Original Article

Patterns of lower extremity innervation in pediatric spinal cord injury

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Study design: Retrospective review.

Objectives: To identify relationships between lower extremity innervation and level of injury, mechanism of injury, and age at injury in a pediatric population with spinal cord injury (SCI). Secondly, relationships between innervation and completeness of injury, time since injury, race, and sex were evaluated.

Setting: Pediatric orthopedic referral hospital, Philadelphia, Pennsylvania.

Methods: Records of 190 subjects, ages 1–21 years, were reviewed. Data collected from the medical record included lower extremity muscle innervation, American Spinal Injury Association (ASIA) level and class, mechanism of injury, age at injury, time since injury, race, and sex. To determine innervation, lower extremity muscles had been tested using surface electrical stimulation and identified as being innervated or denervated. If a muscle responded weakly, strength duration testing was performed. For analysis via logistic regression, subjects were grouped based upon level and mechanism of injury.

Results: A relationship ($P < 0.0001$) was found between ASIA level and lower extremity innervation of all muscles and between length of time since injury and lower extremity innervation for some muscles. Following multiple logistic regression, only ASIA level remained as an independent predictor of lower extremity innervation status.

Conclusion: Our results show that lower extremity innervation does differ based on the level of the injury. Denervation began to be seen with injuries in the lower thoracic region and more predominantly with injuries in the lumbar region. This supports our hypothesis that the incidence of lower motor neuron injuries would increase as injuries became more caudal. Our hypotheses of a relationship between innervation status and mechanism of injury and age at injury were not supported. This information is important in determining treatment strategies, eligibility for electrical stimulation techniques, and potential regenerative strategies.

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Introduction

A spinal cord injury (SCI) may present as an upper motor neuron (UMN) lesion, a lower motor neuron (LMN) lesion or a combination of the two. The LMN is comprised of the ventral gray matter and its connection to skeletal muscle. A lesion to the LMN disconnects and denervates the peripheral skeletal muscle, resulting in muscle atrophy and a loss in both muscle tone and reflexes. The UMN consists of the descending spinal pathways that can influence the LMN. Damage to the UMN presents as paralysis, hyper-reflexia, hypertonia, and spasticity. In this case, the LMN is still intact and

innervated as long as the injury is above the lumbar spinal cord.¹

Lower extremity functional electrical stimulation (FES) is a treatment option only available to individuals without LMN damage to the lower extremities. With FES, electrodes are placed on the skin surface^{2–7} or placed intramuscularly to the muscle or near its nerve supply.^{5–13} Electrical stimulation to the appropriate muscles at the correct timing can allow upright mobility with an assistive device. These systems can increase functional mobility within the user's environment and enhance well-being.^{2,13} Recent research has been exploring the use of electrical stimulation-induced cycling using surface electrodes to improve the cardiovascular and musculoskeletal systems of persons with SCI^{14–19} or

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to promote neurological recovery.¹⁴ Electrical stimulation can also be used to strengthen muscles in persons with SCI in order to prevent further atrophy or to help prepare the person for future FES system implantation.⁷ Again, these techniques are only available for those without damage to the LMN.

Distinguishing between an UMN and LMN lesion will also be important once research strategies for regeneration of the spinal cord become available in the clinical environment. The future of SCI research is developing each day and determining UMN *versus* LMN lesions may have predictive or prognostic value. Neurological recovery requires the presence of an intact LMN for motor recovery, and the muscles of individuals with chronic LMN injuries will likely have lost contractile properties due to irreversible degenerative changes within the muscle fibers themselves²⁰ when regeneration occurs. This presents researchers with additional challenges for these individuals.

Studies have attempted to predict whether the neurological level will correspond to the type of lesion in the lower extremities,¹ upper extremities,²¹ and the bladder.²² Doherty *et al*¹ found a significant trend that the incidence of LMN lesions affecting the lower extremities decreased as the level of injury progressed in a cephalad manner in adults with SCI, using reflex testing to distinguish those with UMN and LMN lesions. Chuang *et al*²² found mixed patterns of UMN and LMN lesions when examining injuries at the thoracolumbar junction and Triolo *et al*²³ reported that 50% of the individuals with injuries below T12 screened for lower extremity FES presented with at least some denervation in the lower extremities.

Recent studies have suggested a relationship between mechanism of injury and extent of damage and denervation. Waters and Adkins²⁴ found that SCI due to motor vehicular accidents (MVA) were associated with fractures and loss of blood and therefore increased the risk of further damage and secondary problems compared to other etiologies of SCI. Traumatic SCI have also been correlated with irreversible damage because they are more likely to harm the nerve roots and affect LMN as they exit the spinal canal.^{25,26} Viral, vascular, and iatrogenic causes of SCI have also been linked to LMN damage.^{21,23}

Differences may exist in patterns of UMN and LMN lesions between injuries sustained by children and by adults. Injuries to the spinal cord can differ in presentation between adults and children. Children up to 8–10 years of age have been shown to have spinal anatomical differences. The vertebral bodies of a child's spine are anteriorly wedged with horizontally oriented facets, resulting in less protection from flexion and rotation injuries.²⁷ This, in combination with ligamentous laxity and incomplete vertebral ossification in the child's spine,²⁸ stresses susceptible areas when exposed to excessive motion, which is often the case with an SCI. This may result in a spinal cord injury without radiographic abnormality (SCIWORA),²⁷ as fractures are less common in children with SCI as compared to adults

with SCI.²⁸ Therefore, the anatomical differences in children's spines could potentially impact whether an UMN or LMN lesion is incurred, as the potential stresses may differ.

The purpose of this paper is to review the findings of a retrospective analysis of lower extremity innervation in children and adolescents with SCI at Shriners Hospital for Children in Philadelphia between the years 1986 and 2003. Specifically, we examined potential relationships or patterns between lower extremity innervation and level of injury, mechanism of injury, and age at injury. We also looked at completeness of injury, time since injury, race and sex as secondary variables. None of these to date have been examined in a pediatric population. We hypothesized that there would be an increased incidence of LMN lesions as injuries became more caudal. Secondly, we hypothesized that there would be an increased incidence of LMN lesions seen with injuries due to gun shot wounds and medical reasons such as viral or surgical problems. Finally, we hypothesized that children injured at a younger age would have an increased incidence of LMN lesions.

Methods

Subjects

The medical records of 199 subjects (ages 1–20 years) seen in the Research Department between 1986 and 2003 were reviewed. Nine of these records (4.5%) were eliminated after review due to discrepancies in information regarding the level of injury. This resulted in a total of 190 subjects in this retrospective analysis (Table 1). All of these subjects had been seen in the Research Department due to their interest in the lower extremity FES research programs offered by the department. All subjects were beyond the period of spinal shock when evaluated. The governing Institutional Review Board approved this retrospective review.

Data collection

The data collected from the chart included lower extremity muscle innervation status, right and left

Table 1 Subject demographics

Variable	
Age at injury (years)	10.5 ± 6.0 ^a
Time since injury (years)	2.9 ± 3.1 ^a
<i>Race</i>	
Caucasian	149
Black	17
Hispanic	15
Other	9
<i>Sex</i>	
Male	112
Female	78

^aMean and standard deviation

American Spinal Injury Association (ASIA) levels (level of injury), mechanism of injury, age at injury, ASIA class (A, B, C, or D), time since injury, race, and sex. The lower extremity muscles of each subject had been tested by a physical therapist using surface electrodes and a portable clinical electrical stimulator. As the records reviewed spanned a period of 17 years, different stimulators had been used for testing. However, all of these stimulators provided sufficient output for creating strong contractions of large lower extremity muscles. ASIA levels and classes had been assessed by physicians, physical therapists, or occupational therapists trained in the use of the test.

Each muscle was identified as being innervated, denervated, or partially denervated based upon the evaluation. A muscle was categorized as innervated if a strong muscle contraction was seen. The minimum manual muscle test scores used to classify a muscle as being innervated were a grade 4 for the quadriceps femoris, hamstrings, anterior tibialis, and gastrocnemius muscles, and a grade 2 for the adductors and gluteal muscles. A lesser grade was used for the adductor and gluteal muscles due to clinical experience with responses of these muscles in relation to the results of strength duration testing. A muscle was classified as denervated if no response of the muscle could be obtained using surface electrodes. With many subjects, this result was confirmed by strength duration testing. If a muscle responded weakly to surface stimulation, strength duration testing was performed to that muscle to determine if the muscle should be classified as innervated, denervated, or partially denervated. A full description of performing and interpreting strength duration curves is identified in Mulcahey *et al.*²¹

Data analysis

Subjects were grouped according to levels of injury (Table 2). These groupings were based upon the authors' hypotheses about potential sources of denervation due to clinical experience and anatomy. First, subjects with cervical (highest level of C5) or thoracic levels of injury through T9 (group C5T9) were grouped together as it was anticipated that this group would present with innervation in all lower extremity muscles except in some explainable circumstances such as a concomitant lumbar fracture. The second group consisted of those subjects with T10 or T11 level injuries (group T10T11) as it was anticipated that denervation may begin to be an issue in this group due to the potential for damage several levels below the neurological level of injury. The third group consisted of subjects with T12 level injuries (group T12). These subjects were separated from the other subjects as denervation may become more common in this group due to the proximity of the lumbar nerve roots and the conus medullaris. However, this group may have denervation more restricted to the lumbar area. The last group consisted of subjects with injuries at L1 and lower (group L1D). It was anticipated that these subjects would show the most extensive

denervation due to the proximity of the conus medullaris and the cauda equina.

Muscles were then reclassified into two categories for analysis: innervated and denervated. The revised definition of denervated included muscles that were determined to be partially or fully denervated. These two categories were combined as each indicates LMN damage. ASIA classifications were grouped as complete (ASIA A) and incomplete (ASIA B, C, and D). Finally, mechanisms of injury were grouped and categorized based on similar mechanisms (Table 3).

Table 2 Subject groups based on level of injury

<i>Variable</i>	<i>Number of subjects</i>
<i>Left ASIA level</i>	
C5–T9	117
T10–T11	30
T12	18
L1 and lower	25
<i>Right ASIA level</i>	
C5–T9	117
T10–T11	27
T12	22
L1 and lower	24
<i>ASIA classification</i>	
Complete	163
Incomplete	26
Unknown	1

C = cervical, T = thoracic, L = lumbar

Table 3 Subject groups based on mechanism of injury

<i>Category</i>	<i>Included mechanisms</i>
Vehicular (<i>n</i> = 105)	Car, truck, or motorcycle accident Pedestrian <i>versus</i> motor vehicle
Sports (<i>n</i> = 25)	Motorcross Winter sports (skiing, sledding, snowboarding, snowmobile) Gymnastics/trampoline Horseback riding Bicycling All terrain vehicle
Gun shot wound (<i>n</i> = 26)	Gun shot wound
Medical (<i>n</i> = 16)	Surgery Tumor Blood clot Transverse myelitis
Other (<i>n</i> = 18)	Fall Hit by object Person to person trauma Roller coaster Noticed at birth/birth injury

Categorical variables (left and right ASIA level, mechanism of injury, ASIA class, race, and sex) and continuous variables (age at injury and time since injury) were analyzed for influence on the probability of innervation of each muscle studied. Variables were analyzed individually for influence on innervation of each muscle using univariate logistic regression. Variables found significant for each muscle studied were entered into a multiple logistic regression to explore joint influences on innervation of that muscle. Variables that remained as independent predictors of innervation were then compared for each muscle studied with odds ratios and 95% confidence intervals calculated.

In order to explore the overall effects on innervation of the potential predictors, a multivariate analysis of variance (MANOVA) was employed combining innervation of all muscles studied simultaneously. Linear contrasts were used for comparison. Differences between levels (rejection of the null hypothesis) and influences of variables were considered significant if the probability of chance occurrence was ≤ 0.05 using a two-tailed distribution.

Results

The data showed a significant relationship ($P < 0.0001$) between left and right ASIA levels and innervation status when each muscle was examined individually. Table 4 displays the number of innervated and denervated muscles for each ASIA grouping. Significant relationships were also found between time since injury and innervation status of the bilateral hamstring, anterior tibialis, and gastrocnemius muscles ($P \leq 0.02$) with an increase in innervation seen with injuries of greater duration. No relationships were found between innervation status and mechanism of injury, age at injury, completeness of injury, time since injury, race or sex.

Following the multiple logistic regression analysis, only the left and right ASIA levels remained as independent predictors of innervation. The likelihood of innervation of individual muscles did vary across ASIA levels and across muscles (Table 5). One consistent finding was that subjects in the C5T9 group displayed a very high likelihood of innervation in all muscles as compared to subjects in the T10T11, T12, and L1D groups. In addition, subjects in the T10T11

group had a greater likelihood of innervation in some muscles as compared to the L1D group bilaterally and to the T12 group on the right. In this analysis, the odds ratios for the bilateral hamstrings and left anterior tibialis were not estimable for some comparisons involving the T12 group. This was due to the lack of occurrence of innervation in the T12 group for those particular muscles.

When innervation status of all muscles was combined, a significant difference in innervation status was found between ASIA level groupings ($P < 0.05$), except between C5T9 *versus* T10T11 on the left and T12 *versus* L1D bilaterally (Table 6). Data were limited in this analysis as only subjects with data for every muscle could be included, resulting in 86 observations on the left and 85 on the right. Data from the other subjects were excluded to avoid skewing the results based on incomplete data.

Discussion

Overall, our results show that the incidence of LMN lesions does differ based on the ASIA level with denervation being more commonly seen with more caudal levels. This supports our hypothesis that the incidence of LMN lesions would increase as injuries became more caudal. Our second and third hypotheses were not supported, as innervation was not related to the mechanism of injury or the age at injury in our sample. However, we did find a relationship between time since injury and innervation of the hamstring, gastrocnemius, and anterior tibialis muscles with increased incidence of UMN lesions seen with injuries of longer duration.

Our results are similar to those found by Doherty *et al*¹ in a study with adults with SCI. Doherty *et al*¹ performed a retrospective review of the medical records of 156 adults with complete T7 to L3 SCI to determine the incidence of LMN *versus* UMN lesions for ASIA level groupings of T7–T9, T10–T12, and L1–L3. The type of lesion was determined by the status of reflexes. The absence of the bulbocavernosus reflex, deep tendon reflexes (Achilles and patellar), and the Babinski sign defined a LMN lesion, whereas their presence defined an UMN lesion. Subjects were tested at least three months postinjury to avoid misinterpreting spinal shock for an LMN lesion. The authors found a significant trend with

Table 4 Actual numbers of innervated (I) and denervated muscles (D) per ASIA grouping

ASIA Group	Quadriceps		Adductors		Gluteus Medius		Gluteus Maximus		Hamstrings		Anterior Tibialis		Gastrocnemius	
	I	D	I	D	I	D	I	D	I	D	I	D	I	D
C5T9	210	29	194	17	198	27	199	26	94	18	92	18	91	15
T10T11	24	21	12	17	27	27	26	28	11	15	10	15	11	16
T12	5	34	6	22	11	26	12	25	0	17	1	17	6	12
L1D	17	32	11	31	6	41	6	42	6	34	6	39	6	37

These numbers represent the combined results for the left and right sides

Table 5 Odds ratios and confidence intervals (in parentheses) for each muscle when comparing between each ASIA level groupings

ASIA Group	Quadriceps	Adductors	Gluteus Medius	Gluteus Maximus	Hamstrings	Anterior Tibialis	Gastrocnemius
<i>Left lower extremity</i>							
C5T9 versus T10T11	10.8 (4.2, 27.7)*	11.6 (3.7, 36.4)*	8.7 (3.4, 22.3)*	10.9 (4.2, 28.4)*	6.95 (1.7, 28)*	5.4 (1.4, 21.2)*	8.2 (1.9, 34.9)*
C5T9 versus T12	36.1 (9.1, 143.4)*	42.3 (9.4, 190.3)*	16.6 (5.0, 55.4)*	13.7 (4.2, 44.3)*	NE	NE	17.1 (2.3, 127.7)*
C5T9 versus L1D	15.5 (5.6, 43.4)*	38.5 (11.2, 132.5)*	52.8 (13.7, 203.5)*	56.2 (14.5, 218.1)*	32.23 (7.1, 147.1)*	31.1 (7.3, 132.7)*	101.6 (13.7, 755.8)*
T10T11 versus T12	3.3 (0.8, 14.3)	3.7 (0.7, 17.9)	1.9 (0.5, 6.9)	1.3 (0.4, 4.4)	NE	NE	2.1 (0.2, 18.6)
T10T11 versus L1D	0.7 (0.2, 2.1)	0.3 (0.1, 1.1)	0.2 (0, 0.7)*	0.2 (0, 0.8)*	0.22 (0, 1.3)	0.2 (0, 1.0)	0.1 (0, 0.7)*
T12 versus L1D	-2.3 (0.5, 10.6)	-1.1 (0.2, 5.8)	0.3 (0.1, 1.6)	0.2 (0, 1.2)	NE	NE	0.2 (0, 2.1)
<i>Right lower extremity</i>							
C5T9 versus T10T11	7.6 (3.0, 19.6)*	8.5 (2.6, 27.3)*	7.1 (2.7, 18.8)*	7.1 (2.7, 18.9)*	5.0 (1.3, 19.8)*	6.5 (1.5, 27.7)*	6.5 (1.5, 27.1)*
C5T9 versus T12	61.2 (12.9, 290.4)*	48.6 (11.2, 210.3)*	30.7 (8.7, 107.9)*	30.7 (8.7, 108.9)*	NE	52.3 (5.6, 488.1)*	23.7 (3.7, 151.7)*
C5T9 versus L1D	10.6 (3.9, 28.6)*	26.8 (8.0, 89.1)*	53.1 (13.7, 206.0)*	55.4 (14.3, 214.2)*	32.8 (7.3, 147.9)*	39.9 (9.0, 176.4)*	35.1 (8.0, 154.0)*
T10T11 versus T12	8.0 (1.5, 41.9)*	5.7 (1.2, 27.6)*	4.7 (1.1, 16.7)*	4.3 (1.1, 16.8)*	NE	8.0 (0.7, 89.1)	3.7 (0.5, 27.3)
T10T11 versus L1D	0.7 (0.2, 2.2)	0.3 (0.1, 1.2)	0.1 (0, 0.6)*	0.1 (0, 0.5)*	0.2 (0, 0.9)*	0.2 (0, 1.0)	0.2 (0, 1.0)
T12 versus L1D	-5.8 (1.1, 31.0)*	-1.8 (0.4, 9.0)	0.6 (0.1, 3.0)	0.6 (0.1, 2.9)	NE	-1.3 (0.1, 14.8)	0.7 (0.1, 5.0)

The odds ratio represents the likelihood of innervation of one group as compared to another. For example, in row 1, an odds ratio of 10.8 means that the C5T9 group has a 10.8 times greater chance of innervation of the left quadriceps as compared to the T10T11 group. An asterisk (*) indicates significance with $P < 0.05$. A negative odds ratio indicates the direction of the comparison only. For example, in row 6, an odds ratio of -2.3 indicates that the T12 group has a 2.3 times less chance of innervation as compared to the L1D group. NE (not estimatable) indicates that the odds ratio could not be estimated due to no occurrence of innervation in one of the groups being compared. In this study, all comparisons labeled as NE resulted from no occurrence of innervation in those muscles for subjects in the T12 category.

Table 6 Results of the MANOVA that combined innervation of all muscles simultaneously to examine overall effect of the predictors

	MANOVA	
	Left	Right
C5T9 versus T10T11	0.1632	0.003
C5T9 versus T12	<0.0001	<0.0001
C5T9 versus L1D	<0.0001	<0.0001
T10T11 versus T12	0.0290	0.0044
T10T11 versus L1D	0.0223	0.0181
T12 versus L1D	0.2355	0.1418

The data are limited to 85 subjects with complete data. A significant difference in innervation status was found between ASIA level categories ($P < 0.05$), except between C5T9 versus T10T11 on the left and T12 versus L1D bilaterally. There were differences between the right and left sides as denervation was not always symmetrical within a subject.

the incidence of LMN increasing as the level of injury progressed in a caudal manner. Subjects with T7–T9 injury levels had predominantly UMN lesions, and LMN lesions presided in the L1–L3 group. In the T10–T12 group, there was a mix of LMN and UMN lesions, making this division of injury seemingly less predictable. In our study, we found differences in innervation status between individuals with T10 or T11 injuries and those with T12 injuries, which might assist with predicting UMN and LMN lesions. Doherty *et al*¹ also found that no subject in the L1–L3 group had a pure UMN lesion, but 5% displayed a combination of both presentations. In our study, one subject with a lumbar ASIA level had a pure UMN lesion and nine (36%) had a mixed presentation.

Chuang *et al*²² examined UMN and LMN lesions in the area of the thoracolumbar (TL) junction, including T11–L2 vertebral levels, in 29 males and six females (mean age 38.5 years) with traumatic SCI of 6 months to 11 years duration. Each subject's injury was classified into one of three groups: below conus lesion (pure cauda equina), combined (cauda equina + conus medullaris), and above conus medullaris lesion. The TL junction is complicated due to the spinal cord's cessation and the exposed nerve roots, where damage could extend further than expected. Magnetic resonance imaging (MRI), electromyography, and urodynamics were used to determine the subjects' status. The results showed that specific neurological problems were less predictable with injuries to the TL junction, except with injury to the cauda equina. In our study, changes began to be seen even in the thoracic area cephalad to the TL junction. We did not attempt to identify if the cauda equina or the conus were affected using MRI. However, our results show that changes in innervation were occurring around the TL junction as differences were seen in overall innervation status between the T10T11 group and both the T12 and L1D groups. However, our results of

greater odds of innervation for the L1D group as compared to the T12 group for several muscles were unanticipated. It is possible that our subjects in the T12 group sustained greater damage at the TL junction, potentially leading to this finding.

In our study, we did find a relationship between time since injury and innervation of the hamstrings, gastrocnemius, and anterior tibialis muscles with an increase in innervation seen with injuries of greater duration. This finding was unexpected as all of our subjects were considered to be past the stage of spinal shock. In our study, time since injury ranged from 7 weeks to 17 years (mean 2.9 ± 3.1 years). Hiersemenzel *et al*²⁹ defined three phases of spinal shock: loss of reflexes, the transition phase (3–8 weeks), and the spastic phase where an increased response is seen for reflexes, spasms, and muscle tone. They reported that spinal shock lasted 2–4 weeks (mean 20 days) in a sample of 18 adult subjects with acute SCI. Therefore, we would not expect our results to be due to spinal shock. In further looking at our data, several confounding variables are possible. It was discovered that subjects who were tested sooner after injury tended to have more caudal injuries. In addition, these subjects appeared to be more likely to have data for the hamstrings, gastrocnemius, and anterior tibialis muscles. These muscles were not routinely tested in these subjects, as most clinical FES applications available in our hospital do not involve these muscles. Therefore, testing of these muscles was inconsistent throughout the time period reviewed.

We found no differences in innervation based upon mechanism of injury or age at injury. The literature presents mixed findings in regard to mechanism of injury and innervation. Doherty *et al*¹ found no relationship between mechanism of injury and innervation, while Mulcahey *et al*²¹ anecdotally reported increased LMN damage in the upper extremities of children whose cervical SCI had viral, vascular, or iatrogenic causes, and Triolo *et al*²³ reported a relationship between SCI due to transverse myelitis and denervation. Despite anatomical differences reported in the spines of children less than 10 years of age, we found no relationship between age at injury and innervation status, indicating that the anatomical differences in children likely do not impact innervation status.

There are several limitations to our study. While we did review the medical records of 190 subjects, data were not always complete. Data were especially lacking for the hamstring, gastrocnemius, and anterior tibialis muscles, where approximately half of the data were not collected during the evaluations. This limitation may have led to the finding of very high likelihood of innervation of two of these muscles when comparing subjects in the L1D group to those in the T12 group. The results of the combined analysis using a MANOVA may also have been affected, as this test requires complete data sets, which were present in only 86 of our 190 subjects. This greatly reduced the number of subjects in each ASIA level grouping and therefore these results should be interpreted cautiously. Another

possible limitation is that multiple testers performed the testing using different clinical stimulators. However, earlier testers did provide education to later testers as all testers were part of a research department that routinely assessed responses using electrical stimulation. In addition, clinical stimulators were chosen that provided the ability to create strong muscle contractions of large muscles. Finally, this was a retrospective review that has inherent limitations in study design.

Conclusion

The results of this study are important in understanding innervation status, especially in relation to level of injury. There are many future implications of innervation status, including spinal cord regenerative strategies as well as applications of FES, which show potential to impact health and possibly neurological recovery. Further study should evaluate our finding of a relationship between innervation status of some muscles and time since injury to determine if this finding is valid or if it is due to our specific sample of subjects.

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