HIGHLIGHTED TOPIC | Fatigue Mechanisms Determining Exercise Performance

A comparison of central aspects of fatigue in submaximal and maximal voluntary contractions

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Magnetic and electrical stimulation at different levels of the neuraxis show that supraspinal and spinal factors limit force production in maximal isometric efforts (“central fatigue”). In sustained maximal contractions, motoneurons become less responsive to synaptic input and descending drive becomes suboptimal. Exercise-induced activity in group III and IV muscle afferents acts supraspinally to limit motor cortical output but does not alter motor cortical responses to transcranial magnetic stimulation. “Central” and “peripheral” fatigue develop more slowly during submaximal exercise. In sustained submaximal contractions, central fatigue occurs in brief maximal efforts even with a weak ongoing contraction (<15% maximum). The presence of central fatigue when much of the available motor pathway is not engaged suggests that afferent inputs contribute to reduce voluntary activation. Small-diameter muscle afferents are likely to be activated by local activity even in sustained weak contractions. During such contractions, it is difficult to measure central fatigue, which is best demonstrated in maximal efforts. To show central fatigue in submaximal contractions, changes in motor unit firing and force output need to be characterized simultaneously. Increasing central drive recruits new motor units, but the way this occurs is likely to depend on properties of the motoneurons and the inputs they receive in the task. It is unclear whether such factors impair force production for a set level of descending drive and thus represent central fatigue. The best indication that central fatigue is important during submaximal tasks is the disproportionate increase in subjects’ perceived effort when maintaining a low target force.

Muscle fatigue in human performance can be defined as any exercise-induced decrease in maximal voluntary force or power produced by a muscle or muscle group (e.g., 11, 27). Multiple processes in the nervous system and muscle contribute to muscle fatigue, many of which begin at the onset of voluntary contraction. Fatigue progresses during exercise and starts to recover when exercise stops. At some time during exercise, fatigue will reduce maximal voluntary force or power measurably. This time will depend on the intensity of muscular activity. If exercise is submaximal, then measurable fatigue can occur without a decrement in task performance as other motor units or muscles are recruited to compensate for those that are fatiguing.

One way to analyze processes contributing to muscle fatigue is to divide them into peripheral and central fatigue. Peripheral fatigue refers to exercise-induced processes that lead to a reduction in force production and that occur at or distal to the neuromuscular junction. It can be demonstrated by a fall in the twitch or tetanic force produced by peripheral nerve stimulation while the muscle is at rest. However, the simultaneous presence of muscle potentiation and fatigue can sometimes make interpretation difficult (e.g., 30). Central fatigue refers to...
more proximal processes and can be defined as a progressive exercise-induced failure of voluntary activation of the muscle (e.g., 27). Central fatigue can be demonstrated by an increase in the increment in force evoked by nerve stimulation during a maximal voluntary effort (3, 71). If extra force can be evoked by motor nerve stimulation during a maximal voluntary effort, some motor units were not recruited or were not firing fast enough to produce fused contractions at the moment of stimulation (36). An increase in this increment (superimposed twitch) signifies central fatigue and means that central processes proximal to the site of motor axon stimulation are contributing to a loss of force. Some central fatigue can be attributed to supraspinal mechanisms (28, 103). For the elbow flexors, transcranial magnetic stimulation (TMS) of the motor cortex elicits superimposed twitches despite subjects’ maximal efforts (28, 107). This indicates that, at the moment of stimulation, motor cortical output is not maximal (some remains untapped) and is not sufficient to activate all motor units to produce maximal muscle force. Therefore, motor cortical output is not optimal. An increase in the superimposed twitch elicited by cortical stimulation during exercise is the marker of supraspinal fatigue.

CENTRAL FATIGUE IN SUSTAINED OR REPEATED MAXIMAL CONTRACTIONS

A maximal voluntary contraction has advantages for the study of central fatigue. First, it tests the entire motor pathway. Second, fatigue develops within seconds. Third, central fatigue can only be measured during maximal efforts as the superimposed twitch then represents a failure to drive the muscle maximally. In contrast, during a submaximal task there is no benchmark for optimal performance of the nervous system. Fourth, the task for the nervous system is “maximal” throughout the exercise (i.e., to drive all the motor units to produce maximal force). By comparison, a submaximal task allows the nervous system to compensate for muscle failure by increasing drive. Fifth, all the motoneurons are undergoing similar processes. That is, all should be recruited and firing at high rates from the start.

During a sustained isometric maximal voluntary contraction, force begins to fall almost immediately and the superimposed twitch evoked by motor nerve stimulation increases (9, 28). Thus central fatigue develops. As the superimposed twitch evoked by motor cortex stimulation also increases, some of the central fatigue is supraspinal (28). At the end of a 2-min maximal elbow flexion, voluntary force falls by ~60% and approximately one-quarter of this fall can be attributed to supraspinal fatigue (28, 106).

Although the underlying mechanisms of central fatigue are complex, slowing of motor unit firing rates has been measured in sustained and repeated maximal efforts (e.g., 8, 10, 63, 85). Observations that muscle contraction and relaxation also slow suggested that changes in the firing rate might match the muscle properties to preserve maximal force output (“muscle wisdom” hypothesis) (63). However, neither slowing of firing rates nor of the muscle always occurs (e.g., 42, 59). Furthermore, the increase in the superimposed twitch implies that some motoneurons slow such that the muscle fibers they innervate no longer produce fully fused contractions despite any slowing of the muscle. Thus the muscle wisdom hypothesis does not hold (e.g., 24). Rather, the mechanisms that contribute to slowing of motor unit firing rates are fundamental to central fatigue. Simplistically, three kinds of actions at the motoneuron pool might lead to motoneuron slowing: a decrease in excitatory input, an increase in inhibitory input, or a decrease in responsiveness of the motoneurons through a change in their intrinsic properties. It is likely that all three actions occur.

Testing of motoneuron excitability during fatiguing contractions shows that the slower firing rates are not due solely to a decrease in excitatory input. During a sustained maximal effort of the elbow flexors, a decrease in the cervicomedullary motor evoked potential (CMEP), measured in the electromyogram (EMG) of the active muscle, suggests that the motoneurons become less responsive to synaptic input (see also 2, 15, 66). The CMEP is a short-latency excitatory response to stimulation of the corticospinal tracts at the level of the cervicomedullary junction (100, 108). As it has a large monosynaptic component and is unaffected by classical presynaptic inhibition, the size of the CMEP reflects motoneuron excitability (38, 74, 77, 108). When motor unit firing rates are high during strong voluntary contractions, a withdrawal of excitatory input (disfacilitation) would reduce firing rates but increase the responsiveness of the motoneurons and the size of the CMEP (65). Thus decreased excitability of motoneurons during sustained maximal contractions does not reflect disfacilitation.

Repetitive activation may decrease the responsiveness of motoneurons to synaptic input. The process known as late adaptation can be demonstrated when motoneurons are given a maintained input (e.g., 33, 44, 87, 96). Initially the motoneurons fire repetitively, but with time, some motoneurons slow their firing rate and others stop (76, 96). Motoneurons recover within 1–2 min after the cessation of current. Similar processes are postulated to occur with synaptic input. Evidence for this in human subjects is limited (75). However, if subjects are given feedback that allows them to voluntarily activate a single motor unit at a constant rate, the longer the unit is active the more drive is required to maintain its firing rate (39). The increase in excitatory input to the motoneuron pool is evidenced by increased surface EMG, which indicates that other motor units have been recruited or are firing more. This result obtained during weak voluntary contractions of a hand muscle is strong evidence that repetitive activation makes active motoneurons less responsive to synaptic input.

Changes in inputs to motoneurons also occur during fatiguing exercise. Those inputs that are most likely to change include reflex inputs from muscle afferents, recurrent inhibition, and descending drive. Altered activity of the large-diameter muscle afferents is not likely to contribute to inhibition of the motoneuron pool, although facilitatory input may be reduced as muscle spindle firing is thought to decrease during maintained contractions (13, 35, 58), and presynaptic inhibition of the Ia afferents may be enhanced during fatigue (79, 82). Firing of Golgi tendon organs is also likely to decrease with the fall in muscle force during fatiguing maximal contractions, and inhibitory input to homonymous motoneurons decreases (49, 111). Small-diameter (groups III and IV) muscle afferents are variously sensitive to noxious mechanical and chemical stimuli so that some increase firing with the accumulation of metabolites in the fatigued muscle (45, 51, 70, 84, 90). The actions of these fatigue-sensitive afferents on motoneurons
is controversial with recent evidence showing inhibition of some human motoneuron pools (such as the elbow extensors) but facilitation of others (such as the elbow flexors) (66). The role of recurrent inhibition in fatigue and how it is controlled are also uncertain. Studies in humans using indirect methods suggest that recurrent inhibition increases during sustained maximal efforts and with experimental activation of nociceptive small-diameter muscle afferents but decreases during submaximal fatiguing exercise (48, 56, 83). Animal studies also suggest decreases in recurrent inhibition with fatigue and firing of small-diameter muscle afferents (40, 109). As recurrent inhibition varies between muscles, it is likely that its role in fatigue will also depend on the muscle involved (41).

Finally, demonstration of supraspinal fatigue using transcranial stimulation of the motor cortex indicates that descending drive becomes suboptimal for force production during sustained and repeated fatiguing maximal contractions (28, 37, 57, 98, 106). This does not necessarily imply that the absolute level of descending drive decreases with the development of fatigue. It is possible that descending drive could stay constant or even increase but might become less effective at driving the motoneurons, which have become less responsive to input. However, even if descending drive has been maintained, the increase in size of the superimposed twitch elicited by cortical stimulation indicates that some motor cortical output remains untapped by maximal effort. There is a failure at the supraspinal level to generate all possible motor cortical output even though the ongoing motor cortical output is insufficient to drive the motor units maximally. A summary of changes is given in Fig. 1.

So what causes motor unit slowing during a sustained maximal contraction? A number of mechanisms probably contribute. It is likely that repetitive activation of motoneurons decreases their response to synaptic input. There may also be an increase in recurrent inhibition. For some muscles, small-diameter muscle afferent activity inhibits the motoneurons. In addition, excitation from muscle spindles may be decreased. Although descending drive may not decrease, it fails to compensate for the changes at the motoneuron pool despite the availability of extra motor cortical output.

EMG responses to TMS give some insight into changes in motor cortical activity during fatigue. During a sustained maximal voluntary contraction (MVC) or during briefer repeated MVCs, the short-latency excitatory response (motor evoked potential or MEP) increases in size (98, 99). This growth indicates an increase in excitability of neurons in the motor cortex compared with the reduction in motoneuron excitability indicated by the decrease in the size of the cMEP (15, 99). Imaging studies also show initial increases in motor cortical activity during sustained MVCs although this can later fall (52). The silent period after TMS is an inhibition of voluntary EMG, which can last for more than 200 ms. The earlier part is due to both spinal and cortical mechanisms whereas the latter part (after ~100 ms) results from intracortical inhibition, probably via GABA_B receptors (e.g., 89). During sustained or repeated fatiguing MVCs, the silent period grows longer (98, 99). Although this might suggest an increase in inhibition within the motor cortex itself, paired-pulse TMS of the motor cortex shows fatigue-related decreases in inhibition attributed to both GABA_A and GABA_B receptor activation (5, 6, see also 73). One suggestion is that the termination of the silent period represents excitatory input overcoming inhibition of the motor cortical output cells, so that impaired voluntary excitation might result in lengthening. However, paired-pulse testing was performed with the muscle at rest between maximal efforts, and it is not known whether inhibition is decreased in a similar way during contraction.

Although changes in the EMG responses to TMS and increases in the superimposed twitch both occur during sustained and repeated MVCs (28, 98, 99), they can be dissociated under some conditions. That is, supraspinal fatigue can be present without changes in the MEP or silent...
period. For example, if the arm is held ischemic at the end of a fatiguing elbow flexion contraction, recovery of the muscle is prevented and the firing of small-diameter muscle afferents is maintained (28). With relaxation, activity in the motor pathway stops, allowing the motor cortex and motoneurons to begin to recover from any changes related to repetitive activation. In this situation, the EMG responses to TMS recover quickly, but supraspinal fatigue does not improve until blood flow to the arm is restored. These results suggest that fatigue-related activity in small-diameter muscle afferents may have a role in supraspinal fatigue. However, this is not an effect on motor cortical excitability, nor on the elbow flexor motoneurons (15, 102). Thus group III and IV muscle afferents may act to limit the circuits that generate motor cortical output.

CENTRAL FATIGUE CAUSED BY SUBMAXIMAL CONTRACTIONS

Like maximal efforts, submaximal muscle activity can also lead to central fatigue. With a sustained voluntary contraction of more than ~15–20% maximal force, subjects eventually reach a point of task failure and are unable to generate the target force (e.g., 61). As active muscle fibers become fatigued, subjects incrementally increase voluntary effort to recruit more motor units and/or increase firing rates (e.g., 1, 7, 19) until the task requires a maximal effort. At this moment, despite subjects’ maximal effort, EMG is reduced compared with that recorded during a MVC of the nonfatigued muscle (25, 78). Although some of the reduction in EMG may be due to changes in the muscle fiber action potential or changes in the summation of motor unit potentials to produce surface EMG, neural drive to the muscle is also reduced as motor nerve stimulation shows poor voluntary activation (54).

To track the development of fatigue during a submaximal task and to determine whether central fatigue occurs before the submaximal task becomes maximal, brief test MVCs can be introduced (e.g., 7, 22, 93, 94, 110). Falls in maximal voluntary force show muscle fatigue during sustained contractions as weak as 5% MVC (93). In contractions of 5–30% MVC, increases in the superimposed twitch evoked by stimulation of the motor nerve during the brief maximal efforts indicate that central fatigue develops concurrently with peripheral fatigue (22, 93, 94, 110). Hence, the development of central fatigue does not require high levels of motor cortical output or recruitment of a large proportion of the motoneuron pool. Surprisingly, central fatigue was not seen in the first dorsal interosseous muscle during stronger contractions (45–75% MVC) (22). This could be due to the shorter duration of these contractions (~1–3 min compared with >6 min for the 30% MVC in the same study), although Zijdewind et al. (110) showed progressive central fatigue that was clear after ~3 min of a 30% MVC of the same muscle.

The demonstration of central fatigue with relatively weak contractions brings forward two questions. First, how does sustained weak activity impair subjects’ ability to drive the muscle maximally? Second, does this impairment of maximal neural drive affect performance of the submaximal task?

Mechanisms of central fatigue in brief MVCs during a sustained weak contraction. TMS of the motor cortex has been applied during occasional brief MVCs in weak sustained contractions (5% and 15% MVC) of the elbow flexors (93, 94). In these efforts, the increasing superimposed twitch evoked by TMS demonstrated that supraspinal fatigue also develops progressively (Fig. 2A). At the same time, the MEP increased in size and the silent period lengthened as in sustained or fatiguing intermittent MVCs. These changes were not as pronounced as during protocols with higher levels of activity but suggest that motor cortical changes are qualitatively similar during fatigue caused by weak or strong contractions (93, 94, 98, 99).

Although the behavior of motoneurons in brief maximal efforts during fatigue produced by sustained weak contractions is not known, maximal motor unit firing rates in triceps brachii decrease when fatigue is produced by intermittent contractions of 50% MVC (104). As in fatiguing MVCs, motor unit slowing is consistent with central fatigue, and the same underlying mechanisms need to be considered. However, one might predict that any changes caused by repetitive activation would be confined to those motoneurons recruited in the weak contraction, whereas altered reflex and supraspinal inputs would affect the whole motoneuron pool and might impair voluntary activation in maximal efforts. Suboptimal descending drive, demonstrated by the increase in the superimposed twitches evoked by cortical stimulation, is clearly an important contributor to central fatigue here, with supraspinal fatigue accounting for approximately one-half to two-thirds of the reduction in maximal voluntary force in prolonged weak contractions (93, 94).

If there is an afferent basis for the development of supraspinal fatigue during very weak static contractions, what is the underlying mechanism? Blood flow varies widely to muscles depending on the force requirements and the type of contraction. Static contractions as low as 5% MVC increase local muscle blood flow up to fourfold with only a mild hyperemia on cessation of the contraction (26, 92). Unlike strong contractions, during low-force static contractions there are variations in local intramuscular pressure and presumably in local perfusion and muscle activity (88, 92). Dependent on muscle fiber distribution within arteriolar territories (18), inhomogeneous metabolic changes and ion fluxes will occur. Sustained contractions of 5% MVC in quadriceps increase the arteriovenous difference for K⁺, and it is likely that the interstitial K⁺ concentration is higher around the fibers and able to activate local group IV muscle afferents (91). Global tissue oxygenation does not seem to be a critical factor (at least for weak contractions of biceps brachii) (12). Consistent with increased firing of nociceptive muscle afferents, discomfort increases in the active muscles during weak sustained contractions (93, 94). Activation of muscle nociceptors by intramuscular hypertonic saline reduces maximal voluntary force (34), and in sustained MVCs, they contribute to supraspinal fatigue (28). Thus these afferents may also reduce voluntary activation in brief MVCs during sustained weak contractions.

While there are some common features of central fatigue caused by submaximal and maximal efforts, there are also differences. Supraspinal fatigue forms a larger proportion of the fall in maximal voluntary force during prolonged low-force contractions than during maximal efforts of 1–2 min and might therefore be considered more important in submaximal efforts. The fall in the level of voluntary activation can be as much or more in the intense exercise, but in intense exercise, peripheral fatigue is much greater (37, 94). A further difference lies in
recovery of voluntary activation after the end of the fatiguing protocol. This occurs within 2–3 min after strong contractions but takes >10 min after long-lasting weak efforts, and after prolonged running, voluntary activation is still depressed after 30–60 min (31, 81). One explanation could involve a continued supraspinal influence of small-diameter muscle afferents. While an immediate drop in blood pressure suggests that firing of small-diameter afferents drops quickly after a 2-min MVC, it is unknown whether these afferents continue to fire after more prolonged contractions. However, motor effects at a spinal level, including reflex depression and increased motoneuron excitability, have been demonstrated as long as 15 min to 1 h after 10–15 min activation (40, 50, 67). It is unclear whether these more lasting motor effects are due to central sensitization or continuing afferent activity, but either mechanism could also contribute supraspinally to impaired voluntary drive. Continued (or de novo) afferent firing may be particularly relevant after exercise like endurance running, which results in muscle damage.

Central fatigue in submaximal contractions. Although central and supraspinal fatigue can only be measured during maximal efforts, these fatigue processes are likely to occur in weak as well as strong contractions. However, it is difficult to test the influence of such processes on the performance of a submaximal task when both voluntary drive to the motoneurons and the force-generating capacity of the muscle fibers are changing continuously. With central fatigue in a MVC, less than maximal force is produced by the muscle despite a constant maximal effort. That is, less force (relative to the muscle’s current maximum) is produced for the same effort. Applying this idea to submaximal contractions is problematic. Indeed, it is not clear whether the concept of central fatigue is meaningful in a submaximal contraction.

During a fatiguing submaximal contraction (see Fig. 3), excitability of the motoneuron pool increases progressively. Evidence of increased excitation includes increases in surface EMG and the MEP evoked by transcranial electrical stimulation (e.g., 7, 21, 86). However, other processes occur at the same time as evidenced by various reflex changes reported during and immediately after sustained submaximal efforts (e.g., 21, 47, 55). Together, these processes result in a heterogeneous pattern of output from the discharging motoneurons. Hence, motor unit firing during fatiguing submaximal contractions is complex, with recruitment of additional units and changes in firing rates. In moderate to strong contractions, firing rates tend to decrease or stay the same, whereas in weaker contractions they tend to increase (e.g., 1, 16, 29, 47, 104). However, the behavior depends on the protocol and muscle. In any muscle, the firing of each motor unit must reflect its firing history and intrinsic properties, as well as the more general influences of inputs to the motoneuron pool. In addition to increasing descending drive, reflex inputs change, including falling muscle spindle activity (58), increasing small-diameter muscle afferent activity, and decreasing recurrent inhibition (56, 83).

Under particular conditions, some afferents may be especially influential in modifying motoneuronal output during submaximal contractions. As examples, alternating recruitment...
of different muscles in very weak contractions (≤5% MVC) of the knee extensors and ankle plantarflexors has been attributed to inhibition between mono- and biarticular synergists by muscle spindle activity (46, 97). The shorter endurance time when maintaining the position of an equivalent weight than when holding an isometric force has been attributed to earlier recruitment of higher threshold motor units because of reduced presynaptic inhibition of Ia afferents (60, 62, 72). In humans, some voluntary drive is thought to be conveyed from the motor cortex to motoneurons via propriospinal neurons with cell bodies at the C3–C4 level (80). Inhibition of drive through these neurons by cutaneous afferents is increased to fatigued muscles and decreased to other active muscles during submaximal efforts (64). In sustained isometric contractions, activity of nociceptive muscle afferents acts differentially on higher threshold motoneurons to compress recruitment thresholds across the motoneuron pool (67). This result is an example of nonuniform response of a motoneuron pool to a reflex input whose synaptic strength varies across the pool (43). It could lead to extra motor unit recruitment but lower firing rates in generating a target force (23). For individual motor units, a fall in firing rate during increasing excitation to the motoneuron pool strongly suggests decreased responsiveness to synaptic input. This could result from repetitive activation as discussed above in relation to MVCs (39). However, it is not known whether such a decrease in firing rate leads to a loss of force, and therefore represents central fatigue, because the contractile properties of the motor unit’s muscle fibers will be changing at the same time (14, 16, 17). Furthermore, opposite changes are possible. The force output of muscle fibers can increase (potentiation) and/or decrease (fatigue) with activity (e.g., 32, 105). Contraction and relaxation may slow with fatigue but could speed up for some fibers, particularly with local increases in muscle temperature (e.g., 20, 32, 47). Given that it is extremely unlikely that the neural machinery exists to control the firing rates of every single motor unit based on its own ongoing contractile performance (27), it may not be sensible to think of central fatigue for a single unit. For example, it may be more efficient, in terms of overall descending drive, to produce force by recruiting new units than by continuing to drive fatigued muscle fibers at a high rate.

There are similar problems in identifying central fatigue for the whole muscle. During a prolonged weak contraction, EMG recorded from the elbow flexors increased progressively but quickly decreased when brief contractions of the same force were performed after the sustained contraction (94). Because there was no recovery of twitch force, the change in the EMG-force relationship suggests that the same target force was produced by a different pattern of motor unit firing during the fatiguing task and after a brief recovery (<1 min). Although this finding suggests that processes related to repetitive activation of motoneurons can alter the way a task is performed, it remains unclear whether additional descending drive was required to maintain force during fatigue and thus whether this is an instance of central fatigue.

One aspect of performance does suggest that central fatigue is important in the performance of submaximal tasks. Subjects’ reports of the effort required to maintain a weak target contraction increase disproportionately to the target force and EMG relative to their maxima. During ~40 min of a 15% MVC, subjects initially reported a “mild” effort of just over 2 and this increased to a “very large” effort of ~7.5 from a maximum of 10, whereas the target force and EMG in the active muscles increased to ~30% and ~35%, respectively, of that recorded during a brief MVC at the time (94) (Fig. 2B). A similar mismatch between perceived effort and motor output also occurred during a prolonged 5% MVC (93). Here, subjects also reported effort during brief contractions to the target force made after the end of the 70-min contraction. Within 1 min of the cessation of the sustained contraction and despite the intervening performance of three brief strong contractions,
subjects’ effort dropped by half. It further recovered to prefatigue levels over 10 min. The immediate drop in effort makes it clear that the sustained nature of the contraction is important in producing this effect.

Changes at a motor cortical level during sustained submaximal efforts are suggested by the EMG responses to TMS. The MEP increases along with the ongoing EMG. As both activity in the motor cortex (4, 53) and firing of motoneurons increase, TMS is expected to evoke more or larger descending volleys, as well as additional motoneuron output. The silent period also lengthens. In prolonged low-force contractions (<20% MVC), it lengthens gradually, whereas in high-force contractions, the silent period first shortens, and does not lengthen until subjects are making close to maximal efforts (for review, see Ref. 101). As the silent period following transcranial electrical stimulation does not change, the lengthening represents a cortical effect (86). Inhibition of voluntary activity by very low-intensity (subthreshold) TMS also increases during a sustained submaximal effort (69). In contrast, as in maximal efforts, paired cortical stimulation shows reduced intracortical inhibition after submaximal fatiguing exercise (68). Thus the influence of inhibitory circuitry on motor cortical function during fatigue remains unclear.

CONCLUSION

There are clear contributions to force loss from spinal and supraspinal factors in maximal contractions performed continuously, intermittently, or superimposed on submaximal contractions. These contributions can be substantial, particularly with fatigue due to prolonged weak contractions. It is more difficult to isolate central changes contributing to force loss during weak sustained contractions, although fatigue processes that impair force production in superimposed maximal efforts must continue to operate. A major problem is identifying “ideal” performance of the neuromuscular system in producing submaximal forces during fatigue. However, the progressive mismatch between perceived effort and muscle output in prolonged weak contractions strongly suggests that central processes do impair some aspects of performance of submaximal tasks.

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