Journal of Electromyography and Kinesiology 24 (2014) 780–788

Contents lists available at ScienceDirect

Journal of Electromyography and Kinesiology

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

2014 ISEK Congress Keynote Lecture

The neural control of coactivation during fatiguing contractions revisited

Jacques Duchateau *, Stéphane Baudry

Laboratory of Applied Biology, ULB Neurosciences Institute, Université Libre de Bruxelles (ULB), Brussels, Belgium

ARTICLE INFO

Article history:
Received 18 August 2014
Accepted 18 August 2014

Keywords:
Motor control
Antagonist muscle
Surface electromyography
Cortical excitability
Spinal excitability

ABSTRACT

In addition to the role of muscle coactivation, a major question in the field is how antagonist activation is controlled to minimize its opposing effect on agonist muscle performance. Muscle fatigue is an interesting condition to analyze the neural adjustments in antagonist muscle activity and to gain more insights into the control mechanisms of coactivation. In that context, previous studies have reported that although the EMG activity of agonists and antagonists increase in parallel, the ratio between EMG activities in the two sets of muscles during a fatiguing submaximal contraction decreased progressively and contributed to a reduction in the time to task failure. In contrast, more recent studies using a novel normalization procedure indicated that the agonist/antagonist ratio remained relatively constant, suggesting that the fatigue-related increase in coactivation does not impede performance. Current knowledge also indicates that peripheral mechanisms cannot by themselves mediate the intensity of antagonist coactivation during fatiguing contractions, implying that supraspinal mechanisms are involved. The unique modulation of the synaptic input from Ia afferents to the antagonist motor neurones during a fatiguing contraction of the agonist muscles further suggests a separate control of the two sets of muscles.

Contents

* Corresponding author. Address: Laboratory of Applied Biology and Research Unit in Applied Neurophysiology, Université Libre de Bruxelles, 808 route de Lennik, CP 640, 1070 Brussels, Belgium. Tel.: +32 2 655 3243.
E-mail address: jduchat@ulb.ac.be (J. Duchateau).

http://dx.doi.org/10.1016/j.jelekin.2014.08.006
1050-6411/

1. Introduction

The amount of activity in antagonist muscles is usually less than that in the agonists during a voluntary contraction. Since the seminal work of Sherrington (1897, 1913) the mechanisms underlying the depression in antagonist activity are known to be largely due to a spinal pathway that reduces the excitability of the motor neurone pool of the antagonist muscles in response to the agonist muscles activation (i.e., reciprocal inhibition). Subsequent studies demonstrated that this pathway involved a disynaptic reciprocal inhibition from muscle spindle afferents from the agonist muscle to the motor neurones of the antagonist muscles (for reviews, see Baldissera et al., 1981; Crone and Nielsen, 1994; Jankowska, 1992; Pierrot-Deseilligny and Burke, 2005). However, some electromyographic (EMG) activity is usually observed in antagonist muscles during a voluntary contraction of an agonist muscle and this is classically referred to as "antagonist coactivation". Although antagonist coactivation can be controlled at spinal levels (Crone and Nielsen, 1989), current knowledge indicates a significant role for supraspinal centres (Bertolasi et al., 1998; Crone and Nielsen, 1994; Hansen et al., 2002; Humphrey and Reed, 1983; Mink and Thach, 1991).

The main objective of the current paper is to revisit the control of antagonist coactivation with a special emphasize on how the nervous system modulates the amount of coactivation during a fatiguing contraction. It seems reasonable to expect that the central nervous system would finely tune the level of coactivation during the course of a fatiguing contraction to minimize the opposing action of antagonist muscles on the agonist activity. Before discussing the possible mechanisms underlying the control of coactivation during fatiguing contractions, this paper will evoke briefly its role and a few technical issues associated with the recording and quantification of coactivation.

2. Definition and technical issues

2.1. Coactivation vs. cocontraction

As already mentioned, coactivation is classically defined as the unintentional concurrent activation of antagonist muscles during the activation of agonist muscles (Kellis, 1998). Coactivation occurs usually during both isometric contractions and movements (Fig. 1). Although cocontraction is often used as a synonym of coactivation (Frey-Law and Avin, 2013), some authors make a distinction between these two terms. In that case, cocontraction has been defined as the deliberate concurrent activation of two antagonistic muscle groups with the purpose of stiffening a joint (Nielsen and Kagamihara, 1992). This condition occurs when an individual attempts stabilize a joint in anticipation of an unpredictable perturbation. For example, an individual standing upright on an unstable surface (foam mat) will cocontract lower leg muscles (ankle plantarflexors and dorsiflexors) much more so than when standing on a stable surface (Baudry and Duchateau, 2012). As the control mechanisms may differ in these two conditions (Nielsen, 1998; Crone and Nielsen, 1994), the current paper focuses on coactivation, which occurs in most of our contractions and movements of daily living.

2.2. Functional role of coactivation

In a series of experiments, Solomonow and colleagues (Baratta et al., 1988; Hirokawa et al., 1991; Solomonow et al., 1988) reported that the pattern of antagonist coactivation in the elbow and knee muscles during maximal isokinetic movements was inversely related to the variations in the moment arm over the range of motion, suggesting that antagonist muscles exert a nearly constant opposing torque during the movement. Accordingly,

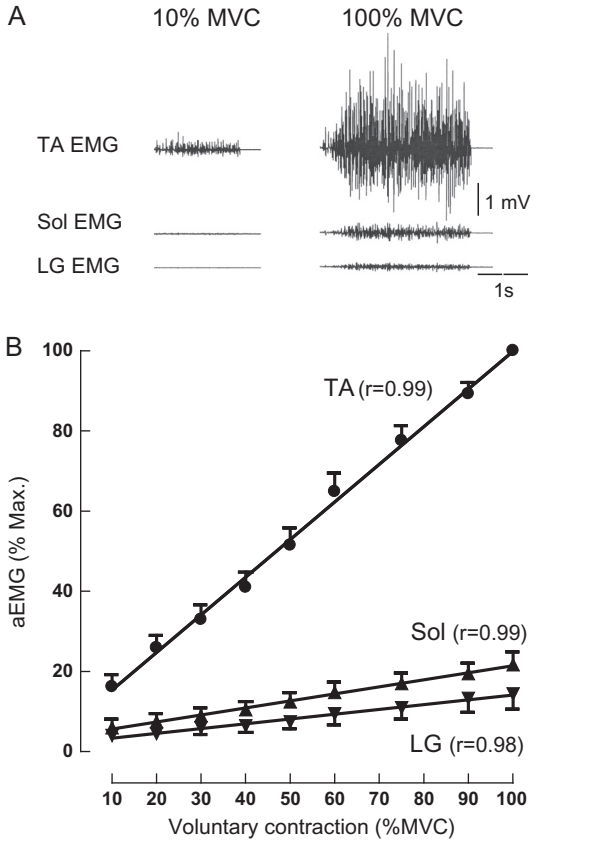

Fig. 1. aEMG – torque relation in agonist and antagonist muscles during isometric ankle dorsiflexion. A, representative traces (A), in one subject, of interference EMG activity in the tibialis anterior (TA), soleus (Sol), and lateral gastrocnemius (LG) muscles during isometric dorsiflexions at 10% and 100% of maximal voluntary contraction (MVC). (B), Relation between average EMG (aEMG) as a function of the dorsiflexion torque for the tibialis anterior (●), soleus (▲) and lateral gastrocnemius (▼) muscles. Data (mean ± SEM; $n = 10$) are expressed as a percentage of their respective values obtained during an MVC and are best fitted by linear relations with r values in brackets.

these authors attributed the role of coactivation to augment ligament function in maintaining joint stability and to equalize the pressure distribution over the articular surface. They also provided evidence that coactivation prevents relative bone displacement (Baratta et al., 1988; Solomonow et al., 1987).

The degree of coactivation does not remain constant across the life span; it has been shown to decrease progressively during childhood (Grosset et al., 2008) and to increase with ageing (Hortobagyi and De Vita, 2006; Klein et al., 2001). Coactivation can also be modified by chronic changes in physical activity. For example, it was found to decrease in the first two weeks of a strength-training program (Carolan and Cafarelli, 1992). Although a decrease in coactivation may, to a certain point, favour the action of the agonist muscles by reducing the opposing force, a minimal amount of antagonist activation appears necessary to optimize torque production by the agonist muscles (Hasan, 2005; Hortobagyi and De Vita, 2006; Loram et al., 2014).

2.3. Cross-talk

Surface EMG is commonly used to quantify muscle activity and thus the level of antagonist coactivation. An important issue associated with EMG is the possible contamination of the recordings by the activity of nearby active muscles. This is classically referred to as "cross-talk" (De Luca and Merletti, 1988; Farina et al., 2004;