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ELECTROMECHANICAL DELAY IN BICEPS BRACHII ASSESSED BY ULTRAFAST ULTRASONOGRAPHY

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ABSTRACT: Using ultrasound we tested the utility of determining the relative contribution of the main muscle structures/mechanisms to the electromechanical delay in the biceps brachii. Nine subjects underwent electrically evoked contractions with the echographic probe maintained over the muscle and the myotendinous junction. No difference was found between the onset of muscle fascicle motion (Dm, 5.57 ± 1.37 ms) and the onset of myotendinous junction motion (Dt, 5.47 ± 1.38 ms), whereas significant differences were found between Dm/Dt and electromechanical delay (approximately 10 ms). Electromechanical delay can be used as a model for studying the effects of neuromuscular disorders or various constraints that affect excitation–contraction coupling and/or muscle force transmission.

Electromechanical delay (EMD) represents the lag time between the onset of electromyographic activity and the onset of the muscle mechanical response.¹ EMD may be influenced by several structures and mechanisms, such as the propagation of the action potential, excitation–contraction coupling, and stretching of the series elastic component (SEC) by the contractile elements.¹ Using ultrasound with a very high frame rate, one recent study² determined the onset of motion for muscle fascicles and myotendinous junctions (MTJs) during contraction of the gastrocnemius medialis (GM) evoked by myoelectrical stimulation. Using this non-invasive methodology it is now possible to determine the relative contribution of the main structures/mechanisms to the EMD. Specifically, it can be used as a model for studying the effects of neuromuscular disorders or various constraints on muscle function. In the case of some myopathies, however, such as Duchenne muscular dystrophy, calf muscles can be difficult to study due to patients' inability to fully extend their legs. In addition, biceps brachii (BB) and GM muscles display different fiber architectures (fusiform and pennated, respectively) that might induce differences in muscle force transmission processes. Thus, the EMD and the relative contribution of the main

structures/mechanisms to the EMD are probably different between these muscles.

Therefore, the aim of this study was to test the utility of determining the relative contribution of the main structures/mechanisms to the EMD using very high frame rate ultrasound in a muscle more accessible in clinical practice (i.e., BB). Performing the study on a fusiform muscle may also have implications for the understanding of muscle force transmission processes.

METHODS

Subjects. Nine healthy men (28.8 ± 4.4 years) volunteered to participate in this study and signed a document of informed consent. The study was conducted according to the Declaration of Helsinki guidelines and has been approved by the local ethics committee.

Measurements. A home-made ergometer was used to measure the force produced by elbow flexors.³ Briefly, subjects sat on a chair with their right upper arm and forearm placed in a 90° flexed position with the wrist supinated. Force was recorded at the level of the wrist using a force sensor (ZF200kg; Scaime, Annemasse, France) at a sampling rate of 5 kHz (MP36; BIOPAC, Goleta, California).

A very high frame rate ultrasound device (64 channels; Lecoer Electronique, Chuelles, France) was used as described previously.² Briefly, radio-frequency (RF) images were acquired at 4 kHz. Then, correlation algorithms between windows of different RF images gave the displacement field due to the contraction.⁴

Protocol. Selective contraction was elicited by means of percutaneous electrical stimulation (one pulse duration = 200 μ s; DS7A; Digitimer, Welwyn Garden City, UK) delivered through two electrodes placed on the motor point and proximal portion of BB muscle. Muscle stimulation was started using a trigger delivered by the ultrafast echographic device.

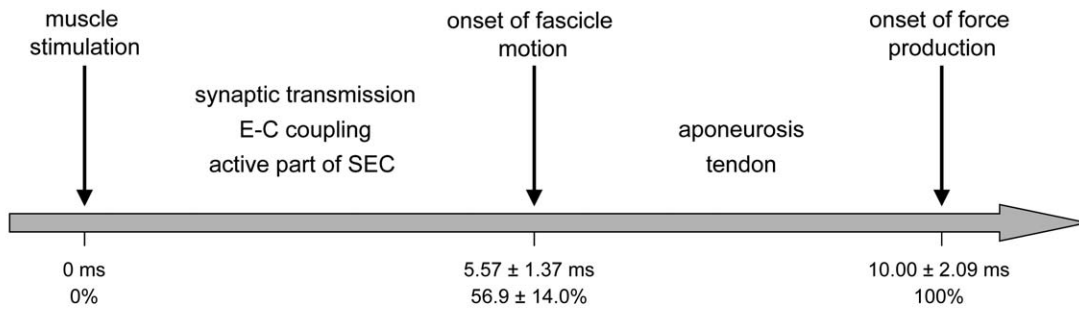
For each subject, two bouts (designated as muscle trials and tendon trials), composed of two electrically evoked contractions, were performed. During the muscle and tendon trials, the echographic probe was maintained on the muscle belly (parallel

Abbreviations: BB, biceps brachii; Dm, onset of muscle fascicle motion; Dt, onset of myotendinous junction; EMD, electromechanical delay; GM, gastrocnemius medialis; MTJ, myotendinous junction; RF, radio-frequency; SEC, series elastic component

Key words: echography; maximal muscle shortening velocity; muscle; slack length; tendon

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A. muscle trials



B. tendon trials

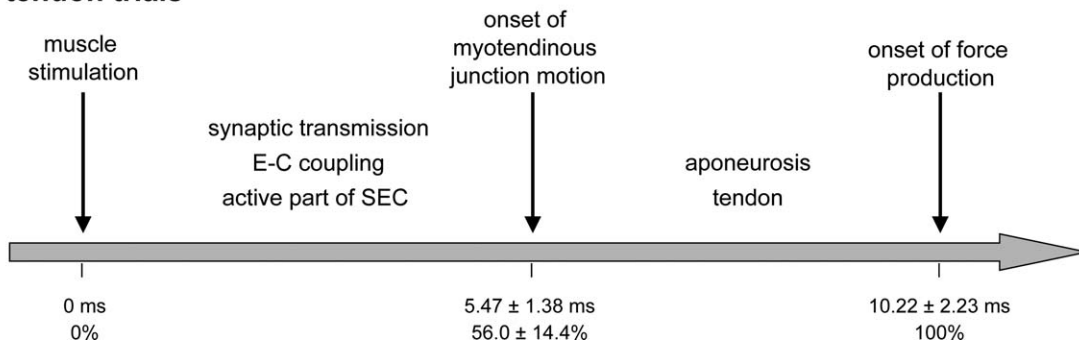


FIGURE 1. Schematic representation of the time lag between the muscle stimulation and onset of fascicle motion, musculotendinous junction motion, and external force. Averaged (\pm standard deviation) onset times across subjects are depicted for muscle trials (**A**) (echographic probe maintained over the muscle belly) and tendon trials (**B**) (echographic probe maintained over the myotendinous junction). Note that the contribution of the aponeurosis in EMD is probably small in this fusiform muscle. EMD, electromechanical delay; Dm, onset of muscle fascicle motion; Dt, onset of myotendinous junction motion.

to the muscle fibers) and on the previously localized distal MTJ of the BB muscle, respectively.

Processing. Ultrasound raw data were processed as described in detail by Nordez et al.² Echographic images were used to determine the region of interest for each contraction (between the two aponeuroses for muscle trials, and on the distal MTJ for tendon trials). Then, a processing method similar to Doppler² was used to measure the tissue motion. EMD was calculated as the time lag between the electrical stimulation and the onset of force production. We also determined the delay between the electrical stimulation and the onset of muscle fascicle motion (Dm, for muscle trials), and between the electrical stimulation and the onset of MTJ motion (Dt, for tendon trials).

Statistical Analysis. Values are reported as mean \pm standard deviation. To determine the repeatability of all measurements, the standard error of measurement (SEM) and the coefficient of variation (CV) were calculated for the two contractions within each bout.⁵ The Dm, Dt, and EMD values were averaged between the two trials and then compared using analysis of variance for repeated measures with orthogonal contrasts as the post hoc test.

RESULTS

SEM and CV values calculated for the two electrically evoked contractions of the muscle trials were 0.25 ms and 4.6% for Dm, and 0.41 ms and 4.4% for the EMD. For the tendon trials, SEM and CV were higher: 1.28 ms and 19.5% for Dt, and 1.01 ms and 11.1% for the EMD. No significant difference ($P > 0.05$) was found between Dm (5.57 ± 1.37 ms) and Dt (5.47 ± 1.38 ms), whereas significant ($P < 0.001$) differences were found between Dm and EMD, and between Dt and EMD. Averaged results across the two contractions for each of the 9 subjects are depicted in Figure 1.

DISCUSSION

EMD values reported in BB (10.00 ± 2.09 ms) are similar to those previously reported by Nordez et al.² (11.63 ± 1.51 ms) in GM. The aim of our work was to assess Dm and Dt by using very high frame rate ultrasound. Dm could be mainly attributed to synaptic transmission, excitation-contraction coupling, and force transmission along the active part of the SEC.² Because the Dm values reported here (5.57 ± 1.37 ms representing $56.9 \pm 14.0\%$ of EMD) are very similar to those reported previously² (6.05 ± 0.64 ms representing $52.5 \pm 5.9\%$ of EMD), one would expect that these mechanisms would be similar between GM and BB.

However, in contrast to results obtained by Nordez et al.,² Dm was not different from Dt. This could be explained by the fact that, in the physiological range, BB length is always beyond the slack length.⁶ Consequently, the muscle–tendon unit was slack during our experiments, leading one to expect that the onset of motion of the MTJ in BB corresponds to a rigid solid displacement due to fascicle shortening. The delay between Dm and the onset of force production (EMD–Dm, i.e., 4.43 ± 1.95 ms, $43.1 \pm 13.9\%$ of the EMD) should be attributed to both unloaded muscle shortening until the slack length is reached and force propagation along the passive SEC (aponeurosis and tendon). This result suggests that, depending on the muscle length, the EMD is also influenced by the maximal shortening muscle velocity.

In conclusion, EMD could be used as a model for studying the effects of neuromuscular disorders or

various constraints that affect excitation–contraction coupling and/or the muscle force transmission.

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