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Low frequency depression of H-reflexes in humans with acute and chronic spinal-cord injury

Sheila Schindler-Ivens and Richard K. Shields

Physical Therapy Graduate Program, The University of Iowa, 2600 Steindler Building, Iowa City, IA 52242–1008, USA

Abstract

We measured low-frequency depression of soleus H-reflexes in individuals with acute ($n=5$) and chronic ($n=7$) spinal-cord injury and in able-bodied controls ($n=7$). In one acute subject, we monitored longitudinal changes in low-frequency depression of H-reflexes over 44 weeks and examined the relationship between H-reflex depression and soleus-muscle fatigue properties. Soleus H-reflexes were elicited at 0.1, 0.2, 1, 5, and 10 Hz. The mean peak-to-peak amplitude of ten reflexes at each frequency was calculated, and values obtained at each frequency were normalized to 0.1 Hz. H-reflex amplitude decreased with increasing stimulation frequency in all three groups, but H-reflex suppression was significantly larger in the able-bodied and acute groups than in the chronic group. The acute subject who was monitored longitudinally displayed reduced low-frequency depression with increasing time post injury. At 44 weeks post injury, the acute subject's H-reflex depression was similar to that of chronic subjects, and his soleus fatigue index (assessed with a modified Burke fatigue protocol) dropped substantially, consistent with transformation to faster muscle. There was a significant inverse correlation over the 44 weeks between the fatigue index and the mean normalized H-reflex amplitude at 1, 5, and 10 Hz. We conclude that: (1) the chronically paralyzed soleus muscle displays impaired low-frequency depression of H-reflexes, (2) attenuation of rate-sensitive depression in humans with spinal-cord injury occurs gradually, and (3) changes in H-reflex excitability are generally correlated with adaptation of the neuromuscular system. Possible mechanisms underlying changes in low-frequency depression and their association with neuromuscular adaptation are discussed.

Keywords

Spasticity; Paralysis; Plasticity; Muscle; Fatigue

Introduction

Individuals with spinal-cord injury (SCI) often display spasticity, a complex motor disorder characterized by a velocity-dependent increase in muscle resistance to passive stretch with exaggerated tendon jerks, caused by hyperexcitability of the stretch reflex (Lance 1981). Hyperreflexia is not an immediate consequence of SCI, nor is it typically present in the

acute stages of recovery. Rather, exaggerated stretch reflexes begin to emerge several weeks after the cord has been damaged (Illis 1995; Dietz et al. 1997). Many mechanisms for spinal spasticity have been proposed (see Katz and Rymer 1989 and Pierrot-Deseilligny 1990 for reviews), but the pathophysiology remains unclear. Even more elusive are the reasons for delayed onset of the condition.

The H-(or Hoffmann) reflex is used to assess the excitability of the monosynaptic reflex loop in neurologically intact and spastic individuals. The H-reflex is a compound muscle action potential elicited by electrical stimulation of afferent fibers in the mixed muscle nerve with subsequent recruitment of motor neurons through monosynaptic connections in the spinal cord (Magladery et al. 1951). The size of the H-reflex is an indication of the number of motor units that are reflexively activated at a given stimulus intensity. Any change in H-reflex amplitude under a given condition reflects changes in the excitability of the reflex pathway.

One approach to assessing changes in the excitability of segmental reflexes is to examine the effects of conditioning stimuli on the size of subsequent reflexes. It is well known that H- and tendon reflexes decrease in amplitude when conditioned with vibration (DeGail et al. 1966; Rushworth and Young 1966; Delwaide 1973; Van Boxtel 1986), passive muscle lengthening (Mark et al. 1968; Delwaide 1973; Gerilovsky et al. 1977; Robinson et al. 1982; Burke et al. 1983; Davies and Lander 1983; Hultborn et al. 1996), and a series of identical reflex stimuli such as paired H-reflexes or tendon taps (Eccles and Rall 1951; Lloyd and Wilson 1957; Meinck 1976; Hoeler et al. 1981; Rothwell et al. 1986; Van Boxtel 1986; Crone and Nielsen 1989; Kohn et al. 1997). However, these depressive effects are reduced or absent in spastic subjects (Calancie et al. 1993).

Low-frequency depression, which is the gradual fall in H-reflex size that occurs when trains of reflexes are elicited at frequencies between 1 and 10 Hz (Lloyd and Wilson 1957), has been shown to be impaired in humans with spasticity from spinal-cord injury (Ishikawa et al. 1966; Calancie et al. 1993) and in spinal-cord-lesioned animals (Gelfan 1966; Thompson et al. 1992; Skinner et al. 1996). Skinner and coworkers (1996) found that, 3 months after spinal transection in the rat, the H-reflex exhibited decreased low-frequency depression compared with control animals at 1, 5 and 10 Hz stimulation frequencies. Also in the rat, Thompson and colleagues (1992) showed that low-frequency depression in animals with contusion injuries of the spinal cord was less than in intact animals at 28 and 60 days post injury. There was no difference in low-frequency depression between injured and control animals at 6 days post injury, suggesting that loss of rate-sensitive depression does not occur immediately. Similar depression was observed regardless of the magnitude of the initial reflex. In human SCI, Calancie and colleagues (1993) demonstrated that low-frequency depression of the H-reflex was enhanced in acutely spinal-cord-injured individuals and reduced in chronically spinal-cord-injured individuals. However, the time course over which low-frequency depression became impaired and its relationship to adaptations in paralyzed muscle were not described.

In the present study, we investigated differences in low-frequency depression of H-reflexes among individuals who were able-bodied, acutely spinal-cord injured, and chronically

spinal-cord injured. We also examined longitudinal changes in low-frequency depression of the H-reflex over 44 weeks following spinal-cord injury. We then examined the relationship between rate-sensitive depression and soleus-muscle-fatigue properties. Our results are consistent with our hypotheses that: (1) chronically paralyzed individuals display a decrease in rate-sensitive depression of the H-reflex when compared with able-bodied subjects, (2) acutely paralyzed individuals display a similar rate-sensitive depression of the H-reflex when compared with the able-bodied subjects, (3) attenuation of rate-sensitive depression in humans with spinal-cord injury occurs progressively after injury, and (4) changes in rate-sensitive depression are generally correlated with muscular adaptations that contribute to increased fatigue.

Materials and methods

Subjects

After giving informed consent, measurements were made from ten male and two female individuals with spinal-cord injury and seven individuals without spinal-cord injury (four males, three females). The mean age, height, and weight of the able-bodied subjects were 29.6 years, 170.6 cm, and 66.89 kg, respectively. Measurements were made from seven individuals with chronic paralysis who had been spinal-cord injured for at least 2 years (7.5 ± 4.9 years, mean $\pm$ SD). Five subjects with chronic paralysis had clinically complete lesions, as evidenced by no motor or sensory function below the level of the lesion. Three subjects with chronic paralysis (C8, C7, and C2) had incomplete lesions with partially intact sensation in their lower extremities, but no motor function. Measurements were made from five individuals with acute paralysis who had been spinal-cord injured for less than 6 weeks (2.8 ± 1.9 weeks, mean $\pm$ SD). All acute subjects had complete motor and sensory loss below the level of their lesion except for subject no. 2 (A2), who had an incomplete lesion at C6 with partially intact sensation, but absent motor function below the level of the lesion. All paralyzed subjects were free of peripheral tibial nerve injury, having only an upper motor-neuron lesion. Background information on the paralyzed subjects is presented in Table 1. The institutional review board approved all experiments in accordance to the ethical standards laid down in the 1964 Declaration of Helsinki. All subjects voluntarily participated and provided written informed consent.

Experimental procedures

Measurements were made from the right, lower extremity in all subjects. Subjects were seated comfortably with the ankle dorsi-flexed to neutral and the knee flexed 90°. The foot was positioned on a rigid plate with a force transducing load cell (Genisco AWU-250) such that the foot was aligned perpendicular to the transducer. The foot was secured to the footplate via an ankle cuff with turnbuckles that directed force through the heel and into the footplate. This fastening system prevented upward movement of the heel during contraction of the ankle extensor muscles and helped to ensure that muscle contractions were isometric. The calibrated accuracy of this system was 1.34% of full scale. The mechanical recording apparatus has been described previously (Shields 1995; Shields and Chang 1997, 1998; Shields et al. 1997).

Surface EMG signals (H-reflexes and M-waves) were recorded with a bipolar silver-silver chloride electrode with a 0.8 cm diameter and 2 cm fixed interelectrode distance. The recording electrode was positioned in parallel with the soleus muscle, approximately 2 cm medial to the midline of the distal calf and distal to the medial head of gastrocnemius. Adjustments in electrode placement were made to optimize H-reflex amplitude, after which the recording electrode was secured with tape. A ground electrode was placed anteriorly on the tibia.

H-reflexes were elicited by transcutaneous electrical stimulation of the tibial nerve at the popliteal fossa. The stimulating electrode was a double-pronged surface electrode, placed such that the cathode was proximal to the anode (Kimura 1989). A custom designed, constant-current stimulator with a current range from 50 μ A to 200 mA and total output capability of 400 V was used to deliver a square wave with a fixed pulse width of 1000 μ s. The stimulator was triggered by a digital pulse from a data-acquisition board (Metrabyte DAS16F) housed in a microcomputer and controlled by custom software. Currents used to elicit H-reflexes were typically in the range of 40–60 mA. The maximal M-wave was determined, and all initial H-reflexes were between 20 and 50% of the M-wave (Crone et al. 1990). After the H-reflex was detected, the stimulating electrode was secured with an orthoplast and Velcro splint and taped to ensure that the electrode remained stationary during the recordings. The computer triggered the stimulator to produce pulses at various repetition frequencies.

Twenty soleus H-reflexes were elicited in a fixed order at each of the following frequencies: 0.1, 0.2, 1, 5, 10 Hz and again at 0.1 Hz. The second bout of 0.1 Hz was administered to verify that the H-reflex returned to its initial size after the higher frequency stimulation had ended and to ensure that activation remained unaltered during the session. If the H-reflex was not fully recovered, then the stimulation conditions were deemed unstable and not accepted. Two able-bodied subjects (N5 and N6) were unable to tolerate 5- and 10-Hz stimulation, and one chronic subject (C8) did not undergo 10-Hz stimulation because of severe spasms during the recordings.

All able-bodied, acute, and chronic subjects underwent H-reflex testing one time, except for one acute subject (A1), who was tested initially within 6 weeks of injury and was then re-tested every 1–2 months for the next 9 months to monitor temporal changes in rate-sensitive depression of the H-reflex. The acute subject (A1) who was studied over time also underwent a modified Burke-fatigue protocol (Burke et al. 1973) to monitor temporal changes in muscle fatigability at 6, 10 and 44 weeks post SCI. The fatigue protocol consisted of a 20-Hz stimulation train delivered every second for 330 ms (seven pulses) for a total duration of 2 min. This protocol is reliable and responsive to the fatigability of human paralyzed muscle (Shields 1995; Shields and Chang 1997; Shields et al. 1997). Peak torque from each of the 120 muscle contractions during the fatigue protocol was recorded on-line and analyzed with custom-designed software. The fatigue index (FI) was calculated by dividing the minimum peak torque by the maximum peak torque. Pearson correlation coefficients between FI and percent H-reflex suppression were calculated at 1-, 5- and 10-Hz stimulation frequencies. A significance level of 0.05 was used to test for correlations different from zero.

Data recording reduction and analysis

Force signals were amplified 500–1000×. EMG signals were onsite preamplified by a factor of 35 before mainframe differential amplification. The EMG amplifiers had an input impedance of 15 mΩ at 100 Hz, a frequency response of 15–1000 Hz, a common mode rejection ratio of 87 dB at 60 Hz, and a gain range of 1000–100,000×. Force and EMG signals were monitored on an oscilloscope and stored on VCR tape via a pulse code modulator (Vetter Digital, model 300). Signals from H-reflex procedures were digitized from tape using a 12-bit resolution analog-to-digital converter and sampled at a rate of 5,000 samples per second.

The peak-to-peak (PP) amplitude of the second through 11th H-reflexes (of the 20 H-reflexes elicited) at each frequency was obtained with peak detection software (Keithly Instruments). The absolute value for mean PP H-reflex amplitude (volts) was calculated at each frequency for each subject. Mean PP values at each frequency were normalized to the PP values obtained at 0.1 Hz. In the one subject (A1) followed over time, the 10th through 19th H-reflexes at 5 Hz and the 11th through 20th H-reflexes at 10 Hz were used for analysis on one occasion, because a spasm occurred during the initial second to tenth H-reflexes. The spasm was supported by an altered ankle extensor force trace.

Because we had a wide range of H-reflex sizes (20–50%), we analyzed the initial H/M ratio among our groups (acute, chronic, able-bodied) and found no differences ($P=0.133$). We also examined the relationship between the percent change in the H/M ratio at various frequencies and the initial H/M ratio. The associated R-square values were 0.16, 0.13, and 0.06 for the chronic, acute, and able-bodied subjects, respectively. This indicates that the initial H/M ratio did not contribute to the magnitude of the suppression. Hence, all H-reflexes were normalized to the 0.1-Hz condition.

We used a two-way repeated-measures analysis of variance (split plot) to determine if H-reflex suppression was different between the chronic and the able-bodied group. We then used a one-way repeated-measure analysis of variance to test for a significant effect of frequency within each group. Two-tailed tests with a significance level of 0.05 were used to test globally for differences between means, after which the appropriate post hoc analysis (Tukey) was used to make all pairwise comparisons. An adjusted significance level was used to account for type-I experimental error.

Results

H-reflex PP amplitude decreased with increasing stimulation frequency in all three groups that were examined (chronic, acute, and able-bodied), but H-reflex suppression was significantly larger in the able-bodied and acute subject groups than in the chronic group. The acutely paralyzed subjects suppressed their H-reflex at higher frequencies, similar to the able-bodied group. The acute subject who was monitored from 6 to 44 weeks following spinal-cord injury displayed a reduction in low-frequency depression over time, eventually responding similarly to the chronic group. The reduction in rate-sensitive depression was associated with increased muscle fatigue, which also progressively increased over the first 44 weeks following spinal-cord injury.

Figure 1 displays representative examples of M-waves and H-reflexes recorded from the able-bodied, chronic, and acute subjects at 0.1, 1, 5, and 10 Hz. For clarity, 0.2 Hz has been omitted from these figures. Each trace is the mean of ten consecutively recorded H-reflexes. In all subjects, robust H-reflexes were observed at 0.1 Hz. In the able-bodied group, H-reflexes decreased substantially and progressively with increasing frequencies. In contrast, chronic subjects displayed a more modest decline in H-reflex amplitude at 1, 5, and 10 Hz. The acute subjects showed a pattern of suppression similar to the able-bodied group. All five of the acute subjects were studied within 6 weeks of their spinal-cord injury.

Figure 2 summarizes the low-frequency depression of H-reflexes in the three groups examined in this study. Able-bodied subjects displayed significant low-frequency depression at 1, 5, and 10 Hz ($P < 0.05$). Indeed, H-reflexes decreased 68% at 1 Hz, 74% at 5 Hz, and 93% at 10 Hz. At 10 Hz, the mean H-reflex amplitude was 2% of the maximum M wave. Acutely paralyzed subjects displayed significant low-frequency depression at 1, 5, and 10 Hz ($P < 0.05$). The H-reflexes decreased 67% at 1 Hz, 88% at 5 Hz, and 95% at 10 Hz. At 10 Hz, the mean H-reflex amplitude was 1.13% of the maximum M-wave. Chronically injured subjects also displayed a significant depression of H-reflexes at 1, 5, and 10 Hz ($P < 0.05$). In chronic subjects, however, H-reflexes decreased only 30% at 1 Hz, 43% at 5 Hz, and 49% at 10 Hz. At 10 Hz, the mean H-reflex amplitude was 24.27% of the maximal M-wave. In able-bodied and the acutely paralyzed subjects, there was a significantly greater depression at 1, 5, and 10 Hz than in the chronic subjects ($P < 0.05$). Hence, the chronic-group's magnitude of H-reflex depression across frequencies was not consistent with the able-bodied and acute-group's depression across frequencies (significant interaction $P < 0.05$). All groups showed high reproducibility in that there was no difference in the mean H-reflex PP amplitudes recorded at the start and the end of the experiment using 0.1-Hz stimulation ($P > 0.05$). Table 2 summarizes each subject's normalized response to an increasing frequency of activation.

Figure 3 displays the temporal data obtained from subject A1, whose H-reflexes were recorded approximately every 1–2 months for a period of 44 weeks post injury. After the initial recordings made at 6 weeks post injury, we observed a decrease in H-reflex suppression at 1, 5, and 10 Hz. As early as 10 weeks post injury, A1 began to show a modest 47% depression in H-reflex amplitude at 1 Hz. This value approached the 30% depression, which was the mean depression at 1 Hz for the chronic group. Modest to absent H-reflex depression at 1 Hz continued over the next 34 weeks. By 18 weeks post injury, only 49% depression was observed at 5 Hz, a value that was similar to the mean value for chronic subjects. The amount of low-frequency depression at 10 Hz decreased gradually between 6 and 18 weeks post injury and later plateaued, showing approximately 73% suppression.

The relationship between soleus fatigability and H-reflex suppression was examined in subject A1 at 6, 10, and 44 weeks post injury. These data are presented in Fig. 4. The FI dropped substantially with increased time post injury, consistent with a transformation to more fatigable fibers (Shields 1995). Specifically, the FI decreased from 0.71 at 6 weeks post SCI to 0.45 at 10 weeks post SCI and 0.24 at 44 weeks post SCI. As the paralyzed soleus muscle became more fatigable, H-reflex suppression also decreased. There was a significant inverse correlation between FI and mean normalized H-reflex amplitude over the

44 weeks at 1 Hz ($r=0.92$), 5 Hz ($r=0.92$), and 10 Hz ($r=1.0$). Hence, as the soleus muscle became more fatigable with time, there was an associated loss of rate-sensitive depression of the H-reflex.

Discussion

The major findings in this study are that: (1) chronically paralyzed subjects exhibit suppression of H-reflexes to a lesser extent than able-bodied subjects and acutely paralyzed subjects, (2) the individual with acute paralysis showed similar H-reflex suppression as the chronics after 44 weeks of paralysis, and (3) there was an association between changes in muscle fatigue and the decrease in H-reflex suppression over time following SCI. Overall, these findings suggest that changes occur within the spinal cord that cause H-reflexes to attenuate their normal suppression at the frequencies tested here. Spinal reorganization with an associated decrease in presynaptic inhibition has been suggested as a plausible mechanism (Delwaide 1973; Calancie et al. 1993; Thompson et al. 1992, 1993; Skinner et al. 1996).

Compared to able-bodied and acutely injured subjects, chronically paralyzed subjects displayed a diminished low-frequency depression of soleus H-reflexes at 1, 5, and 10 Hz and absent low-frequency depression at 0.2 Hz. These data are consistent with previous observations that individuals with chronic SCI display impaired low-frequency depression of H-reflexes (Ishikawa et al. 1966; Calancie et al. 1993). However, our findings suggest that able-bodied and acutely paralyzed subjects displayed similar patterns of suppression across frequencies. Hence, we cannot conclude that acutely spinal-cord-injured humans display greater low-frequency depression than able-bodied subjects (Calancie et al. 1993). The extent of enhanced low-frequency depression after acute paralysis most likely depends on the precise time after injury. Our findings support that, within 6 weeks following injury, the pattern of suppression is similar to that of able-bodied subjects. Hence, the arbitrary definition used to define the acuteness of an injury becomes critical. Calancie and colleagues (1993) studied individuals within 2 weeks of their spinal-cord injury, while our acute injuries ranged from 1 to 6 weeks. However, in our temporal (A-1) study, less suppression was apparent by 18 weeks following injury.

In acute subject no. 1 (A1), who was monitored over time, we observed a transition from high-rate sensitivity to low-rate sensitivity between 6 and 18 weeks post SCI. This time course is similar to that typically involved in the transition from flaccid to spastic paralysis; however, acute subject no. 1 (A1) displayed impaired low-frequency depression before displaying clinically observable signs of spasticity. Longitudinal changes in low-frequency depression of H-reflexes after SCI have not been reported previously for human subjects, but are consistent with observations in the animal model. Low-frequency depression of H-reflexes in the spinally transected rat has been shown to be normal at 6 days post lesion, but significantly reduced at 28, 60, and 90 days post lesion (Thompson et al. 1992; Skinner et al. 1996). Also occurring in the first few weeks after SCI in acute subject no. 1 (A1) was a drop in soleus FI from 0.71 at 6 weeks to 0.45 at 10 weeks. These data suggest that the paralyzed soleus muscle became more fatigable in a relatively short period along with changes in H-

reflex suppression. The implications of concurrent changes in reflex excitability and muscular properties will be discussed later.

Understanding the mechanisms responsible for low-frequency depression is important insofar as we are interested in elucidating the causes of enhanced reflexes in individuals with spasticity. Frank and Fuortes (1957) demonstrated that monosynaptic EPSPs in spinal motor neurons were reduced by volleys in primary afferents without a concomitant change in membrane conductance. They concluded that decreased EPSP size must be caused by a presynaptic mode of inhibition. Because the reflex depression that occurs with vibration, stretch, and repetitive reflex activation appears to be associated with activation of Ia afferents, these depressive phenomenon have been attributed to presynaptic inhibition of primary afferent terminals (Eccles and Rall 1951; Lloyd and Wilson 1957; Delwaide 1973; Thompson et al. 1992; Calancie et al. 1993). It has been suggested that presynaptic inhibition is caused by segmental activation of GABAergic interneurons that synapse axo-axonically on Ia terminals, causing depolarization of primary afferent terminals and reduced neurotransmitter release (Eccles et al. 1963; Eccles 1964; Quevedo et al. 1992). Recently, however, some investigators have challenged the idea that inhibition of H-reflexes in humans is caused by classical presynaptic inhibition.

Hultborn and coworkers (1996) showed in humans that activation of Ia afferents by passive dorsiflexion of the ankle joint inhibited homonymous ankle-extensor H-reflexes for up to 10 s. Inhibition was limited to the pathway excited by the conditioning input and was thought to occur at a pre-motorneuronal level because no change was observed in the ability to elicit motor potentials with magnetic brain stimulation. In parallel cat experiments (Hultborn et al. 1996), it was shown that depressed monosynaptic reflexes were associated with reduced Ia EPSPs in spinal motor neurons without the occurrence of dorsal-root potentials or changes in membrane potential and input resistance of motor neurons. It was suggested that the long-lasting depression differed from classical presynaptic inhibition (Eccles 1964), which is known to be evoked mainly from flexors, have widespread distribution to all muscles in the limb, have a duration limited to several hundred milliseconds, and be accompanied by dorsal-root potentials. Kohn and colleagues (1997) arrived at similar conclusions when they observed that soleus H-reflexes were depressed for seconds when elicited after a single H-reflex or trains of H-reflexes. In contrast, presynaptic inhibition of soleus H-reflexes after peroneal-nerve stimulation produced a reflex depression for less than 1 s.

In our view, impaired low-frequency depression in chronically spinal-cord-injured subjects may be due to loss of classical presynaptic inhibition or impaired homosynaptic depression, as described by others (Eccles and Rall 1951; Hultborn et al. 1996; Kohn et al. 1997). Different forms of inhibition may dominate at different repetition frequencies. At longer interpulse intervals (0.2 Hz), homosynaptic depression may dominate, whereas, at shorter intervals (5 and 10 Hz), classical GABA-mediated inhibition may dominate. Because the magnitude of inhibition was reduced in chronic subjects at long and short interpulse intervals, it is possible that both of these forms of inhibition are altered after SCI. It is interesting to note that rate-sensitive depression was at least partially preserved in chronic subjects, which suggests that adaptations that contribute to impaired low-frequency depression do not completely eliminate it.

Possible explanations for impaired low-frequency depression include adaptive changes of interneurons secondary to the loss of supraspinal control (Delwaide 1973; Mailis and Ashby 1990; Calancie et al. 1993; Skinner et al. 1996), and interneuronal cell loss (Gelfan 1966). Our observation that muscular adaptations were correlated with changes in low-frequency depression merely indicates, in this subject, that changes at the muscular level co-varied with changes at the spinal level. Although the correlation between diminished low-frequency depression and reduced FI may simply represent the co-occurrence of two independent phenomena, it is possible that changes in muscle activation may retrogradely influence reflex excitability. It has been shown that, during trains of high-frequency stimulation, slow motor units that are fatigue resistant exhibit a progressive decrease in EPSP size, while fast-fatigable motor units display less of a decrease or even an increase in EPSP size (Mendell et al. 1995; Munson et al. 1997). Others have suggested that inhibition of monosynaptic reflexes by a variety of conditioning stimuli is greatest when small test reflexes are used and slow motor units are the major contributor to the signal (Lloyd and Wilson 1957; Crone and Nielsen 1990). These observations suggest that coordination may exist between muscle fibers, motor neurons, and synaptic transmission. If this coordination exists as a result of retrograde signaling, as has been recently suggested (Foehring and Munson 1990; Davis and Murphey 1994; Munson et al. 1997), gradual changes in low-frequency depression of H-reflexes in spinal-cord-injured humans could be associated with the apparent up regulation of faster myosin in the soleus muscle in individuals with paralysis (Shields 1995).

In conclusion, individuals with chronic paralysis lose the ability to suppress H-reflexes when compared with able-bodied subjects. The loss is not an immediate consequence of spinal transection, but instead a gradual process associated with adaptations in the entire neuromuscular system. Future studies need to establish the extent to which spinal-cord reorganization can be influenced by muscle activity in individuals with SCI.

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Fig. 1. H-reflexes recorded at 0.1, 1, 5, and 10 Hz for one able-bodied subject (*N4*), one chronic subject (*C2*), and one acute subject (*A2*). Each *trace* is the mean of ten consecutively elicited H-reflexes

Fig. 2.

The mean H-reflex amplitude at each frequency (normalized to 0.1 Hz) in the chronically paralyzed group (*upper left*, $n=7$), the able-bodied group (*upper right*, $n=7$), and the acutely paralyzed group (*lower left*, $n=5$). Asterisks indicate significant within-group differences.

Error bars are standard deviations

Fig. 3. H-reflex amplitude (normalized to 0.1 Hz amplitude) at each frequency for a single subject assessed at 6, 10, 18, 28, 35, and 44 weeks following spinal-cord injury (SCI). Notice the loss of H-reflex suppression at 1, 5, and 10 Hz as the time following injury increased

Fig. 4.
The relationship between the increase in soleus muscle fatigue over 44 weeks and the increase in H-reflex amplitude (loss of suppression) at 1, 5, and 10 Hz for an individual with acute spinal-cord injury followed over time

Table 1

Mean age, mean time post injury, and injury characteristics for each subject. *SCI* Spinal-cord injury

Subject	Age	Years post injury	Level of injury
Chronic SCI			
C1	38	2.1	T8 complete
C2	37	11.6	T1 incomplete
C4	34	4.6	T12 complete
C5	28	11.9	C6 complete
C6	55	9.6	T6 complete
C7	50	2.3	T11 incomplete
C8	33	14.2	T4 incomplete
Mean (SD)	39.4 (9.0)	7.5 (4.9)	–
Weeks post injury			
Acute SCI			
A1	23	6	T10 complete
A2	43	1	C6 incomplete
A3	47	3	C7 complete
A4	32	2	T5 complete
A5	27	2	T5 complete
Mean (SD)	34.4 (10.3)	2.8 (1.9)	–

Table 2

H-reflex amplitudes (normalized to 0.1 Hz condition) for the able-bodied, chronically paralyzed, and acutely paralyzed subjects.

Subject	0.1 Hz	0.2 Hz	1 Hz	5 Hz	10 Hz
Able body					
N1	100	87.80	31.70	24.39	10.98
N2	100	82.91	37.99	34.99	8.97
N3	100	76.95	21.76	14.20	3.68
N4	100	89.70	49.43	48.26	5.00
N5	100	91.53	46.51	—	—
N6	100	91.16	23.86	—	—
N7	100	85.34	10.17	7.20	4.22
Mean (SD)	100	86.48 (5.23)	31.63 (14.13)	25.81 (13.36)	6.57 (2.63)
Chronic SCI					
C1	100	92.42	47.85	26.15	30.88
C2	100	97.17	56.41	62.14	58.12
C4	100	107.33	71.67	67.66	86.12
C5	100	98.39	89.31	56.34	80.44
C6	100	96.11	85.16	66.60	27.55
C7	100	102.99	69.07	66.11	24.73
C8	100	97.89	73.99	41.14	—*
Mean (SD)	100	99.07 (5.30)	69.91 (16.01)	57.50 (15.91)	51.31 (27.55)
Acute SCI					
A1	100	81.42	10.93	11.91	6.14
A2	100	100.47	75.40	25.16	4.20
A3	100	73.80	7.46	7.38	7.37
A4	100	99.65	41.86	9.33	6.55
A5	100	100.18	28.77	5.97	1.15
Mean (SD)	100 (0)	91.10 (12.61)	32.88 (27.54)	11.95 (7.71)	5.09 (2.49)

* indicates that able-bodied subjects could not tolerate the stimulus at these frequencies, while * indicates excessive spasm in chronically paralyzed subject. SCI Spinal-cord injury