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Effect of transient vascular occlusion of the upper arm on motor evoked potentials during force exertion



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ABSTRACT

We previously observed that transient vascular occlusion in volunteers increased the estimation of force exertion with no change in peripheral nerves or muscles. We hypothesized that the primary factor responsible for the overestimation of force exertion during occlusion was the centrally generated motor command, as hypothesized by McCloskey et al. (1974) and McCloskey (1978, 1981). In the present study, we tested the hypothesis that transient vascular occlusion increases the excitability of the primary motor cortex (M1) during force exertion. Healthy human volunteers lay on a bed and squeezed a dynamometer in their right hand. Repetitive gripping forces were exerted at 20%, 40%, or 60% of maximum force, with or without transient (20 s) vascular occlusion of the proximal portion of the right upper arm. During the task, single-pulse transcranial magnetic stimulation was applied to the contralateral M1 to induce motor evoked potentials (MEPs) in the flexor carpi ulnaris (FCU) muscle. The MEP amplitudes were enhanced with occlusion under all conditions, with the exception of 60% contraction. In contrast, no significant difference was observed between the MEP amplitudes obtained from the occluded or non-occluded, relaxed FCU muscle. These results suggest that transient vascular occlusion increases the excitability of M1 only during force exertion.

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1. Introduction

Low-to-moderate intensity resistance exercise with vascular occlusion has been shown to induce increased muscle mass and strength, comparable to that observed after conventional heavy resistance training (Takarada et al., 2000). The physiological mechanism underlying this phenomenon is still not completely understood, but we noticed that subjects reported the need for greater force to lift a weight when the resistance exercise was begun with vascular occlusion.

We previously examined the effects of transient vascular occlusion on force estimation after the application of a pneumatic tourniquet to the proximal end of the upper arm. We observed that such occlusion increased the perceived magnitude of exerted handgrip forces without causing any accompanying changes in activities on electromyography (EMG) or in the efferent/afferent activity of the median nerve (Takarada et al., 2006).

We assumed that the primary factor responsible for the overestimation of force exertion during transient occlusion was the centrally generated motor command, as hypothesized by McCloskey (1978, 1981). Taking into consideration that the primary motor cortex (M1) is a strong candidate for the origin of the motor command, we hypothesized that corticospinal excitability should also be increased by vascular occlusion during force exertion. To evaluate the effect of vascular occlusion on corticospinal excitability, we elicited motor evoked potentials (MEPs) in a hand muscle by applying transcranial magnetic stimulation (TMS) over M1.

Of particular importance was the observation that force overestimation took place rapidly, within 1 min of starting the occlusion (Takarada et al., 2006). However, no previous studies have demonstrated that changes in MEP by vascular occlusion occurred within such a short time scale. In fact, according to McNulty et al. (2002), a decrease in MEPs of hand muscles distal to the ischemic block was observed after 20 min. Of note, this previous study only examined the change in MEP under the resting condition and did not focus on changes in MEP during force exertion. Thus, we further hypothesized that enhancement in MEP by vascular occlusion is only observed during force exertion.

Abbreviations: MEPs, motor evoked potentials; FCU, flexor carpi ulnaris; TMS, transcranial magnetic stimulation; MVCs, maximum voluntary contractions; RMT, resting motor threshold; bEMG, background EMG.

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2. Materials and methods

2.1. Subjects

A total of 12 right-handed volunteers [10 men and 2 women; age, 20–40 years; mean age \pm standard deviation (SD), 28.0 \pm 9.2 years] with no history of neurological or other diseases participated in this study. Six volunteers participated in Experiment 1, and the remaining six participated in Experiment 2. All participants provided written informed consent. The study was conducted in accordance with the Declaration of Helsinki, and all protocols were approved by the Ethics Committee of the Faculty of Sport Sciences, Waseda University (protocol number 104).

2.2. Force measurement

Handgrip force exerted by the right hand was measured using a handgrip device with a strain gauge (KFG-5-120-C1-16; Kyowa Electronic Instruments Co., Ltd., Tokyo, Japan). The measured force was amplified (AD240-A; TEAC Instruments Co., Kawasaki, Japan), digitized (400 Hz), and recorded on the hard disk of a data acquisition computer (Panasonic CF-R4).

2.3. EMG measurement

Surface EMG was recorded from the skin surface of the abductor pollicis brevis and flexor carpi ulnaris (FCU) muscles in the right upper extremity. Bipolar surface electrodes were placed over the bellies of the muscles. EMG signals were amplified, filtered (bandpass, 15 Hz to 10 kHz), and digitized (2 kHz, SYNAMP; Neuroscan, Herndon, VA, USA).

2.4. Experimental procedure

Subjects were positioned in a supine position on a bed. Vascular occlusion was induced by tightly applying a pneumatic tourniquet to the proximal end of the right upper arm (Fig. 1A).

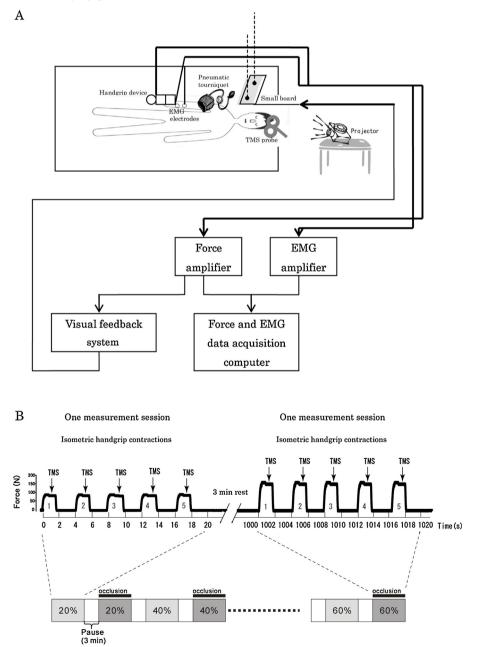


Fig. 1. (A) Schematic representation of data collection systems. (B) Task procedure.

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