Behavior of coactive muscles during fatigue

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PSEK, J. A., AND E. CAFARELLI. *Behavior of coactive muscles during fatigue.* J. Appl. Physiol. 74(1): 170–175, 1993.—Coactivation is antagonist muscle activity that occurs during voluntary contraction. Recently, we showed that the extent of coactivity in the knee flexors decreases after a short period of resistance training of the knee extensors (8). The purpose of the present experiment was to study the time course of coactivation in the knee flexors during fatigue of the knee extensors. Ten male subjects performed repeated submaximal static leg extensions in a low-intensity long-duration and a high-intensity short-duration fatigue protocol until they could no longer produce the required force [time limit of endurance (T_{lim})]. Maximal voluntary contraction (MVC), submaximal force, and surface electromyographic (EMG) activity were measured periodically. Vastus lateralis EMG increased progressively during fatigue of the extensor muscles ($P < 0.05$), resulting in a 38% change from control at T_{lim}. Biceps femoris EMG, which was our measure of coactivation, also increased by ~60% at T_{lim} in each protocol ($P < 0.05$). These observations lead us to conclude that a small but significant force loss during repeated static contractions to T_{lim} is due to an increase in antagonist activity. Moreover, the close correlation between the antagonist and agonist EMG supports the notion of a "common drive" to both motoneuron pools (10).

coactivation; cocontraction; repeated static contractions; electromyography; vastus lateralis; biceps femoris; common drive hypothesis

THERE ARE numerous processes that may be responsible for the loss of force-producing capacity that is characteristic of muscular fatigue. Examples of these are inadequate transmission of electrical activity from the central nervous system to the sarcolemma, impairment of excitation-contraction coupling, and impairment of various metabolic events that provide energy for the contractile machinery (4). It is known that antagonist muscle activity (coactivation) produces force in the opposite direction to that of the agonist muscles during voluntary static and dynamic contractions (7, 8, 17, 18, 20, 23–25, 27). Moreover, we have recently reported that the degree of coactivity decreases in response to a period of resistance training (8). Given this adaptability to long-term exercise, coactivation may also change in response to acute exercise. A decrease in coactivity over the time course of fatiguing contractions would help to extend the limit of endurance. On the other hand, should coactivation increase, it could also contribute to the loss of force-generating capacity associated with fatigue. Consequently, the time to maintain sustained or intermittent contractions at a desired force would be shortened.

It is well known that the force of a static contraction is proportional to the electrical activity that triggers agonist muscle contraction (4, 7, 8, 11, 13). However, neuromuscular adaptations to both training and fatigue alter this proportionality (4, 8, 9, 11, 13). For example, after a period of isometric training of the knee extensors, maximal voluntary contraction (MVC) increases significantly, but vastus lateralis maximal electromyographic activity (EMGmax) remains the same (8, 13). The force-electromyographic (EMG) relationship therefore becomes changed. The relationship between agonist EMG and coactivation may then be observed to determine whether the agonist electrical activity drives coactivation. Fatigue produces a small amount of change in agonist EMG during low-intensity contractions, yet yields a marked EMG increase during contractions held at high intensities (9). By using two different fatigue protocols of high- and low-intensity intermittent contractions, it is possible to relate the behavior of coactivation to two different patterns of increasing agonist EMG. There are thus two main purposes of this experiment: *1*) to determine the behavior of coactive muscles during progressive fatigue, and *2*) to determine whether there is a relationship between coactivation and the agonist drive that persists during fatigue.

METHODS AND PROCEDURES

Subjects and Experimental Model

Ten male students [mean age 25.1 ± 1.6 yr, height 175.3 ± 1.9 cm, and weight 73.5 ± 1.0 (SE) kg] participated in this study. These volunteers reported low to average levels of physical activity, and none were involved in weight training at this time. All experimental procedures were approved by the York University Human Participants Review Committee. All subjects gave their written consent after being informed of the nature of the experiment.

The knee extensors and flexors were used as a model in this experiment because *1*) the hamstring antagonists are large and easily accessible for electrode placement; *2*) the thigh can be stabilized to minimize activity from knee extensor and flexor synergists such as gluteals and hip adductors; and *3*) we have previously observed significant coactivation in the muscles around the knee joint (7, 8).

Techniques

Force measurement. The dynamometer (Fig. 1) was modified from one described by Edwards et al. (12) by

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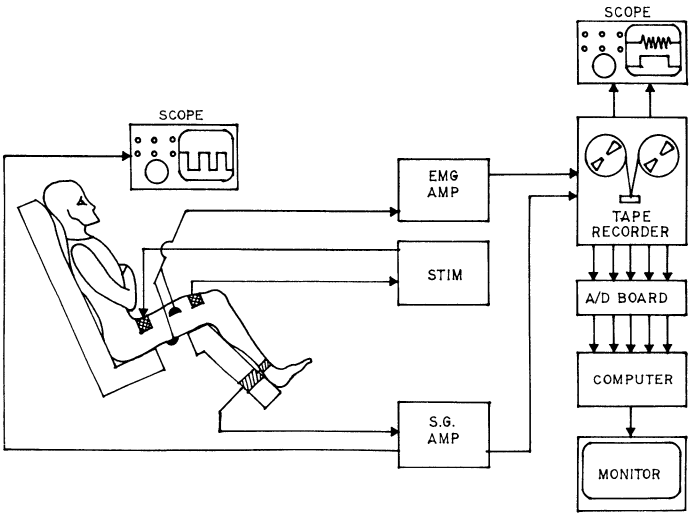

FIG. 1. Schematic drawing of experimental setup. Portion of dynamometer seat was removed to allow access to hamstrings. Force and electromyographic (EMG) signals were amplified with strain gauge (S. G. AMP) and EMG amplifiers (EMG AMP), stored on FM tape, viewed on a storage oscilloscope (SCOPE) for quality control, and digitized [analog-to-digital (A/D) board] for off-line computer analysis. Muscles were electrically stimulated (STIM) by means of stimulating pads placed on their proximal and distal portions.

removing a portion of the seat to allow access to the hamstrings (8). Each subject was strapped securely in the chair by an adjustable seatbelt, and the chair was tilted back to 45° so that the subject's weight was supported by the back and buttocks. The lower leg was secured above the malleoli to a strain gauge. Static leg extension and flexion produced voltage changes within the strain gauge circuitry that were proportional to the forces being applied. The strain gauge was calibrated with known weights before each experiment so that voltage changes could be expressed in newtons.

EMG. Before electrode attachment, the skin was shaved, rubbed with alcohol to reduce impedance, and marked with indelible ink. EMG activity was then recorded with Krusen bipolar silver-silver chloride surface electrodes placed over the belly of the vastus lateralis, 10 cm above the superior border of the patella, and over the long head of the biceps femoris, midway between the ischial tuberosity and the knee crease. Vastus lateralis EMG was representative of quadriceps electrical activity, and biceps femoris EMG was our measure of hamstring coactivation (3, 8, 14).

Signal acquisition and processing. Force voltages were amplified (×10) at the strain gauge, whereas EMG signals were preamplified at the electrodes and then amplified for a total system gain of 2,000. All signals were recorded on FM tape (model D, Vetter), displayed on a 20-MHz digital storage oscilloscope (BK Precision, Dynascan), and sampled at a frequency of 1,000 Hz for off-line computer analysis (Fig. 1). Force amplitude and EMG activity were calculated by a sequence of commands written in a commercially available data acquisition and processing software package (Easyest, Asyst Software Technologies). Both vastus lateralis and biceps femoris EMG signals were full-wave rectified and integrated (integrated EMG) over a 1.5-s epoch to obtain a measure of muscle activation during submaximal and maximal contractions. These values were then expressed in millivolts times seconds. The force associated with submaximal EMG was averaged over the same 1.5-s period, and the maximum voltage amplitude was calculated during MVC analysis. Software routines were used to adjust for periodic baseline shifts and direct current offsets of the amplifiers and tape recorder to ensure that EMG and force values were not inflated.

Protocol

One week before the first data collection, subjects practiced the fatigue protocols as well as maximal extension and flexion contractions; the latter was practiced until MVC measurements were repeatable within 10% of each other. Thereafter, two different fatigue protocols were performed 1 wk apart in counter-balanced order. In the low-intensity long-duration condition (LO protocol), static leg extensions were performed at 30% MVC for 7 s followed by a 3-s rest. This cycle continued until the target force could not be held for ≥4 s. This point was then designated as the time limit of endurance (T_{lim}). The high-intensity short-duration (HI protocol) series of contractions consisted of static 70% MVC extensions held for 3 s with 7-s rest periods intervening. T_{lim} occurred when the required force of 70% could not be attained or held for 1 s. Subjects maintained the alternating cycles of contraction and relaxation by following a pattern of target force production and rest drawn on an oscilloscope placed in front of them (Fig. 1). The oscilloscope was connected to the ankle strain gauge so that knee extension caused a deflection of the trace. In this way, subjects could match force production to the target force.

Measurement Sessions

Before beginning each fatigue protocol, subjects performed 4–5 extensions and then flexion MVCs with a 2-min rest between each contraction. The largest MVC amplitudes were calculated immediately and served as controls. The oscilloscope in front of the subject was then marked at 5, 10, 20, and 40% flexion MVC. Subjects held 4-s contractions at these submaximal forces so that individual biceps femoris (flexion) force-EMG curves could be constructed (Fig. 2). The amount of biceps femoris force produced by coactivation cannot be directly measured; therefore, force-EMG curves were required to estimate the force contributed by coactivation during leg extension. After a 10-min rest, one fatigue protocol was performed, and immediately after T_{lim} an extension and flexion MVC were performed.

Verification of MVC

Subjects were verbally encouraged and were highly motivated to exceed their previous maximum during MVC. However, MVC was also verified by objective means. For this procedure, stimulating pads were placed on the proximal and distal portions of the vastus lateralis and biceps femoris muscles. Trains of eight 75-μs pulses were delivered at 50 Hz to the agonist muscle during randomly chosen extension and flexion MVCs. If a discernable increase was detected in the force record, the contraction was not maximal, and the subject would repeat

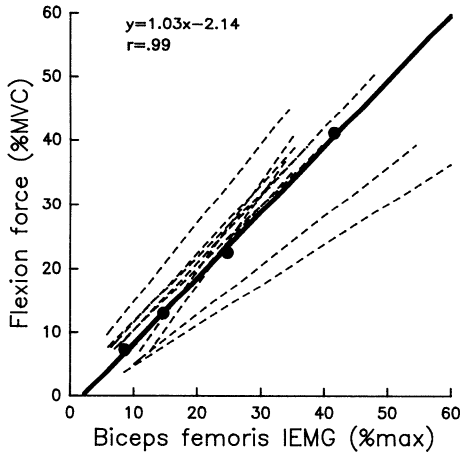

FIG. 2. Flexion force as a function of biceps femoris integrated EMG (IEMG) when subjects performed contractions at ~5, 10, 20, and 40% flexion maximal voluntary contraction (MVC). Dashed lines, regression lines of individual subject's force-EMG data; solid line and equation, regression analysis of mean data.

the MVC. This method is based on the twitch interpolation technique described by Merton (19), who showed that a supramaximal shock delivered during MVC produces no additional tension because of the complete recruitment of motor units discharging at optimal frequency. Bigland-Ritchie et al. (5) modified this technique by utilizing a train of pulses instead of a twitch because the brief tetanized stimulation produces a greater tension (~50% MVC), which does not fatigue as rapidly as a single shock.

Statistical Analysis

One-factor repeated measures analyses of variance (ANOVA) was used to detect any significant changes in control MVC, EMGmax, and coactivation produced before each fatigue protocol. The same analysis on three factors (muscle, protocol, and percent time to T_{lim}) was conducted to determine significant differences in vastus lateralis EMG and coactivation during fatigue of the knee extensors from *time 0* to T_{lim}. Probability levels were set at 0.05 for all analyses, and significant differences were tested with Duncan's multiple range post hoc tests.

RESULTS

Values are means ± SE unless otherwise stated. Subjects had an extension MVC of 807.7 ± 67.9 N and a flexion MVC of 398.7 ± 30.4 N. EMGmax was 548 ± 62 mV · s for leg extension and 591 ± 45 mV · s for leg flexion. Coactivation during extension MVC averaged 14.9 ± 2.2% of biceps femoris EMGmax, which is in agreement with values obtained in similar experiments in our laboratory (7, 8). The MVC, EMGmax, and coactivation values did not differ significantly when recorded on different days.

Length of Fatiguing Protocols

T_{lim} averaged 7.6 ± 1.1 min in the LO protocol and 14.1 ± 2.7 min in the HI protocol. Because of the wide range of T_{lim} values, the time of each contraction was expressed as a relative time (0, 50, and 100% of T_{lim}) for all subjects.

Vastus Lateralis EMG and Coactivation

Figure 3 shows that biceps femoris coactivation increased during the course of fatiguing leg extensions in both protocols. The integrated biceps femoris EMG increased from 32 ± 2 mV · s at *time 0* in the LO protocol to 51 ± 6 mV · s at T_{lim}, and from 61 ± 8 mV · s at *time 0* in the HI protocol to 96 ± 9 mV · s at T_{lim}. The values at T_{lim} constitute a 59.4 and 57.4% change, respectively, in EMG from their initial values. The three-way ANOVA performed on the data showed an interaction between protocol and time of contraction ($P < 0.05$). On analysis of this two-way interaction, the Duncan post hoc test revealed that biceps femoris EMG at T_{lim} was significantly different from biceps femoris EMG at *time 0* for both protocols.

The second purpose of this study was to examine the relationship between vastus lateralis and biceps femoris EMG during fatigue of the knee extensors. Vastus lateralis EMG increased by 40.9% in the LO protocol and 34.0% in the HI protocol at T_{lim} (Fig. 4), showing a similar pattern of rise to that of biceps femoris EMG (Fig. 3). As illustrated in Fig. 5, there is a strong linear correlation ($r = 0.96$) between the two parameters.

Extension and Flexion MVC

An extension MVC was performed immediately after T_{lim} (Fig. 6). Maximal force production was reduced by 29% in the LO protocol and by 17% in the HI protocol, both of which were significantly different from the control MVC ($P < 0.05$). Vastus lateralis EMGmax was not significantly different from control EMGmax. Coactivation during MVC was significantly greater than control biceps femoris EMG only in the HI protocol ($P < 0.05$). There were no significant changes in flexion MVC or biceps femoris EMGmax from control (Fig. 7). This shows that biceps femoris did not fatigue and that the

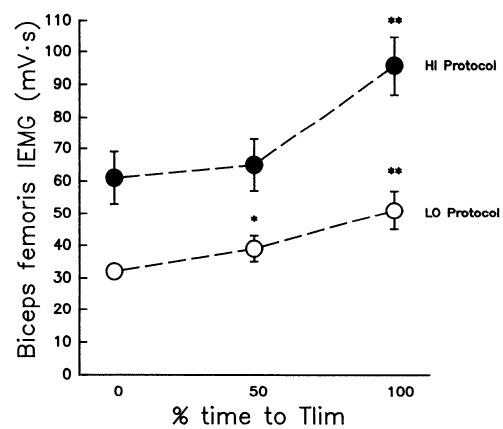

FIG. 3. Biceps femoris IEMG as a function of percent time to time limit of endurance (T_{lim}) during fatigue of knee extensors. Data from first, middle, and last contraction in series of contractions to T_{lim} are shown. Dashed lines connect data points for each condition and are not meant to illustrate rates of change. HI, high-intensity short-duration protocol; LO, low-intensity long-duration protocol. *Significantly different from control (*time 0*). **Significantly different from control and 50% time to T_{lim} (T_{50}) values.

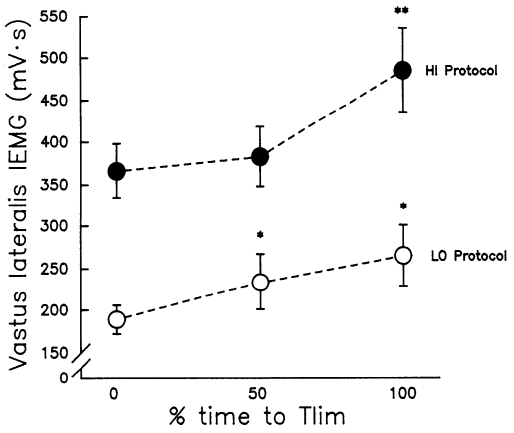

FIG. 4. Vastus lateralis IEMG as a function of percent time to T_{lim} during fatigue of knee extensors. Dashed lines connect data points for each condition and are not meant to illustrate rates of change. *Significantly different from control values. **Significantly different from control and T_{50} values.

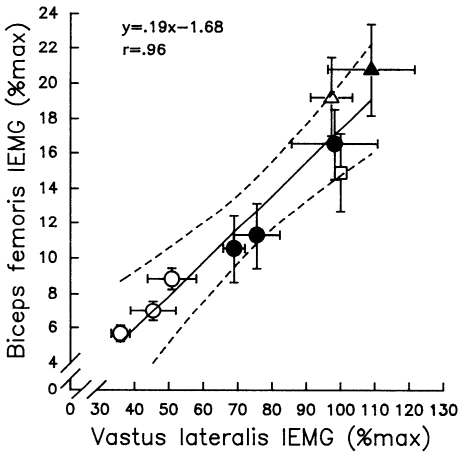

FIG. 5. Relationship between biceps femoris and vastus lateralis IEMG. Data points are means ± SE of IEMG values when expressed as percent of maximal IEMG during contractions at *time 0*, T_{50}, and T_{lim} during LO protocol (open circles) and HI protocol (solid circles), during extension MVC performed immediately after T_{lim} in LO protocol (open triangle) and HI protocol (solid triangle), and during control extension MVC (open square). Dotted lines, 99% confidence interval.

flexion force-EMG relationship was not altered during either protocol.

DISCUSSION

The purpose of this study was to determine the behavior of coactivation during the time course of fatigue and to relate these changes to the agonist electrical activity present during the same period. The results of our experiment showed that biceps femoris coactivation increased during two different patterns of contraction, and that this increase was correlated with vastus lateralis EMG.

Cross Talk

It may be argued that increases seen in biceps femoris coactivity may simply be "cross talk" from the quadriceps. Cross talk occurs when surface electrodes over one muscle register EMG signals from another muscle. However, several researchers have reported the presence of coactivation and did not attribute it to cross talk (3, 7, 24,

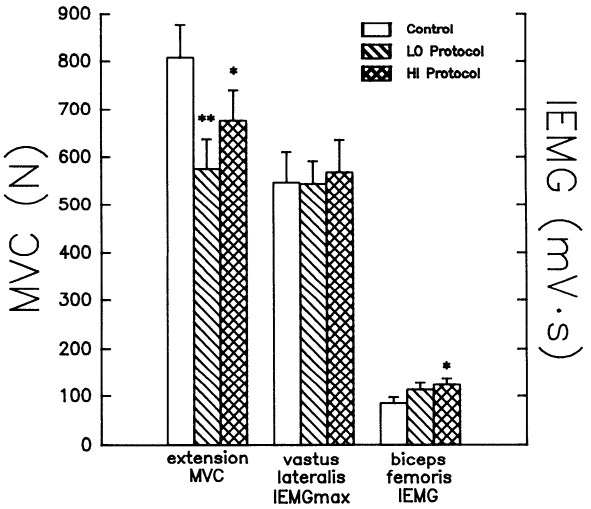

FIG. 6. Extension MVC, vastus lateralis maximal IEMG ($IEMG_{max}$), and biceps femoris coactivation during control and immediately after T_{lim}. *Significantly different from control ($P < 0.05$). **Significantly different from control and *values ($P < 0.05$).

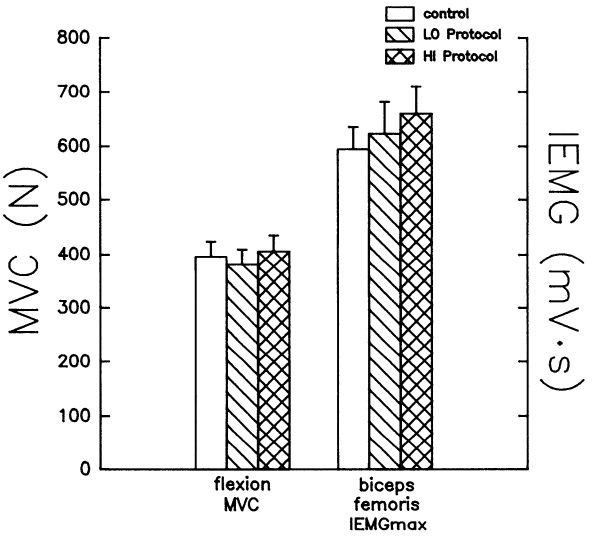

FIG. 7. Flexion MVC and biceps femoris $IEMG_{max}$ at control and immediately after T_{lim}. Note that there are no significant differences among these values.

27). In a recent resistance training study, there was a significant decrease in coactivation, yet no change in agonist EMG activity, which demonstrated the independence of the two EMG signals (8). It has also been shown that there is a high correlation between EMG signals recorded from wire electrodes and the corresponding EMG from surface electrodes placed directly above, indicating a similarity of shape of the two signals (7, 22). Because surface electrodes are placed further from the active muscle, the degree of correlation between the wire electrode signals and the surface electrical activity decreases (7, 22). Given the distance between muscles at the front and back of the thigh, simultaneous EMG signals measured in biceps femoris and vastus lateralis are not at all similar in shape (7), which suggests that there is no cross talk. Moritani et al. (21) found from surface electromyography that the mean mass action potential (M-wave) amplitude of the soleus was only 6% of gastrocnemius M-wave amplitude during percutaneous stim-

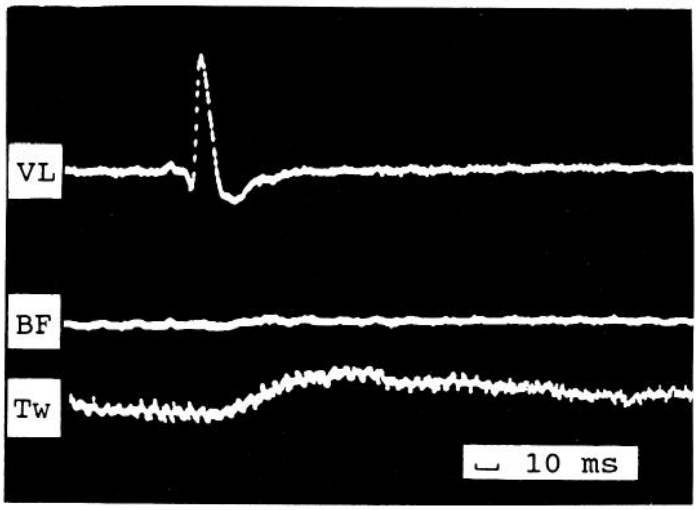

FIG. 8. Surface EMG of vastus lateralis (VL) and biceps femoris (BF), and twitch (Tw) characteristics of quadriceps during direct stimulation of femoral nerve. Presence of an M wave in VL but not in BF illustrates that there is no cross talk between recording electrodes. Signal amplification is same for VL and BF but is considerably less for Tw.

ulation of the medial gastrocnemius. Similarly, Bigland-Ritchie et al. (6) found M waves in adductor pollicis but none in opponens pollicis during ulnar nerve stimulation. These authors concluded that there is negligible signal contamination arising from adjacent muscles by virtue of their close proximity (6, 21). In a replication of these experiments, we also found no M waves in the biceps femoris surface recording during stimulation of the femoral nerve. An example of these results is presented in Fig. 8.

Fatigue

Muscular fatigue is a progressive process that describes the continual loss of force-generating capacity from the onset of muscle contraction to a point at which the desired output can no longer be achieved (3). Even though the capacity for maximal force production decreases, submaximal force output can be achieved by recruitment of additional motor units (1). An increase in hamstring force caused by coactivation detracts from the force produced by the quadriceps. Consequently, recruitment beyond what is required to compensate for fatigue is necessary to counteract coactivation and to maintain constant force output. Thus an earlier recruitment of fast-fatiguing motor units, as well as increased metabolic cost of active antagonist muscles, occurs. This suggests that increasing coactivation may shorten the time to T_{lim} and thereby contribute to the fatigue process.

To evaluate the extent to which coactivation contributes to the progressive decline in extension force-generating capacity during these fatiguing protocols, we first estimated the antagonist biceps femoris force present during extension from the data in Figs. 2 and 3. This value was then expressed as a percentage of the target extension force in each protocol. Coactivity was equivalent to ~6% of the target at *time 0* in the LO protocol. At T_{lim}, however, it had risen to 11.6%. In the HI protocol, the relative values were about the same, 6.7% at *time 0* and 11.2% at T_{lim}. Thus ~11% of the extensor force being generated at T_{lim} in both protocols was actually being used to offset the opposing force of coactivation. We have noted previously that the degree of vastus lateralis coactivation during flexion is about one-half what we observe in biceps femoris during extension (8). The force-EMG curve in Fig. 2 may therefore slightly underestimate the amount of biceps femoris force actually occurring during extension. In any case, T_{lim} would have been extended if coactivation were reduced or eliminated.

Coactivation

Certain situations favor coactivation rather than reciprocal inhibition, depending on the type and velocity of movement and the force produced by the agonist muscles (25). Movements that have the possibility of generating large amounts of force are associated with marked coactivation. These include high-velocity limb displacements (2, 18, 23) and contraction against a resistance (17, 20, 24). During fatiguing contractions, the agonist electrical activity increases to near maximal values in many cases. Virtually maximal contraction forces are generated near T_{lim} to maintain the desired submaximal force. Where large agonist forces are involved, it is generally thought that coactivation serves to prevent possible damage caused by the uneven pressure distribution on the articular surface of the joint created by agonist contraction (3, 27). Acute stress placed on the joint's cartilage would therefore be minimized and focal damage to the cartilage avoided (27). In addition, coactivation may aid the knee's ligaments in maintaining joint stability when the ligaments are overloaded during a forceful contraction (26).

Common Drive

The correlation between vastus lateralis and biceps femoris EMG during fatigue is consistent with the notion of a centrally mediated "common drive" to an agonist-antagonist pair (10). This hypothesis suggests that the central nervous system may control each muscle's motoneuron pool by a single input when both muscles are participating in a specific task (10). Empirical support for common drive can also be found in the observation that coactivation increases with increasing load. For example, the degree of coactivation in the triceps (24) and hamstrings (7) increases proportionately to activity in their agonist muscles. Furthermore, because the force-producing capacity of a muscle is continuously lost during fatigue (3), an increasingly larger proportion of that muscle's motor unit pool must be recruited to maintain the required level of contraction. An increase in agonist EMG occurs and, in accordance with the common drive hypothesis, a high degree of coactivation is expected. Other pathways have been suggested to mediate coactivation, either independently or together with common drive (3, 27). These include the activation of Renshaw cells that exert a feedback inhibition on the Ia-inhibitory interneurons responsible for reciprocal inhibition (15, 16) and the excitation of Ib-afferent interneurons from the Golgi tendon organs (15, 24).

In conclusion, we have quantified the behavior of biceps femoris coactivation during fatigue of the knee extensors and confirmed the presence of coactivation dur-

ing submaximal static contractions. The effects of coactivation during fatigue can be considered paradoxical; by increasing coactivation, susceptibility to joint injury is diminished, but time to T_{lim} is also reduced. Finally, by using two different protocols of fatigue, we have shown that there is a strong correlation between vastus lateralis and biceps femoris EMG, which supports the notion of common drive.

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