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Name of Article: Anaerobic Threshold

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The term “anaerobic threshold” has long been used synonymously with “lactate threshold,” the blood lactate inflection point. This terminology is a source of argument, as the onset of blood lactate accumulation offers no information about anaerobic metabolism. Early models proposed linkages between “muscle oxygen insufficiency, lactate production, and changes in pulmonary ventilation”. These causal linkages were attractive to researchers as lactate threshold could be estimated by observing ventilatory threshold, the point at which pulmonary ventilation and carbon dioxide output begin to increase exponentially. Unfortunately, researchers later discovered that factors such as nutritional status, body mass, mode of exercise, and speed of movement all can affect anaerobic threshold determination.

Studies on patients with McArdle’s syndrome place additional doubt on the AT-VT-LT relationship. McArdle’s syndrome is a disorder where the sufferer lacks the enzyme phosphorylase, rendering them incapable of breaking down glycogen to form lactic acid. Even though McArdle’s syndrome patients are incapable of producing lactic acid, they still demonstrate ventilatory threshold during graded exercise tests. Ventilatory threshold occurred at the usual place on the lactate-workload curve in the absence of blood lactate accumulation. In studies using healthy young male subjects, nutritional status was found to affect the relationship between LT and VT. When glycogen was depleted in the subjects, VT occurred at a lower power output, while LT occurred at higher workload, as compared to GXT results for well fed subjects. Other studies have also found dissociation of the LT-VT relationship following an endurance training protocol.

Researchers sought an explanation as to why LT and VT sometimes occur simultaneously, even though blood lactate accumulation is not necessarily attributed to a lack of oxygen. As exercise intensity increases, fast-twitch muscle fibers are recruited, producing lactic acid regardless of whether or not oxygen is present. Breathing rate during exercise is predominantly controlled by neural factors, mainly the carotid and aortic bodies, which are sensitive to the partial pressure of oxygen, and the hydrogen ion concentration. Peripheral factors such as the pH of the arterial blood and cerebrospinal fluid are of lesser importance. Arterial blood pH is affected both by blood lactate level and venous delivery of carbon dioxide to the lungs. While these factors may cause lactate threshold and ventilatory threshold to occur simultaneously, it does not necessarily indicate that the working muscles lacked oxygen.

Even though lactate threshold and ventilatory threshold occur simultaneously in some instances, it is inappropriate to classify their occurrences as an Anaerobic Threshold. “Redistribution of blood flow from lactate-removing, gluconeogenic tissues to lactate producing, glycolytic tissues”, can cause lactate levels to rise regardless of whether or not the muscles are oxygen deficient. Lactate levels rise simply because production and release of lactate occurs at a rate faster than

removal mechanisms can accommodate. For these reasons, lactate threshold can be accurately determined only by sampling blood during a graded exercise test.

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