Neuromuscular Abnormalities Associated With Spasticity of Upper Extremity Muscles in Hemiparetic Stroke

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Mirbagheri MM, Settle K, Harvey R, Rymer WZ. Neuromuscular abnormalities associated with spasticity of upper extremity muscles in hemiparetic stroke. J Neurophysiol 98: 629–637, 2007. First published June 20, 2007; doi:10.1152/jn.00049.2007. Our objective was to assess the mechanical changes associated with spasticity in elbow muscles of chronic hemiparetic stroke survivors and to compare these changes with those recorded in the ankle muscles of a similar cohort. We first characterized elbow dynamic stiffness by applying pseudo-random binary positional perturbations to the joints at different initial angles, over the entire range of motion, with subjects relaxed. We separated this stiffness into intrinsic and reflex components using a novel parallel cascade system identification technique. In addition, for controls, we studied the nonparetic limbs of stroke survivors and limbs of age-matched healthy subjects as primary and secondary controls. We found that both reflex and intrinsic stiffnesses were significantly larger in the stroke than in the nonparetic elbow muscles, and the differences increased as the elbow was extended. Reflex stiffness increased monotonically with the elbow angle in both paretic and nonparetic sides. In contrast, the modulation of intrinsic stiffness with elbow position was different in nonparetic limbs; intrinsic stiffness decreased sharply from full- to mid-flexion in both sides, then it increased continuously with the elbow extension in the paretic side. It remained invariant in the nonparetic side. Surprisingly, reflex stiffness was larger in the nonparetic than in the normal control arm, yet intrinsic stiffness was smaller in the nonparetic arm. Finally, we compare the angular dependence of paretic elbow and ankle muscles and show that the modulation of reflex stiffness with position was strikingly different.

INTRODUCTION

Damage to descending pathways, as occurs in stroke, results in several forms of motor and/or sensory impairment (Katz and Rymer 1989; Young 1994) and typically results in spasticity, a hallmark of the upper motor neuron syndrome (Burne et al. 2005; Drolet et al. 1999; Duncan et al. 2001; Sehgal and McGuire 1998; Sommerfeld et al. 2004). A widely accepted definition of spasticity, offered by Lance, describes spasticity as a velocity-dependent joint resistance to stretch (Lance 1980). Spasticity is an important phenomenon that can lead to functional limitation, cause pain, and create secondary complications (Burne et al. 2005; Pandyan et al. 2005; Sommerfeld et al. 2004). Numerous investigators have attempted to dissect these complicated phenomena, relying primarily on clinical and/or neurophysiological approaches. Clinical approaches measure the perceived resistance of the spastic joint to passive movement but cannot separate neural from muscular mechanical components. Neurophysiological approaches study neural activities but without measuring mechanical contributions. In short, we do not know yet how to apportion the relative contributions of altered reflex mechanisms, (residing in the neuraxis), and changes in muscle mechanical properties, (residing in the periphery), to the overall impairment.

Two hypotheses have been advanced to account for the features of spasticity. One hypothesis is that the mechanical abnormalities are due to hyperactive stretch reflexes because tendon jerks and reflex electromyograms (EMGs) are increased in spasticity (Gottlieb et al. 1978; Meinders et al. 1996; Powers et al. 1989; Price et al. 1991; Rack et al. 1984; Thilmann et al. 1991). A second, alternative hypothesis is that these mechanical abnormalities result from changes in the intrinsic mechanical properties of spastic muscles and/or passive tissues (Cody et al. 1987; Dietz et al. 1991; Ibrahim et al. 1993; Sinkjaer and Magnussen 1994; Toft et al. 1993). We believe that these controversies arise mainly from a lack of accurate and sensitive tools for separating the contributing torque components. Earlier studies attempted to separate intrinsic and reflex torque and/or stiffness using electrical stimulation (Carter et al. 1990; Sinkjaer and Magnussen 1994; Toft et al. 1991) or nerve block (Allum et al. 1982; Herman and Schaumburg 1968; Hufschmidt and Mauritz 1985; Noth et al. 1984) to suppress the reflex response. Other approaches have used analytical techniques such as Nyquist analysis (Lehmann et al. 1989; Meinders et al. 1996; Price et al. 1991; Rack et al. 1984) to differentiate these components. These studies have met with limited success (Mirbagheri et al. 2000, 2001).

Thus despite intensive investigation, the nature and origin of the mechanical changes in muscle and related tissues associated with spasticity are not yet completely understood. Because spasticity can change the mechanical properties of the neuromuscular system directly and indirectly (Mirbagheri et al. 2001), we have developed and used a novel system identification technique (Kearney et al. 1997; Mirbagheri et al. 2000) to characterize joint dynamic stiffness and to separate the relative contributions of its intrinsic and reflex components. Using this novel technique, we reported that overall ankle dynamic stiffness in stroke survivors was abnormally high in the spastic limb (M. M. Mirbagheri, L. Alibiglou, M. Thajchayapang, W. Z. Rymer, unpublished data). Both intrinsic and reflex stiffness contributed strongly to net joint torque, but the contributions varied sharply with changing ankle joint angle.

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In our current study, we were interested in testing whether abnormalities in intrinsic and reflex stiffness at the elbow were similar to those recorded at the ankle, where neural control mechanisms and motor innervation patterns are systematically different.

Our findings demonstrate that both intrinsic and reflex stiffnesses contribute strongly to abnormally enhanced joint stiffness. However, these abnormalities are strongly position dependent, and this dependency was different for elbow and ankle. Reflex stiffness increased continuously with elbow extension, whereas it reached to its maximum value around neutral position in the ankle then sharply declined as the ankle was further dorsiflexed. In contrast, intrinsic stiffness showed similar behaviors in paretic elbow and ankle joints. However, the modulation of the intrinsic stiffness with the joint angle in the nonparetic sides in stroke subjects was abnormal and different in upper and lower extremities.

These findings demonstrate that the major differences between the upper and lower extremities is the position dependency of the reflex contributions, which may (in turn) be due to different levels of relative tension generated by muscle tissues at the fully extended positions.

METHODS

Subjects

Fourteen chronic, hemiparetic stroke survivors (56 ± 12.7 yr) and 15 aged-matched healthy subjects (53.5 ± 9.6 yr) were examined. The experiment was performed on both paretic and nonparetic elbow joints. Although the nonparetic limb may sometimes have minor detectable impairments (Thilmann et al. 1990), it was designated as a control for the impaired limb because it is not spastic and has similar musculo-tendon architecture and limb mass. However, to control for possible changes in the nonparetic side, we used healthy age-matched subjects as secondary or additional controls.

Stroke subjects met the following inclusion criteria: stable medical condition, absence of aphasia or significant cognitive impairment, absence of muscle tone abnormalities and motor or sensory deficits in the nonparetic arm, absence of severe muscle wasting or overt sensory deficits in the paretic upper limb, and spasticity in the involved elbow muscles for a duration of ≥1 yr.

Table 1 provides details of each stroke subject. All subjects gave informed consent to the experimental procedures, which had been reviewed and approved by Northwestern University IRB Board.

Clinical assessment

All stroke subjects were evaluated clinically using the Modified Ashworth Scale (MAS) to assess spasticity (range 1–5) (Ashworth 1964; Bohannon and Smith 1987). MAS is a conventional clinical measure of spasticity.

Apparatus

The joint actuator (motor) was operated as a position control servo driving the elbow position to follow a command input. The motor’s dynamic response and torque capabilities were much greater than those of the subject’s elbow so that the position change was essentially independent of the subject response. Subjects were seated and strapped to an adjustable, experimental chair with the forearm attached to the beam of the motor by a custom fitted fiberglass cast (Fig. 1). The seat was adjusted to provide shoulder abduction of 80° and align the elbow axis of the rotation with axis of the torque sensor and the motor shaft. An oscilloscope mounted in front of the subject displayed a command signal and provided feedback of low-pass filtered joint torque.

Recording

Elbow position and velocity were measured with a potentiometer and tachometer, respectively. Torque was recorded using a torque transducer mounted between the beam and the motor shaft. Displacements in the flexion direction were taken as negative and those in the extension direction as positive. An elbow angle of 90° was considered to be the neutral position (NP) and defined as zero. Torque was assigned a polarity consistent with the direction of the movement that it would generate (e.g., extension torque was taken as positive). EMGs were recorded from the short head of biceps, brachoradialis, and triceps long and short heads, using bipolar surface electrodes and an active preamplifier (Delsys, Boston, MA). Position, velocity, torque, and EMGs were filtered at 200 Hz to prevent aliasing, and sampled at 1 kHz by a 16 bit A/D converter.

Procedures

RANGE OF MOTION (ROM). ROM was determined with the subjects’ elbow attached to the motor and manually moved to maximum extension and flexion. Mean displacement amplitude were assessed three times by slowly moving the joint until the examiner perceives rapidly increasing resistance or the subject reports discomfort. The typical angular range was from 50° flexion to 75° extension.

PSUEDORANDOM BINARY SEQUENCE (PRBS) TRIALS. We perturbed the elbow joint by applying PRBS position inputs with amplitude of 0.03 rad and a switching-rate of 150 ms to identify the reflex and intrinsic dynamic stiffness. These perturbations contained power over a wide enough bandwidth to identify the dynamics, were tolerated by the stroke subjects and avoided attenuating reflex responses (Mirbagheri et al. 1997).

PARADIGM. Trials were performed at different elbow positions from near full flexion, i.e., −50°, to near full extension, i.e., 75°, at 25° intervals. Each position was examined under the passive condition, where subjects were instructed to remain relaxed.

During and following each trial, the torque and EMG signals were examined for evidence of nonstationarities or co-activation of elbow extensors. If there was evidence of either, the data were discarded and the trial was repeated.

<table>
<thead>
<tr>
<th>Subjects</th>
<th>Age</th>
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<th>Stroke Side</th>
<th>Ashworth</th>
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<tr>
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<td>31</td>
<td>5</td>
<td>Right</td>
<td>3</td>
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<td>2</td>
</tr>
<tr>
<td>S7</td>
<td>43</td>
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<td>1</td>
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<td>S8</td>
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<td>5</td>
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<td>3</td>
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</table>

Analysis procedures

PARALLEL CASCADE IDENTIFICATION TECHNIQUE. Reflex and intrinsic contributions to elbow dynamic stiffness were identified using a parallel cascade technique, described in detail in earlier publications (Kearney et al. 1997; Mirbagheri et al. 2000). Briefly, the method proceeded as shown in Fig. 2.

Intrinsic stiffness dynamics (top pathway) were estimated in terms of a linear impulse response function (IRF) relating position and torque. The reflex pathway (bottom pathway) was modeled as a differentiator in series with a delay, a static nonlinear element (closely resembling a half-wave rectifier), and a dynamic linear element. Reflex stiffness dynamics were estimated by determining the IRF between half-waved rectified velocity as the input and reflex torque as the output. The intrinsic and reflex stiffness IRFs were convolved with the experimental input to predict the intrinsic and reflex torque, respectively.

Linear models were fitted to the estimated intrinsic and reflex IRF curves using the Levenberg Marquardt nonlinear least-square fit algorithm (Press et al. 1985). Intrinsic stiffness was described by a second-order model having inertia, viscous, and elastic parameters. The intrinsic elastic parameter also corresponds to the steady-state, intrinsic stiffness gain.

The reflex stiffness was described by reflex delay and a third-order model having gain, damping, and frequency parameters.

STATISTICAL ANALYSIS. Standard t-test procedures were used to test for significant changes in intrinsic and reflex stiffness parameters due to subject group (e.g., paretic vs. nonparetic, paretic vs. normal, and nonparetic vs. normal). Results with \( P < 0.05 \) were considered significant.

RESULTS

Intrinsic and reflex stiffness

Our recent study shows that both intrinsic and reflex stiffnesses contribute strongly to abnormally high joint stiffness in hemiparetic stroke subjects, but their contributions varied sharply with changing ankle position (M. M. Mirbagheri, L. Alibiglou, M. Thajchayapang, W. Z. Rymer, unpublished data). Because neural control mechanisms and motor innervation patterns are different in upper and lower extremities, we explore whether similar abnormalities are present in the paretic elbow joint musculature.

Figure 3 shows the intrinsic and reflex stiffness parameters plotted against the corresponding control values for all stroke subjects and positions. The dotted line at 45° in each panel indicates what would be expected if there was no change due to stroke. Points above the line indicate abnormal increases following stroke, whereas points below the line indicate decreases.

The figure demonstrates that reflex stiffness gain and intrinsic stiffness gain, two key parameters, changed post-stroke. The reflex stiffness gain values (\( G_R \), Fig. 3A) for all subjects were located above the diagonal line, indicating that \( G_R \) was larger in the paretic than in nonparetic arms of the subjects. Similarly, the intrinsic stiffness gain (\( K \), Fig. 3B) was substantially larger for the vast majority of stroke subjects. \( G_R \) and \( K \) were the only parameters that changed consistently; they increased significantly for most stroke subjects. The other intrinsic and reflex parameters did not change significantly.

Position dependency

We investigated the position dependency of stroke effects; i.e., the differences between paretic and nonparetic sides as elbow angle was changed systematically.

Figure 4 shows group average results for reflex stiffness gain as a function of elbow position for all paretic, nonparetic, and normal groups. \( G_R \) was significantly larger in the paretic than the control elbows at most positions \( (P < 0.004) \), and the difference increased as the elbow was extended. Position dependence was similar in all groups. The reflex stiffness gain continuously increased from full flexion to full extension. The slope of changes was larger in the paretic than in the nonparetic and normal groups. The group behavior was consistent but the inter-subject variability was high at extension positions in the stroke group as demonstrated by the large SE bars associated with the means.

As expected, the nonparetic side of stroke survivors was not similar to healthy subjects; \( G_R \) was larger in the nonparetic than normal elbow, and the differences were significant at extension positions \( (P < 0.037) \).

Figure 5 summarizes the group behavior of intrinsic stiffness for all groups. The intrinsic stiffness gain was significantly larger in the paretic than in the nonparetic and normal elbows at most positions \( (P < 0.01) \). Intrinsic stiffness gain was strongly position dependent; it first decreased sharply from full- to mid-extension, then increased sharply as the elbow was
fully extended. Surprisingly, this position dependency was not consistent in all groups; in nonparetic, $K$ first decreased sharply from full- to mid-extension then decreased slowly and remained invariant.

Intrinsic stiffness gain was modulated differently with elbow angle in nonparetic and normal group and the differences were significant at extension positions ($P < 0.006$).

Comparison between upper and lower extremities

Figure 6A shows the group average of reflex stiffness gain as a function of ankle angle (Mirbagheri et al., unpublished data) in both spastic and nonparetic sides of stroke patients and in healthy subjects. The results from the paretic ankle were similar to those of the paretic elbow (see Fig. 4).

For both ankle and elbow joints, $G_R$ was significantly larger in the paretic than nonparetic and normal joints and was abnormally modulated with joint angle. However, $G_R$ increased continuously as the elbow was moved from full flexion to full extension, whereas it first increased from mid-plantarflexion to mid-dorsiflexion at the ankle and then declined as the ankle moved toward full dorsiflexion.

Nonparetic limbs showed similar abnormal behavior in both joints; $G_R$ was larger in the nonparetic than the normal joints.

Figure 6B compares the group results at the ankle joint for intrinsic stiffness gain as a function of joint angle, for all paretic, nonparetic and normal subjects (Mirbagheri et al., unpublished data).

As for the reflex results, intrinsic stiffness gain was significantly larger in paretic than nonparetic and normal ankle
The comparison of the results of this study to our recent study in lower extremities of stroke subjects (Mirbagheri et al., unpublished data) demonstrates two major points. First, the contributions of the abnormalities are the same; i.e., both intrinsic and reflex components contribute the joint torque disturbances. However, the position dependence of reflex contributions is different; i.e., the enhancement of stretch reflexes plays an important role on a wider range of motion in the elbow than in the ankle joint.

Second, although the reflex stiffness of the nonparetic side is larger for both the upper and lower extremities, the intrinsic stiffness is absolutely larger in the lower extremity but surprisingly is smaller in the upper extremity (i.e., is more compliant). These ideas will be discussed in more detail in the following sections.

Methodological considerations

ADVANTAGES OF THE PARALLEL CASCADE SYSTEM IDENTIFICATION MODEL. The technique is consistent with known physiological behavior. An advanced mathematical model was utilized to characterize dynamic joint stiffness. Under dynamic conditions, joint torque is a function of position and also of its derivatives. The relation between input position and output torque can be used to estimate dynamic joint stiffness, a key system output. Our approach provides estimates of dynamic stiffness because estimates were made for dynamic stimuli, applied at each initial joint angle. We then used a nonlinear, system identification method to characterize the dynamics of both intrinsic and reflex stiffness.

Our systems identification technique has advantages over “First Principles” anatomic/physiological approaches, which are usually mathematical simulations. This is because these approaches are strongly dependent on the selected system structure and only give correct results if the underlying structure corresponds to that assumed. In contrast, our method is not based on any structural assumption because it is a general, nonparametric technique for nonlinear system identification.

Furthermore, our technique is robust because the estimated properties for both passive/intrinsic and reflex pathway are consistent with known anatomic/physiological information. Here net joint torque is related to inertial contributions of the limb segment but especially to visco-elastic properties of muscle and joint tissues. It is the latter component that is most influenced by changes in joint angle in neurological disorders.

In the reflex pathway, the estimated reflex delay corresponds closely to that expected for the monosynaptic Ia pathway (Hugon 1974; Toft et al. 1991), the form of the static nonlinearity (a half-wave rectifier) is consistent with the unidirectional rate sensitivity of the stretch reflex (Kearney and Hunter 1983), the velocity sensitivity of the reflex pathway is consistent with the dynamic response of muscle spindle receptors (Matthews 1972; Poppele and Bowman 1970; Stein 1980), and finally the low-pass dynamics are consistent with muscle activation dynamics (Genadry et al. 1988; Mannard and Stein 1973).

Approach allows simultaneous separation of joint dynamic stiffness components. Intrinsic and reflex torques always appear and change together; it is thus difficult to distinguish the mechanical consequences of reflex activity from those of the intrinsic properties of the joint.
Our parallel-cascade system identification approach characterizes the intrinsic and passive and reflex contributions to overall joint stiffness simultaneously from a single experimental trial. This represents a major advantage over studies that estimate reflex and mechanical contributions separately, such as those comparing mechanical properties before and after eliminating reflexes by electrical stimulation (Carter et al. 1990; Sinkjaer and Magnussen 1994; Toft et al. 1991) or nerve block (Allum et al. 1982; Herman and Schaumberg 1968; Hufschmidt and Mauritz 1985; Lehmann et al. 1989; Noth et al. 1984). Furthermore, these approaches are lengthy and potentially inaccurate because of changes in intrinsic properties associated with elimination of the reflex responses (Davies 1982; Heyters et al. 1994; Noth et al. 1984). In addition, blocking methods cannot completely suppress the reflex response, can be painful and can elicit spasms and co-contraction (Allum et al. 1982). These deficits may result in either over- or under-estimation of intrinsic and/or reflex contributions to overall joint stiffness.

LIMITATIONS. As described, our measurements focus on small amplitude perturbations because they offer an economical way to characterize mechanical and reflex properties swiftly and accurately. Our correlations with Ashworth scores are poor, although we do accept the assessment that the subjects are spastic as determined by clinical examination. It is likely that the clinical and quantitative tests are measuring somewhat different features of the reflex response, although we do examine rigorously the effects of joint angle changes on neuromuscular properties over the full range of motion. Our central thesis, however, is that the relative contributions of reflex and intrinsic mechanisms are subtle and hard to separate without the help of advanced system identification methods.

Abnormalities in neuromuscular properties of spastic extremity

ENHANCED REFLEX STIFFNESS. Our results demonstrate that reflex stiffness gain was substantially larger in stroke than normal subjects at most positions, in some cases, by as much as a factor of seven (Fig. 4). The differences were increased with elbow extension where reflex stiffness was largest for the stroke group. The reflex enhancement was consistent with our ankle data in both stroke (Mirbagheri et al., unpublished data) and SCI (Mirbagheri et al. 2001, 2002) populations although the position dependence was different between upper and lower extremities. The differences were greatest mid-ROM for the ankle (Fig. 6A). This was consistent with increased path length, attributed to enhanced reflex stiffness, by Meinder et al. (1996).

The decline in reflex stiffness at full ankle dorsiflexion (Fig. 6A) could be induced by an increase in tension in muscle and passive tissues of the ankle extensors (Fig. 6B). This could, in turn, result in increased discharge of the group III/IV muscle afferents, which have potent inhibitory effects on reflex stiffness (Schmit et al. 2002). In contrast, the intrinsic stiffness of the elbow showed a relatively smaller increase as the elbow was extended over the range of motion, so that the tension in elbow flexors of spastic elbow did not reach comparable levels.

The enhancement in reflex stiffness gain could be due to different mechanisms. First, this increase could arise from a decrease in reflex threshold as suggested earlier by our group (Powers et al. 1989). One way this could happen is by increased synaptic excitation from descending brain stem pathways, including vestibulo- and reticulospinal pathways. One intriguing possibility is that brain stem monoaminergic pathway activity is increased, inducing changes in serotonin or norepinephrine levels in the spinal cord (Heckman et al. 2003). This mechanism could augment intrinsic motoneuronal voltage-gated conductance, resulting in heightened motoneuron excitability. Furthermore, these changes could be mediated, at least partly, in a bilateral fashion, because the spinal distribution of such pathways is strongly bilateral (Heckman et al. 2003).

Alternatively, the augmented reflex gain could be due to inappropriate recruitment of larger motoneurons in the paretic limb, consistent with our observation in SCI subjects (Mirbagheri et al. 2001). This idea has been supported by others suggesting that CNS reorganizes its neuronal connections following lesions (Carr et al. 1993) by developing new connections including sprouting and strengthening of existing connections to optimize its performance (Carr et al. 1993). Another possible mechanism is increased stretch-evoked synaptic excitation of motoneurons that can arise from γ-motoneuron hyperactivity that increases muscle spindle sensitivity (Prochazka 1989; Taylor et al. 2000) and an increase in sensitivity of excitatory interneurons to muscle afferents. This high sensitivity could arise from increased afferent terminal sprouting, increased postsynaptic receptor hypersensitivity, and reduced presynaptic inhibition (Burke and Ashby 1972; Burke and Lance 1973; Burke et al. 1971; Iles and Roberts 1986; Katz and Rymer 1989; Stein et al. 1995; Young and Shafani 1980). However, a reduction in presynaptic inhibition has been particularly observed in spasticity after spinal cord lesions (Calancie et al. 1993; Knikou 2005). Surprisingly, a reduction in presynaptic inhibition has been reported only for the upper extremity of hemiplegic patients with a unilateral spasticity (Aymard et al. 2000).

INCREASED INTRINSIC STIFFNESS. Our findings demonstrate that the intrinsic stiffness is larger in the paretic than in the normal arms under passive conditions, when subjects are relaxed (Fig. 5). This finding is consistent with findings from our ankle data showing that the intrinsic stiffness was higher in the paretic ankle than in normal groups (Fig. 6B). Although intrinsic stiffness was increased at all angles in both paretic upper and lower extremities, the amount of the change was different at different joint angles. The discrepancies increased with elbow extension and with ankle dorsiflexion, indicating strong position dependency.

Sinkjaer and Magnussen (1994) also reported enhancement in passive stiffness of the ankle joint in stroke subjects. In contrast, Galiana et al. (2005) found no significant differences in the ankle intrinsic stiffness between these two groups because they studied a limited range of positions from mid-plantarflexion to NP, where intrinsic stiffness of the paretic limb is normal (Figs. 5 and 6B). Furthermore, they studied sub-acute patients who had a much shorter time post-lesion as compared with our chronic stroke subjects and, consequently, may not have a substantial muscle fiber remodeling in their spastic limbs.

J Neurophysiol  •  VOL 98  •  AUGUST 2007  •  www.jn.org
This increase of intrinsic stiffness may be due to a shift of the passive length-tension curve to the left, caused, in turn, by lesion-induced changes in structural cellular (Foran et al. 2005; Friden and Lieber 2003) and mechanical properties of spastic muscles and of connective tissues (Booth et al. 2001; Lieber et al. 2004; Marbini et al. 2002; Romanini et al. 1989; Tabary et al. 1976; Trotter and Purslow 1992).

An increase in the intrinsic stiffness can also result from joint contractures, which result from a combination of changes to the properties of the musculo-tendinous unit, the surrounding connective tissues, and the joint itself (Singer et al. 2001; Tabary et al. 1976; Williams and Goldspink 1978). Joint contractures may arise from two mechanisms—immobility and prolonged muscle shortening. This combination of immobilization and shortened positioning may cause several consequences: muscle atrophy, decreasing the number of muscle fibers and strength; muscle fiber shortening, losing sarcomeres and decreasing the range of motion; and an increase in the ratio of collagen to muscle fiber tissue, reducing the muscle compliance (Gracies et al. 1997; Lieber et al. 2004; O’Dwyer et al. 1996).

We assessed potential elbow contractures in our stroke population by recording the passive torque at each initial joint torque angle, prior to applying the perturbation sequence. Using these measurements, the static stiffness of the paretic elbow was derived and shown to be greater than that of either normal controls, or of the nonparetic elbow (−3.5 vs. −0.25 Nm/rad). However, this stiffness value was still well below the intrinsic dynamic stiffness in the paretic joint (20–45 Nm/rad), so the passive stiffness (a measure of contracture) would have contributed little to net joint stiffness.

Abnormalities in ipsilateral extremity (nonparetic versus normal)

Although the tonic stretch reflex is basically absent in clinical assessment of normal subjects, we did report reflex responses for this group, but they were minimal in size as compared with paretic or even nonparetic limbs. The difference lies presumably because the peak velocities are higher in our protocol than in the usual clinical tests of muscle tone. Furthermore, even normal subjects will show reflex responses in relaxed muscles if stretches are sufficiently fast.

Our results showed that reflex stiffness was significantly larger in the nonparetic limbs of the stroke patients than in the normal limbs of healthy subjects. This was the case for both elbow and ankle joints. Furthermore, in the nonparetic ankle, intrinsic stiffness was also slightly larger than in normal controls, but modulated similarly with changes in joint angle. Surprisingly intrinsic stiffness was smaller in the nonparetic elbow (than in normal controls) and modulated differently with increasing elbow angle.

These findings demonstrate that the nonparetic side of the hemiparetic stroke survivor is not normal, consistent with Thilmann et al. (1990), who studied reflex EMG responses to muscle stretch in normals as well as in paretic and nonparetic elbow muscles of stroke survivors. These authors argue that the nonparetic limbs may not be an ideal control for the study of neuromuscular mechanical abnormalities in spastic-paretic muscles, and we would be inclined to agree. We show, in addition, that there are differences evident in both mechanical and reflex components of the response to perturbations.

What physiological mechanisms might explain these changes? Regarding the hyperexcitability of stretch reflexes in the nonparetic limbs, as described earlier, one solution is that brain stem monoaminergic pathways (serotonin and norepinephrine) are bilaterally distributed, and their activity may be enhanced in stroke (Heckman et al. 2003). Alternatively, uncrossed corticospinal fibers may show increased activity, altering the excitability of ipsilateral motoneurons (Martinez et al. 1995; Yanosh et al. 2004).

Regarding reduced intrinsic stiffness of the nonparetic elbow (or increased intrinsic compliance), one possible explanation is that a parallel decrease in descending drive to the nonparetic extremity, following a stroke, promotes some degree of neuromuscular atrophy and an increase in tissue mechanical compliance. This decrease in neural drive is exemplified in the strength deficits in the uninvolved extremity reported in several studies (Colebatch and Gandevia 1989; McCrea et al. 2003; Watkins et al. 1984).

In contrast to the elbow joint, intrinsic stiffness of the ankle joint was slightly larger in nonparetic, as compared with healthy normal limbs (Fig. 6B), perhaps because of consistent increases in limb load in asymmetrical hemiplegic gait, giving rise to some degree of muscle hypertrophy in the nonparetic limb. Such increased loads can presumably be avoided in the upper extremity, where limb loading is not mandatory.

Clinical significance

Our results show that overall dynamic stiffness was significantly larger in stroke than nonparetic subjects due to increases in both intrinsic and reflex stiffness. The relative contributions of these two were strongly dependent on position, and this dependency was different in upper and lower extremity. Thus questions about the nature and origin of spasticity may have quite different answers depending on where in the range of motion and in which extremity tests are made. This can explain some controversies in the literature regarding the nature and origins of mechanical abnormalities associated with spasticity.

The relative contributions of intrinsic and reflex mechanisms and how they change with position are not readily distinguishable by clinical examination of muscle tone. These examinations are carried out by moving the joint through much of its range of motion and by estimating the resistance (Ashworth 1964; Lance 1980; McCrea et al. 2003). This may at least partly explain why we did not find any significant correlation between the modified Ashworth scale and our findings in spastic subjects (Mirbagheri et al. 2001), implying that this clinical evaluation does not measure joint dynamic stiffness and its intrinsic and reflex components.

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