Spasticity: Quantitative Measurements as a Basis for Assessing Effectiveness of Therapeutic Intervention

Justus F. Lehmann, MD, Robert Price, MSME, Barbara J. deLateur, MD, Steven Hinderer, MD, Charles Traynor, BS

ABSTRACT. Lehmann JF, Price R, de Lateur BJ, Hinderer S, Traynor C: Spasticity: Quantitative measurements as basis for assessing effectiveness of therapeutic intervention. Arch Phys Med Rehabil 70:6-15, 1989.

• Spasticity, a common problem in upper motor neuron lesions, frequently results in uncontrolled involuntary motion that interferes with function. A quantifiable method related to the mechanical output of the muscle is needed to test and improve therapeutic intervention. A sinusoidal displacement of 5° was used to measure elastic and viscous stiffness around the ankle at frequencies from 3 to 12Hz. To isolate viscoelastic response, the influences of inertia and equipment drag were eliminated. Test-retest correlation values were 0.953 for elastic and 0.992 for viscous stiffness. The elastic stiffness in 13 spastic subjects under nerve block was significantly higher than that of 13 healthy subjects ($p \leq 0.05$), indicating early changes associated with contracture. Elastic and viscous response is expressed by the total stiffness vector containing both components, the Nyquist diagram. This diagram's pathlength from 3 to 12Hz was calculated and showed high test-retest reliability in healthy subjects. The median pathlength value for the spastic group was 98 Newton-meters/radian (N-m/rad) and, for the normal group, 24N-m/rad, a statistically significant difference ($p \leq 0.0001$). A mathematical model of the spastic response shows that the Nyquist diagram's pathlength relates to reflex loop gain and is independent of the shift in passive properties. The model predicts a shift in passive properties during spastic responses relative to responses measured during nerve block. Thus, subtraction of passive responses measured during nerve block may not isolate the remaining reflex response, but the pathlength measure relating to the reflex response gain was unaffected, allowing evaluation of therapeutic intervention effectiveness.

KEY WORDS: Monosynaptic reflex; Muscle hypertonia; Muscle spasticity; Muscle tonus; Stretch reflex

Spasticity in traumatic brain injury (TBI), stroke, and spinal cord injury (SCI) often interferes with function, limits independence, and may produce secondary complications such as contractures.[9] This is a common problem accompanying a variety of upper motor neuron lesions. Young's group[45] found that 21% of spinal cord injured persons experienced involuntary, uncontrolled movements.

Spasticity is classically defined as a rate-dependent reflex hypertonia triggered by stretch of the spindle. It is often combined with other motor deficits which in TBI, for instance, include rigidity, ataxia, and flexor and extensor synergies. When these coexist with spasticity, they may substantially increase motor control impairment. Imbalance of muscle tone may lead to secondary disabling contractures, eg, equinus deformity of the foot. Spasticity that interferes with function may involve the total spectrum of disability, from upper motor neuron diseases or trauma.[9] In order to study the effectiveness of therapeutic intervention, it is essential to first have an objective, quantifiable method of measuring spasticity.

Therapeutic approaches include drug therapy, nerve and motor point blocks, electric stimulation, exercise programs, and cryotherapy. Most evaluations of these modalities have been based on qualitative or semiquantitative clinical observations, such as tendon tap response, clonus, pendulum swing of a limb, and the Ashworth scale.[3] Clinical observations on drug therapy and other therapeutic modalities suggest that available therapy is of only limited effectiveness, with a trade-off in side effects.[7,8,10,12,14-16,21,25-28,30,35,40,41,43] These findings underline the importance of quantitative, objective methods of evaluation.

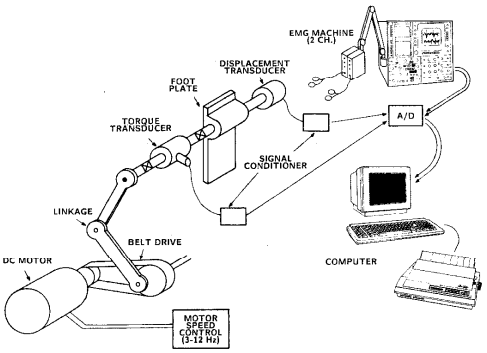


Fig 1—Spasticity measurement system.

Electrodiagnostic and mechanical measurement techniques have been used in an attempt to quantify spasticity. All electromyographic (EMG) measurements have been limited to local, circumscribed sampling of the potentials generated by the muscle, and may therefore not be representative of the action

From the Department of Rehabilitation Medicine, University of Washington, Seattle.

Submitted for publication December 1, 1987. Accepted in revised form July 15, 1988.

This study was supported in part by research grant G00830076 from the National Institute on Disability and Rehabilitation Research, Department of Education, Washington, DC 20202.

No commercial party having a direct or indirect interest in the subject matter of this article has conferred or will confer a benefit upon the authors or upon any organization with which the authors are associated.

Reprint requests to Dr. Lehmann, Department of Rehabilitation Medicine, RJ-30, University of Washington School of Medicine, Seattle, WA 98195

of the total muscle. Other tests, such as the H to M ratios, bypass the spindle. Clearly, interference with controlled muscle function stems directly from the mechanical output of the muscle and relates only indirectly to the EMG.

The torque produced by the mechanical result of the reflex response has been used as a measure of spasticity. However, torque as a measure of resistance to imposed movement is the result not only of the reflex response, but also of the passive properties of the tissues.[5,20,22,33]

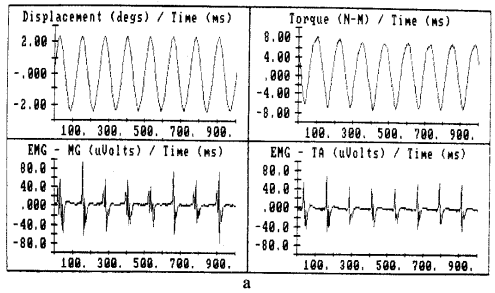
The mechanical stimulus used to elicit the reflex response has been the deformation of the spindle. The most commonly used mechanical stimuli fall into two categories. One attempts to maintain a constant rate of stretch through a "ramp" waveform (ie, an acceleration to constant velocity, and then a deceleration to zero velocity).[19,20,22,29,34] The other uses a sinusoidal displacement. If a ramp waveform is used, it is difficult to produce a stimulus in a comparable fashion at different speeds. The angular range of a joint over which a constant velocity can be achieved is reduced at increasing speeds because there is a finite range of motion available and a finite amount of time required to accelerate and decelerate the limb.

The length at which the muscle is stretched with constant velocity also changes with speed. Hence, the angular range over which the velocity is constant varies, as does the muscle length at which constant rate of stretch begins; these differences influence the reflex response. In addition, the stimulus which excites the reflex response may vary with the duration and magnitude of acceleration to constant speed, the duration and magnitude of the constant speed, and the duration and magnitude of the deceleration. Since all of these stimuli vary with speed, different responses may occur not solely as a result of the speed held constant, but also as a result of the variability of the other stimulus parameters.

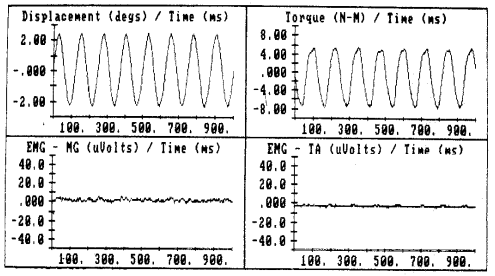
The alternative method is to use sinusoidally oscillating motion imposed upon the joint at different frequencies. Sinusoidal oscillation, both in terms of torque[17] and displacement[5,13,36-38] has been used. Imposing sinusoidal oscillation produces more readily repeatable and controllable stimulus parameters than the ramp method. In order to actually isolate the torque due to reflex response, the following have to be considered: (a) the inertia of the limb[13,38]; (b) the inertia and drag of the measurement system; and (c) the contribution of the passive properties of the tissues.

Researchers have described not only the response in normal subjects to ramp stretch or to sinusoidal variation in torque or displacement, but also responses in patients with various pathologies.[6,17,19,22,34,36,37] In an attempt to assess the contribution of the passive properties of the tissues to the total measured responses, Brown and Associates[5] produced a maximal tetanic contraction of the muscle, a situation where the reflex response could no longer occur. Under these circumstances, they assessed the viscoelastic properties of the tissues. They assumed that these passive properties would not change with the degree of contraction, so that the values they obtained could be applied to different responses using different frequencies.

Undermining this assumption, Rack's group[38] showed some evidence suggesting changes in the passive properties with different levels of voluntary contraction force. Noth and colleagues[33] produced an ischemic block to study the resistance to a superimposed sinusoidal motion of the index finger.



a



b

Fig 2—Computer recorded displacement, torque, and EMG output in spastic subject recorded at 8Hz (a) without nerve blocks; (b) with tibial-peroneal nerve blocks. EMG-MG is the electromyographic activity, in microvolts, of the gastrocnemius muscle. EMG-TA is the electromyographic activity of the tibialis anterior. Positive displacement is dorsiflexion; positive torque is plantar flexion torque generated by the subjects' feet.

After ten minutes, the reflex response seemed to disappear; however, after 20 minutes there was a marked change in the measured properties of the passive tissues.

Hufschmidt and Mauritz,[22] using ramp stretches to the gastrocnemius and soleus, showed that 25 minutes were necessary for a satisfactory ischemic nerve block. In the light of Noth's results, it is questionable whether this method can be used to measure responses in the lower extremity without distorting the measured values. Herman and Schaumberg[20] showed that the difference in the length-tension diagram of the calf at a low rate of stretch was the same with and without a peripheral nerve block. In neither condition was there any EMG activity. It is questionable whether the values thus obtained as a measure of the passive properties of the tissues can be applied to high rates of stretch. It is known, for instance, that the viscous properties are speed dependent.[44] In 1974, Herman and associates[19] suggested lidocaine peripheral nerve blocks to isolate the passive properties of the tissues.

Many of the studies used a sustained voluntary contraction at a level of torque that facilitates the reflex response.[1,13,23,24,36,38] However, not everyone has the ability to produce a steadily maintained voluntary contraction. Voluntary tension maintained during the displacement produces an increase in nonreflex passive resistance and torque.[38] The quantitative relationship between degree of facilitation, in-

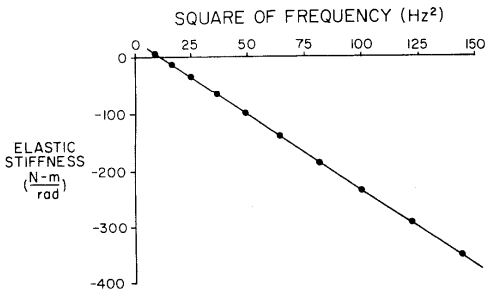


Fig 3—Influence of footplate and foot inertia on elastic stiffness. Linear plot of the elastic stiffness component vs frequency squared. Healthy limb in footplate.

crease in reflex response, and change in passive properties is not known.

All authors who investigated both persons with pathologic conditions and healthy volunteers noted that their reflex responses were different.[6,9,17,18,20,22,34,36,37,39] No comparisons were made with sinusoidal stimulation. Of those who described the effects of ramp stretches at the ankle, only Otis and colleagues[34] and Hufschmidt and Mauritz[22] showed that there was a statistically significant difference between responses of healthy and disabled subjects. Otis and colleagues[34] measured torque at different levels of constant velocity and found that the values differed significantly from the normal values. The disabled group contained 24 paraplegics and five hemiplegics. In the hemiplegics, the unaffected leg was used as a control. Hufschmidt[22] studied an elastic and a velocity-independent plastic resistance (friction) at low angular velocities. In most patients with long-standing spasticity, both of these properties were enhanced.

Mathematical models of the behavior of the passive response of the tissues have been developed by several authors.[23,24,38] Rack's group[38] attempted to mathematically describe the passive and reflex response to varying frequencies of oscillation. These authors developed a simplified model of the tissues and neurologic properties of the ankle. They used data from the literature on tissue properties and reflex response to describe the behavior of the system as exhibited in their experimentation.

From a review of the literature, it was apparent that a system was needed to allow quantitative assessment of spasticity and of the passive properties of the tissues, with adequate reliability and power of discrimination between normal and abnormal responses. Existing mathematical models should be modified to account for and describe experimental results and to be used as an aid to their interpretation.

METHODS

The apparatus used in the study of spasticity is an electromechanical device for imposing sinusoidal rotary movements of varying frequencies (3 to 12Hz) upon the ankle through movement of the foot. The subject's foot is held in place with an adjustable binding attached to an oscillating footplate driven by an electric motor via a crank-linkage mechanism. The crank mechanism maintains the oscillation at ±2.5° about the neutral (90°) ankle position. Some small variation (±0.2°) in this angular range occurs with changing frequency due to compliance of the shaft, linkage, and belt drive system.

Interposed between the crank mechanism and the footplate is a strain gauge torque transducer. A capacitive angular displacement transducer measures footplate angles. A digital motor controller regulates the 1/2hp dc motor's speed precisely to enable testing to be performed at a fixed, prescribed frequency of oscillation, with a maximal controller error of ±0.4%.

Data acquisition was performed by an IBM-PC XT-based computer system sampling at a rate of 500Hz via an analog-to-digital converter.

EMG data obtained with surface electrodes from the gastrocnemius-soleus and tibialis anterior muscles were recorded by a TECA TE4 with two channels and hard copy output. The data were also sampled by the computer system for qualitative purposes. A schematic representation of the complete spasticity measurement system is presented in figure 1.

Subjects

Subjects were selected on the basis of having at least a 5° range of motion around the neutral position of the ankle, with no history of ankle joint pathology. The absence or presence and degree of spasticity was assessed clinically by judging the briskness of tendon tap reflex, presence of clonus, and resistance to passive motion using the five-point Ashworth scale.[3] Spastic subjects not taking any antispasticity medication were selected from a population with upper motor neuron lesions resulting from spinal cord injuries (eight subjects) or traumatic brain injuries (five subjects). Of 26 spastic subjects and healthy controls, 22 were 19 to 27 years of age.

Procedure

Subjects were positioned prone, with the knee straight, on a padded plinth. The footplate and binding were adjusted to ensure a snug fit and optimal alignment of the ankle's axis of rotation with the footplate shaft. All subjects were instructed to relax. Fixed frequencies of oscillation were applied in a random sequence at integral frequencies ranging from 3 to 12Hz. Three trials were performed at each of the ten frequencies for a total of 30 trials per session. Each trial consisted of

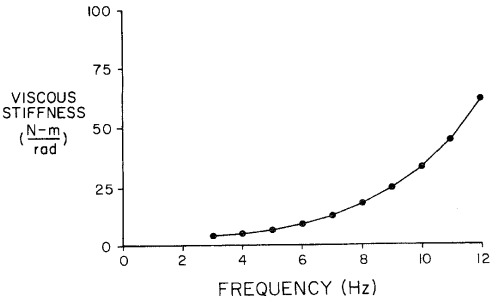


Fig 4—Equipment drag loss vs frequencies; expressed as viscous stiffness of empty footplate.

a 15-second speed stabilization phase and a one-second data sampling phase.

Lidocaine was used to effect peroneal and tibial nerve blocks in the spastic group in order to allow assessment of the passive elastic and viscous stiffness of the tissues after eliminating the reflex response. This technique has been described in earlier publications.[31,32] The block was also used in order to calculate the inertial properties of the foot without interference from the reflex response. Figure 2 shows a typical record of displacement, torque, and EMG vs time in a spastic subject before and after nerve block.

Data Analysis

Raw data from the displacement and torque transducers were subjected to a Fourier analysis in order to decompose the signals into their sinusoidal components. The fundamental frequency, corresponding to the footplate oscillation frequency, was retained. All other frequency components were disregarded in the subsequent analysis.

Since the elastic and viscous responses of the ankle were of primary interest, the effects of inertia and equipment drag were eliminated. To compute the effect of combined footplate and foot inertia, linear regression analysis of the torque component 180° out of phase with the displacement, vs frequency squared, was performed (fig 3). The 180° phase shift and frequency squared relationship occur because the torque induced from inertial effects is proportional to the angular acceleration imposed. Assuming the displacement is described by a sinusoid of frequency ω and amplitude A, the acceleration, α (the second time derivative of displacement) is given by:

$$\begin{aligned}\alpha &= d^2 [A \sin (\omega t)]/dt^2 \\ &= -A\omega^2 \sin (\omega t) \\ &= A\omega^2 \sin (\omega t + 180°)\end{aligned}$$

This calculation assumes that the only contribution to this second order effect (frequency squared) was from inertia. To ensure that the reflex response in spastic subjects did not interfere with the computation of this inertial component, data collected during temporary tibial and peroneal nerve blocks were used.

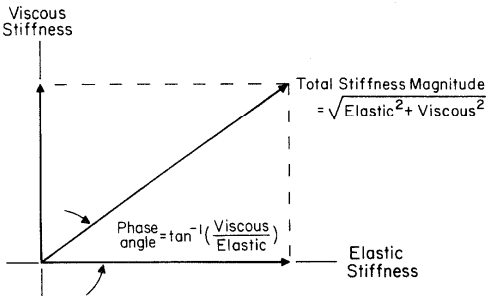


Fig 5—Stiffness components of the Nyquist diagram for a given frequency. The vector sum of the elastic and viscous stiffness components is equivalent to the total stiffness vector. The arc tangent of the ratio of the viscous to the elastic stiffness is the phase angle.

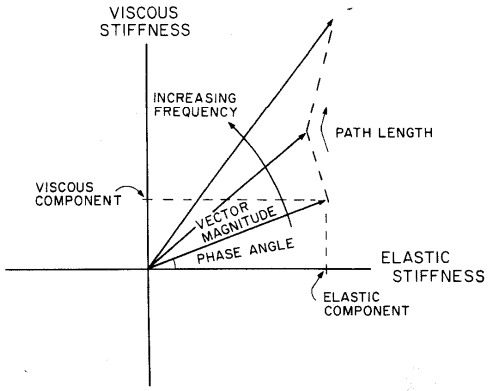


Fig 6—Nyquist diagram of stiffness, its components, and a demonstration of pathlength generation.

Drag was determined by running the equipment without the subject's foot in place. The torque component 90° out of phase with the displacement was determined for each frequency and subtracted from the data. (Figure 4 shows a typical response.) Drag or energy dissipating effects are a function of velocity. Velocity, in turn, is the first derivative of displacement, and for a sinusoidally time-varying displacement, the angular velocity, Ω, is given by:

$$\begin{aligned}\Omega &= d[A \sin (\omega t)]/dt \\ &= A\omega \cos (\omega t) \\ &= A\omega \sin (\omega t - 90°)\end{aligned}$$

Gravitational effects have been neglected due to their relatively minor role. Since the center of gravity is close to the axis of rotation and the displacements small, the variation in torque due to gravity is negligible. In addition, the difference in the gravitational component due to differences in foot mass and center of gravity location is even smaller due to the relatively large contributions of the fixed mass mechanical components. Therefore, comparisons between persons of differing foot mass and configuration can be made without correcting for gravity.

The response of the ankle to sinusoidal displacements was expressed in terms of net stiffness (with inertial and drag stiffness eliminated), and represents the ratio of net torque to displacement. The stiffness is composed of two quantities: that which is in-phase with the displacement (elastic stiffness) and that which is 90° out-of-phase with the displacement (viscous stiffness). The elastic stiffness can be visualized as being due to a torsional spring. The force produced by the spring is proportional to the angular displacement and the constant of proportionality is the elastic stiffness (for a linear system). Similarly, the viscous stiffness can be visualized as being due to a damper whose damping torque is proportional to velocity, the viscosity being the constant of proportionality for a linear system.

The stiffness can be visualized as having a total magnitude at some phase angle with respect to the displacement. The vector sum of the elastic and viscous stiffness components is equivalent to the total stiffness. The arc tangent of the ratio of the viscous to the elastic stiffness is the phase angle (fig 5). These values are expressed as a function of frequency in the form of a Nyquist diagram of angular stiffness.[33] In an effort to express the frequency response over the 3 to 12Hz range by a single value, a pathlength calculation was performed using a straight line approximation method (fig 6) for the stiffness vector, moving sequentially from frequency to frequency.

RESULTS

The inertial response produced a linear plot of elastic stiffness vs frequency squared. Correlation coefficients were 99% or better for the linear plot (fig 3).

An attempt was made to estimate the effect of gravitational forces on the resulting measurements. The footplate was moved through the spectrum of frequencies without a load. For comparison, a prosthetic foot similar in weight to the anatomic foot was then inserted. The elastic and viscous stiffnesses were then determined. The maximal difference in the elastic stiffness was 1.2 Newton-meter/radian (N-m/rad). The maximal difference in the viscous stiffness was 3.6N-m/rad.

To test the accuracy and frequency response of the equipment to elastic loading, two torsion springs of different stiffness were inserted to resist footplate movement. The stiffness was plotted against the frequencies used and closely approached the ideal response, ie, a line at the statically determined stiffness (fig 7). Due to the lack of a suitable viscous standard, no corresponding test for accuracy of viscous measurement was made.

A second set of experiments was performed to assess variability in a group of healthy subjects. Initially, the variability of repeated measurements was assessed. The foot was placed

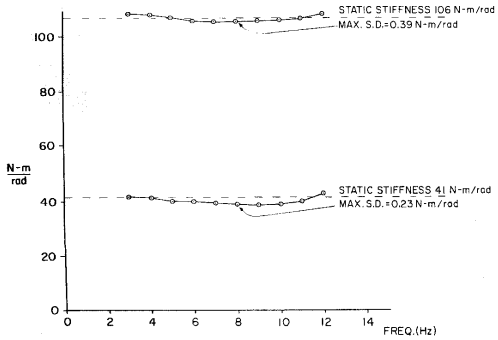


Fig 7—Results of elastic stiffness measurements of torsional springs at frequencies from 3 to 12Hz; recorded for springs of stiffness of 106 and 41N-m/rad. The standard deviation is reported for the point at which it was maximum.

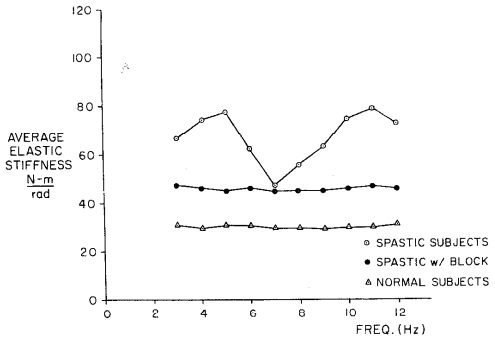


Fig 8—Average elastic stiffness vs frequency for 13 healthy and 13 spastic subjects.

in the binding and three consecutive measurements of the elastic and viscous properties were made at 11Hz. The foot was then taken out of the binding and the process was repeated ten times. In order to eliminate the variability introduced by placing the foot in the binding, the pooled estimate of the standard error of the mean was calculated. For this calculation, the standard error of the mean was determined for each of the three measurements that was obtained for each of the ten placements of the foot in the binding. The values of the pooled estimate for elastic stiffness and for viscous stiffness were 0.21N-m/rad and 0.20N-m/rad, respectively.

In the protocol for each measurement, the foot may be repeatedly placed into the binding and taken out; also, the three measurements for each frequency during one foot placement are averaged, and this average is used as one data point. From the ten foot placement repetitions for one healthy subject at 11Hz, the standard deviation and the standard error of the mean were calculated and found to be 1.46 and 0.47N-m/rad for elastic, and 1.53 and 0.48N-m/rad for viscous. Compared with the previous experiment, the values increased slightly in variability due to the placement, removal, and replacement of the foot in the binding.

The variability of the measurement from person to person was assessed by one single measurement, averaging three trials of elastic and viscous properties at 11Hz for 13 healthy subjects. The standard deviation and standard error of the mean for the elastic properties were 6.85 and 1.87N-m/rad, respectively, and, for the viscous, 10.89 and 3.03N-m/rad, respectively. This variability is small compared with the average magnitude of the actually measured values in normal and spastic persons (figs 8 and 9).

The test-retest reliability was determined for eight healthy subjects at 11Hz. For each subject, two measurements were made within less than one hour and then compared. As a measure of reliability, the Cronbach alpha test was used.[11] The standardized alpha value was 0.953 for elastic stiffness and 0.992 for viscous stiffness.

In order to estimate long-term variability in healthy subjects, five measurements of the elastic and viscous properties at 11Hz were performed with five normal subjects over a period of two

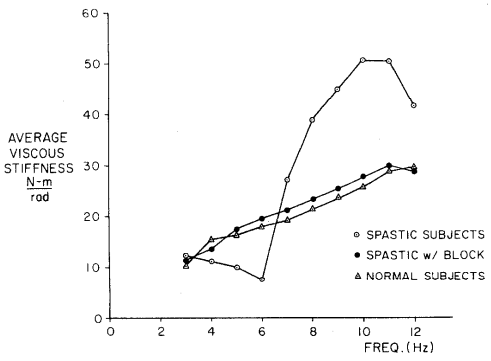


Fig 9—Average viscous stiffness vs frequency for 13 healthy and 13 spastic subjects.

and a half weeks. To eliminate person-to-person variability, the pooled estimate of the standard error of the mean was obtained and found to be 0.8N-m/rad and 1.5N-m/rad for elastic and viscous stiffness, respectively.

In the following experiment, the methodology was tested for its ability to discriminate between normal and spastic behavior.

The mean elastic stiffness in the spastic group was compared with the mean elastic stiffness in the normal group for all frequencies (fig 8). Each group contained 13 persons. The values were compared statistically for each frequency. All but the values at 7Hz were significant at the $p \leq 0.05$ level. The same comparison was made for the viscous stiffness (fig 9). The statistical difference for each frequency was significant at the $p \leq 0.05$ level except at frequencies of 3 to 7Hz. Both the elastic and viscous properties of the spastic group showed a major decrement in value around the mid-range frequencies (6 and 7Hz), a change which Rack's group[36] suggested was due to the timing of the reflex response in relationship to the plate movement in such a way that the reflex response may assist the plate movement. This leads to an overlap of the distribution of the values for the group of spastic subjects with the values obtained in healthy subjects.

The EMG response produced by the stretch reflex was identified by recording the EMG potentials phase related to the footplate movement. In the spastic group, the EMG responses in the gastrocnemius ranged from 51% of all trials at 3Hz to over 90% at frequencies of 10, 11, and 12Hz. The response was clearly frequency dependent. For healthy subjects the response was much less prominent, but also frequency dependent. Three percent of all trials showed an EMG response at 3Hz, with a maximum percentage of 26% at 11Hz (fig 10).

The passive properties of the tissues were studied in spastic subjects after tibial and peroneal nerve blocks were performed to eliminate the spastic reflex response. These responses were compared with the responses of healthy subjects. The results (fig 8) show that there is a significant ($p \leq 0.05$ level) difference between the elastic properties of the passive tissues in the spastic group compared with those of the healthy group. There is no corresponding significant difference between the values of passive viscous stiffness throughout all frequencies (fig 9).

As illustrated in figure 6, the pathlength of the Nyquist diagram was calculated in eight healthy subjects for test and retest. The test-retest reliability was determined using the standardized alpha, yielding a value of 0.907.

The average Nyquist diagram of stiffness at the ankle for the spastic group, with and without nerve blocks, is shown in figure 11. The pathlength values of the Nyquist diagrams were calculated for the spastic group (with and without nerve blocks), and for the healthy group at all frequencies. The responses of 13 spastic and 13 healthy subjects were compared. The results are presented in table 1. The difference between spastic persons' and healthy persons' pathlength is significant at $p \leq 0.0001$, using the Mann-Whitney U test. There was no statistically significant difference between pathlength of the healthy group and the pathlength of the Nyquist diagrams of the passive properties produced by nerve blocks in the spastic group.

Finally, the long-term variability of spasticity measured by the pathlength of the Nyquist diagram was measured and compared with variability in the healthy group. The measurements were taken twice per week over a period of two and a half weeks for a total of five repeat measurements per person, with five persons in each group. The purpose was to obtain a measure of the waxing and waning of muscle tone over a long period of time. In order to eliminate variability from person to person, a pooled estimate of the standard error of the mean was calculated. For this calculation, the standard error of the mean of the five values for each person was obtained. The pooled estimate of the standard error of the mean was 12.7N-m/rad and 2.7N-m/rad for spastic and normal subjects, respectively.

DISCUSSION

The approach to the measurement of spasticity required an elimination of the influence of drag, inertia, and forces of gravity on the operating system in order to isolate viscoelastic properties. Drag and inertia were eliminated for each individual in the study. The influence of the gravitational forces was estimated by measuring elastic and viscous stiffness while the empty footplate vibrated and while it vibrated carrying a prosthetic foot. Results showed that the maximal difference was 1.2 and 3.6N-m/rad for elastic and viscous stiffness, respectively. This difference is negligible, since the two loads—nothing or a prosthetic foot—represent opposite extremes of

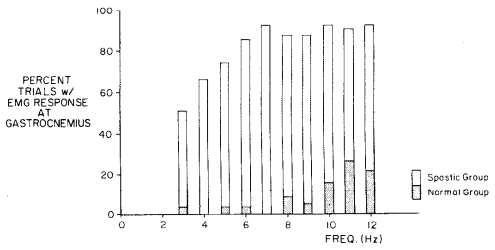


Fig 10—EMG responses in healthy and spastic persons vs frequency.

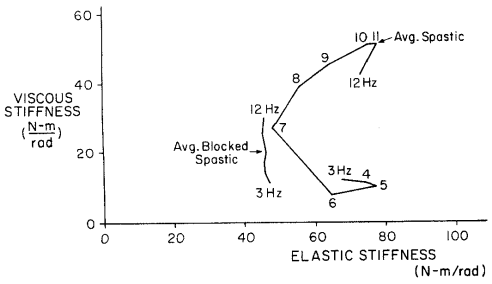


Fig 11—The Nyquist diagram of average stiffness for the spastic group with and without nerve blocks.

possible loads to be carried. The value of the difference is also small compared with the total measurement of viscoelastic properties in both healthy and spastic persons.

Except for a single case, there was no frequency dependence of elastic stiffness using relatively small excursions at the ankle. This was verified by a linear regression analysis which on average produced correlation coefficients of -0.24 for the spastic group with nerve blocks and -0.13 for the healthy group (fig 8). The properties of the viscous stiffness of the spastic group with nerve blocks and the healthy group show a linear increase in viscous stiffness with increasing frequency. This is also verified by linear regression analysis, which yielded a slope of 2.1N-m-sec/rad, an intercept of 6.2N-m/rad, and a correlation coefficient of 0.98 for the blocked spastic group; and a slope of 2.0 N-m-sec/rad, an intercept of 5.8N-m/rad, and a correlation coefficient of 0.99 for the healthy group. These observations are also consistent with a linear viscoelastic model proposed by Hunter and Kearney[23,24] for constant displacement amplitude and mean torque.

Testing the adequacy of measurement by using two elastic springs with different stiffness as a load working against the footplate showed good agreement with the ideal static stiffness of the springs. It also showed that the maximal standard deviations for elastic stiffness at any frequency from 3 to 12Hz within three repeat measurements was small (0.39N-m/rad) (fig 7).

Table 1: Pathlength Values for Spastic, Blocked Spastic, and Healthy Groups from 3 to 12Hz

	Pathlength (N-m/rad)		
Subject	Spastic	Blocked spastic	Healthy
1	512	146	21
2	73	21	13
3	84	33	35
4	109	46	14
5	113	28	25
6	281	17	24
7	60	45	20
8	28	16	18
9	59	18	36
10	515	49	18
11	30	22	37
12	98	35	38
13	173	36	51
Median	98	33	24

Variability of the measurements was minimal for repeat measures at 11Hz on each subject. It increased slightly when the foot was placed in and out of the binding. An additional increase of variability occurred from person to person. However, all of these values are relatively small compared with normal stiffness values, and are minor when compared with values in spastic persons, suggesting the usefulness of this measurement system. Correspondingly, the test-retest reliability was very high, with a value for a Cronbach alpha test of 0.953 for elastic stiffness and 0.992 for viscous stiffness. Finally, the long-term changes in muscle tone in healthy subjects over two and a half weeks were small.

The above analyses of variability were done at the higher frequency of 11Hz because of the overlap of the healthy group's distribution with that of the spastic group at the mid-range frequencies. The assessment of the passive properties of the spastic group did not show an overlap. It showed a clear increase in the elastic stiffness at all frequencies over the healthy comparison group ($p \leq 0.05$), probably indicating changes associated with developing contractures (fig 8). The finding of a difference in elastic stiffness is consistent with previous observations by Hufschmidt[22] and Stolov.[42] The frequency-dependent viscous component of the blocked spastic group was not significantly different compared to that of the healthy group. Hufschmidt and Mauritz[22] could not identify the frequency dependence very well because of the low angular velocities used. However, working with low frequencies, they could determine the frequency independent loss component (ie, plastic resistance), and found some difference.

Thus, it is possible that the change in passive properties may significantly influence the measurement of the spastic response without nerve blocks. Attempts have been made to eliminate the passive factor using nerve blocks and subtracting the passive values from the total response without blocks.[5,20,22,33]

Figure 12 shows a model incorporating the linear viscoelastic properties of muscle and tendon. It includes a reflex loop with significant latency. The active components of the model, ie, the reflex loop, influence the behavior of the model through five parameters: gain, latency, phase shift, natural frequency, and damping ratio.[4] The active component is mathematically represented by a second order, low pass system function. The contribution of the active component is exaggerated in spasticity. A similar model has been used by Rack.[38] The measured passive properties of the spastic group with nerve blocks were used in the model as values for muscle stiffness, viscosity, and friction (table 2). As an estimate, it was assumed that the tendon stiffness in the spastic group was 20 times greater than the stiffness of the muscle. This corresponds to an Achilles tendon with a cross-sectional area of 70mm^2, a length of 300mm,[38] a modulus of 1200N/mm^2,[2] and a computed moment arm of 59mm with respect to the ankle. Muscle stiffness was based on the average linear regression intercept value for elastic stiffness in the 13 persons with peripheral nerve blocks, which represents the series stiffness of muscle and tendon. Muscle viscosity and friction were based on slope and intercept of the average linear regression values of viscous stiffness in the same blocked group.

Since the model is based on linear elements, and since the

analysis methods used to determine the parameters are only strictly valid for a linear system, the model's results may not be valid for other amplitudes and frequencies because the biologic system may not be linear. As such, these parameters would not necessarily apply to the very low frequencies used by Hufschmidt and Mauritz.[22] The purpose of this modeling exercise is not to precisely describe the human ankle response; the model is a great simplification. However, it may aid in understanding the research results.

Using the model (fig 13), the Nyquist diagram of the spastic response ("Model Spastic") was calculated based on the measured passive responses of the spastic group. If the calculated Nyquist diagram is compared with that actually measured in our group of spastic persons ("Measured Spastic Response"), the calculated diagram appears shifted downward and to the left. However, if it can be assumed that the passive properties of the muscle, while the reflex contractions do occur, are different from those under the nerve block condition ("Shifted Passive Properties"), and one recalculates the diagram using the model with increased values for elasticity and friction (table 2), the calculated diagram ("Model Response with Shifted Passive Properties") is shifted into the same position as the experimentally obtained curve ("Measured Spastic Response"). Data published by Rack[38] showing a shift of the position of the Nyquist diagram with changing voluntary muscle force at the ankle may also be interpreted as a result of a change in passive properties due to static muscle contraction. These findings suggest that subtracting passive properties under nerve block conditions from the unblocked spastic response is not adequate to describe the reflex component of spasticity.

The calculated pathlength of the Nyquist diagram has not been changed as a result of using different values for the passive properties (fig 13). Instead, its pathlength expresses the gain of the output through the reflex response, which is independent of the possible change of passive properties during reflex contraction.

This diagram, or its pathlength, seems to be a better quantitative measure of spasticity than subtraction of the passive properties from the total spastic response. That the pathlength of the model is highly sensitive to the gain of the reflex loop is illustrated in fig 14, which is based on using the mathematical model with variation in the gain only.

The fact that the pathlength measure, using all frequencies, discriminates well between the spastic and healthy groups ($p \leq 0.001$) supports the usefulness of this approach and its application to the quantitative study of the effectiveness of therapeutic intervention. The usefulness of this test is also supported by the test-retest reliability of testing healthy persons, which shows a standardized Cronbach alpha of 0.907.

Unlike the statistically significant difference observed between the elastic stiffnesses of the healthy and blocked spastic group, there was no statistically significant difference in the pathlengths of the Nyquist diagrams. This apparent discrepancy can be explained because the pathlength incorporates the frequency dependence of viscous properties, which is the same in both groups. Furthermore, the elastic properties are essentially frequency independent and thus do not contribute to the pathlength. Hence, the pathlengths of these two groups are equivalent.

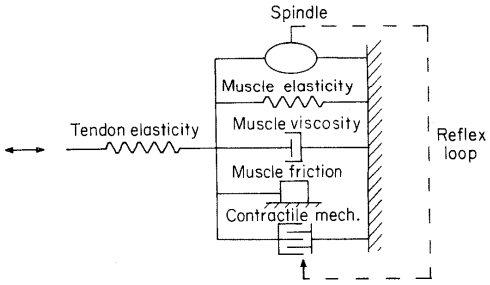


Fig 12—Model basic to viscoelastic properties of muscle and tendon.

CONCLUSION

A quantifiable method was developed to measure degree of spasticity by determining the resistance to a sinusoidal mode of excitation at the ankle. The measured mechanical output was displayed in terms of the total (elastic and viscous) stiffness vector at frequencies from 3 to 12Hz. Based on a mathematical model for the reflex response, the pathlength of the Nyquist diagram represented a measure of the gain of the spastic response. Using the model, it could also be demonstrated that the pathlength of the Nyquist diagram was independent of a change in passive properties of the system. In order to match the location of the actually measured Nyquist diagram in the spastic group, the model suggested that a shift of passive properties had occurred as compared with those properties obtained in the spastic group under nerve block conditions. This finding is consistent with references[38] suggesting that voluntarily sustained contractions change passive viscoelastic properties of the muscle.

The pathlength of the Nyquist diagram discriminated well between abnormal spastic responses and the responses of a healthy group ($p \leq 0.0001$). Also, there was a high degree of test-retest reliability in tests of healthy subjects with a reliability of the correlation coefficient of 0.907. The clinically

Table 2: Values Used With Computer Model

Condition	Muscle stiffness (N-m/rad)	Muscle friction (N-m/rad)	Gain	Delay (msec)
Passive response of spastic group with nerve block*	48.3	6.2	—	—
Spastic response calculated using passive response of spastic group with nerve block**	48.3	6.2	1.1	85
Shifted passive properties*	68.5	23.3	—	—
To fit actually observed spastic response curve**	68.5	23.3	1.1	85

* Tendon stiffness = 966N-m/rad; muscle viscosity = 2.1N-m-sec/rad.
** Tendon stiffness = 966N-m/rad; muscle viscosity = 2.1N-m-sec/rad; phase shift = 0°; natural frequency = 2Hz; damping ratio = 1.

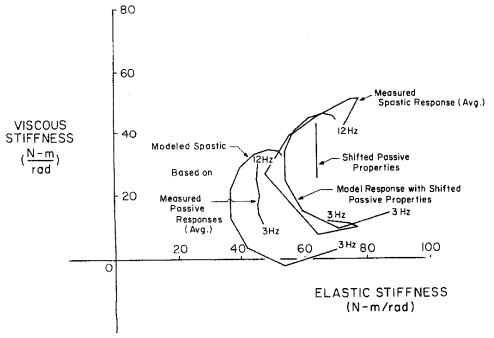


Fig 13—Modeled and experimentally observed Nyquist diagrams of ankle stiffness in the spastic group. If the modeled spastic Nyquist diagram using the passive responses under nerve block is compared with the measured spastic responses, it is shifted down and to the left. However, good agreement of modeled and observed responses can be obtained by shifting the passive properties, based on the assumption that such a shift occurs under muscle contraction. The shift of the passive properties does not change the pathlength of the Nyquist diagram.

observed waxing and waning of spasticity could also be verified by a long-term measurement of the pathlength of the Nyquist diagram in a spastic group, a variability which did not exist in the healthy group.

Comparison of elastic and viscous stiffness in the normal and spastic groups showed that statistical significance was obtained only at higher frequencies. Lower frequencies, especially in the measurement of viscous stiffness, showed an overlap of the distribution due to the timing of the reflex response in relationship to the movement of the footplate, which was assisted by the reflex responses at these frequencies. The other limitation of this comparison is the difficulty of eliminating the contribution of the stiffness due to the passive properties of the tissues to the overall spastic response. Obtaining the passive properties of the tissues in the spastic group under the condition of dual nerve blocks (peroneal and tibial) allowed the demonstration of early signs of contracture to manifest

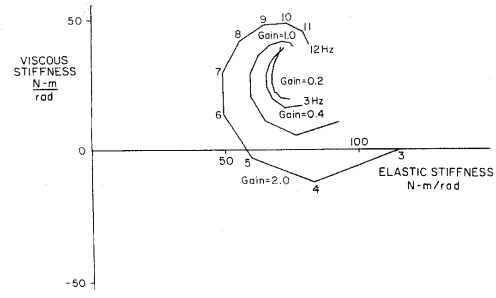


Fig 14—Influence of gain variation on pathlength of Nyquist diagram of spasticity model.

themselves as increased elastic stiffness. Because there is no way of measuring any change in the passive properties due to reflex contraction, it is impossible to isolate the reflex response through subtraction of passive stiffness data. However, since the model indicates that the pathlength of the Nyquist diagram is independent of passive property changes, the early development of contractures or passive property changes associated with reflex contraction has no influence on the quantification of spasticity using the pathlength.

At a high frequency (11Hz), the test-retest reliability in the healthy group was very high for elastic and viscous stiffness (0.953 and 0.992, respectively). The variability of repeat measurements for the same person and for measurements from person to person was very small, much smaller than comparable values measured in normal and spastic persons. The equipment performance was carefully tested and it was possible to eliminate from the raw data the influence of inertia and drag, and it could be demonstrated that the contribution of gravitational forces was minimal. Testing with different spring loads verified the accuracy of the instrumentation. In conclusion, the method described seems to fulfill the requirement for a quantifiable measurement of the mechanical output as it is changed by spasticity, independent of factors which cannot be controlled.

References

1. Agarwal GC, Gottlieb GL: Oscillation of the human ankle joint in response to applied sinusoidal torque on the foot. J Physiol **268**:151–176, 1977
2. Alexander RMcN: Mechanics of skeleton and tendons. *In* Brooks BB (ed): Handbook of Physiology (Section I, The Nervous System; Volume II, Motor Control). American Physiological Society, 1959
3. Ashworth B: Preliminary trial of carisoprodol in multiple sclerosis. Practitioner **192**:540–542, 1964
4. Bawa P, Stein RB: Frequency response of human soleus muscle. J Neurophysiol **39**:788–793, 1976
5. Brown TIH, Rack PMH, Ross HF: Forces generated at the thumb interphalangeal joint during imposed sinusoidal movements. J Physiol **332**:69–85, 1982
6. Burke D, Andrews CJ, Gillies JD: The reflex response to sinusoidal stretching in spastic man. Brain **94**:455–470, 1971
7. Cartlidge NEF, Hudgson P, Weightman D: A comparison of baclofen and diazepam in the treatment of spasticity. J Neurol Sci **23**:117–134, 1974
8. Chyatte SB, Basmajian JV: Dantrolene sodium: long-term effects in severe spasticity. Arch Phys Med Rehabil **54**:311–315, 1973
9. Corcos DM, Gottlieb GL, Penn RD, Myklebust B, Agarwal GC: Movement deficits caused by hyperexcitable stretch reflexes in spastic humans. Brain **109**:1043–1058, 1986
10. Corson RN, Johnson F, Godwin-Austen RB: The assessment of drug treatment in spastic gait. J Neurol Neurosurg Psychiatry **44**:1035–1039, 1981
11. Cronbach LJ: Coefficient alpha and the internal structure of tests. Psychometrika **16**:297–334, 1951
12. Duncan GW, Shahani BT, Young RR: An evaluation of baclofen treatment for certain symptoms in patients with spinal cord lesions. Neurol **26**:441–446, 1976
13. Evans CM, Fellows SJ, Rack PMH, Ross HF, Walters DKW: Response of the normal human ankle joint to imposed sinusoidal

movements. J Physiol **344:**483–502, 1983
14. Fowlks EW, Strickland DA, Peirson GA: Control of spastic states in neurological patients with diazepam. Am J Phys Med **44:**9–19, 1965
15. From A, Heltberg A: Double-blind trial with baclofen (Lioresal) and diazepam in spasticity due to multiple sclerosis. Acta Neurol Scand **51:**158–166, 1975
16. Gaillard JM: Comparison of two muscle relaxant drugs on human sleep: diazepam and parachlorophenylgaba. Acta Psychiat Belg **77:**410–425, 1977
17. Gottlieb GL, Agarwal GC, Penn R: Sinusoidal oscillation of the ankle as a means of evaluating the spastic patient. J Neurol Neurosurg Psychiat **41:**32–39, 1978
18. Hagbarth K-E, Wallin G, Löfstedt L: Muscle spindle responses to stretch in normal and spastic subjects. Scand J Rehabil Med **5:**156–159, 1973
19. Herman R, Freedman W, Mayer N: Neurophysiologic mechanisms of hemiplegic and paraplegic spasticity: implications for therapy. Arch Phys Med Rehabil **55:**338–343, 1974
20. Herman R, Schaumberg H: Alterations in dynamic and static properties of the stretch reflex in patients with spastic hemiplegia. Arch Phys Med Rehabil **49:**199–203, 1968
21. Hudgson P, Weightman D: Baclofen in treatment of spasticity. Br Med J **4:**15–17, 1971
22. Hufschmidt A, Mauritz K-H: Chronic transformation of muscle in spasticity: a peripheral contribution to increased tone. J Neurol Neurosurg Psychiat **48:**676–685, 1985
23. Hunter IW, Kearney RE: Dynamics of human ankle stiffness: variation with mean ankle torque. J Biomech **15:**747–752, 1982
24. Hunter IW, Kearney RE: Invariance of ankle dynamic stiffness during fatiguing muscle contractions. J Biomech **16:**985–991, 1983
25. Jebsen RH, Taylor N, Trieschmann RB, Trotter MJ, Howard CA: An objective and standardized test of hand function. Arch Phys Med Rehabil **50:**311–319, 1969
26. Jebsen RH, Trieschmann RB, Mikulic MA, Hartley RB, McMillan JA, Snook ME: Measurement of time in a standardized test of patient mobility. Arch Phys Med Rehabil **51:**170–175, 1970
27. Jones RF, Burke D, Marosszeky JE, Gillies JD: A new agent for the control of spasticity. J Neurol Neurosurg Psychiat **33:**464–468, 1970
28. Joynt RL, Leonard JA Jr: Dantrolene sodium suspension in treatment of spastic cerebral palsy. Dev Med Child Neurol **22:**755–767, 1980
29. Kearney RE, Chan CWY: Contrasts between the reflex responses of tibialis anterior and triceps surae to sudden ankle rotation in normal human subjects. Electroencephalogr Clin Neurophysiol **54:**301–310, 1982
30. Ketel WB, Kolb ME: Long-term treatment with dantrolene sodium of stroke patients with spasticity limiting the return of function. Curr Med Res Opin **9:**161–169, 1984
31. Lehmann JF, Condon SM, de Lateur BJ, Price R: Gait abnormalities in peroneal nerve paralysis and their corrections by orthoses: a biomechanical study. Arch Phys Med Rehabil **67:**380–386, 1986
32. Lehmann JF, Condon SM, de Lateur BJ, Smith JC: Gait abnormalities in tibial nerve paralysis: a biomechanical study. Arch Phys Med Rehabil **66:**80–85, 1985
33. Noth J, Matthews HR, Friedemann H-H: Long latency reflex force of human finger muscles in response to imposed sinusoidal movements. Exp Brain Res **55:**317–324, 1984
34. Otis JC, Root L, Pamilla JR, Kroll MA: Biomechanical measurement of spastic plantarflexors. Dev Med Child Neurol **25:**60–66, 1983
35. Pedersen E, Arlien-Soborg OP, Grynderup V, Henriksen O: GABA derivative in spasticity. Acta Neurol Scand **46:**257–266, 1970
36. Rack PMH, Ross HF, Thilmann AF: The ankle stretch reflexes in normal and spastic subjects. Brain **107:**637–654, 1984
37. Rack PMH, Ross HF: The role of reflexes in the resting tremor of Parkinson's disease. Brain **109:**115–141, 1986
38. Rack PMH, Ross HF, Thilmann AF, Walters DKW: Reflex responses at the human ankle: the importance of tendon compliance. J Physiol **344:**503–524, 1983
39. Rebersek S, Stefanovska A, Vodovnik L, Gros N: Some properties of spastic ankle joint muscles in hemiplegia. Med Biol Eng Comput **24:**19–26, 1986
40. Rousson M, Terrence C, Fromm G: Baclofen versus diazepam for the treatment of spasticity and long-term follow-up of baclofen therapy. Pharmatherapeutica **4:**278–284, 1985
41. Schmidt RT, Lee RH, Spehlmann R: Comparison of dantrolene sodium and diazepam in the treatment of spasticity. J Neurol Neurosurg Psychiatry **39:**350–356, 1976
42. Stolov WC: The concept of normal muscle tone, hypotonia and hypertonia. Arch Phys Med Rehabil **47:**156–168, 1966
43. Taylor N, Sand PL, Jebsen RH: Evaluation of hand function in children. Arch Phys Med Rehabil **54:**129–135, 1973
44. Woledge RC, Curtin NA, Homsher E: Energetic Aspects of Muscle Contraction. Academic Press, 1985, page 83
45. Young JS, Burns PE, Bowen AM, McCutchen R: Spinal Cord Injury Statistics. Phoenix, Good Samaritan Medical Center, 1982