Alterations in corticospinal excitability with imposed vs. voluntary fatigue in human hand muscles

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Pitcher, Julia B., and Timothy S. Miles. Alterations in corticospinal excitability with imposed vs. voluntary fatigue in human hand muscles. J Appl Physiol 92: 2131–2138, 2002—We aimed to determine whether postexercise depression of motor-evoked potentials (MEPs) could be demonstrated without voluntary muscle activation in humans. Voluntary fatigue was induced with a 2-min maximal voluntary contraction (MVC) of the first dorsal interosseus (FDI) muscle. On another occasion, "electrical fatigue" was induced with trains of shocks delivered for 2 min over the FDI motor point. Five of the twelve subjects also underwent “sequential fatigue” consisting of a 2-min MVC of FDI followed by 20 min of rest and then 2 min of motor point stimulation. Voluntary fatigue induced MEP depression that persisted for at least 20 min. Electrical fatigue induced a transient MEP facilitation that subsided 20 min after the stimulation and became depressed within 30 min. Thus MEP depression can be induced by both voluntary and electrical fatigue. With electrical fatigue, the initial depression is “masked” by transient MEP facilitation, reflecting cortical plasticity induced by the prolonged electrical stimulation. MEP depression probably reflects tonic afferent input from the exercising muscle that alters cortical excitability without altering spinal excitability.

transcranial magnetic stimulation; motor cortex; afferents; plasticity; motor-evoked potential

AFTER A FATIGUING VOLUNTARY muscle contraction, the amplitude of the motor-evoked potential (MEP) produced by transcranial magnetic brain stimulation (TMS) is transiently depressed (2, 4, 9, 16, 18, 25, 26, 28, 31). These changes have been ascribed to intracortical rather than spinal mechanisms that reduce corticospinal output, on the basis of pre- and postexercise assessments of H reflexes and F waves (2, 4, 10, 16, 18, 26, 31) and responses to transcranial electrical brain stimulation (18) and cervicomedullary stimulation (28). All of these methods for examining motoneuronal excitability have failed to show a fatigue-induced alteration in α-motoneuron excitability that could account for the reduced MEP magnitude. Hence, postexercise MEP depression has been thought to reflect suboptimal cortical excitability (9, 28) and possibly “central fatigue” (2, 4, 18). The majority of these studies have not addressed the possibility that the depression may be induced not by intrinsic supra- or intracellular processes per se but by afferent influences from the forcefully contracting muscle that induce a prolonged change in central excitability without altering α-motoneuron excitability. Such changes in cortical excitability (“cortical plasticity”) have been seen after a variety of maneuvers that alter afferent input to the cortex, such as ischemic nerve block (3), repetitive electrical stimulation of peripheral nerves (13, 21, 22), and amputation (6, 24). As in postexercise MEP depression, the changes brought about by these interventions in the periphery are believed to reside in the motor cortex, again because concurrent changes in spinal excitability cannot be demonstrated (22, 27). That is, cortical excitability can be altered by afferent input without necessarily altering spinal excitability.

Electrical motor point stimulation can induce muscle fatigue, but, unlike fatigue induced with voluntary contraction, it does not involve descending corticospinal structures proximal to the site of stimulation. If it is assumed that spinal motoneuron excitability remains unchanged, any poststimulation (i.e., fatigue-related) changes in corticospinal excitability are likely to be due to afferent feedback from the fatigued muscle. However, if MEP depression does not occur after fatiguing motor point stimulation, the implication would be that MEP depression is due to changes in the corticospinal structures associated with descending drive and not from afferent feedback from the fatigued muscle itself.

Hence, the primary aim of the present study was to induce fatigue in an intrinsic hand muscle by electrical motor point stimulation and to determine whether postexercise depression of the TMS-evoked MEP could be demonstrated in the absence of voluntary activation. Only one other study of fatigue-related MEP depression has used electrical motor point stimulation to fatigue the target muscle. McKay et al. (18) stimulated the motor point of tibialis anterior until twitch force declined to 50% of the preexercise value and found no change in MEP amplitude. However, these authors acknowledge that their stimulation protocol (40-Hz pulse trains; 650 ms on, 350 ms off) induces “high-frequency” fatigue, whereas “low-frequency” fatigue...
more closely resembles that induced with voluntary activation (7). Jones et al. (14) showed that the rapid force loss and slowing of the action potential associated with high-frequency fatigue can be reversed if the stimulation frequency is reduced to 20 Hz. Therefore, a second aim was to ascertain whether MEP magnitude was altered after fatiguing motor point stimulation at the more physiological frequency of 20 Hz (see Ref. 1). The intrinsic hand muscle first dorsal interosseus (FDI) was chosen for this study because of its strong corticospinal projection. To ascertain whether changes were specific to the fatigued muscle, we also recorded from abductor digitii minimi (ADM) and abductor pollicis brevis (APB).

Preliminary findings have been presented elsewhere in abstract form (21).

METHODS

Six women and six men, aged 18–52 yr (27.7 ± 7.8 yr), gave informed, written consent to participate in the study. Ethical approval was obtained from the Adelaide University Committee on the Ethics of Human Experimentation, and all experiments were conducted in accordance with the Declaration of Helsinki. Subjects had no relevant medical history and attended the laboratory on two separate occasions not less than 4 days apart. Five subjects returned a third time. All investigations were on the right hand, which was the preferred hand for all but one subject.

Stimulation and EMG recording. Subjects sat with their right hand and forearm secured so that the distal interphalangeal joint of the index finger pressed on a load cell that measured abduction force. The thumb was supported, and the second to fourth fingers were secured with a strap so that only the index finger contributed to the force on the transducer. Surface electromyograms (EMGs) were recorded from the FDI and ADM muscles of the right hand with one electrode over the motor point and the other over the metacarpophalangeal joint. In five subjects, electrodes were also placed over the right APB. EMG signals were digitized and stored on a laboratory interface (model 1401, Cambridge Electronic Design, Cambridge, UK) for off-line analysis.

Single-pulse TMS were delivered by a Magstim 200 magnetic stimulator (Dyfed, UK) with a 90-mm circular stimulating coil held over the vertex of the scalp, oriented so that currents were induced in a posterior-to-anterior direction in the left motor cortex. The MEP threshold was defined as the lowest stimulator output at which at least 5 MEPs with a minimum amplitude of 50 μV were evoked from resting FDI in 10 trials. Each run consisted of 10 TMS delivered at 5-s intervals.

On different occasions, contractile fatigue was induced in the right FDI by voluntary contraction or by electrical stimulation of its motor point. For the latter (“electrical fatigue”), a cathode was placed over the motor point with a large saline-soaked sponge under the palm as anode. Fatigue was induced by stimulation with trains of supramaximal, 100-μs pulses at 20 Hz. A 650-ms-duration train was given each second for 2 min or until twitch force had declined to at least 50% of the prefatigue value. “Voluntary fatigue” was induced by a 2-min isometric maximal voluntary contraction (MVC) against the force transducer.

F and M waves were evoked in FDI by single supramaximal stimuli through surface electrodes placed over the ulnar nerve at the wrist. The amplitudes of the F and M waves before fatigue were determined from two consecutive runs of 20 stimuli given at 4-s intervals (15).

Experimental protocol. The maximal voluntary abduction force of FDI (i.e., the peak force produced in the best of 3 maximal contractions), its response to TMS, and the amplitudes of the F-wave and M-wave responses were determined at the beginning of each experiment. Fatigue was then induced in FDI with electrical stimulation or voluntary activation. Immediately after this, 20 F wave and 20 maximal M waves were recorded. Resting threshold to TMS was determined again, and this was followed as quickly as possible by a run of TMS at 10% of stimulator output above resting threshold. Finally, a single 2-s MVC was performed. This series of trials was repeated 20 min later. In four subjects, MEP trials were repeated every 10 min for 1 h after the electrical fatigue protocol. MVC trials ceased when force recovered to the prefatigue level.

Five subjects (selected pseudorandomly, based solely on availability) returned to perform an additional, combined protocol that consisted of a 2-min isometric MVC of FDI followed immediately by recording of MEPs and of F waves and M waves as before. Then, 20 min later, the electrical fatigue stimulation protocol was carried out while MEP amplitude was still depressed. MEPs, F waves, and M waves were recorded immediately after stimulation ceased and again 20 min later. The combined result of the voluntary fatigue followed by the electrical fatigue is referred to as “sequential fatigue.”

Analyses. EMG signals were analyzed for peak-to-peak amplitude and onset latency by using custom-written graphical programming software (LabVIEW, National Instruments, Austin, TX). The mean MEP and M-wave signals were obtained by ensemble averaging of 10 trials. In trials where background prestimulus EMG exceeded 2 SDs, the individual MEP was rejected and the remaining trials in that run were reaveraged. F waves are expressed as the mean peak-to-peak amplitude of 20 trials (cf. Ref. 19). The F wave-to-M wave ratio was calculated at each time point. The effects of various factors on MEPs, F waves, and M waves were analyzed by using between- and within-factor repeated-measures ANOVA with specific contrasts (SPSS for Windows version 9.0.1, SPSS, 1989-99). The between factor was muscle (up to 3 levels: FDI, ADM, and APB). The within factors were stimulus (up to 3 levels: TMS, F wave, and M wave), pretreatment (2 levels: none or voluntary fatigue, in the sequential fatigue protocol), and time (up to 3 levels: prefatigue, immediately on cessation of motor point stimulation, and 20 min poststimulation).

Where a factor had more than two levels, the pattern of response was assessed by polynomial contrasts. Post hoc analyses were carried out by using Bonferroni’s comparison with corrections. Relationships between variables were assessed by computing Pearson’s product-moment correlation coefficient (r). All comparisons and correlations were two-tailed. Statistical significance was accepted at P ≤ 0.05.

The FDI data for all 12 subjects are presented. A complete data set was available for FDI in all subjects, but, because of the strict criteria that excluded any trials in which background or prestimulus EMG was present in any of the three muscles for the electrical fatigue protocol, complete data for the other two muscles were available in only one subject. Linear interpolation (SPSS) was used to estimate the missing values for ADM and/or APB, but this was done only when all subjects had enough existing data points for the algorithm to estimate a missing value reliably (i.e., data for 2 of 3 time points were required for missing values to be estimated). The algorithm used within- and between-subject trends to esti-
mate the missing values. This gave a maximum of 12 subjects (n = 12) for electrical fatigue. Where estimated missing values are shown in RESULTS, n is denoted as n<sub>emv</sub> and analyses with these data were performed with the appropriate reduction in error degrees of freedom (i.e., by the number of imputed data points). Where actual measured values have been used, the number of subjects is denoted simply as n.

RESULTS

The onset latency of the FDI MEP did not change before or after fatigue in any of the three muscles or in any fatiguing protocol. Resting threshold to TMS was not altered by any of the protocols. Figure 1 shows the individual MEP responses of a typical subject to the different fatiguing paradigms. In contrast to voluntary fatigue (top 3 traces of Fig. 1B), which depressed MEP amplitude for at least 20 min, electrical fatigue (Fig. 1A) induced MEP facilitation for ~10 min. However, this subsided, and, after 20 min, MEP amplitude was depressed. This depression was still evident in this subject 60 min after electrical fatigue.

Voluntary fatigue. Figure 2 shows the group responses of FDI to the voluntary, electrical, and sequential fatigue protocols. The amplitudes of MEPs evoked in FDI when fatigue was induced by voluntary contraction were profoundly depressed for at least 20 min after cessation of the contraction (paired t-test, P = 0.021). The degree of amplitude depression after voluntary fatigue was related to the amplitude of the prefatigue MEP so that the larger the prefatigue MEP, the greater the degree of depression (nonlinear, P = 0.009) (see Fig. 5A). This postexercise depression did not occur in ADM, whose MEPs were not significantly altered by either electrical or voluntary fatigue (Fig. 3). There was a tendency for APB to be depressed immediately after voluntary fatigue, but this was not significant and MEPs recovered to prefatigue levels 20 min after voluntary fatigue.

**Fig. 1.** First dorsal interosseous muscle motor-evoked potential (MEP), M-wave, and F-wave responses of 1 subject to the different fatiguing paradigms. A: “electrical fatigue.” MEP amplitude was facilitated immediately after motor point stimulation and remained so for ~10 min, after which its amplitude fluctuated, but it was still depressed 60 min after the motor point stimulation ceased. B: “sequential fatigue.” Shown first is the profound depression in MEPs that occurred after voluntary maximal voluntary contractions (MVCs) for at least 20 min (“voluntary fatigue”), which is followed by MEPs after 2 min of motor point stimulation was given (sequential fatigue). MEP traces are ensemble averages of 10 consecutive MEPs. C: M waves and F waves during sequential fatigue. M-wave traces are ensemble averages of 10 consecutive trials. F-wave traces are the mean of F waves from 20 consecutive trials.
later. Both FDI M-wave \((P \leq 0.0001)\) and F-wave \((P = 0.02)\) amplitudes were decreased immediately after the contraction, but they had recovered 20 min later (Fig. 1C). However, the F wave-to-M wave ratio did not change significantly over this period.

**Electrical fatigue.** Figure 2A shows that, in the grouped data, in contrast to the depression seen in voluntary fatigue, motor point stimulation was followed by substantial increases in FDI MEP amplitude (mean increase of \(245 \pm 56\%\); \(P = 0.025; n = 12\)). However, 20 min later, this increase no longer differed from prefatigue values (time, \(P = 0.22; n = 12\)). A similar pattern of activity was seen in ADM, but it was not significant because of the highly variable responses across subjects. MEP amplitude was larger in FDI than in APB and exhibited a different pattern of change throughout the time after fatiguing stimulation (muscle \(\times\) time, \(P = 0.002; n_{envelope} = 5\)) (Fig. 3). Whereas FDI amplitude increased, APB amplitude tended to decrease and was significantly smaller than the prefatigue value 20 min after cessation of fatiguing stimulation of FDI (time, \(P = 0.03; n = 5\)) (Fig. 3). Overall, the pattern of change in MEP amplitude induced by electrical fatigue was different in FDI, ADM, and APB (muscle \(\times\) time, \(P = 0.007\)).
Examination of the patterns of amplitude changes of the FDI MEP in individual subjects suggested that both facilitation and depression were present after fatiguing motor point stimulation. In some subjects, facilitation was seen both immediately and 20 min after the fatigue protocol, whereas in others facilitation was transient and MEPs were depressed 20 min after the end of the stimulation. In two subjects, FDI MEP amplitude was depressed immediately in electrical fatigue, as it was in voluntary fatigue (Fig. 4). Therefore, a further series of experiments was conducted in which the FDI MEP amplitudes of four subjects were followed for 1 h after electrical fatigue. These data have been incorporated into Fig. 2A. In these subjects, the initial facilitation (mean increase of 233 ± 89%) lasted ~20 min after motor point stimulation ceased. Thereafter, FDI MEP amplitude remained depressed below prefatigue values for ~40 min, and in two subjects the depression was still evident 60 min after the electrical stimulation (e.g., Fig. 1A). Although there was some intersubject variability in the magnitude and temporal characteristics of the depression, all four subjects showed the same pattern of an initial facilitation followed by a persistent MEP depression after electrical fatigue.

**M-wave amplitude was reduced immediately after motor point stimulation (paired t-test, P = 0.03) but had returned to baseline levels 20 min later (Fig. 2B).** There was no change in M-wave latency or duration, and F wave-to-M wave ratio was not altered at any time point. F-wave amplitude did not alter with electrical fatigue (Fig. 2C).

**Sequential fatigue.** In this protocol, subjects first carried out a 2-min MVC of FDI, which, as before, induced a profound depression in FDI MEP amplitude (Figs. 2A and 1B). This depression persisted for 20 min in all five subjects (mean of 29.0 ± 9.6% of prefatigue MEP). When 2 min of motor point stimulation were then applied to the “depressed” FDI (arrows in Fig. 2), the MEP amplitude initially increased from ~27% of the prefatigue MEP to ~57% (Figs. 2A and 1B) but then declined within 20 min to 41% of its initial amplitude. The MEP amplitude approximately doubled with motor point stimulation in both protocols. However, in marked contrast with electrical fatigue alone (Fig. 5B), the facilitation after electrical fatigue in the sequential protocol was positively correlated with the amplitude of the preceding MEP (Pearson’s r = 0.90; P = 0.002), that is, the smaller the MEP before electrical fatigue, the smaller the degree of facilitation. This, in turn, was related to the degree of depression experienced as a result of the voluntary fatigue component. In summary, therefore, the bigger the initial MEP, the larger the depression evoked by voluntary fatigue.
fatigue and the less the MEP was facilitated by the subsequent electrical stimulation (Pearson’s $r = 0.90$; $P = 0.04$).

M-wave amplitude also decreased immediately after the voluntary fatigue component (paired $t$-test, $P = 0.002$), but it recovered to prefatigue levels 20 min later (Figs. 1C and 2B). When the motor point stimulation was then given, M-wave amplitude did not change further. F-wave amplitude decreased as a result of the voluntary fatigue (paired $t$-test, $P = 0.022$) but had recovered 20 min later (Figs. 1C and 2C). The electrical fatigue component did not alter F-wave amplitude.

Figure 5B shows that the facilitation after electrical fatigue was negatively related to the size of the prefatigue MEP (nonlinear, $P = 0.0006$); that is, small initial MEPs were facilitated relatively more than larger MEPs. This is the opposite relationship to that which was observed with voluntary fatigue (Fig. 5A).

**DISCUSSION**

This study demonstrates that the net corticospinal response after a fatiguing muscle contraction is determined by the combined influence of both excitatory and inhibitory inputs to the motor cortex. MEP amplitudes in human hand muscle are depressed during voluntary fatigue but initially facilitated when fatigue is induced with electrical (motor point) stimulation. This facilitation persists for ~20 min, but thereafter MEP amplitude is depressed for at least 40 min. However, this depression is less than that when the muscle is voluntarily fatigued (Fig. 2A), which may indicate that some weak facilitatory influences are still acting. Motor point stimulation of a voluntarily fatigued (and MEP depressed) muscle induces transient facilitation of the MEP without restoring the amplitude to prefatigue levels.

After a fatiguing MVC, MEPs were depressed in all subjects; however, the incidence and degree of MEP facilitation after electrically induced fatigue was highly variable between subjects. This facilitation was not due to a sustained increase in spinal motoneuron excitability because resting F-wave magnitude was not altered.

**Fatigue or activity dependency?** It must be emphasised that, although we have for simplicity used the expression “fatigue” throughout this paper, there is no evidence that the MEP depression is related to muscle fatigue per se. No study of postexercise MEP depression has identified a level of muscle fatigue that must be reached before MEP depression occurs. Indeed, the corticospinal changes appear not to be related to fatigue as it is often defined, namely, as the inability to maintain a desired force output. In this study, the ability to produce the preexercise MVC force recovered in ~10 min after the fatiguing contraction, but MEP amplitudes remained depressed for ~20 min. Similarly, after the muscle was fatigued electrically, MEP amplitude remained facilitated after MVC had recovered. These results suggest there may be little or no direct relationship between the state of the muscle’s force-producing ability and the level of corticospinal excitability. For the present purposes, it is sufficient to note that voluntary activation of a muscle at an intensity and duration that produces contractile force loss induces MEP depression. We have now shown that motor point stimulation that produces similar force losses induces MEP changes that include depression.

**Does electrical stimulation-induced facilitation mask fatigue-related MEP depression?** The present study shows that the nature of the changes in motor cortex excitability after fatiguing contractions depends on the manner in which the fatigue is induced; that is, voluntary contraction is followed by depression of corticospinal excitability, whereas after electrical motor point stimulation, the MEPs are initially facilitated before being depressed. However, electrical stimulation of afferents can in itself lead to persistent facilitation of MEPs. Hamdy et al. (13) reported that 10 min of high-intensity pharyngeal stimulation increased the excitability of the pharyngeal cortex. Although this finding and several subsequent studies (22, 23) have reported that cortical facilitation is induced by nonfatiguing electrical stimulation of mixed peripheral nerves, the pattern of initial facilitation followed by depression found in the present study has not previously been reported. Hence, it is likely that the intense burst of electrical stimulation of the motor point that we used (which stimulates proprioceptive afferents as well as motor fibers) has several concurrent but opposing effects. It not only fatigued FDI with the accompanying depression of MEPs but may also have induced short-term facilitation of the corticospinal projection to FDI (cf. Ref. 22). In the present study, the net output of the motor cortex reflects both of these processes, that is, the sum of the facilitation from the electrical stimulation plus the depression resulting from the fatiguing contraction.

The facilitation of MEP amplitude with motor point stimulation was proportionally similar in both fresh muscle and voluntarily fatigued (depressed MEP) muscle (i.e., the magnitude was increased approximately twofold, i.e., from 100 to 245% in fresh muscle and from 27 to 57% in the sequential protocol), suggesting that the preceding voluntary fatigue does not affect the response to the subsequent electrical stimulation. However, in the sequential fatigue protocol, the more depressed the MEP, the smaller the facilitation that was induced by electrical stimulation. This suggests that the voluntary fatigue had altered in some way the ability of the FDI area of the cortex to be facilitated.

Thus the observation that the MEPs were initially facilitated after motor point stimulation but then eventually became depressed is consistent with the possibility that the muscle contraction elicited by motor point stimulation depressed the excitability of the motor cortex but that this was masked by the facilitation induced by the afferent stimulation: as this facilitation diminished, the underlying depression then became evident. In the two subjects in whom motor point stimulation did not induce facilitation, the pattern of corticospinal depression strongly resembled the de-
pression that occurred in voluntary fatigue. This supports the idea that the depression is induced by both voluntary and electrical activation of muscles to fatigue but that, in the case of electrical stimulation, it is initially masked by facilitation induced by the stimulation.

In all subjects, voluntary fatigue was followed by depression of cortical excitability. The observation that electrical stimulation most commonly induced an initial facilitation followed by depression of cortical excitability implies that it activates one or more classes of afferents that either are not activated by or are less affected by the voluntary fatigue protocol. These inputs facilitate the cortex but the magnitude and persistence of the facilitation vary among individuals. Unpublished observations from other studies in this laboratory (M. C. Ridding, T. S. Miles, and C. S. Charlton) also suggest that different subjects are more susceptible to facilitation by electrical stimulation than others. This variability may have contributed to the finding that MEP amplitude did not change significantly after motor point stimulation in a study of fatigue-induced MEP changes in the leg (18). However, a more likely explanation for the discrepancy in findings involves the greater density of direct, corticospinal connections controlling the hand muscles compared with those controlling the leg muscles (5).

**Origin of MEP depression.** There are a number of possible sites at which fatigue-related influences could act to reduce MEP amplitude. These include neural pathways projecting to the motor cortex, the cortex itself, corticospinal neurons, and motoneurons. Gandevia et al. (10) showed that, immediately after a 2-min maximal voluntary elbow flexion, the excitability of corticospinal axons is reduced. However, this recovers within 2 min of cessation of contraction and is facilitated thereafter, whereas MEPs remained depressed for many minutes. Thus, although this factor may contribute to the initial phase of postcontraction MEP amplitude depression, it does not persist and is not the primary cause.

Depressed MEPs could also result from depression of motoneuronal excitability. It has been argued that this is not the case in earlier studies using H reflexes and/or transcranial electrical stimulation to test motoneuronal excitability after fatiguing contractions. Because H reflexes are not consistently found in intrinsic hand muscles, we reexamined this issue by measuring F-wave responses (19). Whereas F waves did change during fatigue, the M waves always changed in parallel so that the F wave-to-M wave ratio remained constant. It is therefore most likely that the changes in both the M and the F waves are the result of the well-known changes in membrane properties of fatigued muscles (see Ref. 8 for review); that is, there is no evidence for a change in the excitability of the hand motoneurones during the fatigue induced by either protocol.

It could be argued that at least part of the MEP depression is due to late depression of the M wave. McFadden and McComas (17) demonstrated that, during a 30-s, 20-Hz, fatiguing tetanus of biceps brachii, M waves declined by 50%, recovered to control values within 10 s after stimulation ceased, but 3 min later began to decline again. The decline continued for 3 h to ~42% of control values before recovering over the next 5–6 h. In the present study, M-wave amplitude was depressed by ~25% immediately after fatigue (voluntary or electrical) but had recovered to control levels 20 min later. In the sequential protocol, no further changes to M-wave amplitude occurred after it had recovered after voluntary fatigue. Therefore, the MEP depression evident at or after 20 min cannot be attributed to late depression of the M wave.

The present study therefore supports the prevailing view that the MEP depression is not the result of decreased spinal motoneuron excitability and is therefore likely to reflect a tonic decrease in corticospinal excitability (2, 4, 18, 26, 28). However, the present data extend this view by suggesting that the cortical depression may be induced not by the “effort” of making a sustained MVC but by afferent discharges from the muscle during the fatiguing contraction in a manner analogous to the cortical facilitation induced by afferent stimulation (13, 22, 23). The nature of this afferent discharge, or the fibers involved, is not clear. A range of muscle afferents responds to changes within the muscle during fatiguing contractions. Group III and IV afferents are activated in response to mechanical (e.g., intramuscular pressure) and biochemical (e.g., extra- and intracellular ion concentrations, lactic acid) changes within a muscle and are believed to modulate motoneuronal firing rates during fatiguing contractions (Ref. 12; reviewed in Ref. 11). However, these fiber groups do not mediate postexercise MEP depression (30). Taylor et al. (29) recently suggested that Golgi tendon organs or group III and nonspindle group II afferents might act supraspinally during contraction, modulating descending drive to counteract fluctuations in muscle force output without altering spinal motoneuron excitability. If so, tonic discharges from these fibers during the course of fatiguing contractions would be potential candidates for inducing the corticospinal depression observed after either voluntary or electrically induced fatiguing contractions.

In summary, postexercise MEP depression can be induced in the absence of voluntary muscle activation by electrical motor point stimulation, and this depression is initially masked by a transient cortical facilitation. These results are consistent with the idea that postexercise MEP depression is induced by tonic muscle afferent discharges during contraction that induce prolonged postexercise plastic changes in excitability, either within or “upstream” from the motor cortex, that reduce corticospinal output without concomitant changes in spinal excitability. The afferents responsible are yet to be identified.

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