ORIGINAL ARTICLE

Mechanical and neural changes in plantar-flexor muscles after spinal cord injury in humans

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Study design: Cross-sectional study.

Objectives: To determine the effect of injury duration on plantar-flexor elastic properties in individuals with chronic spinal cord injury (SCI) and spasticity.

Setting: National Rehabilitation Center for Persons with Disabilities, Japan.

Methods: A total of 16 chronic SCI patients (age, 33 ± 9.3 years; injury localization, C6–T12; injury duration, 11–371 months) participated. Spasticity of the ankle plantar-flexors was assessed using the Modified Ashworth Scale (MAS). The calf circumference and muscle thickness of the medial gastrocnemius (MG), lateral gastrocnemius and soleus were assessed using tape measure and ultrasonography. In addition, the ankle was rotated from 10° plantar-flexion to 20° dorsiflexion at 5 deg s^{-1} with a dynamometer, and the ankle angle and torque were recorded. After normalizing the data (the initial points of angle and torque were set to zero), we calculated the peak torque and energy. Furthermore, angle–torque data (before and after normalization) were fitted with a second- and fourth-order polynomial, and exponential (Sten–Knudsen) models, and stiffness indices (SI_{SOP} , SI_{FOP} , SI_{SK}) and $Angle_{SLACK}$ (the angle at which plantar-flexor passive torque equals zero) were calculated. The stretch reflex gain and offset were determined from 0 – 10° dorsiflexion at 50, 90, 120 and 150 deg s^{-1} . After logarithmic transformation, Pearson's correlation coefficients were calculated.

Results: MAS, calf circumference, MG thickness, peak torque and SI_{FOP} significantly decreased with injury duration ($r \text{ log-log} = -0.63, -0.69, -0.63, -0.53$ and -0.55 , respectively, $P < 0.05$). The peak torque and SI_{FOP} maintained significant relationships even after excluding impacts from muscle morphology.

Conclusion: Plantar-flexor elasticity in chronic SCI patients decreased with increased injury duration.

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INTRODUCTION

Motor paralysis following spinal cord injury (SCI) inevitably prevents daily activities such as upright standing and bipedal walking; consequently, physical activity declines in affected patients. Previous studies have indicated that SCI patients experience extreme muscle atrophy,^{1–3} fiber transformation towards a fast-fatigable type^{4–6} and decreased bone mineral density.^{7,8} This musculoskeletal degeneration is attributed largely to the dramatically decreased muscle activity and mechanical stress in the paralyzed limbs, caused primarily by post-SCI motor paralysis.

Quantitative evaluation of the muscle viscoelasticity and tone is important in preventing secondary disorders after SCI. The Modified Ashworth Scale (MAS) is generally used to measure muscle tone and spasticity,⁹ but this score lacks adequate sensitivity to distinguish the contributions from the mechanical and neural components.¹⁰ In a study of the neural profile after SCI, we reported that spinal reflex excitability in participants experiencing both complete and incomplete SCI are significantly higher than the excitability in healthy young participants, but the H_{max}/M_{max} did not change significantly in incomplete SCI participants.¹¹ In addition, the change in $H_{max}/$

M_{max} was not associated with the chronicity of injury in SCI participants. Muscle viscoelasticity is a significant contributor to muscle tone, and it is therefore necessary to understand not only the neural but also the mechanical properties of the paralyzed region in SCI patients. To date, the relationship between the mechanical properties and the duration of injury has not been reported. This information could guide clinicians in determining the appropriate intervention for the patient's pathologic condition.

The objective of the present study is to determine the effect of injury duration on plantar-flexor elastic properties in individuals with chronic SCI and spasticity.

PATIENTS AND METHODS

Participants

Sixteen participants diagnosed with SCI (age, 33 ± 9 years; height, 172 ± 7 cm; weight, 62 ± 10 kg; injury localization, C6–T12; injury duration, 11–371 months) and spasticity (10 complete and 6 incomplete), five participants with complete SCI without spasticity (age, 30 ± 7 years; height, 174 ± 4 cm; weight, 67 ± 14 kg; injury localization, T9–L3; injury duration, 12–213 months) and 13 healthy control participants (age, 27 ± 5 years; height, 169 ± 10 cm; weight, 62 ± 10 kg; 10 male and 3 female) participated in this study. The population demographics

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Table 1 Population demographics

Subject	Sex	Age (years)	Height (cm)	Weight (kg)	Lesion level	Injury type	With spasticity	MAS	Duration of injury (months)	Medication
1	M	20	181	57	C6	Incomplete	Yes	2	11	Gabalon
2	M	30	170	71	T7	Incomplete	Yes	2	14	—
3	M	22	178	64	C7	Incomplete	Yes	2	18	ITB
4	M	45	171	64	T1	Incomplete	Yes	2	19	—
5	M	49	173	70	T3, 4, 5	Incomplete	Yes	1+	52	Dantrium, lendormin
6	M	25	170	54	T5	Incomplete	Yes	1+	63	—
7	M	23	175	61	T10	Complete	Yes	1+	18	—
8	M	27	178	55	T11, 12	Complete	Yes	2	30	—
9	F	27	172	50	T8, 9, 10	Complete	Yes	0	36	—
10	M	27	171	62	T10, 11	Complete	Yes	1	114	—
11	M	37	180	75	T12	Complete	Yes	1	115	Ternelin
12	M	35	173	62	T9, 10	Complete	Yes	1	144	—
13	M	33	174	77	T12	Complete	Yes	1+	144	—
14	M	39	174	77	T8	Complete	Yes	1+	155	—
15	F	40	163	55	T9	Complete	Yes	1+	300	—
16	F	48	153	40	T5	Complete	Yes	1	371	—
17	M	27	173	55	L3	Complete	No	—	12	—
18	M	30	175	58	Th12,L1,L2	Complete	No	—	15	—
19	M	21	178	60	Th9	Complete	No	—	23	—
20	M	33	175	87	Th11,12	Complete	No	—	198	—
21	M	41	168	75	Th10	Complete	No	—	213	—

Abbreviations: MAS, Modified Ashworth Scale; ITB, intrathecal baclofen therapy.
Healthy control subjects ($n=13$): age, 27 ± 6 ; height, 169 ± 10 ; weight, 62 ± 10 .

Table 2 A: partial correlation coefficient between mechanical properties and injury duration (months), excluding muscle architecture and injury level. **B:** associated *P*-values

	Calf circumference (mm)	MG (mm)	LG (mm)	Sol (mm)	Level of injury
A					
Peak torque (Nm)	−0.70	−0.63	−0.80	−0.72	−0.79
Energy (Nm per deg)	−0.70	−0.33	−0.72	−0.58	−0.67
Sl _{SOP} (Nm per deg)	−0.69	−0.57	−0.78	−0.69	−0.77
Sl _{FOP} (Nm per deg)	−0.70	−0.63	−0.80	−0.72	−0.79
Sl _{SK} (Nm per deg)	−0.45	−0.25	−0.62	−0.57	−0.65
Angles _{SLACK} (Nm per deg)	0.58	0.42	0.71	0.63	0.72
B					
Peak torque (Nm)	0.017	0.039	0.003	0.019	0.004
Energy (Nm per deg)	0.017	0.319	0.012	0.077	0.024
Sl _{SOP} (Nm per deg)	0.019	0.070	0.005	0.027	0.006
Sl _{FOP} (Nm per deg)	0.016	0.038	0.003	0.018	0.004
Sl _{SK} (Nm per deg)	0.163	0.461	0.042	0.087	0.032
Angles _{SLACK} (Nm per deg)	0.061	0.201	0.014	0.052	0.013

Abbreviations: LG, lateral gastrocnemius; MG, medial gastrocnemius; Sol, soleus.

are summarized in Table 1. There was no statistical age difference among the three groups (SCI participants with spasticity, SCI participants without spasticity and healthy participants; F -value = 2.075, $P=0.1422$). Each participant provided written informed consent for the experimental procedure, which was approved by the National Rehabilitation Center for Persons with Disabilities (NRCD).

Measurements

The participants were seated with their hip and knee joints angled at 90°. The ankle joint was fixed onto the foot plate of a custom dynamometer.¹² The calf circumference and muscle thickness of the plantar-flexors were measured at 30% of the calf length using a tape measure and ultrasonography (Prosound 2, Aloka, Tokyo, Japan) with a linear array probe (7.5 MHz). The calf circumference, medial gastrocnemius (Gas (MG)) and lateral Gas thicknesses of participants 5, 6 and 8, and four healthy control participants, as well as the

soleus (Sol) thickness of participants 1, 5, 6 and 8, and five healthy control participants, were not obtained. We have stated that this lack of data might lead to type I error. The right ankle joint was rotated from 10° of plantar-flexion to 20° dorsiflexion at 5 deg s^{−1}. During the measurement, ankle joint angle and plantar-flexion torque were recorded at a 1 kHz sampling frequency using a 16-bit analog-to-digital converter (Powerlab, AD Instruments, Bella Vista, NSW, Australia). The muscle activity of the Gas, Sol and tibialis anterior were also measured using surface electromyograms with a bipolar electrode (DE-2.3, Delsys, Boston, MA, USA) spaced 1 cm apart. The electromyograms signal was amplified (The Bagnoli-8 EMG System, Delsys) with band-pass filtering between 20 Hz and 450 Hz. Some participants (#1, 2, 3, 4 and 5 in Table 2) had clonus. In those participants, therefore, we took care not to miss the emergence of symptoms by visually monitoring the joint motion and the surface electromyography displayed on an oscilloscope, and if clonus occurred, the experiment was temporarily stopped. The trial was repeated 6–10 times,

Figure 1 (a) Representative angle and electromyograms (EMG; Gas, Sol, TA) waveform during passive motion test. Broken lines indicate ± 3 s.d. of baseline. (b) Angle and EMG waveform of an excluded participant (participant 3 in Table 1).

and five trials without muscle activity of the lower limb were selected and analyzed further. One participant (Table 1, participant 3) showed remarkable muscle activity exceeding ± 3 s.d. of baseline (Figure 1b), therefore the data from this participant was excluded.

The passive motion test was then followed by a stretch reflex test in the SCI participants with spasticity and healthy control participants only. Stretch reflexes were elicited by quick 10° toe-up rotations of the footplate from 0° with participants at rest. Four different angular velocities ($60, 90, 120$ and 150 deg s^{-1}) were applied five times in each participant in a random order. The inter-stimulus intervals were not constant but were randomized within 4–9 s to avoid anticipation of forthcoming perturbations. After all trials, the MAS of the right plantar-flexor was assessed.

Analysis

Angle and torque data were averaged in five trials and digitally low-pass filtered using a fourth-order zero-lag Butterworth filter¹³ with a 10-Hz cut-off frequency.^{14–16} Three different mathematical models were fitted to the angle (θ)–torque (τ) data as previously described:^{16–27}

- (i) Second-order polynomial (SOP) model (Equation 1)

$$\tau(\theta) = a\theta^2, \quad (1)$$

where a is experimental constant;

- (ii) Fourth-order polynomial (FOP) model (Equation 2)

$$\tau(\theta) = m\theta^4 + n\theta^3 + o\theta^2 + p\theta + q, \quad (2)$$

where m, n, o, p and q are experimental constants;

- (iii) Exponential model similar to the Sten–Knudsen (SK) model (Equation (3))

$$\tau(\theta) = \frac{1}{\alpha} e^{\alpha(\theta - \theta_{\text{slack}})}, \quad (3)$$

where α and θ_{slack} are experimental constants.

Prior to fitting Equation 1, the angle (θ) and torque (τ) data reference points were set to zero. The plantar-flexor, peak torque (torque waveform peak), and

energy (the area between the angle–torque curve and the line torque equaling zero) were calculated from the normalized angle–torque data. In Equations 2 and 3, the raw angle–torque data were used. Equation 3 fitting parameters were determined by the non-linear least square method (Levenberg-Marquardt algorithm²⁸) using the optimization toolbox from MATLAB (The Mathworks, Natick, MA, USA). A total of 1200^{16} data points were used in each fitting. The stiffness indices (SI_{SOP} , SI_{FOP} , SI_{SK}) and $Angle_{\text{SLACK}}$ were determined from these equations as follows:

$$SI_{\text{SOP}} = 2a \quad (4)$$

$$SI_{\text{FOP}} = \text{mean}_{-10 \leq \theta \leq 20} (4m\theta^3 + 3n\theta^2 + 2o\theta + p) \quad (5)$$

$$SI_{\text{SK}} = \alpha \quad (6)$$

$$Angle_{\text{SLACK}} = \theta_{\text{slack}} \quad (7)$$

SI_{FOP} is the average of the derivative of the fourth-order polynomial regression of angle–torque curve across all angle ranges (10° plantar-flexion to 20° dorsiflexion). The determination coefficient (R^2) of the angle–torque curve fitting using SOP, FOP and SK models was also calculated. The reflex responses were analyzed as the peak-to-peak amplitude for 35 ms after perturbation. The SR gain and offset were calculated as the slope and interception of the regression line in the plotted stretch response to angular velocity.

Statistics

One-way analysis of variance was used to compare the determination coefficients (R^2) between the three models, and *post hoc* analysis was performed using Tukey's HSD test. All variables were logarithmically transformed²⁹ except the Gas and Sol offsets, which have negative magnitudes. Then, the Spearman's rank-correlation coefficient for the MAS and the Pearson's product-moment correlation coefficient for the other variables were calculated to determine the relationship between the injury duration and mechanical parameters. In addition, the relationships between all variables and injury localization were

determined using the same procedure. Before logarithmic transformation, MAS values were corrected by designating the minimum data value as one. The partial correlation coefficient, excluding morphological parameters, was also calculated for the mechanical parameters. The statistical significances of the correlation coefficients were assessed for SCI participants with spasticity. Statistical significance was set at $P < 0.05$.

RESULTS

Representative angle–torque data (raw and normalized) and fitting data using the three models are illustrated in Figure 2. In both

Figure 2 Representative angle–torque data fitting using three mathematical models (SOP, FOP and SK). (a) The fitting was good in all models, which were fitted to angle–torque data from participant 12 in Table 1. (b) The SOP and SK model fittings were inferior compared with the FOP model, based on angle–torque data from participant 13 in Table 1.

examples (Figure 2a: participant 12 in Table 1; Figure 2b: participant 13 in Table 1), the FOP model fits the angle–torque data well. However, data fitting using the SOP and SK models is poor in some participants (Figure 2b: participant 13 in Table 1). Similarly, the collective determination coefficient data fitting using the FOP model was very good ($R^2 = 0.999 \pm 0.001$) and significantly higher than in both the SOP ($R^2 = 0.869 \pm 0.135$) and SK (0.904 ± 0.130) models in all the participants ($n = 33$). This trend also occurred and was significant in SCI participants with spasticity (FOP model: $R^2 = 0.999 \pm 0.001$; SOP model: $R^2 = 0.850 \pm 0.120$; SK model: $R^2 = 0.883 \pm 0.114$, $n = 15$). MAS negatively correlated with the injury duration in the SCI participants with spasticity (Figure 3). In addition, the calf circumference and MG thickness were negatively correlated to the injury duration ($n = 13$ excluding the missing data described previously; Figure 3). However, there were no statistically significant relationships between the neural parameters (Gas gain and offset, Sol gain and offset) and injury duration in the group.

Angle–torque representative curves of all participants (excluding participant 3) are shown in Figure 4a. After the polynomial and exponential regression, the SI_{FOP} , SI_{SK} and $Angle_{SLACK}$ were calculated, and Figure 4b was generated by designating the angle and torque reference points in Figure 4a as zero. Using the data in Figure 4a, we calculated the peak torque, energy and SI_{SOP} , and found that the peak torque and SI_{FOP} (stiffness index calculated from the fourth-order polynomial regression) were inversely correlated with the injury duration ($n = 15$).

Muscle morphology theoretically affects passive tension in the muscles and tendons. The effects of these morphologic variables (calf circumference and MG, lateral Gas, and Sol thicknesses) were excluded by calculating the partial correlation coefficients between the mechanical properties (peak torque, energy, SI_{SOP} , SI_{FOP} , SI_{SK} and $Angle_{SLACK}$) and the injury duration (Table 2). The peak torque and SI_{FOP} were negatively correlated, even after excluding the effects of the calf circumference and MG thickness, which significantly decreased with the injury duration. In addition, we confirmed that the injury severity was not correlated with these results (Figure 5).

DISCUSSION

We investigated the effect of injury duration on plantar-flexor elasticity in individuals with chronic SCI and found negative correlations between the clinical index of spasticity (MAS), morphologic parameters (calf circumference and MG thickness), mechanical parameters (peak torque and stiffness index) and injury duration in SCI participants with spasticity. In addition, there were significant partial correlation coefficients between the mechanical parameters and the injury duration excluding morphologic parameters.

Alteration of the ankle joint stiffness after SCI

As clearly shown in Figure 3, the decline in calf circumference and MG thickness with injury duration indicates that muscle atrophy continues in SCI even during the chronic stage. Castro *et al.*² measured the cross-sectional area of the lower musculature (Gas, Sol and tibialis anterior) during the 6 months following spinal injury and showed that the CSA of Gas decreased significantly. The present result showing a selectively decreased MG thickness is consistent with previous findings.² Notably, involuntary muscle activity resulting from clonus and spasm with spasticity might contribute to attenuate atrophy,³⁰ therefore the decreasing spasticity observed in this study, coupled with the immobilization, likely accelerated the muscle atrophy.

The Ashworth scale³¹ remains a major clinical scale for evaluating spasticity,^{32–34} but its validity and reliability are questioned by some

Figure 3 Relationship between mechanical variables and injury duration (months). Complete SCI participants with spasticity (filled circles), incomplete SCI participants with spasticity (open circles), complete SCI participants without spasticity (x), and healthy participants (horizontal bar) are shown. The horizontal dashed line indicates the mean value in healthy participants.

reports.^{35,36} A positive correlation has been reported between the Ashworth scale and indices of clonus and spasm,³⁷ therefore the negative correlation between MAS and the injury duration observed in this study suggest that the frequency of involuntary muscle contraction because of spasm gradually decreases over time after SCI. In evaluating the extent of spasticity over time, we found no statistically significant correlation between the stretch reflex gain and offset in the lower

muscles (MG, Sol) and injury duration. This result is consistent with our previous study¹¹ that found that the stretch reflex peak-to-peak amplitude and stretch reflex peak-to-peak amplitude/Mmax in complete and incomplete SCI participants were not correlated to the time post-injury, suggesting that spinal circuit excitability in SCI patients does not change during the chronic stage. However, several reports showed that Mmax was decreased more in the chronic SCI

Figure 4 (a) Angle–torque curves of plantar-flexor muscles for all complete SCI participants with spasticity (chain), incomplete SCI participants with spasticity (solid), complete SCI participants without spasticity (dashed) and healthy control participants (dotted), excluding participant 3. (b) The angle and torque reference points in **a** were set at zero. (c) Angle–torque curve for SCI participants with spasticity (both complete and incomplete) from **a**, with grayscale intensities of lines indicating injury duration (months). (d) The angle and torque reference points in **c** were set at zero.

participants than in the age-matched healthy control participants,^{38–44} presumably because of the changes in proprioceptor function or muscle atrophy. In these studies, the time-dependent decline in Mmax may be caused by muscle atrophy, but because we did not measure Mmax in our study, we cannot conclude that the spinal reflex excitability in chronic SCI is maintained. Further research is needed to clarify this relationship.

Characteristics of the mechanical properties of the paralyzed ankle joint in SCI

Previous research¹⁶ indicates that the mathematical models used to calculate the mechanical parameters greatly impact the experimental result. Essentially, the change in a mechanical parameter (for example, stiffness) varied depending on the selected mathematical model applied to the angle–torque relation; thus, we evaluated several mathematical models (SOP, FOP and SK) to calculate each mechanical parameter. In this study, the peak torque and stiffness index (SI_{FOP}) calculated from the FOP model were inversely correlated with the injury duration. The determination coefficients for the angle–torque data fitted with the FOP model were significantly higher than the coefficients based on the alternative models (SOP, SK), therefore the result (SI_{FOP}) calculated using the FOP model was deemed to be the most reliable. Notably, SI_{SOP} , SI_{SK} and $Angle_{SLACK}$ calculated using the SOP and SK models were not significantly correlated with the injury duration. This observation likely reflects the inappropriate data fitting observed in some participants and the insufficient sample size; however, the underlying mechanism is unclear.

In addition, work was not significantly correlated with injury duration, which is consistent with a prior study asserting that work is not a spasticity indicator.⁴⁵ Furthermore, the decreased peak

torque and SI_{FOP} over time suggest that plantar-flexor elasticity decreases with injury duration. However, several studies have found that the plantar-flexor passive-elasticity in the chronic SCI patients was higher than observed in the healthy age-matched participants.^{24,26,46} We speculate that plantar-flexor elasticity in SCI patients increases with immobilization during the acute stage of injury (<1 year), and thereafter, elasticity gradually decreases over time due in part to the muscle atrophy observed in this study. In theory, muscle morphology influences passive tension. Thus, we calculated the partial correlation coefficient between mechanical parameters and injury duration to exclude effects from morphological parameters (Table 2); significant relationships persisted, therefore we concluded that tissue elasticity in the plantar-flexor was involved in the mechanical parameter changes.

We cannot determine whether the muscle or tendon caused the plantar-flexor elasticity change. Theoretically, both muscle and tendon arranged in series can affect the total tissue stiffness,⁴⁷ a relationship that has been experimentally confirmed in the plantar-flexors of healthy participants.¹⁷ In contrast, Diong *et al.*²⁴ observed that the Gas muscle stiffness is increased in SCI participants, whereas Olson *et al.*⁶ found that the muscle changes occur at the cell level and reflect muscle fiber transformation from Type I to Type II. In addition, Maganaris⁴⁸ showed that patellar tendon CSA, stiffness, and Young's modulus decreased significantly in chronic (18–288 months after injury) complete SCI patients. Overall, these studies suggest that the tendon rather than the muscle has a primary role in the decreasing plantar-flexor elasticity. Further research is needed to clarify the different changes in muscle and tendon function over time following a SCI.

Figure 5 Relationships between mechanical parameters and injury location. Complete SCI participants with spasticity (filled circles), incomplete SCI participants with spasticity (open circles), complete SCI participants without spasticity (x).

Limitations

We did not measure the range of motion of plantar-dorsiflexion in the SCI participants. Therefore, it is unclear whether contracture occurred in SCI participants. However, we visually confirmed that all

participants achieved a 20° dorsiflexion while keeping the heel in contact with the dynamometer footplate during the passive motion test. In addition, we confirmed that muscle activity in the calf muscles (MG, Sol) and antagonist (tibialis anterior) were low (within the

baseline mean $\pm$ 3 s.d.) during the passive motion test (representative participant in Figure 2a). Thus, the influence of articular alternation and muscle activity on the plantar-flexor torque was minimal, and the passive motion test was conducted appropriately.

In conclusion, the degree of spasticity (MAS), morphologic (circumference and MG muscle thickness) and mechanical (muscle-tendon elasticity) properties of the plantar-flexors in chronic SCI patients with spasticity decreased with the injury duration. Therapeutic intervention, such as FES or BWST, may be needed to prevent these sequelae in chronic SCI patients.

DATA ARCHIVING

There were no data to deposit.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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