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Research article

SPINAL CORD INJURY AND CONTRACTILE PROPERTIES OF THE HUMAN TIBIALIS ANTERIOR

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ABSTRACT

The purpose of this study was to evaluate contractile properties of the tibialis anterior of paralyzed and non-paralyzed subjects. The contractile properties and the fatigability of the tibialis anterior muscle (TA) were tested in 8 spinal cord injured (SCI) and 8 control individuals. The TA was stimulated at frequencies from 10 to 100 Hz to determine a force-frequency curve. A fatigue bout was also performed by stimulating the muscle at 40 Hz every two seconds for three minutes. The SCI muscles produced lower forces overall, but higher forces relative to maximal force at lower frequencies, shifting the force-frequency curve of the SCI group to the left. The half-relaxation time and rate of relaxation at 40 Hz was slower in the SCI muscles than in the control muscles (127 ± 18.4 ms vs. 78 ± 8.7 ms, 6 ± 1.5 $kg \cdot s^{-1}$ 20 ± 4.1 $kg \cdot s^{-1}$ respectively). In addition, force loss and slowing of relaxation during the fatigue protocol were not significantly different between the two groups due to high variability in the SCI group. The TA of the SCI group had slower contractile properties than the control group and fatigability was not significantly different between the SCI and control group. The protocol may be useful to assess training effects during rehabilitation of paralyzed muscle.

KEY WORDS: Muscle, contractility, fatigue, paralysis, paraplegia.

INTRODUCTION

Skeletal muscle is adaptive to changes in chronic use and disuse. These adaptations include modifications in mass, metabolic and contractile properties. Because muscle disuse stems from many causes including injury, neuromuscular disease, microgravity environments, and prolonged bed rest, different rehabilitation programs have been developed to lessen the results of disuse (Bamman et al., 1997; Gerrits et al., 2000a; Martin et al., 1992; Yoshida et al., 2003). Only with a full understanding of the muscular changes following chronic disuse and its probable mechanisms can appropriate and effective treatment techniques be instituted.

Large variability exists in the results of contractile studies on disused muscle. Changes in muscle fiber composition with disuse generally show a decrease in slow fibers and an increase in fast fibers in animal (Leiber et al., 1986; Spector, 1985) and human skeletal muscle (Lotta et al., 1991; Martin et al., 1992; Rochester et al., 1995a; Round et al., 1993). In addition, disused slow muscle often shows a right shifted force-frequency curve, increased shortening velocity, and decreased contraction time, ½ relaxation time (½RT), fusion during a 5 Hz stimulation, twitch/tetanus ratio, and time to peak tension (TPT) compared to the normal slow muscle (Maier et al., 1976; Roy et al., 1984; Spector, 1985; Witzmann et al., 1982). This indicates a switch to a faster muscle as a result of disuse. In contrast to this, one study found that

disused slow muscle became even slower (Dasse et al., 1981).

Most mammalian muscles are composed of a mix of fast- and slow-twitch fibers, so it is particularly important to examine the contractile properties of disused mixed muscles. There are few studies on the adaptation of fast-twitch muscle to disuse and the reported results are conflicting (Dasse et al., 1981; Maier et al., 1976; Roy et al., 1984; Witzmann et al., 1982). The use of a neurotoxin (tetrodotoxin-TTX) causes slowing of the contractile properties, whereas joint fixation has no effect, slows, or speeds up the contractile properties (Dasse et al., 1981; Maier et al., 1976; Witzmann et al., 1982). Additionally, spinal transection alters some of the contractile properties to those seen in faster muscle (Roy et al., 1984) or does not change contractile properties (Lieber et al., 1986). The reasons for the different responses of fast-twitch muscle to different disuse models are not clear.

The effects of disuse seen with spinal cord injury (SCI) in humans are controversial. In a mixed muscle like the human tibialis anterior (TA), with approximately 76% type I and 22% type IIA fibers (Rochester et al., 1995a) prolonged disuse transformed some of the type I fibers into type II fibers and changed some of the contractile properties to those seen in faster muscles (Rochester et al., 1995a; 1995b). Disused quadriceps muscle had a faster rate of force production, a shortened ½ RT (Gerrits et al., 1999) or no change in ½ RT (Gerrits et al., 2001), and less fusion at 10 Hz (Gerrits et al., 1999; 2001). However, in another study it was found that the human disused thenar muscle had a higher twitch /tetanus force ratio and a left shift of the force frequency curve indicating a slowing of contractile properties (Thomas, 1997). Another study found that the ½ RT was slower but the rise time was unchanged in the quadriceps femoris muscle after as little as 6 weeks of disuse (Castro et al., 2000). Bed rest caused significant slowing of TPT and ½ RT (Koryak, 1995). In most studies (Rochester et al., 1995a; 1995b; Thomas, 1997) a great variability among disused muscle was found, so more human studies are needed to clarify the conflicting results.

Additionally, since fast muscles are more fatigable, a conversion of slow to fast fibers should result in a more easily fatigued muscle (Hamalainen and Pette, 1993; Kugelberg, 1973), and greater slowing of contractile properties during a standard fatigue test (Dubose et al., 1987; Rankin et al., 1988). Therefore, the purpose of this study was to investigate the effect of disuse and/or reduced activation observed in spinal cord injury on contractile properties and fatigue resistance in the mixed-fiber tibialis anterior muscle in humans.

METHODS

Subjects

Spinal cord injured (SCI) patients (n=14) and control subjects aged 18 to 65 years were recruited for the study. None of the SCI subjects had been involved in an electrical stimulation program for at least six months prior to participation in this study. Each SCI patient was matched by age and gender to a moderately active control subject. A questionnaire covering gender, age, history of spinal cord injury, time since injury, and participation in physical therapy was obtained from the SCI subjects. The protocol was approved by the Institutional Review Board of Northern Arizona University.

Instrumentation

The test apparatus consisted of a specially designed chair where the knees and ankles were bent at 90 degrees with the soles of the feet resting on a support plate, and secured with a canvas strap similar to that of Reid et al. (1993). The support plate was hinged at the heel, with the front attached to a force transducer (Omega LCAA-200). The plate height was adjusted to accommodate differing subject tibia lengths. Signals from the force transducer were amplified (Grass Instruments, 7P122) and displayed on a chart recorder (Kipp & Zonen, BD112). The force transducer was calibrated before each test protocol using known weights.

Protocol

All of the subjects came to the lab, where the procedures, purpose, and risks associated with the participation were explained and an informed written consent was obtained. Subjects were then seated in the chair as described above. The skin over the right TA was shaved and scrubbed with an alcohol pad. The cathode was positioned over the motor point of the right TA according to a motor point chart (Starkey, 1999) and the anode was positioned 5 cm distally. Stimulation of 100 Hz at 70V to 140V and 0.2-ms pulses was used to make sure that the correct motor point was found. Electrode position was adjusted to elicit the strongest contraction (Reid, et al, 1993). For the control subjects, maximum tolerable voltage was determined. A muscle stimulator (Grass Instruments, S88K) was used to deliver rectangular square wave pulses (0.2 ms pulse duration) and the voltage was increased until a 650-ms stimulus train at 100 Hz was perceived as painful. The voltage was then decreased to just below pain threshold, 70-80V. For the SCI subjects the average voltage of stimulation used for the matched control was employed. In three patients the voltage was increased to 130 or 140V to elicit a muscle contraction. It is likely that

submaximal stimuli were used in both groups because of the protocol (to eliminate pain for controls). It was important, though, in our opinion, to equate the stimuli as much as possible to get as nearly as possible the same stimulation in the two groups. Following the determination of the motor point and the voltage for the electrical stimulation, two force-frequency protocols and one fatigue-recovery protocol for the SCI and control subjects were done as described below.

For the force-frequency protocol, the TA was electrically stimulated at 10, 15, 20, 25, 30, 35, 40, 50, 60, 70, 80, and 100 Hz (650ms train duration, 0.2ms pulse duration). The subjects were given two minutes of rest between each stimulation frequency within the force-frequency test and three minutes rest between each of the two force-frequency protocols. The average force-frequency relationship for all paraplegic subjects were constructed by expressing the forces developed at submaximal stimulus frequencies as a percentage of peak tetanic force developed. If the first and second protocol elicited different forces (>5%), subjects came back for a second visit. This was required for three of the eight control and none of the SCI subjects.

The fatigue-recovery protocol was done three minutes after the force-frequency protocol. In order to produce fatigue, a stimulus train of 650ms at 40 Hz with the same voltage used in the force-frequency protocol was delivered to the TA every two seconds for 180 seconds. Forces and contractile properties were measured for the first three contractions of the fatigue test and for the three contractions immediately following 30, 60, 120, and 180 seconds of the fatigue test. The recovery from fatigue was measured for three successive contractions at 190, 210, 240, 270, 300, and 360s. The fatigue and recovery index were calculated as followed:

- Fatigue Index (FI) = (postfatigue force/prefatigue force) x100
- Recovery Index (RI) = (postrecovery force/prefatigue force) x100

Other studies have shown that the fatigue and contractile properties protocols have a high degree of reproducibility and reliability (Reid et al., 1993; 1994; Williamson and Caley, 1998). Previous unpublished work in our laboratory also indicates that the FI and RI are reproducible.

The TA relaxation properties during the fatigue/recovery protocol were characterized by both the time and the rate of relaxation. The one-half relaxation time (½ RT) was the time it took for the force to drop to one-half of its peak value. The ½ RT was calculated and the average of three consecutive trials were taken at 0, 30, 60, 120, 180 190, 210, 240, 270, 300, and 360 seconds of the fatigue-recovery protocol.

The rate of one-half relaxation time (R½ RT) was determined by differentiating the force output with respect to time during the relaxation phase of the tetani. The R½ RT was normalized to the mean measured force for that contraction. The R½ RT was calculated by taking the quotient of half the peak force divided by the ½ RT and the average R ½ RT of three consecutive trials was taken at 0, 30, 60, 120, 180 190, 210, 240, 270, 300, and 360 seconds of the fatigue-recovery protocol

Statistical analysis

A two-way analysis of variance (two-way ANOVA) with repeated measures was used to determine the force-frequency differences with respect to the frequencies used (10, 15, 20, 25, 30, 35, 40, 50, 60, 70, 80, 100 Hz) and condition (SCI vs. control). A paired t-test with a significance level set at 0.05 was used to test for a difference in ½ RT and R½ RT of the 40 Hz tetani between the SCI subjects and their matched controls. Two-way ANOVA with repeated measures was used to determine differences in force, ½ RT, and R ½ RT over time and condition (SCI vs. control). One-way repeated measures analyses of variance were used to determine differences in FI and RI in the SCI and control groups. All data were reported as mean ± standard error (± SE) and significance levels were set at $p < 0.05$. The Student-Newman-Keuls test was used for the post hoc test.

RESULTS

Of the 14 SCI patients, three were not able to produce any force throughout the experiment and were excluded from the analysis or assignment of a matched control. Another three SCI patients were excluded from the analysis because their TA spasmed with each of the electrical stimuli used during the force-frequency protocol. Since no useable data could be obtained from these subjects, their testing was not completed and they were excluded from the study, along with their matched controls. The experiments were then analyzed on the remaining one woman and seven men (age: 37 ± 6 years) and their age and gender matched controls (age: 37 ± 5). The characteristics of the SCI subjects used for analysis are presented in Table 1.

Force frequency relationship

Figure 1 presents the mean absolute forces produced by the SCI and control groups. The forces produced by the SCI group at 30-100 Hz were significantly lower than the forces produced by the control group

Table 1. Individual data of SCI subjects with respect to age, gender, level of injury, time since injury, occurrences of spasms, use of anti-spasm medication, and completeness of the lesion.

Subject	Age	Gender	Level of Injury	Time since Injury	Spasms	Anti-Spasm Medication	ASIA class*
1	18	man	C3-5	2y, 11m	often	Yes	B
2	20	man	C4-5	1y, 6m	some	Yes	B
3	22	man	C6	7m	often	Yes	A
4	30	man	T11	10y	often	No	A
5	40	man	C6	16y	often	No	B
6	53	man	C3-4	1y, 6m	often	Yes	A
7	65	man	C6-7	4y	often	Yes	A
8	50	woman	T9	2y	some	Yes	A

*ASIA (American Spinal Injury Association, 1992) scale is used to classify the completeness of the lesion. A, sensory and motor complete; B, sensory incomplete but motor complete.

($p < 0.05$). In the SCI group the force produced at 50 Hz was not different from the force produced at 10 Hz, whereas in the control group the force produced at 50 Hz was significantly different from the force produced at 10, 15, and 25 Hz ($p < 0.005$). The highest force produced by the SCI group was 1.5 ± 0.33 kg, which is one third of the 4.3 ± 1.0 kg produced by the control group.

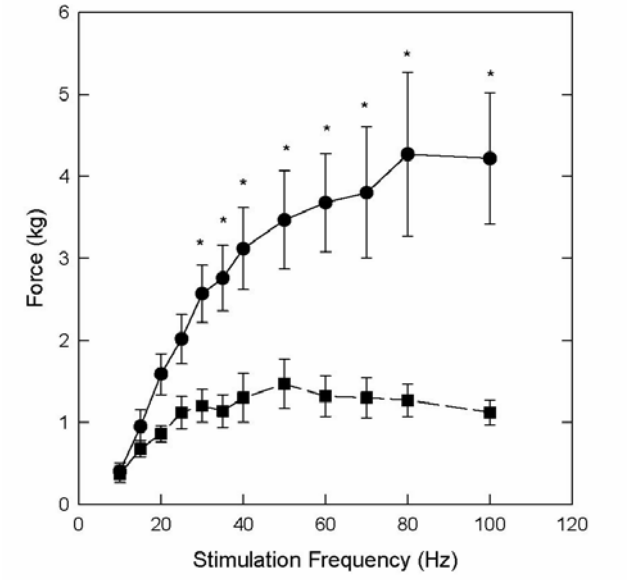

Figure 1. Force-frequency relationship measured in absolute force of TA in SCI (■) and control subjects (●). * Significant difference between groups ($p < 0.05$). (Error bars represent SE).

Figure 2 presents the relative force responses of the SCI and control groups at each frequency. The relative force produced at 10-30 Hz was significantly higher in the SCI group compared to the control group ($p < 0.01$), shifting the force-frequency curve of the SCI group significantly to the left at those stimulation frequencies. Stimulation at 10 Hz elicited 31 ± 7.6 % of the peak tetanic force in the TA of the SCI group, which was significantly higher than the 12 ± 4.2 % of maximal force produced by the TA of the control group ($p = 0.005$). The mean force increased progressively as the stimulation frequency increased from 10 to 50 Hz (50Hz = 95% of peak force) and decreased at frequencies above 50 Hz in the SCI group. In the control group the mean relative force increased progressively from 10 to 80 Hz (80Hz = 96% of peak force).

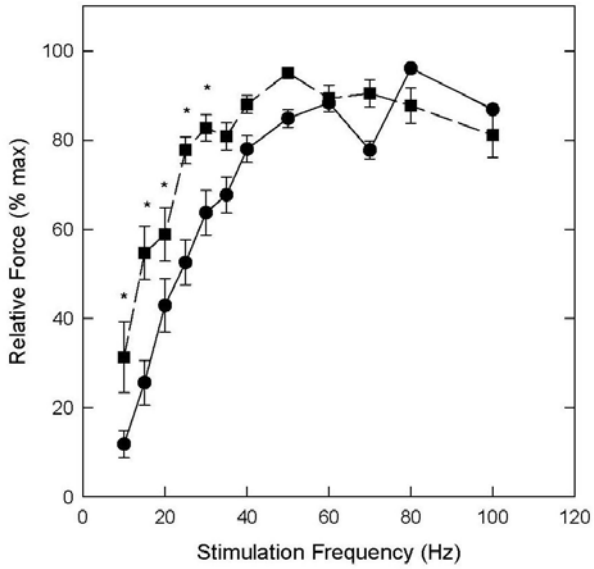

Figure 2. Force-frequency relationship normalized to peak force of TA in SCI (■) and control subjects (●). * Significant difference between groups ($p < 0.05$). (Error bars represent SE).

Contractile speed

The mean tetanic ½ RT produced at 40 Hz for the SCI group was 127 ± 18.4 ms, significantly longer than the 78 ± 8.7 ms produced by the control group ($p = 0.023$). The mean tetanic rate of ½ RT produced at 40 Hz was 6 ± 1.5 $kg \cdot s^{-1}$ for the SCI group, significantly slower than the 20 ± 4.1 $kg \cdot s^{-1}$ produced by the control group ($p = 0.002$).

Fatigue characteristics

Results for the fatigue test are shown in Figure 3. The forces produced by the SCI group were

significantly lower than the control at all times during the fatigue test ($p = 0.015$, see ANOVA Table in Appendix). In addition, forces produced during the fatigue test were significantly lower than the initial force in both groups ($p < 0.001$).

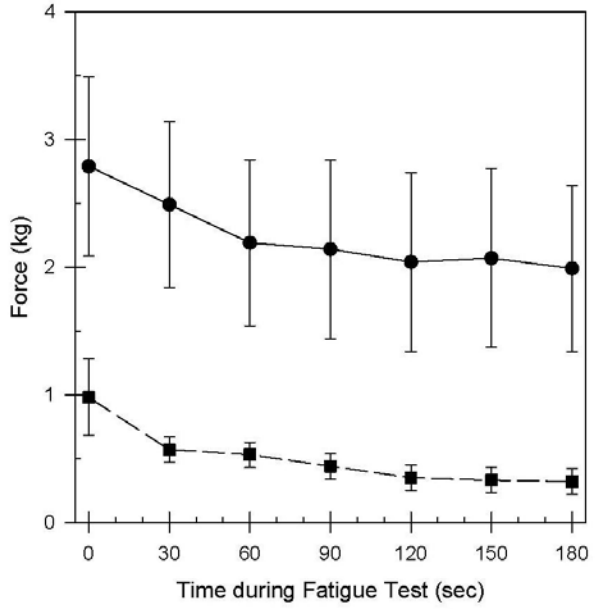

Figure 3. Fatigue protocol results of TA in SCI (■) and control subjects (●). All forces for SCI were significantly lower than those of the control subjects ($p < 0.05$). (Error bars represent SE).

The mean FI at the end of the fatigue test was 45 ± 12.0 and 67 ± 8.3 % for the SCI group and control group, respectively. Due to a high variability in the FI in both the SCI and control groups, the difference between the two groups was not significant ($p = 0.2$). The RI was not significantly different between the two groups.

Contractile properties during the fatigue protocol

½ Relaxation time

Figure 4 presents the mean ½ RT at the beginning and the end of the 3 minute fatigue test and at the end of the three minute recovery test for the SCI and control groups. At the beginning of the fatigue test (time 0-5s), the ½ RT of the SCI group was 150 ± 27.7 ms, which was significantly longer than the 81± 7.79 ms seen in the control group. A significantly longer ½ RT was also observed at the end of the fatigue test in the SCI group when compared to the control group (200 ± 31.9 ms vs. 104 ± 6.6 ms, respectively). At the end of the recovery test, the ½ RT of the SCI group was 127 ± 13.1 ms, which was significantly longer than the 81± 8.5 ms seen in the control group. The ½ RT at fatigue was significantly slower than the initial ½ RT in both the SCI and control group ($p < 0.05$). The ½ RT at the end of the three-minute recovery protocol was not significantly different than the ½ RT at the start of the fatigue protocol for either group.

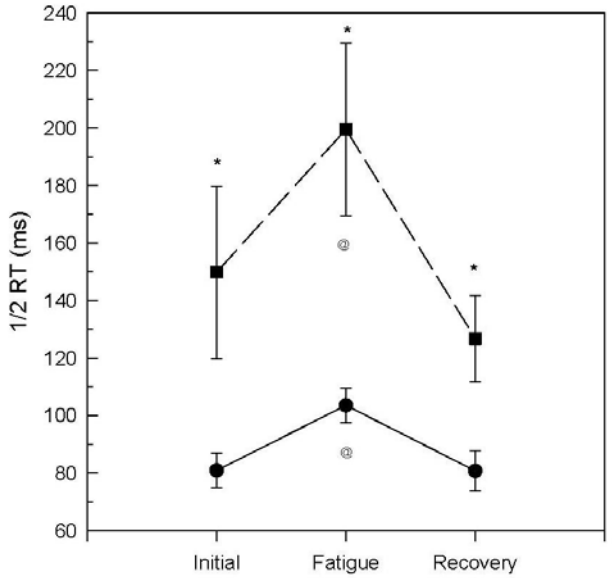

Figure 4. Half relaxation time of TA in SCI (■) and control subjects (●) at initial, fatigue, and recovery stages of the fatigue run. * Significant difference between groups, @ significantly different from initial ($p < 0.05$). (Error bars represent SE).

Rate of ½ Relaxation Time

The R½ RT was significantly slower in the SCI group when compared to the control group at the start of the fatigue test (2.8 ± 0.6 $kg \cdot s^{-1}$ vs.18.5 ± 4.1 $kg \cdot s^{-1}$, at the end of the fatigue test (0.74 ± 0.2 $kg \cdot s^{-1}$ vs. 9.2 ± 2.4 $kg \cdot s^{-1}$, and at the end of the recovery (3.5 ± 1.3 $kg \cdot s^{-1}$ vs.19.4 ± 5.3 $kg \cdot s^{-1}$ ($p < 0.05$). The R½ RT was significantly slower at the end of the fatigue test as compared to the beginning of the fatigue test in the control group ($p < 0.05$). The R½ RT at the end of the 3 minute recovery protocol was not significantly different than the R½ RT at the start of the fatigue protocol for either group.

DISCUSSION

Contractile properties - force frequency relationship, ½ RT, and TPT

The relative TA force frequency curve of the SCI patients was shifted to the left, illustrating that a lower stimulation frequency was needed to produce a similar relative force. The higher twitch/tetanus ratio, the slower ½ RT, and the decreased R½ RT at the 40 Hz tetani in the SCI group all indicate slower contractile properties in the SCI group compared to the control group. This is of special interest because numerous studies have shown that the percentage of type II fibers increases in slow and mixed muscles in SCI patients (Lotta et al., 1991; Martin et al., 1992; Rochester et al., 1995a; Round et al., 1993), consistent with a shift towards a faster muscle.

Most other studies of paralyzed human muscle reported muscle weakness, high twitch/tetanus ratios, and left shifts in the force frequency relationship, all indicators of slower muscle (Gerrits et al., 1999; 2001; Rochester et al., 1995b; Thomas, 1997). However, contractile properties such as ½ RT and TPT in disused muscle of previous studies are very contradictory. A few studies found that the contractile properties changed in the direction of a faster muscle (Gerrits et al., 1999; 2001; Rochester et al., 1995b). Rochester et al. (1995b) found that in the TA the rise time of a twitch and tetanic contraction was faster in SCI patients but ½ RT was faster and slower than the value observed in normal subjects. Gerrits et al. (1999) found faster ½ RT, faster rate of force development, and less fusion at 10 Hz stimulation in the quadriceps muscle of SCI patients when compared to controls. In another study Gerrits et al. (2001) found less fusion at 10 Hz, faster contraction time, but not a faster ½ RT.

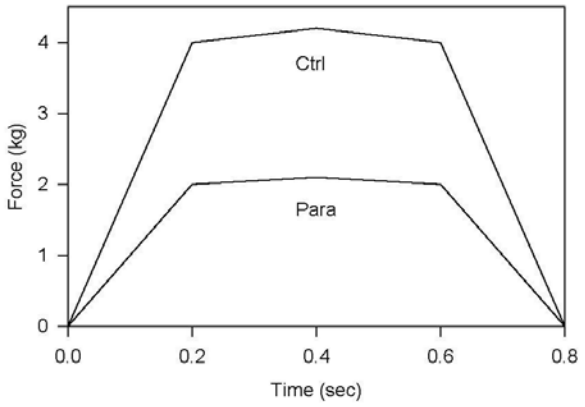

Figure 5 Example of identical ½ RT and TPT in paralyzed and control muscle. Note the slower rate of ½ RT and TPT in paralyzed muscle because of lower force production in paralyzed muscle.

It should be noted that the twitch and tetanic force produced by the muscle of the SCI patients was significantly lower than the force produced by the normal muscle in some of these studies. Therefore, the faster ½ RT and faster TPT may be artifacts of the decreased force production. Miller et al. (1982) noted a similar property in human interosseus muscle and suggested normalizing to maximal force and maximal speed of contraction. For this study we chose to normalize only to force. A more discriminating measurement of the differences between the contractile properties of paralyzed and normal muscles should be the rate of TPT and R½ RT. Figure 5 presents a stylized example where different force production, ½ RT and R½ RT lead to different conclusions. This indicates that ½ RT should not be used when force is dramatically different.

In the study by Rochester et al. (1995b), the force produced by the paralyzed group was one-third of the force produced by the controls. The twitch rise time was measured from 5-95% peak twitch amplitude and the tetanic rise time was measured from 5-50% of peak tetanic amplitude. Due to the lower force produced in the paralyzed group, the rate of rise time should be measured. One should not conclude that the paralyzed TA had a faster rise time.

Several studies reported slower contractile properties. Thomas (1997) found that the TPT and ½ RT in the thenar muscle were slower in some of the SCI patients but not in all of them. Castro et al. (2000) found slower ½ RT but no change in TPT in the quadriceps muscle of SCI patients who have been injured for less than six months. The TA in SCI patients had a slower ½ RT at the beginning of a fatigue test than the control patients (132.2 ± 61.1 for the SCI group and 108.7 ± 3.5 for the control group). Stein et al. (1992) found a comparable twitch contraction time for the TA between SCI patients and controls years after injury. Our study found that three SCI patients had relatively normal ½ RT at 40 Hz during the force-frequency relationship and the 10 and 15 Hz stimulations were unfused. The R½ RT was lower in the three SCI patients when compared to the controls because the force produced at 40 Hz was lower in those three SCI patients. The result demonstrates that contractile properties in the SCI group show great variability. For one patient, the short time between the test and the SCI (seven months) may explain the results. However, Castro et al. (2000) found slower ½ RT in SCI patients who have been injured for less than six months. Two of our subjects had been paralyzed for 10 and 16 years, yet had contractile properties similar to their age- and gender-matched controls.The only difference noted was that neither patient took anti-spasm medication despite the fact that both patients had strong spasms. Dietz et al. (1995) observed that complete SCI patients who took anti-spasm medication (cannabinoids) had less EMG activity and no training effect after five months of treadmill training. It was suggested that human spinal locomotor activity could be influenced by pharmacological drug intake. It is possible that the two patients in this study who had not taken anti spasm drugs recently had different activity levels in their TA, which was responsible for the “normal” contractile properties and lack of fusion at 10 and 15 Hz stimulations. All other patients took anti-spasm medication and their ½ RT were longer than normal and the 10 and 15 Hz stimuli were fused. It is possible that the use of the anti-spasm medication changes the activity of the muscle and consequently the contractile properties, although further studies

are needed to support this suggestion. There is also the possibility that lower blood flow in the SCI could contribute to accelerated fatigue and the 2 min recovery was not sufficient, leading to slowed contraction and relaxation times. We do not believe this to be the case. As mentioned in the methods, no SCI subject had a difference of more than 5% in the forces in the two force/frequency curves.

Animal studies

While not measured in the present study, animal research generally supports that myosin ATPase changes to a faster type with disuse (Lotta et al., 1991; Martin et al., 1992; Rochester et al., 1995a; Round et al., 1993). However, the research on contractile properties is contradictory. Most studies found a left shift in the force-frequency curve indicating that the muscle is slower since more fusion was seen at a lower stimulation frequency (Gerrits et al., 1999; 2001; Rochester et al., 1995b; Thomas, 1997). Studies have found both slower and faster ½ RT and TPT (Castro et al., 2000; Gerrits et al., 1999; 2001; Thomas, 1997). It seems that the activity of myofibrillar ATPase may be unreliable as an indication of the contraction time in chronically disused muscle.

If disuse transforms the myosin ATPase to a faster type, but not all studies find faster contractile properties, then something else must be responsible for the change in contractile properties. One possibility is that disuse changes calcium handling (Castro et al., 2000; Gerrits et al., 2000a; Stein et al., 1992; Stevens and Mounier, 1992). Stevens and Mounier (1992) found that hindlimb suspension increased Ca^{2+} uptake and Ca^{2+} release in the rats soleus muscle.

Two other potential explanations for some of the slowing in ½ RT could be related to the following factors. First, a difference in muscle temperature may have led to slower contractile properties. Gerrits et al. (2000b) found that the core temperature of SCI patients was lower than the core temperature in controls. The heating of the SCI muscle to normal core temperature with ultrasound significantly increased the contractile speed. It is possible that a lower core temperature in the SCI patients may have been responsible for some slowing of the contractile properties. Second, the stimuli given to establish appropriate electrode placement and the stimuli given at the frequencies before the 40 Hz stimulus may have induced some contractile slowing. Dubose et al. (1987) found almost immediate slowing of fast motor units before any changes were noted in force. However, in this study the SCI patients were given a two minute rest interval between each stimulus, therefore it seems unlikely that slowing of contractile properties is due to early fatigue in the SCI group, as discussed earlier. An additional verification of no fatigue from the force/frequency test was that lack of difference between the two trials.

Fatigue properties

The muscles of the SCI group fatigued to 45 ± 12.0 % of their initial value, which was not significantly different from the 67 ± 8.3 % observed in the control group. We found a great variability in both the SCI and control group. The FI of some SCI patients were as low as 0 and 8 % but some were as high as 78 and 83 % which was very close to the range seen in the controls. Because of this large variability no significant difference was found between the two groups. Neither the level of injury nor the length of time after injury appeared to be related to the force output at the end of the fatigue test. The loss of force in this study in the control subjects was similar to that found by Reid et al. (1994), who found force at 3 min of 40-60% of time 0.

These results differ from the findings of previous studies, which reported increased fatigability in SCI patients (Shield, 1995; Shield et al., 1997). However, those studies were done on human paralyzed soleus muscle. Miller et al. (1982) tested the quadriceps muscle of 9 paraplegic patients and found great variability of the residual torque output in SCI patients (from 7.13 to 53.54% of initial output). The level of injury and the time after the injury were not correlated with the residual torque output. Gerrits et al. (1999) found that a 2 minute fatigue protocol decreased the force to 41± 7 % in the SCI group and 65 ± 10% in the control group, showing a significantly greater decline in torque production in the quadriceps muscles of SCI patients. Other studies found a low fatigue resistance in the TA of SCI patients (Lenman et al., 1989) and multiple sclerosis patients (Kent-Braun et al., 1997; Lenman et al., 1989). It was proposed that the low SDH activity and capillary-to-fiber ratio in the TA in these patients were contributing to their fatigability (Kent-Braun et al., 1997; Martin et al, 1992). The previous results, particularly those of Gerrits et al. (1999) are similar to ours, but we report no significant difference.

Contractile properties during the fatigue protocol

½ Relaxation time: In this study the ½ RT was significantly longer at the start and the end of the fatigue test and at the end of the recovery period in the SCI group when compared to the controls. In addition both groups had significantly longer ½ RT at the end of the fatigue test compared to the start of the test. However, two of the SCI patients who had close to control ½ RT (75 and 84 ms), also had much more slowing of ½ RT during the fatigue test. Two

out of the eight SCI patients behaved differently. Reasons for the observed results in the two SCI patients are not clear. One can speculate that the longer ½ RT in the other SCI patients at the beginning of the fatigue test may have been the reason that the ½ RT did not slow more during the fatigue test. In the two SCI patients with times since spinal injury of 10 and 16 years, the only difference was that these two patients did not take anti spasm medication.

Castro et al. (2000) also found a significantly slower ½ RT of the quadriceps femoris muscle at the beginning of the fatigue test in SCI patients who were injured for less than six months. In addition, slowing of ½ RT with force loss during the fatigue test was observed at 24 weeks, but not at six or 11 weeks after SCI. However, the control group showed slowing of ½ RT at both time points.

Rate of ½ RT: The R½ RT takes the low forces produced by the SCI patients into consideration. The SCI group had a significantly slower R½ RT than the control group test (2.8 ± 0.6 $kg \cdot s^{-1}$ vs.18.5 ± 4.1 $kg \cdot s^{-1}$) at the start of the fatigue test. This large difference really shows how slow the TA of SCI patients are at the start of the fatigue test. The R½ RT was significantly lower at the end of the fatigue test when compared to the beginning of the fatigue test in the control group only. The reason why the R½ RT is not significantly lower at the end of the fatigue test in the SCI group, is the very low numbers of the R½ RT in the SCI group.

CONCLUSIONS

The major finding of this study was that the TA in SCI patients had slower contractile properties than the control group. The variability was very high in the SCI group, with three patients having close to normal ½ RT and the other five being much slower. It can be concluded that not all muscle responds the same way to SCI. The slowing of the ½ RT during the fatigue test was seen in both groups. However, variability in the SCI group was very high.

The contractile properties of the SCI patients were different than those of the controls. Treatments to prevent those changes are a prerequisite for rehabilitation in patients who suffered neurological or orthopedic trauma. Our study found differences in the muscle's reaction to disuse, consequently it is important that treatments and rehabilitative procedures are tailored specifically to the patient.

Further, it appears that myosin ATPase activity may not be a clear indicator of the contractile properties of disused muscle in SCI. Many other factors can influence the properties such as synaptic function, sacroplasmic reticulum properties, excitation-contraction coupling, contractile machinery interaction, muscle fiber size and enzymatic activity. Disused muscle cannot be treated like normal muscle. Changes in calcium handling process and enzymatic activity, other protein changes, in addition to myosin ATPase, need to be investigated in future studies in order to clarify the conflicting results.

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KEY POINTS

- Stimulated contractions were tested on controls and spinal cord injured subjects to determine differences in contractile characteristics of the tibialis anterior (ta) muscle.
- Forces were lower in the ta of the spinal cord injured subjects compared to the controls.
- All indices of contractile speed were slower in the spinal cord injured subjects than in the controls.
- The reason for possible differences in contractile capabilities and other biochemical indices of contractile speed in disused muscle need to be further evaluated.

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Appendix Table. This table shows ANOVA results for data collected during the fatigue test.

1. Two way repeated measures ANOVA, Dependent Variable – force.

Source	DF	SS	MS	F	P
Subject	7	55.020	7.860		
Condition	1	85.139	85.139	10.183	0.015
Cond X Subj	7	58.526	8.361		
Time	6	6.392	1.065	14.288	<0.001
Time X Subj	42	3.132	0.0746		
Cond X Time	6	0.217	0.0362	0.356	0.902
Residual	42	4.264	0.102		
Total	111	212.689	1.916		

2. Two way repeated measures ANOVA, Dependent Variable – ½ RT.

Source	DF	SS	MS	F	P
Subject	7	44947.917	6421.131		
Condition	1	59361.333	59361.333	15.906	0.005
Cond X Subj	7	26124.000	3732.000		
Time	2	19874.000	9937.000	6.381	0.011
Time X Subj	14	21801.333	1557.238		
Cond X Time	2	5010.667	2505.333	1.324	0.297
Residual	14	26484.000	1891.714		
Total	47	203603.250	4331.984		

3. Two way repeated measures ANOVA, Dependent Variable – R ½ RT.

Source	DF	SS	MS	F	P
Subject	7	1574.625	224.946		
Condition	1	2148.962	2148.962	14.624	0.007
Cond X Subj	7	1028.624	146.946		
Time	2	391.705	195.852	10.916	0.001
Time X Subj	14	251.193	17.942		
Cond X Time	2	136.120	68.060	7.767	0.005
Residual	14	122.674	8.762		
Total	47	5653.903	120.296		