as generating a lactate threshold. However, it is very difficult to design a study to influence only one variable. One may ask, does reducing $F_{O_2}$ cause an earlier occurrence of a lactate threshold during progressive exercise by reducing oxygen availability at the mitochondria? By stimulating catecholamine production? By shifting more blood flow away from tissues which remove lactate from the blood? Or by some other mechanism? Processes considered essential to the generation of a lactate threshold include: (a) substrate utilisation in which the ability of contracting muscle cells to oxidise fats reaches maximal power at lactate threshold; and (b) catecholaminergic stimulation, for without the presence of catecholamines it appears a lactate threshold cannot be generated. Other mechanisms discussed which probably en-
hance the lactate threshold, but are not considered essential initiators are: (a) oxygen limitation; (b) motor unit recruitment order; (c) lactate removal; (d) muscle temperature receptors; (e) metabolic stimulation; and (f) a threshold of lactate efflux.

Some mechanisms reviewed which may induce or contribute to a ventilatory threshold are the effects of: (a) the carotid bodies; (b) respiratory mechanics; (c) temperature; and (d) skeletal muscle receptors. It is not yet possible to determine the hierarchy of effects essential for generating a ventilatory threshold. This may indicate that the central nervous system integrates a broad range of input signals in order to generate a non-linear increase in ventilation.

Evidence indicates that the occurrence of the lactate threshold and the ventilatory threshold may be dissociated; sometimes the occurrence of the lactate threshold significantly precedes the ventilatory threshold and at other times the ventilatory threshold significantly precedes the lactate threshold. It is concluded that the 2 thresholds are not subserved by the same mechanism.

1. Definition and Diagrammatic Description of the Anaerobic Threshold

The phenomenon of the anaerobic threshold is a controversial issue. The conflict ranges from the possible mechanisms explaining the phenomenon, to the validity and reliability of measurements of it, and to the clinical usefulness of a physiological concept that is not particularly well understood.

It is very difficult, perhaps naive, to define a concept that is not understood. The term 'anaerobic' (see section 5.1) and even the term 'threshold' (Hughson et al. 1987; Yeh et al. 1983) have both come under attack in the recent literature. In order to establish a starting point, anaerobic threshold is defined in this paper as a composite of 2 individual thresholds. During incremental non-steady-state exercise a point is reached at which a subject's ventilation shows a non-linear increase and this is termed the 'ventilatory' threshold. Supposedly coincident with the ventilatory threshold is its hypothesised causative factor, a non-linear increase in blood lactate concentration termed the 'lactate' threshold. A serious problem confounding any attempt to define and describe the anaerobic threshold is the fact that there are sometimes 2 of them: both of which have variously been called the anaerobic threshold in the literature. Figure 1 illustrates, diagrammatically, variations in respiratory parameters that occur with different testing protocols. The anaerobic threshold is indicated by the vertically dashed line. As the time interval between work increments shortens (graphs on the right of figure 1), differential information is obtained supporting the hypothesis of Whipp et al. (1981) that a smooth ramp protocol yields the most information about physiological cardiorespiratory changes occurring during incremental work tasks. The graphs on the right clearly indicate a second threshold. Some authors have discussed the nature of the double threshold. Figure 2 delineates 2 thresholds and 3 associated phases. Skinner and McLellan (1980) hypothesise that: phase I is primarily an aerobic event; the first threshold is termed the aerobic threshold; phase II encompasses an aerobic to anaerobic transition, the second threshold is termed the anaerobic threshold; and phase III is primarily anaerobic. Authors who have identified only 1 threshold, or breakpoint, usually call it the respiratory anaerobic threshold (Palka & Rogozinski 1986) or the ventilatory anaerobic threshold (Reybrouck et al. 1985), although it is not always clear which breakpoint they are referring to. To add further confusion, other investigators have referred to the second threshold as the respiratory compensation threshold (Simon et al. 1983) or the threshold of decompensated metabolic acidosis (Reinhard et al. 1979).

For the sake of clarity in the present discussion, we will identify the thresholds by the parameters used to measure them (Bhambhani & Singh 1985;