Exp Brain Res (2001) 141:446–459
DOI 10.1007/s00221-001-0901-z

RESEARCH ARTICLE

M.M. Mirbagheri · H. Barbeau · M. Ladouceur
R.E. Kearney

Intrinsic and reflex stiffness in normal and spastic, spinal cord injured subjects

Received: 25 January 2001 / Accepted: 5 September 2001 / Published online: 26 October 2001

Abstract Mechanical changes underlying spastic hypertonia were explored using a parallel cascade system identification technique to evaluate the relative contributions of intrinsic and reflex mechanisms to dynamic ankle stiffness in healthy subjects (controls) and spastic, spinal cord injured (SCI) patients. We examined the modulation of the gain and dynamics of these components with ankle angle for both passive and active conditions. Four main findings emerged. First, intrinsic and reflex stiffness dynamics were qualitatively similar in SCI patients and controls. Intrinsic stiffness dynamics were well modeled by a linear second-order model relating intrinsic torque to joint position, while reflex stiffness dynamics were accurately described by a linear, third-order system relating half-wave rectified velocity to reflex torque. Differences between the two groups were evident in the values of four parameters, the elastic and viscous parameters for intrinsic stiffness and the gain and first-order cut-off frequency for reflex stiffness. Second, reflex stiffness was substantially increased in SCI patients, where it generated as much as 40% of the total torque variance, compared with controls, where reflex contributions never exceeded 7%. Third, differences between SCI patients and controls depended strongly on joint position, becoming larger as the ankle was dorsiflexed. At full plantarflexion, there was no difference between SCI and control subjects; in the mid-range, reflex stiffness was abnormally high in SCI patients; at full dorsiflexion, both reflex and intrinsic stiffness were larger than normal. Fourth, differences between SCI and control subjects were smaller during the active than the passive condition, because intrinsic stiffness increased more in controls than SCI subjects; nevertheless, reflex gain remained abnormally high in SCI patients. These results elucidate the nature and origins of the mechanical abnormalities associated with hypertonia and provide a better understanding of its functional and clinical implications.

Keywords Ankle stiffness · Spastic hypertonia · Muscle tone · Spinal cord injury · Stretch reflex · Human

M.M. Mirbagheri · R.E. Kearney (✉)
Department of Biomedical Engineering, McGill University,
3775 University Street, Montreal, QC, Canada, H3A 2B4
e-mail: Robert.Kearney@mcgill.ca
Tel.: +1-514-3986737, Fax: +1-514-3987461

H. Barbeau · M. Ladouceur
School of Physical and Occupational Therapy, McGill University,
Montreal, Canada

Introduction

Spasticity is a motor disorder associated with lesions at different levels of the nervous system due to traumatic injury, multiple sclerosis, cerebral palsy, or stroke (Sehgal and McGuire 1998). Clinical symptoms are diverse; they include hypertonia, autonomic hyperreflexia, flexor or adductor spasms, clonus, Babinski's sign, the clasp-knife phenomena, loss of sensory function, and dyssynergic patterns of contraction (Katz and Rymer 1989; Crozier et al. 1991; Calancie et al. 1993; Waters et al. 1994; Jamshidi and Smith 1996; Perell et al. 1996; Lamontagne et al. 1998; Sehgal and McGuire 1998). There are also performance deficits such as loss of dexterity, paresis, and fatigability (Rymer and Power 1987; Katz and Rymer 1989; Lehmann et al. 1989; Gerhart et al. 1993; Little et al. 1994; Levi et al. 1996; Needham-Shropshire et al. 1997; Thomas et al. 1997, 1998; Drolet et al. 1999). Nevertheless, *hypertonia*, an abnormal increase in muscle tone, is regarded as the defining feature of spasticity (Lance 1980), having both diagnostic and therapeutic significance (Katz and Rymer 1989).

Muscle tone is characterized subjectively as "the sensation of resistance felt as one manipulates a joint through a range of motion, with the subject attempting to relax" (Lance and McLeod 1981). Quantitative, objective evaluation of muscle tone remains difficult, since there is as yet no universally accepted means of measuring it (Berardelli et al. 1983; Verrier et al. 1984; Capaday and Stein 1987; Berger et al. 1988; Powers et al. 1988; Lehmann et al. 1989; Dietz et al. 1991; Katz et al. 1992;

Sinkjaer et al. 1993; Toft et al. 1993; Sinkjaer and Magnussen 1994; Engsberg et al. 1996; Wolf et al. 1996). One approach is to measure the torques evoked by perturbing joint position (Lee et al. 1987; Powers et al. 1988, 1989; Dietz et al. 1991; Ibrahim et al. 1993; Sinkjaer et al. 1993; Sinkjaer and Magnussen 1994). In the passive condition, when subjects are relaxed, there is general agreement that torque responses are greater in spastic subjects than normals (Powers et al. 1988; Ibrahim et al. 1993). The picture is less clear during the active condition, when subjects maintain a voluntary contraction. Some reports indicate that torques are increased (Sinkjaer et al. 1993; Sinkjaer and Magnussen 1994), while others show the spastic responses are unchanged or even decreased compared with those of healthy subjects (Powers et al. 1988; Dietz et al. 1991; Ibrahim et al. 1993). Another approach to the assessment of hypertonia is to measure the mechanical properties of the spastic joint in terms of its overall stiffness (Rack et al. 1984; Lee et al. 1987; Powers et al. 1988; Lehmann et al. 1989; Price et al. 1991; Meinders et al. 1996) or compliance (Gottlieb et al. 1978). Once again, there is no consensus; some report that stiffness is increased in spasticity (Gottlieb et al. 1978; Lehmann et al. 1989; Price et al. 1991), while others find no significant difference from normals (Rack et al. 1984; Lee et al. 1987; Powers et al. 1988; Meinders et al. 1996).

The origin of the mechanical abnormalities associated with spasticity also remains controversial. One hypothesis is that they are due to hyperactive stretch reflexes, since tendon jerks and reflex electromyograms (EMGs) are increased in spasticity (Corcos et al. 1986; Eisen 1987; Lee et al. 1987; Powers et al. 1988, 1989; Fellows et al. 1989; Thilmann et al. 1990, 1991). An alternative hypothesis is that the mechanical abnormalities result from changes in the intrinsic mechanical properties of spastic muscles and/or passive tissues secondary to a lesion (Berger et al. 1982; Hufschmidt and Mauritz 1985; Dietz et al. 1991; Ibrahim et al. 1993; Sinkjaer et al. 1993; Sinkjaer and Magnussen 1994). This view is supported by observations suggesting that reflex torques are not increased in spasticity (Dietz et al. 1981, 1986, 1991; Berger et al. 1982, 1988; Dietz and Berger 1983; Ibrahim et al. 1993).

The present study was designed to address two issues regarding spastic hypertonia: (1) What is the nature of the mechanical changes associated with spastic hypertonia? (2) What are the relative contributions of intrinsic and reflex mechanisms to these mechanical changes? We used system identification techniques to measure ankle dynamic stiffness at different positions through the range of motion (ROM), for passive and active conditions, in both spastic, spinal cord injured (SCI) and healthy (control) subjects. Important factors of this study were: (1) the mechanical properties of the joint were fully described in terms of its dynamic stiffness at operating points through the normal range of motion, and (2) the relative contributions of intrinsic and reflex stiffness were distinguished using a parallel-cascade identification technique.

Our results demonstrate that: (1) overall joint stiffness was substantially greater in SCI subjects than in controls; (2) both intrinsic and reflex mechanical responses were significantly increased in SCI patients, but the major mechanical abnormality arose from increased reflex stiffness; and (3) the relative contributions of reflex and intrinsic changes were strongly dependent on position and condition. Aspects of this work have been presented at several conferences (Mirbagheri et al. 1998a, 1998b).

Methods

Subjects

Nine (five women, four men) SCI patients were examined. All SCI subjects had: (1) clinically evident spasticity, associated with traumatic spinal cord injury with incomplete motor function loss, (2) enough retained voluntary control to generate contractions in the ankle muscle, and (3) long-term spasticity of at least 2 years duration. Table 1 provides details for each SCI subject. The time between the injury and the start of experiment varied from 2.3 to 13 years. The level of the lesion ranged from the fifth cervical vertebra (C5) to the first lumbar vertebra (L1). The cause of injury was motor vehicle accident (MVA) in eight subjects and a fall in one subject. Ten healthy subjects (five women,five men) with a mean age of 26.5±3 years and no history of neuromuscular disease served as controls. All subjects gave informed consent to the experimental procedures, which had been reviewed and approved by McGill University's Research Ethics Board.

Clinical assessment

SCI subjects were evaluated clinically prior to each experiment using: (1) the modified 5-point Ashworth scale (Ashworth 1964; Bohannon and Smith 1987) to assess muscle tone, and (2) the Fugel-Meyer scale (Fugl-Meyer et al. 1975; Fugl-Meyer 1976) to assess function. These scales are the most common clinical assessment methods. To avoid interexaminer variability, the first author performed all evaluations.

In SCI subjects with incomplete motor function loss, the left and right sides are often affected differently. Consequently, both sides of the SCI subjects were assessed, and we examined the side with the highest modified Ashworth scale and lowest maximum voluntary contraction (MVC) both conditions always occurred on the same side.

Apparatus

The apparatus has been described previously in detail (Mirbagheri et al. 2000). In brief, subjects lay supine with their foot attached to the pedal of an actuator by a custom-fitted, fiber glass boot. The knee was held fully extended by sandbags and a knee strap. Joint position and torque were measured with a precision potentiometer and torque transducer, mounted in the actuator. A 90° angle between shank and foot was considered to be the neutral position and defined as zero. Displacements in the dorsiflexing direction were taken as positive and those in the plantarflexing direction as negative. Torque was assigned a polarity consistent with the direction of movement it would generate (e.g., a plantarflexing torque was taken as negative).

EMGs were recorded from the lateral head of the gastrocnemius (GS) and the belly of the tibialis anterior (TA) using bipolar surface electrodes placed parallel to the muscle fiber direction, with an interelectrode distance of 1 cm. The reference electrode was attached over the patella. Position, torque, and EMGs were filtered at 200 Hz to prevent aliasing, and sampled at 1 kHz.

Table 1 Characteristics of spastic, spinal cord injured (SCI) subjects. (*Age* Age at time of examination, *Duration* duration of injury prior to examination, *Level* spinal cord level of injury, *Etiology* cause of injury, *MVA* motor vehicle accident, *PF MVC* maximum voluntary contraction in plantarflexion, *DF MVC* maximum voluntary contraction in dorsiflexion, *Max DF* greatest dorsiflexing position examined, *Ashworth* modified Ashworth scale, clinical assessment of muscle tone, *Fugl-Meyer* Fugl-Meyer scale value, clinical assessment of function, *NM* MVCs not measured owing to spasms)

Subjects	Age (years)	Duration (years)	Level	Etiology	PF MVC (Nm)	DF MVC (Nm)	Max DF (rad)	Ashworth	Fugl-Meyer
BA	45	3	C4-5	MVA	–43	24	0.24	1	32
DF	37	3.5	T1-2	MVA	–15	19	0.24	4	33
FF	43	2.3	T5-6	Fall	–50	30	0.24	2	32
FG	49	3.7	C5-6	MVA	–46	8	0.16	1	23
MS	28	8.8	C6	MVA	–36	1	0.24	4	18
MF	40	2.9	C3-4	MVA	–61	30	0.24	3	31
PO	22	6	C7-T1	MVA	–38	7	0.24	4	31
SC	46	13	C4-5	MVA	NM	NM	0.00	3	30
SM	25	4.3	C5-6	MVA	–24	5	0.16	3	27
Mean	37.2	5.5			–39.1	15.5	0.20	2.8	28.6
SD	10.6	3.7			14.5	11.7	0.08	1.2	5.0
SE	0.3	0.67			0.36	0.75	0.41	0.43	0.18

Procedures

Actuator dynamic response

The electrohydraulic actuator was operated as a position control servo driving ankle position to follow a command input. The actuator's dynamic response and torque capabilities were much greater than those of the subject's ankle, so that the overall response was flat to more than 80 Hz and the position change was essentially independent of the subject's response.

Pseudorandom binary sequence trials

To identify overall stiffness properties and separate the reflex and intrinsic components, we used pseudorandom binary sequence (PRBS) position inputs with amplitude of 0.03 rad and a switching rate of 150 ms (see Fig. 2). These perturbations (1) had a mean velocity low enough to avoid attenuating reflex responses overly, (2) contained power over a wide enough bandwidth to identify the dynamics, and (3) were tolerated well by the SCI subjects (Mirbagheri et al. 1997a).

Pulse trials

Pulse trials were applied to the ankle before and after each PRBS trial as described by Mirbagheri et al. (2000) to control for changes in the subject's "state". Each pulse trial comprised five pulses with an amplitude of 0.03 rad and pulse-width of 40 ms. Pulses were applied with a minimum interpulse interval of 5 s. EMGs from TA and GS and torque were recorded, ensemble-averaged, and compared. Amplitude changes of the reflex EMG or torque responses greater than 20% were taken as evidence of a change in the subject's state, due to fatigue or other factors, and data for the corresponding PRBS trial were discarded. This occurred very rarely; in most experiments no trials were discarded.

Conditions

Trials were conducted at ankle positions from full plantarflexion (PF; –0.48 rad) to near maximum dorsiflexion (DF; +0.24 rad), at 0.08 rad intervals. Each position was examined under both the (1) *passive* condition, where subjects were instructed to remain relaxed, and (2) *active* condition, where subjects maintained a tonic PF contraction of –5 Nm, aided by visual feedback of ankle torque.

Following each trial, the torque and EMG signals were examined for evidence of nonstationarities or coactivation of TA. If there was evidence of either, the data were discarded and the trial was repeated.

Analysis procedures

Parallel cascade identification technique

Reflex and intrinsic contributions to ankle dynamic stiffness were identified using a parallel cascade technique, described in detail previously (Kearney et al. 1997; Mirbagheri et al. 2000). Briefly, the method proceeded as shown in Fig. 1:

1. Reflex EMG dynamics were estimated in terms of a linear impulse response function (VGS_{IRF}) relating half-wave rectified velocity EMG to EMG. The latency of this IRF was used to estimate the conduction delay associated with the reflex pathway.
2. Intrinsic stiffness dynamics, shown in the top pathway, were estimated in terms of a linear impulse response function (PTQ_{IRF}) relating position and torque. The length of PTQ_{IRF} was limited to that of the reflex delay (~40 ms) to prevent contamination by reflex effects.
3. Reflex stiffness dynamics, shown in the bottom pathway, were estimated in terms of a parallel cascade pathway comprising a differentiator (*s*), a static nonlinearity (*N*), and a linear impulse response function (VTQ_{IRF}). The static nonlinearity, which closely resembled a half-wave rectifier, was normalized so that the entire linear gain in the pathway was assigned to VTQ_{IRF}.
4. The intrinsic and reflex stiffness IRFs were convolved with the experimental input to predict the intrinsic and reflex torques, respectively.

Note that the method characterizes the relation between position and torque and does not attempt to predict torque from EMG; EMG is used only to estimate the reflex delay.

Intrinsic compliance

The dynamic inverse of the intrinsic stiffness dynamics (PTQ_{IRF}) was computed to give the intrinsic compliance (TQP_{IRF}). The Levenberg-Marquardt nonlinear least-square fit algorithm (Press et al. 1985) was then used to find the parameter set that minimized the mean squared-error between TQP_{IRF} and the impulse response of the second-order system:

$$TQP_{IRF}(s) = \frac{P(s)}{TQ_1(s)} = \frac{1}{I s^2 + Bs + K} \quad (1)$$

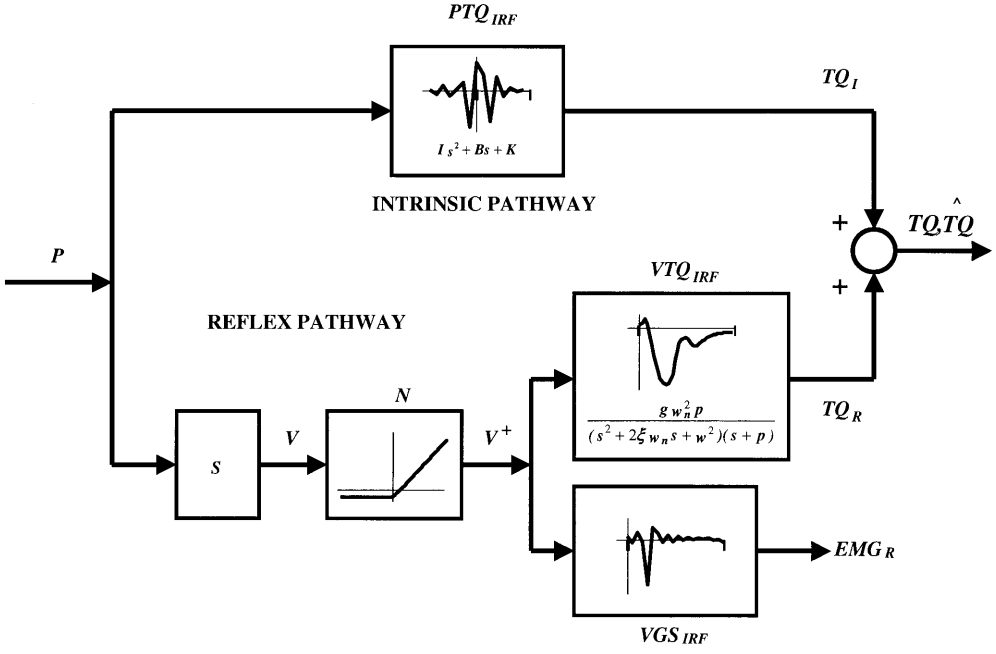


Fig. 1 The parallel cascade structure used to identify intrinsic and reflex stiffness. Experimental data include the position input (*P*), torque (*TQ*) and GS EMG (EMG_R). Intrinsic stiffness is represented in the upper pathway by the intrinsic stiffness impulse response function (PTQ_{IRF}), which has two-sided, high-pass structure. Reflex stiffness is represented in the lower pathway as a differentiator, followed by a static nonlinear element (*N*) and then a linear impulse response function (VGS_{IRF}). The nonlinear element closely resembles a half wave rectifier, and VTQ_{IRF} is low pass in nature. Reflex EMG dynamics (VGS_{IRF}) were characterized by an impulse-like response occurring at a lag of about 40 ms

where *P* is joint angle, TQ_1 is intrinsic torque, *I* is inertial parameter, *B* is viscous parameter, *K* is elastic parameter, and *s* is Laplace variable.

Note that the intrinsic elastic parameter *K* also corresponds to the steady-state gain of the intrinsic stiffness. Typical compliance IRFs and their corresponding second-order fits are shown in the top row of Fig. 3.

Reflex stiffness

Parameters of the reflex stiffness impulse response function (VTQ_{IRF}) were found by fitting the third-order model (Mirbagheri et al. 1995);

$$VTQ_{IRF}(s) = \frac{TQ_R(s)}{V(s)} = \frac{G_R\omega_n^2 p}{(s^2+2\xi\omega_n s+\omega_n^2)(s+p)}e^{-s\tau} \quad (2)$$

where TQ_R is reflex torque, *V* is joint angular velocity, G_R is reflex gain, ω_n is second-order natural frequency, ξ is damping parameter, *p* is first-order cut-off frequency, τ is reflex delay, and *s* is Laplace variable.

Typical reflex stiffness IRFs and their corresponding 3rd-order fits are shown in the top row of Fig. 4.

Statistical analysis

Comparisons between groups and conditions were done using standard t-test procedures. Only results with *P* values less than 5% ($P<0.05$) were considered to be significant.

Results

Experimental data

Figure 2 shows segments of experimental trials for typical SCI (left panel) and control (right panel) subjects at the neutral position for the active condition (–5 Nm plantarflexing contraction). The position inputs (Fig. 2, top row) were almost identical in both cases; the PRBS inputs had a peak-to-peak amplitude of 0.03 rad and a switching interval of 150 ms. The EMG (Fig. 2, second row) and torque (third row) responses were much larger for the SCI than the control subject but were qualitatively similar. Thus, the GS EMG (Fig. 2, second row), displayed a characteristic, unidirectional rate-sensitive behavior in both subjects: a short-latency burst of EMG followed each movement in the dorsiflexing (positive) direction; equivalent movements in the opposite direction evoked no response. However, the peak EMG of the SCI subject (Fig. 2c) was more than 10 times that of the control subject (Fig. 2d). Similarly, both torque responses comprised two distinct components: (1) a symmetric variation correlated with the ankle position and its derivatives with no significant delay, probably representing the contribution of intrinsic mechanics; and (2) a transient, twitch-like increase in torque associated with dorsiflexing displacements only, which we have previously shown to be due to reflex mechanisms (Stein and Kearney 1995). These two components are seen more clearly in the bottom row of Fig. 2, where segments of the torque records are shown on an expanded time scale with the reflex components shaded. The SCI reflex torque was more than four times larger than that of the control subject.

Figure 3 summarizes the intrinsic stiffness analysis of the Fig. 2 data. The dashed curves in the top row are the intrinsic compliance IRFs estimated for the SCI (Fig. 3a) and control (Fig. 3b) subjects. These were similar in shape and magnitude, although the SCI IRF appeared to be more heavily damped, since its oscillation died out more rapidly. Second-order fits to these compliance IRFs, shown by the superimposed solid curves, were very good. In both cases, the variance accounted for

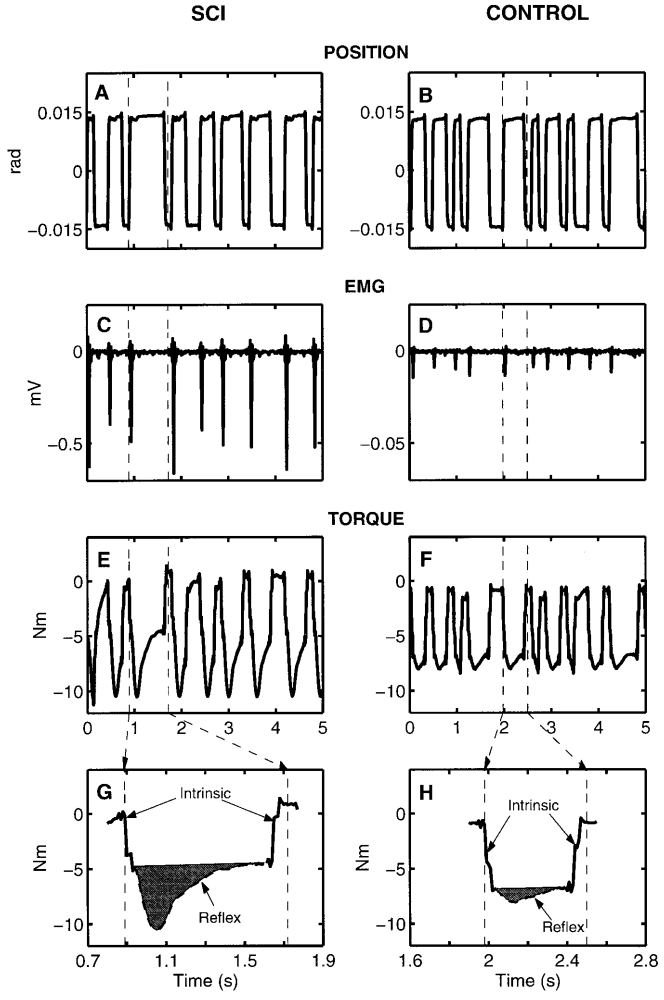


Fig. 2a–h Five-second segments from typical sequence trials for a spastic spinal cord-injured (SCI; *left panel*) and a control (*right panel*) subject. (Neutral position, active condition) **a, b** Position (*P*); **c, d** reflex EMG (EMG_R); **e, f** torque (*TQ*); **g**, **h** a segment of the torque record between *dashed lines* on an expanded scale. The reflex component of the response is *shaded*. Plantarflexing displacements and torques are taken as positive

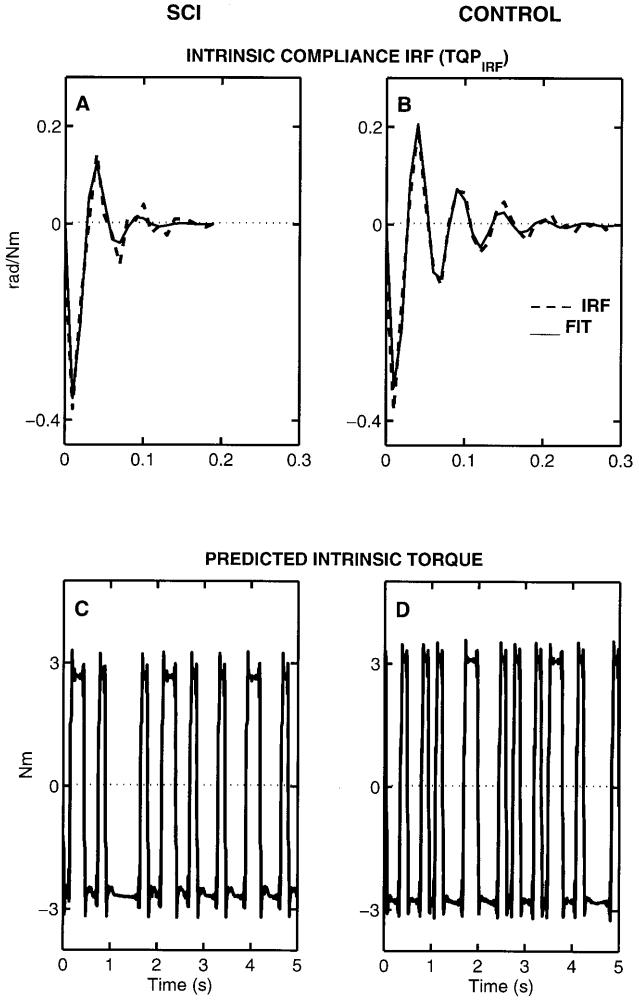


Fig. 3a–d Typical intrinsic dynamics and predicted torques estimated for the SCI (*left panel*) and control (*right panel*; data from Fig. 2). **a, b** Intrinsic compliance (TQP_{IRF}); the *dashed curve* is the nonparametric IRF, the *solid curve* is the parametric fit; **c, d** predicted intrinsic torque

(VAF_{FIT}) was greater than 92%, as was typical of all our data; VAF_{FIT} for the compliance IRF was always greater than 90%. The intrinsic torques predicted by these IRFs, shown in Fig. 3c and d, were comparable in magnitude and waveform, suggesting that the differences in IRF shapes had little effect on the torque responses evoked by our perturbations.

The reflex stiffness analysis for the same data is summarized in Fig. 4. The reflex stiffness IRFs estimated for the spastic (Fig. 4a) and control (Fig. 4b) subjects are shown as dashed lines. Third-order model fits to these reflex stiffness IRFs were very good, as indicated by the superimposed solid curves. These fits were always very good, never accounting for less than 85% of the variance. The amplitude of reflex stiffness IRF for the SCI subject (Fig. 4a) was 4 times that of the control subject (Fig. 4b). In addition, the duration of the SCI reflex stiffness IRF (600 ms), defined as the time taken to decay to 10% of the peak amplitude, was almost twice that of the control (350 ms). The reflex torques predicted by these IRFs reflected these differences; the peak-to-peak torque of the SCI subject in Fig. 4c (~6 Nm) was 4 times that of the control subject in Fig. 4d (~1.5 Nm).

Stiffness during the passive condition

Figure 5 shows the group means and standard errors of intrinsic and reflex stiffness parameters for the passive condition. The narrow standard error bars demonstrate that the overall behavior was very consistent for both groups.

The intrinsic stiffness gain (*K*; Fig. 5a) and viscous parameter (Fig. 5b) were always larger for SCI patients than controls, and the differences increased as the ankle was dorsiflexed. Positions where these differences were

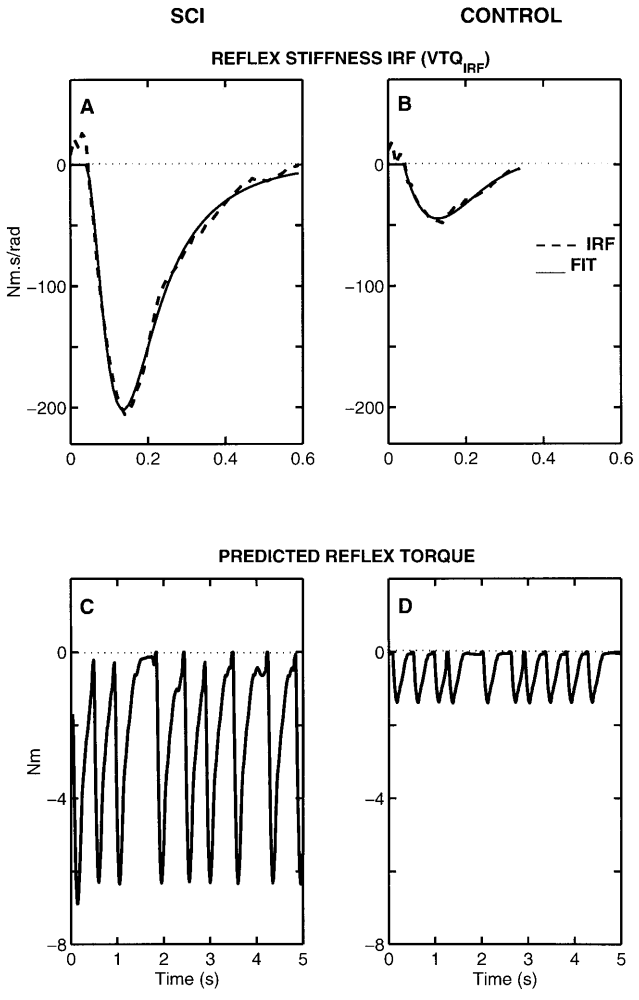
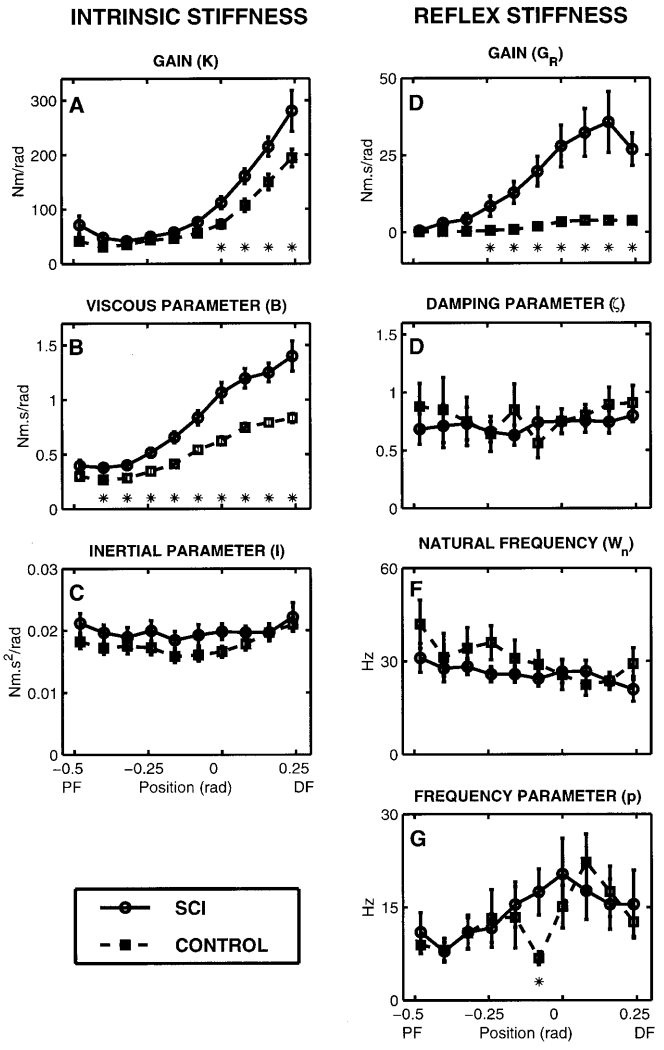


Fig. 4a–d Typical reflex dynamics and predicted torques for the SCI (*left panel*) and control (*right panel*). (Data from Fig. 2). **a, b** Reflex dynamic stiffness (VTQ_{IRF}); the *dashed curve* is the nonparametric IRF estimate, the superimposed *solid curve* is the parametric fit; **c, d** predicted reflex torque



PASSIVE CONDITION

Fig. 5a–g Position dependence of intrinsic and reflex stiffness for SCI and control subjects as functions of position (group means, passive condition). **a** Intrinsic stiffness gain (*K*); **b** intrinsic stiffness viscous parameter(*B*); **c** inertia (*I*); **d** reflex stiffness gain (G_R); **e** damping parameter (ξ); **f** the second-order natural frequency (w_n); **g** the first-order frequency (*p*). *Error bars* indicate ±1 SEM. *Asterisks* represent points where differences between SCI patients and controls are statistically significant ($P<0.05$). (*PF* plantarflexion, *NP* neutral position, 90°, *DF* dorsiflexion)

statistically significant ($P<0.03$) are denoted by asterisks. *K* and *B* increased with ankle dorsiflexion for both groups, as reported previously for control subjects (Mirbagheri et al. 2000). As expected, the inertial (*I*; Fig. 5c) parameter was similar in both groups ($P>0.3$) and was independent of position.

Reflex stiffness gain (G_R; Fig. 5d) was larger for SCI patients than for controls at most positions; the difference increased as the ankle was dorsiflexed and was statistically significant ($P<0.02$) for all positions greater than –0.25 rad. There was no significant difference between the mean values of the other three parameters ($P>0.3$), as shown in Fig. 5e–g. Position dependence was similar in both groups. Reflex stiffness gain increased dramatically as the ankle was dorsiflexed, with changes being larger in the SCI patients than controls. The damping parameter (ξ; Fig. 5e) did not change systematically, the second-order natural frequency (ω_n; Fig. 5f) decreased, and the first-order frequency (*p*; Fig. 5g) increased. Thus, for the passive condition, both intrinsic and reflex stiffness were significantly enhanced in the SCI patients, especially at dorsiflexed positions.

Stiffness during the active condition

Figure 6 shows the group means and standard errors of intrinsic and reflex stiffness parameters for the active condition, where subjects maintained a –5 Nm plantarflexing voluntary contraction. *K* was similar in both groups (Fig. 6a, $P>0.1$) for all positions. Whereas, *B* was

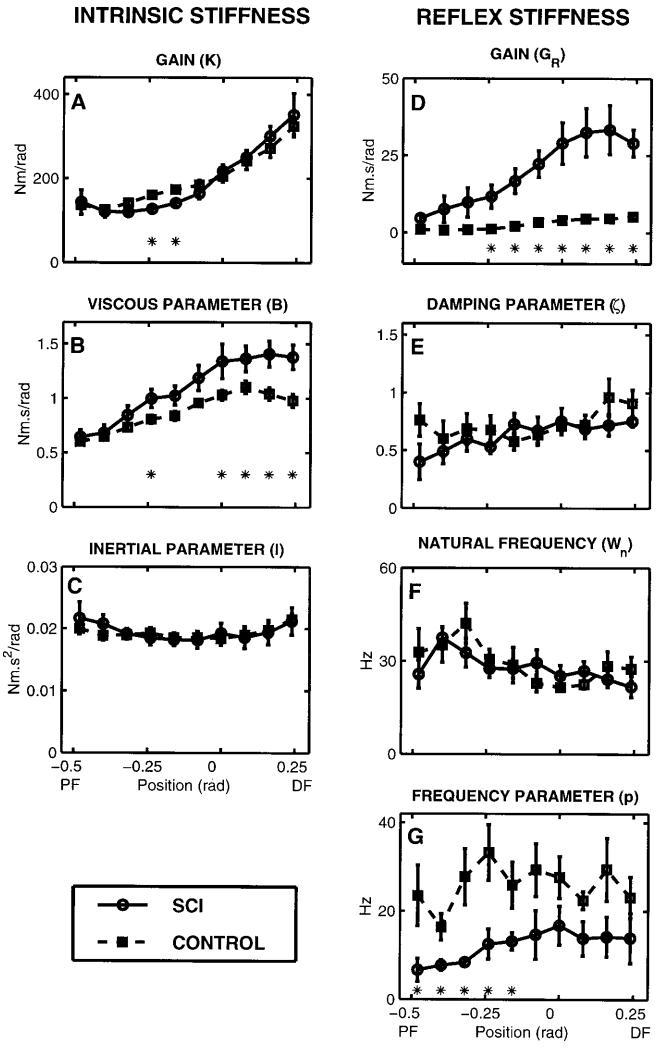


Fig. 6a–g Position dependence of intrinsic and reflex stiffness for SCI and control subjects as functions of position (group means, active condition). **a** Intrinsic stiffness gain (*K*); **b** intrinsic stiffness viscous parameter (*B*); **c** inertia (*I*); **d** reflex stiffness gain (G_R); **e** damping parameter (ξ); **f** the second-order natural frequency (w_n); **g** the first-order frequency (*p*). *Error bars* indicate ±1 SEM. *Asterisks* represent points where differences between SCI patients and controls are statistically significant ($P<0.05$). (*PF* plantarflexion, *NP* neutral position, 90°, *DF* dorsiflexion)

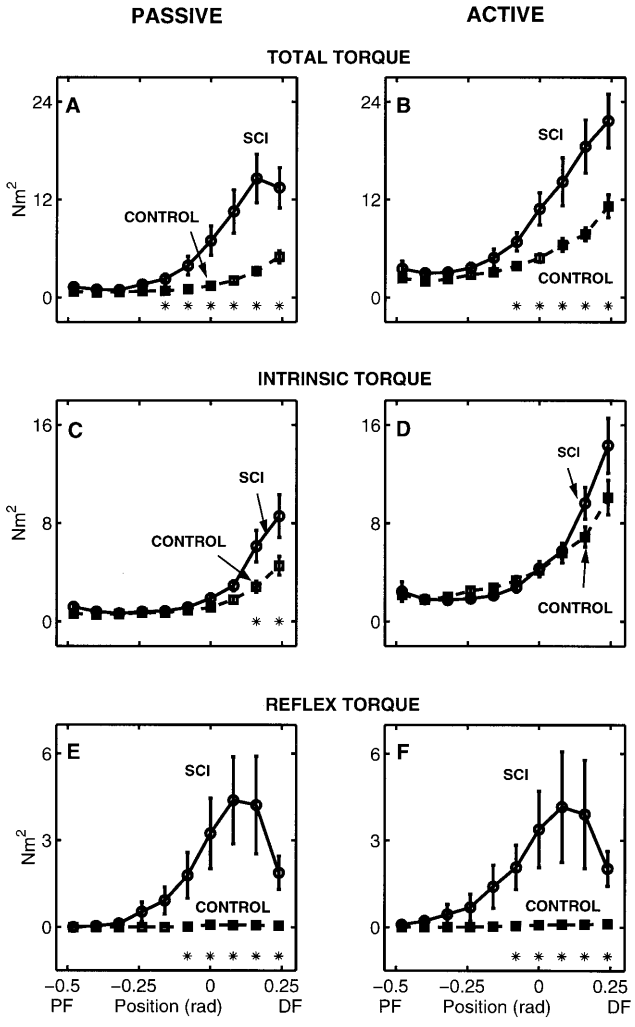


Fig. 7a–f Position dependence of torque variances for the SCI and control subjects (group means for the passive – *left column* – and active conditions – *right column*). **a, b** Total torque variance; **c, d** intrinsic torque variance; **e, f** reflex torque variance. *Error bars* represent ±1 SEM. *Asterisks* represent points where differences between SCI patients and controls are statistically significant ($P<0.05$). (*PF* plantarflexion, *DF* dorsiflexion)

significantly greater for SCI patients than controls ($P<0.05$) and the difference increased with dorsiflexion (Fig. 6b). *I* was similar in both groups ($P>0.25$) and independent of position (Fig. 6c).

As for the passive case, the reflex gain (G_R) was significantly larger in the SCI patients than the controls (Fig. 6d; $P<0.01$), while ξ (Fig. 6e) and ω_n (Fig. 6f) were similar for both groups ($P>0.2$). The major difference from the passive condition was that the first-order cutoff, *p*, was substantially smaller in the SCI group (Fig. 6g; $P<0.05$) for plantarflexed positions. Position dependence of reflex stiffness parameters was similar to that for the passive condition. Thus, for the active condition, reflex stiffness was significantly larger for SCI patients, while intrinsic stiffness was similar.

Intrinsic and reflex torque: absolute contributions

The preceding results demonstrate that the gain and dynamics of both intrinsic and reflex stiffness vary strongly with position and condition. The mechanical consequences of these changes are difficult to predict, since they will depend on the amplitude and frequency of the position input as well as the nonlinearity in the reflex pathway. To examine the mechanical consequences of these changes quantitatively, we computed the variance of the ankle torque as well as the predicted intrinsic and reflex torques as functions of position. Figure 7 summa-

rizes these results for both passive and active conditions. Three major points are apparent:

1. Total torque variance, shown in the top row, was significantly larger for SCI patients than for controls under both passive (Fig. 7a) and active (Fig. 7b) conditions ($P<0.05$); the difference increased as the ankle was dorsiflexed.
2. Intrinsic torque variance was significantly greater in SCI patients at the most dorsiflexed positions (more than 0.1 rad, $P<0.03$) for the passive condition as shown in Fig. 7c. For the active condition (Fig. 7d), there was no significant different between SCI and control subjects.
3. Reflex torque variance was substantially larger in the SCI patients than the controls at all positions from mid-plantarflexion to full dorsiflexion ($P<0.05$) for both passive (Fig. 7e) and active (Fig. 7f) conditions. Reflex torque variance was maximal between the zero (neutral) position and full dorsiflexion.

Intrinsic and reflex torque: relative contributions

Figure 7 describes the absolute contributions made by the intrinsic and reflex mechanisms to total torque variance. To assess their relative importance, we computed the ratio of the reflex torque variance to total torque variance. Figure 8 shows the relative reflex contribution as a function of position for both passive (Fig. 8a) and active conditions (Fig. 8b). Intrinsic contributions were complementary.

Three important points are evident:

1. Reflex torques were much larger relatively in the SCI patients than the controls at all positions for both conditions. The differences were significant for all positions from –0.25 rad through full dorsiflexion. The maximum reflex torque contribution was 40% for SCI patients, while it never exceeded 7% for controls (Fig. 8a).
2. The maximum relative reflex contribution occurred closer to the neutral position, that is, at a more plantarflexed position, than the maximum absolute contribution (Fig. 8e, f). This probably occurred because the intrinsic stiffness, K, increased with position more rapidly than did the reflex stiffness gain, G_R.
3. The relative reflex contribution was somewhat smaller for the active condition than the passive condition at corresponding positions. This reflects the observation that the reflex gain was almost the same for both conditions while the intrinsic gain increased from the active to passive condition.

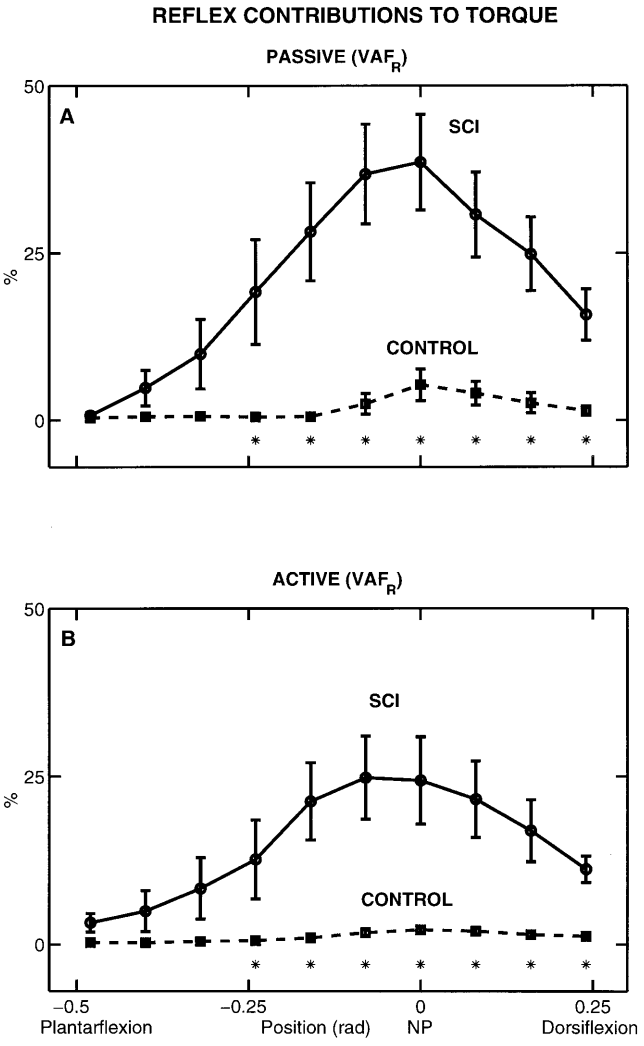


Fig. 8a, b Position dependence of relative reflex contribution to total torque (%VAF_R) for SCI and control subjects. Group means for **a** passive condition, **b** active condition. *Error bars* represent ±1 SEM. *Asterisks* represent points where differences between SCI patients and controls are statistically significant ($P<0.05$). (*NP* neutral position, 90°)

Table 2 Correlation coefficients (r) between stiffness parameters and clinical measures at neutral position (90°) under the passive condition. (K Intrinsic stiffness gain, G_R reflex stiffness gain, *Ashworth* modified Ashworth scale value, clinical assessment of muscle tone, *Fugl-Meyer* Fugl-Meyer scale value, clinical assessment of function)

	K	G_R
Ashworth	0.20	–0.21
Fugl-Meyer	0.34	–0.24

Correlations with clinical evaluation

We looked for correlations between our objective measures of dynamic joint stiffness and clinical assessments. We examined the data from each position and the active and the passive conditions separately but were unable to detect any significant correlation. Table 2 shows the results of the correlation analysis for one data set. These were acquired for the passive condition at the neutral position where the relative reflex contribution was maximal and differences between SCI and controls might be ex-

pected to be most evident. However, even for this case, there were no significant correlations between our quantitative measures of joint mechanics (K and G_R) and the clinical measures (modified Ashworth and Fugl-Meyer scales).

Discussion

Summary of results

We used a nonlinear, system-identification technique to examine the mechanical changes associated with spasticity in the ankle joint of SCI subjects. We believe these results provide the most comprehensive description of the mechanical abnormalities associated with spasticity to date because: (1) the mechanical behavior of the joint was described in terms of its dynamic stiffness at each operating point, (2) intrinsic and reflex stiffness were separated, simultaneously, using a parallel cascade identification technique, (3) the mechanical properties were examined over most of the range of motion, for both passive and active conditions, and (4) a homogenous group of SCI subjects was examined and compared with a control group.

The results of this study provide a number of new insights into spastic hypertonia:

1. First, there is no qualitative difference between joint stiffness and its components between SCI and control subjects. The same model structure worked equally well for both groups. Differences between the two groups were restricted to four parameters, two (K and B) related to intrinsic stiffness and two (G_R and p) related to reflex stiffness.
2. Secondly, the abnormalities in SCI joint stiffness, and its intrinsic and reflex components, vary with position over the range of motion. When the ankle was fully plantarflexed, there was no difference between the overall joint stiffness of SCI and control subjects. In the midrange (–0.2 to 0.2 rad), overall joint stiffness was larger in SCI patients, primarily due to abnormally high reflex stiffness. At full dorsiflexion, increased intrinsic stiffness also contributed to the larger overall stiffness in SCI patients. Thus questions about the nature and origin of hypertonia may have quite different answers depending upon where in the range of motion tests are made.
3. Finally, it demonstrates that differences between the stiffness of SCI and control subjects are smaller during the active than the passive condition. However, this does not result from a decrease in SCI reflex gain; it either increases or stays the same. Rather, it results from a larger increase in the intrinsic stiffness in controls than SCI patients. These findings do not support the hypotheses that (1) spastic reflexes are hyperexcitable for the passive condition but are normal or suppressed for the active condition (Cody et al. 1987; Berger et al. 1988; Dietz et al. 1991; Ibrahim et al. 1993; Sinkjaer et al. 1993; Sinkjaer and Magnussen 1994), and (2) active, spastic muscles generate more torque or stiffness in response to stretch than normals (Dietz and Berger 1983, Dietz et al. 1991; Lee et al. 1987; Ibrahim et al. 1993; Sinkjaer et al. 1993; Sinkjaer and Magnussen 1994).

Taken together, these results provide a better understanding of the nature and origin of the mechanical abnormalities associated with hypertonia. The strong dependence on operating point we have documented can be expected to have important functional and clinical implications and may explain some conflicting results in the literature. These ideas will be discussed in more detail in the following sections.

Methodological issues

The nature and origin of mechanical abnormalities associated with spastic hypertonia are still controversial, despite being the subject of numerous studies. Our results demonstrate that overall dynamic stiffness is increased in spasticity and that these dynamics incorporate a strong, nonlinear, velocity-dependent component. These findings have significant implications for the interpretation of previous studies. In particular, Lee et al. (1987) have characterized stiffness by applying torque steps and measuring the resulting static position change. This approach would provide an estimate of the intrinsic elastic stiffness but would not detect velocity-dependent components such as intrinsic viscosity or reflex stiffness that our results indicate make major contributions to overall stiffness.

Our results are also relevant to studies using linear analysis methods. In some cases sinusoidal inputs were applied and Fourier analysis was used to extract the component of the output at the input frequency and all other components discarded (Rack et al. 1984; Lehmann et al. 1989; Price et al. 1991; Meinders et al. 1996). This analysis procedure explicitly eliminates nonlinear contributions to joint dynamic stiffness; according to our results, this would ignore almost all of the reflex torque. Other studies have used an indirect analysis to relate the "pathlength" of the Nyquist diagram to reflex stiffness (Rack et al. 1984; Lehmann et al. 1989; Price et al. 1991; Meinders et al. 1996). This method was based on a simplified mathematical model that assumed linearity. Our results indicate that reflex stiffness is strongly nonlinear even for small perturbations about an operating point. Consequently, the pathlength approach is likely to provide heavily biased estimates of reflex contributions to overall stiffness

Our parallel cascade identification technique estimated intrinsic and reflex stiffness simultaneously for each experimental trial. This represents a major advantage over studies that estimated reflex contributions by comparing mechanical responses or properties before and after eliminating reflexes using electrical stimulation

(Sinkjaer et al. 1993; Sinkjaer and Magnussen 1994) or nerve block (Herman and Schaumburg 1968; Hufschmidt and Mauritz 1985; Lehmann et al. 1989). As we have discussed in detail previously (Mirbagheri et al. 2000), these approaches are lengthy and risk over- or underestimating reflex contributions because of changes in the intrinsic properties associated with eliminating the reflex responses (Lehmann et al. 1989; Mirbagheri et al. 2000).

Our findings demonstrate that intrinsic and reflex mechanisms were both strongly dependent on position and condition. These strong operating point dependencies could well explain conflicting reports in the literature. For example, according to our results, measures which reflect intrinsic elasticity (Lee et al. 1987) would detect differences between spastic patients and controls during the passive condition but not during the active condition. Similarly, the magnitudes of differences would be very dependent on the position of the joint; they are large at dorsiflexion and small at plantarflexion.

Limitations

The identification methods used in this study provide a dynamic description of the joint stiffness at particular operating points throughout the subject's range of motion. However, these models do not provide a comprehensive description of joint mechanics, since there are a variety of nonlinear effects it does not account for (see Discussion by Stein and Kearney, 1995). As a result, the model changes with the operating point defined by mean position, level of activation, perturbation amplitude and a variety of other parameters. Two effects are particularly noteworthy.

First, the changes in parameters with joint position are of particular importance, since they demonstrate that there is an important position-dependent nonlinearity not yet characterized. Our results describe the behavior at each operating point but not what happens during the transition between operating points. Consequently, these models cannot be used to predict the response to large displacements, such as the ramp displacements used by Rymer's group (Powers et al. 1988), which involve movements between operating points. Our models do, however, provide a convenient, concise summary of how stiffness changes with position in the steady state.

Secondly, it must be recognized that random perturbations will themselves influence the system. We have previously demonstrated that the intrinsic dynamics are not changed by ongoing random perturbations but that reflex stiffness decreases as the average velocity of the perturbation increases (Stein and Kearney 1995; Kearney et al. 1997). We controlled for these effects in these experiments by selecting a set of perturbation parameters that does not unduly attenuate the reflex response and using the same stimulus throughout. In view of the evidence that the stretch reflex gain is lower during movement than rest and modulated systematically throughout cyclic activities (see Brooke et al. 1997), it now appears that any experimental involving ongoing perturbations or movements must account for these effects.

Intrinsic stiffness

We found that, for the passive condition, the intrinsic stiffness gain and viscous parameter were larger in SCI patients than controls (Fig. 5a) and that as a result intrinsic torques were larger (Fig. 7c). This conclusion contrasts with that of Meinders et al. (1996), who found no change in intrinsic stiffness. The different conclusions probably arise because Meinder et al. studied only a narrow range of positions near the neutral position, where our results indicate differences between intrinsic stiffness of SCI patients and controls are small.

The increased intrinsic stiffness in SCI subjects, particularly at dorsiflexion for the passive condition, could be due to the accumulation of connective tissues in atrophic muscle secondary to the lesions (Katz and Rymer 1989; Sinkjaer and Magnussen 1994). In addition, persistent positional deformity often found in SCI subjects may result in changes in the length of muscle fibers and passive tissues (Herman and Schaumburg 1968; William and Goldspink 1978; Tardieu et al. 1982; Hufschmidt and Mauritz 1985; Katz and Rymer 1989; Thilmann et al. 1991).

Reflex stiffness

Our results demonstrate that reflex stiffness gain was substantially larger in SCI patients than in controls at most operating points; in some cases by as much as a factor of 7. The differences were:

1. Greatest at dorsiflexed positions where reflex stiffness was largest for both groups
2. Mirrored by changes in reflex torque variance indicating that the changes in reflex gain have substantial mechanical consequences
3. Large enough to significantly change the relative contribution of reflex mechanisms to overall ankle stiffness. Indeed, reflex contributions were always significantly greater in SCI patients than in controls. At its maximum, reflex stiffness generated as much as 40% of the torque variance in our experiments, while it never exceeded 7% for controls.

It is of interest to compare our results with those of Rymer's group (Lee et al. 1987; Powers et al. 1988, 1989), who attributed the reflex abnormality to a decrease in "reflex threshold." They used a large position ramp as input and defined the reflex threshold to be the position at which the ramp displacement first evoked a reflex response (Powers et al. 1988, 1989). In contrast, we applied small-amplitude PRBS displacements at different positions through the ROM and choose velocity as the input variable rather than position. These method-

ological differences prevent us from comparing our results to those of Rymer directly. However, an indirect comparison is possible. We found that in SCI subjects the reflex gain (Fig. 6d) and reflex torque variance (Fig. 7e,f) were substantial even at plantarflexion where reflex responses were completely absent in control subjects. This indicates that a ramp displacement, such as Rymer used, would first evoke reflex responses at more plantarflexed position in SCI patients than in controls. Consequently, our finding of increased reflex "gain" using velocity inputs is consistent with Rymer's finding of a decreased "threshold" using position inputs.

What physiological mechanisms might explain these changes? The increase in reflex gain in SCI subjects may be attributed to inappropriate recruitment of larger motor units in spastic subjects at rest or low levels of contraction (5–10% MVC) at which only small motor units are recruited in normal subjects. This is supported by our previous findings that reflex latency was shorter in SCI subjects, consistent with the early recruitment of larger motor units with larger, more rapidly conducting axons (Mirbagheri et al. 1998b). This hypothesis would also explain the weakness and fatigability observed in spastic subjects. Thus, early recruitment of large motor units would result in the activation of rapidly fatiguing motor units at low levels of contraction. This would cause inefficient muscle activation, induce early loss of force and fatigue, increase effort, and result in the perception of weakness (Katz and Rymer 1989).

Effects of voluntary contraction

It is widely accepted that spastic muscles behave differently under passive and active conditions (Powers et al. 1988; Dietz et al. 1991; Ibrahim et al. 1993). If true, this might explain some of the conflicting ideas regarding the origin of mechanical abnormalities associated with spasticity. Thus, the reflex hypothesis is supported mostly by studies carried out under passive conditions (Lance 1980; Powers et al. 1988; Fellows et al. 1989; Thilmann et al. 1990), while the evidence for the intrinsic hypothesis comes most from experiments using active conditions (Dietz et al. 1991; Ibrahim et al. 1993; Sinkjaer et al. 1993).

Some studies have concluded that active, spastic muscles generate more torque in response to stretch than normals (Dietz and Berger 1983; Lee et al. 1987; Dietz et al. 1991; Ibrahim et al. 1993), suggesting that intrinsic mechanisms are abnormal in spasticity. This conclusion was based on the observation that, for the active condition, torques evoked by transient inputs in spastic limbs were normal, whereas EMGs were lower; the mechanical properties of the active muscle were not measured directly. Our results, which measured these properties, do not support this interpretation. Figure 9 shows the change in intrinsic stiffness gain (Fig. 9a) and viscosity (Fig. 9b) from the passive to the active condition. Intrinsic stiffness increased with activation for both control and SCI

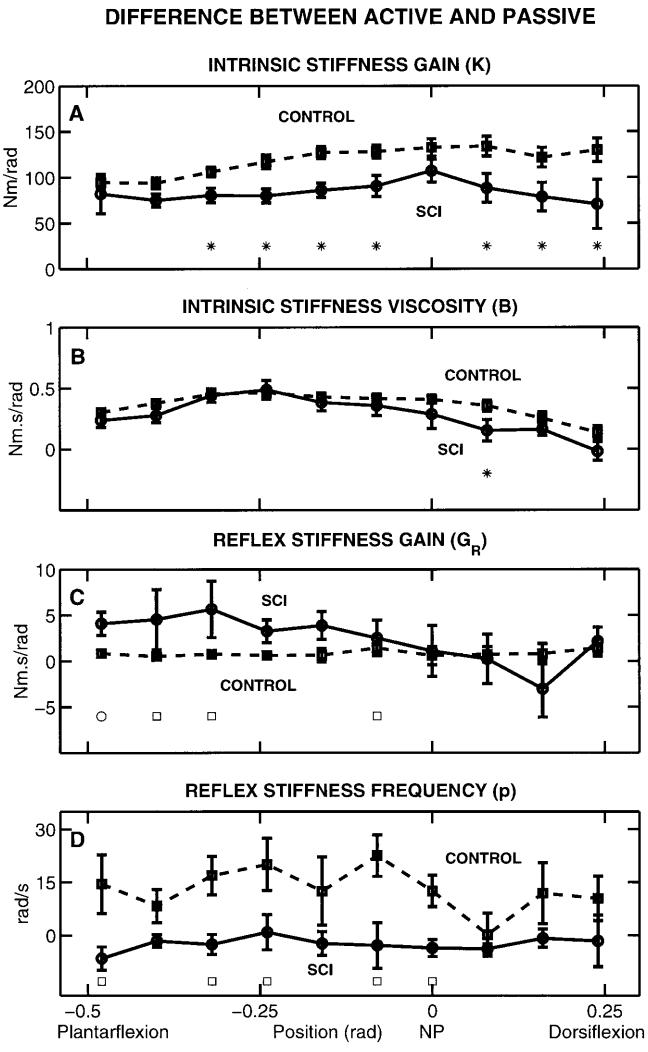


Fig. 9a–d Effects of activation (Group-mean differences between active and passive condition). **a** Intrinsic stiffness gain (*K*); **b** intrinsic stiffness viscous parameter (*B*); **c r**eflex stiffness gain (G_R); **d** reflex stiffness first-order frequency (*p*). *Error bars* represent ±1 SEM. *Asterisks* represent positions where differences between controls and SCI patients are significant. *Squares* and *circles* represent significant differences from zero for controls and SCI patients, respectively. (*NP* neutral position, 90°)

subjects; the increase was smaller for the SCI patients ($P<0.025$). The intrinsic viscous parameter increased with activation in both groups by similar amounts (Fig. 9b; $P>0.1$). Thus, the increase in intrinsic stiffness, and consequently intrinsic torque, with activation is actually less in SCI patients than normals. Why this should be is unknown. Perhaps changes in fiber length associated with spasticity, as discussed here, result in the SCI muscles operating at different points on the length tension curve (William and Goldspink 1978).

It is also widely accepted that spastic reflexes are hyperexcitable for the passive condition but are normal (Ibrahim et al. 1993; Sinkjaer et al. 1993; Sinkjaer and Magnussen 1994) or suppressed (Cody et al. 1987; Berger et al. 1988; Dietz et al. 1991) for the active con-

dition. This view is based on observations of the EMG and overall torque responses evoked by transient inputs (Berger et al. 1988; Dietz et al. 1991; Ibrahim et al. 1993) and by estimates of the magnitude of reflex torque response to pulse displacements obtained by using electrical stimulation to suppress reflexes (Sinkjaer et al. 1993; Sinkjaer and Magnussen 1994). Our results do not support this view, since, as Fig. 9c shows, the SCI reflex gain did not decrease with activation. Rather, at plantarflexed positions it increased with activation, while at dorsiflexed positions it did not change ($P>0.7$). This contrasts with the behavior of normal subjects, where the reflex gain increased by a constant, smaller amount at all positions. Thus, with activation, the differences in the reflex stiffness gain between SCI patients and controls either remain the same or increase.

Figure 9d demonstrates another notable difference in the effect of activation. The first-order frequency (p) did not change with activation in the SCI patients ($P>0.4$), while, for controls, it increased significantly at most positions ($P<0.05$). The natural frequencies, ω_n, were similar in both groups so that the overall bandwidth of the reflex response will be determined by p; this remained constant with activation for SCI patients, while for controls it increased.

In a preliminary simulation study (Mirbagheri and Kearney 2000), we examined the mechanisms underlying the third-order model of reflex dynamics and suggested that the second-order term was related to muscle dynamics while the first-order term was related to the pattern of neural activation. In particular, we found that the first-order term would arise from an oscillatory response of the motor neuron pool in which a single input pulse generates a large peak of activation at the latency of a monosynaptic reflex arc, followed by a several peaks, at intervals of about 140 ms, of decreasing amplitude. Moreover, the value of p was inversely related to the amplitude of the extra bursts. Consequently, the failure of p to increase with activation in SCI subjects suggests that motoneuon response was more oscillatory in SCI patients than in controls. Our inspection of the EMG dynamics (VGS_{IRF}) supported this; oscillations in the reflex dynamics were much more apparent in SCI patients than controls (Mirbagheri et al. 1995; Mirbagheri and Kearney 2000).

Clinical significance

Our results show that overall dynamic stiffness was significantly larger in spastic than normal subjects due to increases in both intrinsic and reflex stiffness. Their relative contributions were strongly dependent on position and condition; reflex contributions dominated at midposition, whereas intrinsic contributions did so at fully dorsiflexed positions. Clinical measures of muscle tone, such as the Ashworth scale, involve manipulating the joint through much of its range of motion and attributing a subjective measure of resistance to reflex mechanisms (Ashworth 1964; Lance 1980). They cannot distinguish the relative contribution of intrinsic or reflex mechanisms cannot or how they change with position. This may explain, at least partly, why we found no correlation between our objective measures of stiffness and the modified Ashworth scale. Perhaps clinical evaluations measure something other than joint dynamic stiffness – such as the way it changes with position.

It is also possible that the failure to detect a correlation between clinical measures of spasticity and our quantitative measures was the result of the random perturbations used in our study. As noted above these would be expected to attenuate the reflex response to some extent. Despite this, reflex stiffness was much greater in the SCI than the control group. Nevertheless, we cannot rule out the possibility that the effects of the perturbation in some way masked the clinical correlations. This might occur if, for example, the reflex responses in SCI patients were more (or less) sensitive to the effects of perturbation than the controls. Preliminary studies examining the effects of the stimulus properties on SCI and control subjects indicate the sensitivities are qualitatively similar in both groups (Mirbagheri et al. 1997a, 1997b); a comprehensive analysis of the effects is forthcoming.

Our results also show that in SCI patients the relative reflex contribution was larger for the passive than the active condition. Reflex torque and stiffness were similar for both conditions, but intrinsic torque and stiffness were much larger for the active than the passive condition. This may explain why hypertonia is more apparent when clinical evaluations are conducted under passive conditions (Lance 1980).

We conclude by noting that spasticity is a syndrome and not a disease; the term is used to describe disorders resulting from a variety of different lesions, having different features, and time courses. The present study controlled for this by using subjects with similar lesions (traumatic SCI patients) of long standing. It is possible that quite different results would have been obtained with spastic subjects of different etiology. Consequently we believe that similar objective, quantitative studies using this identification technique using other patient groups such as stroke, multiple sclerosis (MS), and brain injury would lead to a better understanding of mechanisms underlying spasticity.

References

Ashworth B (1964) Preliminary trial of carisoprodol in multiple sclerosis. Practitioner 192:540–542

Berardelli A, Sabra AF, Hallett M, Berenberg W, Simaon SR (1983) Stretch reflexes of triceps surae in patients with upper motor neuron syndromes. J Neurol Neurosurg Psychiatry 46:54–60

Berger W, Quintern J, Dietz V (1982) Pathophysiology of gait in children with cerebral palsy. Electroencephalogr Clin Neurophysiol 53:538–548

Berger W, Horstmann GA, Dietz V (1988) Spastic paresis: impaired spinal reflexes and intact motor programs. J Neurol Neurosurg Psychiatry 51:568–571

Bohannon RW, Smith MB (1987) Interrater reliability on a modified Ashworth scale of muscle spasticity. Phys Ther 67:206–207

Brooke JD, Cheng J, Collins DF, McIlroy WW, Misaszek E, Staines RE (1997) Sensori-sensory afferent conditioning with leg movement: gain control in spinal reflex and ascending paths. Prog Neurobiol 51:393–421

Calancie B, Broton JG, Klose KJ, Traad M, Difini J, Ayyar DR (1993) Evidence that alterations in presynaptic inhibition contribute to segmental hypo-yperexcitability after spinal cord injured in man. Electroencephalogr Clin Neurophysiol 89:177–186

Capaday C, Stein RB (1987) Difference in the amplitude of the human soleus H-reflex during walking and running. J Neurol Sci 392:513–522

Cody FWJ, Richardson HC, MacDermott N, Ferguson IT (1987) Stretch and vibration reflexes of wrist flexor muscles in spasticity. Brain 110:433–450

Corcos DM, Gottlieb GL, Penn RD, et al. (1986) Movement deficits caused by hyperexitable stretch reflexes in spastic humans. Brain 109:1043–1058

Crozier KS, Graziani V, Ditunno JF, Herbison GJ (1991) Spinal cord injured: prognosis for ambulation based on sensory examination in patients who are initially motor complete. Arch Phys Med Rehabil 72:119–121

Dietz V, Berger W (1983) Normal and impaired regulation of muscle stiffness in gait: a new hypothesis about muscle hypertonia. Exp Neurol 79:680–687

Dietz V, Quintern J, Berger W (1981) Electrophysiological studies of gait in spasticity and rigidity: evidence that altered mechanical properties of muscle contribute to hypertonia. Brain 104: 431–439

Dietz V, Ketelsen U-P, Berger W (1986) Motor unit involvement in spastic paresis. Relationship between leg muscle activation and histochemistry. J Neurol Sci 75:89–103

Dietz V, Trippel M, Berger W (1991) Reflex activity and muscle tone during elbow movements in patients with spastic paresis. Ann Neurol 30:767–779

Drolet M, Noreau L, Vachon J, Moffet H (1999) Muscle strength changes as measured by dynamometry following functional rehabilitation in individuals with spinal cord injury. Arch Phys Med Rehabil 80:791–800

Eisen A (1987) Electromyography in disorders of muscle tone. Can J Neurol Sci 14:501–505

Engsberg JR, Olree KS, Ross SA (1996) Quantitative clinical measure of spasticity in children with cerbral palsy. Arch Phys Med Rehabil 77:594–599

Fellows SJ, Garms E, Thilmann AF (1989) Transient and maintained phasic reflex components in spastic human subjects (abstract). J Physiol (Lond) 420:60

Fugl-Meyer AR (1976) Assessment of motor function in the hemiparetic patients. In: Buerger AA, Tobis JS (eds) Neurophysiological aspects of rehabilitative medicine. Thomas, Springfield, IL, pp 231–250

Fugl-Meyer AR, Jaasko L, Leyman I, Olsson S, Steglind S (1975) The post-stroke hemiplegic patient: method for evaluation of physical performance. Scand J Rehabil Med 7:13–31

Gerhart KA, Bergstrom E, Charlifue SW, Menter RR, Whiteneck GG (1993) Long-term spinal cord injury: functional changes over time. Arch Phys Med Rehabil 74:1030–1034

Gottlieb GL, Agarwal GC, Penn RD (1978) Sinusoidal oscillation of the ankle as a means of evaluating the spastic patient. J Neurol Neurosurg Psychiatry 41:32–39

Herman R, Schaumburg H (1968) Alterations in dynamic and static peroperties of the stretch reflex in patients with spastic hemiplegia. Arch Phys Med Rehabil 49:199–203

Hufschmidt A, Mauritz KH (1985) Chronic transformation of muscle in spasticity: peripheral contribution to increased tone. J Neurol Neurosurg Psychiatry 48:676–685

Ibrahim IK, Berger W, Trippel M, Dietz V (1993) Stretch induced electromyographic activity and torque in spastic elbow muscles. Brain 116:971–989

Jamshidi M, Smith AW (1996) Clinical measurement of spasticity using the pendulum test: comparison of electrogoniometric and videotape analyses. Arch Phys Med Rehabil 77:1129–1132

Katz RT, Rymer WZ (1989) Spastic hypertonia: mechanisms and measurement. Arch Phys Med Rehabil 70:144–155

Katz RT, Rovai GP, Brait C (1992) Objective quantification of spastic hypertonia: Correlation with clinical findings. Arch Phys Med Rehabil 73:339–347

Kearney RE, Stein RB, Parameswaran L (1997) Identification of intrinsic and reflex contributions to human ankle stiffness dynamics. IEEE Trans Biomed Eng 44:493–504

Lamontagne A, Malouin F, Richards CL, Dumas F (1998) Evaluation of reflex- and nonreflex-induced muscle resistance to stretch in adults with spinal cord injury using hand-held and isokinetic dynamometry. Phys Ther 78:964–975

Lance JW (1980) Symposium synopsis. In: Feldman RG, Koella WP (eds) Spasticity: disordered motor control. Year Book, Chicago, pp 485–494

Lance JW, McLeod JG (1981) Disordered muscle tone. In: Physiological approach to clinical neurology. Butterworths, Boston

Lee WA, Boughton A, Rymer WZ (1987) Absence of stretch reflex gain enhancement in voluntarily activated spastic muscle. Exp Neurol 98:317–335

Lehmann JF, Price R, deLateur BJ, Hinderer S, Traynor C (1989) Spasticity: quantitative measurements as a basis for assessing effectiveness of therapeutic intervention. Arch Phys Med Rehabil 70:6–15

Levi AD, Tator CH, Bunge RP (1996) Clinical syndromes associated with disproportionate weakness of the upper versus the lower extremities after cervical spinal cord injury. Neurosurgery 38:79–83

Little JW, Powers RK, Michelson P, Moore D, Robinson LR, Goldstein B (1994) Electrodiagnosis of upper limb weakness in acute quadriplegia. Am J Phys Med Rehabil 73:15–22

Meinders M, Price R, Lehmann JF, Questad KA (1996) The stretch reflex response in the normal and spastic ankle: effect of ankle position. Arch Phys Med Rehabil 77:487–492

Mirbagheri MM, Kearney RE (2000) Mechanisms underlying a third-order parameteric model of dynamic reflex stiffness. Annu Int Conf IEEE Eng Med Biol Soc 22

Mirbagheri MM, Kearney RE, Barbeau H (1995) Parametric modeling of the reflex contribution to dynamic ankle stiffness in normal and spinal cord injured spastic subjects. Annu Int Conf IEEE Eng Med Biol Soc 17:1241–1242

Mirbagheri MM, Kearney RE, Barbeau H, Ladouceur M (1997a) Abnormal stretch reflex mechanics in spastic spinal cord injured spastic subjects. Annu Int Conf IEEE Eng Med Biol Soc 19:1648–1649

Mirbagheri MM, Kearney RE, Barbeau H, Ladouceur M (1997b) Modulation of reflex mechanics with perturbation properties in normal and spastic spinal cord injured subjects. Soc Neurosci Abstr 23

Mirbagheri MM, Kearney RE, Barbeau H (1998a) Abnormal passive and intrinsic stiffness in the spastic ankle. Annu Int Conf IEEE Eng Med Biol Soc 20:2338–2340

Mirbagheri MM, Kearney RE, Barbeau H (1998b) Stretch reflex behavior of spastic ankle under passive and active conditions. Annu Int Conf IEEE Eng Med Biol Soc 20:2325–2327

Mirbagheri MM, Barbeau H, Kearney RE (2000) Intrinsic and reflex contributions to human ankle stiffness: variation with activation level and position. Exp Brain Res 135:423–436

Needham-Shropshire BM, Klose KJ, Tucker ME, Thomas CK (1997) Manual muscle test score and force comparisons after cervical spinal cord injury. J Spinal Cord Med 20:324–330

Perell K, Scremin A, Scremin O, Kunkel C (1996) Quantifying muscle tone in spinal cord injured patients using isokinetic dynamometric techniques. Paraplegia 34:46–53

Powers RK, Marder-Meyer J, Rymer WZ (1988) Quantitative relations between hypertonia and stretch reflex threshold in spastic hemiparesis. Ann Neurol 23:115–124

Powers RK, Campbell DL, Rymer WZ (1989) Stretch reflex dynamics in spastic elbow flexor muscles. Ann Neurol 25:32–42
Press WH, Flannery BP, Teukolsky SA, Betterling WT (1985) Numerical recipes: the art of scientific computing. Cambridge University Press, Cambridge, UK, pp 523–528
Price R, Bjornson KF, Lehmann JF, McLaughlin JF, Hays RM (1991) Quantitative measurement of spasticity in children with cerebral palsy. Dev Med Child Neurol 33:585–595
Rack PMH, Ross RF, Thilman AF (1984) The ankle stretch reflexes in normal and spastic subjects. Brain 107:637–654
Rymer WZ, Power RK (1987) Muscular weakness in incomplete spinal cord injury. Compr Ther 13:3–7
Sehgal N, McGuire JR (1998) Beyond ashworth. Electrophysiologic quantification of spasticity. Phys Med Rehabil Clin N Am 9:949–979
Sinkjaer T, Magnussen I (1994) Passive, intrinsic and reflex-mediated stiffness in the ankle extensors of hemiparetic patients. Brain 117:355–363
Sinkjaer T, Toft E, Larsen K (1993) Non-reflex and reflex mediated ankle joint stiffness in multiple sclerosis patients with spasticity. Muscle Nerve 16:69–76
Stein RB, Kearney RE (1995) Nonlinear behavior of muscle reflexes at the human ankle joint. J Neurophysiol 73:65–72
Tardieu C, Huch de la Tour E, Bert MD, et al.et al. (1982) Muscle hypoextensibility in children with cerebral palsy. 1. Clinical and experimental observation. Arch Phys Med Rehabil 63:97–102
Thilmann AF, Fellows SJ, Garms E (1990) Pathological stretch reflex on the “good” side of hemiparetic patients. J Neurol Neurosurg Psychiatry 53:208–214
Thilmann AF, Fellows SJ, Garms E (1991) The mechanism of spastic muscle hypertonus. Variation in reflex gain over the time course of spasticity. Brain 114:233–244
Thomas CK, Zaidner EY, Calancie B, Broton JG, Bigland-Ritchie BR (1997) Muscle weakness, paralysis and atrophy after human cervical spinal cord injury. Exp Neurol 148:414–423
Thomas CK, Tucker ME, Bigland-Ritchie BR (1998) Voluntary muscle weakness and coactivation after chronic cervical spinal cord injury. J Neurotrauma 15:149–161
Toft E, Sinkjaer T, Andreassen S, Hansen HJ (1993) Stretch responses to ankle rotation in multiple sclerosis patients with spasticity. Electroencephalogr Clin Neurophysiol 89:311–318
Verrier MC, Tatton WG, Blair RDG (1984) Characteristics of EMG responses to imposed limb displacement in patients with vascular hemiplegia. Can J Neurol Sci 11:401–412
Waters RL, Adkins RH, Yakura JS, Sie I (1994) Motor and sensory recovery following incomplete tetraplegia. Arch Phys Med Rehabil 75:306–311
William PE, Goldspink G (1978) Changes in sarcomere length and physiological properties in immobilized muscle. J Anat 127: 459–468
Wolf SL, Segal RL, Catlin PA (1996) Determining consistency of elbow joint threshold angle in elbow flexor muscles with spastic hypertonia. Phys Ther 76:586–600