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Influence of stimulus intensity on electromechanical delay and its mechanisms

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ABSTRACT

Electromechanical delay (EMD) is the time lag between muscle activation and force development. Using very high frame rate ultrasound, both electrochemical and mechanical processes involved in EMD can be assessed. Percutaneous electrical stimulations at submaximal intensity are often used to stimulate a specific target muscle. The aim of this study was to determine whether stimulus intensity alters the delay between stimulation and the onset of muscle fascicules motion (Dm), the onset of myotendinous junction motion (Dt), and force production (EMD). Ten participants underwent two electrically evoked contractions, with the probe maintained either the biceps brachii muscle belly or the distal myotendinous junction of the biceps brachii, for six stimulus intensities (30%, 50%, 70%, 90%, 110% and 130% of the lowest intensity inducing the maximal involuntary force production, Imax). In addition, inter-day reliability was tested in nine participants at both 70% and 90% of Imax. Dm, Dt and EMD were significantly longer ($p < 0.001$) at very low (30% and 50% of Imax) compared to higher intensities (70%, 90%, 110% and 130% of Imax). Inter-day reliability of EMD, Dm, and Dt was good (coefficient of variation ranged from 6.8% to 12.5%, i.e. SEM lower than 0.79 ms). These results indicate that the stimulus intensity needs to be standardized to perform longitudinal evaluation and/or to make between-subject comparisons.

1. Introduction

Electromechanical delay (EMD) is the time lag between muscle activation and force development (Cavanagh and Komi, 1979) and is influenced by both electrochemical processes (e.g., synaptic transmission, excitation–contraction coupling) and mechanical processes (force transmission along the active and passive fraction of the series elastic component, SEC) (Cavanagh and Komi, 1979; Sasaki et al., 2011). Using very high frame rate ultrasound (4 kHz), Nordez et al. (2009) recently determined the relative contribution of these processes to EMD during electrically evoked contractions. More precisely, by measuring the onset of motion for the muscle fascicles and myotendinous junctions of the gastrocnemius medialis they concluded that 47.5% of the total EMD was due to propagation of force along the passive part of the series elastic component (≈20.3% for aponeurosis and ≈27.6% for tendon) (Nordez et al., 2009). Since EMD is modified in case of pathology [e.g., neuropathy (Granata et al., 2000), myopathy (Orizio et al., 1997)] or by training regime (Linford et al., 2006; Grosset et al., 2009), this innovative non-invasive methodology has been proposed to be useful for evaluating the effects of neuromuscular disorders or training/rehabilitation protocols (Hug et al., 2011a).

Because quantification of EMD during voluntary contraction presents some drawbacks associated with the difficulty in precisely detecting the beginning of muscle activation (Hug et al., 2011b), EMD is often quantified during involuntary muscle contractions such as tendon reflex (Häkkinen and Komi, 1983; Zhou et al., 1995; Moore et al., 2002), electrical nerve stimulation (Muro and Nagata, 1985; Grosset et al., 2009; Hopkins et al., 2007; Yavuz et al., 2010), or percutaneous muscle electrical stimulation (Zhou et al., 1995; Muraoka, 2004; Nordez et al., 2009; Hug et al., 2011a; Sasaki et al., 2011). Among them, percutaneous stimulation is preferable because it allows the clinician/researcher to study the EMD of a specific target muscle (Muraoka, 2004; Nordez et al., 2009; Sasaki et al., 2011). However, it is unclear if the stimulus intensity alters EMD. This information is of great interest because performing experiments at submaximal intensities would both limit the discomfort associated with the electrical stimulation and limit activation of adjacent muscles.

Focusing on these potential outcomes, the purpose of the present experiment was to determine whether stimulus intensity alters electromechanical delay in biceps brachii. Using very high frame rate ultrasound, we measured the delay between muscle stimulation and (i) the onset of muscle fascicules motion (Dm), (ii) the onset of myotendinous junction motion (Dt), and (iii) force production (i.e., EMD). It allowed us to isolate the putative effect of intensity on the main structures/mechanisms of EMD. As percutaneous electrical stimulation activates muscles with random and non-selective muscle recruitment in terms of both fiber type (Gregory

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and Bickel, 2005) and spatial organization (Adams et al., 1993), we hypothesised that electrochemical processes are not affected by the stimulation intensity. On the other hand, it seems unclear whether muscle force transmission velocity is influenced by stimulation intensity.

2. Materials and methods

2.1. Participants

Ten active males volunteered to participate in the present study (age: 22.9 ± 2.2 years, height: 181 ± 7.7 cm, body mass: 75.8 ± 8.4 kg). They were informed of the possible risk and discomfort associated with the experimental procedures prior to giving their written consent to participate. This study was conducted according to the Declaration of Helsinki (last modified 2004) and has been approved by the local ethics committee.

2.2. Instrumentation

2.2.1. Ergometer

A schematic representation of the experimental set-up is depicted in Fig. 1. Participants sat on an isokinetic dynamometer (Biodex System 3 Research, Biodex Medical, Shirley, USA) with shoulder abducted at 90° and forearm placed in a 90 flexed position with the wrist in a neutral position. Because of the lack of sensitivity of the isokinetic ergometer to precisely detect the onset of elbow flexion force, a force transducer (SML-50, Interface, Arizona, USA) was incorporated in the ergometer and connected with Velcro straps to the wrist to ensure constant contact (Fig. 1). Isometric elbow flexion force was digitized at a sampling rate of 5 kHz (MP36, BIOPAC, Goleta, California).

2.2.2. Electrical stimulation

Elbow flexion was initiated by means of percutaneous electrical stimulation over the biceps brachii. A constant current stimulator

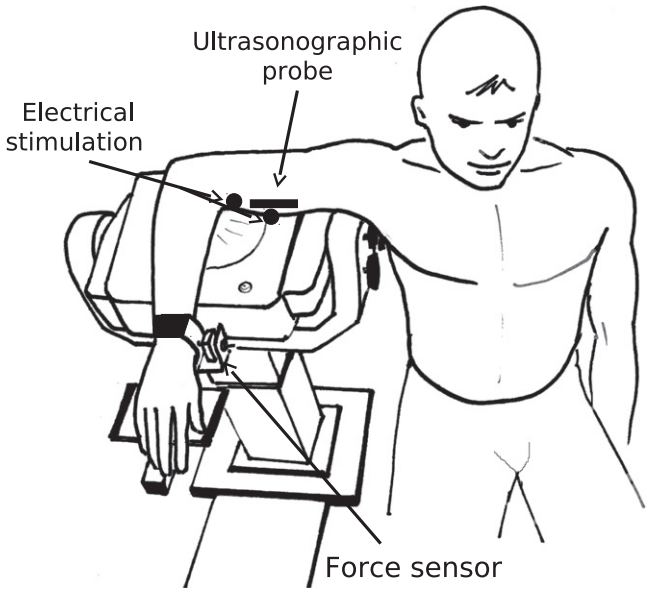

Fig. 1. Schematic representation of the experimental setup. Positioning of the subject with shoulder abducted 90° and forearm placed in a 90 flexed position. The wrist was directly in contact with a force sensor and velcro straps ensured constant contact. Elbow flexion was initiated by percutaneous electrical stimulation over the biceps brachii using two electrodes placed on the motor point and proximal portion of biceps brachii. Each subject underwent two bouts composed of two electrically evoked contractions with the echographic probe maintained over either the biceps brachii muscle belly or the distal myotendinous junction of the biceps brachii muscle.

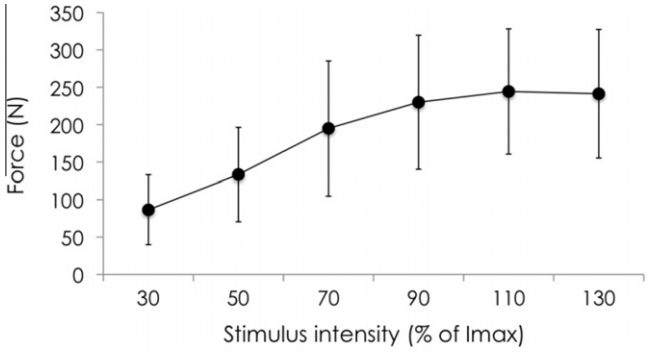

Fig. 2. Dependence of peak twitch force on the stimulus intensity. Values are means ± SD. Relationship between force (Newtons, N) and stimulus intensity (% Imax).

(Digitimer DS7A, Digitimer, Letchworth Garden City, UK) delivered a single electrical pulse (pulse duration = 500 μs, 400 V) through two electrodes (2 × 1.5 cm, Compex, Annecy-le-vieux, France) placed on the main motor point and proximal portion of biceps brachii (Hug et al., 2011a). The motor point was considered as the location inducing the strongest twitch with the lowest electrical stimulation. To determine the minimal stimulation intensity required to induce the maximal elbow flexion force (Imax), the output current was incrementally increased (incremental step of 5 mA) until a maximum force output was reached (Fig. 2). The mean Imax was 98.5 ± 11.3 mA.

2.2.3. Ultrasonography

A very high frame rate ultrasound scanner (Aixplorer, version 4.2, Supersonic Imagine, Aix en Provence, France) coupled with a linear transducer array (4–15 MHz, SuperLinear 15–4, Vermon, Tours, France) was used in « research » mode to acquire raw radio frequency (RF) signals at 4 kHz.

2.2.4. Synchronisation

At the start of each ultrasound acquisition, the scanner sent a transistor–transistor logic (i.e., TTL) pulse to a train/delay generator (Digitimer Ltd, DG2A, Welwyn Garden City, England) which generated a TTL pulse to the electrical stimulator with a 48.00-ms delay to have a sufficient baseline to detect the onset of tissue motion. To check the absence of desynchronization throughout the experiments, TTL pulses from both the ultrasound scanner and the train/delay generator were recorded using the same device as for the force measurements (MP36, Biopac, Goleta, California).

2.3. Protocol

After the previously described recruitment ramp, six electrically evoked contractions were performed at six intensities (30%, 50%, 70%, 90%, 110%, and 130% of Imax). They were applied in a randomized order with 1-min rest between each and two trials were performed for each stimulation intensity (designated as muscle trials and tendon trials). During the muscle and tendon trials, the echographic probe was maintained parallel to the muscle fascicles and on the previously localized distal myotendinous junction of the biceps brachii, respectively. Participants were instructed to be fully relaxed prior to each stimulation.

2.4. Data processing

The data processing was performed using standardized Matlab scripts (The Mathworks, Nathick, USA). First, ultrasonic raw data (i.e., RF signals) were used to create echographic images by apply-