Training in a ballistic task but not a visuomotor task increases responses to stimulation of human corticospinal axons

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Giesebrrecht S, van Duinen H, Todd G, Gandevia SC, Taylor JL. Training in a ballistic task but not a visuomotor task increases responses to stimulation of human corticospinal axons. J Neurophysiol 107: 2485–2492, 2012. First published February 8, 2012; doi:10.1152/jn.01117.2010.—Short periods of training in motor tasks can increase motor cortical excitability. This study investigated whether changes also occur at a subcortical level. Subjects trained in ballistic finger abduction or visuomotor tracking. The right index finger rotated around the metacarpophalangeal (MCP) joint in a split joint. Surface EMG was recorded from the first dorsal interosseus. Transcranial magnetic stimulation over the back of the head (double-cone coil) elicited cervicomedullary motor evoked potentials (CMEPs) by stimulation of corticospinal axons. Responses were recorded from the relaxed muscle before, between, and after two sets of training. In study 1 (n = 7), training comprised two sets of 150 maximal finger abductions. Feedback of acceleration was provided. With training, acceleration increased significantly. CMEPs increased to 248 ± 152% (± SD) of baseline immediately after training (P = 0.007) but returned to control level (155 ± 141%) 10 min later. In study 2 (n = 7), subjects matched MCP joint angle to a target path on a computer screen. After ~30 min of training, tracking improved as shown by increased correlation between joint angle and the target pathway, reduced time lag, and reduced EMGrms. However, CMEPs remained unchanged. These results show that transmission through the corticospinal pathway at a spinal level increased after repeated ballistic movements but not after training in a visuomotor task. Thus, changes at a spinal level may contribute to improved performance in some motor tasks.

corticospinal tract; motoneuron; plasticity

IMPROVED MOTOR PERFORMANCE has long been linked to “plastic” changes in neuronal circuitry. Both structural and functional adaptation (plasticity) have been widely reported to occur in the motor cortex after different types of motor training (for review, see Sanes and Donoghue 2000; Adkins et al. 2006). These cortical adaptations are thought to contribute directly to the enhancements in motor performance.

Studies using transcranial magnetic stimulation (TMS) have shown changes in the cortical representation of the muscles engaged in acquisition of the new motor task. There is an apparent increase in motor cortical excitability after training for hand muscles (Latash et al. 2003; Pascual-Leone et al. 1995), arm muscles (Jensen et al. 2005) and leg muscles (Perez et al. 2004, 2005). Such changes have been interpreted as altered organization in the motor cortex (Klein et al. 2004; Müllbacher et al. 2002; Nudo et al. 1996; cf. Plautz et al. 2000). Reorganization of cortical circuitry has also been demonstrated for repetitive long-term motor practice (e.g., Büttifisch et al. 2000; Classen et al. 1998; Jensen et al. 2005; Pascual-Leone et al. 1995, 1999; Stefan et al. 2005) with potentially permanent changes (Pascual-Leone et al. 1993). Interestingly, repetitive TMS (rTMS) over the motor cortex can interfere with the learning and retention of a behavioral skill and may prevent a change in cortical representation (Carey et al. 2006; Jäncke et al. 2004; Lee et al. 2010; Müllbacher et al. 2002).

Strength training does not increase cortical excitability (Carroll et al. 2002; Jensen et al. 2005; Remple et al. 2001), despite increases in muscle mass (e.g., Hickson et al. 1994; Jones and Rutherford 1987; Komi 1986; McCartney et al. 1988; Narici et al. 1989; Ploutz et al. 1994), muscle protein synthesis, and cross-sectional area of muscle fibers (e.g., Staron et al. 1994). Thus, Carroll et al. (2002) and Jensen et al. (2005) proposed that use of specific muscles without learning might not augment their cortical representation. That is, strength training might not require changes in cortical organization.

Although it is widely recognized that cortical reorganization is related to skill acquisition, much remains unknown about the contribution of changes at a spinal level. It is important to emphasize that responses to TMS are strongly affected by changes in neuronal properties at a subcortical as well as a cortical level (e.g., Rothwell 1997). This implies that neuronal adaptations to motor skill training potentially also occur at a spinal level. There is evidence for short-term plasticity at this site. Investigations of the spinal stretch reflex after motor training have demonstrated changes involving the motoneuron pool, and these appear to be related to the acquisition of specific motor tasks (Carp and Wolpaw 1994; Meunier et al. 2007; Nielsen et al. 1993; Perez et al. 2005; Sale 1988; Wolf et al. 1995; Wolpaw et al. 1994; Wolpaw and Lee 1989). In addition, motoneuronal responses to corticospinal input can be acutely changed after short and prolonged strong voluntary contractions (MVCs) (e.g., Gandevia et al. 1999; Giesebrrecht et al. 2010, 2011; Petersen et al. 2003), and conditioning of the corticospinal-motoneuronal synapse by paired stimulation can alter corticospinal transmission in a way that is consistent with spike-timing-dependent plasticity (Taylor and Martin 2009).

We hypothesize that motor training, which requires brief ballistic efforts generating high motor unit firing rates (Van Cutsen et al. 1998), will favor changes at a spinal level. In contrast, complex tasks such as visuomotor tracking may require cortical reorganization. In this study, our aim was to
investigate whether responses to corticospinal stimulation can be acutely altered by two different types of motor training, namely a simple ballistic motor task and a complex visuomotor tracking task.

METHODS

Subject Groups

Twenty-seven healthy subjects gave written informed consent to participate in the study. The protocol was approved by the local human research ethics committee and performed in accordance with the Declaration of Helsinki. Subjects participated in ~30 min of ballistic training (experiment 1) and/or ~30 min of visuomotor skill training (experiment 2). Subjects were included if cervicomedullary motor evoked potentials (CMEPs) could be evoked in their right first dorsal interosseous muscle (FDI) at rest. CMEPs of sufficient size (at least 0.5 mV amplitude at rest on initial testing) were evoked in 9 of 27 subjects. In addition, in experiment 1 the data from one subject were rejected due to malfunctioning of equipment. Thus, seven subjects (age 36 ± 12 yr, 5 female) were included for experiment 1 and experiment 2 (age 37 ± 12 yr, 5 female). Six subjects took part in both experiments, and the two experiments were on average 3 mo apart. Fifteen of the remaining volunteers, in whom CMEPs could not be evoked, also performed experiment 1 (n = 8, age 29 ± 6 yr, 6 female) and/or experiment 2 (n = 8, age 28 ± 4 yr, 4 female) but without CMEP stimulation.

Experimental Setup

In both experiments, subjects sat with the right forearm pronated and resting on a table with the elbow slightly flexed in a comfortable position (Fig. 1A). Two bandages around the forearm held it tightly in place. The right index finger was positioned in a custom-designed finger splint. The splint permitted free abduction and adduction of the index finger around the metacarpophalangeal (MCP) joint. In both experiments, the thumb was held in a midflexed position. For experiment 1, the third and fourth fingers were strapped down by tape in a straight position. For experiment 2, a weight of 50 g was attached to the splint and hung over a pulley to pull the index finger into adduction. Thus, abduction and adduction movements were controlled by concentric and eccentric activation of FDI; the activity of its antagonist muscle, the first palmar interosseous, was minimized. The third, fourth, and fifth fingers were placed into midflexion to allow free motion of the index finger between 10° of abduction and adduction. Movements further than that were prevented by a mechanical stop.

An accelerometer (5g, ADXL 320, Analog Devices, Norwood, MA) was attached directly to the metal splint perpendicular to the axis of rotation to quantify the acceleration of the index finger during the experiment. Electromyographic (EMG) recordings were taken from the right first dorsal interosseous muscle (FDI). Maximal compound muscle action potentials were evoked by stimulation of the ulnar nerve ($M_{max}$). The axis of rotation was aligned with the axis of the metacarpophalangeal (MCP) joint. During visuomotor training, a 50-g weight pulled the finger into adduction to minimize antagonist activity. Stimulation was delivered at the cervicomedullary junction to evoke cervicomedullary motor-evoked potentials (CMEP). Each set of stimuli included 10 CMEPs (arrows) followed by 2 nerve stimuli to evoke $M_{max}$. With the muscle relaxed, stimuli were delivered before, between, and after 2 sets of training, indicated by the gray blocks.

Fig. 1. Schematic representation of the set up (A) and experimental protocols for the ballistic (experiment 1, B) and the visuomotor (experiment 2, C) training. Electromyographic (EMG) recordings were taken from the right first dorsal interosseous muscle (FDI). Maximal compound muscle action potentials were evoked by stimulation of the ulnar nerve ($M_{max}$). The axis of rotation was aligned with the axis of the metacarpophalangeal (MCP) joint. During visuomotor training, a 50-g weight pulled the finger into adduction to minimize antagonist activity. Stimulation was delivered at the cervicomedullary junction to evoke cervicomedullary motor-evoked potentials (CMEP). Each set of stimuli included 10 CMEPs (arrows) followed by 2 nerve stimuli to evoke $M_{max}$. With the muscle relaxed, stimuli were delivered before, between, and after 2 sets of training, indicated by the gray blocks.
abduction movements. The accelerometer gain was set at 0.96 V/g. The position of the finger in the abduction-adduction plane was signaled by a potentiometer aligned with the axis of rotation of the finger splint. When required, the splint could be rigidly secured to measure isometric index finger abduction force through a force transducer (250 N, Xtran, Melbourne, Australia). Electromyographic activity (EMG) was recorded from the right FDI muscle through self-adhesive surface electrodes (Ag-AgCl). Electrodes were positioned over the midbelly of the muscle and over the second MCP joint. EMG activity from FDI was filtered and amplified (16–1,000 Hz; CED 1902 amplifiers).

All signals were digitized and stored onto a personal computer using a laboratory interface (CED 1401 and Spike2 software; Cambridge Electronic Design, Cambridge, UK). EMG and force were sampled at 2 kHz, and acceleration and position at 1 kHz. During all types of stimulation, voluntary relaxation was constantly monitored and ensured by visual feedback of the EMG signals.

**Stimulation**

**Corticospinal tract.** Magnetic stimulation of the corticospinal tract was performed using a double-cone coil (MagStim, Whittle, Dyfed, UK; 2 coils, each of 12 cm outer diameter, set at an angle of ~90°) behind the back of the subject’s head. The center of the coil was initially placed over the inion with current flowing downwards in the center of the coil (Taylor and Gandevia 2004). The optimal location for corticospinal tract stimulation was assessed by moving the coil laterally and caudally (Martin et al. 2009) and was marked on a tightly fitting cap. For all subjects, CMEPs were elicited in the right FDI with stimulation to the right of the inion. Stimulator intensity was set between 90 and 100% of two simultaneously discharging Magstim 200 stimulators. Throughout the experiments subjects wore earplugs to dampen the sound. As the high stimulus intensity can be uncomfortable, the number of stimuli was kept as low as possible. However, to dampen the sound, the number of stimuli was kept as low as possible. During training, subjects (n = 7) were presented with sets of six consecutive frames (60 s of continuous tracking). After four sets with short breaks between them, subjects had a longer break of ~1 min. One block of training consisted of 3 × 4 sets (overall 72 frames). Subjects completed two blocks of training in total. While most frames were presented randomly, two pairs of the frames were repeated at preselected times throughout the training to simplify analysis. CMEPs and $M_{\text{max}}$ were evoked in sets as described for experiment 1 before (Pre), between (Mid), immediately (Post) and 10 min (Post 10) after the training sets (Fig. 1C). Additional subjects (n = 8) performed the same blocks of training but without cervicomedullary junction or peripheral nerve stimuli.

**Data Analysis**

For each potential evoked by cervicomedullary (CMEPs) or peripheral ($M_{\text{max}}$) stimulation, the area and peak-to-peak amplitude were measured with cursors set appropriately around the waveform. In the relaxed FDI muscle, the changes that occurred in the peak-to-peak amplitude and area were similar. Hence we only report the results for the amplitude of the evoked potentials. To correct for peripheral changes over time, the amplitude of each CMEP was normalized to the amplitude of $M_{\text{max}}$ recorded closest in time. In experiment 1, maximal acceleration was measured for each ballistic abduction movement. $EMG_{\text{rms}}$ amplitude was measured between the start of the EMG burst and the time of maximal acceleration for each movement (55- to 60-ms period). For experiment 2, performance during the task was assessed over predetermined 10-s frames. These frames occurred in pairs at 15 times during the training. For each frame, the relationship between the tracking pathway and the MCP joint angle was assessed by cross-correlation analysis. The bin width used in the cross-correlation analysis was 10 ms. The maximal cross-correlation coefficient (p) and the time lag were calculated. $EMG_{\text{rms}}$ amplitude during each 10-s frame was also measured. Measures were averaged across each pair of consecutive frames before statistical analysis.

**Experimental Protocol**

**Experiment 1:** training in a ballistic task. In experiment 1, we investigated the effects of brisk unilateral index finger abduction movements on the excitability of the corticospinal tract (Fig. 1B). Subjects (n = 7) underwent motor skill training, which involved two blocks of 150 abduction movements of the right index finger. Each block consisted of 15 sets of 10 index finger movements at 0.5 Hz with rest breaks of 30 s between the sets. Finger acceleration was paced by an auditory click, and subjects were given visual feedback of the acceleration on an oscilloscope. Subjects were urged to increase peak acceleration with each trial. Subjects received one set of 10 cervicomedullary junction stimuli (CMEP) and two peripheral nerve stimuli ($M_{\text{max}}$) 10 s apart prior to (Pre), and immediately after, each frame (Fig. 1B). One additional set of stimuli was delivered at ~10 min after the last training block (Post 10). An additional eight subjects performed the blocks of index finger movements without cervicomedullary junction or peripheral nerve stimuli. 

**Experiment 2:** training in a visuomotor tracking task. In experiment 2, we assessed the effects of visuomotor tracking training on the excitability of the corticospinal pathway (Fig. 1C). The training task involved tracking a target pathway formed by two lines on a computer screen positioned in front of subjects. A separate blue line on the screen indicated the subject’s MCP joint angle (i.e., finger position) relative to the target pathway. Subjects performed voluntary index finger abduction and adduction movements within a range of ~10° joint rotation into each direction. The training task was to keep the angular position signal as accurately as possible between the two target lines. The target pathway moved automatically from the left to the right of the screen over 10 s, and subjects had no preview of the target pathway. In total, there were 18 different frames, each of which consisted of unpredictable upward and downward movements. The frames were presented in semi-random order as detailed below.

During training, subjects (n = 7) were presented with sets of six consecutive frames (60 s of continuous tracking). After four sets with short breaks between them, subjects had a longer break of ~1 min. One block of training consisted of 3 × 4 sets (overall 72 frames). Subjects completed two blocks of training in total. While most frames were presented randomly, two pairs of the frames were repeated at preselected times throughout the training to simplify analysis. CMEPs and $M_{\text{max}}$ were evoked in sets as described for experiment 1 before (Pre), between (Mid), immediately (Post) and 10 min (Post 10) after the training sets (Fig. 1C). Additional subjects (n = 8) performed the same blocks of training but without cervicomedullary junction or peripheral nerve stimuli.

**Statistical Analysis**

All group data are presented in the text as means ± SD. In the figures, CMEP group data are normalized to $M_{\text{max}}$ and then expressed as a percentage of control values and shown as means ± SE. For experiment 1, acceleration and $EMG_{\text{rms}}$ during the ballistic task were analyzed using two-way ANOVAs for comparison of group (with or without CMEPs; between-subject factor) and time (within-subject factor), with each training set (10 index finger abduction movements) compared with the first training set. For experiment 2, the visuomotor training, the maximal cross-correlation coefficient (p), time lag, and $EMG_{\text{rms}}$ were also analyzed using two-way ANOVAs for comparison of group (with or without CMEPs; between-subject factor) and time (within-subject factor) with subsequent frames compared with the first frame. The CMEP data (normalized to $M_{\text{max}}$) for each experiment were analyzed using one-way repeated-measures ANOVAs with training time points as a within-subject factor (4 levels: Pre, Mid, Post, and Post 10). ANOVAs were performed on the CMEP values normalized to $M_{\text{max}}$. Mauchly’s test of sphericity was used, and Greenhouse-Geisser corrections were applied where needed.
In other analyses, Pearson product-moment correlations were performed between some measures to test whether changes in the evoked responses were related to behavioral changes. For each subject, CMEP amplitudes (normalized to M\text{max}) were expressed as a percentage of values recorded prior to training, and averaged for each recording time point. For experiment 1, cross subject correlations were sought between average CMEPs (recorded at Mid and Post) and average acceleration and average EMG\text{ rms} recorded over the last 30 movements of each training block and expressed relative to the initial 10 training movements. For experiment 2, correlations were performed between CMEPs (recorded at Mid and Post) and EMG\text{ rms}, cross-correlation coefficient and time lag calculated for the pre-selected pair of traces at the end of each training block. EMG\text{ rms} was expressed as a percentage of initial values, and cross-correlation coefficient and time lag were expressed as differences.

RESULTS

Experiment 1: Training in a Ballistic Task

Subjects undertook training of ballistic finger abduction while CMEPs were measured before, between, and after training.

Index Finger Acceleration

Figure 2A shows raw traces from an individual subject. These indicate about a twofold increase in index finger acceleration and EMG from the first to the final training set. For the group who received cervicomedullary stimulation, peak acceleration increased from 34.7 ± 19.1 m/s\textsuperscript{2} to 88.7 ± 53.4 m/s\textsuperscript{2} (Fig. 2B), while EMG went from 1.1 ± 0.3 mV to 1.2 ± 0.4 mV (Fig. 2C). For the group who received no cervicomedullary stimulation, peak acceleration increased from 28.1 ± 6.1 m/s\textsuperscript{2} to 81.9 ± 34.4 m/s\textsuperscript{2}, and EMG went from 1.2 ± 0.2 mV to 1.3 ± 0.2 mV. Two-way ANOVA showed that acceleration increased significantly (F\textsubscript{1,72,22,3} = 15.767; P < 0.001) with no difference between the subject groups (F\textsubscript{1,13} = 0.069; P = 0.797). EMG increased with marginal significance (F\textsubscript{8,2,106,6} = 1.995; P = 0.052) and with no difference between the subject groups (F\textsubscript{1,13} = 0.959; P = 0.345).

Corticospinal Excitability

Prior to the ballistic training, the average amplitude of CMEPs was 0.7 ± 0.6 mV. Immediately after the first block of acceleration training, CMEPs increased in all but one subject to an average of 1.5 ± 1.1 mV, and after the second block of training to a mean of 1.5 ± 1.2 mV or 248 ± 152% of control (Fig. 3, A and B). Ten minutes after training finished, CMEPs had returned towards control amplitude (0.9 ± 0.8 mV). ANOVA showed significant changes in CMEP amplitude over time (F\textsubscript{3,18} = 11.62, P < 0.0005). Tests of within-subject contrasts revealed a significant effect after the first and the second training set (Post vs. Pre, P = 0.002; also Mid vs. Pre, P = 0.024). On average, M\text{max} had an amplitude of 21 ± 5.8 mV and did not change significantly throughout the experiment.

Correlations

There was a significant positive correlation between the change in index finger abduction acceleration over the first block of training and the change in size of CMEPs after the first block of training (r = 0.913; P = 0.0041; Fig. 4). No other significant effects were detected.

Fig. 2. Performance (peak acceleration and EMG activity) during the ballistic task. A: overlaid traces from an individual subject show acceleration into abduction and EMG of the right FDI muscle in the first 5 ballistic movements of the first set (Start) and the last 5 movements of the last set (End). Arrows indicate the peak accelerations achieved in the trials. B: group data (means ± SE) of peak acceleration of the index finger abduction movements expressed as m/s\textsuperscript{2}. Two blocks of training were performed (1–15 and 16–30 training sets). C: group mean data (± SE) of EMG\text{ rms} amplitude (mV) during the ballistic movements. Data are averaged across the 10 movements in each set. Subjects who received cervicomedullary stimulation (n = 7) are shown as filled circles. Subjects who did not receive stimulation (n = 8) are shown as open circles.

Experiment 2: Training in a Visuomotor Tracking Task

Subjects practiced a visuomotor tracking task while CMEPs were measured before and after training.

Visuomotor Tracking

Figure 5 shows the improvement in performance of an individual subject in tracking the same 10-s pathway at the beginning (Start: maximal cross-correlation coefficient ρ = 0.76; 278-ms
Corticospinal Excitability

Prior to training, CMEPs had a mean amplitude of 0.45 ± 0.41 mV. After the first block of visuomotor tracking, mean CMEP amplitude was 0.34 ± 0.34 mV, and after the second block, it was 0.33 ± 0.31 mV (Fig. 6). CMEPs evoked 10 min after training were 0.54 ± 0.61 mV. There was no significant difference in CMEPs between time points (F3,18 = 0.939, P = 0.443). M_max amplitudes showed no significant change over time (before: 17.8 ± 1.8 mV, after 19.3 ± 1.6 mV; F8,48 = 0.285; P = 0.968).

Correlations

There were no significant relations between maximal cross-correlation coefficient, time lag, EMG activity, and CMEPs.

DISCUSSION

We investigated responses in the FDI muscle to magnetic stimulation at the cervicomedullary junction (CMEP) to detect changes in corticospinal excitability following two types of motor training. As in previous studies, we found that ~30 min of training in a ballistic movement or in visuomotor tracking improved performance of the task. Our novel finding is that ballistic training induced large changes in excitability at a subcortical level. Following the ballistic task, CMEPs increased ~2.5 times compared with baseline, and tended to recover 10 min after the training. In contrast, after visuomotor tracking, CMEPs were unchanged.

Previous studies in which subjects trained to improve the acceleration of digit movements have shown significant increases in motor potentials evoked by magnetic stimulation over the motor cortex (MEP) (e.g., Carroll et al. 2008; Lee et al. 2010; Rogasch et al. 2009; Ziemann et al. 2004). However, these changes can depend on the intensity of stimulation, with larger changes of MEPs elicited with higher intensity stimuli (Carroll et al. 2008). When increases in MEPs occurred, they could last for ≥30 min (Rogasch et al. 2009; Ziemann et al. 2004). In our study, despite the small size of the potentials, CMEPs consistently increased in all subjects by the end of the training but were no longer significantly increased 10 min later. As CMEPs are evoked through stimulation of corticospinal axons at the cervicomedullary junction, the increase in ampli-
titude indicates alteration in the corticospinal pathway at a subcortical level. Single motor unit studies have shown that there is a strong monosynaptic contribution to CMEPs in the hand muscles (e.g., Ugawa et al. 1991). Hence, the increase in size of the CMEP in FDI most likely occurred through an increase in the excitability of the motoneurons or through an increase in corticospinal synaptic efficacy. Because non-monosynaptic excitation of the motoneurons will also contribute to the CMEP, changes in spinal interneurons cannot be ruled out.

Only one previous study has examined responses to brainstem stimulation after training in a ballistic task (Müllbacher et al. 2002) and no consistent changes in CMEP amplitudes were found, but only three subjects were tested. The authors proposed that the change in MEPs induced by the practice was predominantly a function of altered motor cortical excitability. In the current study, the significant correlation across subjects between the increase in CMEP amplitude and the increase in acceleration at the end of the first set of 150 movements suggests that the change at a spinal level in the corticospinal pathway may contribute to improved motor performance. However, the relationship was no longer present after the second training set, although CMEPs were still facilitated. As increases in the MEP can last longer than those seen here in the CMEP (Rogasch et al. 2009; Ziemann et al. 2004), it seems likely that changes in excitability can occur at both a cortical and spinal level with training in a ballistic movement. However, it is not possible to measure excitability changes at a cortical level without consideration of the size of spinal changes.

After practice of visuomotor tracking with the index finger, performance improved. However, CMEP amplitudes did not change in a consistent way. CMEPs decreased in some subjects and increased in others. This variability was unexpected as CMEPs evoked in FDI by magnetic stimulation are reliable within testing sessions (Martin et al. 2009) and consistent changes were found after the ballistic movements. The changes in CMEPs across subjects did not show associations with the improvements in visuomotor tracking, but we cannot rule out other differences in task performance which may have influenced individuals’ responses. A similar task performed with the elbow and the ankle resulted in increased MEPs elicited in biceps and tibialis anterior, respectively, by transcranial magnetic stimulation. In contrast, responses evoked by transcranial electrical stimulation were unchanged (Jensen et al. 2005; Perez et al. 2004). This suggests that the training alters motor cortical excitability in this complex task. The finding of unaltered responses to transcranial electrical stimulation is consistent with the lack of consistent change in CMEP size in the current study (Perez et al. 2004). Both responses test transmission in the motor pathway through the spinal cord to the muscle, and their lack of change implies that motoneuron excitability is unaltered. In contrast, H-reflexes are reduced with various types of skill acquisition including visuomotor tracking with ankle movement, cycling with variable resistance, and implicit learning of a movement sequence (Lungu et al. 2010; Mazzocchio et al. 2006; Meunier et al. 2007; Perez et...
al. 2005, 2006). However, the decreases are not thought to represent altered motoneuron excitability. There is evidence that the changes following visuomotor tracking are due to altered presynaptic inhibition acting through primary afferent depolarization of Ia afferents (Perez et al. 2005, 2006), whereas changes in homosynaptic postactivation depression contribute after training in the cycling task (Meunier et al. 2007).

Our results show improved efficacy in transmission through the corticospinal pathway at a spinal level after repeated brief efforts in which subjects attempted to maximize motoneuronal output and no change after fine skilled movements of the index finger. Moderate to strong isometric contractions of more than 5- to 10-s duration also alter CMEPs (Giesebrecht et al. 2010, 2011; Petersen et al. 2003). The pattern of changes varies between muscles, but, for FDI, such contractions lead to smaller responses for ~8 min (Giesebrecht et al. 2011). Thus, the increase seen in the current study is specific to the training task. While we acknowledge that changes in motoneuronal or interneuronal excitability could result in the observed increase in the CMEP, we postulate that the change relies on the pattern of repeated high frequency bursts of voluntary synaptic input to the motoneurons and is a marker of plasticity at a spinal level.

We have recently reported that human corticomotoneuronal synapses can be both facilitated and depressed by repeated pairs of presynaptic and postsynaptic potentials with appropriate interstimulus intervals (Taylor and Martin 2009). This suggests that these synapses may be susceptible to long-term facilitation and depression following appropriate timed synaptic inputs. An alternative explanation for the current findings is that excitatory afferent input to the motoneurons continues for several minutes after repeated ballistic movements. For example, small-diameter fatigue-sensitive muscle afferents are excitatory to some motoneuron pools and might have some relatively long-lasting effects (Martin et al. 2006, 2008). However, the depression rather than facilitation of CMEPs in FDI after fatiguing isometric MVCs argues against this (Giesebrecht et al. 2011).

Animal studies have shown that training for muscle strength can increase excitatory synapses onto motoneurons, whereas training for skilled movement leads to cortical reorganization (Adkins et al. 2006; Remple et al. 2001). Operant conditioning of H-reflexes can alter motoneuron properties (Carp and Wolpaw 1994; Feng-Chen and Wolpaw 1996; for review, Wolpaw 2007). In humans, the consequences at a spinal level of training for contraction speed or resistance training for strength are less clear. Increases in initial EMG underlie increases in the rate of force development, and motor units produce more doublets, but whether this greater motoneuron output is due to extra output from the cortex or an increased response of the motoneuron pool because of changes at the spinal level is not known (Aagaard et al. 2002; Gruber and Gollioher 2004; Van Cutsem et al. 1998). Similarly, after strength training, some studies have suggested that motoneuron output during maximal efforts is increased by increased corticospinal input (Aagaard et al. 2002; Chen et al. 2001; Del Balso and Cafarelli 2007). However, studies using cortical stimulation generally suggest that corticospinal excitability is decreased in a task-specific manner (Carroll et al. 2002; Jensen et al. 2005; Schubert et al. 2008).

Thus, it seems likely that there are long-term adaptations in the human spinal cord with strength training, but they are currently difficult to define. We suggest that the changes in motoneuronal responses that occur after short-term practice might reflect an initial step in adaptation and could contribute to more persistent reorganization at a spinal cord level with repeated practice.

In conclusion, during practice of a ballistic motor task, subjects improved peak acceleration of an index finger movement. The motor responses to corticospinal stimulation at a subcortical level increased significantly. The increase in CMEP size suggests an increase either in excitability of the motoneurons or in efficacy of synaptic transmission. Thus, changes at a spinal level may contribute to improvements in motor performance with practice.

REFERENCES


