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Endurance Training Of Trained Athletes- An Electromyogram Study

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Abstract— **Little is known about the mechanism that improves the capabilities of athletes by high, intensity interval training (HIT). This study was conducted to determine the neurophysiological changes due to HIT. Changes in surface electromyography (SEMG) in well-trained endurance cyclists due to the training were identified. Seven subjects (maximal oxygen uptake [VO2max] 64.6 ± 4.8 ml.kg-1.min-1, mean ± SD) undertook a 3 week training intervention, replacing ~ 15% of their weekly endurance training with 6 sessions of laboratory-based HIT (8 x 5 min work bouts at 82% of PPO [~85% VO2max], with 60 sec active recovery at 100 W). SEMG was used to assess neuromuscular changes before and after the 3 wk training program. During the first and sixth training session, SEMG was recorded. To determine the effects of the HIT program on performance, subjects performed a 40 km time trial (TT40) before and after the training intervention. The frequency of SEMG is a measure of the muscle fatigue and hence was used to identify the variation of the signal properties. Three weeks of intensified training decreased the mean power frequency of the SEMG signal during the latter stages of HIT (interval seven) 50.2 ± 5.1 to 47.5 ± 4.2 Hz (P<0.05). The preliminary conclusions of these experiments suggest that high-intensity interval training enhanced endurance performance and reduced the fatiguing of the muscles. It is suggested that this was possibly due to recruitment of addition slow-twitch motor units.**

I. INTRODUCTION

THE effect of endurance training on sedentary individuals is pronounced and the mechanism is relatively well understood. When already well-trained athletes undertake endurance training, there is a subtle change in their abilities and improvement of their athletic performance is less well-defined. Well-trained endurance athletes are rarely able to enhance endurance performance by increasing the volume of training. Laursen and Jenkins [11] have suggested the use of high-intensity interval training (HIT), in conjunction with endurance training for improving the performance of these athletes.

Previous studies of the effect of short-term (~ 3 wk) intensified training have reported improvements in athletic performance [4, 5, 8, 10], accompanied by shifts in substrate metabolism (from carbohydrate to fat). There have been mixed responses of enzyme activity after intensified training [1, 4, 7].

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However, intensified interval training has been reported to increase in vivo skeletal muscle buffering capacity. The mechanisms to explain why high-intensity interval training (HIT) improves endurance performance in already well-trained athletes are inconclusive.

Previous studies have examined metabolic, ionic and respiratory parameters in an attempt to correlate changes in these variables with changes in performance parameters. However, they cannot fully explain the reason why performance is enhanced.

One mechanism that has been proposed to explain improvements in endurance performance of athletes is a change in neuromuscular recruitment patterns [5, 12]. But there appears to be little known about the neuromuscular response to HIT and hence it is not possible to understand its role in enhancing performance in response to HIT. De Luca et al. [6] and Lucia et al [2] observed that during contraction, there appears to be a "functional reserve" of motor units. These motor units are not readily available but can form part of the increased motor unit activity after training and hypothesised that part of the training–induced adaptation is that the athlete is able to "learn" to fully activate some of the motor units that were not previously active. These results suggest that one adaptation to strenuous endurance training in elite endurance athletes is possibly the ability to recruit an additional number of motor units in the working muscles (i.e., vastus lateralis).

Currently the most suitable technique to measure muscle recruitment patterns is Surface Electromyography (SEMG). It has also been proposed that the SEMG frequency spectrum would be affected by morphological differences in the fibre type and size, via differences in conduction velocities [9]. The spectrum compression of SEMG is also a measure of the onset of muscle fatigue due to change in recruitment pattern. The enhancement of the neuromotor system would result in less pronounced reduction of the spectrum. However, SEMG recordings with elite athletes are scarce because of the logistics difficulty. As a result, there is a lack of understanding of the process by which the athletes improve their capacity due to HIT. An improved understanding of the mechanism will dispel doubts of the effectiveness of HIT and will also help better design the training method for the athletes. This paper reports the research aimed at determining the effect of HIT on motor recruitment by measuring changes in SEMG among elite athletes (cyclists).

II. METHODOLOGY

Seven highly trained cyclists or triathletes who had been riding 300 □ 30 km.wk-1 (mean □SD) and who had not undertaken any high-intensity interval training for at

least 6 weeks prior to the investigation, participated in this study. Their mean details are; age 30 years, height 177.8 cm, body mass 75.4 Kg, sum of 7 skin folds 75.2 mm, maximal oxygen uptake (VO2max) 64.6 ml kg-1 min-1 and peak power output (PPO) of 370.9. This study was conducted in accordance with the guidelines of the Human Research Ethics Committee of RMIT University.

Subjects underwent preliminary testing and then undertook a 3 wk training intervention that required subjects to replace ~ 15% of their weekly training with 6 sessions of HIT. After this they were tested again. This was done to ensure familiarity with equipment and testing procedures and to determine stability of their performance. On their first visit to the laboratory, each subject was weighed and underwent anthropometric measures for the sum of seven skinfolds (biceps, tricep, subscapular, abdominal, supraspinae, mid-thigh and medial gastrocnemius). Subjects were provided with a standard diet for 36 hr before the maximal test consisting of 50 kcal. kg^{-1} BM, composed of 63% carbohydrate (8 g . kg^{-1} BM), 20% fat, and 17% protein. During all exercise testing sessions heart rate (HR) was monitored using a Polar Accurex Plus (Polar Electro OY, Kemple, Finland). Each interval session was conducted under standard lab conditions (i.e. 50% relative humidity, 20-22°C) with subjects cooled using a fan (wind speed of ~7 $m.s^{-1}$) and provided with water *ad libitum* throughout exercise.

Subjects performed a cycling test on an electronically braked cycle ergometer (Lode Groningen), set up for each individual requirement and recorded for repeatability. Each cycle consisted of a the pedal going through 360°, or, for the pedal of one foot to start and return back to 12 O'clock position. The test commenced at a work rate equivalent to 3.33 $W.kg^{-1}$ body mass for 150 sec and increased by 50 W for another 150 sec. After the second stage, the exercise intensity was increased by 25 W every 150 sec until the subject fatigued. Fatigue was defined when the cyclist could no longer maintain a cadence above 60-70 rpm. Data obtained from the maximal test was used to determine the power output corresponding to ~80% of PPO (~85% VO_{2max}) which was the workload for the HIT sessions.

SEMG signals were recorded from the belly of the vastus lateralis (VL) and vastus medialis (VM) using a pair of active surface electrodes (Ag/AgCl, Meditrace, 200-30) using a bipolar configuration, along the longitudinal lines of the muscle fibres. The VL electrodes were placed 4 cm apart at the mid-point between the head of the greater trochanter and the lateral condyle of the femur. The VM electrodes were placed 2 cm apart at ~ 55°, 2 cm medially from the superior border of the patella. A reference electrode was placed over the iliac crest. All sites were shaved, abraded and cleaned with alcohol in order to keep inter-electrode impedance below 1,000Ω. Recording electrodes and cables were firmly taped to the skin. SEMG was recorded using an AMLAB EMG amplifier (AMLAB Technologies, Lewisham, Australia)

A. Data Analysis

Small sections of the raw EMG were extracted for analysis during the first and last HIT sessions. For frequency analysis small slices of data were chosen manually from the middle of the cycle for a length of 100 ms (Figure 1). The small sections were chosen to avoid sections of the cycle where the muscles under investigation are not (or minimally) active and isolates identical phases of each cycle. The data was taken during the later stages of the first interval (after ~ 4 min and 50 sec) and at the same time during the seventh interval. For each dynamic segment of SEMG data, the power spectral density (PSD), root mean square (RMS) for two cycles and the mean power frequency (MPF) of the PSD calculated (Figure 1). All data was analysed using MATLAB™.

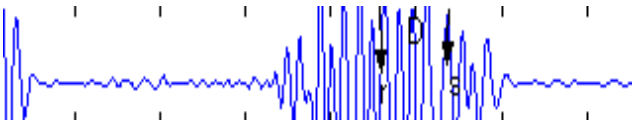

Fig 1 Segmentation of SEMG

III. RESULTS AND OBSERVATIONS

It can be observed (figure 2) that there was no significant variation of the magnitude of SEMG in response to HIT. The variation between RMS of SEMG was less between HIT session one to HIT session six at the start and end of the exercise- for both, interval one (25.6 ± 10.1 to 20.9 ± 7.9 mV) and interval seven (21.9 ± 8.0 to 20.0 ± 8.3 mV).

It is also observed (Figure 4) that there was a significant drop in MPF from the training session one to training session six during interval seven (50.2 ± 5.1 to 47.5 ± 4.2 Hz, $P<0.05$). There were no significant changes in MPF determined in workout one (52.9 ± 5.6 to 51.3 ± 4.3 Hz).

Figure 4 show a significant reduction in MPF between intervals one and seven during the first training session (52.9 ± 5.6 to 50.2 ± 5.1 Hz) and the last training session (51.3 ± 4.3 to 47.5 ± 4.2 Hz), $P<0.05$. From Figure 3, it is also observed that there is a significant reduction in overall MPF after HIT.

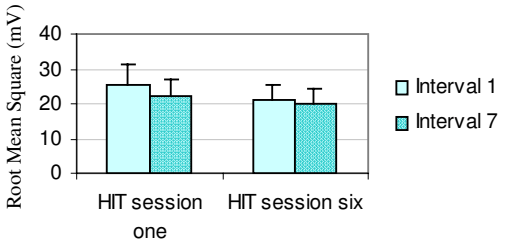

Fig 2. Changes in RMS from interval one to seven, before and after the 3 wk training intervention

Based on these observations, it can be stated that there is a change in the neuromotor mechanism of the athlete due to HIT. While there is no observable change in the magnitude, there is a marked change in the spectral content of SEMG which suggests. This suggests that before the

HIT, different motor units were getting recruited simultaneously near the end of the exercise; the athlete was able to maintain the normal recruitment pattern after the training. The authors are unable to confirm the reason for this change and maybe attributable to change in the morphology of the muscle and

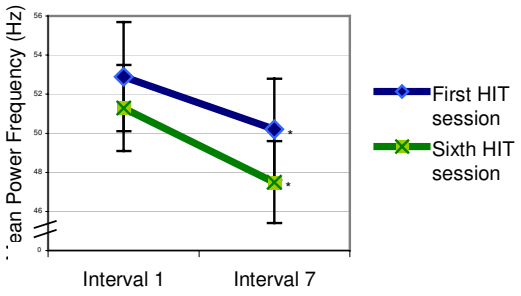

Figure 3 Mean power frequency (MPF) from interval one to interval seven in training sessions one and six Values are mean ± SD. $P<0.05$

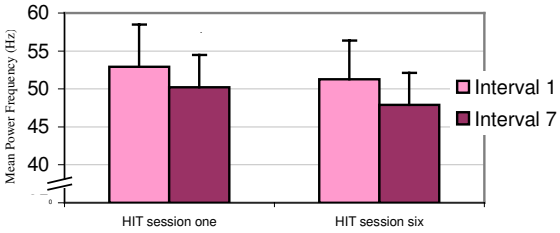

Fig 4. Changes in Mean Power Frequency from interval one to seven, before and after the 3 wk training intervention

suggest that more experiments have to be conducted to be able to determine the cause.

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