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The effect of active pedaling combined with electrical stimulation on spinal reciprocal inhibition

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ABSTRACT

Objective: Pedaling is widely used for rehabilitation of locomotion because it induces muscle activity very similar to locomotion. Afferent stimulation is important for the modulation of spinal reflexes. Furthermore, supraspinal modulation plays an important role in spinal plasticity induced by electrical stimulation. We, therefore, expected that active pedaling combined with electrical stimulation could induce strong after-effects on spinal reflexes.

Design: Twelve healthy adults participated in this study. They were instructed to perform 7 min of pedaling. We applied electrical stimulation to the common peroneal nerve during the extension phase of the pedaling cycle. We assessed reciprocal inhibition using a soleus H-reflex conditioning-test paradigm. The magnitude of reciprocal inhibition was measured before, immediately after, 15 and 30 min after active pedaling alone, electrical stimulation alone and active pedaling combined with electrical stimulation (pedaling + ES).

Results: The amount of reciprocal inhibition was significantly increased after pedaling + ES. The after-effect of pedaling + ES on reciprocal inhibition was more prominent and longer lasting compared with pedaling or electrical stimulation alone.

Conclusions: Pedaling + ES could induce stronger after-effects on spinal reciprocal inhibitory neurons compared with either intervention alone. Pedaling + ES might be used as a tool to improve locomotion and functional abnormalities in the patient with central nervous lesion.

1. Introduction

Reciprocal inhibition (RI) between agonist and antagonist muscle is mediated by the Ia inhibitory interneurons. RI modulates reciprocating lower extremity movements such as walking and pedaling. In humans with incomplete spinal cord injury (SCI), spinal inhibitory reflexes mediated by GABA and glycine are reduced (Calancie et al., 1993; Boorman et al., 1996; Okuma et al., 2002). Reduction in disynaptic glycinergic reciprocal Ia inhibition is thought to contribute to abnormal muscle coactivation during locomotion (Fung and Barbeau, 1989).

It has been reported that spinal activity-dependent plasticity could be induced with pedaling (Motl et al., 2003) and electrical stimulation (Perez et al., 2003). Pedaling is a rhythmic movement that induces muscle activity very similar to locomotion. It is widely used for rehabilitation of patients with central nervous system (CNS) lesions (Fujiwara et al., 2005). Phase-dependent modulation of RI can be seen during pedaling (Pyndt et al., 2003). The after-effects of pedaling on spinal RI, however, have not been thoroughly investigated.

Sensory electrical stimulation induces long lasting plasticity in reciprocal Ia inhibition in intact humans (Perez et al., 2003). Therefore, sensory input from electrical stimulation might play a role in the induction of use-dependent plasticity.

Supraspinal modulation plays an important role in spinal plasticity induced with electrical stimulation. Fujiwara et al. (2011) demonstrated that plastic changes of spinal reciprocal inhibitory

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networks induced with sensory electrical stimulation could be modulated by transcranial direct current stimulation (tDCS), which could change the motor cortex excitability in a polarity specific manner. Christensen et al. (2000) showed that active pedaling significantly activated bilateral primary motor cortices compared with passive pedaling. It was suggested that active pedaling could increase motor cortex excitability. We therefore expected that active pedaling combined with electrical stimulation could induce stronger after-effects on spinal reciprocal inhibitory neurons when compared with either intervention alone.

To test this hypothesis, we applied the following three paradigms, active pedaling, electrical stimulation and the combination of pedaling and electrical stimulation (pedaling + ES), and studied their after-effects on spinal RI in healthy adults.

2. Methods

2.1. Participants

Twelve healthy volunteers (mean age 26.2 ± 3.6 years, eight males and four females) participated in this study. The study was approved by the institutional ethics committee (Tokyo Bay Rehabilitation Hospital, No. 4-1), and all the participants gave their written informed consent in accordance with the declaration of Helsinki.

2.2. Pedaling

We instructed the participants to perform 7 min of active pedaling at a comfortable speed (mean ± standard deviation, 57 ± 6 rpm). The pedal resistance was set at 5 Nm with an isotonic mode that maintained a constant pedal resistance throughout the pedal cycle, regardless of the pedal speed (Fujiwara et al., 2003). We used a recumbent cycle ergometer (StrengthErgo240, Mitsubishi Electric Co., Tokyo, Japan) that can achieve a highly precise load control (coefficient of variation, 5%) over a wide range of pedaling resistance (0–240 Nm), even at the lower end of resistance. It also has an adjustable seat with a backrest and seat belt for trunk stabilization. The seat height was 51 cm, and the crank length was 17 cm. The distance from the seat edge to the crank axis and the height of the pedal axis were adjusted so that the knee extension angle was −10° from full extension when the participants extended their knees maximally. Pedals with straps allowed the participants to maintain firm contact between their feet and the pedals during the experiments. The trunk was also strapped with a seat belt to the backrest, whose tilt angle was set at 10° from the horizontal to assure seating comfort during all experimental conditions.

2.3. Pedaling combined with electrical stimulation (pedaling+ES)

We instructed subjects to perform 7 min of pedaling at a comfortable speed (mean ± standard deviation, 57 ± 6 rpm) as the active pedaling paradigm. During pedaling, the crank angle was monitored. The 0° crank angle was defined as the point at which maximal hip flexion was attained during pedaling. The electrical stimulation was applied throughout the extension phase of pedaling during which the crank angle changed from 0° to 180°. Electrical stimulation was controlled by a personal computer running LabVIEW 2009 (National Instruments Corp., Austin, Texas, USA). The common peroneal nerve (CPN) was stimulated with adhesive electrodes (HV-LLPAD, 2 × 2 cm, Omuron Corp., Kyoto, Japan) just below the fibular head. The stimulus intensity was adjusted to the motor threshold level for the tibialis anterior muscle (TA) without peroneal muscle activation. The stimulation parameter was a biphasic current with a frequency of 30 Hz and a pulse duration of 0.3 ms.

2.4. Electrical stimulation (ES)

Electrical stimulation was applied for 7 min at the same stimulus interval and parameters as the pedaling + ES. The stimulus interval was determined according to each participant's comfortable speed (mean ± standard deviation, 57 ± 6 rpm), which was measured on a different day, and was set individually. Subjects sat on the ergometer with their feet fixed to the pedals, with the crank angle set at 90°.

2.5. Experimental paradigm

At intervals of over 1 week, subjects randomly received pedaling + ES, pedaling alone and electrical stimulation alone. Before, immediately after, 15 and 30 min after the three paradigms, H reflex and disynaptic RI were assessed.

2.6. Reciprocal inhibition (RI)

RI was assessed using a soleus H-reflex conditioning test paradigm. Ten conditioned and 10 test H reflexes were averaged at each time point. The H reflex was elicited by stimulating the tibial nerve at the popliteal fossa (1-ms rectangular pulse). The test soleus H reflex amplitude was maintained at 15–20% of the M max for each block of trials (Crone et al., 1990). Conditioning stimulation to the CPN was delivered below the fibular head. Stimulus intensity of the conditioning stimulation was set at the motor threshold (MT). MT was defined as a 100-μV response of the TA. The conditioning-test stimulus interval was set at 0, 1, 2 and 3 ms. The conditioning stimulation was triggered randomly (0–3 ms) once every 4 s by a personal computer with LabVIEW 2009 and was kept at this level during the experiment. The optimal interval for stimulating the CPN to produce disynaptic RI was determined at the beginning of each session and used throughout. The mean values of the test and the conditioned test H reflexes were determined. The amount of RI (%) was defined as: [(mean test H reflex amplitude − mean conditioned H reflex amplitude)/mean test H reflex amplitude] × 100.

We carefully considered the following measurements in order to maintain stable the H-reflex because H-reflexes are susceptible to neurological activity including posture and movements, volitional attention. Participants were comfortably seated in an ergometer seat with a seatbelt for trunk stabilization during measurement. The right leg was fixed to an immobile pedal with hip flexed (80°) and knee flexed (90°), and ankle was fixed at 0° dorsiflexion. These joint positions were monitored and kept during measurements. We asked participants to relax during measurements in order to avoid movement and mental focus. In addition, we carefully checked the SOL and TA EMG to confirm the resting state of these muscles. Therefore, we believe the countermeasures to minimize the influences of neurological activity.

2.7. Statistical analysis

Two-factor repeated measures ANOVA was used to analyze the effects of paradigm (pedaling + ES, pedaling only, and ES only) and time (before, immediately after, 15 min after, and 30 min after). The changes of test H amplitudes of each condition were analyzed with one-factor repeated measure ANOVA with main factor of time (before, immediately after, 15 min after, and 30 min after). A one-factor repeated measures ANOVA was used to analyze the baseline of test H amplitudes and amounts of RI. Post hoc testing to determine significant comparisons was done using two-tailed multiple *t*-test with Bonferroni correction. Statistical analyses were performed using SPSS 15.0 for Windows. A value of $p < 0.05$ was used as an indicator of statistical significance.