Fatigue-related impairment of neural drive to muscle

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The amplitude of the surface-detected electromyogram (EMG) increases during sustained, submaximal isometric contractions. This increase in EMG is due to an increase in motor unit recruitment and discharge rate that is necessary to maintain a target force in the presence of a declining force capacity of the contractile machinery. We have found, however, that the eventual inability to maintain force at a target level is due in part to an inadequate level of neural drive to muscle.

Evidence for impaired neural drive

We recently examined changes in evoked (M wave) and voluntary EMG signals associated with sustained contractions at different submaximal force levels (Fuglevand et al., 1992). Thirty-two subjects were assigned to one of three groups which differed as to the target force of the fatigue task: 20, 35, or 65% of the maximum voluntary contraction (MVC) force. The fatigue task involved a sustained isometric abduction of the index finger at the target force. Abduction force and first dorsal interosseus EMG were monitored throughout the task, which was terminated when the force dropped below 90% of the target for more than 2 seconds. M waves were elicited prior to and immediately after the fatigue task. The rectified average EMG, the mean frequency of the EMG power spectrum, and the average force were determined for each 10% epoch of the fatigue task.

The average endurance times for the 20, 35, and 65% target force groups were 534 s, 246 s, and 66 s, respectively. The mean power frequency declined in parallel during the fatigue task for all three groups and reached a similar endpoint value (50, 43, and 45% of the initial value for the 20, 35, and 65% MVC groups, respectively) suggesting that metabolite-induced changes in the propagation velocity of muscle fiber action potentials were similar for the three groups. The amplitude of the voluntary EMG increased from 19.3 to 45.2% of the pre-fatigue MVC value for the 20% MVC group, from 31.5 to 54.5% for the 35% MVC group, and from 59.6 to 81.4% for the 65% MVC group (Fig. 1). Voluntary EMG, therefore, failed to reach maximum levels (pre-fatigue MVC) during the fatigue task for any of the three force groups.

The fatigue task also caused a significant depression of M-wave amplitude for all three groups; immediately after the fatigue test, M-wave amplitude was 76.2, 73.6, and 88.3% of pre-fatigue amplitude for the 20, 35, and 65% MVC groups. The decline in M-wave ampli-

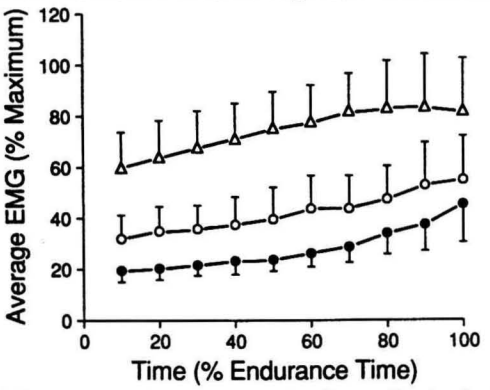

Figure 1. Mean (SD) changes in voluntary EMG amplitude during sustained isometric contractions at 20 % (•), 35% (o), and 65% (Δ) of MVC force (from Fuglevand et al., 1992).

tude, which reflects neuromuscular-propagation impairment, was greatest for the low-target force conditions. In a separate set of experiments involving nine subjects, the EMG from MVCs performed immediately after the fatigue task were diminished relative to pre-fatigue levels (61.9, 65.3, and 71.0% of pre-fatigue MVC for the 20, 35, and 65% MVC groups).

Potential mechanisms contributing to impaired neural drive

The fatigue-related deficit in voluntary EMG probably represents the net effect of at least six processes that limit the neural excitation of muscle (Fig. 2). These include: decreased excitatory input to motor neurons from the motor cortex (Maton, 1991) and muscle spindle afferents (Macefield et al., 1991); increased inhibitory input to motor neurons from metabolite-sensitive muscle receptors (Bigland-Ritchie et al., 1986) and Renshaw cells (McNabb et al., 1987); a reduction in motor neuron excitability (Kernell & Monster, 1982); and impaired neuromuscular propagation (Fuglevand et al., 1992). We suggest that impaired neural drive to muscle during sustained contractions contributes significantly to the decreased force capacity of the neuromuscular system.

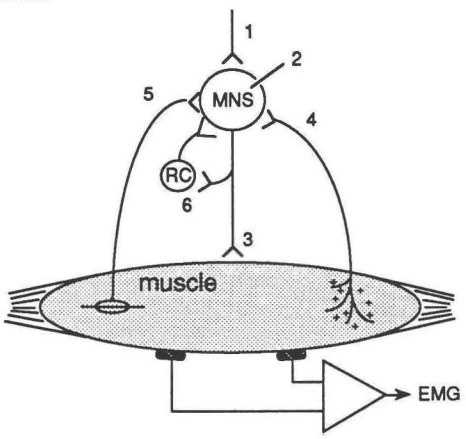

Figure 2. Processes that may interact to impair neural drive to muscle, as reflected in the surface EMG, during sustained contractions. 1) decrease in excitatory input from motor cortex 2) adaptation in motor neuron (MN) excitability 3) impairment of neuromuscular propagation 4) inhibition from metabolite-sensitive receptors 5) decrease in muscle spindle feedback 6) increased recurrent inhibition via Renshaw cells (RC).

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