ABSTRACT: The modulation of the soleus H reflex in response to functional electrical stimulation (FES) of the rectus femoris (RF) muscle and its overlying skin was examined in 11 normal adults and 6 patients with a clinically defined complete spinal cord injury (SCI). Stimulation of RF at twice motor threshold (MT) resulted in a long-lasting (>1,000 ms) and significant reduction (50–70% of control) in the size of the soleus H reflex in all normal subjects tested. For five of the SCI subjects, 2MT stimulation of RF induced a 55–60% reduction in the soleus H reflex that was also long-lasting (>160 ms). In the remaining SCI subject, 2MT stimulation resulted in an initial period of significant H-reflex facilitation (0–14 ms) that was followed by a longer-lasting inhibition commencing 60 ms after the cessation of the conditioning stimulation. Decreasing the strength of stimulation to below that required to generate a clear contraction in RF resulted in mixed facilitatory and inhibitory actions that were subject dependent. The changes in H-reflex excitability resulting from FES highlight the potential use of FES in the management of hypertonicity in SCI but also suggest that the central actions of FES need to be considered when FES gait restoration programs are designed.


REFLEX EFFECTS OF INDUCED MUSCLE CONTRACTION IN NORMAL AND SPINAL CORD INJURED SUBJECTS

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The abnormal muscle tone occurring in spasticity has traditionally been considered to have a reflex element that is associated with abnormal stretch reflex pathways.19 Accordingly, decreasing the excitability of afferents, interneurons, or motoneurons has often been the focus of research into the mechanisms generating spasticity and its subsequent treatment. In this respect, electrical therapies including transcutaneous electrical nerve stimulation (TENS) for pain relief and functional electrical stimulation (FES) for muscle re-training or gait restoration in hemiplegia and spinal cord injury (SCI) have often been reported to decrease spasticity and to improve residual motor control.3,6,12,16,19,20

In patients with SCI, FES directed to the quadriceps, hamstrings, or the peroneal nerve has been reported to reduce the severity, frequency, and duration of clonic and tonic extensor spasms based on clinical assessment.1 In addition to studies reporting an antispasticity effect of electrical stimulation, there are sufficient data to suggest that the afferent feedback arising from electrical stimulation can influence the excitability of widespread motoneuron pools.15,24 The afferent feedback induced by FES will include the direct activation of populations of cutaneous and muscle afferents together with activity generated from receptors influenced by the mechanical events occurring in the stimulated and surrounding musculature.20

The reflex actions associated with this multimodal afferent feedback are largely unknown, but it can be hypothesized that, if this feedback acts to influence spasticity, it should have a measurable effect on spinal reflex pathways. Accordingly, we have investigated the conditioning influence that FES of the rectus femoris (RF) muscle has on the amplitude of the

Abbreviations: C–T, conditioning test; EMG, electromyography; FES, functional electrical stimulation; MT, motor threshold; MVC, maximum voluntary contraction; RF, rectus femoris; SCI, spinal cord injury; ST, sensory threshold; TENS, transcutaneous electrical nerve stimulation

Key words: functional electrical stimulation; H reflex; muscle contraction; reflex excitability; spasticity; spinal cord injury

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soleus H reflex in both normal and complete SCI patients. Part of this study has been published in abstract form.9,18

MATERIALS AND METHODS
Experiments were conducted with the approval of our local ethics committee (South Glasgow University Hospitals NHS Trust, Glasgow, UK) and the informed consent of all volunteers. Eleven normal men (aged 23–38 years) and six men with SCI (aged 30–44 years) from thoracic regions, ranging from T4 to T12, of traumatic origins participated. The time from injury varied from 3 to 16 years (9 ± 5; mean ± SD). All SCI patients were considered to have a complete spinal lesion on the basis of clinical examination except for subject 5, who demonstrated preserved joint sensation from the digits of his right foot. The degree of spasticity at the ankle joint for each SCI subject was assessed using the Ashworth scale,2 and was graded as 2 for subjects 2, 3, 4, and 6, and 0 for the remaining subjects. The SCI subjects recruited to this study also form the subject group used in a previous study.17 All reflex testing was performed with subjects seated. During the experiment, the subject’s legs and feet were restrained to prevent movement during reflex testing and to ensure that the RF contraction induced by FES was isometric. The hip, knee, and ankle angles were fixed at 120°, 160°, and 110°, respectively.

H-Reflex Testing. The soleus H reflex was evoked by monopolar electrical stimulation of the right posterior tibial nerve at the popliteal fossa using a 1-ms pulse generated by a constant current stimulator (model DS7A; Digitimer, Welwyn Garden City, UK), triggered once every 10 s and recorded by surface electromyography (EMG) via disposable pre-gelled Ag/Ag Cl electrodes (N-10-A; Medicotest, Ølstykkee, Denmark). The EMG signal was amplified and bandpass filtered (10 Hz–1 kHz; Neurolog, Digitimer) before being sampled at 2 kHz (CED 1401 and laboratory interface running Spike 2 software; Cambridge Electronic Design, Cambridge, UK). At the start of each experimental session, the intensity of tibial nerve stimulation was adjusted to evoke a control test reflex on the ascending part of the H-reflex recruitment curve for normal and SCI subjects. The amplitude of this control reflex was adjusted to be between 15–30% of the maximal M wave,10,22 for all subjects tested. This reflex was then conditioned by RF stimulation at varying intensities and intervals. The digitized EMG signals were full-wave rectified and the size of the M wave and H reflex were estimated from the area under the respective waveforms. The mean size of the unconditioned and conditioned test reflexes were calculated from a minimum of 20 evoked reflexes, and H-reflex data was only accepted if no significant differences (one-way ANOVA) could be detected in the size of the M wave recorded under control and experimental conditions. In each sequence of testing, the mean size of the unconditioned control reflex was measured at least twice and was alternated with the conditioned reflexes.

Conditioning Stimuli. Two circular self-adhesive electrodes (electrode type 879300R; PALS, Fallbrook, California) were positioned on the skin of the right leg at sites close to motor points of the RF muscle. Using a constant current stimulator (DS57AH, Digitimer), single shocks (pulse width 200 µs) of increasing intensity were used to determine the sensory (ST) and motor thresholds (MT) of normal subjects and the MT of SCI subjects. ST was defined as the lowest intensity of single-shock stimulation perceived by the subjects, whereas MT was defined as the lowest stimulus intensity that evoked an observable twitch of the RF muscle. All subsequent conditioning stimuli were presented as trains of pulses graded with respect to ST and MT in normal subjects (on average ST was equivalent to 0.13MT), and MT in SCI subjects. The conditioning stimulation train consisted of five pulses with an interpulse interval of 43 ms (equivalent stimulation frequency 23 Hz) repeated once every 10 s. In total, four stimulation intensities were used as conditioning stimuli in tests on the normal subject group (2, 1, and 0.7 × MT; 1 × ST) and three for tests on SCI subjects (2, 1, and 0.7 × MT). With the stimuli below MT, no observable mechanical response could be detected in the stimulated muscle. Stimuli above 2MT were not tested, as this was perceived as painful in a pilot study on normal subjects. Conditioning stimuli preceded the test H reflex by variable delays measured from the time of the last pulse in the conditioning stimulus train (Fig. 1). This allowed the reflex to be observed free from stimulation artifacts. The conditioning–test (C–T) intervals investigated are listed in Table 1. For each stimulus intensity and C–T interval, 20 repeat measures were taken. To avoid muscle fatigue due to FES, rest periods were given between each set of measurements and the size of the unconditioned H reflex was rechecked.

Measurement of Electrically Induced RF Contraction. The average strength and duration of the isometric contraction resulting from 2MT stimulation of RF was measured in five normal subjects and nor-
malized to the maximum voluntary contraction (MVC) level of each subject. The subjects were seated in an identical posture (hip = 120°, knee = 160°) to that used in reflex testing experiments, except that their lower leg was strapped to a strain-gauged beam that was aligned to measure knee extension moments. MVC was estimated from the maximum moment generated during a minimum of three attempted maximal knee extensions, and the mechanical response to electrical stimulation at 2MT was estimated by averaging the moments recorded for 20 episodes of stimulation repeated once every 10 s. Similar measurements were not obtained from subjects with SCI.

**Statistical Analysis.** For each subject, the conditioned H reflexes were expressed as percentages of the mean size of the control reflex, and one-way ANOVA was applied to the data. Bonferroni post hoc tests for multiple comparisons were performed to determine at which C–T interval the conditioned reflex was different from the control and whether statistical differences within the conditioned reflexes at the C–T intervals investigated could be established. In addition, pooled data (all subjects) grouped according to the C–T interval were compared using a repeated measure analysis of variance. A statistical comparison (ANOVA) was also performed on the M waves associated with the unconditioned H reflexes and those associated with the conditioned H reflexes for each subject tested. If significant differences between the sizes of the M waves were identified, the H-reflex data were discarded and the test repeated. Statistically significant differences were established at a 95% confidence interval. The results are presented as mean values and standard deviation (SD) of the mean.

**RESULTS**

**Mechanical Response to RF Stimulation.** Examples of the average (n = 20) knee moment recorded in response to stimulation of RF at 2MT are illustrated in Figure 2A for the five normal subjects in which it was measured. As can be seen, the contraction begins during the stimulus train and the peak force is reached approximately 40 ms after cessation of the stimulus train, following which time the muscle relaxes. With the stimulation parameters used, the peak isometric knee moment was equivalent to 19 ± 1.36% MVC (mean ± SD) with a contraction rise time of 0.21 ± 0.02 s and a relaxation time of 0.16 ± 0.01 s (Fig. 2B). The consistent level of force output in the tested group indicates that normalization of the stimulus intensity to MT provides an acceptable way to quantify stimulus conditions.

**Effect of RF Stimulation on the Soleus H Reflex.** In Figure 2, the effect of RF stimulation at 2MT on the average rectified H reflex at a C–T interval of 2 ms in a normal (Fig. 2C) and an SCI subject (Fig. 2D) is shown. In both cases, the size of the conditioned H reflex is reduced significantly compared with the control reflex. This reduction in the H reflexes occurred without any apparent change in associated M waves, indicating that stimulation conditions remained stable despite the ongoing RF contraction. The depression of the H reflex was significant in all 11 normal subjects and in 5 of the 6 SCI patients tested.

In the normal subject group, the H reflex was reduced by 50% at short C–T intervals (0–16 ms) and remained at a significantly reduced level compared to control values at the longer C–T intervals tested (400–1,000 ms), reaching a mean level of a 65 ± 12% of control values 1 s after the cessation of RF stimulation (Fig. 3A). The reductions in H-reflex size observed in the short interval range (0–16 ms) would have occurred during the active contraction of the muscle, whereas at the longer intervals tested in the normal subjects, the muscle was at rest (see Fig. 2A). The results for the five SCI subjects that demonstrated a depression in H-reflex magnitude are illustrated in Figure 3B. Here the reduction in the size of the H reflex across the interval range tested is between 50 ± 6.4% and 67 ± 7.9% of control reflex values. As in the normal subjects, the short interval range (0–16 ms) is likely to coincide with a period of increasing muscular tension, whereas the longer C–T intervals tested (40–160 ms) should correspond to periods where the muscle is relaxing.
both subject groups, there were no statistically significant differences between the amount of H-reflex depression seen at short and long C–T intervals, suggesting that the process generating the reduction in H-reflex size is established early and is long lasting. In addition, the overall pattern of changes in H-reflex excitability following RF stimulation at 2MT in the normal group and most of the SCI group is similar in the 0–16 ms range of C–T intervals ($P > 0.05$).

In one SCI subject (subject 5), an increase in the amplitude of the soleus H reflex was observed at short C–T intervals (0–12 ms) following stimulation of RF at this intensity. Data from this subject were not included in the overall analysis of the SCI subject group but are shown separately in Figure 3C. In this subject, the H-reflex size peaked with an amplitude 68 ± 7% greater than control values at a C–T interval of 4 ms (Fig. 3C). For intervals ranging from 14 to 60 ms, the change in the H-reflex size did not differ from control values ($P > 0.05$), but at longer C–T intervals (80 to 160 ms), a significant decrease in reflex size occurred (Fig. 3C), the reflex amplitude being reduced by 60 ± 10.7% compared to control values.

**Table 1.** Conditioning–test intervals used at each stimulation intensity for both groups.

<table>
<thead>
<tr>
<th>Stimulation strength</th>
<th>2MT</th>
<th>MT</th>
<th>0.7MT</th>
<th>ST</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal subjects</td>
<td>0–16 (2 ms steps)</td>
<td>0–8</td>
<td>0–8</td>
<td>0–4</td>
</tr>
<tr>
<td>SCI subjects</td>
<td>0–16 (2 ms steps)</td>
<td>0–8</td>
<td>0–8</td>
<td>Not applicable</td>
</tr>
</tbody>
</table>

MT, motor threshold; SCI, spinal cord injury; ST, sensory threshold.

*Time in parentheses denotes the incremental steps tested within a range of intervals.*

*FIGURE 2.* (A) Waveform averages of RF muscle force obtained from five normal subjects recorded during FES-induced muscle contraction at 2MT and expressed as a percentage of the MVC for each of the subjects tested. The FES period is depicted by the horizontal bar aligned below the force records. (B) Histogram illustrating the mean (±SD) duration for the time to peak tension and the mean (±SD) relaxation time following RF muscle stimulation at 2MT for the five normal subjects. (C) The average, rectified H reflex ($n = 20$) recorded under control conditions without RF conditioning (solid line) and following RF-induced muscle contraction at 2MT (dashed line) in a normal subject. (D) The average rectified H reflex ($n = 20$) recorded under control conditions without RF conditioning (solid line) and following RF-induced muscle contraction at 2MT (dashed line) in an SCI subject. For the conditioned H reflexes shown in (C) and (D), the C–T interval was 2 ms. Note that the reduction in H-reflex amplitudes for both subjects occurred without significant changes in M wave.
Effects of RF Stimulation at Different Intensities on the Soleus H Reflex. For stimulation intensities that did not evoke a clear muscle contraction, the effects were investigated over a restricted range of C–T intervals (Table 1). Unlike the data for 2MT stimulation, no overall pattern of facilitation or depression of the H reflex emerged that described the behavior of the entire sample (Fig. 4). Considering the results from the normal subjects, the left-hand column of the graphs in Figure 4 shows that the H reflex for only two subjects (subjects 4 and 6) was facilitated at each of the conditioning intensities examined. Facilitatory effects were seen in other subjects at some stimulus intensities but not others, e.g., subjects 5 and 8. In subjects 9 and 11, another pattern of H-reflex modulation was observed. These subjects demonstrated an increase in the magnitude of the H reflex following conditioning at ST, no significant effect at 0.7MT, and a depression in H-reflex size at MT. Significant reductions in H-reflex size were also seen in subjects 2, 3, 7, and 10 for all stimulation intensities tested, with subject 1 showing a reduction in H reflex at ST and MT.

For each C–T interval, a statistical analysis was performed to determine whether the size of the conditioned H reflex varied with changes in the conditioning stimulus intensity. The inhibitory or facilitatory effects for individual subjects did not differ significantly with different intensities of RF stimulation below 2MT. This suggests that at conditioning intensities below that required to evoke a clear muscle contraction, the effects observed depend on the subject tested rather than on the strength of stimulation.

DISCUSSION

Feedback Associated with FES. By directly exciting axons innervating muscle fibers, transcutaneous electrical stimulation of RF at 2MT will stimulate populations of cutaneous and muscle afferents lying...
within the vicinity of the stimulation area. Unlike the single-shock electrical stimulation of the femoral nerve used in many studies investigating human reflex pathways, the type of afferents recruited by the FES used in this study is difficult to predict. Nevertheless, RF stimulation at 2MT was not perceived as painful by normal subjects, indicating that nociceptive afferents in the skin or muscle were not significantly activated by the stimulus train. There was also no obvious flexion reflex activity induced by such

FIGURE 4. Individual subject H-reflex responses to RF stimulation at different intensities of conditioning stimulation. (A) ST, (B) 0.7MT, and (C) MT intensity. The data shown represents the mean size (±SD) of the pooled soleus H reflex obtained for all C–T intervals tested at each intensity of conditioning stimulation (see Table 1). The data was pooled in this manner because no significant differences were detected in the H-reflex size for individual subjects in relation to the C–T intervals tested. In (A), only data for normal subjects are shown; in (B) and (C), data from normal and SCI subjects are presented. In each graph, the abscissa identifies each subject with a number, and the ordinate shows the conditioned H-reflex size expressed as a percentage of the control H reflex.
stimulation in normal or SCI subjects. However, at this intensity, the stimulation of the α-motoneuron axons that evoke the RF contraction will be accompanied by the direct recruitment of afferent axons of greater or equivalent diameter. This implies that, at this stimulus intensity, an antidromic volley in a population of α-motoneuron axons together with a mixed afferent volley composed of activity in a population of cutaneous (Aβ fibers) and muscle afferents is generated. In addition to these direct effects, the evoked contraction will result in modulation in the discharge of muscle spindles and Golgi tendon organs located within RF and other muscle groups affected by the evoked contraction. Based on recordings of human group Ia afferent activity during isometric contractions,13,21 the evoked contraction will decrease RF muscle spindle afferent activity during the period of increasing force output. In contrast, afferent output from Golgi tendon organs should show the opposite behavior, with the output of group Ib afferents reaching a peak when the force output is greatest. Thus, at 2MT, it is likely that the stimulus train evokes feedback generated by both direct and indirect effects. Importantly, the indirect effects can be predicted to reflect the time course and magnitude of the induced contraction (Fig. 2A,B). At lower stimulus intensities, the contribution of direct and indirect effects will vary.

Given that stimuli at ST or 0.7MT did not generate either a direct or reflex contraction in the RF, a contribution from movement-related afferent feedback and recurrent pathways can be discounted, and only the direct effects of electrical stimulation of afferent fibers in the vicinity of the stimulating electrodes need to be considered. Furthermore, as no contraction was evident in RF, it is likely that the recruitment of low-threshold group I afferents is limited with stimulation at 0.7MT, and the main afferent volley at this stimulation intensity thus occurs in cutaneous fibers that lie close to the stimulation electrodes where the current density is greatest. However, the weak and asynchronous contraction observed with stimulation at MT suggests that only a small number of α-motoneurons are stimulated and that any movement-related afferent feedback is short lasting. Thus, the grading of stimulation intensity alters the contributions from direct and indirect forms of FES-induced feedback and allows some insight into their relative effects on the excitability of the soleus H reflex.

**FES-Induced H-Reflex Depression.** The observation that the long-lasting reduction of H-reflex activity only became evident in most subjects with stimulations at 2MT and was either not present, weak, or reversed at lower stimulation intensities strongly suggests that this reflex depression was not the result of sustained postsynaptic actions resulting from the direct electrical activation of afferents fibers. The H reflex was triggered at varying intervals following the final pulse in the RF stimulation train. Thus, at C–T intervals of 0 ms or more, the H-reflex afferent volley will reach the soleus motoneuron pool more than 5 ms after the fastest component of the afferent volley induced by the last shock in the RF stimulus train.14 This dictates that the short-lasting (2–3 ms) quadriceps group Ia facilitation of soleus cannot be observed in these experiments. However, the short-latency Ib inhibition from quadriceps to soleus, which terminates the Ia facilitation, has a duration of approximately 10 ms and may therefore contribute to the depression of H reflexes at C–T intervals ranging from 0–8 ms.14,23 At C–T intervals longer than 8 ms, a contribution from Ib inhibitory pathways can only occur if significant temporal facilitation occurs in this pathway. Given the time course of Ib inhibition (~10 ms) and the interstimulus interval used to generate the stimulus train (43 ms), this seems unlikely. The significant inhibition of the soleus H reflex at all C–T intervals greater than 10 ms for all normal subjects and five of the six SCI subjects is therefore unlikely to result from sustained postsynaptic inhibition generated by the direct electrical activation of RF afferents. A sustained inhibition resulting from the activation of cutaneous afferents is also unlikely given the mixed effects seen with low-intensity stimulation and the reported lack of reflex effects when a pure cutaneous stimulus, generating the same sensation as femoral nerve stimulation, is applied to the anterior aspect of the thigh.14,23 Nevertheless, a cutaneous contribution to the inhibition seen at short C–T intervals (0–10 ms) cannot be entirely excluded.

If direct electrical excitation of afferents cannot satisfactorily account for the duration of the observed depression of the soleus H reflex with stimulation at 2MT, it is worth considering whether contraction-induced changes in afferent feedback may be involved in generating the long-lasting H-reflex depression. In normal subjects, the maximal force generated by the evoked muscle contraction occurred on average 40 ms after the cessation of stimulation, indicating that, for C–T intervals in the 0–16 ms range, the H-reflex volley arrives at the spinal cord while the isometric force output is still increasing. As described previously, during this phase of increasing force output, the activity of muscle spindle afferents will be reduced and Ib afferent ac-
tivity will be increasing. It is therefore possible that the ongoing contraction contributes to the depression of the soleus H reflex by a combination of a group Ia disfacilitation and a postsynaptic Ib inhibition of soleus motoneurons. However, although our experiments cannot exclude this possibility, the presence of significant inhibition long after the end of the evoked contraction in normal subjects suggests that this postsynaptic mechanism cannot account for the entire period of H-reflex depression. Similarly, in the SCI group, C–T intervals between 40 and 160 ms also result in significant H-reflex depression. Given that these intervals should correspond to the period when the RF muscle is relaxing, such H-reflex depression argues against a major contribution from postsynaptic effects related to the contraction-induced changes in Ia or Ib afferent activity.

Although, postsynaptic Ib inhibition could contribute to the depression seen during the rising phase of the evoked contraction with stimulation at 2MT, other mechanisms need to be considered to explain the prolonged period of depression in both the normal and SCI subjects. With stimulation at this intensity, synchronous direct stimulation of α-motoneuronal axons occurs. The antidromic volley that this generates will result in a significant excitation within recurrent inhibitory pathways from quadriceps to soleus. It is known that femoral nerve stimulation at intensities exceeding MT generates a long-lasting (∼40 ms) depression of the H reflex in a manner consistent with heteronymous recurrent inhibition from quadriceps to soleus. Therefore, it is highly probable that recurrent inhibitory pathways also contribute to the depression of the H reflex in the 40-ms period following RF stimulation at 2MT. However, recurrent inhibition is unlikely to be maintained for a period much greater than 40 ms. Consequently, additional mechanisms that can generate prolonged excitability changes in the soleus H reflex need to be considered.

One mechanism that can lead to very long-lasting depression of the H reflex is presynaptic inhibition of soleus Ia afferents. Previous studies in human subjects have demonstrated that passive movements that stretch the quadriceps muscle produce a marked and long-lasting depression of the soleus H reflex by presynaptic inhibition. In addition, taps to the patella tendon inhibit the soleus H reflex, supporting the view that activity in stretch-sensitive muscle afferents from quadriceps can contribute to the presynaptic inhibition of the soleus H-reflex pathway. In walking and non-walking decerebrate cats, sinusoidal stretches and vibration of the quadriceps tendon also decrease the soleus H reflex in a manner consistent with such presynaptic inhibition. Similarly, weak stretches of the biceps femoris muscle evoke a profound depression of the soleus H reflex that can last up to 400 ms. In our experiments, the electrical stimulation of afferents from RF and the changing pattern of afferent activation that occurs in response to movement-related feedback could all contribute to a long-lasting presynaptic inhibition of soleus Ia afferents. This suggests that FES can evoke changes in the excitability of spinal reflex pathways that are similar to those that occur with passive movements.

Presynaptic inhibition of soleus Ia afferents, heteronymous group Ib inhibition, and recurrent inhibition may all play a part in the observed depression of the H reflex in our experiments. The long duration of the H-reflex depression strongly suggests that presynaptic mechanisms dominate in the conditioning effect produced by FES-induced muscle contraction. Furthermore, the demonstration that this depression of H-reflex amplitude can be observed in normal and SCI subjects strongly suggests that it is a spinal phenomenon and is not dependent on descending pathways. Our findings also support the belief that the afferent feedback resulting from this form of intervention may have potential as a means of reducing hypertonicity due to abnormal stretch reflex activity. It is therefore likely that reports of FES decreasing the severity of tonic extensor spasms in SCI subjects may be a consequence of presynaptic inhibition of group Ia afferent terminals. However, in one of our SCI subjects, FES facilitated the soleus H reflex at C–T intervals between 0 and 14 ms. This subject differed from the remainder of the SCI group in that he displayed a partial preservation of sensation from his right foot. Although we do not believe that the preserved sensory status of this subject accounts for the difference in results, it is likely that the facilitation is postsynaptic in origin and significant activity in the responsible pathway could be considered a contraindication to FES treatment of extensor hypertonicity in some SCI subjects.

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