Adaptations in coactivation after isometric resistance training

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CAROLAN, B., AND E. CAFARELLI. *Adaptations in coactivation after isometric resistance training.* J. Appl. Physiol. 73(3): 911–917, 1992.—Twenty sedentary male university students were randomly assigned to an experimental or a control group. The experimental group trained the knee extensors of one leg by producing 30 isometric extension maximal voluntary contractions (MVC) per day, three times per week for 8 wk. After 8 wk of training, extensor MVC in the trained leg increased 32.8% ($P < 0.05$), but there was no change in vastus lateralis maximal integrated electromyographic activity (IEMGmax). The most important finding was that the degree of hamstring coactivation during extension MVC decreased by ~20% ($P < 0.05$) after the 1st wk of training. Less pronounced adaptations occurred in the untrained leg: extension MVC force increased 16.2% ($P < 0.05$), hamstring coactivity decreased 13% ($P < 0.05$) after 2 wk of training, and vastus lateralis IEMGmax was unchanged. The same measures in legs of the control group were not changed during the study. There were no changes in flexion MVC, biceps femoris IEMGmax, or the degree of quadriceps coactivity during flexion MVC in either leg of the control or experimental group. A reduction in hamstring coactivity in the trained and untrained legs indicates that these muscles provide less opposing force to the contracting quadriceps. We conclude that this small but significant decrease in hamstring coactivation that occurs during the early stages of training is a nonhypertrophic adaptation of the neuromuscular system in response to static resistance training of this type.

coactivity; cocontraction; nonhypertrophic adaptations; hypertrophy; electromyogram; neural adaptation

ISOMETRIC RESISTANCE TRAINING increases the maximal force-generating capacity of skeletal muscle (1, 17, 29, 35). In humans this is usually accompanied by what appears to be a proportionally smaller increase in muscle cross-sectional area (10, 17, 32, 34). This discrepancy between increased force production and the degree of muscle hypertrophy has been attributed to nonhypertrophic adaptations, such as increased maximal neural drive to the muscle (12, 13, 20, 23, 30) or an increase in the degree of motor unit discharge synchronization (22). Increased electromyographic (EMG) activity, a measure of muscle activation during maximal voluntary contractions (MVC), has been observed in some (12, 13, 20, 23, 25) but not all (5, 10, 32) instances after resistance training.

We have taken a somewhat different approach to the problem of nonhypertrophic adaptations by examining the components that contribute to net force production. Measuring the force produced by a single muscle or muscle group requires that an indirect measure of joint torque be made at the point of force application. This is a measurement of the net force produced by the agonist muscle and the opposing coactivated antagonist muscle. Antagonist coactivation is often excessive in novel complex tasks but can be reduced with practice (28). In some cases, less antagonist coactivation is associated with a high skill level or familiarity with a movement (27, 31). If antagonist coactivation decreases as a result of resistance training, some portion of the increase in maximal force production would be independent of hypertrophy. The purpose of this experiment was to determine whether there is a reduction in hamstring coactivation after 8 wk of isometric resistance training of the quadriceps. A significant reduction in coactivation would constitute a nonhypertrophic adaptation that could account for some of the increase in MVC.

METHODS

Experimental Model and Subjects

The quadriceps femoris muscle group was chosen for training in this experiment for several reasons. *1*) It can be almost completely isolated from its synergists by use of a dynamometer similar to that of Edwards et al. (9). *2*) Force, EMG, and metabolic adaptations to training in this muscle group have been studied often (12, 13, 15, 17). *3*) The antagonist hamstring group is large and easily accessible for EMG electrode placement. When this model is used, it is necessary to clarify terms; we measured the force of the knee extensors and flexors and assumed that it was caused by activation of the quadriceps femoris and hamstring groups, respectively. The degree of activation of these groups was estimated from the EMG monitored over the surface of the vastus lateralis and the biceps femoris muscles. Our assumption was that these two muscles were representative of their constituent groups. We based this assumption partially on the data of Hakkinen and Komi (12), who showed that maximal surface EMG in these muscles is similar, and also on our own experiences with them (5, 6, 10).

Twenty sedentary male volunteer subjects were recruited from the university population. None had participated in weight training within the year before the onset of the study, and all reported a low level of physical activity. After an explanation of the protocol, the subjects gave their informed consent to participate in the experiment. The experimental procedures were approved by

the York University Human Participants Review Committee, and the subjects were paid for their participation.

Experimental Design and Protocol

Before beginning the experiment, each subject practiced by producing maximal and submaximal contractions of the extensors and flexors of both legs in the dynamometer. Practice continued until both flexion and extension MVC force was repeatable. Subjects were then matched according to MVC force, and the pool was randomly divided into a control and an experimental group of equal size.

The experimental group trained the extensor muscles of the dominant leg (TR) while the same muscles of the untrained leg (UT) served as a within-group control. Neither the dominant (C1) nor the nondominant leg (C2) in the control group was trained.

Extensor MVC and vastus lateralis maximal EMG activity (EMGmax) and EMG activity in the coactive biceps femoris were measured before the experimental group began training (PRE) and after 1, 2, 4, and 8 wk of training. Flexion MVC, biceps femoris EMGmax, and quadriceps coactivity were measured at the same intervals. These parameters were also measured at 25, 50, and 75% of flexor MVC for construction of force-EMG curves. Measurements at these intervals were made from the TR, UT, and C1 legs only. Measurements from the C2 limb were made only in the PRE session and again after 8 wk to determine whether the measurement sessions at *weeks 1, 2,* and *4* were sufficient to cause a training effect in the C1 limb.

The extensor muscles were trained by subjects performing 30 isometric MVC/day, 3 days/wk for 8 wk. Each MVC was held for 3–4 s, and ≥30 s separated each MVC. Training sessions were conducted in the same dynamometer used for measurement, and each session was separated by ≥48 h.

To avoid contraction and possible training of the UT leg, surface electrodes were placed over the vastus lateralis during each training session, and the EMG signal was displayed on an oscilloscope in front of the subject. Activation of the UT leg during TR leg contraction could therefore be detected and was discouraged. In fact, once the subjects learned to activate only the TR leg during the practice session, we saw almost no activation of the UT leg during the 8 wk of training.

Measurement Sessions

During the measurement sessions, the subjects were seated in the dynamometer, and padded nonextensible plastic cuffs were secured to the legs with Velcro straps just proximal to the malleoli. The cuffs were attached to strain gauges designed to measure either extension or flexion force. The amplified strain gauge output was displayed on a voltmeter in front of the subject and provided visual feedback of the force produced.

First, subjects were instructed to produce a single 4-s MVC of the extensor muscles. This procedure was repeated four times, and the largest of the four contractions was taken as MVC. To ensure maximization, superimposed shocks were delivered to the muscle during two of the MVCs. There was a rest interval of ≥1 min between all maximal contractions.

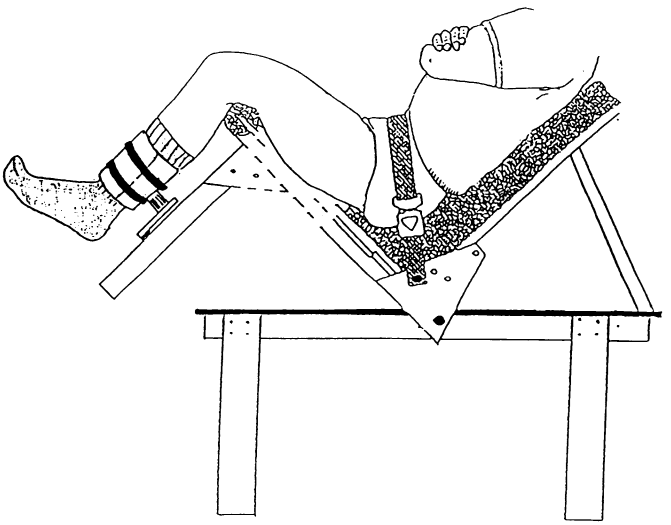

FIG. 1. Dynamometer modified from Edwards et al. (9). Seat beneath hamstrings was removed to provide access to biceps femoris muscle. Dynamometer was also tilted to allow most of subject's weight to be supported by buttocks.

Once MVC force was determined, visual feedback was provided to indicate 25, 50, 75, and 100% MVC. With this feedback as a guide, each submaximal force was produced twice, and each was held for 4 s. The protocols for determining MVC and submaximal forces were then repeated for the flexor muscles of the same leg and then repeated again for both muscle groups on the contralateral leg.

Techniques

Force. The chair dynamometer (Fig. 1) was modified from that of Edwards et al. (9). A portion of the seat was cut away to give access to the hamstring muscles, and the dynamometer was tilted backward so that most of the subject's weight was supported by the buttocks. The hips and knees were fixed at 90°, and the hips were stabilized with a seatbelt; the legs were secured to the strain gauges proximal to the malleoli. Force applied to the strain gauges in either direction produced voltage changes proportional to the applied force. These voltages were amplified (×10), simultaneously recorded on FM tape (model D, Vetter), displayed on the voltmeter, and digitized downstream at 1,024 Hz (Data Translation DT2801) for computer analysis. The strain gauges were calibrated with known weights before each measurement session, and the system was zeroed with the subject in the apparatus to account for the weight of the leg.

EMG. A Krusen bipolar silver-silver chloride surface electrode with an interelectrode distance of 2 cm was positioned over the vastus lateralis approximately one-third of the muscle's length from the point of insertion into the patellar tendon. A second bipolar electrode was placed over the biceps femoris half the distance between the ischial tuberosity and the crease of the knee. The position of the electrodes was marked with indelible ink to ensure consistent electrode placement throughout the experiment. EMG signals were preamplified at the electrodes and amplified again downstream (total system

gain 2,000). All EMG signals were high-pass filtered at 8 Hz and simultaneously stored on FM tape, displayed on a storage oscilloscope, and digitized (1,024 Hz) for off-line computer analysis.

The digitized EMG signals were full-wave rectified and integrated on a microcomputer (Zenith 386 Sx) with commercially available software (Asystant Plus, Keithly-Asyst Technologies). The rectified EMG signal was integrated over a 1.5-s epoch that coincided temporally with maximal force output during MVC (IEMGmax). During submaximal force production, the 1.5-s epoch was taken during constant force output and expressed as a percentage of IEMGmax. Similarly, the IEMG values of the coactive muscle during MVC were expressed as a percentage of IEMGmax when that muscle was producing MVC.

Percutaneous stimulation. To verify MVC, short trains of supramaximal shocks were delivered percutaneously to the muscle through large (10 × 10 cm) malleable surface stimulating electrodes (Medelco) placed over the proximal and distal ends of both the quadriceps and the hamstring muscle groups. A supramaximal shock was defined as one in which the voltage was 10% greater than the shock that produced a maximal twitch. During MVC, a 160-ms train of 130- to 150-V shocks at 50 Hz and 0.05-ms duration was delivered from a Grass stimulator (model S88). These trains produced forces of ~50% MVC, which were more clearly visible than a single twitch when superimposed on a near MVC. If the stimulation did not produce a discernible increase in the force record, it was assumed that all motor units were maximally activated (3, 7, 21). However, if the stimulation did produce an increase in force, the record was discarded, and the procedure was repeated until MVC was achieved.

Statistical Analysis

Data from the TR, UT, and C1 legs were collected at five time points during the study (PRE and 1, 2, 4, and 8 wk after the study began). However, data from the C2 legs were collected only at PRE and 8-wk intervals to determine the effects of the measurement sessions at 1, 2, and 4 wk on the C1 leg. Because there were missing data cells in the C2 leg, a three-factor analysis of variance could not be conducted. Therefore, separate two-factor analyses of variance were conducted on the experimental and control group data.

Experimental group. Two-factor repeated-measures analyses of variance were used to determine differences in MVC force, IEMGmax, and antagonist coactivity. Comparisons were made between TR and UT legs at five different time points (PRE and 1, 2, 4, and 8 wk).

Control group. The same analyses were conducted to determine differences in MVC force, IEMGmax, and antagonist coactivity in the control subjects. However, comparisons were made between the C1 and C2 limbs at only two time points (PRE and 8 wk). From these comparisons it could be determined whether there was a training effect from the measurement sessions at 1, 2, and 4 wk.

Probability levels were set at 0.05 for all analyses, and significant differences were tested with Duncan's multiple range post hoc tests.

TABLE 1. *Age, height, weight, and extension and flexion MVC of subjects in control and experimental groups before training*

Group	Age, yr	Height, cm	Weight, kg	MVC, N	
				Quadriceps	Hamstring
Control	22.1±0.9	177.9±4.4	76.2±3.2	760.2±44.7	509.4±42.6
Experimental	21.4±0.7	176.4±4.7	77.7±3.6	835.7±60.8	540.1±42.7

Values are means ± SE for 10 subjects in each group.

RESULTS

The descriptive statistics, including the initial extension and flexion MVC values for the experimental and control groups, are shown in Table 1. There were no significant differences between the groups in any of these parameters at the onset of the study, nor was there a significant change in the weight of the subjects between PRE and 8 wk.

Extension Contractions

After 8 wk of isometric resistance training, the TR leg extension MVC force increased 32.8% ($P < 0.05$), from 835.7 to 1,109.8 N, while that of the UT leg increased 16.2% ($P < 0.05$), from 743.5 to 864.3 N. Figure 2 shows that TR limb extension MVC increased progressively throughout the training. These increases were significant after 1 wk of training and again after 8 wk of training ($P < 0.05$). UT limb extension MVC increased significantly after 2 wk ($P < 0.05$) but did not change further during the remainder of the experiment. There were no significant adaptations in extension force in either the C1 or C2 leg over the course of the experiment.

Figure 3 shows the EMG activity in the biceps femoris during extension MVC over the course of the experiment. There was a significant main effect of weeks, indicating a decrease in biceps femoris coactivity in both the TR and UT limb with training ($P < 0.05$). Biceps femoris coactivation in the TR leg decreased from 14.7 to 11.5% of EMGmax after 1 wk ($P < 0.05$), whereas UT leg coactivation decreased from 10.7 to 9.3% of maximum over the same time ($P < 0.05$). Thereafter, coactivation in both of these limbs did not change. In contrast, there were no significant differences in the degree of biceps femoris coactivation in the control legs between the PRE and 8-wk measurement sessions.

Vastus lateralis IEMGmax was not altered in any limb over the course of the study (Fig. 4). Thus maximal activation of the quadriceps muscles during extension MVC was not increased by training.

Flexion Contractions

There were no significant alterations in flexion MVC force in any leg. With the hip and knee fixed at 90°, the force-generating capacity of the hamstrings was ~65% of the MVC force of the quadriceps. There were no significant changes in vastus lateralis coactivity during flexion MVC over the course of the study. However, the degree of quadriceps coactivity was approximately one-half that of the hamstrings when the hamstrings were acting as

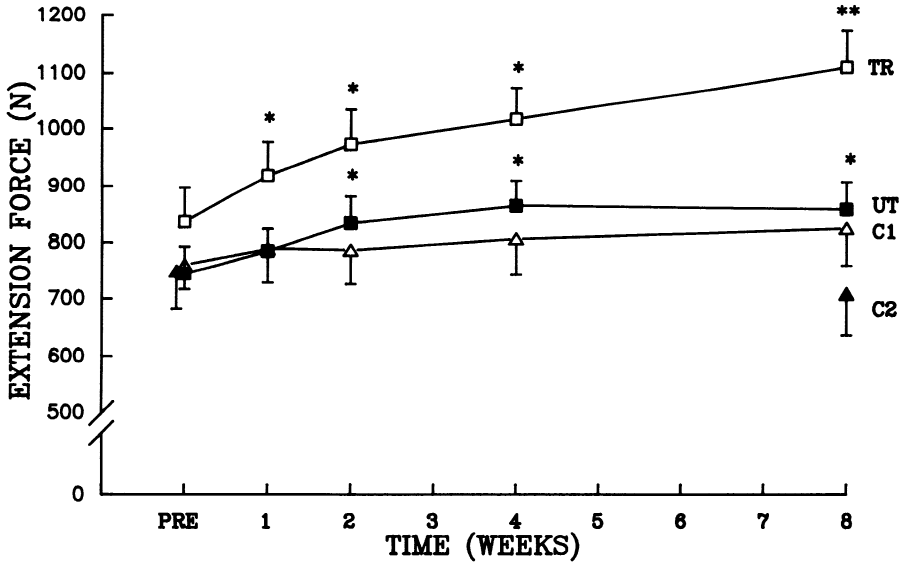

FIG. 2. Extension maximal voluntary contractions (MVC) in trained (TR), untrained (UT), and control (C1 and C2) limbs during training. MVC increased in TR (32.8%, $P < 0.05$) and UT limbs (16.2%, $P < 0.05$). * Significantly different from PRE values; ** significantly different from PRE and *values.

antagonists during quadriceps MVC. Also, there were no significant changes in biceps femoris IEMGmax during flexion MVC.

It is not possible to measure directly the amount of flexion force during coactivation; therefore, we estimated it from a force vs. EMG plot to determine how much of the increase in extension MVC could be accounted for by reduced coactivation. The force vs. EMG plot for the TR leg over a range of flexion forces (Fig. 5) is curvilinear, like the curves for other muscles (18, 19, 33, 36). This curved function shows that at lower EMG levels there is more force produced per unit EMG than there would be if the relationship were linear.

Figure 5 also illustrates the reproducibility of the surface EMG. Although the data are expressed here in relative terms, neither the absolute flexion MVC nor the absolute biceps femoris EMG changed after training. There is no systematic grouping of the pre- or posttraining data, and the curved function of the form shown in Fig. 5 fits the data quite well. When these EMG data are submitted to regression analysis, the correlation coefficient is 0.92, the slope of the relation is 0.96, and the intercept is 0.77.

DISCUSSION

The major finding of this experiment was that hamstring coactivation during extension MVC decreased significantly after 8 wk of static resistance training. Extension MVC force increased significantly, but there was no change in vastus lateralis IEMGmax, indicating no increase in the degree of activation of this muscle. There were no adaptations in control group hamstring coactivation, extension MVC force, or vastus lateralis IEMGmax.

The magnitude of the increase in MVC observed in the TR leg in this study is similar to what others have found with comparable resistance overloads (10, 17, 23, 34). Adaptations in the TR leg were rapid; force increased 10% after only 1 wk of training, and there was also a small but significant increase in UT leg MVC force. Increases in force production during the early stages of training are usually attributed to neural adaptations that are manifested in an increased EMGmax (12, 13, 20, 23, 30). It has been suggested that increased EMGmax may occur when some individuals are initially unable voluntarily to activate all motor units of a muscle maximally (3, 30). This

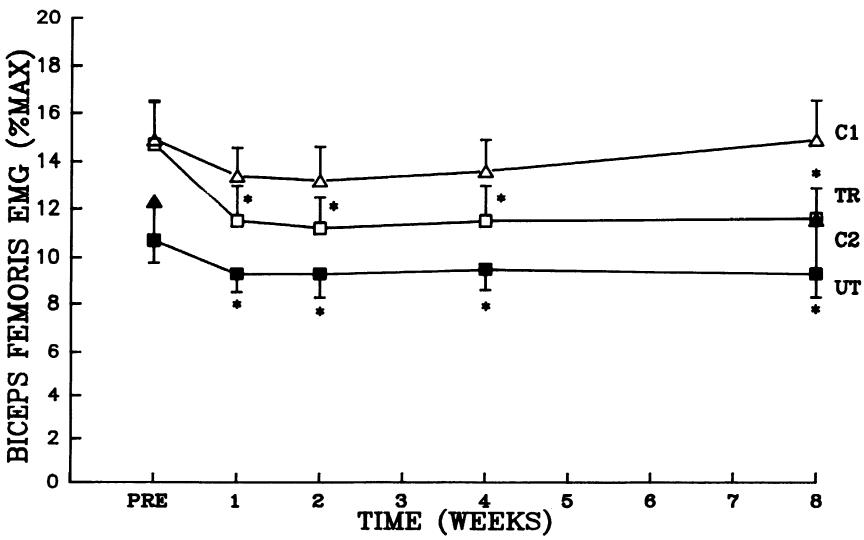

FIG. 3. Biceps femoris EMG during extension MVC in TR, UT, C1, and C2 limbs during training. Hamstring coactivity in each limb is expressed as a percentage of biceps femoris IEMGmax measured during flexion MVC. Hamstring coactivity decreased from 14.9 to 11.5% in TR limb ($P < 0.05$) and from 10.7 to 9.3% in UT limb ($P < 0.05$). * Adaptations significantly different from PRE values. There were no significant differences between PRE and 8-wk values in C1 and C2 limb biceps femoris EMG levels.

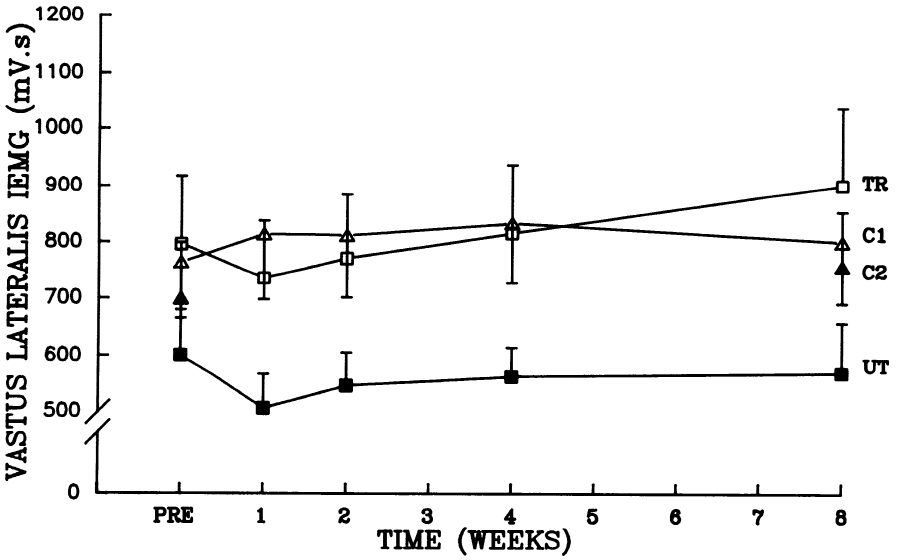

FIG. 4. Vastus lateralis IEMGmax during extension MVC. There were no changes in vastus lateralis IEMGmax in any leg over course of study.

inhibition may diminish over the course of training as a subject becomes more accustomed to the movement and would result in increased EMGmax and force (30). In the present study, however, all subjects were able to generate reproducible MVCs that were verified with superimposed shocks before the training began (3, 21). Furthermore, we found no evidence of adaptations in EMGmax in any leg in response to training, which replicated earlier results from this and other laboratories (6, 11, 21, 37). Thus we found no evidence of a reserve in neural drive or force-generating capacity that would contribute to an increase in extension MVC force.

Of some concern in studies of coactivation is the possibility of cross talk between the recording electrodes and adjacent muscles. We previously showed that the EMG activity picked up over the biceps femoris in this same preparation is not correlated with EMG activity from the vastus lateralis during an extension MVC (6). In the present study there were different degrees of adaptation in hamstring coactivity in the TR, UT, and control legs that would not occur if coactivity were due to cross talk. Furthermore, Moritani et al. (24) found that near-maximal stimulation of the medial gastrocnemius produced an M-wave over the gastrocnemius and the soleus muscles. However, the mean M-wave amplitude at the soleus was only 6% of gastrocnemius M-wave amplitude. On the basis of their observations, we reason that any cross talk between electrodes over the vastus lateralis and biceps femoris would be even less because of the size of the upper leg and the greater distance between these two muscles.

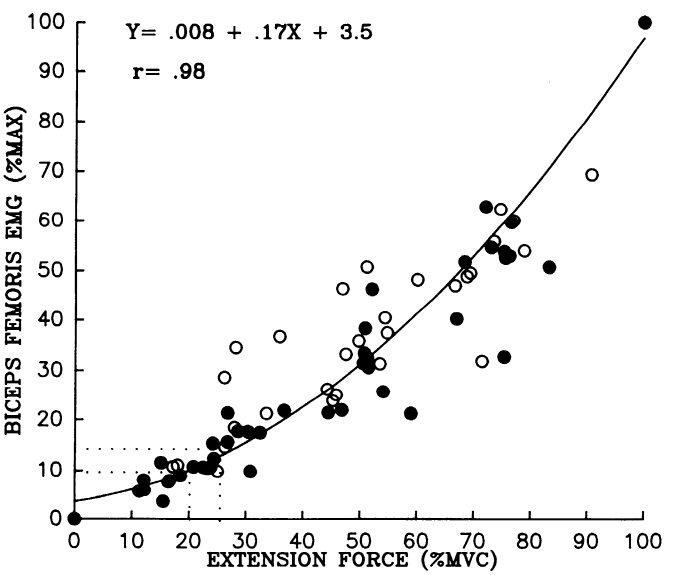

FIG. 5. Relationship between biceps femoris EMG and flexion force in TR leg before (open symbols) and after 8 wk of training (closed symbols). Second-order regression fit to data indicates that 14.9% of biceps femoris activity (*top dotted line*) corresponds to ~25% of flexion MVC. Decline in coactivity to 11.5% (*bottom dotted line*) corresponds to 20% flexion MVC. This decrease in hamstring coactivity in TR leg occurred after 1st wk of training.

Changes in coactivation could be a learned adaptation manifested as an improvement in coordination or skill (29). The dynamometer employed in this experiment restricts movement of the knees and stabilizes the hips. It seems reasonable for coactivation to be reduced, inasmuch as its functions of stabilizing joints (2) and evenly distributing pressure across articular surfaces (31) are assisted by these external devices. Reducing hamstring coactivation requires no conscious effort and therefore is likely mediated by mechanisms in the central nervous system. It has been suggested that coactivation is facilitated by Renshaw cell firing, which inhibits the Ia-inhibitory interneurons (16) by excitation of the Ib interneurons from the Golgi tendon organs (14, 27, 31) or by direct descending motor pathways (14, 31). Attenuation of any or all of these pathways would reduce coactivation. It is relevant in this regard that hamstring coactivation during extension MVC was reduced in both the TR and UT legs; this suggests that the central adaptations responsible for reduced coactivation cross over from the TR to the UT leg.

On the basis of the amount of biceps femoris EMG activity in the TR leg before training (14.7% hamstring IEMGmax), the flexors were generating 25% of their maximal force during extension. After 1 wk, coactivity had decreased and produced only 20% maximal flexion force during the same maneuver. This reduction corresponds to 28 N of flexion force. Extension MVC increased by 83 N during the same period. Thus the de-

crease in coactivation can account for approximately one-third of the increase in mean extension MVC after the 1st wk of training. However, the decrease in coactivation reached a plateau after 1 wk, whereas extension MVC force continued to increase. Of a total increase of 274 N in extension MVC after 8 wk, only 28 N of the increase were accounted for by reduced coactivation. If coactivation had not declined, the TR limb MVC force would have increased 246 N instead of 274 N. Thus after 8 wk of training, only 10.2% of the total increase in TR limb force was accounted for by adaptations in hamstring coactivity.

Because adaptations in antagonist coactivation do not account for all the nonhypertrophic increases in quadriceps MVC, the possibility of additional changes occurring elsewhere in the neuromuscular system must be entertained. For example, there is evidence that the radiological density from muscle CAT scans increases after training (17). This suggests that the packing density of myofibrils within a cell increases. However, radiological density can increase without an increase in the actual packing density of fibers (8). Furthermore, it is uncertain whether a rearrangement of the muscle matrix is possible or what effect such an adaptation would have on force production. There are also speculations that the angle of pennation of individual fibers might adapt so that they become more parallel to the direction of pull or that intermediate connective tissue attachments are formed along the length of the fiber (17). These hypotheses have not yet been investigated.

It has been suggested that adaptations such as increased neural drive or the synchronization of motor units may create a nonhypertrophic increase in agonist MVC (12, 13, 20, 22, 23, 30). However, it is unclear how an increase in neural drive (EMGmax) results in increased force production. The surface EMG signal is proportional to the number of motor units recruited and their firing frequency (4). Normally, all motor units in a muscle are maximally activated during MVC (21), and resistance training does not increase the number of muscle cells through hyperplasia (11). EMGmax could therefore increase after training only through an increase in the firing frequency of a significant portion of the motor unit pool. To our knowledge, there is no evidence to suggest that this occurs. The notion that the synchronization of motor units will increase maximal force-producing capacity is equally doubtful, inasmuch as electrical stimulation of a muscle with completely synchronous trains of supramaximal shocks does not produce more force than MVC (21).

In summary, we have offered evidence that, as a result of isometric training, the human neuromuscular system expresses nonhypertrophic adaptations that tend to maximize force-producing capacity. These adaptations are manifested as a reduction in coactivation of the antagonist muscles in both the trained and untrained limbs. This observation suggests a central mechanism. We suggest that coactivation should not be ignored when the force of agonist contractions is measured; in some of our subjects it was as high as 22% of EMGmax during isometric contractions and may be more during isokinetic movements (26). Force created by the coactivated hamstrings deters from net quadriceps force and should be considered when the adaptation to training of any muscle in a complex muscle-bone-joint system is evaluated.

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