EMG Changes in Human Thenar Motor Units With Force Potentiation and Fatigue

C. K. Thomas, R. S. Johansson, and B. Bigland-Ritchie

INTRODUCTION

For a muscle to develop tension, its surface membranes have to be depolarized, a process that is reflected in the EMG activity. Studies in various mammals have shown that changes in EMG generally do not parallel changes in force. For example, the twitch force from most cat motor units increases significantly in response to brief periods of high-frequency stimulation, with little or no change in EMG potentials (Burke et al. 1973; Dum and Kennedy 1980; Kernell et al. 1975). Likewise, during muscle fatigue, the EMG may not change, may increase, or may decline for units that show large or intermediate force declines. In units that are more fatigue resistant, either no change or potentiation of EMG may accompany the decline in force (Burke et al. 1973; Celichowski et al. 1991; Enoka et al. 1992; Gardiner and Olha 1987; Hamm et al. 1989; Sandercoc et al. 1985). Furthermore, changes in EMG potentials, when present, usually recover before the force is restored (Sandercoc et al. 1985). In summary, these data suggest different processes underlie the changes in EMG and force that occur during human thenar motor unit activity.

Motor unit EMG and force in human muscles at the level of single motor units. It is difficult to follow the activity of the same motor unit during prolonged contractions that involve strong forces. Thus most previous studies on long-term changes in EMG and force have been restricted to weak voluntary contractions and to changes in either the force or the EMG of low threshold units rather than on the relationships between these parameters (e.g., Carpenter et al. 2001; Christensen and Sjøgaard 1999; Nordstrom and Miles 1990; Stephens and Usherwood 1977). However, simultaneous recording of single motor unit force and EMG during weak and strong contractions have been made in spinal cord–injured individuals in whom only a few motor units in a muscle remained under voluntary control. In repeated voluntary contractions that elicited fatigue in these motor units, the unit EMG was either well maintained or declined (Thomas and del Valle 2001).

Intraneural stimulation of individual motor axons in healthy humans (Westling et al. 1990) has been applied in one previous study to examine the force-EMG relationship. Fuglevand et al. (1999) report that the EMG potentials of motor units in intrinsic and extrinsic hand muscles were unchanged despite depression of force. In contrast, using trains of pulses at 40 Hz and stimulation of the median nerve through the skin, Chan et al. (1998) found that the amplitude and area of the evoked potentials from thenar motor units more than doubled for fatigable units. Fatigue resistant units showed little change in EMG.

Using intraneural stimulation, we have previously characterized single human thenar motor units with respect to their potentiation of twitch force and fatigue of tetanic force (Thomas et al. 1990, 1991a,b; Westling et al. 1990). In this study, we analyze the changes in unit EMG that accompany these changes in force.

METHODS

Single motor unit EMG and force were recorded from the thenar muscles of 12 healthy subjects in response to intraneural stimulation of their motor axons in the median nerve at the level of the upper arm (Westling et al. 1990). All subjects gave their written informed consent to participate in these experiments, conducted in accordance with the Declaration of Helsinki.

Experimental setup

As described previously (Westling et al. 1990), each subject reclined in a dental chair with the right forearm supinated, extended, and resting in a vacuum cast supported by a platform (Fig. 1). Clay was molded to the shape of the hand to stabilize it, and U-shaped metal clamps were pressed into the clay to restrain the fingers. Three electrodes made of braided
Baseline fluctuations from the pulse pressure and respiration were minimized in two ways (Westling et al. 1990). First, using signals from the pulse detector, all single stimuli and trains of stimuli were delivered to the thenar motor axons during periods when the force baseline remained relatively flat (typically 50–100 ms after peak pulse pressure). Second, the force baseline was reset electronically to a defined level just before the delivery of single stimuli or just before the first stimulus in a train of pulses.

Data collection and analysis

EMG recorded from the distal and proximal surfaces of the thenar muscles, and the abduction and flexion forces were each sampled on-line at 3,200 and 400 Hz, respectively, using SC/Zoom (Physiology Section, IMB, Umeå University, Sweden). All data analyses were done off-line. The vector sum of the measured abduction and flexion force components represented the magnitude of the evoked forces.

Up to 10 twitches recorded before and after the series of pulse trains at different tetanic frequencies were averaged and are referred to as initial and potentiated twitches, respectively, because these tetanic stimuli increased the twitch force significantly (Thomas et al. 1990). We characterized EMG potentials obtained from both the distal and the proximal channels by amplitude, duration, and area. The sum of the durations and areas measured for the first and second phase of an EMG potential (as defined by isoelectric crossings) represented the duration and area of the potential, respectively. EMG amplitude was calculated as the peak-to-peak voltage (Fig. 2A). The corresponding EMG parameters and the peak twitch force of each unit obtained after stimulation at different frequencies were normalized to the respective initial values to show the changes in twitch EMG and force induced by this stimulation.

During the fatigue protocol, the EMG and force responses from five trains of pulses were averaged every 20 s (i.e., trains at 0–4 s, 18–22 s, etc.) to examine the changes in these signals over time. The amplitude, duration, and area of the first and last EMG potentials in these averaged responses were analyzed for both the distal and proximal EMG signals to evaluate changes across (long-term) and within trains (short-term) because decrements in EMG may result in force declines. Peak force was also measured.

Time-dependent changes in the EMG amplitude, duration, and area for the first potentiation in the train were calculated by normalizing the values at 20, 40, 60, 80, 100, and 120 s to the respective values at 0 s. Changes in the parameters of the last EMG potential in these same trains were assessed by normalizing the values to the respective parameters for the first EMG potential at 0 s. Thus at 120 s, these data represent the changes in EMG parameters after the delivery of 1,560 pulses. Fatigue (force decline) was calculated by dividing the peak force measured every 20 s by the force measured at 0 s.

Statistics

Separate repeated measures ANOVAs with EMG channel (distal vs. proximal) and test point (initial vs. potentiated) as factors were used to compare EMG amplitude, duration, and area before and after tetanic stimulation at frequencies between 5 and 100 Hz. Likewise, repeated-measures ANOVAs with EMG channel (distal vs. proximal) and train number (7 levels corresponding to 7 times) were used to assess EMG changes during the fatigue protocol. One-way repeated-measures ANOVAs were used to examine effects of the tetanic stimulation and the fatigue protocol on twitch forces. Tukey HSD tests were used in post hoc analyses that involved pairwise comparisons of data. Relationships between parameters were analyzed using Pearson or product-moment correlations. Use of other statistical analyses is indicated in the Results section. Mean ± SD values are given, and the threshold for statistical significance was P < 0.05.
RESULTS

Presented here are EMG data from single human thenar motor units previously characterized with respect to potentiation of twitch force and fatigue of tetanic force (Thomas et al. 1990, 1991a,b; Westling et al. 1990). We also address the changes in unit EMG that accompany the force changes, focusing on the 23 units that were subjected to brief trains of stimulation at frequencies between 5 and 100 Hz (Thomas et al. 1991a) and to fatigue (Thomas et al. 1991b).

Figure 2A shows the EMG and twitch force recorded from a single thenar motor unit when single pulses were first delivered to its motor axon. These pulses were followed by a series of trains of pulses delivered at frequencies from 100 to 5 Hz (Fig. 2B) and by more single pulses (Fig. 2C). For this unit, the twitch force increased by 94% after these trains of stimuli compared with the force of the initial twitches, whereas there were only small changes in the corresponding EMG potentials. Overall, the potentials recorded from the distal muscle surface were similar in shape to those from the proximal muscle surface, but typically larger in amplitude. Furthermore, the distal and proximal EMG was of opposite polarity for 22 of the 23 units studied (96%). This suggested that the end plates of most motor units lay near the center of the muscles.

Changes in unit EMG with twitch force potentiation

Brief periods of stimulation at frequencies between 5 and 100 Hz caused significant increases in EMG amplitude (P < 0.05), twitch potentiation

<table>
<thead>
<tr>
<th>Amplitude, µV</th>
<th>Range</th>
<th>Duration, ms</th>
<th>Range</th>
<th>Area, µVs</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Distal EMG</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Proximal EMG</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Force</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Twitch potentiation</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Initial</td>
<td>149 ± 108</td>
<td>19–497</td>
<td>9.1 ± 2.2</td>
<td>6.0–15.0</td>
<td>0.34 ± 0.21</td>
</tr>
<tr>
<td>Potentiated</td>
<td>160 ± 129</td>
<td>21–623</td>
<td>9.6 ± 2.4</td>
<td>6.1–15.6</td>
<td>0.37 ± 0.25</td>
</tr>
<tr>
<td>% initial</td>
<td>105 ± 7</td>
<td>93–125</td>
<td>105 ± 4</td>
<td>97–116</td>
<td>109 ± 8</td>
</tr>
<tr>
<td>First potential</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>at 0 s</td>
<td>168 ± 136</td>
<td>21–659</td>
<td>9.4 ± 2.1</td>
<td>6.3–14.4</td>
<td>0.44 ± 0.28</td>
</tr>
<tr>
<td>at 120 s</td>
<td>190 ± 154</td>
<td>21–715</td>
<td>9.9 ± 2.4</td>
<td>6.3–15.3</td>
<td>0.52 ± 0.32</td>
</tr>
<tr>
<td>Last potential</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>at 0 s</td>
<td>175 ± 153</td>
<td>20–746</td>
<td>8.5 ± 2.4</td>
<td>5.2–15.0</td>
<td>0.39 ± 0.26</td>
</tr>
<tr>
<td>at 120 s</td>
<td>175 ± 161</td>
<td>21–773</td>
<td>9.7 ± 2.6</td>
<td>5.4–15.4</td>
<td>0.47 ± 0.31</td>
</tr>
<tr>
<td>Force, % initial</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>at 120 s, % initial</td>
<td>113 ± 10</td>
<td>100–132</td>
<td>105 ± 7</td>
<td>93–119</td>
<td>120 ± 14</td>
</tr>
</tbody>
</table>

Values are means ± SD. Numbers under Fatigue are 40 Hz force.

J Neurophysiol • VOL 95 • MARCH 2006 • www.jn.org
increased twitch force ($P < 0.001$), and area ($P < 0.001$; Table 1). Increases in each EMG parameter were seen in all but four units (83% of units; Fig. 3). The EMG potentials recorded from the distal muscle surface were similar in duration ($P = 0.31$) to those recorded from the proximal muscle surface, but significantly larger in amplitude ($P < 0.01$) and area ($P < 0.05$). There was no interaction between EMG channel (distal vs. proximal) and test point (initial vs. potentiated) for any of the EMG variables. Initial and potentiated peak-to-peak EMG amplitudes for distal and proximal records were positively correlated, as were the respective EMG areas and durations (all $r > 0.96$).

The stimulation at frequencies between 5 and 100 Hz also increased twitch force ($P < 0.001$). Twitch force potentiation occurred in all but two units (91% of units), but its magnitude varied widely between units. Potentiated twitch forces ranged from 93 to 290% of those recorded initially (Table 1; Thomas et al. 1990). There was a significant positive correlation between the initial and potentiated twitch forces ($r = 0.82, P < 0.001$). In relative terms, the potentiation of twitch force was greater than the increases in any of the EMG parameters ($P < 0.05$ in all instances, paired $t$-test; Fig. 3). However, across units, no significant relationships were evident between the relative changes in any of the EMG parameters and twitch force (Fig. 4). The $r$ values for the EMG amplitude, duration, and area for the 23 units analyzed in this study were $-0.23$, $0.37$, and $0.15$, respectively. The lack of correlations between the changes in the EMG parameters and force were further emphasized by a weakening of the corresponding correlations ($r = 0.04, 0.26$, and $0.14$) when we included data from another seven units stimulated at frequencies between 5 and 100 Hz (Thomas et al. 1991a) but not subjected to the fatigue protocol.

Changes in unit EMG during fatigue of tetanic force

Fatigue was induced in each unit by stimulating its motor axon for 2 min with trains of 13 pulses at 40 Hz delivered once per second. Figure 5 exemplifies the changes in the EMG and force during this protocol for three motor units. For the more fatigable units (Fig. 5, A and B), the first EMG potentials of the trains increased in amplitude, duration, and area during the fatigue protocol. For the last train, the EMG amplitudes for these two units were 132 (Fig. 5A) and 110% (Fig. 5B) of that recorded for the first train. The corresponding values for the duration of the potentials were 114 and 118% initial. Area data were 144 and 111% initial. The evoked tetanic forces declined to 41 (Fig. 5A) and 49% of initial (Fig. 5B). In contrast, the force for the more fatigue resistant motor unit had only fallen by 8% after 2 min of stimulation (Fig. 5C). However, the amplitude, duration, and area of the first EMG potentials in the trains increased to 128, 103, and 136% initial, respectively. Figure 5 also shows changes in the last EMG potentials in the trains. For one of the more fatigable units, the amplitude of the last potential in the train at 120 s declined during the fatigue protocol, but its duration increased resulting in an increase in its area (Fig. 5A). The duration and area of the EMG also increased for the other fatigable unit, whereas the amplitude was maintained (Fig. 5B). The value of all three EMG parameters increased for the more fatigue-resistant unit (Fig. 5C).

Considering the first EMG potentials in the trains, significant changes occurred in the average unit data for all three EMG parameters during the 2 min of intermittent stimulation at 40 Hz (all $P < 0.001$). The values of all parameters gradually increased. The increase in EMG amplitude was significant at 20 s, duration at 60 s, and area at 40 s (Fig. 6, A–C). The distal EMG amplitudes were larger than the proximal EMG amplitudes 144 and 111% initial. The evoked tetanic forces declined to 41 (Fig. 5A) and 49% of initial (Fig. 5B). In contrast, the force for the more fatigue resistant motor unit had only fallen by 8% after 2 min of stimulation (Fig. 5C). However, the amplitude, duration, and area of the first EMG potentials in the trains increased to 128, 103, and 136% initial, respectively. Figure 5 also shows changes in the last EMG potentials in the trains. For one of the more fatigable units, the amplitude of the last potential in the train at 120 s declined during the fatigue protocol, but its duration increased resulting in an increase in its area (Fig. 5A). The duration and area of the EMG also increased for the other fatigable unit, whereas the amplitude was maintained (Fig. 5B). The value of all three EMG parameters increased for the more fatigue-resistant unit (Fig. 5C).

Changes in unit EMG during fatigue of tetanic force

Fatigue was induced in each unit by stimulating its motor axon for 2 min with trains of 13 pulses at 40 Hz delivered once per second. Figure 5 exemplifies the changes in the EMG and force during this protocol for three motor units. For the more fatigable units (Fig. 5, A and B), the first EMG potentials of the trains increased in amplitude, duration, and area during the fatigue protocol. For the last train, the EMG amplitudes for these two units were 132 (Fig. 5A) and 110% (Fig. 5B) of that recorded for the first train. The corresponding values for the duration of the potentials were 114 and 118% initial. Area data were 144 and 111% initial. The evoked tetanic forces declined to 41 (Fig. 5A) and 49% of initial (Fig. 5B). In contrast, the force for the more fatigue resistant motor unit had only fallen by 8% after 2 min of stimulation (Fig. 5C). However, the amplitude, duration, and area of the first EMG potentials in the trains increased to 128, 103, and 136% initial, respectively. Figure 5 also shows changes in the last EMG potentials in the trains. For one of the more fatigable units, the amplitude of the last potential in the train at 120 s declined during the fatigue protocol, but its duration increased resulting in an increase in its area (Fig. 5A). The duration and area of the EMG also increased for the other fatigable unit, whereas the amplitude was maintained (Fig. 5B). The value of all three EMG parameters increased for the more fatigue-resistant unit (Fig. 5C).

Considering the first EMG potentials in the trains, significant changes occurred in the average unit data for all three EMG parameters during the 2 min of intermittent stimulation at 40 Hz (all $P < 0.001$). The values of all parameters gradually increased. The increase in EMG amplitude was significant at 20 s, duration at 60 s, and area at 40 s (Fig. 6, A–C). The distal EMG amplitudes were larger than the proximal EMG amplitudes 144 and 111% initial. The evoked tetanic forces declined to 41 (Fig. 5A) and 49% of initial (Fig. 5B). In contrast, the force for the more fatigue resistant motor unit had only fallen by 8% after 2 min of stimulation (Fig. 5C). However, the amplitude, duration, and area of the first EMG potentials in the trains increased to 128, 103, and 136% initial, respectively. Figure 5 also shows changes in the last EMG potentials in the trains. For one of the more fatigable units, the amplitude of the last potential in the train at 120 s declined during the fatigue protocol, but its duration increased resulting in an increase in its area (Fig. 5A). The duration and area of the EMG also increased for the other fatigable unit, whereas the amplitude was maintained (Fig. 5B). The value of all three EMG parameters increased for the more fatigue-resistant unit (Fig. 5C).
tudes ($P < 0.02$), but the durations ($P = 0.51$) and areas were similar ($P = 0.44$) for the two EMG channels. There was no interaction between EMG channel (distal and proximal) and train number for any of the EMG variables. The general potentiation of the first EMG potential in a train was accompanied by a force decline ($P < 0.001$) that was manifest in every unit (Fig. 6D; Table 1). The force decline was significant at 60 s (Thomas et al. 1991b). However, as shown in Fig. 7 A–C, there were no significant correlations between the relative changes in force and any of the EMG parameters (amplitude: $P = 0.15$, duration: $P = 0.06$, area: $P = 0.22$ for distal EMG).

As with the first EMG potentials in the trains, the last potentials in the trains also increased in amplitude with fatigue ($P < 0.05$) but more significantly in duration ($P < 0.001$) and area ($P < 0.001$). Again, the distal EMG amplitudes were larger than the proximal EMG amplitudes ($P < 0.02$), whereas the durations ($P = 0.62$) and areas ($P = 0.36$) were similar. No interaction was observed between EMG channel (distal and proximal) and the number of the train for any of the EMG variables. The increases in EMG duration and area were both significant at 40 s, whereas the increase in EMG amplitudes only reached significance at 60–80 s when considering the distal and proximal EMGs separately.

Figure 8, A–C, shows the EMG changes that occurred during the fatigue protocol by comparing EMG parameters obtained for the first and last potential of the last train (at 120 s) with the first potential of the first train (at 0 s). For the first EMG potential of the last train, the amplitude, duration, and area remained higher than their initial values for 100, 83, and 96% of units, respectively. The corresponding percentages for the last potential of the trains were 61, 48, and 65%.

In separate ANOVAs, we compared the first and last potentials in the first (0 s) and last (120 s) 13-pulse trains. Overall, the first potential had a longer duration than the last one at 0 s ($P < 0.002$), and the duration increased from the first to the last train ($P < 0.001$). Furthermore, an interaction between the position of the potential in the train and train number ($P < 0.001$) indicated that the longer duration of the first potential compared with the last in a train only occurred for the first train, whereas there was little effect on the duration in the last train (Fig. 8E). For the amplitude, there was no effect of the potential’s position in the train ($P = 0.16$), but overall, the amplitude increased from the first to the last train ($P < 0.05$). However, an interaction between these factors ($P < 0.05$) suggested that this increase in amplitude largely was accounted for by an increase in the amplitude of the first potential in the last train (Fig. 8D). Finally, the area of the potential increased with both the potential’s position in the train ($P < 0.001$) and from the first to the last stimulus train ($P < 0.01$; Fig. 8F). There was no interaction between these two factors. Also in these ANOVAS, we included the EMG channel as a factor and, again, it only had an effect on the amplitude ($P < 0.02$), which was larger for the distal pair of electrodes.

Units whose EMG potentials had slowed the most by the end of the fatigue test had greater reductions in EMG amplitude (Fig. 9A; $P < 0.001$), larger increases in EMG area (Fig. 9B; $P = 0.014$), and were the more fatigable units (Fig. 9C; $P < 0.01$). There were no significant correlations across units between the relative change in force and relative change in EMG amplitude or area. This together with the observation that the EMG area for most units exceeded the initial values at the end of the fatigue protocol (Fig. 8C) suggested that the force loss related to processes beyond the electrical excitation of the muscle fibers.

**Discussion**

These data show that thenar motor unit EMG potentials and twitch force change to different extents and largely independently after the delivery of brief trains of pulses at different frequencies between 5 and 100 Hz. Furthermore, when the tetanic forces were reduced by brief trains of stimuli at 40 Hz...
for another 2 min, the EMG potentials potentiated even further. These divergent changes in EMG and force (Figs. 4 and 7) suggest that different processes control increases in EMG, potentiation of twitch force and fatigue of tetanic force. These issues have not been explored previously in humans at the level of single motor units.

Changes in unit EMG with twitch force potentiation

Stimulation at various frequencies between 5 and 100 Hz induced significant increases in EMG amplitude, duration, and area, changes that were accompanied by even greater increases in twitch force (Fig. 3). Significant increases in evoked EMG have also been seen in whole human muscles after a brief maximal voluntary contraction (e.g., Hicks et al. 1989) or maximal evoked contractions (e.g., Duchateau and Hainaut 1985). In cat hind limb or foot muscles, however, twitch force potentiation also occurred in almost all motor units examined, but no changes were reported for the EMG waveforms (Burke et al. 1973; Dum and Kennedy 1980; Kernell et al. 1975). Potentiation of EMG amplitude with muscle activity can result from increases in Na⁺/K⁺ pump activity and from greater synchronization of muscle fiber potentials (Hicks and McComas 1989; McComas et al. 1994). Our results suggest that increased synchronization may not fully explain the potentiation of EMG amplitude in experiments on whole human thenar muscles since the duration of most of our unitary EMG potentials increased rather than decreased in response to brief stimulation at different frequencies (Figs. 3 and 4). Increases in EMG potential duration probably reflect the slowing of muscle fiber conduction velocity that occurs when extracellular K⁺ increases with exercise (Juel 1988). In comparison, potentiation of twitch force after repetitive stimulation is proposed to relate to phosphorylation of myosin regulatory light chains (Grange et al. 1998; Sweeney et al. 1993). That the EMG and force data changed to quite different extents further suggests...
that different mechanisms underlie the potentiation of thenar unit EMG and twitch force in response to stimulation at different frequencies (Fig. 3). Likewise, there were no reliable correlations between the changes in unit EMG parameters and twitch force (Fig. 4).

Changes in unit EMG with fatigue

Two minutes of intermittent stimulation at 40 Hz resulted in significant potentiation of all EMG parameters, whereas the force of the motor units decreased. That the changes in EMG parameters were largely unrelated to the changes in force show that the changes in EMG signals do not predict motor unit fatigue and vice versa. These results also show that excitation of the sarcolemma and transmission across the neuromuscular junctions was effective and changes in these processes cannot explain the force loss. Similar conclusions have been reached in studies of single cat and rat motor units (Burke et al. 1973; Celichowski et al. 1991; Enoka et al. 1992; Hamm et al. 1989; Sandercock et al. 1985), where it has been suggested that force loss relates to changes in phosphate metabolism, intercellular Ca$^{++}$ handling and altered cross-bridge kinetics (Edman 1995; Westerblad and Allen 1991; Westerblad et al. 1998).

As for motor units in cat or rat hind limb muscles, the duration and area of the EMG potentials increased with fatigue in human thenar units, consistent with data of Chan et al. (1998). However, for thenar units, there was usually an increase in the amplitude of the first EMG potential in each train of stimuli (Fig. 6A), whereas more variable amplitude effects have been observed in cat units exposed to the same fatigue protocol (Enoka et al. 1992; Sandercock et al. 1985). These results may reflect differences in relative contraction intensity. Trains of pulses at 40 Hz evoke near maximal force in human thenar units (Thomas et al. 1991a) but cause unfused contractions in most cat motor units (Bottermann et al. 1986; Kernell et al. 1983). Thenar units are also relatively fatigue resistant compared with the units in many cat hind limb muscles (Thomas et al. 1991b), so the largest changes in EMG parameters do not necessarily occur in fatigable units (force <75% of the initial force after 2 min of stimulation), as is typical for motor units in cat and rat muscles (Celichowski et al. 1991; Enoka et al. 1992; Gardiner and Oilha 1987; Hamm et al. 1989; Sandercock et al. 1985).

Although Enoka et al. (1992) suggested that measurements of either EMG amplitude and duration or area are adequate to assess EMG changes, further comparison of results across studies is difficult because of the general lack of consensus on how to analyze EMG signals. Sometimes an entire train of potentials is averaged, which masks possible EMG changes occurring within the train (Figs. 5 and 8). The increases in unit EMG duration and area during fatiguing contractions may reflect slowing of muscle conduction velocity or fatigue-induced variability in conduction velocity across the different fibers of a motor unit (Gydikov et al. 1979; Stalberg 1966). Both these factors, in turn, will influence the amplitude of the unitary EMG potential. Furthermore, during high-frequency stimulation, a temporal overlap between succeeding EMG
cles (Hicks et al. 1989), whereas M-waves are maintained at initial levels as force declines during sustained maximal voluntary contractions (Bigland-Ritchie et al. 1982). After small daily amounts of chronic stimulation, the force of cat muscles declines in response to repeated stimulation but the EMG is maintained well (Kernell et al. 1987). These motor unit and whole muscle data emphasize that changes in EMG signals are poor indicators of motor unit and muscle force or vice versa. Thus inappropriate adjustments in force may occur when electrical stimulation is used to restore behaviors in paralyzed muscles if changes in EMG are used to control the stimulation parameters. Furthermore, when interpreting surface EMG data obtained during voluntary muscle contractions, it is important to consider that the magnitude of motor unit potentials may change in addition to alterations in motor unit recruitment and firing rate.

General implications

These data show that thenar unit EMG signals usually became larger both when twitch force potentiated and when additional stimulation induced declines in tetanic force. These activity-dependent dissociations between EMG and force must underlie some of the discrepancies in EMG–force relationships seen at the whole muscle level. For example, early increases in M-wave amplitude accompany force declines that result from repeated brief maximal voluntary contractions of thenar muscles (Hicks et al. 1989), whereas M-waves are maintained at initial levels as force declines during sustained maximal voluntary contractions (Bigland-Ritchie et al. 1982). After small daily amounts of chronic stimulation, the force of cat muscles declines in response to repeated stimulation but the EMG is maintained well (Kernell et al. 1987). These motor unit and whole muscle data emphasize that changes in EMG signals are poor indicators of motor unit and muscle force or vice versa. Thus inappropriate adjustments in force may occur when electrical stimulation is used to restore behaviors in paralyzed muscles if changes in EMG are used to control the stimulation parameters. Furthermore, when interpreting surface EMG data obtained during voluntary muscle contractions, it is important to consider that the magnitude of motor unit potentials may change in addition to alterations in motor unit recruitment and firing rate.

ACKNOWLEDGMENTS

The authors thank C. Kostov for help with some data analysis and B. Mas for help with the figures.

GRANTS

This research was funded by National Institutes of Health Grants NS-30226, NS-14756, and HL-30026, the Swedish Research Council (08667), and the 6th Framework Program of the EU (project IST-001917).

REFERENCES


