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Original Article

Abductor hallucis for monitoring lower-limb recovery after spinal cord injury in man

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Study design: Electromyogram (EMG) study on patients with acute spinal cord injury (SCI).
Objectives: We hypothesized that subjects with mild to moderate acute SCI would have a higher probability of recovering function in intrinsic muscles of the foot compared to more proximal lower-limb muscles, based on the relative density of corticospinal tract innervation to these different motoneuron pools.
Setting: Miami and Syracuse, USA.
Methods: We conducted repeated measures of EMG during voluntary contractions from lower-limb muscles in subjects with acute traumatic SCI. For this study, analysis was restricted to those subjects who had either no recruitment (ie 'motor-complete') or limited recruitment (ie 'motor-incomplete') in any lower-limb muscle of either leg during the initial evaluation, and all of whom had converted to a motor-incomplete status in one or both legs at the time of final evaluation. Recruitment of the abductor hallucis (AbH) muscle during contraction attempts was judged as being either 'present' or 'absent', based upon the presence or absence of EMG-based volitional motor unit recruitment.
Results: A total of 70 subjects were included in this study. Of these, 58 had motor-incomplete injury at or rostral to the T10 vertebral level, and another 12 had injury caudal to T10. In the former group, the AbH muscle showed a recovery probability that was considerably higher than that of other lower-limb muscles. Quite the opposite pattern was seen in persons with injury caudal to T10. In these subjects, recruitment was more common in proximal muscles of the thigh (psoas and quadriceps), and least common in the AbH muscle.
Discussion: For persons with SCI at or rostral to the T10 vertebral level, the AbH muscle proved to be an earlier and more sensitive indicator of lower-limb contraction recovery following acute SCI compared to other lower-limb muscles. Including this intrinsic muscle of the foot as part of a neurologic assessment of muscle function after SCI should increase the test's sensitivity to preserved (or restored) supraspinal motor influence over lower-limb motoneuron pools, and is recommended.
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Keywords: SCI; human; recovery; EMG; voluntary recruitment; lower-limb; corticospinal tract

Introduction

Multiple systems of the body are disrupted following spinal cord injury (SCI), and monitoring recovery could involve a whole host of measurements, depending upon the system or systems of interest. Recovery of the ability to volitionally contract and relax a given muscle that was initially paralyzed after the injury is a concept that is universally understood as representing a positive development in the recovery process.[1,2] Originally developed for testing strength in patients with polio, the 'Manual Muscle Test' (MMT) utilizes scores of 0 (no palpable contraction) through 5 (normal strength) for describing muscle contractile strength during a maximal voluntary contraction.[3] Testing of contraction ability in specific muscle groups generally falls under the guise of a neurologic evaluation, of which there are a number of variants that are specific for spinal cord injury.

One such neurologic examination advocated by the American Spinal Injury Association (ASIA) involves a combined motor and sensory protocol that can be administered in an acute hospital setting. The origins of

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this test date back a number of decades.[4,5] Slight variants of this test have served as the primary outcome measure of neurologic status in a number of multi-center studies of drug treatment strategies to minimize secondary injury after acute SCI.[6,7] Like its predecessors, the ASIA scale includes testing of a number of muscles in both upper and lower-limbs. In its most recent version, the upper limb muscle groups for testing include elbow flexors, wrist extensors, elbow extensors, long finger flexors, and small finger abductors (an intrinsic muscle of the hand).[8] Lower-limb muscle groups tested with the ASIA protocol include hip flexors, knee extensors, ankle dorsiflexors, long toe extensors (ie extensor hallucis longus), and ankle plantar-flexors. Absent from the ASIA muscle testing scheme, or any other neurologic assessment protocol specific for the SCI population, is inclusion of an intrinsic foot muscle, such as abductor hallucis (AbH).

Evidence from primate studies indicates that spinal motoneurons innervating distal muscles of the lower limb receive a disproportionately strong input from the contralateral motor cortex relative to more proximal lower-limb muscles.[9] In humans, AbH is more easily recruited (ie has a lower threshold) by transcranial magnetic stimulation (TMS) in awake humans than other lower-limb muscles,[10] and the same is true in the anesthetized human when tested with transcranial electrical stimulation during spine surgery.[11] These findings point to a relatively prominent role of the AbH in cortically controlled voluntary movements. This being the case, we hypothesized that in persons with SCI who are initially unable to voluntarily contract any lower-limb muscle but who then regain some contraction ability, the AbH muscle should be among the first of the lower-limb muscles to show this recruitment.

Methods

We studied subjects with acute traumatic SCI. All subjects were examined at least two times following their injury. The initial examination was attempted within the first week of injury, and subsequent evaluations were scheduled on a diminishing frequency over the next 1-year period. To be included for study, all subjects either: (1) had no voluntary contraction in any of their lower-limb muscles (ie they were 'motor-complete') within either leg during the initial examination, but regained contraction in at least one lower-limb muscle at some later time (ie they converted to a 'motor-incomplete' status in one or both legs); or (2) they were motor-incomplete at the initial examination, but were very weak in one or both legs. We expected the cauda equina might be involved for lesions to vertebrae at or caudal to T11 (based upon radiology report), hence we stratified our subject population into those with injury at or rostral to the T10 vertebral level, and those with injury caudal to T10. The study protocol was approved by the University of Miami's Institutional Review Board, and all subjects provided their consent (for adults) or assent (for children) to participate.

During each subject's acute phase, a mobile instrumentation rack containing electrophysiology equipment was wheeled to his or her room for data collection. Upon discharge, follow-up evaluations were conducted in a laboratory setting of the same hospital. We used pairs of self-adhesive surface electrodes to record electromyogram (EMG) from the following lower-limb muscles or muscle groups bilaterally: hip flexors (including psoas major (psoas)), quadriceps (quads), hamstring (hams), tibialis anterior (TA), soleus, and AbH (an intrinsic muscle of the foot that both abducts and plantar-flexes the great toe). EMG records were filtered (100 Hz–5 kHz), amplified (gain = 10 k), and recorded on digital tape for later analysis. Subjects could hear the EMG activity as it was played back through a loudspeaker. When seated in an upright position, subjects could also observe the EMG interference pattern as it scrolled across a computer screen.

At the acute stage, all subjects were lying supine at the time of testing. At later stages, subjects were tested in a seated position, either in a dental chair or within the subject's own wheelchair. Subjects were asked to contract each muscle or muscle group as forcefully as possible when given the command 'contract', and then to relax that contraction only when instructed to do so. All contractions were isometric; resistance to limb movement was provided as needed by one of the investigators, in a manner consistent with a typical manual muscle test.[3] We discouraged subjects from using strategies which might elicit involuntary contractions (eg breath-holding; pressing down on the thighs), as these can trigger spasms. If there was doubt as to whether a contraction might in fact be due to spasticity, subjects were asked to immediately replicate the contraction. Spasm-based EMG recruitment typically could not be repeated until 10 s or more had elapsed, whereas volitional EMG could be initiated and stopped upon command.

Voluntary contractions of individual muscles were graded on a 6-point scale (0–5) based upon the EMG interference pattern produced. A previous study from this laboratory showed a good relationship between this EMG score and scores of manual muscle testing in subjects with both acute and chronic SCI, allowing this EMG measure to be substituted for manual muscle testing.[12] EMG scoring was performed off-line from the tape record by one of the investigators. In an effort to minimize investigator bias, this person did not participate in the subject evaluations. Data are reported from subjects with significant weakness in one or both legs during the initial evaluation, corresponding to a mean EMG score in that limb of 2.0 or lower (ie either no recruitment at all, or recruitment that was inadequate to provide useful function). Once this inclusion criterion was met, the study concentrated on the presence or absence of AbH recruitment. In this regard, even minimal EMG recruitment (ie a single motor unit potential discharging a few action potentials at a slow rate) within this muscle during a contraction attempt was enough to constitute a score of 'present', provided

the subject could either maintain the unit's discharge until asked to relax, or could replicate the contraction within 2–3 s of the past recruitment. These conditions helped ensure that a score of 'present' was not assigned to recruitment caused by involuntary contractions.[13] Finally, traumatic injury to the spinal cord resulting in partial preservation (or recovery) of motor function typically leads to an asymmetry in strength between left and right sides. For this reason, data from left and right legs were treated independently (ie as individual 'cases'), in accordance with a previous study from Waters et al.[13] Thus the number of cases presented within the Results exceeds the number of subjects tested.

It was also due to the high incidence of asymmetry in lower-limb function that we did not utilize ASIA categories to describe our subject population. The main point of this study was to examine the pattern of recovered (or preserved) contraction ability in a subject's leg. Many subjects included in this study had adequate strength in one leg for it to be functional, yet the other leg had muscles with little or no contraction ability. We felt it would add confusion to talk about a subject's leg as being 'motor-complete' (in the event that there was no recruitment) yet the subject might best be described by an ASIA 'C' or even ASIA 'D' descriptor. Further confusion would result from consideration of some of the subjects in this study with injury caudal to T10, in whom motor sparing or recovery was evident, yet they would be classified as ASIA 'A', due to perianal anesthesia.

Results

Results were obtained from 70 subjects (40. 4 ± 16.2 years of age; 57 males and 13 females). The average duration from injury to the initial evaluation for this population was 1.0 ± 0.9 weeks; 31 of these subjects were first examined within 3 days of injury. The three most common causes of injury were motor vehicle accident (43%), falls (24%), and gunshot wound (10%). This subject population forms a subset of a much larger group of subjects with acute SCI, whose EMG-based time course of recovery for multiple upper- and lower-limb muscles[14] and tendon-tap responses[15] are presented elsewhere.

In total, 58 subjects sustained a primary SCI at or rostral to the T10 vertebral level. Based on findings at the initial evaluation, these subjects either had no voluntary contraction in any lower-limb muscle of either leg ($n = 18$ subjects), or had measurable – but limited – voluntary contraction ability in one or both legs (mean EMG score for all muscles of the leg < 2.0; $n = 40$ subjects). By restricting our analysis to those motor-incomplete subjects with a mean EMG score in a lower limb of less than 2.0, we excluded the many subjects in our larger sample[14] who had sustained a mild form of SCI, and had functional, bilateral contraction in most or all of the six lower-limb muscles being studied, even at the most acute stage following injury.

An additional 12 subjects had sustained their injury at a bony level at or caudal to T11. All subjects in this thoraco-lumbar group showed significant weakness in one or both legs (ie mean EMG score for all muscles of the leg < 2.0). Since one cannot rule out a combination of spinal cord, conus medullaris, and cauda equina injury in these subjects, they were treated separately from persons with injury at or rostral to the T10 vertebral level.

SCI at or rostral to T10

The AbH muscle had a much higher probability of recovery after SCI, at an earlier time-point, than other lower-limb muscles in this population. Beginning with subjects who had no voluntary contraction in one or both legs at the initial evaluation, Figure 1 illustrates those cases in which muscle recruitment was noted to recover in *only one* lower-limb muscle at some later evaluation. There were 31 cases that met these conditions. In 24 of these cases, the AbH muscle was the first (and only) muscle to show voluntary recruitment. Of the remaining seven cases (ie the 'exceptions' to the more common finding of AbH being first-recruited), hamstring was first-recruited most often ($n = 5$), with single examples of quadriceps and soleus first-recruitment.

In these seven exceptions, six showed AbH recruitment at a subsequent evaluation. Figure 2 plots the time (in weeks) post-injury when a non-AbH lower-limb muscle was first recruited (filled circles) and the time of the subsequent evaluation when the AbH itself was now seen to be recruited by voluntary contraction (open circles). In six of these cases, AbH recruitment lagged recruitment in a different lower-limb muscle by only a few days or weeks. We could not directly determine whether AbH was recruited in the lower limb of the subject depicted as 'Exception number 1' of Figure 2 (asterisk), because he was transferred from our hospital shortly after his second evaluation (ie that study in which we first observed recruitment in his hamstring muscle, while AbH was silent during contraction attempts). Approximately 1 month after this subject's
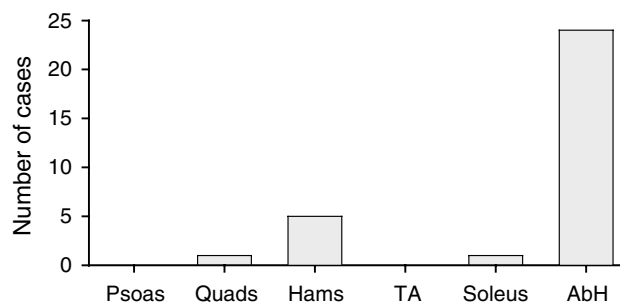

Figure 1 Number of cases showing the first muscle recruited in which subjects were initially motor-complete in one or both legs, and subsequently recovered voluntary contraction in only one lower-limb muscle. From subjects with injury at or rostral to T10. Total number of subjects = 25; total number of cases = 31 (ie cases were based upon findings from both legs of six subjects, and from one leg in 19 subjects)

transfer, we learned from his surgeon that the subject was moving all joints in that limb, including his big toe in a plantar-going manner. Without direct confirmation of this movement, however, we elected to omit an open circle for this subject in Figure 2.

An example of this recovery is illustrated in Figure 3, showing EMG records from the right lower limb in a subject who sustained a C3 fracture, leaving him initially motor-complete in that leg. At the first evaluation showing he was no longer motor-complete (6.5 months following his injury), we noted recruitment of one or more motor units in his AbH muscle during attempts to contract his quadriceps ('2' in Figure 3a), soleus ('5' in Figure 3a; note 2 attempts were made), and AbH

muscles ('6' in Figure 3a; 2 attempts were made). No other lower-limb muscle showed recruitment during voluntary contraction attempts at this time. However, by 10 months post-injury (Figure 3b) the subject was now able to recruit multiple muscles on command in his leg, including quadriceps ('2'; 2 attempts), tibialis anterior ('4'; 2 attempts), soleus ('5'), and AbH ('6'). In fact, the subject had difficulty relaxing the AbH muscle during this evaluation, so that voluntary contraction attempts show recruitment superimposed upon tonic discharge of multiple single motor units in this muscle.

Careful examination of the 'hams' trace of Figure 3b reveals what appears to be a marginal increase of EMG during the attempted hamstring ('3') contraction. This level of activity was scored a '0' at this time. However, at the next evaluation in this subject following the 10-month study shown in Figure 3b, this subject was in fact contracting his hamstring upon command (score of '1') in a reproducible manner (not shown).

Figures 1–3a presented findings from subjects who were initially motor-complete after SCI in one leg (or both legs) and then progressed to a motor-incomplete status with confirmed recovery in only one of the six lower-limb muscles examined for that leg. What about cases in which: (1) the transition from motor-complete to motor-incomplete status was accompanied by EMG recruitment within more than one lower-limb muscle at the time of evaluation; or (2) subjects had recruitment of 1 or more muscles at the *initial* evaluation following SCI (but were still very weak in that limb)? Figure 4 shows which muscles were recruited in these cases. In every such case, AbH was always one of the muscles recruited (100% probability). This figure also shows that, with the exception of the AbH muscle, there did not appear to be any substantial difference in recruitment probability between proximal (eg quadriceps) and distal (eg soleus) lower-limb muscles within these cases.
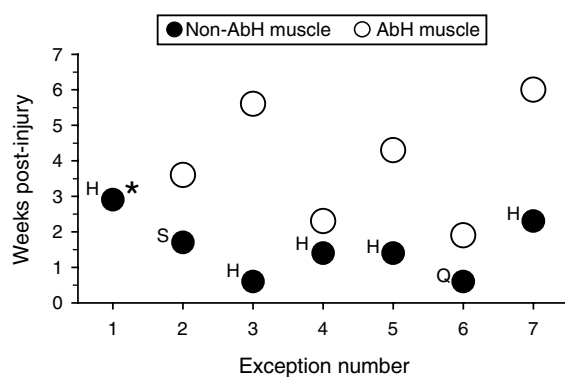

Figure 2 Expansion of findings from Figure 1. Weeks postinjury in which a lower-limb muscle other than AbH was first-recruited (filled circles), and the later time when AbH was then found to contract in that subject (open circles). Exception #2 was soleus ('S' in the figure); exception #6 was quadriceps ('Q'); other exceptions were hamstring ('H'). Asterisk (*) denotes the one case in which direct follow-up for confirmation of AbH recruitment was not possible, due to transfer of this subject to another hospital
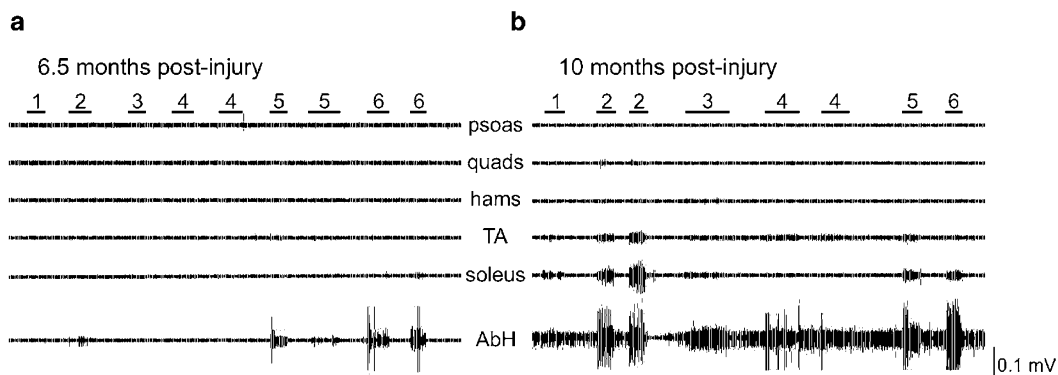

Figure 3 Data from a 50-year-old male with C3 fracture due to blunt trauma. Raw EMG traces depict activity in six right-side lower-limb muscles during attempted contraction of each muscle in isolation. The timing and site of contraction attempt is indicated by horizontal lines just above the upper-most data trace in each of the records. The beginning and end of each line indicates when the subject was asked to 'contract' and 'relax' the muscle, respectively. In 'a', only the AbH muscle is recruited during attempts to contract the quadriceps ('2'), soleus ('5', attempted two times) and abductor hallucis ('6', attempted two times) muscles. Approximately 3.5 months later ('b'), multiple muscles are contracting during voluntary movement attempts. In addition, there is considerable resting discharge in the AbH muscle, upon which voluntary efforts are superimposed. Horizontal calibration bar = 5 s

In our larger population with acute SCI at or rostral to T10,[14] a total of 120 subjects were either motor-incomplete in one or both legs at the time of initial evaluation, or were motor-complete initially and converted to a motor-incomplete status at some later time. In six of these subjects, one of their legs showed some recovery, but the other remained motor-complete throughout our follow-up period. In another seven subjects, EMG could not be recorded from multiple muscles (including AbH) of one leg due to casts or wound dressings. Therefore, there were a total of 227 cases ((2*120)−6−7) in which AbH recruitment probability after SCI at or rostral to T10 (bony level) could be compared to that of other lower-limb muscles. In each and every one of these cases – with the sole exception illustrated by asterisk in Figure 2 – AbH was one of the lower-limb muscles recruited in a given leg by voluntary contraction attempts. Usually AbH was the first muscle, or tied as the first of several muscles, to show recruitment following injury, other than the few remaining exceptions depicted in Figure 2. Even in these few cases, the AbH motoneuron pool was receiving volitional movement command signals within a few more weeks post-injury, such that EMG recruitment was soon evident in this muscle during voluntary contraction attempts.

SCI caudal to T10

A very different picture of muscle recruitment emerged when examining persons with SCI caudal to T10 who were weak (mean EMG score <2.0) in their lower limb(s) at the time of examination. The probability of seeing recruitment in proximal lower-limb muscles in this population was considerably higher compared to that for distal lower-limb muscles, as shown in Figure 5. In fact, the probability for seeing psoas recruitment in this population approached 100% (22 of 24 possible cases within the 12 subjects examined). Conversely, the probability for AbH recruitment was only 29%, the lowest of any of the six muscles examined in this population. We never saw a single case within this cohort of subjects in which AbH was the sole muscle recruited. Similarly, there were no cases among these subjects in which AbH recruitment preceded recovery of recruitment of other lower-limb muscles.

Ventilator-dependence and AbH recruitment

We had a number of subjects in our larger study[14] whose injury to the rostral cervical spine left them fully ventilator-dependent, with no signs of volitional recruitment in any of the 24 upper and lower-limb muscles we typically examined bilaterally. Five of these individuals (three ASIA 'A'; two ASIA 'B', based on initial evaluation) evolved to a motor-incomplete status by virtue of their recovering recruitment of at least one lower-limb muscle, even while they remained ventilator-dependent. In four of these cases, the AbH was the first (or only) muscle to be recovered, while hamstring was
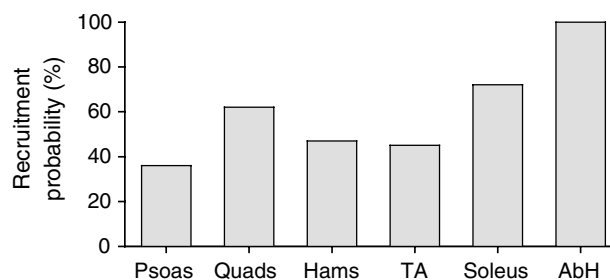

Figure 4 Probability that a muscle could be recruited by voluntary effort, based on findings from the first evaluation in which that subject's leg was found to be motor-incomplete. All data from subjects with SCI at or rostral to T10 who, at the time of initial evaluation, were weak (average EMG score in lower limb muscles < 2.0) or completely paralyzed in the leg. For the latter subjects, recruitment at some later evaluation was noted in two or more lower-limb muscles (ie muscles were 'tied' in their recovery . Total number of cases = 47, from 36 subjects
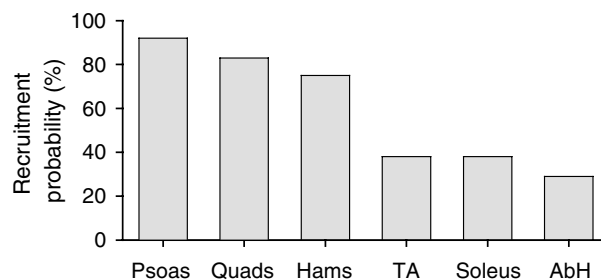

Figure 5 Data from 12 subjects with SCI at or caudal to T11 who presented with significant clinical weakness (mean EMG score in that lower-limb ⩽ 2.0) at the time of initial evaluation. Columns represent each muscle's recruitment probability during contraction attempts. In this case, the AbH had the lowest probability of showing recruitment (29%; seven of 24 possible cases), whereas the probability of psoas recruitment was highest (92%) in this population

first-recovered in one subject. Two of these subjects remained ventilator-dependent until their death. Survival periods for these two subjects following the experiment in which AbH recruitment was first seen were 4 and 18 months, respectively. We carried out multiple follow-up examinations prior to each subject's death, but did not see recruitment in more than the AbH muscle in either case. The other three subjects showed more widespread recovery of muscle function prior to subject death (one case) or extubation (two cases), but none evolved beyond an ASIA 'C' category.

Discussion

The incidence of 'incomplete' traumatic SCI, in which there is retention or recovery of some level of neurologic function in segments caudal to the injury, appears to be increasing compared to values reported from the 1960s and 1970s.[16] In our larger acute study – which included persons with gunshot injury – incomplete SCI accounted

for two-thirds of our subject population,[14] compared to values that did not exceed 45% in any of the three NASCIS trials.[6,17,18] The scope and breadth of research into animal-based SCI treatments continues to expand, leading to predictions that clinical trials of treatment strategies to optimize functional recovery are inevitable.[19–23] Success in such trials will necessitate optimal subject selection and the utilization of outcome measures both sensitive to and specific for the treatment effects.[24] Detailed neurologic testing will almost certainly be a cornerstone of these outcome measures, as it has been for pharmacological clinical trials after acute SCI.[6,17,18,25]

At this time, such neurologic testing does not include the AbH muscle, or any of the intrinsic foot muscles. However, the present study shows that for persons with SCI at or rostral to T10, the AbH muscle: (1) has a higher probability of recovering from traumatic SCI compared to other lower-limb muscles; and (2) recovers from paralysis earlier than other lower-limb muscles. These findings argue that evaluation of the AbH would increase the sensitivity of the neurologic examination in establishing lower-limb motor recovery over that of existing measurement devices (eg the ASIA scale).

The ASIA motor assessment includes three muscles acting on the foot (tibialis anterior and soleus) and great toe (extensor hallucis longus), all of which lie adjacent to the tibia/fibula of the shank. The assessment does not include AbH or any other intrinsic muscle of the foot. This aspect of standard evaluations may account for the conflict between findings of the present study and those of several previous studies.[13,26–29] Specifically, a review of recovery after traumatic SCI stated that 'Proximal extensors recover weeks before distal flexors.' after Brown-Séquard syndrome SCI.[26] Data for this conclusion were based on direct findings from two subjects and a review of the literature.[27] Late conversion to 'incomplete' status following initial, complete quadriplegia was reported in six of 61 subjects; three of these subjects recovered minimal levels of lower-limb motor function, but the specific muscles in which contraction was seen were not specified.[13] In a study of 23 subjects with motor-incomplete and acute SCI, quadriceps recovery was compared to that in 'toe flexors and toe extensors'[28] or just 'toe extensors'.[29] These authors reported that 'toe movement' recovery preceded that of quadriceps contraction in only one case, matched quadriceps recovery in eight cases, and lagged quadriceps recovery in 14 cases. In these studies, the nature of the toe movements (ie the identity of those muscles presumed to be causing movements) was not specified,[28–30] but was most likely that of great toe dorsiflexion (ie via the extensor hallucis longus (EHL)).

We did not routinely monitor EHL for EMG recruitment, even though this muscle's function is tested as part of the ASIA protocol. In our protocol, we asked persons to contract AbH by having them point (ie plantar-flex) their great toe (ie '... like a dancer.'). We then asked subjects to try and spread their toes apart. This latter movement attempt is accompanied by dorsiflexion of the great toe in virtually all subjects with normal or partial lower-limb motor function. Had the EHL been recruited by this movement, our electrodes overlying the TA muscle would have registered this activity, since volume conduction of a contracting muscle's EMG through a neighboring, electrically silent muscle is common.[12,31–33] In fact, we never saw a case in which there was EMG from electrodes overlying the TA muscle in a subject with SCI at or rostral to the T10 level, unless the subject could also voluntarily contract the AbH muscle.

One can understand how the AbH has been overlooked from a clinical standpoint. For gait and ambulation, even limited power in the hip flexors and quadriceps can, when coupled with appropriate prosthetic support, be adequate for walking.[34] Conversely, contraction in the AbH muscle alone, without contributions from more proximal limb musculature, has little functional benefit. Instead, the presence of an AbH contraction signal can confirm that at least one axon (presumably from the corticospinal tract[35]) is conducting action potentials spanning a person's injury site, and that this axon (or these axons) are mediating the command signals to contract that muscle. In those cases of severe and high-cervical SCI (eg requiring total ventilator support), such knowledge may become crucial when considering whether or not to initiate highly-invasive interventions (eg intramedullary cellular transplantation), the decision for which may center on a subject's degree of 'completeness', or lack thereof.

Ours is not the first report demonstrating the role of the AbH as an early indicator of lower-limb recovery following SCI. In a review of diagnosis and prognosis of acute cervical SCI, flexion of the great toe was specifically associated with a good prognosis for functional return.[36] Nevertheless, the findings of the present study are not consistent with more recent publications, which describe the pattern of lower-limb muscle recovery following traumatic SCI. Several possibilities emerge to account for these differences. First, as pointed out above, the standard (ie ASIA-sanctioned) neurologic assessments after SCI do not include foot intrinsic muscles, hence any volitional movement of the great toe may be overlooked (or treated as the initial emergence of flexor-related spasticity) by the examining clinician. Also, our use of EMG recordings lead to a more sensitive measure of voluntary contraction compared to the traditional manual muscle test (ie palpation and visualization of movement). This is because recruitment of literally one single motor unit will be evident on EMG, yet the mechanical force caused by that unit's activity – particularly if its discharge rate is relatively low and limited twitch fusion has occurred – could easily be missed by palpation or visualization. Even so, and as a consequence of the relatively small mass of the great toe, limited contractile forces acting on this joint will have a much higher likelihood of causing visible joint movement compared to forces acting about the ankle, knee, or hip.

Several lines of evidence suggest that the AbH muscle in the foot receives a disproportionately large output from motor cortex in normal man. Of lower-limb muscles, AbH is typically the first-recruited using threshold-level repetitive transcranial electrical stimulation of motor cortex in the anesthetized patient during spine surgery.[11,37] Thresholds for recruiting this muscle with single-pulse transcranial magnetic stimulation in awake, neurologically intact subjects are typically equal to or lower than those for other lower-limb muscles.[10] In persons with incomplete cervical spinal cord injury – most of whom had been injured at least 1 year prior to testing – we showed that AbH was significantly more likely to be recruited by TMS than other lower-limb muscles, including hamstring, tibialis anterior, and soleus.[10] In that study of chronic SCI, every instance in which a subject could recruit at least one lower-limb muscle in the leg included AbH as one of the muscles (or the only muscle) recruited by TMS.[10] Exactly, the same situation was encountered in the present study in persons with acute SCI, using voluntary contraction as the outcome measure.

Investigations using transcranial electric stimulation have concluded that direct, monosynaptic connections between motor cortex and tibialis anterior motoneurons are highly probable.[38] Support for this argument comes from TMS studies of multiple lower-limb muscles, although AbH was not included in these studies.[39,40] In the primate, Jankowska and colleagues compared intracellular responses of distal *versus* proximal hindlimb motoneurons in response to direct electrical stimulation of contralateral motor cortex. They found monosynaptic inputs to both populations of motoneurons, but motoneurons to distal muscles showed greater synaptic efficacy, indicating a greater density of innervation from motor cortex to these distal muscles.[9]

If there are indeed a larger number of corticospinal axons innervating motoneuron pools of intrinsic foot muscles (including the AbH) compared to lower-limb muscles of the thigh, then simple probability would argue that a larger absolute number of these axons may escape permanent damage after motor-incomplete SCI. This argument is based upon the premise that axons within the corticospinal tract are not arranged in a laminar pattern within the lateral tracts, but are instead randomly dispersed throughout the ventrolateral cord. While such an argument is not consistent with the original report of 'central cord syndrome' by Schneider et al,[41] it is supported by other studies in humans.[42–45]

Summary

Persons with a history of acute, traumatic spinal cord injury at or rostral to T10 and who started with, or evolved to, a motor-incomplete status in lower-limb musculature were virtually always able to contract the AbH muscle when tested with EMG recordings, whereas contraction probability was lower, and took longer to become apparent, for more proximal lower limb muscles. Our results indicate that AbH provides an earlier and more accurate indicator of supraspinal influence over motoneuron pools to lower extremity muscles than other lower-limb muscles in the majority of persons who sustain SCI. This being the case, we propose that the sensitivity of standard neurological examinations to evaluate preservation of descending supraspinal influence over lower-limb motoneuron pools would be improved by inclusion of AbH into the group of 'key' muscles being tested. The AbH muscle is easily accessed, even in patients with acute SCI who are bedridden, the movement needed is easily comprehended (ie '… point your big toe down, like a dancer.'), and the small mass of the toe allows for easy visualization of movement, even with minimal motor unit recruitment.

Acknowledgements

References

1 Ditunno Jr JF. Predicting recovery after spinal cord injury: a rehabilitation imperative. *Arch Phys Med Rehabil* 1999; **80:** 361–364.

2 Iseli E, Cavigelli A, Dietz V, Curt A. Prognosis and recovery in ischaemic and traumatic spinal cord injury: clinical and electrophysiological evaluation. *J Neurol Neurosurg Psychiatry* 1999; **67:** 567–571.

3 Kendall FP, McCreary EK. *Muscles. Testing and Function.* Williams & Wilkins, Baltimore, MD 1983.

4 Kirshblum SC, O'Connor KC. Predicting neurologic recovery in traumatic cervical spinal cord injury. *Arch Phys Med Rehabil* 1998; **79:** 1456–1466.

5 Hadley MN. Clinical assessment after acute cervical spinal cord injury. *Neurosurgery* 2002; **50:** S21–S29.

6 Bracken MB *et al.* Administration of methylprednisolone for 24 or 48 hours or tirilazad mesylate for 48 hours in the treatment of acute spinal cord injury. Results of the Third National Acute Spinal Cord Injury Randomized Controlled Trial. National Acute Spinal Cord Injury Study. *JAMA* 1997; **277:** 1597–1604.

7 Geisler FH, Coleman WP, Grieco G, Poonian D. The Sygen multicenter acute spinal cord injury study. *Spine* 2001; **26**(24 Suppl)**:** S87–S98.

8 Marino RJ *et al.* International standards for neurological classification of spinal cord injury. *Spinal Cord Med* 2003; **26**(1)**:** S50–S56.

9 Jankowska E, Padel Y, Tanaka R. Projections of pyramidal tract cells to a-motoneurones innervating hindlimb muscles in the monkey. *J Physiol* 1975; **249:** 637–667.

10 Calancie B, Alexeeva N, Broton JG, Suys S, Hall A, Klose KJ. Distribution and latency of muscle responses to transcranial magnetic stimulation of motor cortex after spinal cord injury in humans. *J Neurotrauma* 1999; **16:** 49–67.

11 Calancie B, Harris W, Brindle GF, Green BA, Landy HJ. Threshold-level repetitive transcranial electrical stimulation for intraoperative monitoring of central motor conduction. *J Neurosurg* 2001; **95:** 161–168.

12 Calancie B, Molano MDR, Broton JG, Bean JA. Relationship between EMG and muscle force after spinal cord injury. *J Spinal Cord Med* 2001; **24:** 19–25.

13 Waters RL, Adkins RH, Yakura JS, Sie I. Motor and sensory recovery following complete tetraplegia. *Arch Phys Med Rehabil* 1993; **74:** 242–247.

14 Calancie B, Molano MR, Broton JG. EMG for assessing the recovery of voluntary movement after acute spinal cord injury in man. *Clin Neurophysiol* 2004, in press.

15 Calancie B, Molano MR, Broton JG. Tendon reflexes for predicting movement recovery after acute spinal cord injury in humans. *Clin Neurophysiol* 2004, in press.

16 National Spinal Cord Injury Statistical Center BA. Spinal Cord Injury: Facts and Figures at a Glance. *J Spinal Cord Med* 2002; **25**(2)**:** 139–140.

17 Bracken MB *et al.* Efficacy of methylprednisolone in acute spinal cord injury. *JAMA* 1984; **251:** 45–52.

18 Bracken MB *et al.* A randomized, controlled trial of methylprednisolone or naloxone in the treatment of acute spinal-cord injury. *N Engl J Med* 1990; **322:** 1405–1411.

19 Geller HM, Fawcett JW. Building a bridge: engineering spinal cord repair. *Exp Neurol* 2002; **174:** 125–136.

20 Hulsebosch CE, Hains BC, Waldrep K, Young W. Bridging the gap: from discovery to clinical trials in spinal cord injury. *J Neurotrauma* 2000; **17:** 1117–1128.

21 Jones LL, Oudega M, Bunge MB, Tuszynski MH. Neurotrophic factors, cellular bridges and gene therapy for spinal cord injury. *J Physiol* 2001; **533:** 83–89.

22 Bunge MB. Bridging areas of injury in the spinal cord. *Neuroscientist* 2001; **7:** 325–339.

23 Burns AS, Ditunno JF. Establishing prognosis and maximizing functional outcomes after spinal cord injury: a review of current and future directions in rehabilitation management. *Spine* 2001; **26**(24 Suppl)**:** S137–S145.

24 Burns AS, Lee BS, Ditunno Jr JF, Tessler A. Patient selection for clinical trials: the reliability of the early spinal cord injury examination. *J Neurotrauma* 2003; **20:** 477–482.

25 Geisler FH, Coleman WP, Grieco G, Poonian D. Measurements and recovery patterns in a multicenter study of acute spinal cord injury. *Spine* 2001; **26**(24 Suppl)**:** S68–S86.

26 Little JW, Ditunno JF, Stiens SA, Harris RM. Incomplete spinal cord injury: neuronal mechanisms of motor recovery and hyperreflexia. *Arch Phys Med Rehabil* 1999; **80:** 587–599.

27 Little JW, Halar E. Temporal course of motor recovery after Brown-Sequard spinal cord injuries. *Paraplegia* 1985; **23:** 39–46.

28 Graziani V, Tessler A, Ditunno JF. Incomplete tetraplegia: sequence of lower extremity muscle recovery. *J Neurotrauma* 1995; **12:** 121.

29 Ditunno Jr JF, Graziani V, Tessler A. Neurological assessment in spinal cord injury. In: Seil FJ (ed). *Neuronal Regeneration, Reorganization, and Repair*. Lippincott-Raven: Philadelphia, PA 1997, pp 325–333.

30 Graziani V, Crozier KC, Selby-Silverstein L. Lower extremity function following spinal cord injury. *Top Spinal Cord Injury Rehabil* 1996; **1:** 46–55.

31 De Luca CJ, Merletti R. Surface myoelectric signal cross-talk among muscles of the leg. *Electroencephalogr Clin Neurophysiol* 1988; **69:** 568–575.

32 Farina D, Merletti R, Indino B, Nazzaro M, Pozzo M. Surface EMG crosstalk between knee extensor muscles: experimental and model results. *Muscle Nerve* 2002; **26:** 681–695.

33 van Vugt JPP, van Dijk JG. A convenient method to reduce crosstalk in surface EMG. *Clin Neurophysiol* 2001; **112:** 583–592.

34 Kirshblum S, Ho CH, House JC, Druin E, Nead C, Drastal S. Rehabilitation of spinal cord injury. In: Kirshblum S, Campagnolo DI, DeLisa JA, (eds). *Spinal Cord Medicine*. Lippincott Williams & Wilkins: Philadelphia, PA 2002, pp 275–298.

35 Nathan PW. Effects on movement of surgical incisions into the human spinal cord. *Brain* 1994; **117:** 337–346.

36 Stauffer ES. Diagnosis and prognosis of acute cervical spinal cord injury. *Clin Orthop* 1975; **112:** 9–15.

37 Calancie B, Harris W, Broton JG, Alexeeva N, Green BA. 'Threshold-level' multipulse transcranial electrical stimulation of motor cortex for intraoperative monitoring of spinal motor tracts: description of method and comparison to somatosensory evoked potential monitoring. *J Neurosurg* 1998; **88:** 457–470.

38 Maertens de Noordhout A *et al.* Corticomotoneuronal synaptic connections in normal man. An electrophysiological study. *Brain* 1999; **122:** 1327–1340.

39 Brouwer B, Ashby P. Corticospinal projections to lower limb motoneurons in man. *Exp Brain Res* 1992; **89:** 649–654.

40 Bawa P, Lemon RN. Recruitment of motor units in response to transcranial magnetic stimulation in man. *J Physiol* 1993; **471:** 445–464.

41 Schneider RC, Thompson JM, Bebin J. The syndrome of acute central cervical spinal cord injury. *J Neurol Neurosurg Psychiatry* 1958; **21:** 216–227.

42 Levi ADO, Tator CH, Bunge RP. Clinical syndromes associated with disproportionate weakness of the upper versus the lower extremities after cervical spinal cord injury. *Neurosurgery* 1996; **38:** 179–185.

43 Bunge RP, Puckett WR, Becerra JL, Marcillo A, Quencer RM. Observations on the pathology of human spinal cord injury. A review and classification of 22 new cases with details from a case of chronic cord compression with extensive focal demyelination.. In: Seil FJ (ed) *Advances in Neurology*. Raven Press, Ltd: New York 1993, pp 75–89.

44 Collignon F, Martin D, Lenelle J, Stevenaert A. Acute traumatic central cord syndrome: magnetic resonance imaging and clinical observations. *J Neurosurg* 2002; **96** (1 Suppl)**:** 29–33.

45 Bolger C, Wigfield C. Image-guided surgery: applications to the cervical and thoracic spine and a review of the first 120 procedures. *J Neurosurg* 2000; **92:** 175–180.