

## Voluntary neuromuscular activation is enhanced when paired with a mechanical stimulus to human plantar soles

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### Abstract

The purpose of this investigation was to determine if the location and the timing relative to muscle activation onset, of a mechanical stimulus applied to the soles impacted the neuromuscular activation associated with a voluntary movement. The subjects completed a series of dorsiflexion or plantarflexion movements during which a stimulus was applied to either the heel or ball of the foot at one of three time periods relative to the initiation of the agonist muscle. Surface electromyography from the tibialis anterior and soleus was collected during the movements. The results show that if the stimulus was applied shortly before agonist muscle activation, regardless of stimulation site, the neuromuscular activity associated with the movement was greatly increased.

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It is well documented that individuals unable to experience bipedal gravitational loading will begin to develop lower limb muscle atrophy that, if not countered, will eventually result in a variety of motor behavior deficits. One possible countermeasure to muscle atrophy for people unable to regularly exercise is the use of a mechanical stimulus to the plantar surface of the feet. Supporting evidence for this concept comes from the work of De-Doncker et al. [5], who reported that rat soleus muscle atrophy was significantly attenuated as a result of mechanical foot stimulation during a hindlimb suspension protocol. Previous work with humans has shown that stimulation of the cutaneous and muscle receptors in the soles can result in enhanced neuromuscular activation in free-floating astronauts [12,13] and in individuals lying prone [1]. These findings are in agreement with reports of enhanced neuromuscular activation resulting from direct electrical stimulation to cutaneous nerves and/or receptors [2,9,11,16]. If sensory input can be used to 'drive' neuromuscular activation, it is reasonable to suggest that the increased activity may serve to attenuate muscle atrophy.

The enhanced activity resulting from a mechanical stimulation of the soles may result from cutaneous and/or muscle

reflexes [2,11,16], since such a stimulus could potentially activate both cutaneous and muscle receptors. Alternatively, it has been suggested that afferent input can function to modify motoneuron thresholds such that a voluntary descending command results in the activation of a greater number of motoneurons, compared to conditions without enhanced input [10,14].

Regardless of the mechanism, the neuromuscular response can be modified by the timing of the stimulus relative to muscle activation onset, the stimulation site and the nature of the accompanying afference. For example, it has been reported that enhanced responses are observed when an electrical stimulus is paired with constant sensory input from the soles [1]. Other reports indicate if a stimulus precedes voluntary muscle activation, the magnitude of the response is increased [1,8,10,14]. Additionally, varying the location of plantar stimulation can lead to different responses, particularly during specific phases of a task such as locomotion or cycling [3,6,7,17,18,20]. Finally, it has been shown that the neuromuscular response to a stimulus is greatest when paired with a voluntary contraction of a muscle [4,11,15,16]. These reports strongly suggest that both spatial and temporal factors play a significant role in the subsequent response to somatosensory stimuli. Our general hypothesis was that somatosensory stimulation of the soles, when paired with voluntary contractions, would result in

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increased neuromuscular activity. More specifically, we hypothesized that neuromuscular activity levels would be dependent on the spatial location of the stimuli on the soles and the timing of the stimuli relative to voluntary muscle onset. If somatosensory stimulation of the soles is to be reliably used to enhance neuromuscular activity and counter muscle degradation, it is crucial to gain a more complete understanding of the temporal and spatial relationships associated with plantar stimulation and neuromuscular activation. Here we report the findings of an investigation that assessed the neuromuscular responses to mechanical stimulation of the sole at two sites and at three different time intervals relative to agonist muscle onset.

In this study, 14 healthy adults (seven males, seven females, age range 19–36) served as volunteer subjects. All subjects were free of pain, sensory-motor deficits and provided written consent as required by the University Institutional Review Board. The task comprised five conditions of seated right ankle dorsiflexion movements and five conditions of plantarflexion movements. The subjects were instructed to perform ankle motions as rapidly as possible using a maximal voluntary effort and to hold for one count before releasing the ankle. During the movements, 15 psi of pressure was applied to either the heel or the ball of the foot using a rapidly inflating and deflating 6 × 6 cm pad. The pad reached full inflation within 3 ms of pressure initiation and remained inflated for approximately 150 ms. The stimulus pad activated both cutaneous and foot muscle receptors. None of the subjects experienced any pain with this magnitude of pressure, as ascertained by self-report. The pressure was applied at one of three time periods relative to agonist muscle activation onset (i.e. tibialis anterior (TA) for dorsiflexions and soleus (SO) for plantarflexions). These time periods were labeled 'early' (150–50 ms prior to muscle initiation), 'coincident' (49 prior to 49 after muscle initiation) and 'late' (50–150 after muscle initiation). This combination of stimulus sites (heel or ball of foot), movement direction, and pressure application time period resulted in ten different experimental conditions. To standardize the pressure experienced by each subject, chair height was adjusted such that the ankle, knee and hip angles were 90° while the sole rested on the pressure pad. A goniometer was used to ensure that the subjects achieved the standardized body segment angles. Each condition consisted of 20 ankle movements followed by a 5 min rest period to prevent fatigue. To ensure that the pressure stimulus was applied during the designated time period relative to agonist onset, a simple visual reaction time protocol. By providing a fixed warning interval accompanied by practice, the subjects became accurate in their estimation of when the reaction stimulus would be delivered and initiated their agonist activity such that movement initiation would coincide with the reaction stimulus. The delivery of the pressure stimulus was manipulated within the reasonably fixed interval of the

warning stimulus and agonist muscle onset such that 'early', 'coincident' and 'late' experimental conditions could be produced. The order of the condition presentation was counterbalanced to prevent order effects. Data were obtained during two control conditions consisting of plantar and dorsiflexion movements without a pressure stimulus. Data were also obtained during trials when the pressure stimulus was applied without an accompanying ankle movement. This experimental design enabled us to determine if where the stimulus was applied on the sole and when it was applied affected the resulting neuromuscular response.

Prior to data collection, the subjects were instrumented with preamplifier surface electromyogram (EMG) electrodes secured over the TA and SO muscles. During data collection, root mean square surface EMG, and electrical signals designating the reaction time circuitry and pressure pad activation were sampled at 500 Hz. The electronic signals associated with the reaction time circuit, pressure pad activation and EMG were stored on a desktop personal computer. Prior to data analysis, electronic cursors were used to assess the electronic records of each movement to determine if the pressure stimulus met the specified criterion for the designated condition. Of the 20 trials in each condition, the first ten that met the temporal conditions were analyzed. For each subject, and each condition, the selected data records were then averaged, with the reaction stimulus serving as the synchronization point. The resulting EMG waveforms were separated into three analysis epochs of 100 ms each with 'time zero' representing the initiation of muscle activation. The positive integrated area (PIA) within each analysis epoch was obtained using the cursors. Group means and standard errors were calculated for each epoch. The PIA data were assessed using repeated measures analysis of variance. The analyses comprised one between-subject factor, gender, and three within-subject factors: pressure location, timing and analysis epoch.

The data indicate two significant findings: (1), voluntary muscle activation can be enhanced when paired with plantar somatosensory stimulation; and (2), the enhancement is greatest if the stimulus occurs just prior to the initiation of voluntary activation, regardless of stimulation site. During dorsiflexion, both the early and coincidental pressure conditions were significantly different from the control at each epoch, regardless of stimulation site. The early pressure condition was greater than the control for the first and second 100 ms epochs, and lower on the third epoch ( $F = 19.968$ ,  $P = 0.001$ ). Coincidental pressure produced an enhanced response at the first epoch and a reduced response in the second and third epochs ( $F = 14.534$ ,  $P = 0.002$ ). Fig. 1 provides an example of averaged EMG waveforms obtained in the control and 'early' dorsiflexion condition. It can be observed that in the experimental condition, the activation magnitude is greater than that obtained during the control condition. These differences

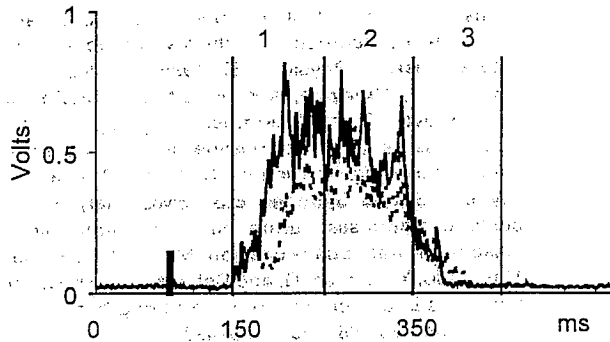


Fig. 1. An exemplar TA EMG waveform separated into muscle burst analysis epochs. Compared with the control group (broken line), 'early' application of pressure to the heel (solid line) resulted in enhanced EMG activity in the first and second epochs. The solid dark line that bisects the abscissa represents the time when the mechanical stimulus was applied.

are further illustrated in Fig. 2 using normalized average EMG activity obtained from the first analysis epoch. Although statistical significance was only found in the dorsiflexion conditions, the trend towards greater EMG activity was also observed during plantarflexions, regardless of stimulation site. Additionally, there were no gender effects. Trials during which the stimulation was applied without an accompanying contraction did not result in an increase in neuromuscular activation.

The present data are in accord with several authors [1,10,14] who observed that electrical stimulation of cutaneous nerves produced maximal effects on neuromuscular activation if applied prior to voluntary activation. Our data extend those observations to mechanical stimuli presented to the sole during voluntary movement. We suggest two possible mechanisms that may operate in concert or inde-

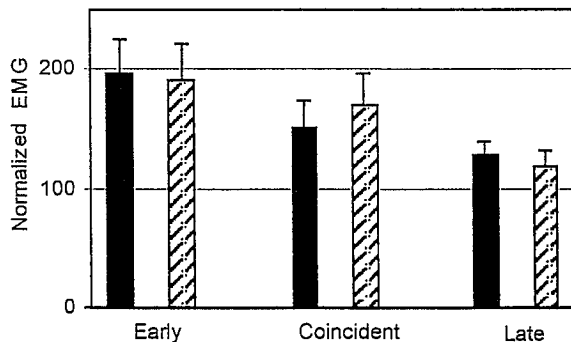


Fig. 2. Normalized mean EMG obtained from the first 100 ms analysis epoch (+SEM). The filled columns represent data from the dorsiflexion conditions and the hatch columns represent data from the plantarflexion conditions. The data were amplitude normalized using each subject's PIA obtained during the first analysis epoch of the dorsi or plantar flexion control condition as the divisor. The control condition is therefore represented as 100 along the ordinate. The neuromuscular activation levels obtained in the 'early' and 'coincident' dorsiflexion conditions were significantly different ( $F = 19.968$ ,  $P = 0.001$ ) from the control condition.

pendently to produce increased activation. One is that the increased activity resulted from cutaneous and/or muscle reflexes in response to the stimulus. If so, such reflexes are likely to be of spinal origin given the time frame in which increased activation occurred relative to the time of the stimulus. We only found statistically significant increases in activation in the first 200 ms of the early condition and the first 100 ms of the coincident condition. Thus, the plantar stimulus had less effect the longer the muscle had been electrically active prior to the presentation of the stimulus. Once the muscle had been fully activated, as in the 'late' condition, the stimulus did not impact the magnitude of activation. Given that the 'early' stimulus occurred at least 50 ms prior to voluntary activation, the enhanced activation observed in this condition was evident at a latency similar to those of reflexes observed in response to electrical cutaneous or muscle stimulation [1,18,19]. Our data are consistent with the idea that when a somatosensory stimulus is presented just prior to voluntary activation, a reflex response may be evident during the early phases of the voluntary muscle activation and would therefore contribute to enhanced activation. The temporal accuracy of the pressure stimulus was made possible through the use of the fixed warning interval reaction time protocol. This enabled the subject to activate the agonists in advance of the stimuli. The 'early' preparation may have slightly modified the responses to the pressure stimulus relative to a protocol relying on an unexpected movement stimulus. Future work will explore this possibility.

While the timing of our enhanced responses relative to the stimulus supports the concept that the increased activation may be reflexive in nature, the fact that the enhanced activation was only observed when the stimulus was coupled with a voluntary activation, suggests other mechanisms may have contributed. In particular, it has been argued that afferent input may function to modify motoneuronal thresholds such that a given descending movement command will increase the number of activated motoneurons [2,10,14]. Our data is consistent with this possibility given that sole afferent input generated during the 'early' condition would presumably be arriving at the motoneuronal pool during the same time period as the descending command. Therefore, the afferent input could be expected to have its largest influence during this period with progressively less influence as the motoneuron pool reached full activation during our maximal effort contractions. This possibility is consistent with the observed response pattern indicating the longer the muscle was activated prior to plantar stimulation, the less enhanced activation, or even inhibition, occurred. Whatever the underlying mechanisms contributing to the response, our data indicate that controlled timing of a mechanical stimulus to the sole, increased lower limb neuromuscular activation. This finding suggests that further research into the possibility that plantar stimulation can be used to enhance neuromuscular activation, and thereby serve to attenuate muscle atrophy, is warranted.

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