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Cathodal transcranial direct current stimulation over the Cz increases joint flexibility

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ABSTRACT

Joint flexibility depends on both mechanical and neural factors. However, the contribution of neural factors is not fully understood. To test the hypothesis that the sensorimotor cortex is involved in joint flexibility, we investigated whether transcranial direct current stimulation (tDCS) over the Cz modifies ankle and wrist flexibility in healthy human participants. In eight male participants, range of motion of the left ankle and wrist were measured during a passive-dorsiflexion test. We also assessed passive torque, which represents involuntary resistance to dorsiflexion at the ankle. Participants performed passive-dorsiflexion tests before and after anodal, cathodal, and sham tDCS over the Cz. The current was applied for 10 min with an intensity of 2.0 mA during anodal and cathodal tDCS. Cathodal tDCS resulted in a 10.5% increase in range of motion of the ankle, but no significant increase in range of motion of the wrist. Neither anodal nor sham tDCS had a significant effect. Cathodal tDCS over the Cz may have affected neural factors, such as perception of joint angle or pain, because the passive torque at 0°, 5°, 10°, and 15°, which indicates mechanical effects, did not change. These results suggest that the sensorimotor cortex is involved in joint flexibility.

1. Introduction

Flexibility is one of the components of fitness that is thought to be associated with exercise performance (Wilson et al., 1992) and incidence of muscular injury (Wilson et al., 1991). Flexibility is commonly evaluated by assessing joint range of motion (ROM), which depends on both mechanical and neural factors (Avela et al., 1999; Behm et al., 2013; Evetovich et al., 2003; Guissard and Duchateau, 2004, 2006; Guissard et al., 2001; Magnusson et al., 1996; Mizuno et al., 2013a,b; Morse et al., 2008; Wilson et al., 1992). While many studies of the mechanical factors have concluded that the muscle-tendon unit is important for joint ROM (Evetovich et al., 2003; Magnusson et al., 1996; Mizuno et al., 2013a,b; Morse et al., 2008; Wilson et al., 1992), the neural factors that affect joint ROM have not been fully investigated. For instance, it has been demonstrated that stretch tolerance, which means tolerance to stretching-induced pain, is one of the important limiting factors that affect the increase in joint ROM after static stretching (Magnusson et al., 1996; Mizuno et al., 2013a,b). A previous study has reported that the increase in joint ROM immediately after and 5 min after static stretching for 5 min was due to both changes in mechanical factors related to the muscle-tendon unit and increased stretch tolerance, while the increase in joint ROM 10 min and 15 min after stretching was due to increased stretch tolerance alone (Mizuno et al., 2013a). It has also been suggested that stretch tolerance might be related to the central nervous system (Magnusson, 1998), but the mechanism of stretch tolerance is still unclear. Furthermore, although a few studies have demonstrated the effect of spinal excitability on joint ROM, as measured using the H-reflex or tendon reflex (Avela et al., 1999; Behm et al., 2013; Guissard and Duchateau, 2004; Guissard et al., 2001), the contribution of the cerebral cortex to joint ROM remains unknown. However, because the cerebral cortex is involved in proprioception (Lephart et al., 1998) and some imaging studies have reported that the primary somatosensory cortex (S1) is involved in both pain perception and limb movement (Antal et al., 2008; Bingel et al., 2004; Bushnell et al., 1999; Dobkin et al., 2004; Francis et al., 2009; MacIntosh et al., 2004; Peyron et al., 2000; Porro et al., 2002), the excitability of the cerebral cortex associated with proprioception and cognitive function may also affect joint ROM.

Recently, transcranial direct current stimulation (tDCS), a non-invasive neuromodulation technique, has been used to modify

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cortical excitability (Nitsche and Paulus, 2000; Priori et al., 1998). tDCS modulates regional brain activity by altering the membrane potential of neurons (Liebetanz et al., 2002; Nitsche and Paulus, 2000). Furthermore, tDCS can increase or decrease cortical excitability in a polarity-dependent manner (Boggio et al., 2008; Fregni et al., 2006a,b; Jeffery et al., 2007; Matsunaga et al., 2004; Nitsche and Paulus, 2000; Tanaka et al., 2009); that is, anodal tDCS can enhance cortical excitability, while cathodal tDCS can diminish it. Polarity-dependent changes in cortical excitability induced by tDCS are mediated by the activity of sodium and calcium ion channels in the neuronal membrane, and by the effectivity of receptors for N-methyl-D-aspartate-neurotransmitters (Liebetanz et al., 2002; Nitsche et al., 2003). Using this technique, Antal et al. (2008) demonstrated that cathodal tDCS over S1 decreased laser-stimulated pain perception. A recent meta-analysis also reported that anodal tDCS over the primary motor cortex (M1) increased sensory and pain thresholds, while anodal tDCS over S1 increased pain threshold (Vaseghi et al., 2014). These studies have suggested that modulation of S1 and/or M1 is related to pain perception (Antal et al., 2008; Vaseghi et al., 2014), although the effect of polarity remains controversial.

As mentioned above, cognitive function may affect joint ROM, especially considering that pain perception is a limiting factor for joint flexibility (Mizuno et al., 2013a,b). Therefore, we predicted that tDCS stimulation to the S1 and/or M1 would modulate joint ROM as a result of modulating pain perception. Thus, in this study, to test the hypothesis that the sensorimotor cortex is involved in joint flexibility, we examined whether the application of tDCS over the sensorimotor foot area could modify ankle ROM in healthy participants. The position of the sensorimotor foot area corresponds to the Cz, in reference to a previous study (Marshall et al., 2013). We positioned the electrode over the Cz to stimulate the foot sensorimotor cortex.

2. Materials and methods

2.1. Participants

Ten healthy men volunteered for the study, and the final cohort consisted of eight men (mean ± SD; age, 25 ± 3 years; height, 170.8 ± 2.9 cm; weight, 65.3 ± 5.0 kg). Two participants were excluded based on the results of the Smirnov–Grubbs rejection test ($p < 0.01$) because the coefficient of variance (CV) and the value range for ankle ROM, wrist ROM, or passive torque at maximal dorsiflexion angle during pre-stimulation over a 3-trial period were too high [Subject 1: CV for ankle ROM, outlier (34.7), overall (10.4 ± 9.9); Subject 2: range for passive torque at maximal dorsiflexion angle, outlier (17.4 Nm), overall (4.9 ± 5.2 Nm)]. The results of tDCS for the outliers are summarized in the Supplementary Table 1. All participants had specifically studied sports science in graduate school. These men were right-leg-dominant, and none had a history of recent musculoskeletal injury or neuromuscular disease specific to the lower limb. They also had no history or current signs or symptoms of neurological or psychiatric disorders. The participants provided written informed consent for their participation in the experiments, which were conducted according to the principles of the Declaration of Helsinki. All participants were fully informed of the purposes, procedures, and possible risks of the study. The experimental protocol was approved by the Human Subjects Committee at Chukyo University Graduate School of Health and Sports Sciences.

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2.2. Experimental design

The present study was conducted as a single-center trial, as all participants were evaluated at a single study location. The experiment was designed as a double-blind trial. The order of tDCS stimulation was randomized, and the experimental design also contained sham stimulation as a control trial. All subjects repeatedly participated in anodal tDCS, cathodal tDCS, and sham tDCS experiments.

The participants visited the laboratory on four occasions, and the visits were separated by at least 24 h to prevent interference effects, based on a previous study that indicated that the effect of anodal tDCS at 2 mA for 10 min diminished 60 min after stimulation (Tanaka et al., 2009). The first visit involved a familiarization trial, and the subsequent three visits included the following experimental conditions: (a) anodal tDCS, (b) cathodal tDCS, and (c) sham tDCS. During the familiarization trial, each participant practiced the passive-dorsiflexion test to minimize any potential learning effects and to adjust to the procedures. During the experimental trials, the participants underwent a pre-stimulation passive-dorsiflexion test, followed by one of the three types of stimulation, and a post-stimulation passive-dorsiflexion test (Fig. 1a). The post-stimulation passive-dorsiflexion test was performed immediately after 10 min-tDCS. During the passive-dorsiflexion test, we measured passive torque (i.e., involuntary resistance to passive dorsiflexion) and ROM of the ankle and wrist. Before the pre-stimulation passive-dorsiflexion test, participants walked on a treadmill at 100 m/min for 5 min as a warm-up.

2.3. Passive-dorsiflexion test

To determine passive torque and joint ROM, each participant underwent a passive-dorsiflexion test. The passive-dorsiflexion test was performed using an approach similar to that described in previous studies (Mizuno et al., 2013a,b; Morse et al., 2008). To assess ankle ROM, participants were secured to an isokinetic machine (Biodex System 3, Biodex, NY, USA) with their knee in full extension and the footplate fixed to their left foot. The lateral malleolus was aligned with the axis of the dynamometer. In this study, all reported ankle angles reflect the angle of the footplate, and the ankle angle was defined as 0° when the footplate was perpendicular to the floor. To assess wrist ROM, the participants were secured to an isokinetic machine (Biodex System 3, Biodex, NY, USA) with their left elbow flexed 90° and the grip held in their left hand. The styloid process of the ulna was aligned with the axis of the dynamometer. All reported wrist angles reflected the angle of the wrist attachment, and the wrist angle was defined as 0° when the wrist attachment was parallel to the floor. Values were defined as positive for dorsiflexion of the ankle and the wrist. The foot and wrist of the participant were passively and isokinetically dorsiflexed at a speed of 1°/s from -30° for the foot and from 0° for the wrist, to the angle at which the participant felt discomfort and stopped the dynamometer by activating a safety trigger (Fig. 1b). To prevent reflex contraction due to pain, we defined the angle at which the participant subjectively felt discomfort as the maximal dorsiflexion angle. The maximal angle of the footplate or the wrist attachment was defined as the joint ROM. During this test, the passive torques generated on the footplate were measured when the ankle was submaximally dorsiflexed (i.e., 0°, 5°, 10°, and 15°) and at maximal dorsiflexion.

To correct for the effects of the weight of the attachment and the lower limb on torque, all measurements of passive torque were gravity corrected. Gravity correction was performed at -30° because passive torque around the ankle has been shown to be near zero at this angle (Kawakami et al., 1998). Therefore, to correct for gravity, we set the passive torque at zero at 30° of