

Neural inhibition during maximal eccentric and concentric quadriceps contraction: effects of resistance training

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Aagaard, P., E. B. Simonsen, J. L. Andersen, S. P. Magnusson, J. Halkjær-Kristensen, and P. Dyhre-Poulsen. Neural inhibition during maximal eccentric and concentric quadriceps contraction: effects of resistance training. *J Appl Physiol* 89: 2249–2257, 2000.—Despite full voluntary effort, neuromuscular activation of the quadriceps femoris muscle appears inhibited during slow concentric and eccentric contractions. Our aim was to compare neuromuscular activation during maximal voluntary concentric and eccentric quadriceps contractions, hypothesizing that inhibition of neuromuscular activation diminishes with resistance training. In 15 men, pretraining electromyographic activity of the quadriceps muscles [vastus medialis (VM), vastus lateralis (VL), and rectus femoris (RF)] was 17–36% lower during slow and fast (30 and 240°/s) eccentric and slow concentric contractions compared with fast concentric contractions. After 14 wk of heavy resistance training, neuromuscular inhibition was reduced for VL and VM and was completely removed for RF. Concurrently, electromyographic activity increased 21–52, 22–29, and 16–32% for VL, VM, and RF, respectively. In addition, median power frequency decreased for VL and RF. Eccentric quadriceps strength increased 15–17%, whereas slow and fast concentric strength increased 15 and 8%, respectively. Pre- and posttraining median power frequency did not differ between eccentric and concentric contractions. In conclusion, quadriceps motoneuron activation was lower during maximal voluntary eccentric and slow concentric contractions compared with during fast concentric contraction in untrained subjects, and, after heavy resistance training, this inhibition in neuromuscular activation was reduced.

neuromuscular activation; muscle strength; neural efferent drive; eccentric activation deficiency; force inhibition

IN TERMS OF CONTRACTILE CHARACTERISTICS, the force- or moment-velocity relationship that is measured in vivo during maximal voluntary activation of the quadriceps femoris muscle (3, 45) deviates markedly from that obtained from isolated, in vitro muscle preparations (28), especially when comparing eccentric contractions. In addition, a marked “plateauing” of angle-specific

concentric quadriceps moments is observed at low angular velocities (2, 3, 10, 47). It has been speculated that the plateauing phenomenon may be due to a force-inhibiting neural mechanism that reduces the voluntary neural drive to the quadriceps muscle during maximal voluntary contractions (10, 47). Interestingly, superimposition of electrical stimulation onto maximal voluntary quadriceps contraction yields an increase in eccentric, but not concentric, moment of force (4, 46), indicating that unique neuromuscular activation patterns are present during maximal eccentric quadriceps contraction. Furthermore, this enhancement of eccentric strength was observed in sedentary subjects but was absent in strength athletes (4), suggesting that the underlying mechanisms may be modulated by training. Indirect evidence of a tension-limiting mechanism was presented in a recent study in which maximal eccentric quadriceps moment was significantly lower than that predicted (150% of maximal isometric moment) from extrapolation of the stretch-induced increment in moment observed at different intensities of voluntary effort (43). Thus the existence of a neural regulatory mechanism that limits the recruitment and/or discharge of motor units during maximal voluntary eccentric quadriceps contraction was proposed (43).

More direct evidence suggests that neuromuscular activation of the quadriceps muscle is reduced with certain types of high-tension muscle loading. Electromyographic (EMG) activity is markedly lower during maximal voluntary eccentric and slow concentric quadriceps contraction compared with that of fast concentric contraction (38, 44). The exact mechanisms responsible for this inhibition in neuromuscular activation are unknown. Although evidence of a preferential recruitment of type II motor units and derecruitment of type I units has been demonstrated for submaximal eccentric muscle contraction in humans (25, 31), this may not hold true for maximal eccentric quadriceps contraction (42).

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Motoneuron activation is modulated by both inhibitory Ib afferent feedback from Golgi organs and excitatory Ia and group II afferent feedback from muscle spindles (17). In human muscle, changes in evoked H-reflex and V-wave responses suggest that the net influence from these reflex pathways and the influence of central descending pathways may be altered in response to resistance training (1, 36), which causes maximal muscle contraction strength to change accordingly (1). Regardless of the specific mechanisms involved, neuromuscular activation is expected to increase after heavy resistance strength training (35). However, it is not well understood whether, and to what extent, the reduction in neuromuscular quadriceps activation during eccentric and slow concentric contraction changes with training. Therefore, the aim of the present study was to compare neural activation during maximal concentric vs. eccentric quadriceps contraction before and after a period of heavy-resistance strength training. It was hypothesized that the regime of heavy resistance training would result in diminished motoneuron inhibition and enhanced neuromuscular activation.

METHODS

Subjects. Fifteen men volunteered to participate in the study (body mass 73.2 ± 5.8 kg, height 179 ± 4 cm, age 23.5 ± 3.4 yrs, means \pm SD). All subjects gave their informed consent to the procedures of the study. None of the subjects had any history of previous knee or muscle injury or had previously participated in systematic strength training. The conditions of the study were approved by the local ethics committee.

Resistance training. The training consisted of 14 wk of progressive, heavy-resistance strength training (38 training sessions). All training was surveyed and supervised by the authors of the study. Training loads ranged between 6–10 repetition maximum (RM), except for the first 10 days (5 sessions), in which slightly lower loadings were used (10–15 RM). Very heavy loadings (6–8 RM, increased number of sets) were used in the final 4 wk of the study. Obligatory training exercises were 1) hack squat, 2) incline leg press, 3) isolated knee extension, 4) hamstring curls, and 5) calf raises. For the hack squat, 4 sets were performed each session, whereas all other exercises were performed in 4 sets in weeks 1–10, followed by 5 sets in the final 4 wks.

Isokinetic strength measurements. Maximal concentric and eccentric quadriceps muscle strength was measured as maximal voluntary knee extension moments exerted in an isokinetic dynamometer (Kinetic Communicator, Chattecx, Chattanooga, TN). The reliability and validity of this dynamometer are described elsewhere (15). Subjects were seated in a rigid chair and firmly strapped at the hip and distal thigh. The rotational axis of the dynamometer was aligned to the lateral femoral epicondyle, and the lower leg was attached to the dynamometer lever arm above the medial malleolus, with no static fixation of the ankle joint. Within each subject, identical positioning of the seat, backrest, dynamometer head, and lever arm length were used for pre- and posttraining. All measurements were performed using the right leg. Measurements were preceded by 10 min of warm-up, followed by a number of submaximal preconditioning trials in the dynamometer. Subjects were familiarized

with the dynamometer and the procedures of the experiments on separate occasions.

Maximal concentric and eccentric quadriceps contractions were performed during slow and fast knee extension (knee joint angular velocities 30 and 240°/s, respectively). Range of knee joint excursion was 10 to 90° (0° = knee fully extended). The order of velocities was random. On-line visual feedback of the dynamometer forces was given to the subject on a computer screen, and successive trials were performed, separated by a rest period of at least 30 s, until peak moment could not be improved any further. This procedure ensured that the voluntary muscle moments were truly maximal. Typically, 6–7 trials were performed at each velocity and contraction mode to fulfill this goal. All recorded moment signals were corrected for the effect of gravity (2). Total work and average moment were determined for each trial.

EMG measurements. EMG signals were obtained from medial vastus lateralis (VL), medial vastus medialis (VM) and medial rectus femoris (RF) muscles by using bipolar surface electrodes (Medicotest Q-10-A) with a 1.8-cm inter-electrode distance and a recording area of 330 mm². After careful preparation of the skin (shaving, abrasion, and cleaning with alcohol), electrode pairs were placed ~15, 13, and 20 cm above the patella for VL, VM, and RF muscles, respectively. The exact electrode positions were carefully measured for each subject to ensure that pre- and posttraining recording sites were identical. EMG electrodes were connected directly to small, custom-built preamplifiers taped to the skin. The EMG signals were led through shielded wires to custom-built differential instrumentation amplifiers, with a frequency response of 10–10,000 Hz and a common mode rejection ratio better than 100 dB. The preamplifiers lowered the impedance, which effectively prevented movement artifacts. Neither passive movements nor tapping the leg produced any visible artifacts. No significant crosstalk was observed between EMG channels as revealed by cross-correlation analysis.

To monitor the degree of hamstring involvement, EMG signals also were obtained in the biceps femoris caput longus and semitendinosus muscles. Care was taken to ensure identical pre- and posttraining recording sites. The magnitude of antagonist hamstring EMG did not change with training, neither when expressed in absolute units (μ V) nor when percentage hamstring co-activation was calculated according to procedures described previously (38), thus expressing antagonist hamstring EMG relative to that recorded in separate trials of maximal hamstring contraction performed at identical contraction modes and speeds.

Signal processing. Synchronous sampling of the dynamometer strain gauge signal, lever arm position, and EMG signals was performed at 1,000 Hz analog-to-digital conversion rate using an external A/D converter (dt2801-A, Data Translation, Marlboro, MA). The strain gauge signal was converted to newtons and multiplied by the individual lever arm length to calculate moment of force (torque). All recorded moments were corrected for the influence of gravity on the lower limb. At each respective contraction mode and velocity, the trial with highest work (i.e., largest moment-angle curve area and thus greatest average moment) was identified in each subject and selected for further analysis. A similar selection criterion was used in previous studies (38, 44).

During the latter process of analysis, all EMG signals were smoothed by using a linear envelope, as recommended by Winter (48), which consisted of digital high-pass filtering at a 5-Hz cutoff frequency followed by full-wave rectification and subsequent low-pass filtering at a 10-Hz cutoff frequency. All filtering routines were based on fourth order, zero-lag But-

terworth filters (48). To allow a comparison of neuromuscular activation between contraction modes and speeds, EMG was integrated and divided by integration time to yield the average EMG in the 30–70° range of knee joint excursion, according to procedures described previously (38, 44). Likewise, the average moment exerted in the 30–70° range was calculated. To quantify the degree of motoneuron inhibition (i.e., the reduction in neuromuscular activation) between speeds and contraction modes, EMG and moment obtained during slow concentric and slow-to-fast eccentric contraction conditions were normalized relative to that measured during fast concentric contraction (38, 44). Median power frequency (MPF) was determined from the raw EMG signals using a 256- or 2,048-point FFT spectral analysis for the fast and slow contractions, respectively. Before FFT transformation, a detrend procedure was performed to remove any dc-component (offset) and, subsequently, a Hanning window function was applied. To compare the conditions of a more flexed knee joint angle and high muscle forces with those of more extended joint angles and corresponding lowered muscle forces, EMG and moment also were averaged and normalized in the 60–90° and 10–40° ranges of motion (0° = full extension), respectively, according to the specific procedures described above.

Statistical methods. Unless otherwise stated, data are reported as group means \pm SE. The Friedman two-way ANOVA by ranks for related samples (7) was used to evaluate the change in neuromuscular activation of the respective quadriceps muscles and the change in maximal contractile quadriceps strength with training. Furthermore, the Friedman test was used to test the variation of these parameters across contraction modes and speeds.

RESULTS

Neuromuscular activation and contractile strength. Moment-angle and EMG-angle curves from typical trials are displayed in Fig. 1. Quadriceps muscle strength was higher during eccentric than concentric contractions, both pre- and posttraining ($P < 0.001$; Table 1). In contrast, before training, quadriceps EMG was lower during slow and fast eccentric and slow concentric contractions compared with that recorded during fast concentric contraction ($P < 0.001$; Table 1). To quantify the reduction in neuromuscular activation between speeds and contraction modes, the EMG and moment recorded during slow and fast eccentric and slow concentric contraction conditions were normalized relative to that of fast concentric contraction (Fig. 2, see METHODS). Compared with fast concentric contraction, quadriceps EMG was lowered 26–29, 34–36, and 17–24% for VL, VM, and RF, respectively, during eccentric and slow concentric contraction before training ($P < 0.001$; Fig. 2). Posttraining eccentric and slow concentric VL and VM EMGs remained lower (16–22%, $P < 0.001$) compared with that of fast concentric contraction, albeit to a significantly lesser extent than pretraining ($P < 0.01$; Fig. 2). Normalized VM EMG showed no increase during slow concentric contraction ($P > 0.05$; Fig. 2). Eccentric and slow concentric EMG of the RF muscle were not different from that of fast concentric contraction after training ($P > 0.05$; Fig. 2). In addition, absolute EMG amplitudes and maximal quadriceps strength increased with training ($P <$

0.001; Table 1). Thus slow and fast eccentric quadriceps strength increased 15 and 17%, respectively (from 227 ± 10 to 261 ± 10 Nm at 30°/s and from 229 ± 7 to 269 ± 9 Nm at 240°/s), whereas slow and fast concentric quadriceps strength increased 15 and 8%, respectively (190 ± 6 to 219 ± 7 Nm at 30°/s and 128 ± 4 to 139 ± 4 Nm at 30°/s; $P < 0.001$).

MPF. MPF was similar for each muscle in eccentric and concentric contractions and during slow and fast velocities ($P > 0.05$; Table 1). Pretraining MPF differed in the order VM < VL < RF, except during fast eccentric contraction, in which both VM and VL < RF ($P < 0.001$). Posttraining, the MPF difference was VM and VL < RF for all contraction types ($P < 0.001$). In addition, lower MPF was observed after training for VL during all concentric and eccentric contraction conditions and in RF during fast eccentric contraction ($P < 0.001$; Table 1).

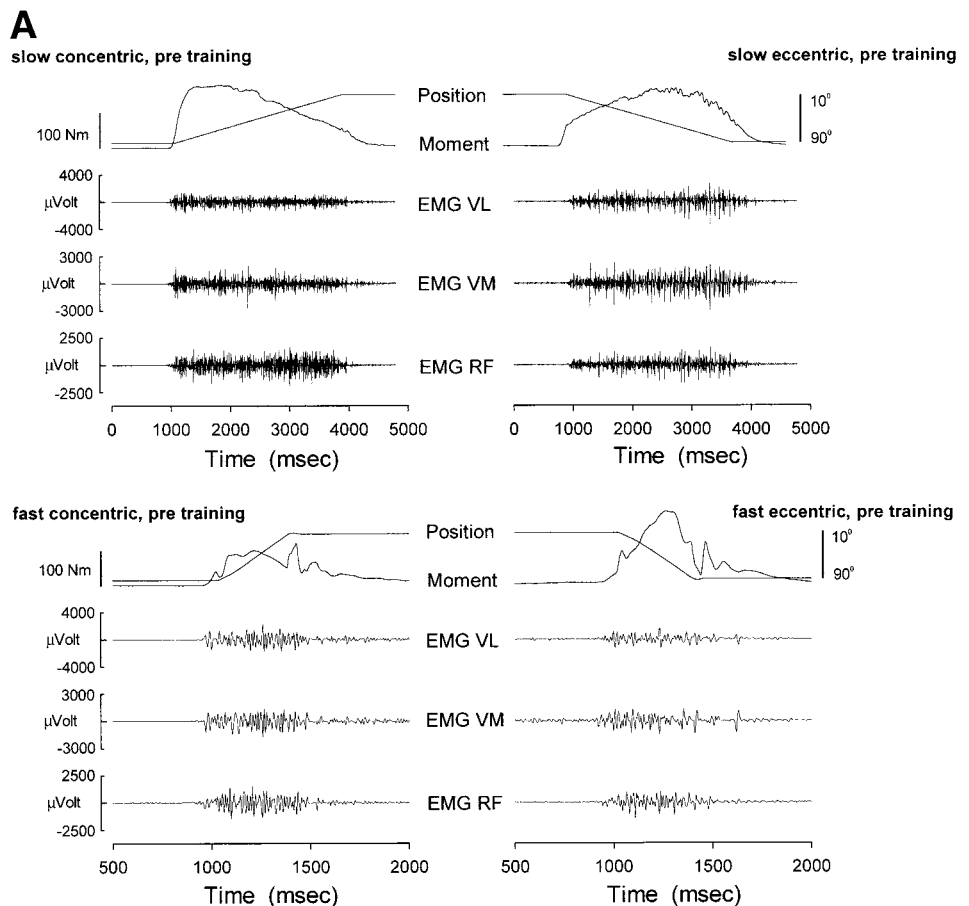
DISCUSSION

Several interesting findings were observed in the present study. First, pretraining neuromuscular activation was significantly lower during maximal voluntary eccentric and slow concentric contraction compared with fast concentric contraction, which indicates significant neural inhibition before training. Second, for the first time, it was demonstrated that this inhibition in motoneuron activation was reduced or completely removed after resistance training. Third, the removal of inhibition with training was paralleled by marked increases in neuromuscular activation and maximal quadriceps strength during slow concentric and eccentric contraction conditions.

Neuromuscular activation. Neuromuscular activation increased after the training period, particularly during eccentric and slow concentric muscle contractions (Table 1, Fig. 3). Several studies have reported increases in muscle EMG amplitude, suggesting increased neural drive to muscle fibers in response to resistance training (18–20, 33). However, not all studies have been able to show changes in maximal muscle EMG after resistance training (32, 40). The limitations associated with the recording of muscle surface EMG are discussed herein.

Existence of a neural force-inhibiting mechanism. The force-velocity or moment-velocity relationships recorded during maximal voluntary activation of human skeletal muscle often deviate markedly from those recorded in isolated in vitro muscle preparations (28), especially for maximal eccentric contraction (2, 3, 44–46). A plateauing of angle-specific concentric quadriceps moment is observed at low angular velocities (2, 3, 10, 47). It was proposed that this plateauing phenomenon is due to a force-inhibiting neural mechanism that is responsible for reducing the neural drive to the quadriceps muscle during maximal, high-tension contraction conditions (10, 47). By superimposing brief tetanic electrical stimulation onto maximal voluntary quadriceps contractions, indications of a decrease in

Fig. 1. Typical recordings of knee extension moment and neuromuscular activation of quadriceps femoris muscle, measured during maximal voluntary concentric and eccentric contractions both pretraining (A) and posttraining (B). *Top*: slow (30°/s) concentric (*left*) and eccentric (*right*) quadriceps contractions. *Bottom*: fast (240°/s) concentric (*left*) and eccentric (*right*) contractions. In each panel, upper tracings show measured knee extension moment (moment; *left*) and knee joint angle (position 10–90°, 0° = full extension; *right*). Lower tracings display raw electromyogram (EMG) signals recorded from the vastus lateralis (VL), vastus medialis (VM), and rectus femoris (RF) muscles. During fast contraction, irregular force peaks were seen in limb acceleration and deceleration phases. These movement phases were omitted from analysis, which was restricted to the middle 40° of range of motion (see METHODS for details). Note the different time axis scaling between slow and fast contractions. Also, pretraining EMG was lower during eccentric compared with concentric contractions, indicating significant neural inhibition. Furthermore, quadriceps contraction strength and EMG amplitudes increased after training, especially during eccentric and slow concentric contractions.



neuromuscular activation are seen during maximal eccentric quadriceps contractions (4, 46). Interestingly, the deficiency in eccentric activation was observed in sedentary subjects but not in strength athletes (4), suggesting that the underlying mechanisms may be modulated by training. Furthermore, maximal eccentric quadriceps moment has been estimated to correspond to ~150% of the maximal isometric moment (from extrapolation of the stretch-induced increment in moment observed at different intensities of voluntary effort) (43). However, because voluntary eccentric quadriceps moment was markedly lower than this predicted value, a neural regulatory mechanism that limits recruitment and/or discharge of motor units was proposed to exist (43). In the present study, the EMG recorded during maximal isokinetic knee extension clearly demonstrates that neuromuscular activation of the quadriceps muscle is lower during conditions of high contractile forces, i.e., in slow concentric and slow and fast eccentric contraction (Fig. 2). Lower neuromuscular quadriceps activation during maximal voluntary eccentric and slow concentric contraction has been reported by several authors (4, 6, 21, 29, 38, 39, 44). The data of the present study are the first to demonstrate that this inhibition in neuromuscular quadriceps activation may diminish (VL and VM) or even disappear completely (RF) as a result of resistance training (Fig. 2). Previous studies found marked in-

creases in maximal eccentric quadriceps strength (3, 8, 21–23, 37, 41) and reduced strength plateauing during slow concentric contraction (3, 10) in response to resistance training. It was speculated that the increase in eccentric strength was attributable to changes in neural drive, recruitment pattern efficiency, and inhibition of protective mechanisms (41). Interestingly, each of these specific adaptation mechanisms were observed in the present study; thus enhanced neuromuscular activation (increase in EMG; Table 1), reduced motoneuron inhibition (Fig. 2), and, although less substantiated, signs of more synchronized motoneuron activation (discussed below) all seem to have occurred with the present training regime. Few studies have addressed the influence of training on neuromuscular activation during maximal eccentric vs. concentric muscle contraction. Neuromuscular quadriceps activation was observed to increase during both eccentric and concentric contractions after resistance training (21–23). Higbie and co-workers (21) further analyzed their data and found that eccentric quadriceps activation (VL and VM) remained lower than that measured during concentric contraction, despite overall increases in neuromuscular activation after 10 wk of concentric- or eccentric-only resistance training performed by 35 women in an isokinetic dynamometer (21). The apparent discrepancy between their data and the present results (RF muscle) probably arises from differences in training

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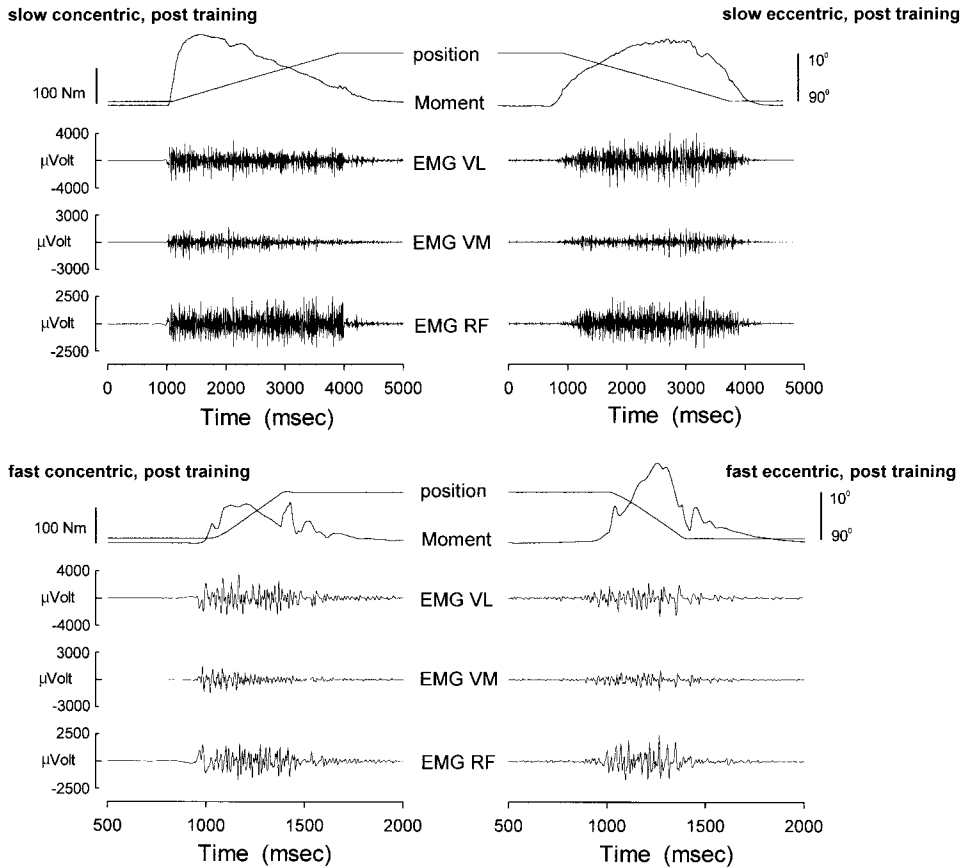


Fig. 1—Continued.

protocols. In the present study, three different exercises were employed for the quadriceps muscle, using very heavy loads (average 8 RM), whereas a single exercise was used in the study by Higbie et al., with slightly lower loads (isokinetic knee extension, arguably 10 RM loads). In addition, 30 sessions were performed in that study, whereas the subjects of the present study completed 38 sessions. Furthermore, men and women may show markedly different responses to resistance training in terms of gains in maximal eccen-

tric quadriceps strength. In a study by Colliander and Tesch (9), women displayed no further increase in maximal eccentric quadriceps strength during the latter phase of training (*weeks 8–12*), whereas men continued to show increases in eccentric strength (9).

Factors responsible for the inhibition in neuromuscular activation. The exact mechanisms responsible for the inhibition in quadriceps motoneuron activation remain unidentified. During maximal voluntary muscle contraction, the magnitude of neuromuscular activa-

Table 1. Quadriceps strength and average EMG and MPF during concentric and eccentric quadriceps contractions

	Fast Eccentric		Slow Eccentric		Slow Concentric		Fast Concentric	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post
Moment, Nm	229 ± 7	269 ± 9†	227 ± 10	261 ± 10†	190 ± 6	219 ± 7†	128 ± 4	139 ± 4†
EMG, µV								
VL	333 ± 30	443 ± 37†	346 ± 36	454 ± 47†	318 ± 26	484 ± 56†	493 ± 51	596 ± 58†
VM	423 ± 39	544 ± 65†	407 ± 32	498 ± 55†	426 ± 43	500 ± 49	675 ± 69	679 ± 84
RF	289 ± 25	381 ± 40†	288 ± 29	361 ± 38†	318 ± 36	369 ± 37†	392 ± 38	407 ± 39
MPF, Hz								
VL	92.7 ± 4.6	83.8 ± 4.3†	91.0 ± 4.1	81.8 ± 3.7†	91.2 ± 3.4	78.6 ± 2.8†	91.2 ± 3.6	80.3 ± 3.4†
VM	76.9 ± 4.2	75.6 ± 3.2	75.1 ± 2.7	75.6 ± 3.0	72.7 ± 2.8	76.7 ± 4.0	72.8 ± 2.9	78.3 ± 3.6
RF	113.5 ± 5.8	98.3 ± 4.8*	110.3 ± 5.8	102.2 ± 4.1	112.6 ± 5.8	104.9 ± 4.1	106.6 ± 4.5	104.3 ± 4.1

Values are means ± SE. Quadriceps strength (moment) and neuromuscular activation [average electromyographic (EMG) activity and median power frequency (MPF)] obtained during slow (30°/s) and fast (240°/s) concentric and eccentric quadriceps contractions, before (Pre) and after (Post) 14 wk of heavy resistance training. Changes in normalized EMG, with training, are shown in Fig. 2. VL, vastus lateralis; VM, vastus medialis; RF, rectus femoris. Numeric changes from pre- to posttraining: **P* ≤ 0.05; †*P* ≤ 0.001.

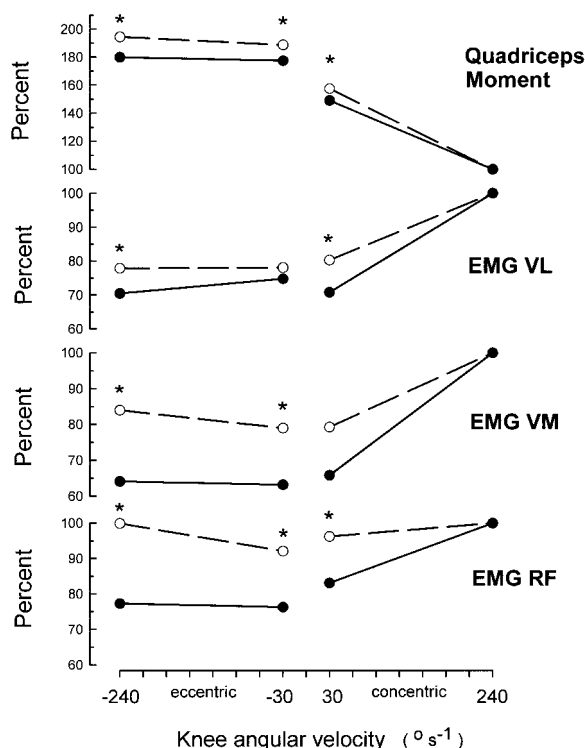


Fig. 2. Normalized quadriceps muscle strength (moment) and EMGs before (solid lines) and after (dashed lines) resistance training (means; $n = 15$). To quantify the magnitude of neuromuscular inhibition across speeds and contraction modes, EMG and moment were expressed relative to that measured during fast, concentric contraction. For the purpose of clarity, SE is not shown. Absolute values \pm SE are given in Table 1. *Changes pre- to posttraining, $P \leq 0.01$.

tion is regulated not only via central descending pathways but also through sensory reflex pathways, including group Ib afferents from Golgi organs in the muscle-tendon complex and group Ia and group II afferents from muscle spindles (17). The pattern of motoneuron activation may differ substantially between eccentric and concentric muscle contraction (14). Although evidence of preferential recruitment of type II motor units and derecruitment of type I units has been demonstrated during submaximal eccentric muscle contraction in humans (25, 31), this has not been confirmed for maximal eccentric quadriceps contractions (42). More likely, the decrease in neural activation could be caused by inhibitory feedback via sensory group I and II afferents. The sensory Ib afferents from Golgi organs in the muscle-tendon unit, together with the Ia and group II afferents from the muscle spindles, converge onto the entire motoneuron pool, not only from the muscle itself (homonymous afferent pathways) but also from antagonist muscles (heteronymous pathways) (17). The extensive localization of Golgi organs within the muscle itself (26) allows stress forces to be monitored in every portion of the muscle-tendon unit. Furthermore, Golgi organs have extremely low thresholds in response to active contraction, and evidence exists that the inhibition exerted from Golgi organs actively modulates motor output throughout the entire physiological range of force gradation (24, 26). In the spinal

cord, the Ib afferents from the Golgi organs excite inhibitory interneurons, which, in turn, are influenced by higher central nervous system centers via descending corticospinal pathways (i.e., rubrospinal and reticulospinal tracts). In addition, the sensory outflow of the Golgi organs reaches the cerebellum and cerebral sensory cortex. During voluntary contraction, Ib inhibition of homonymous motoneuron is reduced, probably as a result of presynaptic gating from supraspinal centers (26). This depression in Ib afferent inhibitory action increases with the force of contraction (26), which causes the gain of the Ib force feedback to vary during voluntary contraction. Thus the expression of maximal voluntary muscle force is influenced by numerous sensory afferent pathways, which, in turn, are modulated by dynamic gating control via central descending pathways. The present data suggest that the overall influence from these pathways may change with training. As an alternative explanation, the decrease in neuromuscular quadriceps activation could arise as a result of sensory afferent inflow from the anterior cruciate ligament (ACL). Evidence of a reflex pathway from the human ACL that modulates the activity of the knee musculature was recently reported (13). Furthermore, it is well known that forceful contraction of the human quadriceps muscle may induce significant tensile stress loads on the ACL at positions close to full knee extension (see Ref. 2 for review). To support the existence of an inhibitory reflex pathway excited by ACL stress forces, more pronounced reductions in neuromuscular activation would be expected to occur at extended joint positions. However, this does not appear to be the case (Fig. 3). Consequently, autogenic inhibition from Golgi organs seems to be a more likely candidate in accounting for the reduction in neuromuscular quadriceps activation during maximal eccentric vs. concentric contraction.

Changes in EMG MPF with training. Evidence of selective recruitment of type II muscle fibers during submaximal eccentric contraction in the triceps surae muscle group (31) and in some muscles of the hand (25) exists. In contrast, MPF did not differ between eccentric and concentric contractions in the present study, suggesting that maximal eccentric quadriceps contraction does not involve selective type II muscle fiber recruitment. In support of this, previous studies have been unable to find a difference in MPF between maximal eccentric and concentric quadriceps contractions (39, 42). In the present study, decreases in MPF and elevations in EMG were observed after training in the VL muscle (during all contraction conditions) and the RF muscle (fast eccentric contractions only). Similar findings were previously interpreted as indicating a more synchronous pattern of motoneuron activation (30). It should be noted, however, that the EMG power spectrum obtained by use of surface electrodes may not readily reflect actual motor unit firing rates (5, 12). Therefore, no firm conclusions can be made from the present data. In fact, it may be that selective recruitment of type II motor units during eccentric contraction (causing MPF to increase) was masked by a rela-

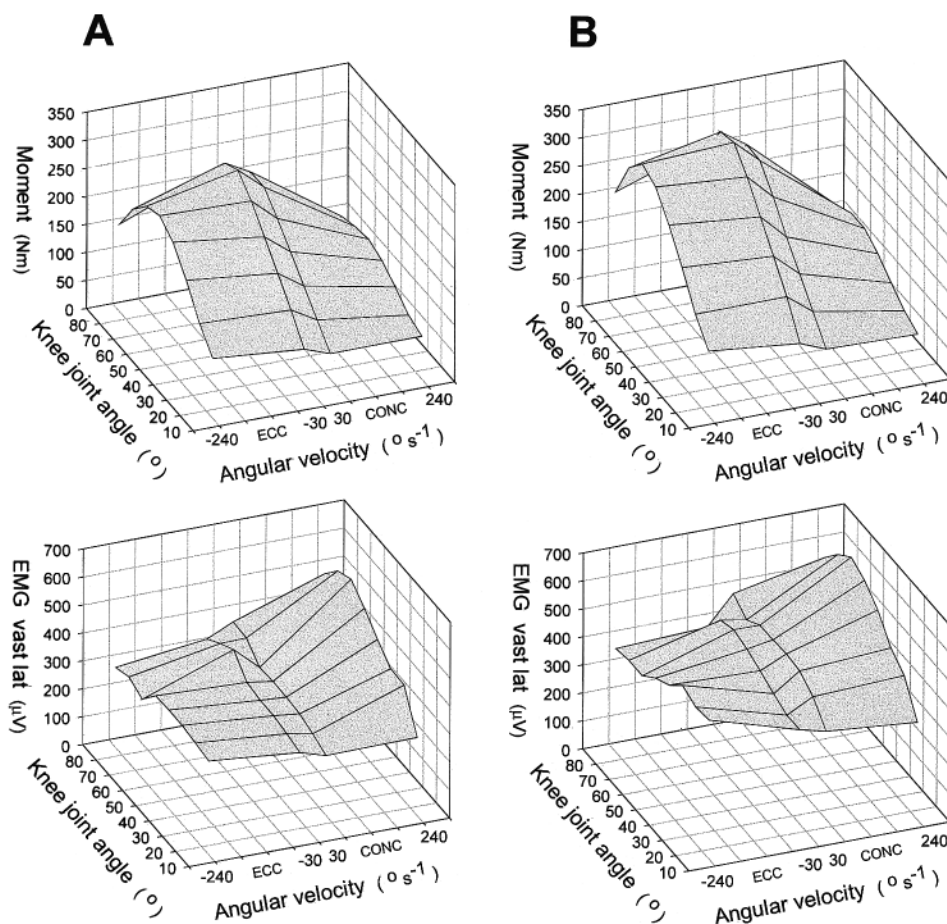


Fig. 3. Maximal quadriceps moment (*top*) and VL EMG (*bottom*) as a function of knee joint angle (averaged in 10° intervals, 0° = full knee extension) and angular velocity (slow = $30^\circ/\text{s}$, fast = $240^\circ/\text{s}$) and contraction mode [eccentric (ECC) and concentric (CONC)], before (A) and after (B) training ($n = 15$). Values for SE are omitted for the purpose of clarity. Averaged within the $60\text{--}90^\circ$ knee joint angle interval, pretraining VL EMG was 26–31% lower during slow concentric and eccentric contraction and 47% lower during fast eccentric contraction compared with fast concentric contraction ($P < 0.001$). In contrast, no reductions in EMG were observed at extended knee joint positions, i.e., when averaged at $10\text{--}40^\circ$ ($P > 0.05$). Similar trends were observed for VM and RF.

tive increase in synchronization due to the large size of type II motor units (causing MPF to decrease) as well as the appearance of more marked “on-off” activation patterns (also causing MPF to decrease). The recordings of the present study may provide some support of this notion, because very large EMG spikes separated by almost-silent interspike periods were typically observed during eccentric contractions (Fig. 1A, *right*). However, recordings of intramuscular EMG would be needed to further clarify this point.

Use of different training and testing devices. In agreement with a number of previous studies (3, 27, 32–34), maximal muscle strength and neuromuscular activation were evaluated using a dynamometer that was distinctly different from the machines employed during training. In result, the potential effects of improved learning or adaptation to the test situation itself were minimized (27, 34). In contrast, reporting the maximal load (e.g., 1 RM) that could be lifted in the specific machines or in the squatting movements used during training may result in muscle strength data that are markedly biased by “learning effects” (27, 34). Such strength values may often increase 50–100% within a few months of training, which does not reflect true adaptation in maximal muscle strength.

Implications for training and rehabilitation. Data reported by Hortobagyi and co-workers (23) suggest that training that involves true maximal eccentric

loadings may be more effective than concentric-only training in removing inhibition and creating a more uniform pattern of motoneuron activation between eccentric and concentric contraction modes (see Ref. 23, Fig. 3). However, the fact that concentric-only training is also effective in increasing quadriceps strength during eccentric and slow concentric contractions (3, 37) suggests that neuromuscular inhibition decreases, even with this type of training.

Limitations associated with the recording of muscle EMG. In accordance with the present findings, previous studies have demonstrated increases in muscle EMG amplitudes after resistance training (18–20, 33). It should be recognized, however, that electrode positions and muscle volumes may vary despite careful measuring procedures, thereby making changes in surface EMG a somewhat imprecise estimate of the actual change in neural drive to the muscle with training. Moreover, there are studies that have failed to show increase in maximal EMG after resistance training of the quadriceps muscle (32, 40) and selected hand muscles (11, 16). The compound surface EMG signal is comprised of a summation of several thousand action potentials (5) that cause a significant portion of the signal to be inherently stochastic in nature (12). Consequently, the sensitivity of the EMG signal as a training measure will depend on the specific EMG processing routines used (i.e., filtering algorithm, cutoff

frequencies, etc.), which may partially explain the lack of increase in neuromuscular activation in previous studies. Nevertheless, despite the bias inherent to surface EMG data, the present data demonstrate a clear elevation in EMG amplitude during maximal eccentric quadriceps contraction after training.

In conclusion, significant neural inhibition (i.e., reduced neuromuscular activation) was observed during maximal voluntary eccentric and slow concentric quadriceps contractions. This inhibition was reduced after a 14-wk regime of heavy resistance training. Although diminished, inhibition remained present for the VM and VL muscles, whereas it was completely abolished for the RF. Concurrently, increases in neuromuscular activation and maximal quadriceps strength were observed. MPF was not different between eccentric and concentric contraction conditions, which may indicate that maximal eccentric quadriceps contraction does not involve selective recruitment of type II motor units. Because decreases in MPF and increases in EMG were observed after training for the VL and, to a lesser extent, the RF, the possibility exists that the muscle fibers in these muscles were activated in a more synchronous manner after the period of resistance training.

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