J Appl Physiol 103: 402–411, 2007.
First published 19 April 2007; doi:10.1152/japplphysiol.00477.2006.

HIGHLIGHTED TOPIC | *Neural Changes Associated with Training*

Adaptations in the activation of human skeletal muscle induced by short-term isometric resistance training

Christopher Del Balso and E. Cafarelli

Kinesiology and Health Science, Faculty of Science and Engineering, York University, Toronto, Ontario, Canada

Submitted 1 January 2006; accepted in final form 2 January 2006

Del Balso C, Cafarelli E. Adaptations in the activation of human skeletal muscle induced by short-term isometric resistance training. *J Appl Physiol* 103: 402–411, 2007. First published 19 April 2007; doi:10.1152/japplphysiol.00477.2006.—This study employed longitudinal measures of evoked spinal reflex responses (Hoffman reflex, V wave) to investigate changes in the activation of muscle and to determine if there are "linked" neural adaptations in the motor pathway following isometric resistance training. Twenty healthy, sedentary males were randomly assigned to either the trained ($n = 10$) or control group ($n = 10$). The training protocol consisted of 12 sessions of isometric resistance training of the plantar flexor muscles over a 4-wk period. All subjects were tested prior to and after the 4-wk period. To estimate changes in spinal excitability, soleus Hoffman (H) reflex and M wave recruitment curves were produced at rest and during submaximal contractions. Recruitment curves were analyzed using the slope method (H_{slp}/M_{slp}). Modulation of efferent neural drive was assessed through evoked V wave responses (V/M_{max}) at 50, 75, and 100% maximal voluntary contraction (MVC). After 4 weeks, MVC torque increased 20.0 ± 13.9% (mean ± SD) in the trained group. The increase in MVC was accompanied by significant increases in the rate of torque development (42.5 ± 13.3%), the soleus surface electromyogram (60.7 ± 30.8%), voluntary activation (2.8 ± 0.1%), and the rate of activation (48.7 ± 24.3%). H_{slp}/M_{slp} was not altered by training; however, V/M_{max} increased 57.3 ± 34.2% during MVC. These results suggest that increases in MVC observed in the first few days of isometric resistance training can be accounted for by an increase in the rate of activation at the onset of muscle contraction. Augmentation of muscle activation may be due to increased volitional drive from supraspinal centers.

Hoffman reflex; V wave; soleus

ADAPTIVE ALTERATIONS in the neuromuscular system can be induced in response to specific types of training. It has been well established that progressive isometric resistance training can lead to increases in maximal contractile torque (42, 43). However, the specific mechanisms responsible for this adaptation during the first few weeks of training are not fully understood. Adaptive changes in neural function following training are commonly investigated using the surface electromyogram (EMG) as an indicator of change in efferent neural drive. Several investigations have reported increases in integrated EMG (37, 39) with resistance training; however, this has not been demonstrated consistently (38, 43).

Disproportionate increases in maximal voluntary contraction (MVC) torque and muscle cross-sectional area (CSA) following resistance training have been reported with a variety of techniques, including computerized tomography (17), and magnetic resonance imaging (38). Small but significant increases in muscle CSA have been observed, but it is difficult to attribute all of the relatively large increases in MVC to muscle hypertrophy at the onset of training. A case has therefore been made to implicate neural factors for the increase in maximal voluntary contraction (MVC) during the first few weeks of resistance training.

Although the effect of resistance training on muscle morphology has been thoroughly examined, less is known about the specific neural mechanisms responsible for training-induced increases in MVC. Increasingly, intramuscular recordings and EMG normalization procedures have been used successfully to reduce some of the limitations of the use of surface EMG alone. As well, measurements of evoked spinal reflex responses (Hoffman reflex, V wave) have been used to examine the changes in neural function that occur with training (3, 20).

The Hoffman (H) reflex is often used to assess the excitability of the α-motor neuron pool in vivo. The H reflex may best be interpreted as an estimate of "net spinal excitability", which includes the summation of excitatory and inhibitory descending and afferent synaptic input, presynaptic inhibition, and intrinsic motor neuronal properties (15). Cross-sectional studies have shown decreased spinal excitability in power-trained and sprint athletes compared with endurance athletes (10, 34, 44). Longitudinal investigation has shown increased evoked H reflex responses during MVC following 14 wk of dynamic resistance training (3), but no change during MVC following five weeks of electromyostimulation training (20). Lack of consistency in the results of these studies may be due to different training protocols and/or methodological constraints.

The V wave is an electrophysiological variant of the H reflex (48, 52) that has been proposed as an estimate of efferent neural drive from spinal α-motor neurons during voluntary activation of muscle (3, 46). This has recently been verified by Levenez et al. during fatigue (32). To elicit a V wave, supramaximal stimulation is applied to a mixed peripheral nerve during muscle contraction. In motor neuron axons, orthodromic action potentials result in activation of the target muscle. Simultaneously, antidromic action potentials collide

Address for reprint requests and other correspondence: E. Cafarelli, 346 Bethune College, York Univ., 4700 Keele St., Toronto, Ontario M3J 1P3, Canada (e-mail: ecaf@yorku.ca).

The costs of publication of this article were defrayed in part by the payment of page charges. The article must therefore be hereby marked "*advertisement*" in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.

with a second wave of orthodromic action potentials generated as a result of descending input. Collision between these antidromic and orthodromic potentials results in a cancellation of the two signals. As a result, the orthodromic reflex volley in Ia-afferent axons is allowed to pass to the muscle, where it is recorded as the V wave. Increases in V wave amplitude have been observed in response to resistance training (3, 20); however, not all have observed a corresponding change in MVC (46).

The evoked spinal reflex responses have distinct utility in resistance training investigations. While both the H reflex and V wave may reflect training-induced changes that occur at the spinal level (i.e., alterations in excitability of the α-motor neuron pool, presynaptic inhibition), the V wave may also be indicative of training-induced changes mediated supraspinally, as it is thought to provide an estimate of neural drive in descending corticospinal pathways (3).

The purpose of the present investigation was to elucidate adaptive alterations in neural function during four weeks of isometric resistance training. We have attempted to link changes in descending volitional drive, the spinal cord, the α-motor neuron pool, and the rates at which muscle is activated and develops torque to an early increase in MVC. Longitudinal measures of evoked H reflex and V wave responses were employed to determine if changes in spinal excitability and/or volitional drive *1*) occur in conjunction with an augmentation of skeletal muscle activation, and *2*) can collectively account for an increase in MVC torque observed following isometric resistance training.

METHODS

Subjects. Twenty healthy, sedentary males were randomly assigned to either the trained (23.8 ± 4.8 y, 176.0 ± 8.6 cm, 80.7 ± 10.0 kg, mean ± SD) or the control group (22.8 ± 3.0 y, 179.6 ± 7.6 cm, 79.7 ± 11.7 kg, mean ± SD) for this repeated measures study. Each was screened for any existing medical conditions or history of right knee or ankle injury. Applicants who were more than recreationally active, diagnosed with neurological pathologies, or on prescription medications, nutritional supplements, or ergogenic aids were excluded from the study.

All participants attended an orientation session to allow for familiarity with the experimental apparatus and protocol, and to sign an informed consent document outlining the experimental procedures and potential side effects of the protocol. Monetary compensation was provided for participation in the study. The protocol was in accordance with the Declaration of Helsinki and approved by the York University Human Participants Review Committee.

Apparatus. Subjects sat upright in a seat mounted on a hydraulic base that was secured to a large platform. A custom-made dynamometer (York University Machine Shop) clamped to the same platform fixed the right hip, knee, and ankle at 90° of flexion. Velcro straps secured the heel and forefoot to an aluminum plate, to which a strain gauge was bonded. Padded clamps placed above the flexed knee joint and against the anterior aspect of the lower leg prevented movement during isometric contractions of the plantar flexors. With the knee joint at 90° of flexion, the contribution of the two-joint gastrocnemius to the production of plantar flexion torque is considerably reduced (13, 28). A three-point seat belt was used to ensure the subject was appropriately restrained when experimental measures were made.

Experimental protocol. All participants attended the laboratory for one experimental session prior to a 4-wk regimen. In this session, initial measures of MVC, voluntary activation, and twitch torque were obtained off-line from the same recording. Subsequently, baseline H reflex and M wave recruitment curves were generated at rest (0% MVC) and during muscle activation (10% MVC). Finally, V wave amplitudes and the associated voluntary activation were recorded and calculated at contraction intensities of 50, 75, and 100% MVC. Upon completion of baseline measures, the trained group engaged in the first of 12 sessions in the isometric resistance training protocol. For the trained group, experimental measures were made in each session for the duration of the protocol. Evoked spinal reflex responses were obtained at the mid-point of the protocol, prior to training on *day 7*, and at the end of the four weeks. Control group experimental measures were only obtained pre-post to avoid the possibility of a training effect.

Training. The trained group attended three sessions per week, consisting of 6 sets of 10 MVCs (3–4 s duration) of the plantar flexor muscles of the right leg. MVCs were performed at a rate of 4 contractions/min, with a 2-min rest between sets. Training took place in the same apparatus as was used for data collection. MVC and voluntary activation were recorded and calculated in each training session. In addition, surface EMG was recorded to allow for assessment of electrical changes as the training program progressed. Post-training experimental measures were made no earlier than 24 h and no later than 48 h after completion of the final training session.

Electromyography. To record the electrical activity of the soleus, a bipolar silver-silver chloride surface electrode (EQ; Chalfont, PA) with a 0.8-cm diameter and an interelectrode distance of 2.0 cm was positioned just lateral to the mid-dorsal line of the leg, ~5 cm distal to the lateral head of the gastrocnemius. The skin under the electrode was shaved, exfoliated, and cleaned with a 70% ethanol solution before placement of the recording electrode. To ensure accurate positioning of the recording electrode, subjects were required to perform preliminary isometric plantar and dorsiflexion contractions. Any activity in the EMG channel during dorsiflexion would reflect pick-up of electrical activity from another muscle (i.e., tibialis anterior) and required repositioning of the recording electrode. In addition, the shape of the M wave accompanying an electrically induced twitch was monitored; anything other than the typical biphasic shape would imply pick-up of electrical activity from another muscle (i.e., gastrocnemius) and would again require repositioning of the recording electrode. Day-to-day variation in recording electrode placement was avoided through measurement and marking (permanent marker) of the area. A strap electrode soaked in water and covered in plastic wrap to prevent evaporation was used as a ground, positioned proximal to the recording electrode over the gastrocnemius muscle belly. The EMG signal was preamplified at the source and then passed through a variable gain second stage amplifier. The amplitude of the voluntary surface EMG was derived from the root mean square (RMS) of a 0.5-s epoch coinciding with peak torque but excluding the superimposed M wave.

To estimate the maximal rate of soleus muscle activation, we measured the rate of increase of the surface EMG from the onset of electrical activity to the point that maximal rate of MVC torque development ($+dF/dt_{max}$) occurred in the torque recording. To make this calculation, the surface EMG signal was full-wave rectified and then integrated as a function of time. The slope of a line fit to the integral by the method of least squares was then taken to represent the rate at which the soleus was electrically activated (43). Figure 1 provides a visual representation of the calculation of the rate of soleus muscle activation.

Electrical stimulation. Twitches, M waves, H reflexes, and V waves were elicited through electrical stimulation of the right tibial nerve with carbonized rubber electrodes, each covered with a conductive gel. The cathode (2.5 × 2.5 cm) was placed in the popliteal fossa, directly over the nerve, and the anode (4 × 5 cm) was positioned over the patella. Both electrodes were fixed securely to the limb with a Hypafix dressing retention sheet.

Electrical stimuli (duration = 1 ms) were produced by a constant current stimulator (Digitimer model DS7A; Hertfordshire, England). A stimulus intensity of 110% was used for measurement of twitches,

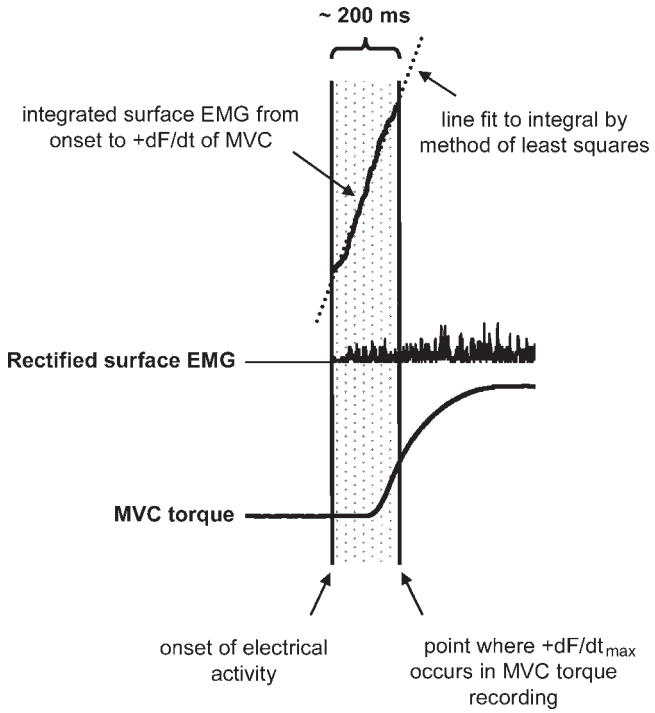

Fig. 1. Calculation of rate of activation of soleus. The slope of the line fit to the integral of the rectified surface electromyogram (EMG), from the onset of electrical activity to the point where $+dF/dt_{max}$ occurred in the torque channel (~200 ms), was taken as the rate of soleus muscle activation. MVC, maximal voluntary contraction.

maximal M waves (M_{max}), V waves, contractile properties, and calculation of voluntary activation via twitch interpolation. Percent voluntary activation was calculated as [1-(superimposed twitch/maximal twitch)] × 100; (6).

Maximum voluntary contraction. To obtain recordings of MVC, subjects were instructed to contract the right plantar flexors as hard and fast as possible. To ensure optimal measurement of MVC, standardized verbal encouragement was employed according to the criteria set forth by Gandevia (16). In this experiment, MVC was determined as the average of the three largest torques produced within 10% of each other. This value was used subsequently to determine relative submaximal contraction intensities. Data from MVC trials were also used to determine $+dF/dt_{max}$.

H reflex recordings. H reflex responses obtained during actual voluntary contraction represent a more functional estimate of training-induced changes in spinal excitability (3, 55). Accordingly, H reflex and M wave recruitment curves were produced at rest and during a voluntary contraction of 10% MVC. Curves were produced by progressively increasing the intensity of electrical stimulation to the tibial nerve. A series of 60 1-ms pulses, beginning at 15% of the stimulus intensity required to evoke a maximal twitch response (I_{max}), was used to generate each set of curves. Current was increased in intensity with an inter-stimulus interval of 4 s, until M_{max} was elicited. Stimulation intensity was increased by 1% of I_{max} initially, to ensure an accurate depiction of the developmental slope of the H reflex curve. For the second half of the curve, stimulation intensity was increased by 2% of I_{max}, producing a recruitment curve consisting of 60 data points. Additional stimuli were applied when necessary to ensure that M wave amplitude reached a plateau. Peak-to-peak H reflex and M wave amplitudes were measured online and displayed as a function of time in a data channel, such that the recruitment curves could be viewed as they were generated. To ensure inactivity of the soleus while recording recruitment curves at rest, surface EMG was fed through a window discriminator to an audio amplifier. Any increase in the EMG above baseline noise would produce an audio signal, and the subject would be instructed to relax; any affected stimuli were repeated. For production of H reflex and M wave recruitment curves during muscle activation, torque was kept within 0.5% of the target torque of 10% MVC via visual feedback.

To attain measures of spinal excitability, H reflex data were analyzed using the slope method (H_{slp}/M_{slp}) previously described (27). In this method, the peak-to-peak amplitudes of the H reflex and M waves were normalized to the M_{max} amplitude and plotted as a function of stimulus intensity to create H reflex and M wave recruitment curves for each subject. H_{slp}/M_{slp} involves using the ratio of the slopes of the H reflex and M wave recruitment curves. The slopes were taken from the linear regression lines calculated for both the H reflex and M wave recruitment curves. The analysis of H_{slp} included all data points between H reflex threshold and M wave threshold. The analysis of M_{slp} included all data from the threshold of the M wave to 90% of the maximal M wave. Sample recruitment curves for one subject are shown in Figs. 2*A* and *B*.

V wave recordings. The V wave as a measure of efferent neural drive has been discussed previously (46, 48, 52). In the present investigation, V waves were produced via supramaximal electrical stimulation (i.e., 110% of the current which produced a maximal twitch) delivered during randomized sets of five 3- to 4-s voluntary contractions of 50, 75, and 100% MVC. Subjects were given 30-s

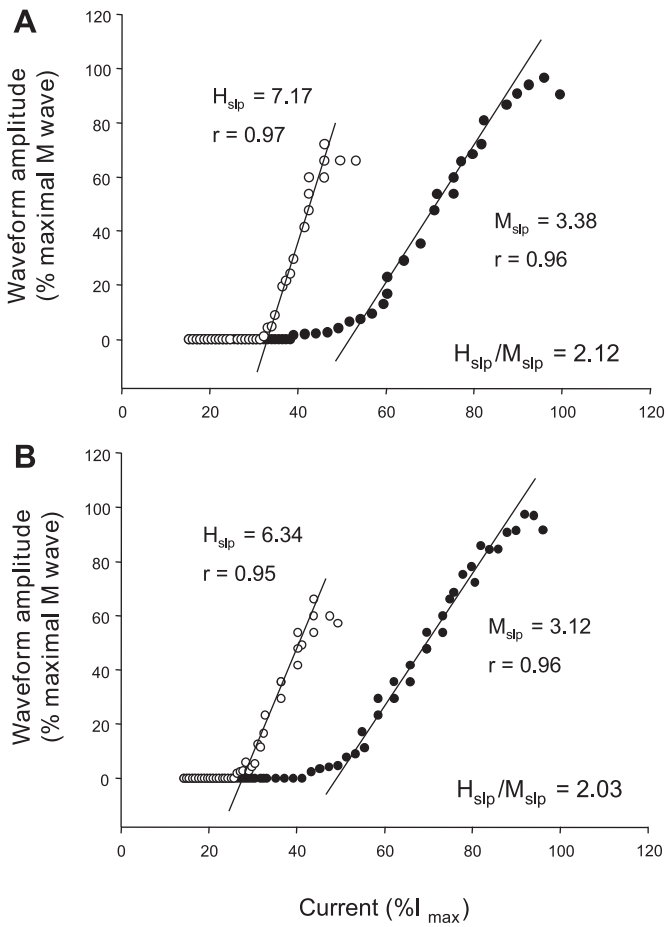

Fig. 2. Hoffman (H) reflex and M wave recruitment curves produced at rest, for one control subject. Peak-to-peak H reflex amplitude (○) and M wave amplitude (●) are plotted as a function of increasing current intensity. H reflex and M wave recruitment curves produced at rest (*A*, pretest; *B*, posttest) show the slope method (H_{slp}/M_{slp}) used for assessment of spinal excitability. Recruitment curves produced during muscle activation (10% MVC) are not shown. I_{max}, maximal twitch response.

rests between contractions, and 2-min rests between sets. Measurement of peak-to-peak amplitude of the V wave was normalized to that of the maximal M wave (V/M_{max}). Maximal twitch responses were elicited after each contraction for the purpose of calculating the associated voluntary activation.

V wave analyses were performed at the time of the pretest on *day 1*, prior to training on *day 7* (i.e., training group only), and during the posttest on *day 13*. Posttest V wave data were collected twice (posttest-V1, posttest-V2) so that we could assess changes in both V wave amplitude and percent voluntary activation after training. The second data collection (posttest-V2) was performed using the same absolute MVC torque as the pretest to calculate contraction intensities of 50, 75, and 100% MVC.

Data processing. Amplified torque and EMG signals were pulse-code modulated (Vetter digital, Model 4000A; Rebersberg, PA) and stored on videocassette (Sony VCR, Model SLV-N80) for subsequent off-line analysis. Digitized signals (micro1401, CED; Cambridge, UK) were filtered and processed using Spike2 for Windows (version 5.12, CED; Cambridge, UK). Torque was digitized at 1,000 Hz and low-pass filtered (50 Hz cutoff). Soleus EMG was digitized at 2,500 Hz and high-pass filtered (20 Hz cutoff).

Statistical analysis. All statistical analyses were conducted with Statistica (version 6.0, Statsoft; Tulsa, OK). To determine spinal excitability, recruitment curves were fit by linear regression and the correlation coefficient was always greater than 0.92. Repeated-measures ANOVA followed by contrast analyses with a modified Bonferroni correction procedure were performed to determine the effect of training day for each variable. Trained group data and control group data were analyzed separately. Control group data were used to test repeatability of experimental measures, allowing one-way analyses of trained group data to delineate the time-course of adaptations to the isometric resistance training protocol. Data are presented as mean ± standard deviation.

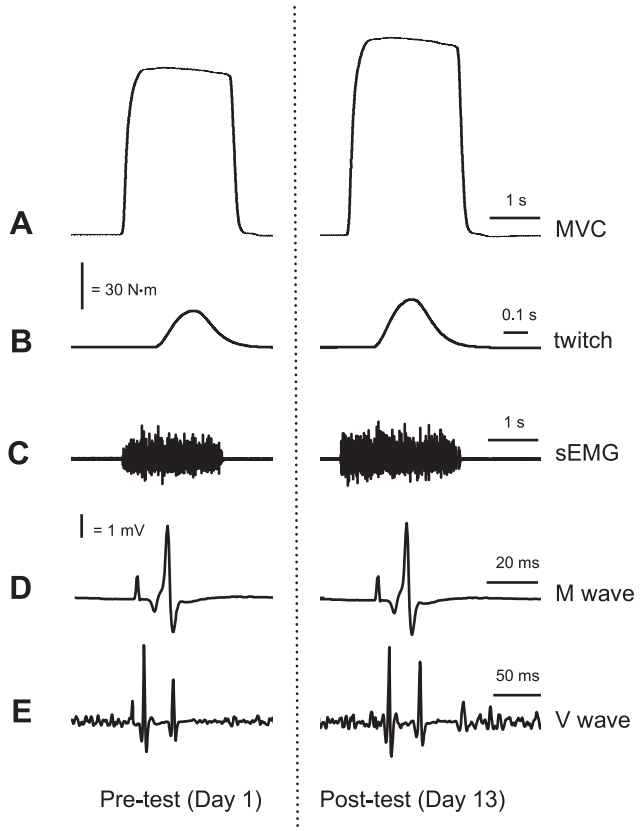

Fig. 3. Sample recordings from one trained subject. Recordings of MVC torque (*A*), twitch torque (*B*), surface EMG (sEMG, *C*), M wave (*D*), and V wave (*E*) are shown. Differences between the pretest and posttest can easily be observed.

RESULTS

There were no significant differences between the trained and control groups with regard to anthropometric data, nor were there any differences between the groups in any of the pretest measures. Figure 3 shows sample pre-post recordings from one trained subject.

Voluntary torque and activation. MVC torque increased 20.0 ± 13.9% ($P < 0.001$) in the trained group (*day 1*, 106.6 ± 18.0 N·m; *day 13*, 128.0 ± 17.8 N·m), while no change was observed in average MVC torque for the control group (*day 1*, 104.6 ± 25.7 N·m; *day 13*, 103.5 ± 25.3 N·m). The increase in MVC torque observed for the trained group was different from baseline by the third training day ($P < 0.005$) and remained as such for the remainder of the protocol (Fig. 4*A*). The MVC $+dF/dt_{max}$ increased in a manner similar to that of MVC, becoming significantly greater than the pretest value by *day 3* ($P < 0.005$), and increasing 42.5 ± 13.3% overall ($P < 0.002$; *day 1*, 454.2 ± 113.1 $N \cdot m \cdot s^{-1}$; *day 13*, 647.2 ± 86.3 $N \cdot m \cdot s^{-1}$; Fig. 4*B*).

The increase in MVC torque in the trained group was accompanied by an increase in voluntary activation assessed by means of both surface EMG as well as twitch interpolation. Because there was no change in M wave amplitude after training, we did not normalize the surface EMG signal to the M wave. The RMS-dervied soleus surface EMG (EMG_{RMS}) increased 60.7 ± 30.8% from *day 1* to *day 13* ($P < 0.001$; Fig. 5*A*). The increase in EMG_{RMS} initially became significant by *day 7*, the midpoint of the training protocol ($P < 0.001$). Voluntary activation assessed by means of twitch interpolation significantly increased 2.8 ± 0.1% ($P < 0.001$) over the course of training (Fig. 5*B*). By *day 3*, voluntary activation had significantly increased from 97.2 ± 1.3% on *day 1* to 99.1 ± 0.7% ($P < 0.002$).

Figure 5*C* shows that the rate at which the soleus was voluntarily activated during MVC increased with training. We observed a 48.7 ± 24.3% ($P < 0.001$) increase in the activation rate of the soleus for the trained group (*day 1*, 0.086 ± 0.040 $mV \cdot s^{-1}$; *day 13*, 0.128 ± 0.031 $mV \cdot s^{-1}$). By *day 3*, the rate had become significant ($P < 0.002$), and it remained as such until the completion of the protocol.

Contractile properties. Maximal evoked twitch torque increased 36.2 ± 24.8% ($P < 0.003$) following training. This increase had become significant by *day 5* ($P < 0.004$). Both the twitch $+dF/dt_{max}$ and rate of twitch torque relaxation ($-dF/dt_{max}$) increased significantly. Twitch $+dF/dt_{max}$ increased significantly by *day 5* ($P < 0.005$), and by the end of training there was a 33.1 ± 26.2% ($P < 0.006$) increase. Twitch $-dF/dt_{max}$ also increased significantly by *day 5* ($P < 0.002$) and exhibited an increase of 33.1 ± 31.4% ($P < 0.002$) overall.

H reflex analysis. Using the H_{slp}/M_{slp} method for analysis of recruitment curves, we observed no significant changes in spinal excitability for either the trained group or the control group, at rest or when recruitment curves were produced during muscle activation (10% MVC; Table 1). There were

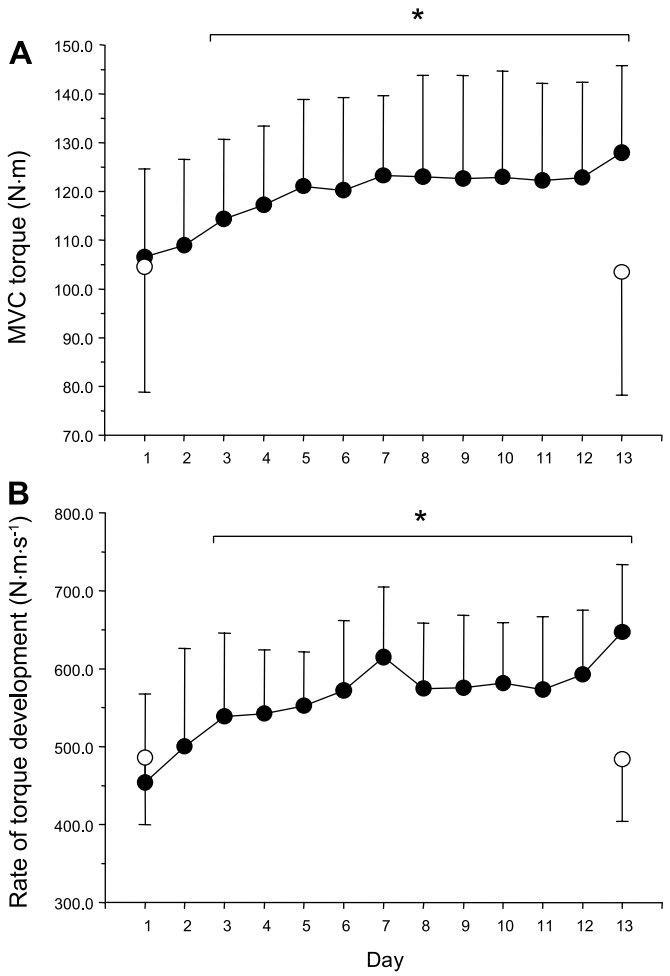

Fig. 4. MVC torque and rate of torque development. Mean ± SD. Trained group (●), control group (○). Trained group MVC torque (*A*) and the rate of torque development (*B*) increased 20.0 ± 13.9% and 42.5 ± 13.3% respectively, after 4 wk. * denotes a value significantly different from *day 1* ($P < 0.008$).

also no differences in spinal excitability between the rest and 10% MVC conditions in either group at any point in the investigation.

V wave analysis. Peak-to-peak V wave amplitude normalized to the maximal M wave increased significantly in all 10 trained subjects at each contraction intensity (Fig. 6). At 100% MVC, V/M_{max} increased 57.3 ± 34.2% from pretest (0.40 ± 0.18) to posttest-V1 (0.62 ± 0.21). V/M_{max} on *day 7* (0.56 ± 0.22) was also different from baseline.

In posttest-V2, subjects completed the V wave protocol using the absolute MVC torque value from the pretest to calculate contraction intensities. For the trained group, posttest-V2 V/M_{max} differed significantly from posttest-V1 at 50, 75, and 100% MVC (Fig. 6). The voluntary activation associated with each contraction intensity was also significantly different (i.e., 50, 75, and 100% MVC). Compared with the pretest, posttest-V2 V/M_{max} and voluntary activation values were similar at each contraction intensity. In other words, after a training-induced increase in MVC torque, there was no change in the required neural drive or activation for the same absolute torque production.

DISCUSSION

There have been few attempts to determine the sequence and time-course of adaptations that occur in the human central nervous system (CNS) in response to resistance training and to link these adaptations to augmented muscle performance (2, 3, 20). Moreover, there is considerable variability within the

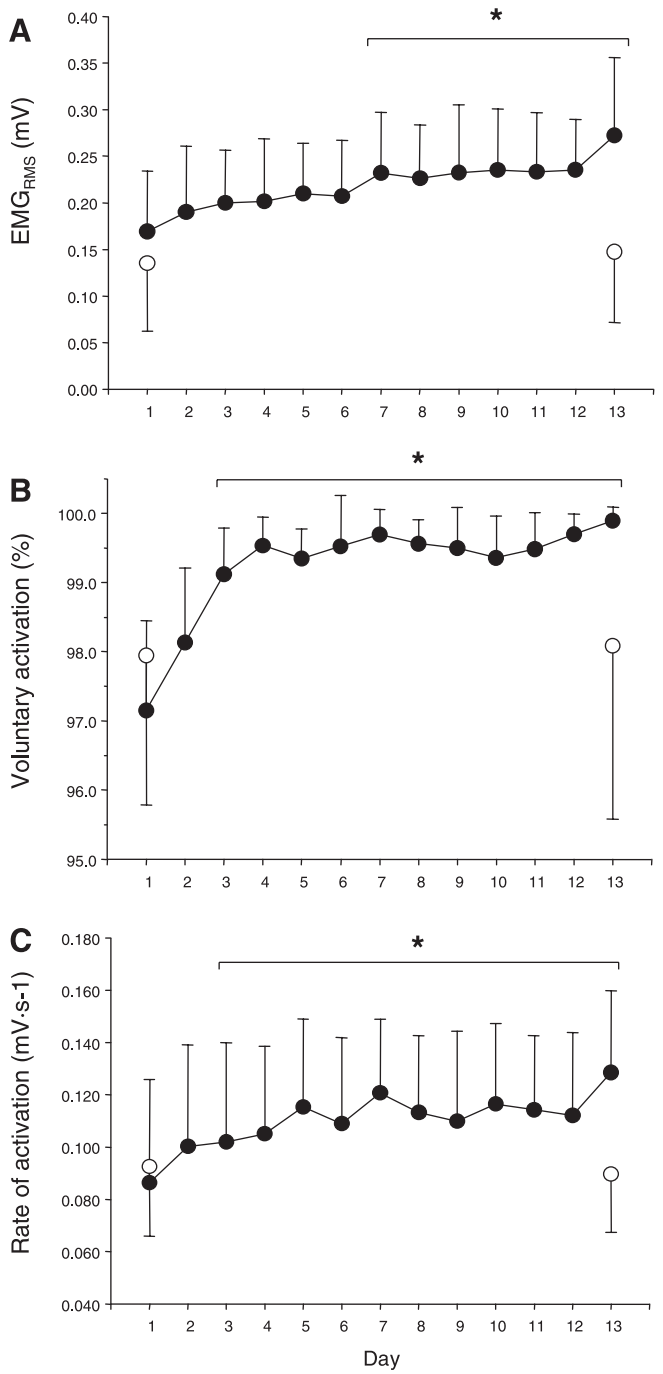

Fig. 5. Soleus surface EMG, voluntary activation, and rate of activation. Mean ± SD. Trained group (●), control group (○). The respective training-induced increases of 60.8 ± 30.8% and 48.8 ± 24.3% in root mean square-derived surface EMG (EMG_{RMS}; *A*) and rate of activation of soleus (*C*) are shown above. The 2.8 ± 0.074% increase in voluntary activation (*B*) was calculated via twitch interpolation, [1-(superimposed twitch/maximal twitch)] × 100. * denotes a value significantly different from *day 1* ($P < 0.008$).

Table 1. *H reflex analysis*

	H_{slp}/M_{slp}		
	Pretest	*Day 7*	Posttest
Training group			
rest	1.77±1.08	1.64±0.81	1.86±0.96
10% MVC	1.55±0.51	1.48±0.74	1.49±0.77
Control group			
rest	1.87±0.79	NA	1.62±0.58
10% MVC	1.27±0.44	NA	1.28±0.53

All values are mean ± SD. A ratio of the developmental slopes of the H reflex and M wave recruitment curves (H_{slp}/M_{slp}) was taken as an index of spinal excitability. No significant changes were observed for either trained or control group at rest or during muscle activation (10% MVC). Nor were there any differences in spinal excitability between the rest and 10% MVC conditions in either group at any point in the investigation.

literature that does exist. For example, Aagaard and colleagues (2, 3) utilized a 14-wk, *isotonic* training protocol which involved a number of exercises to strengthen the entire lower limb. Their experimental measures were made using *isometric* contractions of the knee extensors (2) and the plantar flexors (3) at the onset and upon completion of the 14-wk protocol, but not during. With this training protocol there would likely have been significant muscle hypertrophy to which the CNS would have had to adapt. The experimental design did not permit them to compare adaptations in V wave and H reflex responses with changes in activation and rate of torque development *in the same muscle*, nor were they able to provide insight into very early adaptations in the likely absence of hypertrophy. Gondin et al. (20) used a protocol similar to the one in the present experiment. However, training consisted of stimulating the triceps surae electrically, thereby eliminating the effects of asynchronous voluntary activation. Again, experimental measures were only made prior to and after five weeks of electromyostimulation training. This did not allow for daily assessment of adaptations within the CNS, as are made in the present investigation. The most important aspect of the present work is that it suggests a linked chain of events that occur almost immediately in response to isometric resistance training. In this very short time period, it is unlikely that any muscle hypertrophy occurred. In addition, we suggest that the series of adaptations observed here may be related to changes in cortical excitability following a similar isometric training protocol, as shown in a recent publication from our laboratory (23).

The adaptive response of the neuromuscular system to the progressive isometric resistance training protocol was rapid and resulted in a significant increase in MVC torque after only two training sessions and a 20% increase after four weeks. This increase is comparable to other studies that use similar training protocols (9, 23, 42). An increase in MVC of this magnitude may occur via changes in muscle fiber size or architecture (2), but it is unlikely that changes in muscle morphology are responsible for the MVC increase after only two training days.

Increases in MVC in the first 2–3 wk of training are not associated with an increase in either muscle CSA (4) or increased myofibrillar density (12). While it can be argued that protein synthesis rates are elevated after just a single bout of resistance exercise (19, 33), soleus muscle protein synthesis after a single bout of resistance exercise was shown to be a third of that which has been consistently reported for vastus lateralis (51). In addition, rates of protein degradation are also elevated after a single bout of resistance exercise (7, 41). Further, even if a positive net protein balance is achieved after a single bout of resistance exercise (40), whether or not this contributes to the immediate accretion of contractile proteins has yet to be determined.

In our investigation, the time-course of increases in both the rate of electrical activation of the soleus muscle and the maximal rate of voluntary torque development were similar to that of MVC. After just two training sessions in which the soleus had been active a total of ~7 minutes, significant increases in all three measures were observed. An increase in the rate of electrical activation of soleus cannot be assumed to translate directly into an increase in MVC torque; however, our data suggest that there is a relationship between the two. If, at the beginning of contraction, the rate at which the soleus is electrically activated is increased, it is likely that there would be an increased rate of torque production. Figure 7*A* shows the rate of torque development as a function of the rate of electrical activation of soleus; the two are highly correlated ($r = 0.95$, $P < 0.001$). It is also likely that an increased rate of torque development serves to increase MVC (2). MVC torque as a function of torque development is illustrated in Fig. 7*B*. Again, the correlation is high ($r = 0.95$, $P < 0.001$).

Parallel increases in the rate of torque development and EMG amplitude have previously been observed after resistance training (2, 53). Accompanying an increase in rate of torque development, Aagaard et al. (2) observed a marked increase in both EMG amplitude and rate of rise of the integrated EMG in the initial contraction phase. These findings are replicated in the present data set. A 60% increase in the surface EMG as well as a 49% increase in the rate of electrical activation of soleus was observed over four weeks. Thus modulation of volitional drive could be the cause of the 43% increase in the

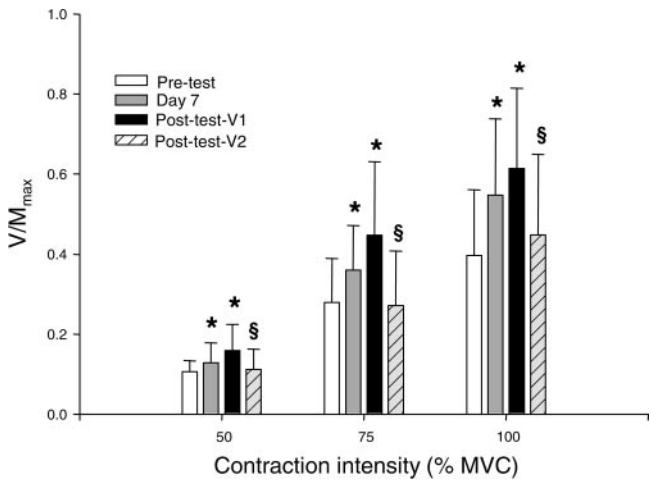

Fig. 6. V wave amplitude (trained group only). Mean ± SD. V wave amplitude normalized to the maximal M wave (V/M_{max}) is plotted as a function of contraction intensity. V/M_{max} increased significantly at each contraction intensity from pretest (*day 1*) to posttest-V1 (*day 13*), notably increasing 57.3 ± 34.2% at 100% MVC. The absolute MVC torque value from the pretest was used to calculate contraction intensities in posttest-V2. Posttest-V2 V/M_{max} differed significantly from posttest-V1; however, it was not different from the pretest value. * denotes a value significantly different from the pretest value ($P < 0.02$). § denotes a value significantly different from posttest-V1 ($P < 0.001$).

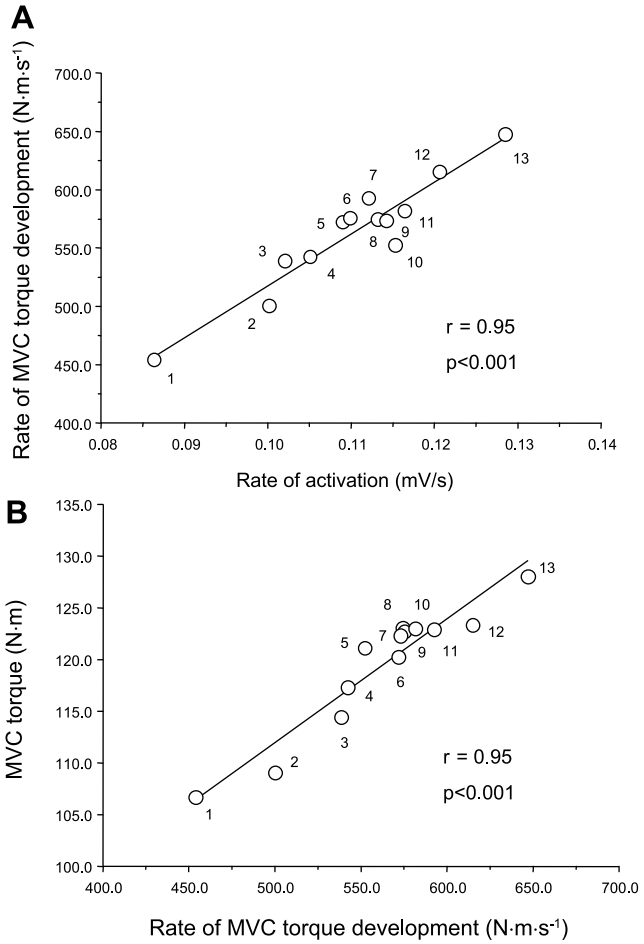

Fig. 7. Rate of torque development, rate of activation, and MVC torque (trained group only). Numbered data points (○) indicate the training day on which measures were taken. Linear regression (solid line) reveals a significant correlation between the averaged increases in the rate of torque development and the rate of activation of soleus (*A*). Linear regression also reveals a significant correlation between the averaged increases in MVC torque and rate of torque development (*B*).

rate of voluntary torque development observed here. Specifically, alteration of motor unit (MU) recruitment patterns and/or changes in the rate coding of motor units may be responsible. Increases in the soleus surface EMG and rate of activation indicate that either one or both of these neural strategies has been employed.

The production of MVC torque is contingent upon the extent of MU recruitment and MU firing rates. Changes in recruitment and discharge rates have been observed following dynamic resistance training. MU recruitment thresholds for first dorsal interosseous (29) and tibialis anterior (53) muscles were observed to decrease following training. Also following resistance training, synchronization (i.e., the simultaneous activation of numerous MUs) was estimated based on surface EMG and observed to increase in human thenar muscles (36). Likewise, MU synchronization was found to be greater in strength-trained subjects versus controls in a protocol that measured MU discharges directly (47). Van Cutsem et al. (53) reported an increase in rate of torque production, together with a six-fold increase in the incidence of discharge 'doublets' in the firing pattern of individual MUs following resistance training. Doublets (interspike interval <10 ms) have been observed in the firing pattern of single motor neurons at the onset of rapid voluntary contraction and during reflex contraction (18). As previously suggested (1), the firing of doublets at the onset of contraction may serve to enhance the initial generation of torque by taking advantage of the catch-like property of skeletal muscle (8), which would increase the rate of torque development. Together, an alteration of MU recruitment patterns and the elevated incidence of doublets at the onset of contraction represent feasible mechanisms for the increases in the rate of torque development and MVC observed here.

An increase in V/M_{max} may result from enhanced drive in descending corticospinal pathways, elevated motor neuron excitability due to peripheral inputs, or alterations in presynaptic inhibition (3). In other words, V wave amplitude is affected by changes which occur at both spinal and supraspinal levels. For this reason, it is necessary to assess changes in both evoked reflex responses (i.e., H reflex and V wave) to allow the determination of sites mediating neural adaptations in response to resistance training.

To assess changes in spinal excitability, H reflex and M wave recruitment curves were analyzed by means of the H_{slp}/M_{slp} method, which provides an estimate of "reflex gain" (15). Normalizing the H_{slp} to the M_{slp} provides a measure of excitability that represents a significant portion of the α-motor neuron pool and minimizes the effects of postural change, electrode placement, skin resistance, and the effect of collision (15). Despite this, the present investigation showed no significant changes in spinal excitability in the trained group at rest or during voluntary activation of the soleus.

To date, longitudinal investigations of training-induced alterations in spinal excitability have shown inconsistent results. No changes in maximal H reflex amplitude normalized to the maximal M wave amplitude (H_{max}/M_{max}) at rest (3, 20, 35) have been observed. During MVC, Gondin et al. (20) observed no change, while Aagaard et al. (3) observed a 20% increase in H_{max}/M_{max}. Lagerquist et al. (31) recently demonstrated no change in H_{max}/M_{max} obtained with a 10% EMG background contraction. The inconsistency is likely the result of different training protocols and/or methodological limitations. Still, a lack of change in the H reflex response observed here suggests that elements of the H reflex pathway may not be directly involved in chronic adjustments in response to isometric resistance training.

V wave amplitude in the present study significantly increased over four weeks of resistance training. V/M_{max} increased at all contraction intensities, notably increasing 57% during MVC. Previously, in response to 14 wk of dynamic resistance training, similar increases in soleus V wave amplitude (~50%) were observed to accompany 22–30% increases in MVC (3). Following five weeks of electrical stimulation training, an 81% increase in soleus V wave amplitude accompanied a 22% increase in MVC torque (20). With no change in spinal excitability, the increased V wave amplitude observed here is the result of increased collision in α-motor neuron axons between electrically evoked antidromic action potentials and orthodromic potentials from voluntary contraction. Enhanced volitional drive increases MU recruitment and augments MU firing rate, thus increasing efferent action potentials in α-motor neuron axons. Therefore, any increase in descending volitional drive will produce an increased cancellation of the antidromic action potentials, allowing more of the electri-

cally evoked reflex volley of action potentials to reach the muscle, resulting in an increased V wave amplitude (3, 52).

A rapid change in the activation of soleus was induced by the training. At present, twitch interpolation is the best method available for measuring voluntary activation in whole muscle. Maximal voluntary activation assessed with this technique yielded an increase of about 3% after four weeks. Compared with the 20% increase in MVC torque, the change in voluntary activation of soleus approximated based on twitch interpolation appears inadequate as an explanation for the relatively large increase in MVC (see also Ref. 20). The small change in voluntary activation may be attributable to the near-maximal estimation of activation at the onset of the training protocol. In addition, the twitch interpolation technique may be limited in its assessment of voluntary activation because the relationship between contraction intensity and superimposed twitch amplitude is non-linear, particularly at near-maximal torques (6, 14). It has been suggested that in the soleus, this nonlinearity may be a result of the proximity of the common peroneal nerve to the tibial nerve in the popliteal fossa, leading to antagonist activation from stimulus spread (50). The twitch interpolation technique may therefore best be interpreted as an indicator of change in the activation of muscle; however, it may not necessarily provide a proportional increase compared with MVC.

The possibility of a change in muscle morphology after four weeks of isometric resistance training cannot be excluded, since 36 and 33% increases in twitch amplitude and twitch torque development, respectively, were observed over the duration of the protocol. Increases such as these are often attributed to changes in the force-generating capacity of the contractile component, namely, muscle fiber hypertrophy. Alternatively, the way force is transmitted within as well as from muscle (i.e., via the series elastic component) could account for these changes independent of any change in the contractile elements. The rate of torque development depends both on the stiffness of the series elastic component and on the torque-velocity characteristics of the contractile component (26). Increases in tendon stiffness and Young's modulus of human tendon structures have been shown to accompany an increase in MVC following 12 wk of isometric resistance training (30). Due to a pre-post experimental design, delineation of the time-course of changes in tendon structures is not possible based on the aforementioned results. Even so, stiffer tendon structures are suitable for transmitting the force to tendon more effectively (11) and would result in an improved rate of twitch torque development (54), as was observed in the present investigation.

There is evidence that the contractile history of skeletal muscle may facilitate the production of torque (45). Postactivation potentiation (PAP) represents another possible explanation for the increased twitch amplitude observed here. PAP alters excitation-contraction coupling through phosphorylation of myosin regulatory light chains (25), which makes actin and myosin more sensitive to Ca^{2+} (49). In addition to increasing maximal twitch torque, preceding contractile activity (i.e., MVC) has been shown to increase rate of twitch torque development (45). Over the course of the isometric protocol, as MVC increased, the evoked maximal twitch that followed MVC trials is likely to have been subject to a greater deal of potentiation. It follows that greater PAP would function to increase the rate of twitch torque development (22) as well as increase twitch torque (21, 24).

Our isometric resistance training protocol induced a significant increase in MVC torque after just two training sessions. This was accompanied by increases in the rate of torque development and the rate of activation of soleus. While no apparent changes in the elements of the H reflex pathway took place following resistance training, the increase in V wave amplitude is indicative of an enhanced descending volitional drive to the muscle (51). Evidenced additionally by the increase in the surface EMG, it is this change in efferent neural drive that likely produced increases in both our measures of voluntary activation and the rate of electrical activation. Collaboratively, the results from evoked spinal reflex responses indicate augmented drive from supraspinal centers. Future investigation of adaptive alterations in neural drive following resistance training should include reliable measures of both evoked spinal reflexes, as only in conjunction may sites mediating neural adaptations be determined. Earlier recruitment of large MUs, increased synchrony, elevated MU firing rates, and/or an increased incidence of doublets at the onset of contraction are the mechanisms likely responsible for the increases in the rate of torque development, and subsequently MVC torque (53). In summary, we have attempted to establish a pathway of training-induced changes from the motor cortex to the muscle which explains the increase observed in MVC (Fig. 8).

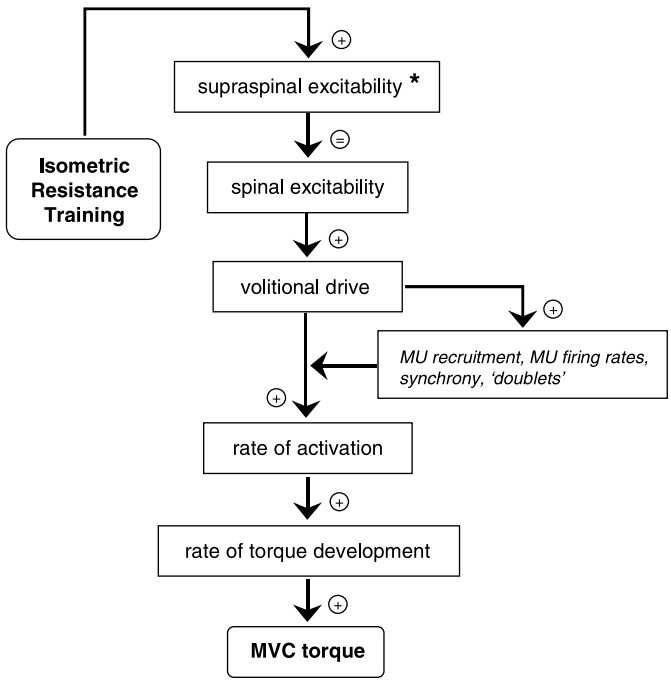

Fig. 8. Summary of adaptations. The results of the present investigation suggest resistance training-induced neural adaptations occur at multiple sites along the pathway from the motor cortex to muscle. No change in "spinal excitability" was observed from H_{slp}/M_{slp} analysis. The increase in "volitional drive" was observed through analysis of V/M_{max}. The increase in "rate of activation" was derived from the surface EMG. The increase in the "rate of torque development" was derived from the torque recording and observed during MVC trials. Italicized mechanisms were not directly measured. * denotes data from Ref. 22 were collected from tibialis anterior. See DISCUSSION for further explanation.

Our data suggest the increase in MVC torque may be attributed to an increased rate of activation, secondary to an increased descending volitional drive following isometric resistance training. The novelty of the present investigation is that it describes a chain of events that may account for the very early adaptations to isometric resistance training. Using transcranial magnetic stimulation (TMS), we have recently reported increased cortical excitability following the same training protocol in tibialis anterior (23). This suggests that the very early changes in the present study may reflect changes occurring supraspinally. However, because there are some differences between soleus and tibialis anterior with respect to strength of corticomotoneuronal connections and responses to TMS (5), this association must be carefully interpreted. Nevertheless, our V wave data suggest that this increased supraspinal excitability serves to increase descending volitional drive to the muscle. Consequently, the muscle is activated more rapidly and generates torque more rapidly. This increased rate of torque development is highly correlated with the increase in MVC observed following isometric resistance training. Still, it is likely there are numerous changes within the nervous system occurring simultaneously, which only when taken together result in the adaptations commonly observed in the first few weeks of resistance training.

ACKNOWLEDGMENTS

The authors thank Dr. Lisa Griffin of the University of Texas at Austin for assistance in preparation of the manuscript.

GRANTS

This study was supported by National Sciences and Engineering Research Council Grant A-6655 to E. Cafarelli.

REFERENCES

1. **Aagaard P.** Training-induced changes in neural function. *Exerc Sport Sci Rev* 31: 61–67, 2003.
2. **Aagaard P, Simonsen EB, Andersen JL, Magnusson P, Dyhre-Poulsen P.** Increased rate of force development and neural drive of human skeletal muscle following resistance training. *J Appl Physiol* 93: 1318–1326, 2002.
3. **Aagaard P, Simonsen EB, Andersen JL, Magnusson P, Dyhre-Poulsen P.** Neural adaptation to resistance training: changes in evoked V-wave and H-reflex responses. *J Appl Physiol* 92: 2309–2318, 2002.
4. **Akima H, Takahashi H, Kuno SY, Masuda K, Masuda T, Shimojo H, Anno I, Itai Y, Katsuta S.** Early phase adaptations of muscle use and strength to isokinetic training. *Med Sci Sports Exerc* 31: 588–594, 1999.
5. **Bawa P, Chalmers GR, Stewart H, Eisen AA.** Responses of ankle extensor and flexor motoneurons to transcranial magnetic stimulation. *J Neurophysiol* 88: 124–132, 2002.
6. **Belanger AY, McComas AJ.** Extent of motor unit activation during effort. *J Appl Physiol* 51: 1131–1135, 1981.
7. **Biolo G, Maggi SP, Williams BD, Tipton KD, Wolfe RR.** Increased rates of muscle protein turnover and amino acid transport after resistance exercise in humans. *Am J Physiol Endocrinol Metab* 268: E514–E520, 1995.
8. **Burke RE, Rudomin P, Zajac FE 3rd.** Catch property in single mammalian motor units. *Science* 168: 122–124, 1970.
9. **Cannon RJ, Cafarelli E.** Neuromuscular adaptations to training. *J Appl Physiol* 63: 2396–2402, 1987.
10. **Casabona A, Polizzi MC, Perciavalle V.** Differences in H-reflex between athletes trained for explosive contractions and non-trained subjects. *Eur J Appl Physiol Occup Physiol* 61: 26–32, 1990.
11. **Cavagna GA, Citterio G, Jacini P.** Effects of speed and extent of stretching on the elastic properties of active frog muscle. *J Exp Biol* 90: 131–143, 1981.
12. **Claassen H, Gerber C, Hoppeler H, Luthi JM, Vock P.** Muscle filament spacing and short-term heavy-resistance exercise in humans. *J Physiol* 409: 491–495, 1989.
13. **Cresswell AG, Loscher WN, Thorstensson A.** Influence of gastrocnemius muscle length on triceps surae torque development and electromyographic activity in man. *Exp Brain Res* 105: 283–290, 1995.
14. **Dowling JJ, Konert E, Ljucovic P, Andrews DM.** Are humans able to voluntarily elicit maximum muscle force? *Neurosci Lett* 179: 25–28, 1994.
15. **Funase K, Imanaka K, Nishihira Y.** Excitability of the soleus motoneuron pool revealed by the developmental slope of the H-reflex as reflex gain. *Electromyogr Clin Neurophysiol* 34: 477–489, 1994.
16. **Gandevia SC.** Spinal and supraspinal factors in human muscle fatigue. *Physiol Rev* 81: 1725–1789, 2001.
17. **Garfinkel S, Cafarelli E.** Relative changes in maximal force, EMG, and muscle cross-sectional area after isometric training. *Med Sci Sports Exerc* 24: 1220–1227, 1992.
18. **Garland SJ, Griffin L.** Motor unit double discharges: statistical anomaly or functional entity? *Can J Appl Physiol* 24: 113–130, 1999.
19. **Gibala MJ, MacDougall JD, Tarnopolsky MA, Stauber WT, Elorriaga A.** Changes in human skeletal muscle ultrastructure and force production after acute resistance exercise. *J Appl Physiol* 78: 702–708, 1995.
20. **Gondin J, Duclay J, Martin A.** Soleus- and gastrocnemii-evoked V-wave responses increase after neuromuscular electrical stimulation training. *J Neurophysiol* 95: 3328–3335, 2006.
21. **Gossen ER, Sale DG.** Effect of postactivation potentiation on dynamic knee extension performance. *Eur J Appl Physiol* 83: 524–530, 2000.
22. **Grange RW, Vandenboom R, Houston ME.** Physiological significance of myosin phosphorylation in skeletal muscle. *Can J Appl Physiol* 18: 229–242, 1993.
23. **Griffin L, Cafarelli E.** Transcranial magnetic stimulation during resistance training of the tibialis anterior muscle. *J Electromyogr Kinesiol* 2006. In press. Published online 4 August 2006; doi:10.1016/j.jelekin.2006.05.001.
24. **Hamada T, Sale DG, MacDougall JD, Tarnopolsky MA.** Postactivation potentiation, fiber type, and twitch contraction time in human knee extensor muscles. *J Appl Physiol* 88: 2131–2137, 2000.
25. **Hodgson M, Docherty D, Robbins D.** Post-activation potentiation: underlying physiology and implications for motor performance. *Sports Med* 35: 585–595, 2005.
26. **Jewell B, Wilkie D.** An analysis of the mechanical components in frog's striated muscle. *J Physiol* 143: 515–540, 1958.
27. **Kalmar JM, Del Balso C, Cafarelli E.** Increased spinal excitability does not offset central activation failure. *Exp Brain Res* 173: 446–457, 2006.
28. **Kawakami Y, Ichinose Y, Fukunaga T.** Architectural and functional features of human triceps surae muscles during contraction. *J Appl Physiol* 85: 398–404, 1998.
29. **Keen DA, Yue GH, Enoka RM.** Training-related enhancement in the control of motor output in elderly humans. *J Appl Physiol* 77: 2648–2658, 1994.
30. **Kubo K, Kanehisa H, Ito M, Fukunaga T.** Effects of isometric training on the elasticity of human tendon structures in vivo. *J Appl Physiol* 91: 26–32, 2001.
31. **Lagerquist O, Zehr EP, Docherty D.** Increased spinal reflex excitability is not associated with neural plasticity underlying the cross-education effect. *J Appl Physiol* 100: 83–90, 2006.
32. **Levenez M, Kotzamanidis C, Carpentier A, Duchateau J.** Spinal reflexes and coactivation of ankle muscles during a submaximal fatiguing contraction. *J Appl Physiol* 99: 1182–1188, 2005.
33. **MacDougall JD, Gibala MJ, Tarnopolsky MA, MacDonald JR, Interisano SA, Yarasheski KE.** The time course for elevated muscle protein synthesis following heavy resistance exercise. *Can J Appl Physiol* 20: 480–486, 1995.
34. **Maffiuletti NA, Martin A, Babault N, Pensini M, Lucas B, Schieppati M.** Electrical and mechanical H_{max}-to-M_{max} ratio in power- and endurance-trained athletes. *J Appl Physiol* 90: 3–9, 2001.
35. **Maffiuletti NA, Pensini M, Scaglioni G, Ferri A, Ballay Y, Martin A.** Effect of electromyostimulation training on soleus and gastrocnemii H- and T-reflex properties. *Eur J Appl Physiol* 90: 601–607, 2003.
36. **Milner-Brown HS, Stein RB, Lee RG.** Synchronization of human motor units: possible roles of exercise and supraspinal reflexes. *Electroencephalogr Clin Neurophysiol* 38: 245–254, 1975.
37. **Moritani T, deVries HA.** Neural factors versus hypertrophy in the time course of muscle strength gain. *Am J Phys Med* 58: 115–130, 1979.
38. **Narici MV, Hoppeler H, Kayser B, Landoni L, Claassen H, Gavardi C, Conti M, Cerretelli P.** Human quadriceps cross-sectional area, torque and neural activation during 6 months strength training. *Acta Physiol Scand* 157: 175–186, 1996.

39. **Narici MV, Roi GS, Landoni L, Minetti AE, Cerretelli P.** Changes in force, cross-sectional area and neural activation during strength training and detraining of the human quadriceps. *Eur J Appl Physiol Occup Physiol* 59: 310–319, 1989.
40. **Phillips SM, Parise G, Roy BD, Tipton KD, Wolfe RR, Tamopolsky MA.** Resistance-training-induced adaptations in skeletal muscle protein turnover in the fed state. *Can J Physiol Pharmacol* 80: 1045–1053, 2002.
41. **Phillips SM, Tipton KD, Aarsland A, Wolf SE, Wolfe RR.** Mixed muscle protein synthesis and breakdown after resistance exercise in humans. *Am J Physiol Endocrinol Metab* 273: E99–E107, 1997.
42. **Pucci AR, Griffin L, Cafarelli E.** Maximal motor unit firing rates during isometric resistance training in men. *Exp Physiol* 91: 171–178, 2006.
43. **Rich C, Cafarelli E.** Submaximal motor unit firing rates after 8 wk of isometric resistance training. *Med Sci Sports Exerc* 32: 190–196, 2000.
44. **Rochcongar P, Dassonville J, Le Bars R.** [Modification of the Hoffmann reflex in function of athletic training (author's transl)]. *Eur J Appl Physiol Occup Physiol* 40: 165–170, 1979.
45. **Sale DG.** Postactivation potentiation: role in human performance. *Exerc Sport Sci Rev* 30: 138–143, 2002.
46. **Sale DG, MacDougall JD, Upton AR, McComas AJ.** Effect of strength training upon motoneuron excitability in man. *Med Sci Sports Exerc* 15: 57–62, 1983.
47. **Semmler JG, Nordstrom MA.** Motor unit discharge and force tremor in skill- and strength-trained individuals. *Exp Brain Res* 119: 27–38, 1998.
48. **Stanley EF.** Reflexes evoked in human thenar muscles during voluntary activity and their conduction pathways. *J Neurol Neurosurg Psychiatry* 41: 1016–1023, 1978.
49. **Sweeney HL, Bowman BF, Stull JT.** Myosin light chain phosphorylation in vertebrate striated muscle: regulation and function. *Am J Physiol Cell Physiol* 264: C1085–C1095, 1993.
50. **Todd G, Gorman RB, Gandevia SC.** Measurement and reproducibility of strength and voluntary activation of lower-limb muscles. *Muscle Nerve* 29: 834–842, 2004.
51. **Trappe TA, Raue U, Tesch PA.** Human soleus muscle protein synthesis following resistance exercise. *Acta Physiol Scand* 182: 189–196, 2004.
52. **Upton AR, McComas AJ, Sica RE.** Potentiation of "late" responses evoked in muscles during effort. *J Neurol Neurosurg Psychiatry* 34: 699–711, 1971.
53. **Van Cutsem M, Duchateau J, Hainaut K.** Changes in single motor unit behaviour contribute to the increase in contraction speed after dynamic training in humans. *J Physiol* 513 (Pt 1): 295–305, 1998.
54. **Wilson GJ, Murphy AJ, Pryor JF.** Musculotendinous stiffness: its relationship to eccentric, isometric, and concentric performance. *J Appl Physiol* 76: 2714–2719, 1994.
55. **Zehr PE.** Considerations for use of the Hoffmann reflex in exercise studies. *Eur J Appl Physiol* 86: 455–468, 2002.