THE SLOW COMPONENT OF OXYGEN UPTAKE AND THE MEAN POWER FREQUENCY OF EMG DURING HEAW INTENSITY EXERCISE

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The purpose of this study was to elucidate the mechanisms causing the slow component of oxygen uptake ($\dot{V}O_2$), by investigating changes in exercising muscle electrical activity. Volunteers performed square-wave dynamic knee extension exercise. Surface EMG of the rectus femoris was used to measure mean power frequency (MPF) and integrated (IEMG) values for each contraction. $\dot{V}O_2$ was measured breath-by-breath using a mass spectrometer. During 'heavy' intensity exercise eliciting a $\dot{V}O_2$ slow component and muscle fatigue, there was no decrease in MPF, which contrasts with 'severe' exercise where the MPF decreased as muscle fatigue occurred. Results from this study suggest a change in fibre recruitment from slow-twitch to oxygen-inefficient high-frequency fast-twitch fibres during fatiguing exercise as the cause of the $\dot{V}O_2$ slow component.

KEY WORDS: electromyography, frequency analysis, oxygen uptake, slow component.

INTRODUCTION: An important limiting factor for athletic performance is maximal oxygen uptake ($VO_{2\,max}$) as exercise above this level can only be sustained for a short period before fatigue occurs. During exercise at work rates below the lactate threshold (θ_L) oxygen uptake ($\dot{V}O_2$) is characterised by a monoexponential function, with a steady-state achieved. At work intensities above θ_L, $\dot{V}O_2$ is greater than that predicted from the linear $\dot{V}O_2$-work rate relationship found below θ_L. A slow component of $\dot{V}O_2$ is superimposed on the monoexponential function, which leads to a delayed steady-state or a continuous increase in vo, (Whipp and Wasserman, 1972).

There are several proposed sources of this oxygen 'excess', including increased use of non-exercising muscle, and changes in exercising muscle efficiency due to changes in fibre-type, changes in temperature, or local metabolite accumulation (Poole et al., 1994). Several studies have investigated and shown support for the fibre recruitment model using various strategies (for example, Barstow et al., 1996). One study used electromyography (EMG) as the investigative tool (Scheuermann et al., 1998). However, the frequency characteristics of the EMG signal were not considered. The current paper is the first to combine EMG frequency analysis and $\dot{V}O_2$ measurements in specified intensity domains.

The purpose of this study is to elucidate the mechanisms causing the slow component of oxygen uptake by investigating changes in mean power frequency (MPF) of exercising muscle. This study thereby intends to contribute to our understanding of muscle energetics and the limits to athletic performance.

METHODS: Dynamic knee extension exercise with constant deflection and contraction frequency was performed by six healthy young males seated on a modified standard exercise bench. For all tests, the subject breathed at constant frequency through a mouthpiece attached to a turbine and mass spectrometer (QP9000, CaSE, Biggin Hill, UK), to measure volume and O_2 concentrations respectively, for subsequent calculation of pulmonary gas exchange variables. Bilateral surface electrodes were placed on the rectus femoris for measurement of electrical activity and subsequent contraction-by-contraction calculation of integrated and frequency variables (MT8, MIE Medical Research Ltd., Leeds, UK). Data were collected at 1000Hz, with a 20Hz high pass filter.

The first test was an incremental test to the limit of tolerance for estimation of θ_L, $VO_{2\,max}$ the difference between $VO_{2\,max}$ and $\dot{V}O_2$ at θ_L (A). Each subject performed three square wave tests at 'moderate' (30% θ_L), 'heavy' (25%Δ above θ_L), and 'severe' (75%Δ above θ_L)

intensities. Moderate exercise lasted 10 minutes. Heavy and severe exercise continued to volitional exhaustion. Each test was performed on a different day and at the same time of day.

RESULTS: It was found that $\dot{V}O_2$ profiles for each exercise intensity were similar to those previously described for cycle ergometry and consistent within and between subjects, with a steady state achieved for moderate exercise, a slow component for heavy (Figure 1), and a rapid increase to $\dot{V}O_2$ for severe exercise.

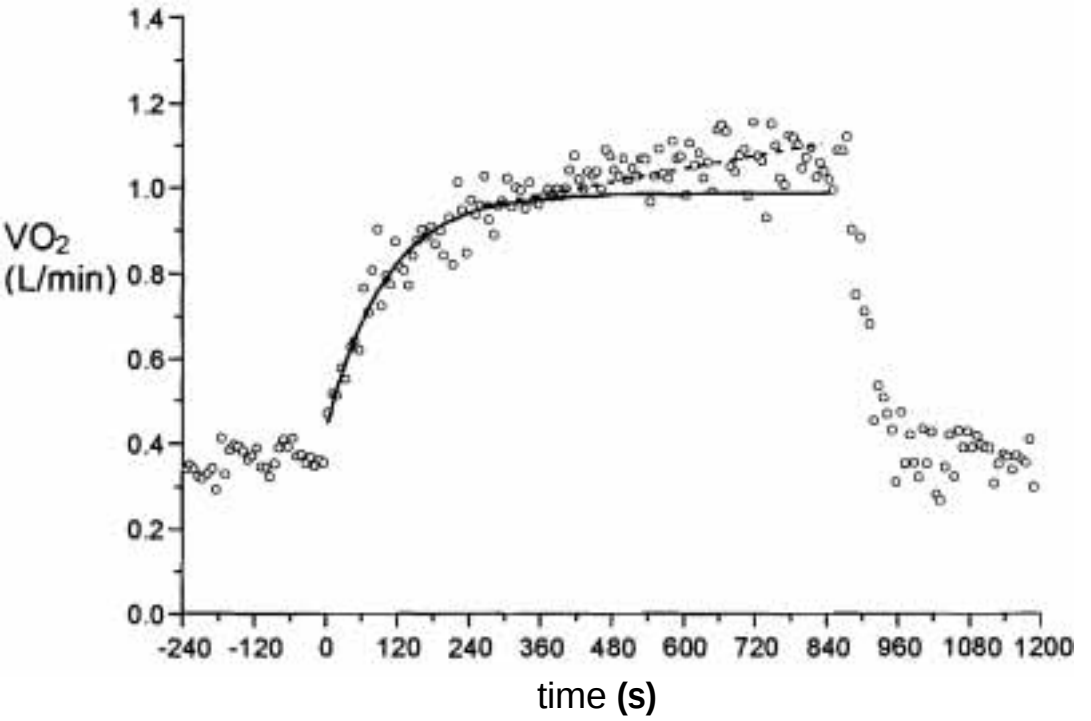

Figure - 1 Breath-by-breath VO_2 profile for one trial in the heavy domain. This is typical for the heavy domain both in these experiments and previously published work. A monoexponential function is fitted (solid line), and the oxygen "excess" (the slow component) is shown (dashed line).

For the EMG variables in the moderate (non-fatiguing) and severe (rapidly fatiguing) domains, and the integrated EMG in the heavy (fatiguing) domain, patterns agree with previous work. For each test in the heavy domain, MPF at t=60s, t=180s, t=360s, and t=720s was calculated and normalised to the value at 60s. The mean and standard deviation of the MPF of all the tests were calculated, and are shown in Figure 2. The dashed line indicates a continuous decrease in MPF, which is expected for fatiguing exercise. At approximately 360s, the data deflect from the dashed line, and an increase in MPF is evident at 720s.

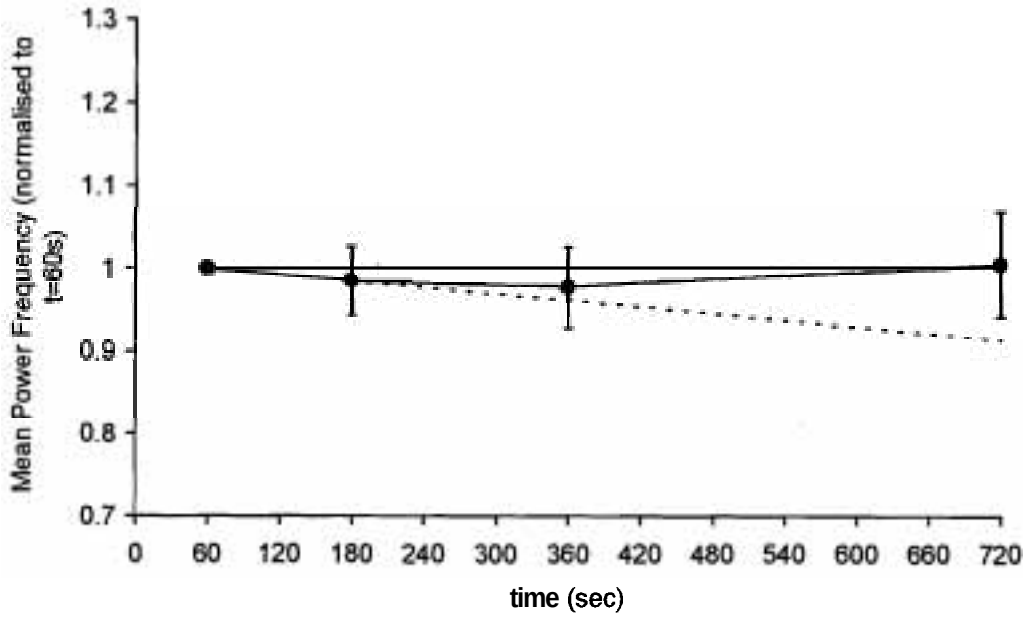

Figure 2 - Mean and standard deviation of MPF for all experiments in the heavy domain **(n=36)** at **t=60s, t=180s, t=360s,** and **t=720s.**

DISCUSSION: In the severe domain, most muscle fibres are active at the start of exercise to produce the large forces required. There is a rapid production of hydrogen, and efflux of potassium ions throughout the exercise. This impairs the flux of sodium and potassium ions across the muscle membrane, which is responsible for the muscle fibre action potential (AP) (Milner-Brown and Miller, 1986). The AP conduction velocity (CV) decreases, and causes a decrease in the MPF. Very few, if any fresh fibres are available for recruitment, so to maintain the same work-rate the already activated muscle fibres have to work harder. This is reflected in the increase in IEMG throughout severe exercise bouts.

In the heavy domain, fewer fibres are required at the start of the bout to carry out the task than in the severe domain and the fibres recruited are predominantly small diameter slow-twitch fibres. As the exercise progresses and fatigue continues, more and more large diameter fast-twitch fibres are recruited. MPF is dependent on CV, which in turn is influenced by fibre diameter - the larger the diameter, the faster the CV (Kupa ef *al.*, 1995). Here, two factors influence the MPF and they work in opposite directions: (i) cation imbalances causing the MPF to decrease, which occurs in both severe and heavy domains, and (ii) an increase in the average diameter of the population of fibres producing the force, causing the MPF to increase. The latter occurs only in the longer-duration heavy exercise, which is evident in Figure 2.

This argument can also be applied to explain the $\dot{V}O_2$ kinetics in heavy exercise. Late recruitment of fast-twitch fibres, which are less oxygen-efficient than slow-twitch fibres, can account for the $\dot{V}O_2$ slow component.

CONCLUSION: In this study we have focused on the changes in EMG MPF during heavy intensity exercise. A delayed upward deflection of the MPF from an initial downward trend was observed, which implies a change in recruitment from slow- to fast-twitch muscle fibres. This observation has interesting implications for studies of muscle energetics and $\dot{V}O_2$ kinetics.

These results support the view that a change in fibre-type recruitment during fatiguing exercise can account for the $\dot{V}O_2$ slow component. An understanding of the performance-limiting effects of delayed fast-twitch recruitment is necessary for devising training and competition strategies for athletes.

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