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Full Length Article

Reduced cortical voluntary activation during bilateral knee extension

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ABSTRACT

Introduction: Reduced neural drive is mainly thought to explain the bilateral deficit phenomenon, i.e. the difference in maximal isometric voluntary contraction (MVC) between unilateral and bilateral contractions. The aim of the present study was to further document if bilateral knee extension is associated with changes in voluntary activation level assessed by both peripheral nerve electrical stimulation and transcranial magnetic stimulation.
Methods: Fourteen subjects performed unilateral and bilateral knee extensions with both superimposed femoral electrical nerve stimulation and transcranial magnetic stimulation in order to assess voluntary activation (VA_{FNES}) and cortical voluntary activation (VA_{TMS}), respectively.
Results: There was no difference in MVC force of the tested leg when involved in unilateral and bilateral knee extensions ($p = 0.87$). However, a significantly reduced VA_{FNES} ($-2.1 \pm 2.4\%$; $p = 0.01$) and VA_{TMS} ($-1.6 \pm 2.7\%$; $p = 0.04$) have been evidenced during bilateral knee extension.
Discussion: It is hypothesized that counterbalances could have masked the decrease of voluntary activation during bilateral contraction.

1. Introduction

The bilateral deficit refers to a lower maximal isometric voluntary contraction (MVC) during bilateral contractions (i.e. with both limbs acting simultaneously) than the sum of unilateral MVCs performed separately by the two limbs. This has been well documented for upper limb muscles (Post et al., 2007; Van Dieen, Ogita, & De Haan, 2003), as well as lower limb muscles, especially knee extensors (Buckthorpe, Pain, & Folland, 2013; Butler, Crowell, & Davis, 2003; Hakkinen, Kallinen, et al., 1996; Howard & Enoka, 1991; Koh, Grabiner, & Clough, 1993; Kuruganti, Murphy, & Pardy, 2011; Matkowski, Martin, & Lepers, 2011; Secher, Rube, & Elers, 1988; Van Dieen et al., 2003). Although the mechanisms involved in the bilateral deficit are still a matter of debate, reduced neural drive to the agonist muscles is often hypothesized as the main potential explanation.

Surface electromyography (EMG) has been widely used to investigate neural changes associated with the bilateral deficit. Some studies reported bilateral deficit with concomitant reduced agonist EMG amplitude (commonly assessed through its

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root mean square value) (Koh et al., 1993; Oda & Moritani, 1994; Van Dieen et al., 2003; Vandervoort, Sale, & Moroz, 1984), while others failed to evidence such a parallel (Buckthorpe et al., 2013; Howard & Enoka, 1991; Kawakami, Sale, MacDougall, & Moroz, 1998; Matkowski et al., 2011; Schantz, Moritani, Karlson, Johansson, & Lundh, 1989). These equivocal findings may result from the known limitations of EMG to detect small changes in motor unit activity (Farina, Holobar, Merletti, & Enoka, 2010), and then its inability to match small changes in force as observed for the bilateral deficit (Howard & Enoka, 1991). Similar discrepancies are reported when the changes in agonist neural drive are investigated with the interpolated twitch technique (Merton, 1954). Such a technique allows the calculation of the voluntary activation level (VA) by measuring the amplitude of the superimposed twitch elicited by peripheral electrical nerve stimulation. Indeed, while Van Dieen et al. (2003) reported a bilateral deficit that was associated with decreased VA, Matkowski et al. (2011) demonstrated no VA changes and hypothesized that the observed bilateral deficit was rather due to changes in antagonist co-activation. Moreover, others studies failed to evidence difference in MVC between unilateral and bilateral contractions despite significant (Herbert & Gandevia, 1996) and non-significant (Jakobi & Cafarelli, 1998) decreased VA during bilateral contractions. Transcranial magnetic stimulation (TMS) may also be used to evoke superimposed twitches during contractions. Cortical VA assessment using TMS was recently demonstrated to be reliable for knee extensors muscles (Goodall, Romer, & Ross, 2009; Sidhu, Bentley, & Carroll, 2009) and may highlight the presence of suboptimal drive from the motor cortex to the involved muscles during MVC (Goodall, Howatson, Romer, & Ross, 2014). Since previous reports using functional magnetic resonance imaging demonstrated an altered activation of the precentral gyrus during bilateral contraction of the index finger, suggesting decreased input to the motor cortex (Post et al., 2007), cortical VA assessment may be an interesting tool to further understand the bilateral deficit phenomenon.

The aim of the present study was to further document if bilateral knee extension is associated with changes in VA assessed by both peripheral nerve electrical stimulation and transcranial magnetic stimulation. Decreased VA, and notably cortical VA, was hypothesized to be observed with reduced muscle force production during bilateral contractions.

2. Methods

2.1. Subjects

Fourteen young and healthy males (age: 22 ± 3 years; height: 177 ± 5 cm; body mass: 73 ± 9 kg) participated in this study. Subjects were regularly involved in physical activity (2–4 h per week). Written informed consent was obtained from all subjects prior to their participation. This study conformed to the standards from latest revision of the *Declaration of Helsinki* and was approved by the local ethics committee. All participants were free of lower-limb injury during the previous three months, contraindications to TMS, acute and chronic neurological disorders and trauma. They were instructed to abstain from caffeine for a minimum of 12 h before the testing session.

2.2. Experimental protocol

Measurements were assessed at least 1 week after a familiarization session with the experimental procedures. The design of the experimental protocol is illustrated in Fig. 1. After a standardized warm-up, 2 maximal knee flexions of the dominant leg were performed. Maximal voluntary knee extension (MVC) was then determined from 2 unilateral (UL) maximal contrac-

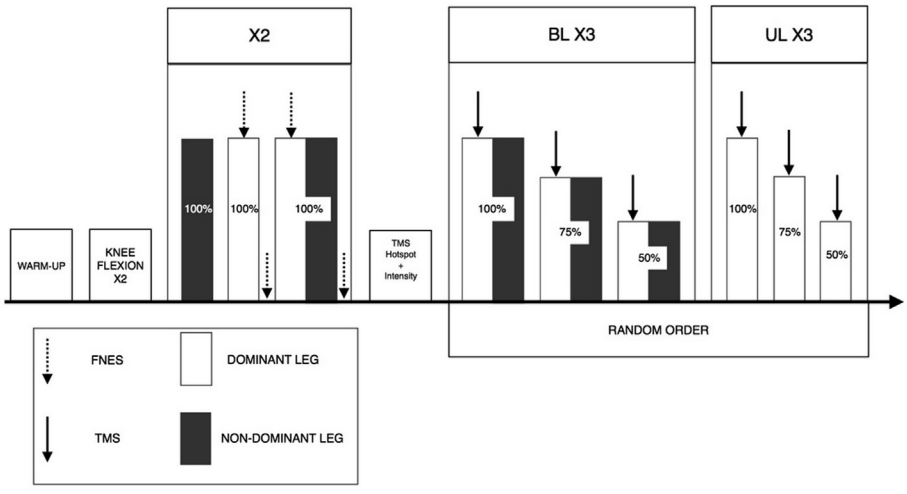

Fig. 1. Illustration of the experimental protocol.