Neurophysiological Responses After Short-Term Strength Training of the Biceps Brachii Muscle

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Abstract

Kidgell, DJ, Stokes, MA, Castricum, TJ, and Pearce, AJ. Neurophysiological responses after short-term strength training of the biceps brachii muscle. *J Strength Cond Res* 24(11): 3123–3132, 2010. The neural adaptations that mediate the increase in strength in the early phase of a strength training program are not well understood; however, changes in neural drive and corticospinal excitability have been hypothesized. To determine the neural adaptations to strength training, we used transcranial magnetic stimulation (TMS) to compare the effect of strength training of the right elbow flexor muscles on the functional properties of the corticospinal pathway. Motor-evoked potentials (MEPs) were recorded from the right biceps brachii (BB) muscle from 23 individuals (training group; $n = 13$ and control group; $n = 10$) before and after 4 weeks of progressive overload strength training at 80% of 1-repetition maximum (1RM). The TMS was delivered at 10% of the root mean square electromyographic signal (rmsEMG) obtained from a maximal voluntary contraction (MVC) at intensities of 5% of stimulator output below active motor threshold (AMT) until saturation of the MEP (MEP_{max}). Strength training resulted in a 28% ($p = 0.0001$) increase in 1RM strength, and this was accompanied by a 53% increase ($p = 0.05$) in the amplitude of the MEP at AMT, 33% ($p = 0.05$) increase in MEP at 20% above AMT, and a 38% increase at MEP_{max} ($p = 0.04$). There were no significant differences in the estimated slope ($p = 0.47$) or peak slope of the stimulus–response curve for the left primary motor cortex (M1) after strength training ($p = 0.61$). These results demonstrate that heavy-load isotonic strength training alters neural transmission via the corticospinal pathway projecting to the motoneurons controlling BB and in part underpin the strength changes observed in this study.

Key Words transcranial magnetic stimulation, controlled strength training, biceps brachii, electromyography, corticospinal

Introduction

Changes in maximal voluntary contraction (MVC) force after a period of strength training have been attributed to adaptive modifications in the neuromuscular system (see review by Folland and Williams [14]). Neural adaptations have been suggested to account for the rapid increase in strength within the first 2–4 weeks of a strength training program (14); however, the specific mechanisms contributing to this adaptation are not well understood. Proposed neural mechanisms may range from an increase in neural drive to subtle changes in motor unit behavior, suggesting that there is no single mechanism responsible for the increase in strength and that adaptations probably extend to both supraspinal and spinal regions (14). Given that it still remains unclear what mechanisms contribute to the rapid development in strength after a period of heavy-load controlled strength training, the purpose of this study was to examine the contribution of the corticospinal tract after 4-weeks of upper limb strength training on strength development.

Adaptations in neural function after strength training have usually been investigated and quantified via changes in the amplitude of the muscle electromyogram (EMG) and more recently after single motor unit recordings (12). An increase in the amplitude of the surface EMG (sEMG) signal has, by default, been interpreted as an increase in neural drive, therefore contributing to the increase in force. Changes in neural drive can be investigated by recording evoked spinal cord responses such as the Hoffman Reflex (H-reflex), which is used to determine the level of motoneuron excitability and the magnitude of presynaptic inhibition of muscle spindle Ia afferents (29). Alternatively, the volitional wave (V-wave) that is a variant of the H-reflex can be used to quantify training-induced modifications in efferent motoneuronal output (1). Elevated H-reflexes and V-wave amplitudes have been reported after maximal dynamic and isometric strength

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training (7,11), suggesting enhanced neural excitability in descending corticospinal tracts. However, adjustments in H-reflex and V-wave amplitude after strength training may arise as a result of changes in the intrinsic properties of Ia afferents, such as presynaptic inhibition, intrinsic motoneuron properties, and changes in motoneuron firing rate (23). A limitation of these techniques is the difficulty in quantifying the site of adaptation (e.g., supraspinal or spinal) because neither technique directly measures the involvement of the primary motor cortex (M1) or corticospinal tract. The M1 and corticospinal tract are perhaps the primary supraspinal structures that are involved in modulating voluntary force production (3); therefore, changes in descending pathways should be measured with the appropriate technique.

Changes in M1 and corticospinal excitability can be measured using transcranial magnetic stimulation (TMS). Transcranial magnetic stimulation enables the assessment of corticospinal excitability during voluntary contractions in humans and has recently been used in strength training research (6,13,16,23,26,36). The TMS applied over the M1 can induce a series of descending volleys in the corticospinal tract, which in turn, causes a muscle response referred to as a motor-evoked potential (MEP). Changes in MEP amplitude are thought to reflect adjustments in the physiological strength of corticospinal cell projection onto the spinal motoneuron pool. Corticospinal excitability may also be measured by plotting the relationship of the MEP amplitude in response to stimulation at a range of stimulus intensities resulting in a sigmoid curve that reflects corticospinal excitability. The slope of the curve is influenced by the excitability of corticospinal cells underneath the stimulating coil and the spatial distribution of the excitable elements of the M1 and corticospinal pathway (5).

Motor skill practice studies have provided convincing evidence for a task-dependent adaptation in corticospinal output with suggested mechanisms of adaptation, including increased excitability of populations of corticospinal neurons projecting to the muscles involved in the skilled task (30,32), unmasking of latent synapses (2), and functional reorganization of the M1 (33). It has been hypothesized that strength training may also result in a similar adaptation, because the skilled element of strength training exercises (10). In light of this, TMS has recently been used in an attempt to determine the role of the corticospinal tract after a period of strength training. However, results have been inconsistent, and this may be attributed to the different training paradigms used, muscles trained and/or the different methods used to assess corticospinal excitability. For example 4 weeks of moderate to heavy-load isometric strength training of the first dorsal interosseous (FDI) muscle, decreased corticospinal excitability, despite a 33% increase in strength (6). Similarly, 4 weeks of strength training of the biceps brachii (BB) muscle increased strength by 31%; however, this was associated with a decrease in corticospinal excitability (24). In contrast to these findings, Beck et al. (4) demonstrated increased MEP amplitude after 4 weeks of ballistic strength training of the tibialis anterior (TA). In support of this, Griffen and Cafarelli (16) after 4 weeks of strength training of the TA muscle, found a 32% increase in MEP amplitude, suggesting that strength training resulted in a task-specific adaptation within the corticospinal tract. However, it is difficult to compare the data across studies because different muscles and TMS protocols have been used. Furthermore, few studies have actually assessed isotonic strength training on MEPs evoked during voluntary contraction (24). Therefore, the purpose of this study was to investigate whether short-term controlled strength training stimulated changes in human corticospinal excitability after 4-week strength training of the BB muscle. We compared the effects of heavy-load controlled strength training on corticospinal conduction and excitability at active motor threshold (AMT), 20% above AMT and at maximum MEP amplitude (MEP_{max}) during 10% of MVC background muscle activation. It was hypothesized that 4 weeks of heavy-load controlled strength training would increase muscle strength, and this would be reflected by an increase in corticospinal excitability providing evidence for a corticospinal mechanism for strength development.

Methods

Experimental Approach to the Problem

Twenty-three healthy participants (10 men and 13 women, 26.8 ± 7.3 years) were randomly allocated into either a strength training (6 men, 20.3 ± 3.4 years and 7 women, 24.5 ± 3.0 years) or a control group (5 men, 27.6 ± 7.9 years and 5 women, 29 ± 6.2 years). All participants were right handed, as assessed by the Edinburgh handedness inventory (28), and, although being physically active and healthy in noncompetitive recreational activities, none of the participants had involvement in any kind of strength training in the previous 2 years. All participants gave written, informed consent to the experimental procedures, which conformed to the Declaration of Helsinki and were approved by the Human Research Ethics Committee of the University. Participants assigned to the strength training group were required to undertake 12 supervised strength training sessions over a 4-week training period. Participants assigned to the control group completed no training. At the beginning and at the end of the training period, each subject participated in a testing session that involved the following: (a) strength testing to evaluate maximal voluntary dynamic elbow flexor muscle strength (1 repetition maximum [1RM]) and maximal root mean square electromyography (rmsEMG) during an isometric MVC and (b) single pulse TMS applied to the hemisphere projecting to the right BB. All testing posttraining was conducted within 48 hours of the final supervised strength training session.

Subjects

Twenty-three people without a history of neurological disease volunteered to participate in the study (10 men and

13 women, 26.8±7.3 years). Subjects were randomly allocated either to a strength training condition (n = 6 men and 7 women 13), or to a control condition (n = 10, 5 men and 5 women). The handedness of the subjects was determined according to the Edinburgh Handedness Inventory (28). This questionnaire provided a measure of hand preference that is based on the hand used to perform a range of daily activities (e.g. writing, holding a soon, etc.). All subjects gave written, informed consent to the procedures of the study, which conformed to the Declaration of Helsinki and were approved by the Human Research Ethics Committee of the university.

Procedures

Maximum Strength Testing. Participants in both groups performed a standard unilateral 1RM test for the right arm. After the protocol of Munn et al. (27), participants were asked what they believed their 1RM elbow flexion strength was, and this load served as their initial starting weight. Participants performed the 1RM test standing, holding a weighted dumbbell with one hand, with their elbow in full extension, forearm supinated, and the opposite arm placed behind their back while standing against a wall to prevent excessive body movement. Participants were then asked to flex their arm and lift the dumbbell as if doing a standard "biceps curl." If the trial was successful, the weight of the dumbbell was increased accordingly (0.5-kg increments) on each trial after a 3-minute recovery to minimize the development of muscular fatigue. This procedure continued until the subject could no longer complete one repetition and their prior trial served as their 1RM elbow flexion strength (27).

Arm Circumference. To determine whether there was any change in muscle hypertrophy as a result of the strength training program, arm circumference of the right upper arm was measured with a tape measure. Specifically, arm circumference was determined at the largest circumference of the upper arm while participants attempted a strong contraction of the elbow flexors in a shortened position, with the shoulder at 90° flexion and the forearm 45° to the upper arm (27).

Strength Training Procedures. The strength training group performed heavy-load strength training (80% of their 1RM) of the right elbow flexors only, 3 times per week for 4 weeks (12 sessions in total). All training was supervised within the laboratory, and participants were instructed to train in the same way as tested, that is, with the contralateral limb placed behind their back. Biceps curls with a dumbbell were performed by undertaking flexion–extension movements of the elbow with the forearm supinated. The participants performed 4 sets of 6–8 repetitions at 80% 1RM with a 3-minute recovery period between sets (27). Participants were required to perform each repetition with a repetition timing of 3-second concentric and 4-second eccentric, because previous research has demonstrated that slow velocity strength training is associated with a greater level of neural adaptation compared to high velocity strength training alone and it is thought such adaptations may be mediated by peripheral feedback mechanisms thus contributing to strength development (21, 22). The principle of progressive overload was employed throughout the training period to maximize the training response (33). Specifically, when participants could complete 4 sets of 8 repetitions, at the beginning of the next training session, the training weight (kg) was increased by 5%.

Electromyography and Transcranial Magnetic Stimulation. Surface EMG activity was recorded from the right BB muscle using bipolar Ag–AgCl electrodes. Two electrodes were placed 2 cm apart over the BB muscle, located by manual muscle testing and placed over the belly of the muscle, with the third reference electrode (ground electrode) placed over the bony prominence at the elbow (lateral epicondyle). The area of electrode placement was prepared by shaving and cleaned with 70% isopropyl alcohol. The site was marked with permanent marker and continually maintained by the investigator and participant, to ensure no differences in electrode placement occurred relative to the innervation zone before and after the 4-week training period. Electromyographic signals were amplified (×1,000) with bandpass filtering between 10 Hz and 1 kHz and digitized at 1.5 kHz for 500 milliseconds using custom-designed software (National Instruments V4.0, Austin, TX, USA). The surface rmsEMG was calculated from a 500-milliseconds segment occurring during the asymptote of the MVC (15). To obtain the MVC, participants were seated in a chair with the elbow flexed to 90°, as measured by an electronic goniometer (Biometrics, Ladysmith, VA, USA), and with their hand in a supinated position. A dynamometer (Microfet2, Hoggan Health Industries, West Jordan, UT, USA) was positioned on a modifiable bench so the dynamometer was inside the participant's forearm at the level of the wrist. The participant was then instructed to flex the elbow against the dynamometer as forcefully as possible for 3 seconds. Three attempts, with a 2-minute rest between each attempt were performed. The trial with the highest MVC and rmsEMG level was recorded and subsequently used to determine background muscle activity during the TMS protocol. The standard criteria for measurement of MVCs were fulfilled and included a period of familiarization (before data collection) and verbal encouragement, feedback of rmsEMG displayed on a computer monitor at the eye level, standardized verbal encouragement provided by the investigators and the rejection of a trial in the case the participant felt it was not a maximal effort (15).

The TMS testing followed the established protocols of Pearce and Kidgell (31). The MEPs were evoked by TMS of the contralateral motor cortical area projecting to the BB using a Magstim 200^2 stimulator (Magstim Co, Spring Gardens, United Kingdom), with a 70-mm figure of 8-coil

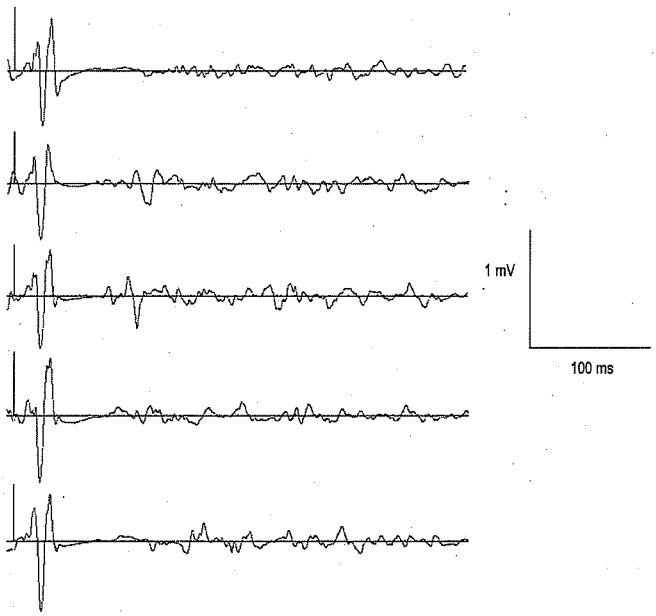

Figure 1. Example of 5 raw motor-evoked potential (MEP) sweeps (500 milliseconds) from 1 participant during the transcranial magnetic stimulation (TMS) trials. The black spike represents the stimulus artifact.

placed tangential to the skull in an antero-posterior direction, so that the current flowed in a counterclockwise direction for activating the left M1 (right-side muscles). For reliability of coil placement, participants wore a snugly fitting cap, positioned with reference to the nasion–inion and interaural lines (31). The cap was marked with sites at 1-cm spacing in a latitude-longitude matrix to ensure reliable coil position throughout the testing protocol and for repeated testing sessions over the period of the study. The cap was checked constantly to ensure that no changes in cap position occurred. Sites near the estimated center of the BB area (4–7 cm lateral to the vertex) were explored to determine the site at which the largest MEP amplitude was observed, via visual inspection of the MEP waveform (Figure 1). This site was defined as the "optimal" site (31). At the optimal site, MEP stimulus–response curves were measured by delivering 2 sets of 5 TMS stimuli at intensities (5% of stimulator output steps) from a level below the participant's AMT until the plateau of MEP amplitude (i.e., until the amplitude did not increase with increased stimulation). The AMT was defined as the intensity at which an MEP could be obtained with at least 5 of the 10 stimuli with a peak–peak amplitude being greater than 200 μV during 10% of MVC rmsEMG (35). The MVC rmsEMG was determined from the participant who performed an isometric MVC of their BB muscle on the bench with their elbow flexed to 90° and was used to control for background muscle activity during TMS trials. Each set of 5 stimuli was delivered during a controlled, low level voluntary contraction of the BB muscle at 10% (±3%) of MVC rmsEMG (35). Feedback of the participant's rmsEMG level was displayed on a computer monitor positioned 1.5 m away at the eye level using custom-built software (National Instruments V4.0). Each stimulus was delivered in random intervals every 10–12 seconds to avoid stimulus anticipation and 30-second rest was provided between each set of stimuli to reduce the possibility of muscular fatigue.

Statistical Analyses

All MEPs collected ($n = 10$, 2 sets of 5 500-millisecond recordings, at each stimulus intensity from below participant's AMT to MEP_{max}, see Figure 1 for an example) were displayed and averaged online for visual inspection, in determining the optimal site, and then stored off-line for further analysis. Stimulus–response curves were constructed according to the protocol of Carroll et al. (6).

Stimulus intensity was plotted against MEP amplitude, and the data were fitted with a 3 parameter sigmoid equation:

$$\mathrm{MEP}(s) = \frac{\mathrm{MEP_{MAX}}}{1 + e^{m(S50-s)}},$$

where s is stimulus intensity, m is the estimated slope, $S50$ is the estimated peak slope, and MEP_{max} is the measured maximum the participant's MEP amplitude reached in a given trial. A nonlinear data fit iterative model to each participant's data using SPSS17.0 (SPSS Inc, Chicago, IL, USA) was applied. This procedure estimated the values for m and $S50$ and provided a measure of the curves fit to the data. All iterative fits significantly fitted the data.

All data were first screened for normal distribution. To have sufficient data to test for questions of normality, all MEP parameters (AMT, 20% above AMT and MEP_{max}) and dynamic 1RM strength data were used to establish the distributional properties. No variable's z-score of skew or kurtosis was excessive. Further, Shapiro–Wilk tests showed MEP amplitude at 20% above AMT, MEP_{max}, and dynamic 1RM strength variables were clearly normally distributed (20% above AMT, $SW = 0.9$, $p = 0.8$; MEP_{max}, $SW = 0.9$, $p = 0.2$; 1RM strength, $SW = 0.8$, $p = 0.1$). Although the MEP amplitude at AMT was apparently not normally distributed

(SW = 0.7, p = 0.01), this violation was only mild after examination of frequency histograms and detrended Q-Q plots and was not considered sufficient to warrant a more conservative analytic strategy. Consequently, it was decided to treat the data as essentially normal in distribution. To identify changes in the functional properties of the corticospinal pathway, the slope and plateau values of the stimulus-response curve were used to characterize the physiological strength of corticospinal connections projecting onto the spinal motoneuron pool innervating the right BB. Latency was calculated from stimulus artifact to MEP onset and MEP peak-to-peak amplitude was cursored and measured (32). Furthermore, MEP sweeps (n = 10) obtained at AMT, 20% above AMT and MEP_{max} were analyzed to quantify changes in membrane excitability and corticospinal cell recruitment after the strength training intervention (17). To test the hypothesis that unilateral strength training increases contralateral strength and corticospinal excitability, a 2-way analysis of variance, and Fisher's least significant difference procedure for post hoc testing, for the right arm was used to compare group interaction (trained vs. control) by testing session (pre vs. post) for each dependent variable (elbow flexion strength, rmsEMG, MEP latency, and amplitude). Test–retest reliability of the participants' girths, strength, and TMS data was assessed by applying coefficient of variation (CoV) using the method by Hopkins (20) and correlation coefficient. Data are presented as means (±*SD*) and effect size (ES) conventions were used for small (0.25), medium (0.5), and large (0.8) comparative effects (7). The level of significance used for all tests was set at $p \leq 0.05$.

Results

All participants completed the 4-week training intervention. Reliability in all measures (girths, strength, and TMS) in both the trained and control groups, before the strength training intervention, were demonstrated with a CoV of less than 1% (r = 0.99) for arm girth measures; 4.5% (r = 0.98) for strength measures and 3% for MEP amplitude between participants was variable and to be expected (38); however *intra*-participant MEP amplitudes (taken under 10% of MVC tonic contraction) were reliable with a CoV of 3.0% at AMT, 0.9% at 20% of AMT, and 1.2% at MEP_{max} (37).

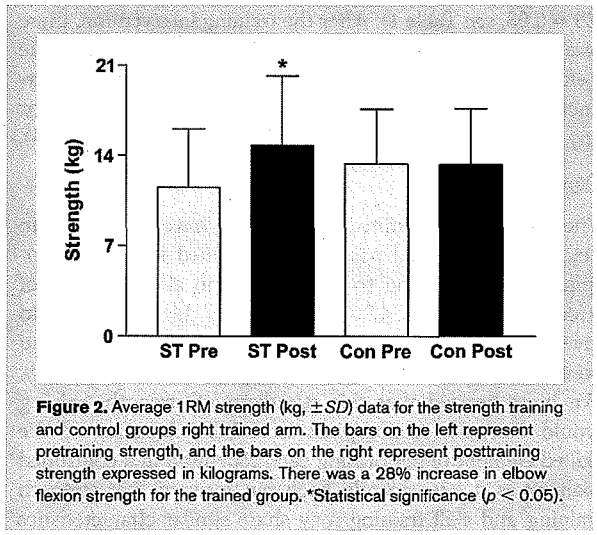

Figure 2. Average 1RM strength (kg, ±*SD*) data for the strength training and control groups right trained arm. The bars on the left represent pretraining strength, and the bars on the right represent posttraining strength expressed in kilograms. There was a 28% increase in elbow flexion strength for the trained group. *Statistical significance ($p < 0.05$).

Voluntary Muscle Strength

Figure 2 represents absolute changes (kg, ± *SD*) in strength after the training intervention for the training and control groups. There were no significant differences in dynamic elbow flexion strength (1RM) at baseline between the control and trained groups for the right arm (p = 0.86). After the 4-week training intervention, 1RM elbow flexion strength increased by 28% (p = 0.0001, ES = 0.67) in the trained group (11.5 ± 4.5 to 14.8 ± 5.2 kg; Figure 2). There were no differences in 1RM elbow flexion strength for the control group (13.3 ± 4.2 to 13.2 ± 4.3 kg; p = 0.34).

Arm Circumference

No significant differences were observed in muscle girths between groups at pretraining (right arm trained group pre 31.9 ± 5.6 cm vs. control group pre 31.3 ± 5.2 cm, p = 0.4). No significant differences in arm girths were observed within and between groups after the training period (right arm trained group post 32.2 ± 4.9 cm vs. control group post 31.4 ± 3.3 cm, p = 0.3).

Muscle Activation–rmsEMG

There were no significant differences at pretraining for group mean right BB MVC rmsEMG activity between the groups (control, right arm: 0.41 ± 0.24 mV; trained, right arm: 0.50 ± 0.20 mV, p = 0.5). There were also no differences after training to pretraining values within or between the groups (control, right arm: 0.41 ± 0.21 mV; trained, right arm: 0.58 ± 0.17 mV, p = 0.5). Further, no interaction was found between groups by training (p = 0.7). Similarly, no differences were observed between rmsEMG at 10% of MVC contraction pre and posttesting sessions (precontrol, right arm: 0.04 ± 0.02 mV; pretrained, right arm: 0.05 ± 0.02 mV, p = 0.4; postcontrol, right arm: 0.04 ± 0.02 mV; posttrained, right arm: 0.05 ± 0.01 mV, p = 0.5).

Latency

No significant differences in latency duration were seen between groups at 20% above AMT at pretraining (left M1, p = 0.2). After the training intervention, there was no significant difference in latency duration pre vs. posttraining in both trained (left M1: 13.1 ± 0.8 vs. 12.9 ± 0.3 milliseconds, p = 0.3) and control groups (left M1: 12.9 ± 0.50 vs. 12.8 ± 0.5 milliseconds, p = 0.4).

Active Motor Threshold and Motor-Evoked Potentials

Mean group data for the control and the trained groups for percentage of stimulator output at AMT are shown in Table 1.

Table 1. Mean ± *SD* data for percentage of stimulator output at AMT (%), MEP amplitude at AMT, 20% above AMT and MEP_{max} (mV) before and after the 4-week strength training intervention for the control and trained groups left M1.*†

Group		Stimulator output at AMT (%)		AMT amplitude (mV)		MEP amplitude (mV) @ 20% above AMT		MEP_{max} (mV)	
		Pre	Post	Pre	Post	Pre	Post	Pre	Post
Control	Left M1	49.37 ± 6.7	45.62 ± 7.7	1.04 ± 0.72	0.93 ± 0.19	2.87 ± 1.42	2.76 ± 0.93	3.35 ± 1.81	3.39 ± 1.34
Trained	Left M1	50.0 ± 10.4	49.5 ± 9.6	0.82 ± 0.28	1.26 ± 0.51†	2.45 ± 0.92	3.26 ± 1.19†	2.81 ± 0.96	3.89 ± 1.09†

*AMT = active motor threshold; MEP = motor evoked potential; MEP_{max} = saturation of the MEP; M1 = primary motor cortex.
†Significant time effect, $p < 0.05$.

There were no significant differences at pretraining for the percentage of stimulator output at AMT within and between the trained and control groups left M1 ($p = 0.3$). After the training period, there were no significant differences for percentage of stimulator output at AMT between the trained and control groups (control left M1 vs. trained left M1; $p = 0.8$; Table 1).

Table 1 displays mean data for the control and trained groups for mean MEP amplitude at AMT, 20% above AMT and MEP_{max} between groups before and after strength training. There was no significant difference in mean MEP amplitude at AMT at baseline between groups (left M1: $p = 0.16$). The MEP amplitude at AMT increased by 53% ($p = 0$ 0.01, ES = 1.07) in the left M1 in the trained group after the training intervention. There were no significant differences ($p = 0.32$, ES = 0.2) in the mean MEP amplitude at AMT in left M1 in the control group after the training intervention. Further, there were no interaction effects between the groups ($p = 0.2$, ES = 0.4).

There were no significant differences in the estimated slope (m) of the input–output curve after strength training in the trained group (pre: 0.16 AU ± 0.06 AU, post: 0.15 AU ± 0.05 AU, $p = 0.4$, ES = 0.18) for the left M1. Furthermore, no significant differences were identified for $S50$ after the training intervention for the left M1 (left M1: 4.9 AU ± 3.7 AU, post; 5.6 AU ± 4.8 AU, $p = 0.6$, ES = 0.16). There were no difference in the mean MEP amplitude at 20% above AMT at pretraining between groups (left M1: $p = 0.4$, ES = 0.33).

There were also no significant differences ($p = 0.6$, ES = 0.09) in the MEP amplitude for the left M1 at 20% above AMT in the control group; however, there was a significant increase (33%) ($p = 0.05$, ES = 0.9) in the trained group after the intervention. No significant interaction effect was observed between the groups (left M1: $p = 0.1$, ES = 0.4).

There were no significant differences in mean MEP_{max} amplitude at pretraining between groups (left M1: $p = 0.4$; ES = 0.08, see Table 1). After the training intervention, there was a 38% increase in the amplitude of the MEP_{max} in the Left M1 ($p = 0.02$, ES = 1.1). There were no significant differences ($p = 0.8$, ES = 0.02) detected for the control group or any significant interaction effects (left M1: $p = 0.3$, ES = 0.4).

Discussion

There have been limited studies that have attempted to determine the neural adaptations confined to the corticospinal tract projecting to the upper limb after a period of strength training (6,24,26). The aim of this study was to investigate the corticospinal responses after heavy-load controlled isotonic strength training of the elbow flexors. We hypothesized that this paradigm of strength training would increase corticospinal excitability because of the skilled element of performing each repetition in a controlled manner as it has recently been put forward that strength training and skill training may share similar characteristics

that result in changes in corticospinal output (10,31). The main findings of the study were the significant increases in 1RM strength, in the absence of muscle hypertrophy and increased MEP amplitude at and above AMT. We found a 28% increase in 1RM strength after the strength training intervention concurring to previous short-term strength training studies that have used both isotonic and isometric contractions across a range of upper and lower limb muscles (11,16,24,26).

It has been proposed that increased excitability of populations of corticospinal cells projecting to spinal motoneurons controlling the trained muscles, may account for some of the observed increases in strength (23). The finding in this study of increased MEP amplitude at, and above AMT evoked by TMS demonstrates that heavy-load strength training altered the excitability of the corticospinal tract projecting to spinal motoneurons innervating the BB muscle. These findings are consistent with increases in corticospinal excitability after strength training that have previously been reported by Griffen and Cafarelli (16) and Beck et al. (4), however, inconsistent with the findings from Lee et al. (26), Carroll et al. (6), and Jensen et al. (24), who reported either a decrease or no change in corticospinal excitability. The factors that may contribute to the potential differences across studies, most likely reside in the different muscles subjected to strength training, the type of strength training employed, the conditions in which TMS was elicited and the strength of corticospinal projection to the spinal motoneurons innervating the trained muscles being different. For example, Lee et al. (26) strength trained the wrist abductors by performing 4 sets of 8 repetitions at 75% 1RM and increasing up to 85% 1RM. Also, TMS was applied to M1 during wrist abduction and extension and, as acknowledged by the authors, there may have been a lack of training and testing specificity (i.e., cortical stimulation during wrist abduction was obtained, but not adduction, although participants trained though wrist adduction). Further, the authors also suggested, which is in accordance with previous research (19), that voluntary activation of the extensor carpi radialis brevis was high for all participants at pretraining, suggesting that there was little room for improvement in neural drive. Similarly, the earlier study by Carroll et al. (6) strength trained an intrinsic hand muscle (FDI), where participants completed 4 sets of 6 repetitions at 70–85% 1RM. Each repetition was performed slowly; however, the exact repetition timing (tempo) was not provided, despite repetition tempo being an important component to exercise prescription and strength development (27). Likewise, Jensen et al. (24) had participants perform bicep curls for 4 weeks. The exact load lifted by participants throughout the training period, the timing of each repetition and how progressive overload was applied were not reported. Therefore, it may be necessary that all parameters of a strength training program (e.g., exercise selection, training load, repetition speed, and progressive overload) be accurately monitored. In this study, we precisely controlled the timing to perform each repetition and increased the training load by 5% as soon as a participant could complete 4 sets of 8 repetitions. Another unique aspect to this study was that we actually started training novice participants at 80% of their 1RM, without any reported contraindications, while other studies started training at a lower intensity with a gradual increase up to 80%. Therefore, the novelty of this study was the use of a heavy-load in novice participants, the controlled timing to perform each repetition and this may explain the observed differences in corticospinal excitability between this study and that of others. Further, the consistent finding between this and that of Griffin and Cafarelli (16) and Beck et al. (4) study appears to reside in the type of strength training performed and the manner in which the repetitions were performed. Griffen and Cafarelli (16) and Beck et al. (4) isometrically strength trained the TA in a rapid manner (6 × 10 and 4 × 10 MVCs, respectively), demonstrating an increase in both strength and corticospinal excitability. It appears that training intensity and the manner in which the repetitions are performed are important for increasing neural transmission via the corticospinal pathway. Rapid isometric contractions and maximal strength training have previously been shown to elicit increases in corticospinal drive (8,11); therefore, the use of a heavy resistance in this study adds to the suggestion that training load and the manipulation of repetition velocity (i.e., tempo) is important for stimulating changes in strength that are mediated by the nervous system. The consistent findings between studies (i.e., 4, 8, 11,16, and this study) have important practical implications by demonstrating that to maximize strength gains via changes in neural control, strength and conditioning coaches should focus on exercise technique, training intensity, and repetition velocity.

Because repetitive skill training (both short term and long term) has been shown to increase neural excitability within populations of corticospinal cells (30), it may be important that the type of strength training prescribed should focus on skilled movements that challenge the nervous system (10,24). For example, increases in corticospinal excitability after ballistic strength training, which requires acceleration and deceleration forces has been shown to increase corticospinal excitability (4,36). Even though it has been suggested that corticospinal excitability may be unchanged after strength training because of low task complexity and limited peripheral feedback (21,24), repetitive movements against resistive loads that require a certain level of task complexity may underpin the corticospinal responses observed within this study and that of others (36). The time to complete each repetition employed in the present strength training program (3-second concentric and 4-second eccentric) increased the skilled element of performing a standard bicep curl exercise, leading to increased peripheral feedback, which has resulted in increased corticospinal excitability and this is consistent with previous research (21,22). Therefore, purposefully controlling the repetition tempo during both the concentric

and eccentric phases in this study has resulted in increased task complexity and peripheral feedback, which has led to increased corticospinal excitability. The repetition tempo used was based on our previous work that demonstrated increased corticospinal excitability when participants performed the same task, with the same force levels, under different levels of precision, being the timing of the movement (31). Therefore, we specifically prescribed a repetition tempo that would increase the level of precision and this may have contributed to an increase in task complexity and thus altered neural transmission via the corticospinal pathway.

The increase in the mean amplitude of the descending corticospinal volley at MEP_{max} after strength training lends support to the concept of activity-dependent changes in corticospinal output and adaptive changes within the intrinsic properties of the corticospinal tract after strength training. These changes in corticospinal excitability (e.g., changes in corticospinal cell recruitment) likely reflect changes in cortical synapse number and/or synaptic strength (2). Further, the strength training program used has resulted in some form of adaptation in the efficacy of existing corticospinal connections projecting onto the spinal motoneurons controlling the BB and these changes in MEP amplitude reflect the "unmasking" of dormant pre-existing corticospinal connections (2). Such connections are widespread and exhibit activity dependent modifications in synaptic strength within the corticospinal pathway after the acquisition of novel tasks by activating excitatory corticospinal cells (18). Because AMT and MEP amplitude are 2 related, but independent measures of corticospinal excitability, the observed changes in MEP amplitude at and above AMT, suggest that the strength training intervention has resulted in a shift in the balance between inhibitory and excitatory inputs onto cortical and/or spinal motoneurons. Moreover, the change in MEP amplitude above AMT, demonstrates an increase in the number and size of the descending volleys generated by the cortical stimulus or from an increase in the number of corticospinal cells activated. Overall, these changes suggest that, in the strength training group, there has been a change in the level of cortical and/or spinal excitability.

Although we have reported increased corticospinal excitability, given the divergent pattern of corticospinal cell projection onto spinal motoneurons and that MEPs evoked by TMS represent the entire corticospinal tract (9), it is possible that changes in strength may be related to adaptations in neural circuits not confined to the corticospinal tract. There may have been small adaptive changes at multiple sites within the central nervous system (CNS), which combined may have altered the way in which the BB was activated. There may also have been modifications in spinal motoneuron activity, subcortical neurons that are innervated by corticospinal tract fibers, and alterations within intracortical circuits. The observed changes in corticospinal excitability after strength training could simply reflect changes from input from other neural circuits that influence the excitability of existing corticospinal cells. Aagaard et al. (1) demonstrated significant increases in evoked V-wave responses during MVCs after strength training, suggesting increased efferent drive (descending drive) from higher neural centers leading to increased α-motorneuron excitability. This concept of increased descending drive has recently been supported by Del Balso and Cafarelli (8) and by Fimland et al. (11), who both reported increased supraspinal drive after 4 weeks of strength training a lower limb muscle. Because the corticospinal tract as a whole includes cortical circuitry, the motorneuron pool and its intrinsic properties, and spinal interneuronal pathways (35), increased excitability of the motorneuron pool and changes in presynaptic inhibition may have occurred and could explain the increases in strength observed. However, Griffen and Cafarelli (16) suggested that the larger MEP after training resulted from an increase in motor unit recruitment, indicating multiple potential sites of adaptation within the CNS after strength training (25). Because single motor unit behavior and spinal cord reflexes were not performed in this study, it cannot therefore, be excluded that modifications in the efficacy of neural transmission across synaptic connections between corticospinal fibers and spinal motoneurons may have occurred, thus in part explaining the changes in strength.

In light of this, this investigation demonstrated, using TMS, an increase in MEP amplitude during a 10% rmsEMG background contraction at stimulus intensities at and above AMT after 4 weeks of strength training the BB muscle. These data suggest increased excitability of pre-existing but dormant connections within corticospinal cells projecting onto the motorneuron pool innervating the BB and adaptive changes within the stimulus–response properties of the corticospinal tract, possibly contributing to the changes in strength. It cannot be ruled out that changes in excitability or inhibition within other neural circuits in the nervous system not confined to the M1 and corticospinal tract were also involved. Despite this, the data extend on the current research by demonstrating changes in corticospinal excitability; however, further research is still required as there still remains contradictory evidence for neural adaptations confined to the M1 and corticospinal tract after a period of strength training. Therefore, at present, it is difficult to identify the specific neural mechanism that contributes to the observed changes in strength, suggesting that the adaptive neural response to strength training are most likely because of multiple mechanisms confined to both cortical and subcortical regions of the nervous system. In an attempt to explore the corticospinal responses to strength training in greater detail, future studies should investigate the task-dependent effects of different types of strength training by comparing isometric strength training to dynamic strength training to determine if the corticospinal responses are task dependent. In addition, future investigations should also examine the corticospinal responses after different repetition timing of movement and the effect of heavy-load strength

training (such as that used in this study) on different muscles, such as the deltoid.

Practical Applications

The findings from this study have a number of practical applications to strength and conditioning practitioners. For example, the data demonstrate that controlled tempo isotonic strength training increases corticospinal drive onto spinal motoneurons, and this results in improved force production and this has important implications for neuromuscular rehabilitation. Purposefully controlling the tempo of each repetition increases neural drive that may be modulated by mechanisms associated with increased peripheral feedback (21,22). Increasing peripheral feedback and proprioception are important for regaining strength after injury. For the strength and conditioning practitioner, these data demonstrate that heavy-load controlled strength training increases neural drive and would support the notion for prescribing heavy-load training to induce maximal strength increases.

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