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Electromyography Assessment of Muscle Recruitment Strategies During High-Intensity Exercise

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1. Introduction

Electromyography (EMG) is an experimental technique concerned with the recording and analysis of myoelectric signals. Since the EMG signal detected on the surface of the skin directly reflects the recruitment and firing characteristics of the detected motor units within an area, EMG activity can be used to study the neuromuscular activation of muscles within postural tasks, functional movements, work conditions and treatment/training regimes (Basmajian and De Luca 1985). Furthermore, EMG activity has been correlated on multiple occasions with fatigue-related events occurring within the muscle (Bigland-Ritchie et al. 1986a; Bigland-Ritchie and Woods 1984; Moritani et al. 1986; Nordlund et al. 2004). Other chapters in this book describe the use of EMG for various applications. In the exercise sciences, EMG activity is typically used to explore muscle recruitment strategies (*i.e.,* time-dependent, internal, physiological modifications) and, thereby, understand the complex relationship between the development of locomotor muscle fatigue and the cortical regulation of exercise intensity (Billaut et al. 2010; Marino 2004). Neuromuscular fatigue can be induced by sustained muscular contractions. It is essentially accompanied by external manifestations such as the inability to maintain a desired force output, muscular tremor, and localized pain. The effects of this fatigue are localized to the muscle or group of synergistic muscles performing the contraction. According to several authors (Bigland-Ritchie 1984; Fitts 1994; Gandevia 2001; Merton 1954; Szubski et al. 2007), this fatigue may have its source peripherally (within the muscle tissue or neuromuscular junction) and/or centrally (within the brain and spinal cord). During fatigue the EMG activity may display two typical characteristics. The first is a change in amplitude (Figure 1), whereby additional motor units are recruited or already-active motor units are de-recruited. The second characteristic is a shift of the EMG power frequency spectrum (Figure 2), which shows the relative electrical activity contributed by slow (on the left) or fast (on the right) motor units. A leftward shift suggests increased stimulation of smaller, slow, fatigue-resistant motor units.

However, studies have mainly explored constant work-rate tests, along with incremental tests to maximum effort. In these examples, ecological validity has been limited due to work

rate being either fully or partly dictated by the protocol; this excludes the individual subjective assessment of the task. Because performance in competitive events depends largely upon pacing strategies (Figure 3) (Billaut et al. 2011; Hettinga et al. 2006; Palmer et al. 1997; Paterson and Marino 2004; St Clair Gibson et al. 2006), it is necessary to investigate the neuromuscular responses to self-paced exercise to further understand the role of the central nervous system (CNS) in the regulation of exercise performance. Thus, a more realistic paradigm for future research in the exercise sciences is one that would permit the individual to use sensory cues to adjust the effort along with the fatigue process. In fact, Marino and colleagues (2011) recently re-emphasised that bringing the brain (and subsequent muscle recruitment strategies) into modern fatigue research represents the next phase in the unravelling of the fatigue process.

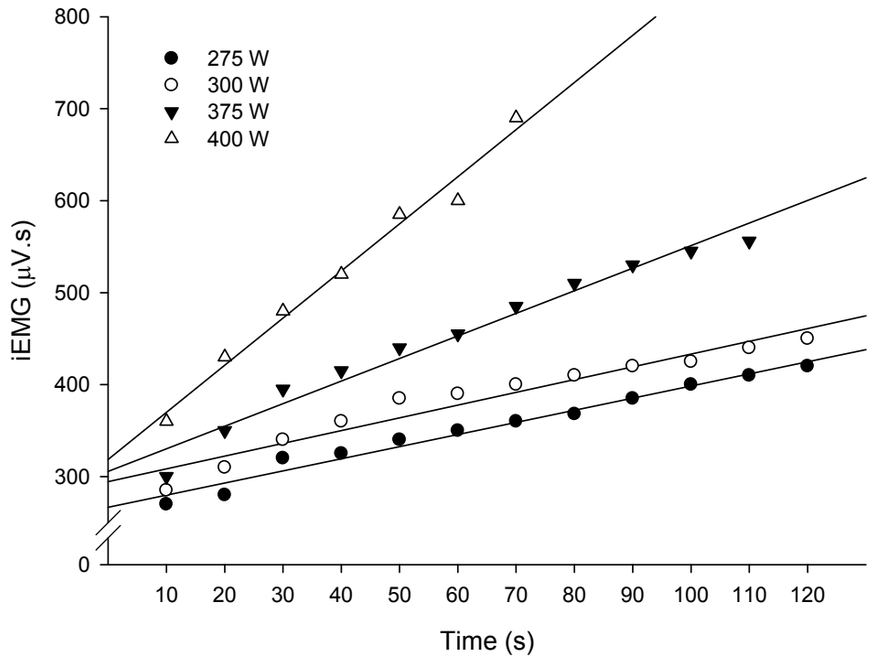

Fig. 1. Typical increase in EMG amplitude (represented on the y axis as integrated EMG, µV.s) with time (s) at constant intensities (275, 300, 350 and 400 watts). The increase in iEMG indicates the recruitment of additional motor units by the central nervous system to maintain the required power output as time (and fatigue) goes on. It can be seen that the slope of the linear regression (rate of rise in iEMG as a function of time) is higher at higher intensities because fatigue occurs more quickly. It was therefore suggested that the non-invasive analysis of iEMG slope coefficient could provide a sensitive measure of motor unit fatigability. Modified from Moritani et al. (1993).

Traditionally, endurance exercise has been researched extensively but our understanding of the factors that regulate muscle recruitment during very high-intensity exercise is much poorer. This is surprising since 1) many sports (*e.g.*, team and court sports, athletics) involved burst of "all-out" activity (Billaut and Bishop 2009; Spencer et al. 2005), and 2) newly-developed training regimes targeted at improving health include repetitions of sprint exercise (Gibala 2007). Certainly, a greater understanding of neural recruitment strategies

during high-intensity tasks would lead to better training programs to enhance fitness in athletes and patient populations.

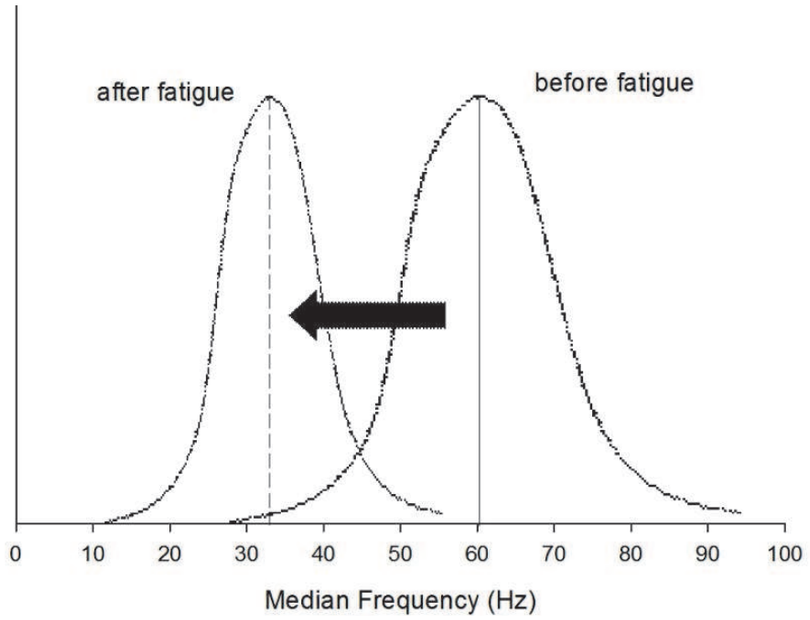

Fig. 2. Typical changes in EMG power frequency spectrum (Hz) of a muscle during a fatiguing contraction. The solid line indicates the pre-fatigue state, and the dash line indicates the fatigued state. The leftward shift in the EMG power frequency spectrum indicates the recruitment of more fatigue-resistant motor units (with lower firing frequency) to cope with the task constraint. The non-invasive analysis of the power spectra could provide a sensitive measure of motor unit fatigability that may reflect the activities of different types of muscle fibers.

Performing a sprint at "all-out" intensity requires very high levels of neural drive (typically +/– 5000 μV in elite sprinters) (Ross et al. 2001); therefore failure to activate fully the contracting musculature can theoretically decrease force and power production and, thereby, impair the ability to sprint. Whilst not a well-studied mode of exercise, several studies have demonstrated that the fatigue that develops during single and repeated sprints is associated with changes in muscle recruitment strategies which ultimately originate within the CNS. The aims of this chapter are 1) to use most recent data to describe the behaviour of the EMG signal (serving as a surrogate for muscle recruitment) during sprint exercise using traditional and innovative analysis techniques, and 2) to give some insights into the main mechanisms thought to contribute to the regulation of muscle recruitment and the fatigue process during sprint exercise.

2. A contemporary view of the fatigue phenomenon

For over a century, neuromuscular fatigue has been viewed and researched as a finite quantity of essential (metabolic and/or cardiovascular) resources causing exhaustion, independent of any regulation by the CNS (Allen et al. 1995; Bassett and Howley 2000; Fitts 1994; Hill 1924; Shephard 2009). This view has encouraged the interpretation that exercise results in linear changes in metabolism, in energy provision, and in the cardiovascular,

respiratory, thermoregulatory, and hormonal responses, among many others. Ultimately, demand exceeds capacity in one or more systems, which causes them to fail. As a result, this failure to maintain homeostasis in the active muscles causes the termination of exercise. Overall, this has produced a "brainless" physiology (Marino 2004; Marino et al. 2011; Noakes 2011; Noakes et al. 2001) that is still currently taught in most exercise science classes throughout the world. In contrast, increasing evidence has accumulated in the last few years suggesting an anticipatory regulation of exercise intensity (Billaut et al. 2011; Kayser 2003; Marino 2004; Noakes 2011; Noakes et al. 2001; Noakes and St Clair Gibson 2004; St Clair Gibson et al. 2006; St Clair Gibson and Noakes 2004; Tucker et al. 2004). This mechanism allows feedback from varied sources to influence the magnitude of the feed-forward neural drive that determines the quantity of muscle mass recruited (*i.e.,* the number of motor units recruited in the exercising limbs). In this model, therefore, an athlete's pace is continuously regulated via the action of varied physiological and psychological inputs before and during exercise (Noakes 2011; Noakes and Tucker 2008; St Clair Gibson and Noakes 2004). In other words, the brain is able to anticipate a future failure and modify accordingly and in "real time" muscle recruitment and, hence, exercise intensity to ensure homeostasis is protected.

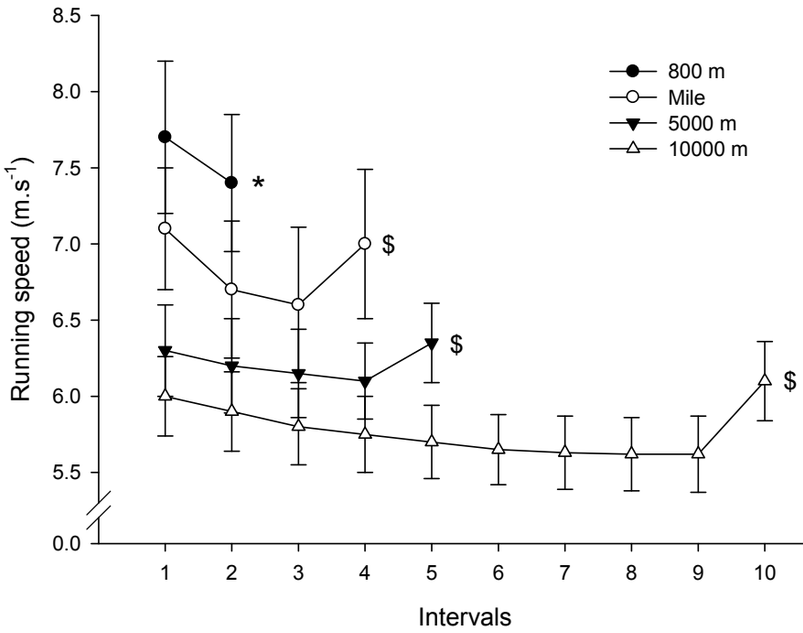

Fig. 3. Average running speed (meter.s^{-1}) for each interval during world-record performances in 800-m, 1-mile, 5000-m and 10000-m events. * Significantly slower than the first lap ($P < 0.05$). $ Significantly faster than the preceding interval ($P < 0.05$). This figure clearly shows different paces selected by athletes at the beginning of every race that depends upon the distance to run. The presence of an end-spurt is also shown in "endurance-type" events. Modified from Tucker et al. (2006a).

In this perspective, the results from several recent studies conducted on well-trained athletes and patient populations reveal that during high-intensity, constant-load and self-paced exercises participants terminate the task with a given level of severe locomotor muscle fatigue (assessed via quadriceps twitch force) that appears to be never exceeded under 'normal' exercise conditions, despite manipulations of exercise performance (Amann

2011; Amann et al. 2006a; Duffield et al. 2010; Romer et al. 2007; Saey et al. 2003). This end-exercise level of peripheral muscle fatigue has been purported to be task specific and to vary across participants (for review see (Amann 2011)). So, overall, in the early years of the 21st century, several scientists worldwide agree that the CNS constantly monitors and deliberately regulates and limits the development of peripheral fatigue in the exercising limbs (via changes in the extent of muscle recruitment). This may presumably be to avoid overtraining / overexertion and potentially long-lasting harmful consequences to critical organs (Amann 2011; Calbet 2006; Kayser 2003; Marino et al. 2011; Noakes 2011; Nybo and Secher 2004). Several physiological variables (*e.g.,* afferent feedback from exercising, fatiguing muscles (Amann et al. 2008; Amann et al. 2009), cerebral oxygenation (Billaut et al. 2010; Nielsen et al. 1999), rate of heat storage (Marino 2004; Marino et al. 2004; Tucker et al. 2006b)) would potentially be closely monitored depending upon the exercise conditions, and would influence more or less the CNS to attenuate muscle recruitment.

To the author's best knowledge, the presence of pacing during short and "all-out" sprints has been examined in one study. The authors (Billaut et al. 2011) deceived the participants to evaluate the degree of pacing depending upon the number of sprints to be performed. Astonishingly, the anticipation of performing fewer sprints (*i.e.,* 5 instead of 10) resulted in enhanced muscle recruitment (as evidenced by higher EMG activity in the lower limb), which was associated, in turn, with a higher mechanical output during the first 5 sprints. On the other hand, when participants were not informed of the actual sprint number they recruited less muscle and, hence, exhibited a lower mechanical output profile, despite similar levels of encouragement and similar perceived exertion. These results demonstrated that pacing occurs during short repeated-sprint efforts in anticipation of the number of sprints that are included in the trial (Billaut et al. 2011). Furthermore, it may be speculated from these findings that the pacing strategy adopted by these athletes has been mostly subconscious, because it is not clear how athletes can consciously regulate their muscle recruitment and power output precisely enough to account for such variations at this very high intensity and in this short time frame. Importantly, a key measure in all these experiments has been the central motor drive, namely muscle recruitment, which is directly assessed via EMG activity. Therefore, the investigation of EMG events holds a critical role in understanding humans' ability to cope with physical challenges.

3. Electromyographic events during single-sprint exercise

Although not extensively studied, changes in skeletal muscle recruitment may contribute to performance decrement during maximal sprint exercise. Vandewalle and colleagues (1991) observed a parallel decline in power output and integrated EMG of the *vastus lateralis* (average values not reported by the authors) during a 45-second cycle sprint, and suggested a progressive attenuation of spatial and/or temporal recruitment of motor units during the exercise. These findings are supported by another study that reported a ~15% ($P < 0.05$) decline in the integrated EMG of the plantar flexors and a ~15–19% ($P < 0.05$) reduction in the median frequency of the EMG power spectrum of the plantar flexors and the knee extensors during a 30-s Wingate anaerobic cycle sprint (Greer et al. 2006). While Hunter et al. (2003) did not observe any change in the EMG amplitude of the *vastus lateralis* muscle during a 30-second cycle sprint (whereas power output declined), the authors reported a shift of the mean power frequency towards lower values (–14.7%; $P < 0.05$). These acute frequency changes may be caused by an accumulation of metabolites and a consequent

decrease in muscle pH, and/or some form of neural control through reflex regulation of muscle force to prevent excessive metabolic perturbations (De Luca 1997; Juel 1988; Noakes 2011; Noakes et al. 2004; St Clair Gibson et al. 2001). Unfortunately, skeletal muscle function was not examined in these studies (via maximal voluntary contraction and twitch technique), which precludes from drawing firm conclusions about the (maximal allowed) level of peripheral muscle fatigue at the end of these tasks. Clearly, more studies using electrically- or magnetically-evoked stimulation need to be conducted to clarify whether central motor drive parallels the power output decline observed during a single maximal effort and whether it is regulated in an anticipatory manner to limit development of excessive limb muscle fatigue.

4. Electromyographic events during repeated-sprint exercise

4.1 Muscle recruitment strategies

The first investigations of the changes of the EMG signal (serving as surrogate for muscle recruitment) provoked by "all-out" repeated sprints have only been conducted recently (Billaut and Basset 2007; Billaut et al. 2006; Giacomoni et al. 2006). These studies have examined the EMG activity of the quadriceps muscles during brief, maximal, isometric voluntary contractions of the knee extensors performed before and immediately after the ten 6-s sprints separated by 30 s of passive rest. In every case, the maximal knee extensors force was reduced after the sprints (average: ~12%; $P < 0.05$) and this was accompanied by a higher EMG activity (average: ~15%; $P < 0.05$). The authors also observed a concomitant decrease in frequency components, which suggests a modification in the pattern of muscle fibre recruitment and a decrease in conduction velocity of active fibres (Basmajian and De Luca 1985; De Luca 1997; Gerdle and Fugl-Meyer 1992; Linnamo et al. 2000; Moritani et al. 1986). In fact, during the final sprints, the relative contribution of less-powerful type I muscle fibres involved in the production of power may have increased as a result of the greater fatigability of type II muscle fibres, highly solicited during this type of exercise (Casey et al. 1996; Gerdle and Fugl-Meyer 1992; Komi and Tesch 1979; Ross et al. 2001). However, the conclusions from these studies may have been confounded by methodological factors. Indeed, while investigating EMG activity during isometric contractions may greatly reduce muscle movements underneath EMG electrodes and artefacts due to wire movements, and thus ease signal processing (Farina et al. 2004; Merletti and Lo Conte 1997), it is now well accepted that physiological responses and neuromuscular fatigue are highly task specific (Hunter 2009; Maluf and Enoka 2005). Accordingly, results and conclusions would be highly specific to the task performed, hence, the isometric contraction, with less relevance to the actual fatigue that develops during the repeated sprints.

More recently, muscle recruitment strategies have been investigated during sprints, and authors have reported a concurrent decline in mechanical performances and the amplitude of the EMG signal in primer-mover muscles (Billaut and Smith 2009; Billaut and Smith 2010; Mendez-Villanueva et al. 2007, 2008; Racinais et al. 2007; Smith and Billaut 2010). For example, Billaut and Smith (2010) demonstrated a ~15% decline ($P < 0.05$) in the quadriceps muscles EMG activity from the first to the twentieth cycle sprint of 5 s separated by 25 s of rest. Importantly, such a reduction in agonist muscle recruitment during repeated cycle sprints has been strongly correlated ($r = 0.91$ to 0.98, $P < 0.05$, Figure 4) with the decline in mechanical output (Billaut and Smith 2009; Billaut and Smith 2010; Mendez-Villanueva et al. 2008). That being said, it is interesting to note that when fatigue is moderate (*e.g.*, fatigue

index or sprint decrement score is ~10% or lower), studies have typically reported a steady level of muscle recruitment during repeated-sprint exercise (Billaut and Basset 2007; Billaut et al. 2005; Girard et al. 2008; Hautier et al. 2000; Matsuura et al. 2007). However, when the neuromuscular fatigue level is more substantial (> 20%), a concurrent decline in mechanical performance and the amplitude of EMG signals has consistently been reported across sprint repetitions (Billaut et al. 2011; Billaut and Smith 2010; Mendez-Villanueva et al. 2007, 2008; Racinais et al. 2007; Smith and Billaut 2010). Furthermore, a reduction in the knee flexor EMG amplitude (*i.e.,* antagonist muscle) has been observed during fifteen cycle sprints of 5 s separated by 25 s of rest (Hautier et al. 2000). Overall, these data suggest that under conditions of considerable neuromuscular fatigue development the failure to fully activate the contracting musculature may become an important factor limiting performance during "all-out" sprint repetitions.

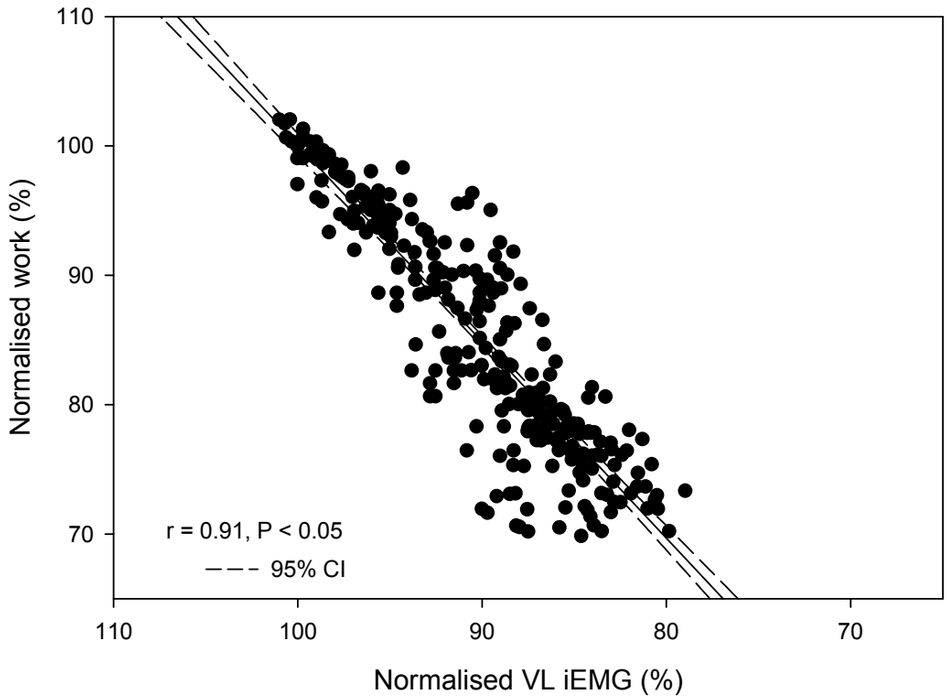

Fig. 4. Relationship of EMG amplitude (integrated EMG) of the *vastus lateralis* muscle (expressed as a percentage of initial-sprint value) to mechanical work (expressed as a percentage of initial-sprint value) performed over twenty 5-s cycle sprints interspersed by 25 s of rest. The correlation shows that the greater the drop in EMG amplitude the greater the drop in mechanical work [r = 0.91 (95% Confidence Interval, 0.75;0.97)]. The short-dashed lines represent 95% confident intervals. Reproduced from Billaut and Smith (2010) with permission.

4.2 Timing of muscle activation

Thus far, the above sections have particularly highlighted the importance of the "quantity" of EMG activity or muscle recruitment in the production of maximal power. During "all-out" actions, the timing of muscle activity also comes into play in determining neuromuscular performance. In fact, during sprint exercises maximal power production

requires muscles to be fully activated during shortening and fully relaxed during lengthening phases (Neptune and Kautz 2001). Thus, when studying sprint exercises, the inter-muscle coordination patterns (*i.e.,* the kinematics of muscle activation and deactivation) should also be taken into account when analysing the fatigue process (Neptune and Kautz 2001; Prilutsky and Gregor 2000; Van Ingen Schenau et al. 1992). Muscle coordination is defined as a "distribution of muscle activation or force among individual muscles to produce a given combination of joint moments" (Prilutsky and Gregor 2000), and, therefore, can be studied from both EMG and force patterns of individual muscles. The appropriateness of using EMG recordings for studying muscle coordination has been discussed on several occasions and the reader is referred to the following detailed reviews (Hug 2011; Hug and Dorel 2009). Studies that focus on muscle coordination usually report muscle activity profiles (*i.e.,* EMG patterns; Figure 5). Typically, a linear envelope of the EMG signal is first computed by rectification (Hug and Dorel 2009). Then, the EMG is related to mechanical events (*e.g.,* foot switches, force signal), and is time normalized. Finally, a representative EMG profile is obtained for every muscle by averaging the linear envelopes for a number of consecutive cycles or trials. EMG profiles are characterised by bursts of activity. Muscle coordination is presumably modulated by the neural command in order to generate an appropriate neural control strategy or more simply reflects task constraints (Hug 2011; Tresch and Jarc 2009). Thus, it provides an attractive simplifying strategy to study the control of complex movements in sports and daily activities. Because this approach allows describing multiple patterns of muscle activity in an integrative fashion, it is believed to be a useful way of examining muscle coordination.

As this is a rather innovative approach of using EMG signals to study the brain regulation of exercise capacity, very few data are available during sprint exercise. To the author's best knowledge, the only study on coordination patterns in sprints has demonstrated that the agonist–antagonist activation strategy is altered during ten 6-s cycle sprints. The *biceps femoris* muscle was activated earlier within a pedaling cycle presumably to account for the loss of force of the prime movers in order to maintain the transfer of force to the pedal. Importantly, this locomotor alteration occurred before any changes in the "quantity" of muscle recruitment (*i.e.,* EMG amplitude), which suggests that muscle activation sequence is an important parameter in the regulation of exercise intensity (Billaut et al. 2005). Admittedly, much work lies ahead to substantiate this hypothesis and fully comprehend the neural processes behind the complex regulation of muscle actions. Future studies devoted at challenging muscle coordination and investigating how the brain copes with task constraints are warranted.

4.3 At least two potential causes for down-regulation of muscle recruitment

While it is suspected that multiple feedback originating from various locations within the body alter the output of the spinal motor neuron pool, and therefore muscle recruitment, research into neuromuscular fatigue during "all-out" sprints has defined at least two potential influences. The mechanisms that lead to a decreased output of the spinal motor neuron pool are still not well understood, especially during repeated sprints. Nevertheless, it is now clear that the CNS receives sensory input from muscle afferents (*e.g.,* muscle spindles, Golgi tendon organs, group III and IV fibres) which are integrated into the determination of central motor drive. Evidence is accumulating that the purpose of this reflex mechanism may be to limit the development of excessive peripheral muscle fatigue (Amann 2011; Kayser 2003; Marino et al. 2011; Noakes 2011). This consensus is supported by experimental studies showing that pre-existing locomotor muscle fatigue has a dose-

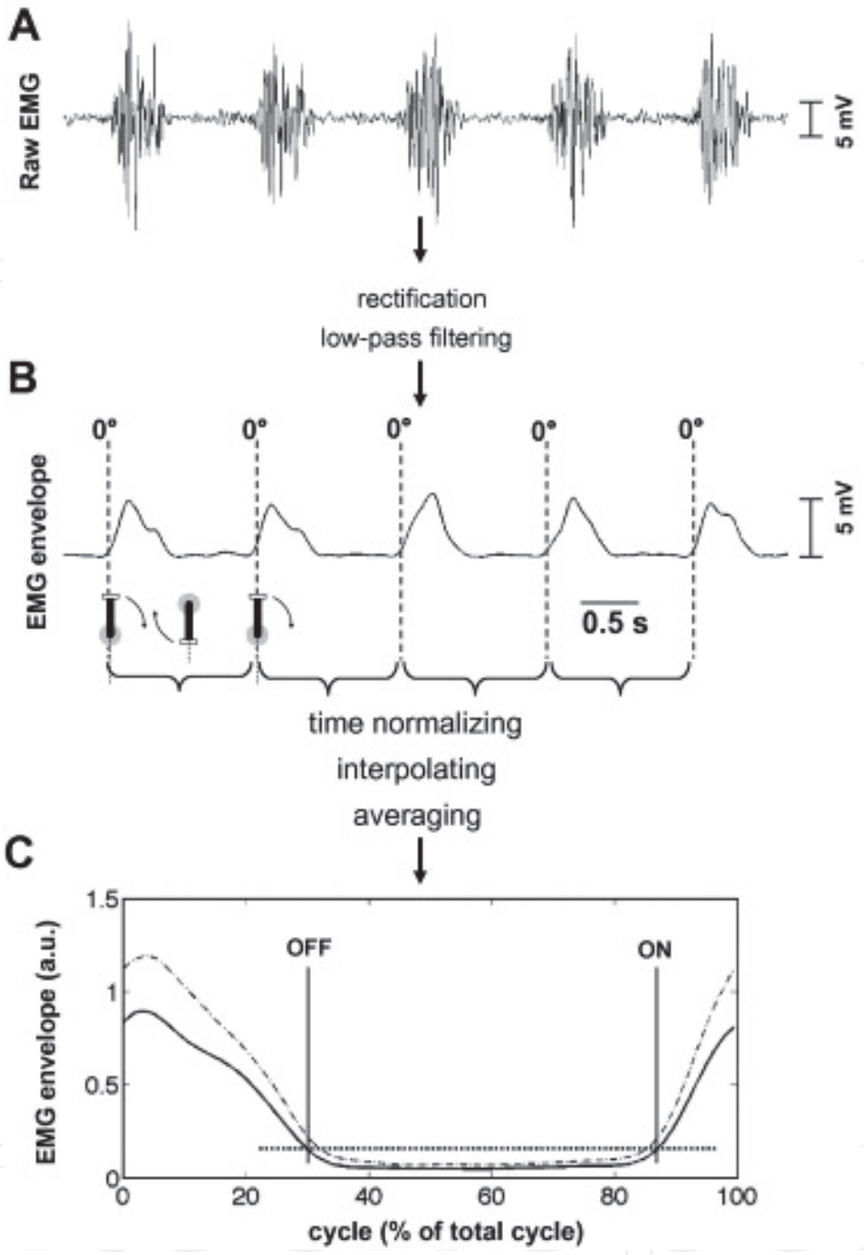

Fig. 5. Example of surface electromyographic signal processing to examine muscle coordination strategies. Panel A displays a raw EMG signal recorded from the *vastus lateralis* muscle during pedalling. The linear envelope is first computed by rectification and low-pass filtering (9 Hz in this example). All cycles are then extracted using a trigger pulse that is delivered at the top dead centre (crank angle = 0°; Panel B) and time normalised. Finally, a representative EMG profile is obtained for every muscle by averaging the linear envelopes for a number of consecutive cycles (Panel C). Onset and offset values are then determined from this averaged pattern using an EMG threshold (fixed at 20% of the peak EMG recorded during the cycle in this example; horizontal dashed line). Reproduced from Hug (2011) with permission.

dependent, inverse effect on muscle recruitment and power output during a 5-km time trial (Amann and Dempsey 2008). Specifically, the higher the level of pre-existing fatigue, the lower the muscle recruitment and power output during the trial. Furthermore, in a nicely-designed series of studies Amann and colleagues (2008; 2009) presented convincing results that sensory feedback from fatiguing muscles restrict muscle recruitment and, therefore, the exercise-induced development of peripheral fatigue during whole-body endurance exercise. However, whether the central projection of afferent feedbacks from fatigued muscles changes according to the continuous vs. intermittent nature of the exercise task remains unknown. It is intuitive that the dramatic metabolic disturbances that occur within the exercising muscle during "all-out" and repeated sprints (for review see (Billaut and Bishop 2009)) influence the central projection of these feedbacks and thus affect the central neural drive during such intense tasks (Billaut et al. 2005; Billaut and Smith 2010; Mendez-Villanueva et al. 2008; Racinais et al. 2007). That being said, the manipulation of intramuscular pH, for example, by oral administration of sodium bicarbonate does not influence EMG activity and mechanical performance during repeated cycling sprints (Matsuura et al. 2007). Further studies manipulating the metabolic "milieu interieur" during sprint repetitions to examine the effects on centrally-regulated muscle recruitment are needed.

A second potential line of inquiry for fatigue research during "all-out" activity is the influence of oxygenation on motor neuron activity. Recently, studies have been performed to further unravel changes in muscle activation. In this perspective, it is worth noting that the drop in EMG amplitude observed during repeated sprints has been shown to be strongly correlated ($r = 0.80$ to 0.95, $P < 0.05$) with a decline in arterial blood oxygen (O_2) saturation (Billaut and Smith 2009; Billaut and Smith 2010). This relationship is also very similar in men and women, suggesting that there is no sex dimorphism in this phenomenon (Billaut and Smith 2009) and demonstrating the strong recurrence of this phenomenon. So, is it simply coincidence that muscle recruitment falls concomitantly with arterial saturation? One may interpret existing correlative evidence (Billaut et al. 2010; Billaut and Smith 2009; Billaut and Smith 2010) to mean that the CNS deliberately down-regulates muscle activity to keep peripheral fatigue within "tolerable" limits. We further tested this hypothesis and used a hypoxia paradigm to alter O_2 delivery to tissues and evaluate its impact on muscle recruitment. The reduction in prefrontal cortex oxygenation induced by acute hypoxia was correlated ($r = 0.89$ to 0.92, $P < 0.05$) with the changes in EMG amplitude of active muscles during ten 10-s cycle sprints (Smith and Billaut 2010). This shows that the larger the brain deoxygenation, the lower the muscle recruitment. At this point however, it is important to acknowledge that the use of surface EMG as a sole determinant of the neural response is questionable and requires caution, even though its rate of change throughout the exercise may be used as an index of the rate of motor unit recruitment (Amann et al. 2008; Amann et al. 2006b; Amann et al. 2007; Romer et al. 2007). Taken as it is (*i.e.,* without neuromuscular stimulation technique), our EMG data are compatible with the general finding that hypoxia has an effect on cortical and subcortical functions of the CNS, and thereby, on motor neuron excitability and synaptic neurotransmission. This hypothesis is consistent with the findings that systemic hypoxaemia and insufficient brain oxygenation depress central neural drive (Amann et al. 2006a; Amann and Kayser 2009; Bigland-Ritchie et al. 1986b; Dillon and Waldrop 1992; Dousset et al. 2003). The latest research has strengthened this consensus by showing that scalp-recorded brain cortical activity (as assessed via electroencephalography combined with localized electromagnetic tomography) is affected during whole-body

exercise at high intensity (Brummer et al. 2011; Schneider et al. 2010) and in a hypoxic environment (Schneider and Struder 2009). The use of electromagnetic tomography during supra-maximal exercise is restricted by head movement artifacts, but remains a powerful tool to shed light on muscle recruitment strategies in future years.

5. Conclusion

It is becoming clear that the CNS modulates muscle recruitment (both quantity of muscle mass and coordination strategies) to cope with task constraints during high-intensity exercise. It may do so presumably to counteract the increasing fatigue process and limit the development of peripheral muscle fatigue to a non-harmful level. Exercise physiologists and sport scientists can no longer pretend that conscious or subconscious decision making plays no role in the fatigue process and, ultimately, in the regulation of exercise intensity. Perhaps, as Marino and colleagues wrote, "*this means that studying fatigue as a closed feedback loop will no longer suffice. At this stage in our understanding of the brain, bringing feed-forward mechanisms of the brain into fatigue process*" is necessary to promote greater understanding of human physiology (Marino et al. 2011). Now is an appropriate time to move the field of exercise physiology forward and acknowledge the controlling role of the brain. Understanding these complex neural strategies during sprint exercise constitutes a necessary step if we want to implement such tasks in interventions designed to enhance performance and health. And in this quest, the recording of surface EMG activity holds potential.

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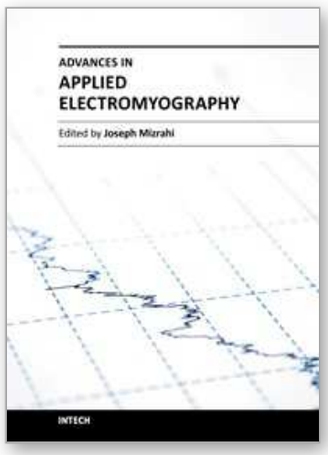

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The electrical activity of the muscles, as measured by means of electromyography (EMG), is a major expression of muscle contraction. This book aims at providing an updated overview of the recent developments in electromyography from diverse aspects and various applications in clinical and experimental research. It consists of ten chapters arranged in four sections. The first section deals with EMG signals from skeletal muscles and their significance in assessing biomechanical and physiologic function and in applications in neuro-musculo-skeletal rehabilitation. The second section addresses methodologies for the treatment of the signal itself: noise removal and pattern recognition for the activation of artificial limbs. The third section deals with utilizing the EMG signals for inferring on the mechanical action of the muscle, such as force, e.g., pinching force in humans or sucking pressure in the cibarial pump during feeding of the hematophagous hemiptera insect. The fourth and last section deals with the clinical role of electromyograms in studying the pelvic floor muscle function.

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