

The factors limiting force production and exercise endurance time have been briefly described, together with some of the changes occurring at various sites within the muscle and central nervous system. Evidence is presented that, in fatigue of sustained maximal voluntary contractions (MVC) executed by well-motivated subjects, the reduction in force generating capacity need not be due to a decline in central nervous system (CNS) motor drive or to failing neuromuscular transmission, but can be attributed solely to contractile failure of the muscles involved. However, despite this conclusion, both the integrated electromyogram (EMG) and the mean firing rate of individual motor units do decline progressively during sustained MVC. This, however, does not necessarily result in loss of force since the parallel slowing of muscle contractile speed reduces tetanic fusion frequency. It is suggested that the range of motoneuron firing rates elicited by voluntary effort is regulated and limited for each muscle to the minimum required for maximum force generation, thus preventing neuromuscular transmission failure and optimizing motor control. Such a CNS regulating mechanism would probably require some reflex feedback from the muscle.

CHANGES IN MUSCLE CONTRACTILE PROPERTIES AND NEURAL CONTROL DURING HUMAN MUSCULAR FATIGUE

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Everyone has experienced the sensations of fatigue, and the increasing difficulty of continuing a given level of physical exercise. Neuromuscular fatigue is sometimes defined as "an inability of a muscle or group of muscles to sustain the required or expected force."¹² This terminology has led to the concept that the onset of fatigue is delayed and commences only after a protracted period of exercise. However, much evidence suggests that the physiological events underlying fatigue commence at the onset of activity, although they cannot always be readily detected. For example, if, while a subject is making a sustained or repeated submaximal ef-

fort, one asks him periodically to make a brief maximal voluntary contraction (MVC), the maximum force he can exert declines progressively with time. At the limit of endurance, or the "point of fatigue," the required force (initially submaximal) has become the best the subject can possibly do; i.e., this is now his new, fatigued maximal force generating capacity (Fig. 1). Hence, endurance time declines as the imposed load increases, and is determined by the rate of decline of the MVC. Thus, for our purposes, we define neuromuscular fatigue as *any reduction in the force generating capacity* of the total neuromuscular system regardless of the force required in any given situation.

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ENDURANCE TIME

The threshold for fatigue is that level of exercise which just cannot be sustained indefinitely (here defined as > 45 minutes). Rohmert²⁸ studied the relationship between the force exerted by a muscle during sustained isometric contractions, expressed as a percentage of its maximum capacity, and the time for which that force can be held. For maximal contractions the force starts to fall immediately. For other force levels the endurance time declines roughly exponentially as the force held is increased. He found that this unique relationship ap-

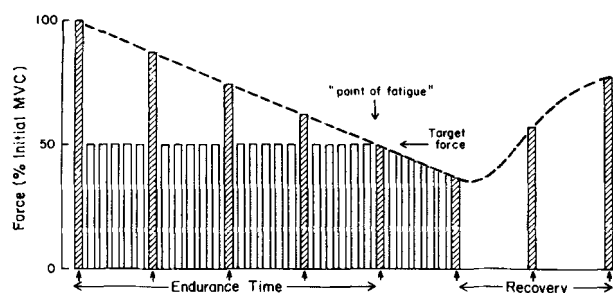


FIGURE 1. Schematic representation of fatigue from intermittent submaximal contractions. Changes in maximum force generating capacity are shown by brief MVC contractions executed periodically throughout the exercise and recovery times.

plied to sustained contractions of a wide range of human limb muscles, all of which demonstrated a fatigue threshold at about 15% MVC (Fig. 2A).

Recently Bellemare and Grassino⁵ have examined this relationship for intermittent contractions of the human diaphragm. They found that endurance time depends not only on the relative force exerted during each contraction but also on the time for which the force is held during each duty cycle; i.e., the force-time integral per breath ($\int FT$). The force-time integral can be calculated under any conditions from measurement of only the mean force exerted and the duty cycle. When breathing against variable inspiratory resistances at a given frequency, they found that fatigue could be induced by either increasing the force of each con-

traction or by increasing the inspiratory time (i.e., the duty cycle). However, no fatigue resulted using any breathing pattern if $\int FT$ (termed the tension time index) was less than 0.15. This relationship thus provides a means of measuring the fatigue threshold for any pattern of intermittent contractions, as shown in Fig. 2B. They also found that whenever $\int FT$ exceeds this threshold the endurance time is limited and can be reasonably accurately predicted in a manner similar to that observed by Rohmert.²⁸ Our preliminary (unpublished) studies suggest that this calculation of the fatigue threshold and the endurance time also holds for fatigue from intermittent contractions of various limb muscles.

EFFECTS OF BLOOD FLOW LIMITATION

When a muscle contracts, the intramuscular pressure rises. If this increases above a critical level it causes progressive restriction of the blood flow and thus limits the rate of energy supply. For sustained isometric contractions the fatigue threshold generally corresponds with the onset of blood flow limitation.¹⁹ The longer endurance times associated with fatigue from intermittent contractions at any force level clearly result from the unrestricted blood flow and reactive hyperemia which partly replenish this supply during each period of relaxation.⁵ The muscle energy demands are roughly proportional to $\int FT$, while its replenishment is a function of the period of relaxation. Hence, the

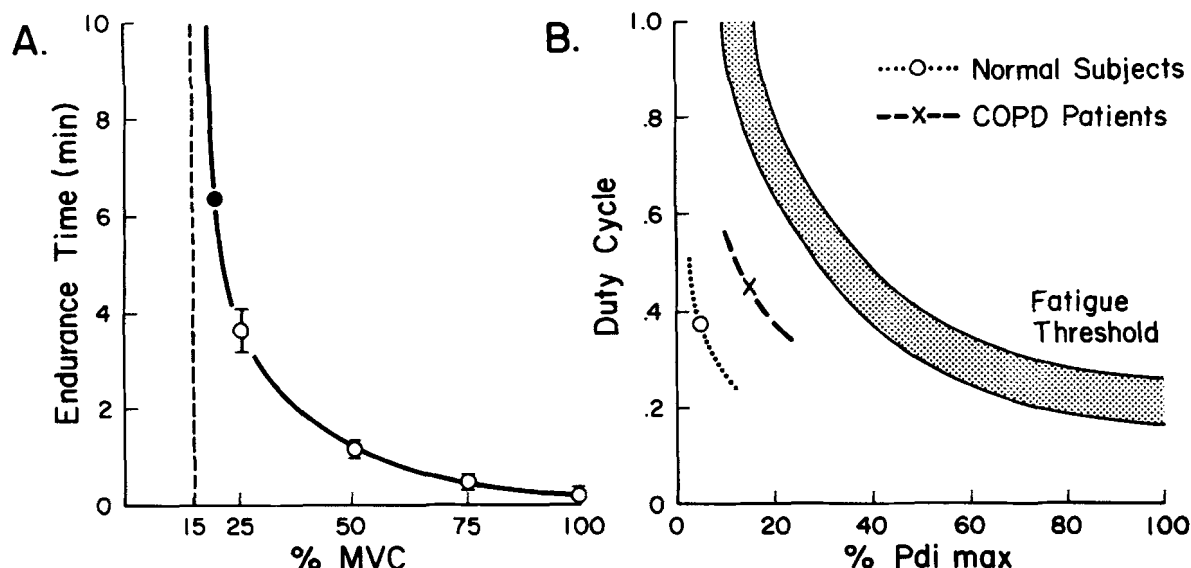


FIGURE 2. (A) Endurance times of sustained isometric contractions of different muscles as a function of the % MVC held. The dotted line indicates the fatigue threshold (Adapted from Rohmert, 1960.²⁸). (B) The fatigue threshold for intermittent contractions of the human diaphragm as a function of % maximal transdiaphragmatic pressure (Pdi) and contraction duty cycle. Values obtained from normal subjects (O) and COPD patients (X) breathing at rest show both have considerable reserves before fatigue is likely to occur (Adapted from Bellemare and Grassino, 1982.⁵).

rate of fatigue is highly dependent on the duty cycle which affects both parameters.

NEUROMUSCULAR FATIGUE

Despite its obvious importance in everyday life, the limitations it imposes on many types of athletic performance, or the critical role it may play in various neuromuscular diseases (including respiratory failure), remarkably little is known of the mechanisms underlying neuromuscular fatigue during human voluntary contractions, or at which of the many possible sites it occurs. Most studies have sought a single cause; yet the generation of a voluntary (or involuntary) contraction involves a sequence of events, any of which may fail.

The various potential sites of failure can be divided into three general categories: those which lie within the central nervous system (CNS), those concerned with neural transmission from CNS to muscle, and those within the individual muscle fibers. Undoubtedly, functional changes occur at all these sites as activity proceeds. Moreover, although muscular fatigue can be measured in terms of reduced force (or work) generating capacity, it is also accompanied by many other measurable changes such as a shift in the electromyogram (EMG) power spectrum, slowing of muscle conduction velocity and muscle contractile speed, and the accumulation of H^+ , lactate, and other metabolites. A major question yet to be answered is which of these events determine performance and which are simply incidental by-products. Moreover, the changes which are rate-limiting may depend on both the particular muscles employed and on the type of exercise from which fatigue results. It is therefore unwise to treat fatigue as one single phenomenon. All sites should be examined under a variety of fatigue-inducing types of exercise. Since each of these major sites are currently thought by different groups^{1,20,23,29} to constitute the major cause of force loss, finding those which are not responsible is as valuable as investigating those that are.

In studies on isolated muscles, stimulated either directly or via their motor nerves, there is general agreement¹⁵ that the force declines whenever the energy demands can no longer be met by the rate of ATP supply; and that this decline of force is increased by the accumulation of metabolites such as lactic acid or by a fall in intracellular pH. The excitability of the muscle membrane and of the t-tubular system may also be impaired if the electrolyte gradients across the cell membrane can no longer be maintained, and K^+ etc. accumulates in

the extracellular spaces. This condition is particularly likely to occur in response to high frequency rates of stimulation, and would then contribute to impairment of excitation/contraction coupling (Ca^{++} release per impulse). These topics have been thoroughly reviewed elsewhere.¹⁵ Henceforth they will be referred to collectively as "muscle contractile failure."

Muscle contractile failure also plays an important role in fatigue induced during human voluntary contractions such as are encountered in normal physical exercise. For example, a progressive reduction in the rate of adenosine triphosphate (ATP) and phosphocreatine supply, together with accumulation of lactate and H^+ , have been amply demonstrated in studies employing nuclear magnetic resonance and muscle biopsy techniques.^{15,30} Impaired excitation/contraction coupling has been implicated as a contributing common factor in fatigue induced by diverse forms of exercise ranging from sustained maximal isometric contractions to bicycle pedaling and bench stepping,¹⁷ and also in respiratory failure attributed to fatigue of the diaphragm muscle.² But its relative importance is still unclear.

However, when considering fatigue during exercise of intact human subjects additional factors operating outside the muscle must also be considered. For example, the force will obviously fall if the motor drive from the CNS declines below the level required for sufficient muscle activation, as may happen if the subject simply lacks motivation or is not prepared to tolerate the increasing sensations of discomfort. This problem is of particular concern when dealing with patients, and is difficult to evaluate objectively. Alternatively, there may be physical limitations to the capacity of the central motor drive system to sustain sufficiently high levels of activity. The force will also fall if the motor drive is prevented from reaching the muscle by failure of peripheral electrical transmission; e.g., the development of neuromuscular block. Our recent work has attempted to answer the question: to what extent is loss of force during fatigue of sustained human MVCs due to failure of the CNS to maintain adequate muscle activation, rather than to failure of muscle contractility per se; and how is the force-generating capacity influenced by changing patterns of neural drive?

FATIGUE OF CENTRAL ORIGIN

The terms "central fatigue" or "fatigue of central origin" refer to conditions in which the decline in force can be related to a reduced motor drive fail-

ing to maintain muscle activation. Traditionally it has been believed that, even in the unfatigued state, the CNS is not capable of recruiting and maximally activating all motor units by voluntary effort. However, in 1954 it was shown that the force of a MVC of the adductor pollicis muscle matched that from supramaximal tetanic stimulation of the ulnar nerve.^{7,23} Recently, Belanger and McComas,³ using a different method, have shown that most human muscles can be maximally activated by voluntary effort. Merton²³ also concluded that for the adductor pollicis full muscle activation could be sustained during fatigue when the contraction was maintained with maximum effort for up to 3 minutes, since maximal nerve stimulation failed to increase the falling force. We have subsequently confirmed this observation many times, but we have found that this maximum force can only be sustained by highly motivated and trained subjects.¹¹ Similar results were also found using the quadriceps muscle¹² stimulated either via the femoral nerve or percutaneously; i.e., maximal stimulation of a constant fraction of the whole muscle. Only for three of the nine subjects studied did the force from maximal voluntary effort consistently decline more rapidly than that from stimulation; and even these subjects were always able to regenerate force to match that from maximal stimulation during brief "extra efforts," which they were unable to sustain continuously. Thus, during maximal contractions lasting for 45–60 seconds central fatigue does not appear to be an insurmountable factor despite up to 50% loss of overall force-generating capacity. However, in exercise of longer duration, declining motor drive may well limit force production, a topic which requires further investigation. Preliminary results suggest that central fatigue need not limit force generation by limb muscles during fatigue of intermittent, submaximal contractions of limb muscles lasting up to 20 minutes.⁹ However, when fatigue is induced in a similar manner in the human diaphragm, reduced CNS motor drive appears to be a major factor.⁴

The concept of central fatigue receives support from several investigations,¹ including those of Ikai et al.²⁰ who found a faster force loss during intermittent MVCs of the adductor pollicis muscle than resulted from brief periods of maximal nerve stimulation. Various methodological factors may have influenced their results. First, it is difficult to make sure each contraction is maximal when executed as fast as one per second, particularly when the muscle is slowed by fatigue and proprioceptive

sensations are impaired. Second, they stimulated the ulnar nerve in the upper arm rather than at the wrist. Substantially greater force was generated by nerve stimulation than by the voluntary contractions, even in the absence of fatigue. This suggests some cross stimulation of additional muscle synergists, which may not have been used during the voluntary efforts and thus remained unfatigued.

FAILURE OF PERIPHERAL NEUROMUSCULAR TRANSMISSION

Even if the motor drive from the CNS remains adequate throughout each contraction, full electrical excitation of all motor units may not be maintained if conduction of the resultant nerve impulses to the muscle contractile mechanism becomes impaired.

Propagation of action potentials along motor axons rarely fails; but when a nerve-muscle preparation is stimulated continuously, failure of propagation between nerve and muscle can easily be demonstrated. It develops at a rate which increases with the stimulus frequency.²¹ This failure may occur presynaptically at nerve terminal branches, postsynaptically from a decrease of endplate excitability, or, less frequently, from a depletion of synaptic transmitter substance.²¹ Since failure of transmission can readily be demonstrated *in vitro* and also during high frequency stimulation of human muscles *in vivo*,^{13,27} and since high rates of maximal motor nerve stimulation are required to match the force of a voluntary contraction, it has often been assumed that neuromuscular block must also be a principal cause of fatigue during voluntary contractions.^{21,27} This, however, may not necessarily be so.

The effectiveness of electrical propagation across the neuromuscular junction and along the muscle surface membrane can be assessed during voluntary contractions by recording from the muscle surface the mass action potential (M wave) evoked by superimposing single maximal shocks to the motor nerve. If neuromuscular transmission or muscle membrane excitability is impaired, a reduction in M wave amplitude and area results. Changes in the ability of the muscle surface membrane to propagate action potentials can be separated from those of neuromuscular transmission by comparing the results of nerve stimulation with those from isolated animal (or human) muscle excited directly in the presence of curare, where no neuromuscular transmission is possible. For the adductor pollicis muscle, Merton²³ observed no decline in the amplitude of the surface M wave

evoked periodically during more than 3 minutes of isometric MVC despite almost total loss of force. He therefore concluded that there had been no failure of electrical transmission at any of these sites.

In 1972, using a similar technique for fatigue of the first dorsal interosseous muscle, Stephens and Taylor²⁹ challenged Merton's²³ conclusions. They found that the surface EMG decreased parallel with the force, and this was accompanied by an apparent decline in the evoked M waves. They therefore concluded that neuromuscular block was indeed the main cause of force loss, at least during the first minute of sustained maximal effort.

In our similar experiments,¹⁴ again on the adductor pollicis muscle, we found no decline in the amplitude of the surface M wave during a 60-second MVC. The total M wave area actually increased somewhat due to a slight slowing of conduction velocity which prolonged the wave form. Similar results were also obtained when using the first dorsal interosseous muscle, so that differences between the underlying fatigue mechanisms of these two muscles, as suggested by Clamann and Broecker,¹⁶ seem unlikely. The discrepancy between our results and those of Stephens and Taylor²⁹ may, however, be due to differences of methodology.¹⁴

Perhaps the most convincing evidence for the maintenance of full muscle activation and the integrity of neuromuscular transmission comes from the report of Merton et al.²⁴ that the loss of force during a sustained MVC cannot be restored by massive direct stimulation of the adductor pollicis muscle fibers themselves. The stimulus currents used were so great that even conduction over the muscle surface membrane may have been bypassed. Moreover, Merton et al.²⁴ recently found no decline in the muscle mass action potential evoked by periodic stimulation of the human motor cortex during fatiguing MVCs. These two important observations seem to demonstrate conclusively the absence of any physiological mechanism limiting electrical propagation during fatigue at any site in the motor pathway, either within the CNS or peripherally.

SUMMARY

In general, the available evidence indicates that, in normal human subjects, reduced muscle activation by the CNS is not an insurmountable factor limiting force generation during fatigue from sustained MVCs of limited duration. However, more work is required using additional muscle groups

and different forms of exercise before a general conclusion can be reached. Nor does it imply that a reduction in central drive is not a major factor in determining reduced performance in everyday life, competitive athletics, labored breathing, or in various pathological conditions. However, in those situations in which it can be demonstrated that the CNS continues to provide adequate muscle activation, and this is not blocked by failure of peripheral transmission, all motor units must continue to respond with a fully fused tetanus. The force loss can then be attributed solely to failure of contractile processes within each muscle fiber.

Measurement of Motor Drive. Changes in the degree of muscle excitation by the CNS can be assessed by recording the integrated EMG (IEMG) from the surface of a muscle. During a series of brief, non-fatiguing submaximal contractions of graded intensity, the IEMG increases progressively with the force exerted. The IEMG/force relationship is not necessarily linear, but appears to be unique and repeatable for each muscle.^{8,31} Thus, the IEMG, when expressed as a percentage of that seen in a maximal contraction, provides an index of the degree of muscle excitation by the CNS.

When a submaximal contraction is held at constant force, or regularly repeated at the same intensity until the limit of endurance, the surface IEMG increases progressively. This has generally been attributed to the recruitment of additional motor units and to increases in their firing rates, a process required to counteract the increasing muscle contractile failure of those fibers already active. However, part of this increase may be due to both a slowing of muscle conduction velocity which increases the duration and area of each muscle fiber potential recorded, and also to increasing synchronization between the discharge rates of different motor units. Both these processes tend to increase the surface recording but do not necessarily reflect any increase in muscle excitation.^{8,25} These factors should therefore be taken into account before changes in the IEMG can be directly related to changes in motor drive. In contrast, during a sustained maximal contraction the surface EMG declines roughly in proportion to the force.^{11,13,29}

ADAPTATION OF NEURAL CONTROL TO OPTIMIZE FORCE PRODUCTION DURING FATIGUE

Stimulation Experiments. In 1979, we suggested that the decline in the surface IEMG recorded during a sustained MVC might result from a progres-

sive reduction in motoneuron discharge rates as fatigue develops.^{13,15} This suggestion was based on the observation that none of the characteristics of fatigue seen during a sustained MVC could be reproduced when the nerve was stimulated at any constant frequency. Prolonged stimulation at 50–80 Hz, the minimum rates required to match the initial force of a MVC, resulted in too rapid a loss of force, which was clearly due to failure of peripheral electrical propagation (M wave reduction). Reducing the stimulus frequency after a period of stimulation restored the M wave and increased the force. The rate of force loss, the typical decline in IEMG, and the well-maintained M wave seen in a sustained MVC could be well imitated if the muscle was initially stimulated at high rates and then the stimulus rate progressively reduced from 80 to 20 Hz. Thus, a reduction in excitation frequency may serve to minimize force loss (rather than cause it) by preserving electrical transmission in fatigued muscle.

When isolated curarized mouse muscles were stimulated directly in the same manner, the force and action potential changes were similar to those seen in human muscle stimulated via the motor nerve.¹⁵ In the isolated muscle, a reduction of the

extracellular $[Na^+]$ or increases in $[K^+]$ the bathing medium reduced propagation and increased the rate of force loss in the same way as increasing the stimulus frequency. We suggested^{13–15} that the loss of force during high-frequency fatigue may not be entirely due to neuromuscular block, but also, in part, to a reduced excitability of the muscle fiber membrane due to changes in the extracellular electrolyte composition which undoubtedly occur whenever the excitation frequency remains high.

Changes of Single Motor Unit Firing Rates. Using conventional recording electrodes, the firing rates of individual human motoneurons during high force voluntary contractions have seldom been measured with confidence because the signals are generally contaminated by interference from other surrounding active units (Fig. 3A). We have recently succeeded in recording uncontaminated single unit potentials from several muscles of normal human subjects during MVCs using tungsten microelectrodes.^{6,10} Their amplitude, duration, and shape showed them to be action potentials from single muscle fibers (Fig. 3B and C). In brief non-fatiguing maximal contractions the average firing rate of more than 300 units recorded from the

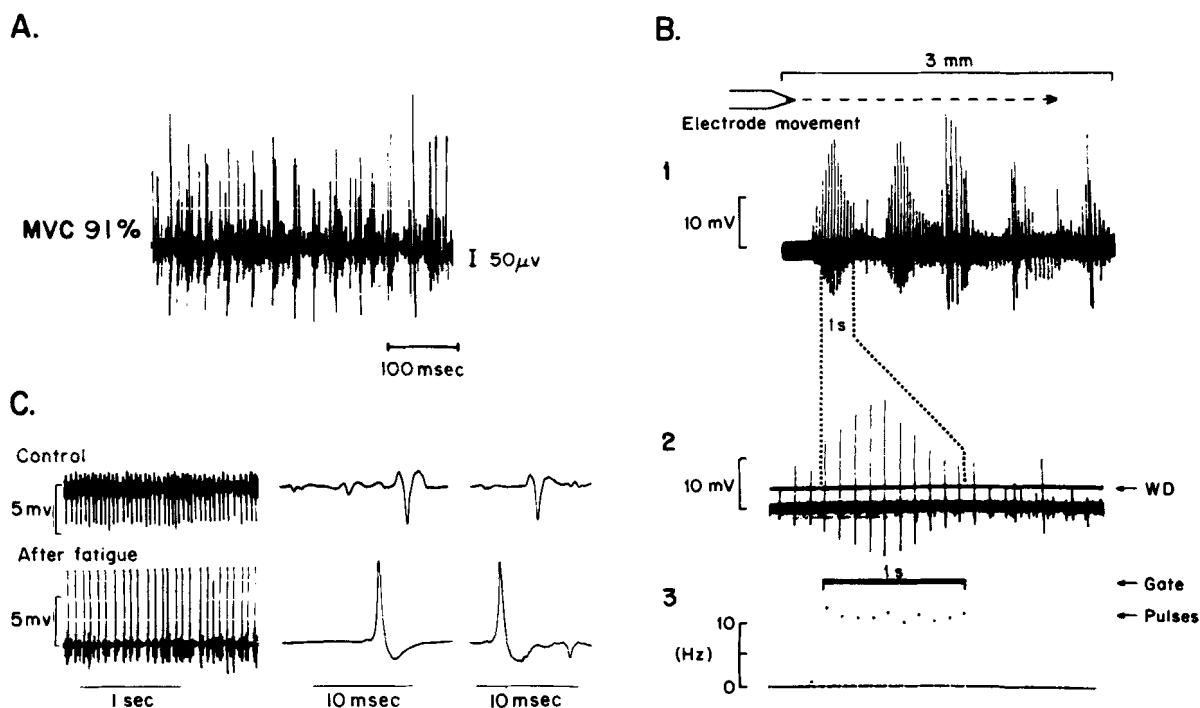


FIGURE 3. (A) Motor unit potential recorded during high force contractions using conventional electrodes. (B) 1) Trains of potentials recorded from different muscle fibers as a tungsten electrode is advanced through muscle during MVC contraction (soleus). 2) High speed display of the train just described. 3) Pulses displayed through window discriminator (WD above) showing regularity of intervals and mean frequency. (C) Trains of potentials recorded from adductor pollicis muscle before and after fatigue, with the wave forms of individual spikes from each train.

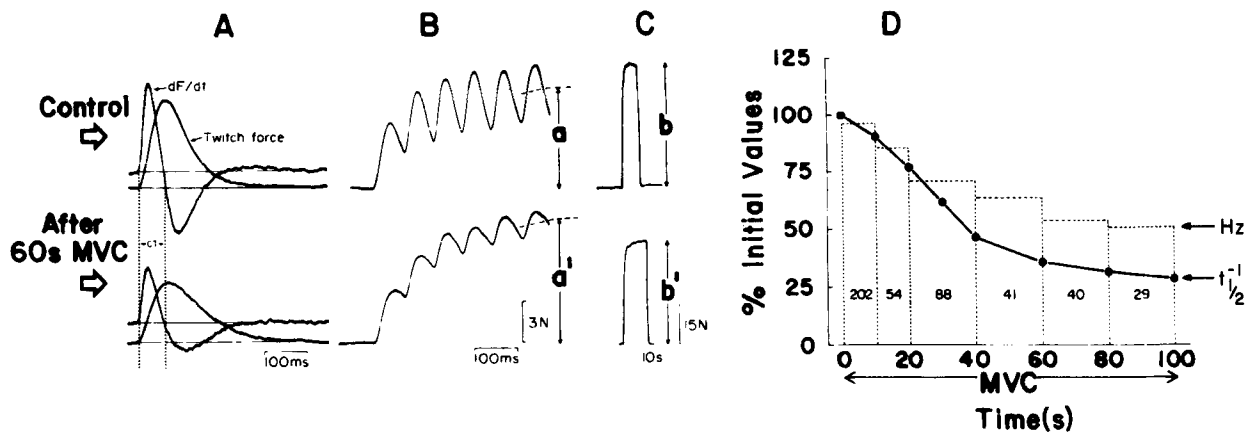


FIGURE 4. (A) Twitches, (B) unfused tetani (7 Hz), and (C) 50-Hz tetanic responses from adductor pollicis muscle before and after fatigue. In A the force record has also been differentiated (dF/dt) for measurement of contractions and relaxation times. (D) Changes in relaxation rate ($t_{1/2}^{-1}$) during 100 seconds of sustained MVC superimposed on corresponding changes in mean motoneuron firing rates (Hz).

adductor pollicis muscles of five subjects was 29.8 ± 6.4 Hz (mean \pm SD). During prolonged maximal effort, force and firing rates always declined. Between 30–60 seconds and 60–90 seconds after the onset of each contraction the rates were 18.8 ± 4.6 Hz ($n = 78$) and 14.3 ± 4.4 Hz ($n = 62$), respectively. The percent decline in mean motoneuron firing rate paralleled, and appeared to account for, that of the surface EMG recorded simultaneously.^{10,11}

These results, therefore, provide direct evidence for a reduction of motoneuron firing rates during this type of fatigue. Why then does this not cause loss of force?

Contractile Speed and EMG Changes During Fatigue.

It is well established that fatigue is characterized, not only by loss of force, but also by slowing of the muscle contractile speed. The frequency with which successive twitches summate and tetanic fusion occurs depends on the total time required for each twitch contraction and relaxation.¹⁸ Thus, for any muscle or motor unit the minimum excitation frequency required to generate maximum force and tetanic fusion is proportional to its contractile speed. In twitches recorded before and immediately after a 60-second MVC of the adductor pollicis muscle we found¹¹ no significant change in twitch contraction time (CT), but relaxation time was prolonged by about 50% (Fig. 4A). When stimulated at a given subtetanic rate (e.g., 7 or 10 Hz), after fatigue the degree of summation increased, as did the fraction of the remaining tetanic force (Fig. 4B and C). Figure 4D shows the percent reduction in relaxation rate recorded during a sustained

MVC superimposed on the corresponding changes in single motor unit firing rates. Since both decline roughly in parallel, the reduction in motoneuron firing rates need not necessarily change the degree of tetanic fusion; that is, force loss is due only to muscle contractile failure and is not affected by the reduction in excitation rate. This, in turn, suggests some reflex CNS mechanism which may regulate motoneuron firing rates to match changes in muscle contractile speed.

Motor Unit Discharge Rates in Muscles With Different Contractile Properties. To determine whether, in other situations, motoneuron firing rates are also regulated to match differences of contractile speed, the frequencies of single motor unit potentials were recorded, during nonfatiguing maximal voluntary contractions, from the biceps brachii and soleus muscles chosen because of known differences in their fiber type composition and contractile speeds. The results were compared with differences in their respective twitch contraction and relaxation times.⁶

For the biceps brachii and adductor pollicis muscles, the MVC mean firing rates (\pm SD) recorded from about 300 units in each muscle were 31.1 ± 10.1 Hz and 29.9 ± 8.6 Hz, respectively, while for the soleus muscle they were only 10.7 ± 2.0 Hz. The biceps and adductor pollicis muscles had similar twitch contraction and half-relaxation times (60–70 msec), while for soleus these were each about 50% longer; these values agree well with those found for single fast and slow twitch human motor units, respectively.¹⁸ Thus, the mean firing rates for each muscle varied roughly in pro-

portion to the differences between their intrinsic contractile speeds. This finding provides further support for the notion that, during maximal voluntary contractions, motoneuron firing rates are geared to match the contractile requirements of the individual muscles they supply.

Functional Consequences. These results define the upper limit of motoneuron firing rates that can be sustained by voluntary effort. They also suggest that in any muscle these firing rates, either before or after fatigue, do not exceed the minimum required for maximum force production. In either condition, once maximum force is generated no useful purpose is served by further increases in excitation frequency.

Figure 5A and B shows the range of motoneuron firing rates recorded from the human adductor pollicis muscle during maximum efforts before and after a 60-second MVC, together with the corresponding relaxation rates. The force generated when the motor nerve is stimulated at comparable rates is shown in Fig. 5C. To generate maximum tetanic force in the unfatigued muscle, supramaximal shocks at 50–80 Hz must be delivered. Yet the same force can be produced in an MVC where the

mean motoneuron discharge rate is only 30 Hz. Motor nerve stimulation at 30 Hz results in 85% of this force. Thus, the remaining 15% of the force is probably generated by those units which fire at rates from 35–50 Hz; presumably those of highest recruitment threshold and fastest contractile speed. It also seems likely that those with the lower discharge rates (15–25 Hz) are the slowest, low threshold units with tetanic fusion rates similar to those observed in the slow soleus muscle. After fatigue and contractile slowing, the mean motoneuron discharge rate declines to about 15 Hz. The now reduced maximum force still matches that from supramaximal tetanic nerve stimulation. Thus, after fatigue, if the same argument is applied, the new relationship between excitation frequency and relative force generation for the muscle as a whole has shifted toward the lower frequency range (see dotted line, Fig. 5C).

During fatigue the slowing of muscle contractile speed shifts the force/frequency relation towards the lower frequency range. This allows all motor units to remain fully activated despite a substantial reduction in motoneuron discharge rates. If the initial frequency required to generate full force in the unfatigued muscle was maintained, it would soon become markedly supratetanic after contractile slowing. As pointed out earlier, the reduction in discharge rate probably provides a safeguard against failure of neuromuscular transmission. More importantly, it must also serve to optimize force regulation by limiting the range of discharge rates to correspond closely to those in which force production can be modulated. This can only occur if the range of discharge frequencies available to each motor unit is limited to those which correspond to the steeper parts of the force/frequency response curve (Fig. 5C). These must vary between different motor units according to the contractile properties of their individual constituent muscle fibers. Also, since these properties change with fatigue, a regulatory mechanism must exist within the CNS to match the motoneuron discharge rates to the changing contractile speed of the motor units they supply. Such regulation would presumably require some sensory feedback from the individual muscles innervated.^{8,11,15} Such a mechanism may be particularly important for the small hand muscles such as the first dorsal interosseous and adductor pollicis, where all motor units are thought to be recruited in contractions of less than 50% MVC,^{22,26} above which rate-coding becomes the sole means for further force regulation.

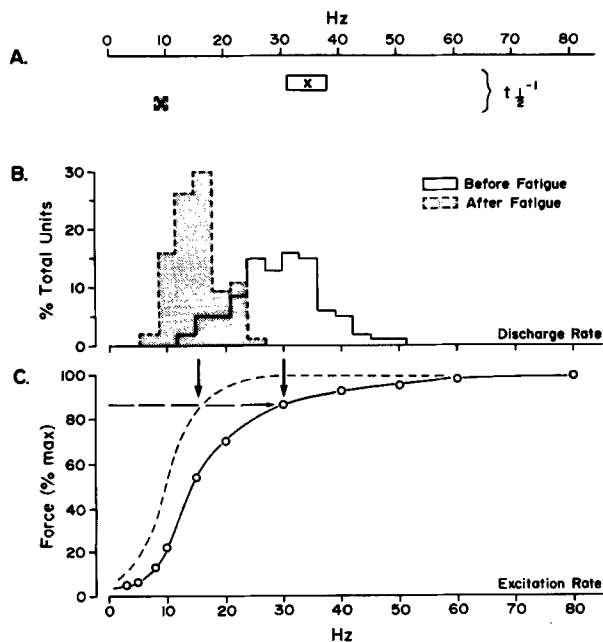


FIGURE 5. Adductor pollicis measurements at onset and termination of 60-second MVC. (A) Relaxation rates, $t_{1/2}^{-1}$ (mean \pm SD). (B) Motoneuron firing rates. Arrows show mean rates recorded. (C) % Maximum force generated by nerve stimulation before fatigue (solid line) and predicted after contractile slowing (dotted line).

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