Neuroscience Letters 576 (2014) 11–16

Contents lists available at ScienceDirect

Neuroscience Letters

journal homepage: www.elsevier.com/locate/neulet

Changes in muscle coordination and power output during sprint cycling

Steven J. O'Bryan[a], Nicholas A.T. Brown[b], François Billaut[a,c], David M. Rouffet[a,b,*]

[a] Institute of Sport, Exercise and Active Living (ISEAL), Victoria University, PO Box 14428, Melbourne, VIC 8001, Australia
[b] Department of Biomechanics and Performance Analysis, Australian Institute of Sport, PO Box 176, Belconnen, ACT 2616, Australia
[c] Institut national du sport du Québec, 1000, avenue Émile-Journault – bureau 1.72, Montréal, Qc H2M 2E7, Canada

HIGHLIGHTS

- Power deficit is accompanied by significant alterations in muscle coordination.
- Large reductions in the EMG activity of RF and GAS muscles are observed during 30-s cycling sprint.
- Co-activation between GAS and proximal mono-articular muscles are dramatically reduced.
- Co-activation analysis complements information provided by biomechanical models.
- Coordination is affected by central/peripheral fatigue or change in movement strategy.

ARTICLE INFO

Article history:
Received 17 September 2013
Received in revised form 27 March 2014
Accepted 13 May 2014
Available online 23 May 2014

Keywords:
EMG
Muscle activation
Bi-articular muscles
Fatigue
Co-activation
Power transfer

ABSTRACT

This study investigated the changes in muscle coordination associated to power output decrease during a 30-s isokinetic (120 rpm) cycling sprint. Modifications in EMG amplitude and onset/offset were investigated from eight muscles [*gluteus maximus* (EMG_{GMAX}), *vastus lateralis* and *medialis obliquus* (EMG_{VAS}), medial and lateral *gastrocnemius* (EMG_{GAS}), *rectus femoris* (EMG_{RF}), *biceps femoris* and *semitendinosus* (EMG_{HAM})]. Changes in co-activation of four muscle pairs ($CAI_{GMAX/GAS}$, $CAI_{VAS/GAS}$, $CAI_{VAS/HAM}$ and $CAI_{GMAX/RF}$) were also calculated. Substantial power reduction ($60 \pm 6\%$) was accompanied by a decrease in EMG amplitude for all muscles other than HAM, with the greatest deficit identified for EMG_{RF} ($31 \pm 16\%$) and EMG_{GAS} ($20 \pm 14\%$). GAS_{onset}, HAM_{onset} and $GMAX_{onset}$ shifted later in the pedalling cycle and the EMG offsets of all muscles (except GAS_{offset}) shifted earlier as the sprint progressed ($P < 0.05$). At the end of the sprint, $CAI_{VAS/GAS}$ and $CAI_{GMAX/GAS}$ were reduced by $48 \pm 10\%$ and $43 \pm 12\%$, respectively. Our results show that substantial power reduction during fatiguing sprint cycling is accompanied by marked reductions in the EMG activity of bi-articular GAS and RF and co-activation level between GAS and main power producer muscles (GMAX and VAS). The observed changes in RF and GAS EMG activity are likely to result in a redistribution of the joint powers and alterations in the orientation of the pedal forces.

1. Introduction

Performance during sprinting events (running, cycling) is strongly determined by the ability to produce and maintain maximal levels of muscular power. However, the ability of the central nervous system (CNS) to optimally coordinate the activation of the different muscles is also very important as it determines how the muscular forces are distributed across the joints and how the external force is orientated. If power production during sprint performances has been well documented [6,8,17], muscle coordination has received less attention, particularly during fatiguing sprints.

Previous studies have provided detailed analyses of muscle coordination during fatigue-free sprint cycling, through the investigation of muscle activation patterns during the pedalling cycle [5,20]. Surface EMG (EMG) studies of fatigue-free sprint cycling show that *gastrocnemius* (GAS) and quadriceps muscles [*vasti* (VAS) and *rectus femoris* (RF)] are maximally activated, while some discrepancies have been reported regarding activation level of the hamstrings (HAM) and *gluteus maximus* (GMAX) muscles [5,20]. Considering the high activation level of most lower-limb muscles

* Corresponding author at: Institute of Sport, Exercise and Active Living (ISEAL), Victoria University, PO Box 14428, Melbourne, VIC 8001, Australia.
Tel.: +61 3 9919 4384.
E-mail address: david.rouffet@vu.edu.au (D.M. Rouffet).

http://dx.doi.org/10.1016/j.neulet.2014.05.023
0304-3940/

during fatigue-free sprint cycling, neuromuscular fatigue (central and peripheral) is likely to occur during prolonged periods of sprint cycling (i.e. 30-s) [12], which may manifest as changes in muscular EMG activity (amplitude and/or timing).

During a maximal 30-s cycling sprint, EMG activity of the quadriceps has been shown to decrease by ~8% [4] or remain unaltered [9,13,19], whereas GAS muscles have shown a decrease of up to 15% [9]. A manipulation to the timing of activation for *biceps femoris* (BF) muscles during fatiguing sprint cycling has also been shown [2]. Unfortunately, none of these studies [2,4,9,13,19] analyzed changes in EMG activity using the recommendations made by recent extensive reviews [3,12]. Consequently, it seems difficult to describe the link between changes in muscle coordination with power deficit during fatiguing sprint cycling from this previous data.

Martin and Brown [16] demonstrated that the power produced at the three lower-limb joints decreases at different rates during a maximal 30-s isokinetic (120 rpm) cycle sprint. In reference to biomechanical models of pedalling [25,27], it is possible that alterations in joint power [16] could be further understood by investigating changes in muscle co-activation. Van Ingen Schenau [25] showed that co-activation between GMAX/RF and VAS/HAM is essential to transfer muscular forces across the hip and knee joints and to optimize the orientation of the pedal forces. A decrease in co-activation between VAS/HAM has been suggested during fatiguing cycling sprints [2,10]. However, Zajac et al. [27] further suggests that co-activation between the ankle plantar flexors and proximal muscles (GMAX and VAS) is a key factor for optimizing power transfer across the ankle joint to the crank during cycling. Interestingly, Martin and Brown [16] observed the largest reduction in joint power at the ankle level over the course of a fatiguing cycling sprint (63%). However, no EMG studies have investigated changes in co-activation between the ankle plantar flexors and proximal muscles during fatiguing sprint cycling.

The aim of the present study was to investigate changes in the EMG activity (amplitude and onsets/offsets) of the lower-limb muscles and associated modifications in muscle co-activations during a fatiguing 30-s cycling sprint for which detailed biomechanical data has been reported [16]. Changes in the EMG activity of five major lower-limb muscle groups were investigated by using gold standard methods for EMG normalization [3,20], calculation of EMG profiles [5,12] and quantification of co-activations [15]. Extending on the biomechanical findings of Martin and Brown [16], it was hypothesized that marked changes in the EMG activity of the ankle plantar flexor muscles (amplitude and timing) and/or co-activation of this muscle with proximal muscles would occur during the course of a fatiguing 30-s cycle sprint.

2. Materials and methods

Ten active males volunteered to participate in this study (age 24 ± 3 years; body mass 83.7 ± 9.7 kg). Six were amateur team sport players (Australian Rules football, basketball and netball), three participated in individual sports (swimming, tennis and sprint athletics) and one was undertaking regular resistance training, with an overall training load of 5.3 ± 1.7 h/week. Written informed consent was obtained from each participant and all testing was approved by Victoria University's Human Research Ethics Committee.

Exercise was conducted on an electronically braked cycle ergometer (Excalibur Sport; Lode®, The Netherlands) which sampled power output at 5 Hz. Crank length equalled 175 mm and pedal straps were utilized to fasten feet into the pedals. Participants remained seated with hand position in the dropped portion of the handlebars during the sprint.

A familiarization session took place no earlier than 48 h prior to testing. Following a warm-up [5-min cycling at 1 W/kg and 80 revolutions per minute (rpm)], each participant completed a practice 5-s sprint followed by 5 min of passive rest. For the 30-s sprint, participants were required to overcome the inertia of the flywheel and reach a cadence close to 120 rpm within 5 s to start time. Participants were stopped from pedalling and instructed to place the right crank angle at 45° before receiving a verbal 5-s countdown. For the sprint, the ergometer was set in isokinetic mode and cadence fixed at 120 rpm [16]. Power output was calculated over each pedalling cycle and normalized in reference to the peak power achieved during the sprint (P_{PEAK}).

EMG signals were recorded from eight muscles of the left lower-limb [gluteus maximus (EMG_{GMAX}), rectus femoris (EMG_{RF}), vastus lateralis (VAS_{LAT}), vastus medialis obliquus (VAS_{MED}), semitendinosus (ST), biceps femoris long head (BF), gastrocnemius medialis (GAS_{MED}) and gastrocnemius lateralis (GAS_{LAT})]. Muscles from the same functional group were averaged to produce EMG_{GAS} (GAS_{LAT} + GAS_{MED}), EMG_{VAS} (VAS_{LAT} + VAS_{MED}) and EMG_{HAM} (ST + BF). Dual surface electrodes of 10 mm diameter and inter-electrode distance of 20 mm (Noraxon dual electrodes, Noraxon USA Inc., Scottsdale, AZ) were used to record the EMG signals, with electrode location defined following SENIAM's recommendations [11]. The reference electrode was placed over the superior aspect of the medial sacral crest. Prior to electrode application, skin was shaved, lightly abraded and cleaned with an alcohol swab to reduce skin impedance. Tubular netting was worn to limit movement artefacts. All signals were recorded continuously at 1500 Hz via a wireless receiver (Telemyo 2400 GT, Noraxon Inc., USA) connected to a notebook. A reed switch attached to the ergometer frame was aligned with a magnet attached to the left crank in order to identify 0/100% (TDC) of the left pedalling cycle from +3 V pulse recorded in a channel of the EMG system.

EMG signals were recorded and processed using Noraxon software (MyoResearch XP version 1.07.41). Raw EMG signals were pre-amplified, band pass filtered (10–500 Hz) and full wave rectified. Following full wave rectification, the EMG signals were root mean squared with a 100 ms moving rectangular window to create a linear envelope. Each linear envelope was synchronized with the TDC sensor and time normalized to 100 points (100% of pedalling cycle, TDC-TDC) to create EMG activity profiles, which were then normalized in reference to the individual maximum value obtained during the sprint [5,12,20]. An average EMG profile was constructed for each muscle group and for each 6-s interval. From each of the EMG profiles (i.e. 60), we calculated the average EMG amplitude and the EMG onset/offset of activation via determining a threshold of three standard deviations above the minimal EMG value [21].

Co-activation values were calculated from the normalized EMG activity profiles using the Co-activation Index (CAI) employed by Lewek et al. [15]. Based on the biomechanical models of cycling, changes in CAIs were calculated for VAS/HAM ($CAI_{VAS/HAM}$) and GMAX/RF ($CAI_{GMAX/RF}$) [25] as well as VAS/GAS ($CAI_{VAS/GAS}$) and GMAX/GAS ($CAI_{GMAX/GAS}$) [27].

Power output, EMG amplitude, onset/offset and CAIs was recorded for each pedalling cycle before average values were calculated for each variable over five even time intervals of 6 s duration (equivalent of 20% total sprint duration and 12 consecutive pedalling cycles). All data was analyzed using SPSS software (version 21, SPSS Inc., Chicago, IL). A two-way repeated measures ANOVA (muscle/CAI*time) with LSD post hoc comparisons was performed to evaluate differences between EMG (amplitude and onset/offset) and CAIs over each 6-s time interval of the sprint. Changes over time for mean power, individual EMG and CAIs were investigated via one-way repeated measures ANOVA. The significance level for all statistical tests was set at $P < 0.05$ and all data in text is reported as mean ± standard deviation.