

## ORIGINAL ARTICLE

# Muscle fatigue characteristics in paralyzed muscle after spinal cord injury

CA Pelletier and AL Hicks

Department of Kinesiology, McMaster University, Hamilton, Ontario, Canada

**Study design:** The study design used is cross-sectional.

**Objectives:** The aim of this study is to examine muscle contractile and excitability characteristics during fatigue of the tibialis anterior in six individuals with chronic spinal cord injury (SCI) and matched able-bodied (AB) controls.

**Setting:** McMaster University, Hamilton, ON, Canada.

**Methods:** Muscle compound action potential (M-wave) characteristics, muscle twitch properties, and summated force were examined during a 2 min fatigue protocol of intermittent bursts at 30 Hz (4 s tetanus, 2 s rest) or maximal voluntary contraction (MVC). Evoked twitch responses were followed during a recovery period.

**Results:** M-wave amplitude was smaller in SCI ( $2.5 \pm 1.6$  mV in SCI,  $5.7 \pm 3.2$  mV in AB) at baseline, but there was no change in M-wave amplitude or area during fatigue in either group. There was an increase in M-wave duration toward the end of recovery in the SCI group. Peak torque (PT) was not different between groups at baseline ( $3.8 \pm 1.8$  Nm in SCI,  $3.7 \pm 0.6$  Nm in AB); PT potentiated significantly during fatigue in the AB, but not SCI group. There was significantly greater fatigue of both PT (43% decline) and summated force (57% decline) in the SCI group compared with the AB group (13% increase and 22% decline for PT and MVC, respectively).

**Conclusion:** The dorsiflexor muscles in people with SCI are significantly more fatiguable than those in AB controls, but decreases in muscle excitability do not seem to be an important contributor to the increased fatigability. The mechanisms behind the increased fatigue must lie distal to the muscle membrane.

*Spinal Cord* (2011) 49, 125–130; doi:10.1038/sc.2010.62; published online 8 June 2010

**Keywords:** muscle fatigue; contractile properties; electrical stimulation; spinal cord injury

## Introduction

One of the most important implications of the changes that occur to skeletal muscle after spinal cord injury (SCI) is a decreased fatigue resistance in the paralyzed muscle.<sup>1,2</sup> This results in many functional limitations and can inhibit rehabilitation efforts. Fatigue resistance has been shown to decrease rapidly for the first 1.7 years post-injury, with smaller decreases occurring in subsequent years.<sup>3</sup>

Although the mechanism for the increased muscle fatigability in the SCI population is unknown, a current theory of neuromuscular fatigue in the able-bodied (AB) population involves a decrease in muscle excitability due to increased extracellular  $K^+$ . The sodium potassium pump ( $Na^+/K^+$  ATPase) is largely responsible for maintaining concentration gradients of  $Na^+$  and  $K^+$  across the sarcolemmal membrane,

allowing for muscle excitability to be maintained during repetitive activation. When an action potential arrives at the membrane, depolarization is required to elicit a contraction. Given the small extracellular space surrounding muscle fibers, repeated depolarization can quickly lead to significant changes in the extracellular concentration of  $K^+$ , which would be predicted to affect membrane excitability.<sup>4</sup>

Earlier work has shown a substantial decrease in  $Na^+/K^+$  ATPase concentration in the vastus lateralis of people with complete SCI;<sup>5</sup> however, it is not known whether this decrease in pump concentration has any significant effect on membrane excitability or fatigue resistance.

Muscle membrane excitability can be non-invasively assessed with the compound muscle action potential (M-wave), and changes in M-wave amplitude and area have been used as an indirect measure of  $Na^+/K^+$  ATPase activity.<sup>4,6</sup>

To date, the potential role of muscle excitability in contributing to fatigability in paralyzed muscle has only been examined in the soleus muscle. Shields<sup>2</sup> concluded that changes in muscle membrane excitability were not solely

Correspondence: Dr AL Hicks, Department of Kinesiology, McMaster University, 1280 Main Street West, Hamilton, Ontario, Canada L8S 4K1.  
E-mail: hicksal@mcmaster.ca

Received 1 February 2010; revised 8 April 2010; accepted 23 April 2010; published online 8 June 2010

responsible for muscle fatigue as decreases in M-wave amplitude did not coincide with decreases in torque. A later study, also in the soleus muscle, found a 20% decrease in M-wave amplitude, a 40% increase in M-wave duration, no change in M-wave area, and an 80% decrease in peak torque (PT) after intermittent tetanic stimulation in participants with chronic, but not acute (<6 weeks post-injury) SCI.<sup>7</sup> Furthermore, M-wave amplitude was found to completely recover 5 min after the stimulation trial, whereas PT remained depressed.<sup>7</sup>

Contrary to the extensive body of literature pertaining to muscle fatigue in AB human beings, the various mechanisms behind neuromuscular fatigue have not been fully resolved in paralyzed muscle. Earlier work has shown markers of oxidative capacity to be correlated with increased muscle fatigue in participants with SCI.<sup>8–10</sup> Other possibilities include the fiber-type transformation in which paralyzed muscle experiences a shift to a predominance of type II fatiguable fibers<sup>8–11</sup> and impairments in Ca<sup>2+</sup> handling.<sup>12</sup> The limited number of studies examining the mechanisms of fatigue in paralyzed muscle combined with the highly variable nature of SCI makes it difficult to draw any definitive conclusions. Characteristics of muscle fatigue in people with SCI seem to vary with fatigue protocol, stimulation intensity, and the muscle being investigated. The purpose of this study was to examine the potential role of a decline in muscle excitability as a contributor to impaired fatigue resistance in the ankle dorsiflexors in people with SCI. M-wave changes during a period of intermittent tetanic stimulation were assessed in the ankle dorsiflexor muscles of people with SCI, and compared with a similar protocol in AB individuals. It was predicted that the SCI group would show a greater decrease in M-wave amplitude throughout the fatigue protocol when compared with AB controls. It was also predicted that the SCI group would show decreased fatigue resistance as evidenced by a more rapid and significant decline in PT and a slower recovery.

## Materials and methods

### Participants

Six participants with chronic SCI (5 male, 1 female; age 44 ± 13.9 years) and six age- and sex-matched AB controls (age 45 ± 14.3 years) volunteered for this study. All participants were recreationally active and none had participated in any form of muscle stimulation training. Characteristics of the study participants with SCI are summarized in Table 1. Inclusion criteria for the participants with SCI included >1 year post-injury, upper motor neuron lesion level higher than T8, American Spinal Injury Association classification A–C, and wheelchair dependent. Informed consent was obtained from all participants and procedures were approved by the McMaster University Research Ethics Board.

### Apparatus

All measurements were conducted on the right ankle dorsiflexor muscle. Subjects either remained in their wheel-

**Table 1** SCI group characteristics

Participant	Age	Sex	ASIA classification	Level of injury	Years since injury
1	55	M	C	C3	14
2	33	M	A	C6/7	14
3	42	M	A	T7	4
4	49	M	A	C5/6/7	2
5	24	M	A	C6	1.5
6	61	F	C	T1	1.5

Abbreviations: ASIA, American Spinal Injury Association; SCI, spinal cord injury.

chair (SCI) or a height-adjustable chair (AB) with the right knee flexed at 90° and the lower leg and foot secured in an isometric boot apparatus. For both the SCI and AB participants, the ankle was placed at 20° PF. This angle has been previously determined as optimal for evoked dorsiflexor torque in the SCI and AB populations.<sup>13</sup> The common peroneal nerve was stimulated with rubber-stimulating electrodes (3 cm diameter). Muscle electrical activity was collected using two surface electromyogram electrodes (Kendall Medi-Trace 133, Mansfield, MA, USA) placed 5 cm apart on the muscle belly of the tibialis anterior.

A constant current stimulator (Digitimer Ltd, Model 3072, Welwyn Garden City, Hertfordshire, England) was used to deliver single 50 μs rectangular pulses and 4 s tetanic trains (30 Hz) to the peroneal nerve for assessment of peak evoked twitch (PT) and tetanic torque (TT), respectively. The stimulation frequency for the tetanus (30 Hz) was selected based on the plateau of the force–frequency relationship for the tibialis anterior in people with SCI previously determined in our laboratory.<sup>14</sup> The optimal stimulation intensity was determined for each participant by gradually increasing the current until there were no further increases in evoked twitch torque.

### Procedure

Muscle contractile properties and M-wave characteristics were assessed before, during, and after a 2 min fatigue trial of intermittent tetanic stimulation in the SCI group, and a similar trial of intermittent maximal voluntary contractions (MVCs) in the AB group. The fatigue protocol consisted of 4 s tetanus at 30 Hz (or MVC), followed by 2 s rest. A single muscle twitch was elicited half-way through each 2 s rest period. A 15 min recovery period followed the fatigue protocol and consisted of 17 addition twitches spaced at increasing intervals. Specifically, the muscle contractile properties measured were peak evoked twitch torque (PT), maximum compound action potential amplitude (M-wave amplitude), area (M-wave area), duration (M-wave duration), time to PT, maximum rate of torque development,  $\frac{1}{2}$  relaxation time, and maximum rate for torque relaxation. A fatigue index (% decline in torque) was calculated for both the decline in PT and decline in TT or MVC, using the following formula:  $(\text{Torque}_{\text{baseline}} - \text{Torque}_{2\text{min}}) / \text{Torque}_{\text{baseline}} \times 100$ .

**Table 2** M-wave characteristics at baseline, during a 2 min fatigue protocol, and during a 15 min recovery period

	Baseline	Post-fatigue	1 min recovery	5 min recovery	15 min recovery
<b>Amplitude (mV)</b>					
SCI	2.5 ± 1.6 <sup>a</sup>	2.9 ± 2.4	2.9 ± 1.9	2.9 ± 2.1	2.7 ± 1.9
AB	5.7 ± 3.2	5.9 ± 3.8	5.7 ± 3.7	5.7 ± 3.6	5.9 ± 3.4
<b>Area (mV · ms)</b>					
SCI	24.9 ± 14.8	30.8 ± 23.2	27.4 ± 15.7	32.0 ± 19.9	29.0 ± 20.6
AB	34.8 ± 30.5	35.9 ± 26.0	37.8 ± 31.0	34.4 ± 30.4	32.5 ± 28.2
<b>Duration (ms)</b>					
SCI	34.9 ± 9.1	44.8 ± 18.2	42.4 ± 12.1	53.9 ± 22.5 <sup>a</sup>	47.3 ± 12.5 <sup>a</sup>
AB	27.8 ± 7.3	28.9 ± 3.3	31.7 ± 8.1	28.6 ± 9.8	25.5 ± 7.1

Abbreviations: AB, able-bodied; SCI, spinal cord injury.

<sup>a</sup>Indicates a significant difference between groups ( $P < 0.05$ ). Values are mean ± s.d.

### Statistical treatment

Muscle fatigue characteristics were analyzed with a two-factor analysis of variance with one between subjects factor (SCI vs AB) and one within subjects factor (time). Significance was set at  $P < 0.05$ , and Tukey *A post hoc* tests were used to examine significant differences between means. Throughout the text, values are reported as mean ± s.d. and mean ± s.e. in figures.

## Results

### M-wave characteristics

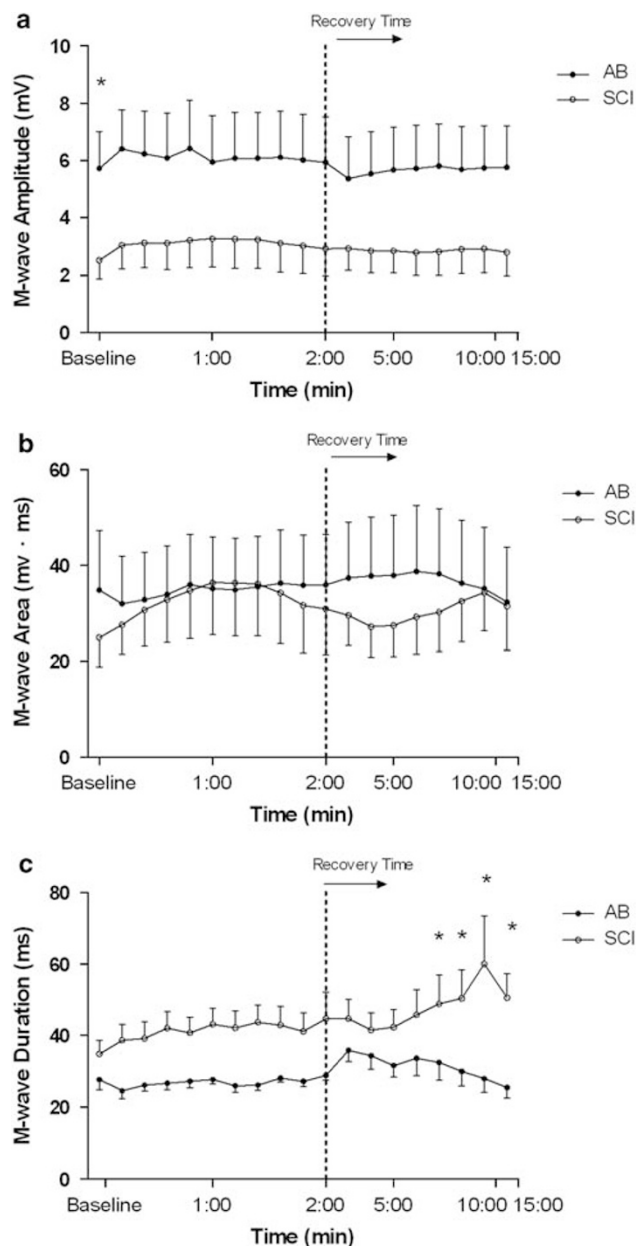
M-wave amplitude was greater in the AB group at baseline with values of  $2.5 \pm 1.6$  mV in SCI and  $5.7 \pm 3.2$  mV in AB. There was no significant difference in M-wave duration ( $34.9 \pm 9.1$  in SCI,  $27.8 \pm 7.3$  in AB) or area noted between groups at baseline. In both the SCI and AB groups, M-wave amplitude and area were maintained throughout the fatigue protocol and through recovery. M-wave duration increased during the recovery period in the SCI group only. These results are shown in Table 2 and Figure 1.

### Summated torque

Both groups showed decreases in TT or MVC (Figure 2) from baseline to the end of the fatigue protocol, with a significantly greater fatigue index being observed in SCI compared with AB ( $57.2 \pm 19.9\%$  vs  $22.4 \pm 13.9\%$ , respectively). Fatigue index results are shown in Figure 3.

### Muscle contractile properties

Changes in muscle contractile properties from baseline through to the end of the recovery stage are depicted in Table 3. There was no significant difference in PT between groups at baseline ( $3.8 \pm 1.8$  Nm in SCI,  $3.7 \pm 0.6$  Nm in AB); however, both groups responded differently to the fatigue protocol. There was significant twitch potentiation noted in the AB group (Figure 4) that lasted throughout the 2-min fatigue protocol. In contrast, despite some early potentiation of PT in the SCI group (not statistically significant), twitch



**Figure 1** M-wave amplitude (a), area (b), and duration (c) during a 2 min fatigue protocol followed by a 15 min recovery period in AB and SCI groups. The dotted horizontal line represents the transition between fatigue and recovery. Error bars represent s.e. \*Denotes significant difference between groups ( $P < 0.05$ ).

torque dropped below baseline within 1 min of the fatigue protocol and was significantly decreased from baseline at the end of fatigue. A significantly ( $P < 0.05$ ) greater fatigue index for PT was observed in the SCI group ( $42.8 \pm 41.3\%$  in SCI,  $-13.2 \pm 22.9\%$  in AB). With regard to contractile speed, both time to PT and maximum rate of torque development were similar between groups at baseline; however, maximum rate of torque development slowed significantly in the SCI group over the course of fatigue (Table 3). Similarly, maximum rate for torque relaxation and  $\frac{1}{2}$  relaxation time were not significantly different between groups at baseline, but there

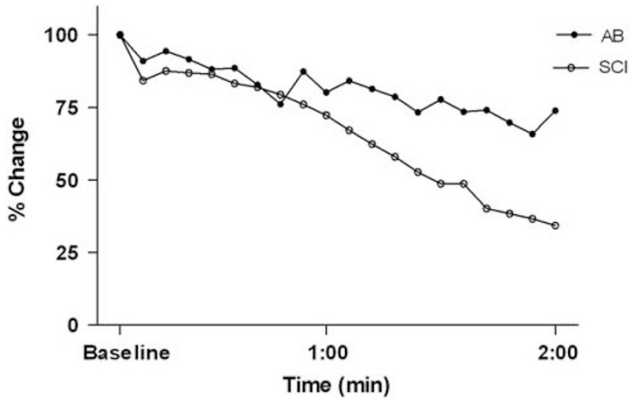


Figure 2 Percent change in tetanic torque and maximal voluntary contraction torque during a 2 min fatigue protocol in AB and SCI groups.

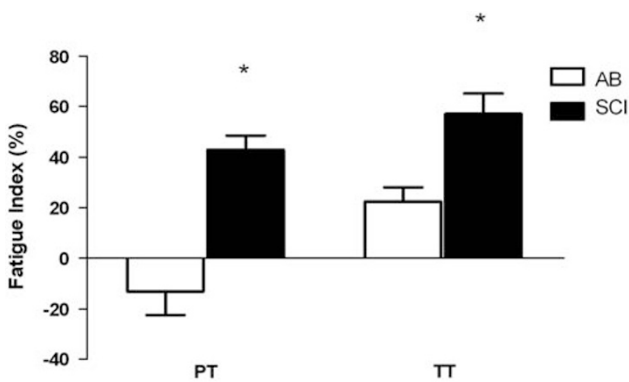


Figure 3 Fatigue index for AB and SCI groups after a 2 min fatigue protocol. Error bars represent s.e. \*Denotes significant difference between groups ( $P < 0.05$ ).

was a trend for relaxation rates to slow in the SCI group at the end of fatigue ( $28.9 \pm 28.7 \text{ Nm s}^{-1}$  in SCI,  $71.7 \pm 38.7 \text{ Nm s}^{-1}$  in AB;  $P = 0.054$ ).

Discussion

The findings of this study suggest that changes in muscle excitability are not a major contributing factor to the decrease in fatigue resistance after SCI. Although the SCI group displayed a greater fatigue index for both PT and TT, there was no indication of a decrease in muscle excitability

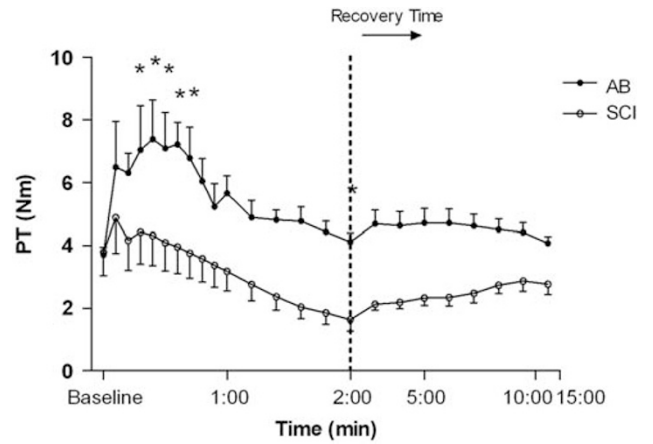


Figure 4 Peak twitch torque at baseline, during a 2 min fatigue protocol, and 15 min recovery period in AB and SCI groups. The horizontal dotted line represents the end of the fatigue protocol. Error bars represent s.e. \*Denotes significant difference between groups ( $P < 0.05$ ).

Table 3 Muscle contractile properties before and after a 2 min fatigue protocol and 15 min recovery period

	Baseline	Post-fatigue	1 min recovery	5 min recovery	15 min recovery
<i>PT (Nm)</i>					
SCI	3.8 ± 1.8	1.6 ± 0.9 <sup>a</sup>	2.3 ± 0.6	2.7 ± 0.8	2.6 ± 0.9
AB	3.7 ± 0.6	4.1 ± 0.7	4.7 ± 1.1	4.3 ± 0.7	3.7 ± 0.5
<i>RTD (Nm s<sup>-1</sup>)</i>					
SCI	116.6 ± 38.5	67.4 ± 55.4 <sup>a</sup>	94.9 ± 55.0	93.4 ± 50.9	89.0 ± 34.1
AB	135.2 ± 83.4	178.2 ± 2.5	185.7 ± 4.9	164.5 ± 89.0	128.9 ± 67.3
<i>RTR (Nm s<sup>-1</sup>)</i>					
SCI	42.2 ± 18.9	28.9 ± 28.7	35.1 ± 35.9	39.2 ± 33.0	41.9 ± 35.8
AB	50.0 ± 48.5	71.7 ± 38.7	54.9 ± 29.2	74.0 ± 52.1	67.1 ± 39.8
<i>TPT (ms)</i>					
SCI	96.2 ± 26.2	140.7 ± 67.1	119.9 ± 59.5	126.0 ± 72.0	107.0 ± 57.0
AB	94.7 ± 17.7	87.9 ± 18.2	119.8 ± 20.6	115.2 ± 24.5	100.3 ± 18.4
<i>½ RT (ms)</i>					
SCI	108.0 ± 28.3	178.2 ± 28.2	145.7 ± 27.5	149.9 ± 35.5	143.7 ± 54.2
AB	87.0 ± 13.0	110.2 ± 41.4	110.0 ± 13.2	104.2 ± 25.1	88.7 ± 18.3
<i>TT or MVC (Nm)</i>					
SCI	10.5 ± 6.6	3.6 ± 1.1	—	—	—
AB	38.6 ± 15.6	29.8 ± 5.5	—	—	—

Abbreviations: AB, able-bodied; MVC, maximal voluntary contraction; PT, peak torque; RTD, maximum rate of torque development; RTR, maximum rate of torque relaxation; SCI, spinal cord injury; TPT, time to peak torque; TT, tetanic torque; ½ RT, ½ relaxation time.

<sup>a</sup>Indicates a significant difference between groups ( $P < 0.05$ ). Values are mean ± s.d.

as assessed by changes in M-wave amplitude or area. These findings are similar to earlier work that has shown changes in M-wave characteristics to be disassociated with changes in muscle torque during fatigue in the paralyzed soleus muscle.<sup>7</sup>

M-wave amplitude was greater in the AB group at baseline, which supports earlier findings<sup>13</sup> and is likely a reflection of the significant muscle atrophy after SCI. The fact that M-wave amplitude and area stayed relatively constant over the course of fatigue in the SCI group suggests that the previously reported decrease in  $\text{Na}^+/\text{K}^+$  pump concentration after SCI does not contribute to muscle fatigability.<sup>5</sup> Muscle membrane excitability seemed to be maintained quite effectively in paralyzed muscle during the 2-min fatigue protocol. Although there was an increase in M-wave area in the SCI group, this was likely associated with the increase in M-wave duration rather than increased  $\text{Na}^+/\text{K}^+$  pump activity, as there was no corresponding increase in M-wave amplitude. The increasing M-wave duration in the SCI group during recovery is suggestive of a slowing of action potential conduction across the sarcolemma membrane, a finding that has been demonstrated previously in the soleus muscle.<sup>7</sup>

As predicted, the SCI group showed increased fatigue compared with the AB group as evidenced with an increased fatigue index for both PT and TT. This increased fatigability in paralyzed muscle has been reported previously in the soleus and quadriceps muscle groups.<sup>1-3</sup> However, based on the M-wave results of this study, and from earlier work,<sup>2,7</sup> a failure in  $\text{Na}^+/\text{K}^+$  pump activity does not appear to play a significant role in the increased fatigability during a relatively short period of maximum stimulation in people with SCI. This was an unexpected finding given the reported decrease in  $\text{Na}^+/\text{K}^+$  pump concentration after SCI.<sup>5</sup> One possible explanation may be that the previously reported decrease in  $\text{Na}^+/\text{K}^+$  pump concentration in the vastus lateralis muscle is not transferable to the tibialis anterior muscle. An alternative possibility is that despite a decrease in pump concentration, an increase in the efficiency of the remaining  $\text{Na}^+/\text{K}^+$  pumps may assist in maintaining membrane excitability. As muscle excitability does not seem to be playing a major role in the fatigability of paralyzed muscle, it is important to consider the involvement of other mechanisms for fatigue. Muscle oxidative capacity, which provides an indication for the potential for aerobic metabolism, has been previously shown to decrease after SCI and has been correlated with an increase in fatigability in the tibialis anterior<sup>8,9</sup> and vastus lateralis.<sup>10</sup> Another common theory of muscle fatigue involves the impaired control of calcium ( $\text{Ca}^{2+}$ ) release into the sarcoplasmic reticulum.<sup>15</sup> Alterations in the function and concentration of  $\text{Ca}^{2+}$  ATPase have been suggested after SCI to explain the slowing of relaxation time<sup>12</sup> and changes in sarcoplasmic reticulum  $\text{Ca}^{2+}$  ATPase isoform expression.<sup>11</sup>

The phenomenon of enhanced force after a brief maximal summated contraction is known as post-activation potentiation or post-tetanic potentiation, depending on whether the stimulus is voluntary (post-activation potentiation) or electrically stimulated (post-tetanic potentiation). Potentiation can prevail over muscle fatigue, particularly in muscles with a high percentage of type II fibers.<sup>16,17</sup> In the present

study, there was a significant post-activation potentiation observed in the AB group, whereas the SCI group showed only a brief period of potentiation. This result is contrary to what was expected due to the reported fiber-type transformation after SCI.<sup>8-11</sup> Although the different fatigue protocols (electrically induced vs voluntary) may be a contributing factor to the group differences, twitch responses have been shown to be similar regardless of the method used to induce potentiation.<sup>18</sup> Thus, it appears that paralyzed muscle is unable to maintain twitch potentiation and succumbs to fatigue sooner than AB controls during periods of intermittent maximal contraction.

The results of this study extend the findings of increased fatigability in paralyzed muscle after SCI to the ankle dorsiflexor muscle group. During 2 min of intermittent tetanic stimulation, a decrease in muscle excitability does not appear to be a significant contributing factor to the decreased fatigue resistance after SCI; the mechanisms more likely relate to changes in excitation-contraction coupling and/or muscle oxidative capacity. It is important that future research further explore the mechanisms behind the decreased fatigue resistance after SCI and to consider the associated clinical implications. As many rehabilitation strategies (for example, functional electrical stimulation) are dependent on the muscle being able to withstand repeated contractions and to delay the onset of fatigue, a greater understanding of the process of muscle fatigue in paralyzed muscles will assist in the optimization of rehabilitation efforts.

## Conflict of interest

The authors declare no conflict of interest.

## References

- 1 Gerrits HL, de Haan A, Hopman M, van der Woude LHV, Jones DA, Sargeant AJ. Contractile properties of the quadriceps muscle in individuals with spinal cord injury. *Muscle Nerve* 1999; **22**: 1249-1256.
- 2 Shields RK. Fatigability, relaxation properties, and electromyographic responses of the human paralyzed soleus muscle. *J Neurophysiol* 1995; **73**: 2195-2206.
- 3 Shields RK, Chang Y, Dudley-Javoroski S, Lin C. Predictive model of muscle fatigue after spinal cord injury in humans. *Muscle Nerve* 2006; **34**: 84-91.
- 4 Hicks A, McComas AJ. Increased sodium pump activity following repetitive stimulation of rat soleus muscles. *J Physiol* 1989; **414**: 337-349.
- 5 Ditor DS, Hamilton S, Tarnopolsky MA, Green HJ, Craven BC, Parise G *et al.*  $\text{Na}^+$ ,  $\text{K}^+$ -ATPase concentration fiber type distribution after spinal cord injury. *Muscle Nerve* 2004; **29**: 38-45.
- 6 Hicks A, Fenton J, Garner S, McComas AJ. M wave potentiation during and after muscle activity. *J Appl Physiol* 1989; **66**: 2606-2610.
- 7 Shields RK, Chang Y, Ross M. Neuromuscular propagation after fatiguing contractions of the paralyzed soleus muscle in humans. *Muscle Nerve* 1998; **21**: 776-787.
- 8 Martin TP, Stein RB, Hoepfner PH, Reid DC. Influence of electrical stimulation on the morphological and metabolic properties of paralyzed muscle. *J Appl Physiol* 1992; **72**: 1401-1406.
- 9 Rochester L, Chandler CS, Johnson MA, Sutton RA, Miller S. Influence of electrical stimulation of the tibialis anterior muscle in paraplegic subjects. 1. Contractile properties. *Paraplegia* 1995; **33**: 437-449.

- 10 Gerrits HL, Hopman MTE, Offringa C, Engelen BGM, Sargeant AJ, Jones DA *et al.* Variability in fibre properties in paralysed human quadriceps muscles and effects of training. *Pflugers Arch* 2003; **445**: 734–740.
- 11 Talmadge RJ, Castro MJ, Apple DF, Dudley GA. Phenotypic adaptations in human muscle fibers 6 and 24 wk after spinal cord injury. *J Appl Physiol* 2002; **92**: 147–154.
- 12 Castro MJ, Apple DF, Rogers S, Dudley GA. Influence of complete spinal cord injury on skeletal muscle mechanics within the first 6 months of injury. *Eur J Appl Physiol* 2000; **81**: 128–131.
- 13 Pelletier CA, Hicks AL. The length-tension relationship of human dorsiflexor and plantarflexor muscles following spinal cord injury. *Spinal Cord* 2010; **48**: 202–206.
- 14 Rodrigues L, Pilutti L, Hicks A. Force-frequency relation and fatigue of paralyzed human tibialis anterior muscle. *Appl Physiol Nutr Metab* 2007; **32**: S75.
- 15 Favero TG. Sarcoplasmic reticulum  $Ca^{2+}$  release and muscle fatigue. *J Appl Physiol* 1999; **87**: 471–483.
- 16 Hamada T, Sale JD, MacDougall JD, Tarnopolsky MA. Interaction of fibre type, potentiation, and fatigue in human knee extensor muscles. *Acta Physiol Scand* 2003; **178**: 165–173.
- 17 Vandervoort AA, Quinlan J, McComas AJ. Twitch potentiation after voluntary contraction. *Exp Neurol* 1983; **81**: 141–152.
- 18 Alway SE, Hughson RL, Green HJ, Patla AE, Frank JS. Twitch potentiation after fatiguing exercise in man. *Eur J Appl Physiol* 1987; **56**: 461–466.