

# Effects of supramaximal exercise on the electromyographic signal

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*Br J Sports Med* 2003;37:296–299

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Accepted 8 July 2002

**Aim:** To determine the neuromuscular recruitment characteristics during supramaximal exercise.

**Methods:** Ten healthy subjects completed the Wingate anaerobic test (WAT) cycling protocol. Electromyographic (EMG) data and rate of fatigue were recorded throughout the cycling.

**Results:** The mean (SD) rate of fatigue (decrease in power output) was 44.5 (8.6)%. No significant change was found in EMG amplitude. A significant decrease ( $p < 0.01$ ) in mean power frequency spectrum was found over the 30 second period.

**Conclusions:** During WAT, mean power frequency spectrum was attenuated with no decline in EMG amplitude, which may be caused by an accumulation of metabolites in the periphery. However, it is also possible that the feedback loop from intramuscular metabolism to the central nervous system is unable, within the 30 second period of the WAT, to affect neural recruitment strategy.

The Wingate anaerobic test (WAT) has been used extensively by researchers and sports practitioners to examine maximal power output and as a standard exercise task to analyse athletes' responses to supramaximal exercise.<sup>1</sup> The WAT was originally based on a 30 second cycling test described by Cumming.<sup>2</sup> The 30 second period was considered suitable for stressing the glycolytic pathway.<sup>3</sup> This period was chosen because subjects were able to maintain an all out effort for 30 seconds, unlike tests of 45 and 60 seconds, where subjects repeatedly moderated their starts for fear of not finishing.<sup>1</sup>

The WAT has been described as an exercise test that will provide an index of a subject's anaerobic capacity.<sup>4</sup> Hussain *et al*<sup>5</sup> showed a significant decrease in pH from baseline levels immediately after the WAT. A lowered pH has often been associated with severe symptoms of fatigue.<sup>6,7</sup> However, it is not clear how these changes in pH affect electromyographic (EMG) amplitude, the frequency spectrum, and the relation between them during the WAT.

Maximal power is a function of instantaneous force and velocity.<sup>1</sup> During fatiguing maximal cycling, the phasic acceleration and deceleration of the lower limb muscles limits the period for which the muscle can maintain an optimal velocity.<sup>8</sup> The only study to have examined neuromuscular fatigue during the WAT was that of Vandewalle *et al*,<sup>9</sup> who concluded that the observed decline in EMG amplitude was due to the occurrence of central fatigue. However, this study is limited for two reasons. Firstly, the WAT protocol used meant that the test was not supramaximal; as mentioned previously, subjects will pace themselves to successfully complete a 45 second WAT as opposed to the all out effort seen during a 30 second WAT.<sup>1</sup> Secondly, EMG amplitude was measured without EMG spectrum analyses; therefore, conclusions about whether a task is central or peripheral are speculative. Other studies have shown a decline in EMG spectrum during maximal running sprint activities<sup>10</sup>; however, the relevance to WAT can be questioned because of the different muscle groups used and the biomechanical action involved. Green<sup>11</sup> proposed that only incremental supramaximal motorised treadmill tests and the WAT enable alactic work capacity to be determined and provide more information on performance ability than constant load tests. However, in a method using placing of an EMG electrode on a working muscle, the technique required to perform the task and the variability between subjects have to be considered. Clearly, the cycling action required to perform

the WAT requires far less technique than running, therefore less variability, resulting in a more reliable, comparable EMG signal.

To our knowledge, the EMG signal during a 30 second WAT has not been examined. Therefore we recorded changes in integrated EMG (IEMG) and mean power frequency spectrum (MPFS) while 10 subjects completed the WAT.

## METHODS

Ten healthy men volunteered for this study. The mean (SD) age and body mass of the subjects were 21.4 (2.6) years and 72.3 (8) kg respectively. All subjects were physically active and each signed an informed consent before the study. The research and ethics committee of the University of Cape Town Faculty of Health Sciences approved the study.

### Maximal isometric voluntary contraction (MVC)

To normalise the EMG signal, the MVCs of the subject's right knee extensor muscles were measured on an isokinetic dynamometer (Kin-Com, Chattanooga Group Inc, Hixson, Texas, USA). Subjects sat on the dynamometer, and their hips, thighs, and upper bodies were firmly strapped to the seat. In this position their hip angle was at 100° flexion. The right lower leg was then attached to the arm of the dynamometer at a level slightly above the lateral malleolus, and the axis of rotation of the arm was aligned with the lateral femoral condyle. The arm of the dynamometer was then set so that the knee was at a 60° angle from full leg extension. Each subject performed four submaximal familiarisation contractions before performing two maximal MVCs, the latter of which were used for all subsequent analyses. All subjects were verbally encouraged to exert maximal effort during both MVCs.

### Experimental design

After performing the MVCs, the subjects warmed up with light cycling and stretching. The WAT consisted of one 30 second sprint, performed on an electrically braked cycle

**Abbreviations:** WAT, Wingate anaerobic test; EMG, electromyographic; IEMG, integrated EMG; MPFS, mean power frequency spectrum; MVC, maximal isometric voluntary contraction

**Table 1** Fatigue index for each subject throughout the 30 second Wingate anaerobic test

Subject	Fatigue index (%)
1	53.8
2	34.0
3	38.0
4	49.8
5	45.8
6	50.7
7	51.7
8	32.1
Mean (SD)	44.5 (8.6)

ergometer (Watsystem; ITC Corp, Bloomington, IN, USA) with a preset load of 0.09 kg/body weight.<sup>12</sup> The subjects were given five seconds of loadless pedalling to reach maximum pedal speed and were subsequently instructed to sustain maximal cadence during the 30 seconds once the correct load was applied. All subjects received verbal encouragement throughout the 30 seconds.

The rate of fatigue during the WAT was calculated by using the following equation as described previously by McCartney *et al*<sup>8</sup>:

$$\text{Fatigue index (\%)} = ((\text{peak power} - \text{lowest power}) / \text{peak power}) \times 100$$

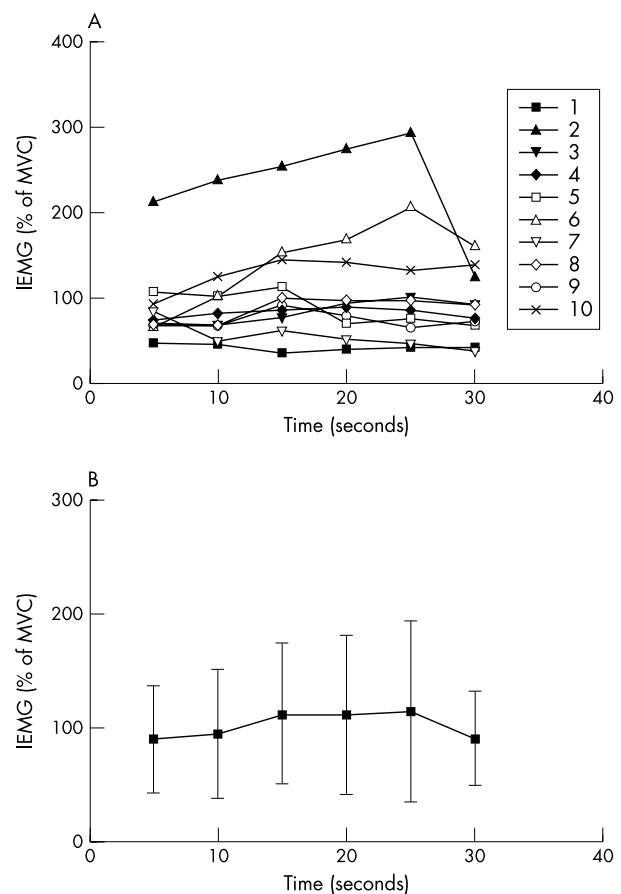
where power is measured in W.

### EMG testing

Before maximal isometric strength testing on the Kin-Com isokinetic dynamometer, EMG electrodes were attached to the subject's lower limb midway between the superior surface of the patella and the anterior superior iliac crest of the belly of the rectus femoris. The overlying skin on the muscles was carefully prepared. Hair was shaved off, the outer layer of epidermal cells abraded, and oil and dirt were removed from the skin with an alcohol pad. Triode electrodes (Thought Technology, Montreal, Quebec, Canada) were placed on the muscle sites as described above, and linked via a fibre-optic cable to the Flexcomp/DSP EMG apparatus (Thought Technology) and host computer. The electrodes were heavily taped down with cotton swabs to minimise sweat induced interference. The EMG data were filtered with a 50 Hz line filter to prevent electrical interference from electrical sources and was automatically anti-aliased by the hardware (Thought Technology). Each activity was sampled at a 1984 Hz capture rate for five second bouts. Recordings were taken on the second maximal isometric trial and during the cycling trial at 5, 10, 15, 20, 25, and 30 seconds, yielding a raw signal. MVC EMG data were recorded before the cycle ride to ensure normalisation of EMG in the trial. The raw data were divided into seven five second epochs. The first epoch included all data collected during the second MVC trial, and the remaining six epochs included data collected on the ride at 5, 10, 15, 20, 25, and 30 seconds.

The EMG signals were full wave rectified, and movement artifacts removed using a high pass, second order Butterworth filter with a cut off frequency of 15 Hz, then smoothed with a low pass, second order filter with a cut off frequency of 5 Hz. This was performed using MATLAB gait analyses software. These integrated data (IEMG) were used for subsequent analyses.

The spectrum of the frequency for each epoch of data collected during the cycle ride was assessed using the raw EMG data by using a fast Fourier transformation algorithm. The analyses for frequency spectrum were restricted to frequencies in the 5–500 Hz range, because the EMG signal



**Figure 1** Total electromyographic amplitude (IEMG) as a percentage of maximal isometric voluntary contraction (MVC) at five second intervals for individual subjects (A) and as a group (B) over a 30 second Wingate anaerobic test.

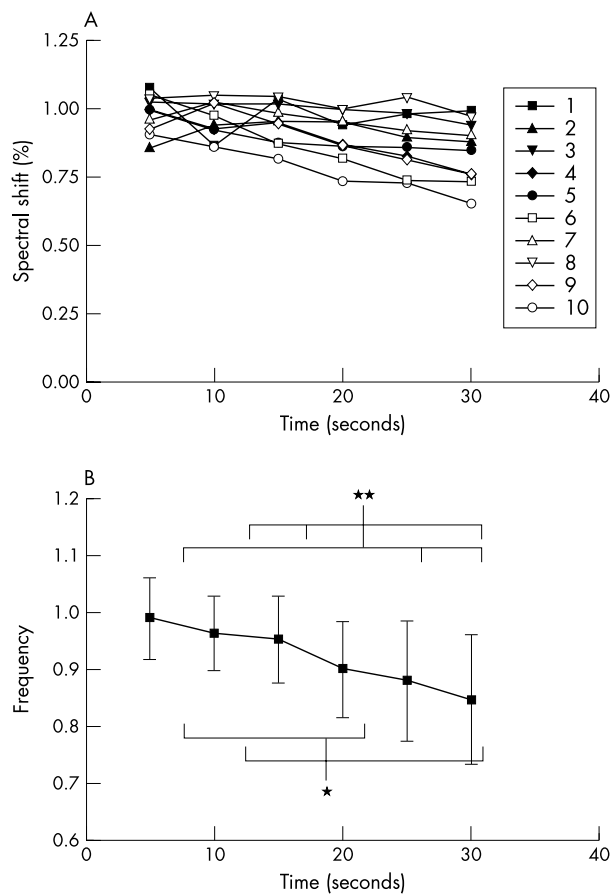
content consists mostly of noise when it is outside of this bandwidth. The frequency spectrum from each epoch of data was compared with that derived from the MVCs, and the amount of spectral compression was estimated. This technique was performed as described by Lowery *et al*,<sup>13</sup> which is a modification of the work of Lo Conte and Merletti<sup>14</sup> and Merletti and Lo Conte.<sup>15</sup> The spectrum of the raw signal of each epoch was obtained and the normalised cumulative power at each frequency was calculated for each epoch. The shift in centile frequency was then examined—that is, at 0%...50%...100% of the total cumulative. The centile shift was then estimated by calculating the mean shift in all centile frequencies throughout the mid-frequency range (5–500 Hz). This method has been suggested as a more accurate estimate of spectral compression than median frequency analyses, which uses a single value of (50th) centile frequency.<sup>13</sup> This change in MPFS data was used for subsequent analyses.

### Statistical analysis

Data are presented as mean (SD). Significant differences over time were assessed using a one way analysis of variance with repeated measures. Where significant changes occurred a Scheffe's post hoc test was applied to identify individual differences over time. Relations among variables were assessed using the Pearson correlation coefficient. Significance was accepted at  $p < 0.05$ .

### RESULTS

The mean (SD) rate of fatigue calculated from the decrease in power during WAT was 44.5 (8.6)% (table 1). Data from subjects



**Figure 2** Mean power frequency spectrum at five second intervals during the Wingate anaerobic test for individual subjects (A) and as a group (B). There was a highly significant ( $p < 0.01$ ) reduction over 30 seconds. \*\* $p < 0.01$ : 5 seconds v 25 seconds; 5 seconds v 30 seconds; 10 seconds v 30 seconds; 15 seconds v 30 seconds. \* $p < 0.05$ : 5 seconds v 20 seconds; 30 seconds v 10 seconds.

9 and 10 could not be recorded because of technical difficulties with the cycle ergometer.

IEMG normalised to MVC showed a large individual variation, with one subject showing a drop off in IEMG in the final five seconds (fig 1A), although, as a group, there was no significant change in IEMG during WAT (fig 1B). However, MPFS normalised to MVC was significantly reduced by 14.7% over the 30 second WAT ( $p < 0.01$ ) (fig 2B) and the individual variation was far less than for IEMG (fig 2A). A correlation of  $r = 0.56$  (NS) was found between fatigue index (%) and MPFS (%) decline during WAT.

## DISCUSSION

The main finding in this study was the significant drop off in MPFS coupled with a relatively unchanging IEMG despite a mean fatigue index of 45%.

The decline in force during the WAT was similar to that found previously.<sup>1</sup> The decline in MPFS may be caused by an accumulation of metabolites and a consequent decrease in pH, resulting in the slowing down of the muscle fibre conduction velocity.<sup>16</sup> This decline in MPFS is similar to the results shown in both isometric and isokinetic fatiguing protocols.<sup>17</sup> This occurrence could be as a result of a reduced central drive,<sup>16, 18</sup> but no change in motor unit recruitment.<sup>19</sup> The precise reason for the fall in MPFS in this study remains unclear; however, the 30 second time frame for the feedback loop between central motor drive and intramuscular metabolism at this high level of fatigue may be too short, but, given more time, could have come into effect.

## Take home message

During the Wingate anaerobic test, no change in motor unit recruitment strategy was shown despite the high rate of fatigue and decline in frequency power. We conclude that this was due to the accumulation of metabolites in the periphery and/or an ineffective afferent command to the central nervous system to affect neural recruitment strategies. These neural recruitment strategies usually act as a protective mechanism to slow the body down before damage can occur.

IEMG showed no significant change throughout the WAT. We suggest that this may be caused by minimal afferent signal from the active muscles to influence a reduction in central drive. This being the case, no protective mechanism would be used to initiate changes to adapt to the high level of fatigue occurring. It has been suggested that additional motor units are progressively recruited to compensate for the reduction in contractility due to the impairment of fatigued motor units, resulting in an increasing IEMG.<sup>20</sup> This would also cause a decline in the propagation velocity of the action potentials along the muscle fibres<sup>21, 22</sup> and the recruitment of new, non-fatigued, motor units to replace the fatigued ones.<sup>23</sup> These mechanisms will cause an increase in the duration of the recorded motor unit action potentials resulting in a reduction in MPFS. However, because the larger motor units become recruited, a larger IEMG is usually recorded. In our study, the IEMG showed no significant changes throughout the WAT, agreeing with our suggestion. Furthermore, Degtyarenko *et al*<sup>24</sup> speculated that the central nervous system modulates input from metabolic receptors less effectively than input from mechanical receptors. Therefore it is possible that, during the short (30 second) period of WAT, the afferent command from the metabolic receptors to the central nervous system is insufficient to cause a reduction in central drive to result in an altered IEMG pattern. The other compounding factor may be from mechanical receptors; it could be argued that both the controlled range of movement for the leg and support of body weight during WAT do not allow for any significant stretch of the muscle spindle to affect the mechanical receptor. This could also result in insufficient signalling to the central nervous system to cause a reduction in central drive and IEMG.

Exercise fatigue leads to a decline in neural drive, resulting in a loss of force.<sup>25</sup> However, the present results show no change in IEMG, which does not indicate a decline in central drive. Indeed, Vandewalle *et al*<sup>26</sup> using a 45 second WAT protocol found a decrease in IEMG amplitude. This is in agreement with the findings of Bigland-Ritchie *et al*,<sup>26, 27</sup> Moritani *et al*,<sup>28</sup> and Petrofsky and Lind,<sup>29</sup> who found a decline in IEMG and MPFS throughout MVC. It was concluded that these observations were the result of a decline in central drive to ensure the optimal force output by avoiding peripheral transmission failure.<sup>26, 30, 31</sup> Therefore, during a 30 second WAT, a protective mechanism may not be used.

Interestingly, the individual variation for IEMG was far greater than for MPFS. Subject 2 displayed a far higher overall IEMG (over 200% of MVC) than other subjects. The IEMG in this subject then rapidly declined in the final five seconds, although the fatigue index was one of the lowest. This suggests that the subject may have subconsciously attempted the WAT submaximally as opposed to supramaximally, which could be evidence of a protective mechanism, as described by St Clair Gibson *et al*.<sup>32</sup> In conclusion, during WAT, MPFS was attenuated with no decline in IEMG. The significant reduction in MPFS may have been caused by an accumulation of metabolites in the periphery. However, it is possible that a 30 second period is ineffective for the feedback loop between intramuscular metabolism and the central nervous system to

affect motor unit recruitment strategies. Further investigation is required to measure both metabolite accumulation and changes in neural firing patterns during the WAT.

## ACKNOWLEDGEMENTS

This research was supported by the Harry Crossley Research Fund of the University of Cape Town, and the Medical Research Council of South Africa.

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