Distinct Patterns of Motor Unit Behavior During Muscle Spasms in Spinal Cord Injured Subjects

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Thomas, C. K. and B. H. Ross. Distinct patterns of motor unit behavior during muscle spasms in spinal cord injured subjects. J. Neurophysiol. 77: 2847–2850, 1997. Surface electromyograms (EMG) and force were recorded during repeated involuntar y spasms of paralyzed triceps surae muscles of four men with chronic cervical spinal cord injury. The firing rates of 78 medial gastrocnemius (MG) motor units also were recorded intramuscularly with tungsten microelectrodes. Spasms typically involved a relatively rapid rise, then a more gradual fall in triceps surae EMG and torque. Motor unit firing rates either increased and then decreased with the spasm intensity (54%) or were relatively constant (26%), firing mainly at 2–10 Hz. The remaining units (20%) produced trains that included one or several doublets. Mean peak spasm firing rates were 18 ± 9 Hz (mean ± SD) for rate modulated units and 11 ± 10 Hz for units with little or no rate modulation. Some motor units fired at rates comparable with those recorded previously during maximum voluntary contractions performed by intact subjects. Others fired at rates below the minimum usually seen when normal units are first recruited (<6 Hz). Doublets (interspike interval <10 ms) often repeated every 123–333 ms, or were interspersed in trains firing at low steady rates (<11 Hz). This study shows that rate coding for many motor units appears to be similar whether descending motor input is intact or whether it has been reduced severely by spinal cord injury. In contrast, rate modulation in other units appears to depend mainly on voluntary motor commands.

METHODS

Individuals with chronic (>1 yr) cervical SCI who lacked voluntary control of trunk and leg muscles as assessed by manual muscle evaluation, whose paralyzed triceps surae muscles were prone to contract involuntarily, and who took no medication to dampen spasms were chosen as subjects. Ethical approval and informed written consent were obtained. The leg most susceptible to spasms was studied. During an isometric ramp voluntary contraction of control muscle, both force and electromyograms increase because additional motor units are recruited and/or because there is an increase in the rate at which active cells fire. These motor units usually fire at 6–10 Hz when first recruited (Monster and Chan 1977) and at 15–60 Hz during maximum voluntary contractions. Rates also vary between units, between muscles, and between subjects (Bellemare et al. 1983). Such rate modulation is considered to depend largely on the intensity of descending motor drive reinforced by input from local and afferent inputs (Binder et al. 1996). Thus motor unit firing rates are reduced during both maximal and submaximal voluntary contractions after afferent feedback has been temporarily blocked by local anesthesia (Gandevia et al. 1990) or when conduction in central nervous system motor pathways is impaired, as in stroke or multiple sclerosis (Gemperline et al. 1995; Rice et al. 1992).

Involuntary muscle spasm is common in muscles paralyzed by spinal cord injury (SCI; muscles where voluntary EMG is absent). Many subjects can trigger spasms at will by trivial stimuli such as light touch or a minor postural shift. These spasms thus would seem to provide a unique opportunity to examine the extent of motor unit rate modulation in muscles to which maximal voluntary drive fails to produce EMG. To our knowledge, no investigations have monitored motor unit activity during such involuntary spasms. The aim of the current study was to examine motor unit firing rates during spasms of medial gastrocnemius muscles paralyzed chronically by SCI. With largely afferent inputs and spinal circuits to contribute to the excitation of the medial gastrocnemius (MG) motor pool, one might expect to record lower than normal motor unit firing rates when these muscles contract involuntarily. In fact, considerable rate modulation was recorded in approximately half the MG motor units. These findings suggest that inputs that remain years after traumatic injury to the human cervical cord are sufficient to excite one group of spinal MG motoneurons, but that others rely mainly on excitatory drive from higher brain centers for rate modulation. Some of these data have been published as an abstract (Ross and Thomas 1994).

INTRODUCTION

During an isometric ramp voluntary contraction of control muscle, both force and electromyograms increase because additional motor units are recruited and/or because there is an increase in the rate at which active cells fire. These motor units usually fire at 6–10 Hz when first recruited (Monster and Chan 1977) and at 15–60 Hz during maximum voluntary contractions. Rates also vary between units, between muscles, and between subjects (Bellemare et al. 1983). Such rate modulation is considered to depend largely on the intensity of descending motor drive reinforced by input from local and afferent inputs (Binder et al. 1996). Thus motor unit firing rates are reduced during both maximal and submaximal voluntary contractions after afferent feedback has been temporarily blocked by local anesthesia (Gandevia et al. 1990) or when conduction in central nervous system motor pathways is impaired, as in stroke or multiple sclerosis (Gemperline et al. 1995; Rice et al. 1992).

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The person sat with the knee flexed 60° (full knee extension, 0°), the ankle in the neutral position, and the foot strapped to a plate beneath which was a force transducer to register isometric spasm strength. Bipolar EMG was recorded from the surface of MG, lateral gastrocnemius (LG) and soleus. For MG and LG an active electrode (2 cm diam) was placed on each muscle belly. For soleus, this electrode was on the muscle midline below the gastrocnemius outline. Indifferent electrodes were placed on the distal tendon. Intramuscular EMG was recorded from MG using a monopolar tungsten electrode (Hagbarth and Vallbo 1969). A leg spasm was induced by a minute change in posture (e.g., hip angle) or light brief touch of the skin on the side of the test knee. In each experiment, two to five spasms were evoked 5–10 min apart, most lasting <30 s. The needle electrode was inserted into MG and moved slightly within the muscle during a spasm until a train of potentials could be recorded from a motor unit, which then was held as long as possible (Fig. 1). Between spasms the electrode was repositioned in MG to ensure sampling from different motor units. Each person performed three such experiments, 1–8 days apart (mean: 5 days). Another experiment was performed on each subject to confirm triceps surae muscle paralysis. Subjects made
FIG. 1. Distinct patterns of involuntary medial gastrocnemius motor unit activity recorded during same spasm (A and B) or a different experiment (C) performed by 1 subject. Insets: superimposed traces of consecutive potentials of units identified. Note 2 units are shown in A, 1 of which has a linked potential (right). Large potentials have been truncated (B) to show smaller potentials. Dips in torque probably reflect activation of muscles other than triceps surae.

RESULTS

Data were recorded from four men (26–39 yr) who had chronic SCI (6–20 yr) at C5 or C6 from diving accidents. All the test triceps surae muscles were paralyzed in that each scored zero on manual muscle evaluation, and no surface EMG was recorded during attempted voluntary contractions or when high-intensity magnetic cortical stimulation was delivered during these voluntary efforts.

Torque and MG surface EMG during spasms recorded from one person on two different days are illustrated in Fig. 1, A and C. The EMG and torque typically increased relatively rapidly and then declined more gradually. Mean spasm rise and fall times were 3.0 ± 1.5 s and 11.2 ± 10.3 s, respectively (n = 33). Peak spasm torque was 13 ± 7 Nm (n = 13) which was <20% of that evoked by 20-Hz stimulation of soleus muscles paralyzed by chronic SCI (Shields 1995). Thus the present spasms of paralyzed triceps surae muscles were weak.

Distinct patterns of motor unit activity recorded during spasms are illustrated in Fig. 1, A–C, each of which shows (top to bottom): torque, rectified MG surface EMG, superimposed sequential wave-forms from identified MG units to illustrate their unitary nature, instantaneous firing frequencies recorded during each 2 s for all motor units that increased or decreased rate with changes in spasm strength or showed little or no rate modulation (Fig. 2B). Torque and MG surface EMG during spasms recorded

Irrespective of firing pattern, units with high mean rates were generally recorded near spasm peak intensity (taken as time zero). Mean rates within 1 s on either side of time zero were 18 ± 9 Hz (Fig. 2A) and 11 ± 10 Hz (Fig. 2B). The
Spinal motoneurons fire in response to voluntary commands from higher motor centers and also when excited by local inputs. During maximum voluntary contractions of neurologically intact subjects, motoneuron discharge rates generally range from 15 to 60 Hz (Bellemare et al. 1983). However, Gandevia et al. (1990) have shown that, when motoneurons are deprived temporarily of afferent input by local anesthesia of their motor nerve, maximum voluntary efforts fail to elicit motoneuron firing rates >20 Hz. Thus it appears that normal maximum firing rates can be achieved only when the excitability of motoneurons is enhanced by facilitation from muscle spindles (Hagbarth et al. 1986) and/or other afferent sources. The motor unit firing rates recorded here during spasms of paralyzed triceps surae muscles show what occurs in the reverse situation. That is, afferent and spinal input is intact but the descending voluntary motor drive is reduced to the extent that no palpable contraction or surface EMG was recorded during attempted voluntary contractions of triceps surae muscles or when high-intensity cortical stimulation was given during these voluntary efforts. The changes in surface EMG recorded during spasms of paralyzed triceps surae muscles and the rate modulation behavior of 54% of the MG motor units were similar to those seen in weak voluntary contractions (Monster and Chan 1977). Although some of this rate modulation may reflect alterations in the number, distribution, and effectiveness of synapses with chronic paralysis and/or adoption of fast-type motor unit properties (Cope et al. 1986), such adaptations may not be universal (Thomas 1997). Removal of descending input also lowered the minimum triceps surae unit firing rate to 2 Hz (Fig. 2A). Other triceps surae motor units fired doubles, possibly a reflection of delayed depolarization in motoneurons, as suggested for doubles recorded during weak voluntary contractions (Bawa and Calancie 1983). The remaining triceps surae motor units did not change their discharge rate with torque, behavior not seen during ramp-force voluntary contractions. The present data suggest that afferent input, spinal systems, and any other inputs that remain but that are not accessible to voluntary drive or high-intensity cortical stimulation can induce substantial rate modulation and doubles in a subset of MG motoneurons. The negligible firing rate changes in other motor units, despite changes in EMG and torque, suggest these cells depend more on voluntary drive and/or other afferent inputs for rate modulation.

The authors thank Dr. Marc Binder for constructive comments, Dr. Tucker and A. Del Valle for help during experiments, and Dr. Klose for statistical advice.

This research was funded by the United States Army Research Office Grant DAAH04-94-G-0425, The Miami Project to Cure Paralysis, the New Zealand Neurological Foundation Grant 35-341, and the University of Otago.

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Received 19 August 1996; accepted in final form 19 December 1996.

REFERENCES


Received 19 August 1996; accepted in final form 19 December 1996.


