Quantitative Comparison of Passive Motion and Tendon Reflex Responses in Biceps and Triceps Brachii Muscles in Hemiplegic or Hemiparetic Man

BY DAVID G. SIMONS, M.D.* AND A. G. A. BINGEL, M.D.†

Abstract: Quantitative Comparison of Passive Motion and Tendon Reflex Responses in Biceps and Triceps Brachii Muscles in Hemiplegic or Hemiparetic Man

The clinical impression that severe hyperreflexia is not consistently associated with severe spasticity in hemiplegia or hemiparesis due to vascular pathology was tested by objective measurement in 11 patients. The intensity of tendon reflex responses of the triceps and biceps brachii muscles was measured as the reflex sensitivity ratio (evoked twitch tension/stimulating hammer force). The degree of spasticity was measured as the threshold rate of elbow flexion or extension above which a myotatic response was elicited, determined by both a sudden increase in passive resistance and a burst of antagonist electromyographical activity.

Rate thresholds were ranked ordered for flexion data and for extension data with their corresponding reflex sensitivity ratios. Numerous individual discrepancies stood out. Correlation coefficients between rate thresholds and reflex sensitivities determined at two elbow angles were —0.52 and —0.35 during extension, and —0.47 and 0.03 during flexion.

Differences in rate of stretch, differences in the sensory structures stimulated, and differences in the distortion of the organization of motor control may account for the difference in responses to the two tests.

A number of authors have considered exaggerated contraction of muscle subjected to passive stretch an essential component to spasticity in hemiplegic or hemiparetic patients.1–4 Among clinical neurologists, increased ease of elicitation, irradiation, and clonic repetition of tendon reflexes with Babinski’s sign are accepted criteria of spasticity.5 These criteria strongly emphasize hyperreflexia. Both features, reaction to passive stretch and hyperreflexia, are separately measurable.

Creed et al.8 sharply distinguish in cats the “static reaction” of the stretch reflex in response to elongation of the muscle contrasted to the “phasic reaction” of the stretch reflex in response to the tendon tap. They attributed this difference in response to the marked difference in the synchronization of the afferent nerve impulses to the spinal cord. There also may be significant differences as to which afferent end organs are responding and their type of response. This “static reaction” of the stretch reflex response can be measured as a rate threshold. The rate is expressed as the angular velocity of elbow flexion or extension in...
<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Side hemi</th>
<th>Age</th>
<th>Time insult to test, wks.</th>
<th>Strength*</th>
<th>Ankle clonus hemi side</th>
<th>Babinski hemi side</th>
<th>Hemiplegic upper extremity sensornism</th>
<th>Spasticity</th>
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<tbody>
<tr>
<td>1</td>
<td>R</td>
<td>76</td>
<td>5</td>
<td>4</td>
<td>4</td>
<td>+</td>
<td>normal ?†</td>
<td>Flexor spasm at rest‡</td>
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<tr>
<td>2</td>
<td>L</td>
<td>41</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>+</td>
<td>Marked resistance at moderate rates, flex. &amp; ext. slight during extension</td>
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<td>11</td>
<td>0</td>
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<td>hypo, pain &amp; touch</td>
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<tr>
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<td>L</td>
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<td>+</td>
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</tr>
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<td>+</td>
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<td>65</td>
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<td>+</td>
<td>hyper?†</td>
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<td>R</td>
<td>45</td>
<td>15</td>
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<td>L</td>
<td>55</td>
<td>16</td>
<td>6</td>
<td>5</td>
<td>0</td>
<td>+</td>
<td>slight hypo, touch</td>
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<tr>
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<td>+</td>
<td>normal ?†</td>
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<td>L</td>
<td>51</td>
<td>20</td>
<td>5</td>
<td>4</td>
<td>+</td>
<td>+</td>
<td>hypo, pain</td>
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<tr>
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<td>R</td>
<td>58</td>
<td>37</td>
<td>5</td>
<td>5</td>
<td>8</td>
<td>8</td>
<td>hyper, pain</td>
</tr>
</tbody>
</table>

*For scale, see text.
†Patient responses not considered reliable.
‡Evaluation by P.T.
§Data missing.
The threshold is the rate at which stretch reflex responses are observed in response to activation of the elbow joint. This response is typified by, but not restricted to, the clasp-knife phenomenon. The "phasic reaction" may be measured as the force of the muscular twitch response to tendon tap. The rate threshold is concerned with the presence or absence of an evoked myotatic response when tested at various rates of passive arm movement. The tendon reflex response is concerned with the amplitude of response to a constant tendon tap force. This rate of stretch produced by the blow of the reflex hammer on the tendon is unphysiologically rapid compared to the functional rates of stretch produced with passive arm movements.

Clinical experience with hemiplegic or hemiparetic patients supports the distinction drawn by Creed et al. Many spastic patients show both "static" and "phasic" reactions of the stretch reflex. Some patients show predominantly one, other patients predominantly the other. This paper examines both factors quantitatively in the same patients.

Methods
The 11 patients in this study were referred from the neurology and the physical medicine and rehabilitation wards of the Houston VA Hospital. Each patient had been diagnosed as suffering from hemiplegia due to either right or left intracranial vascular pathology. The average age of the group was 57 years, ranging from 41 to 76 years. Table 1 presents specific details of the clinical condition of these patients observed within two weeks of the time that they were tested in the laboratory. The strength scale was rated:

0 = no muscular contraction; 1 = muscular contraction without perceptible force; 2 = movement with maximal assistance; 3 = movement with minimal assistance; 4 = movement without assistance; 5 = movement against partial manual resistance; and 6 = movement against full manual resistance.

The passive motion (PM) test established a rate threshold from measurements of arm angle, biceps and triceps electromyogram (EMG) and the force resisting passive motion (fig. 1). To measure resistance to passive motion, a bicycle wheel was mounted horizontally on a table and fitted with a splint to receive the patient's forearm for testing. The table permitted adjustment of the height of the wheel to suit the patient. The plastic splint transmitted all rotational forces through a strain gauge tensiometer to the wheel. This recorded any resistive force to passive motion exerted by the patient's arm. A wooden handle permitted manual rotation of the wheel. A hollow pad over the axle of the wheel received the medial epicondial to assure centering of the elbow joint over the axis of the wheel. Stops adjustable every 1.6° insured a positive mechanical limit to the range of motion (ROM) during the passive motion tests. Limit switches which were actuated by the stops automatically initiated and terminated timing of each PM test within 5 mm of the mechanical limit.

The force of the resistance to passive motion was measured with a Statham Instruments, Inc., Gold Cell UC 3 fitted with a UL 50 lb force transducing head, and a Statham UR 5 amplifier which was calibrated to full scale readings of ±2.5 or occasionally ±5.0 kg if necessary.

The goniometer used a Bourns 10,000 ohm single-turn potentiometer which had conductive plastic elements with ±0.5% linearity and a ball-bearing supported shaft. This potentiometer was the attachment for two thin aluminum arms each of which fastened onto the patient's arm with velcro in two places above and below the elbow. Elbow extension approached the 180° reading, and elbow flexion approached zero degree. One arm of the goniometer aligned with the ulna and the other with the humerus. The wrist was in the neutral position halfway between pronation and supination.

To measure EMG three Beckman biopotential electrodes were connected to an amplifier developed in this laboratory. It provided a fixed gain of 1,000 and an input impedance of 100 megohms. Its frequency response was flat between 50 and 1,000 Hz, which is fully adequate to measure surface electrodes interference pattern EMG.

Forearm angle values were recorded on the X-axis of the XY plotter and force values on the Y-axis. In addition, all three measures were
Instrumentation arrangement for measurement of tendon reflex responses. The reflex hammer held in the operator's hand is fitted with a strain gauge sensor to record the impact force. The other strain gauge sensor measures the response twitch tension against the wheel, which is locked in position for this test. The three electrodes are used to record biceps EMG and are connected to the preamplifier strapped on the subject's chest.

The tendon reflex (TR) test (fig. 2) established the amplitude of twitch tension in response to tendon tap. It used a clinical reflex hammer that was adapted with a Statham Gold Cell No. UC 3. The gold cell was fitted with a UL 50 lb transducer head which was interposed between the rubber hammer head and the handle to measure the stimulus force. This measured the relative decelerative force developed as a result of impact over the region of the tendon. This stimulus signal was amplified by another Statham UR 5 amplifier and recorded on the oscillograph using a full scale range of 0 to 20 kg. The twitch response tension was measured with the tensiometer mounted on the wheel.

**PROCEDURE**

The patient was instrumented with the goniometer and electrodes while seated in his wheel chair with his arm extended at right angles from his side and with his elbow placed over the axis of the wheel. The handrest on the wheel held his forearm in the neutral position midway between flexion and extension as it is positioned clinically for reflex testing. By attaching the goniometer directly to the patient's arm rather than measuring the angle of the wheel, the measurement of elbow angle was independent of shoulder motion and thus required no stabilization of the torso. The EMG electrodes were applied to the skin over the palpably most prominent portion of the muscle belly with the arm in position on the wheel and with the elbow at 90°. The positioning of the patient is illustrated in figures 1 and 2.

The stops to limit the range of motion during testing of resistance to passive motion were set by slowly moving the arm until limited by mechanical obstruction, rapidly increasing force due to compression of biceps tissue, or pain. The mechanical pins were then inserted locking the stops in position. The initial baseline test was then conducted at an average rate of approximately 5°/sec going from the flexion stop to the extension stop, holding five seconds, and then returning to the flexion stop. If no myotatic reflex response was observed in the record it established the baseline XY plot at a subthreshold rate. The appearance of a myotatic response in subsequent tests established a rate as being above threshold. It was defined as a burst of EMG activity concurrently associated with a sudden increase in resistance to passive motion. A subthreshold rate was a rate at which this myotatic response was not observed. If a myotatic response was observed, the cycle was repeated at a slower rate until a subthreshold value was obtained. Test cycles of increasing angular velocity were then executed in ten, five, two and one seconds corresponding to 10, 20, 50 and 100°/sec tests. A clean sheet of graph paper that was printed on a translucent base was placed in the plotter for each test. All three measures were recorded on the oscillograph which was operated at a speed to make the goniometer trace move across the record at approximately a 45° angle.

Tendon reflex responses were measured by first locking the wheel so that the tensiometer mounted on it would record twitch tension in response to tendon tap. The biceps were first tested with the arm at 90° and then at 45° (which was halfway between 90° and full flexion). A minimum of ten taps were applied to the index finger placed over the biceps tendon at each arm angle. The mean hammer force for all subjects at 45° was 0.79 kg and at 90° was 0.67 kg. The triceps TR values were measured first at 90° and then at 135° by tapping the triceps tendon at the elbow. The mean hammer tap forces were 0.60 kg at 90° and 0.74 kg at 135°. The amplitude of the hammer force and tension responses were recorded on the Brush oscillograph.

The analyses of the XY plots were made by laying the higher rate records over the baseline records using a lighted x-ray viewbox. In this way any sudden increase in resistive force due to a myotatic reflex response became clearly appar-
A passive stretch reflex response was validated by looking for the appropriate burst of EMG activity on the oscillograph record at the corresponding arm angle. The rate was measured as the average rate of angular motion as determined by the computer from the goniometer and limit switches. The threshold value was defined as the lowest average rate of elbow motion which elicited a passive stretch reflex response. This applied whether the threshold-subthreshold rate values overlapped or not.

The deep tendon reflex responses were measured as the amplitude of muscular twitch tension response as distinguished from mechanical oscillation of the system due to the hammer blow. To correct for the expected variations in the force of the manually applied sequential hammer taps, the results were expressed as the reflex sensitivity ratio (twitch response tension in kg divided by the stimulating hammer force in kg).

**Results**

Patients have been measured with the described method (a) for their threshold rates of resistance to passive elbow flexion and (b) for their threshold rates of resistance to passive elbow extension, each of which is compared to the response to (c) triceps tendon tap and (d) biceps tendon tap.

Figure 3 shows an example of the results obtained on the XY plotter for the resistance to passive motion test. This plot illustrates a baseline record and a positive myotatic response. The corresponding oscillograph record (fig. 4) illustrates a flexion suprathreshold stretch reflex response.

The data obtained during extension (biceps spasticity) testing for threshold rate were rank ordered according to threshold rate in table 2. This table also presents the corresponding reflex sensitivity ratios for both 45° and 90° of elbow angle.

One would expect a high-rate threshold to be associated with a low amplitude of TR myotatic response. Two notable exceptions to this expectation are seen in table 2 (biceps stretch). Patient number 3 (showing the fourth highest rate threshold of 11 patients) was clearly above average in amplitude of TR response. Conversely, patient number 9, who showed the fourth from lowest threshold rate, had next to the lowest TR response amplitude. The scattergram for these data (fig. 5A) indicates no clearly defined relationship. A regression line is included for both the 45° and 90° elbow angle positions. It assumes that whatever relation is present is linear. The correlation coefficient, r, for biceps reflex response values versus threshold rate was −0.52 at 45° and −0.35 at 90°. Both are negative, but weak correlations which are far from −1.0.
TABLE 2
Biceps Spasticity and Hyperreflexia Measured as Twitch Tension Response on Hemiplegic Side

<table>
<thead>
<tr>
<th>Rank order, patient no.</th>
<th>Threshold rate, extension, °/sec</th>
<th>Ratio of biceps tens./hammer force at 45°</th>
<th>Ratio of biceps tens./hammer force at 90°</th>
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<tr>
<td>4</td>
<td>255</td>
<td>0.23</td>
<td>0.24</td>
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<td>11</td>
<td>136</td>
<td>0.06</td>
<td>0.03</td>
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<td>8</td>
<td>118</td>
<td>0.18</td>
<td>0.78</td>
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<td>3</td>
<td>48</td>
<td>0.50</td>
<td>0.24</td>
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<tr>
<td>1</td>
<td>43</td>
<td>0.20</td>
<td>0.09</td>
</tr>
<tr>
<td>2</td>
<td>25</td>
<td>*</td>
<td>0.54</td>
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<tr>
<td>5</td>
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<td>1.0</td>
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<tr>
<td>7</td>
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<td>1.0</td>
<td>2.0</td>
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<tr>
<td>10</td>
<td>2.2</td>
<td>0.60</td>
<td>0.35</td>
</tr>
<tr>
<td>6</td>
<td>2.0</td>
<td>*</td>
<td>1.09</td>
</tr>
</tbody>
</table>

Mean hemiplegic side: 61.7°
Mean contralateral side: Not applicable

*Data not reported because patients not tested at this angle.

TABLE 3
Triceps Spasticity and Hyperreflexia Measured as Twitch Tension Response on Hemiplegic Side

<table>
<thead>
<tr>
<th>Rank order, patient no.</th>
<th>Threshold rate, flexion, °/sec</th>
<th>Ratio of triceps tens./hammer force at 90°</th>
<th>Ratio of triceps tens./hammer force at 135°</th>
</tr>
</thead>
<tbody>
<tr>
<td>4</td>
<td>154</td>
<td>0.10</td>
<td>0.16</td>
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<tr>
<td>11</td>
<td>149</td>
<td>0.03</td>
<td>0.07</td>
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<tr>
<td>7</td>
<td>119</td>
<td>0.29</td>
<td>0.65</td>
</tr>
<tr>
<td>9</td>
<td>107</td>
<td>*†</td>
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<tr>
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<td>0.22</td>
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<td>3</td>
<td>64*</td>
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<td>0.13</td>
</tr>
<tr>
<td>2</td>
<td>55</td>
<td>0.18</td>
<td>*†</td>
</tr>
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</table>

Mean hemiplegic side: 98.6°
Mean contralateral side: Not applicable

*Since subject No. 3 measured 0.96 kg of resistance to passive motion compared to 0.70 kg of resistance by subject No. 3 at the same rate, subject 5 was ranked as more rate sensitive.
†Patients 6 and 10 were tested in flexion to only 74 and 56°/sec with no myotatic reflex responses observed. They could not be rank ordered since other patients exhibited thresholds at larger rate values.
‡Patient not tested at this angle.

In a similar manner, data obtained from tests of the threshold rates during flexion (triceps spasticity) are rank ordered in table 3 and compared with their corresponding tendon tap reflex sensitivity ratios. In table 3, patient number 7 showed a marked discrepancy. He had the third from highest rate threshold, 119°/sec, yet the highest (not lowest) reflex sensitivity ratio of 0.65 at 135° of elbow angle. The scattergram for these data, figure 5B, indicates no clearly defined relationship. A regression line is included for tests at an elbow angle of 90°. At this angle r = −0.47 which suggests a moderate degree of negative correla-
Discussion

The measured results corroborate the clinical observation that the degree of hyperreflexia seen in hemiplegia or hemiparesis which is due to intracranial vascular pathology is not necessarily an indication of the degree of spasticity. This is shown by the low correlation coefficients obtained between threshold values and corresponding reflex sensitivity ratios, and by a few marked discrepancies between the two measures. The negative correlations indicate that more severe spasticity (a lower rate threshold value) shows some tendency to be associated with greater hyperreflexia (higher reflex sensitivity ratio).

Bishop et al. demonstrated marked differences in the degree and pattern of hyperreflexia among hemiplegic patients. They identified two types of response patterns among 15 patients. Seven identified as type 1 showed symmetrical responses to H-reflex and Achilles tendon tap on the involved side. They did not relate this observation to spasticity, but clearly demonstrated that stroke patients exhibited different patterns of aberration in the organization of their motor control.

If the commonly implied concept that spasticity is simply due to hyperactive stretch reflex activity were valid, then why is there not a consistent direct relationship? Spasticity is a complex phenomenon which may be dissected into a number of specific related factors such as phasic and tonic stretch response sensitivity, muscle viscoelastic properties, contracture of skeletal connective tissue structures, and aberrations in the organization of motor control. This study measured spasticity just in terms of dynamic stretch characteristics which are affected by distortion of the organization of motor control.

One could expect the dynamic stretch characteristics of spasticity to relate closely to the degree of hyperreflexia if both depended exclusively upon the dynamic stretch sensing function of muscle spindles. The difference observed between them may be due to: (1) a large difference in the rate of application of the two types of stimuli; (2) a likely difference in the receptor systems stimulated; and (3) the distortion of normal spinal level reflex relationships by the upper motor neuron lesions. The monosynaptic stretch reflex as observed in animals responds sensitively to fusimotor control and also to multiple presynaptic inhibitory influences within the spinal cord. The tendon tap stimulates intramuscular sensors with an unaccustomed high rate of stretch, but should impart little stimulus to joint position receptors. Passive motion, on the other hand, provides commonly realized rates of stretch with adequate stimulation of both.
intramuscular stretch receptors and joint receptors.

Evidence suggests that the joint receptors may be important. The knee joint of the cat shows an elaborate system of receptors which mediate velocity of joint movement, acceleration and deceleration, extremes of joint displacement, and pain. These articular receptors have been shown to be important to limb muscle reflexes in cats and in man. Position sense was abolished in the great toe of man when the capsule of the metatarsal-phalangeal joint was infiltrated with local anesthetic, indicating that functional joint receptors are essential to position sense. Freeman and Wyke deduced that a major contribution of the articular receptors to articular reflexes was mediated through a bias effect on fusimotor neuron activity. The major contribution of joint receptors to position sense also suggests an important cortical input. The absence of awareness of joint position should not eliminate afferent input at the spinal level in stroke patients. Joint receptors thereby might contribute to spasticity through distorted organization of spinal reflexes and through distortion of long loop reflex arcs that include supraspinal levels. To test this the position sense deficit should be measured at the elbow, a special examination which was not included in this study.

Hyperactive tendon reflexes indicate disturbed functional motor organization related to intramuscular sensory receptors, but probably indicate little or nothing about the function of the joint receptors. The passive motion test for spasticity responds to the combined contribution of the two receptor systems.

The manner in which the central nervous system organizes and responds to the multiple sources of afferent information varies considerably from one normal individual to the next and appears to vary considerably more among hemiplegic or hemiparetic patients.

The distinction between spasticity and rigidity may be characterized clinically by the difference between the rapid buildup of passive resistance associated with the clasp-knife phenomenon versus the feeling of "lead pipe" resistance, which shows a relatively constant resistance to passive motion regardless of the rate at angular velocities above threshold. The former is the picture of spasticity generally associated with hemiplegia and hemiparesis. To distinguish between this response and rigidity, which is generally associated with parkinsonism, would raise a question not explored in this study. Clinicians do not ordinarily expect to find rigidity with hemiplegia and hemiparesis of cardiovascular origin. Since both spasticity and rigidity exhibit rate thresholds the distinction is not critical to this study.

The precision with which threshold rates were determined was limited by test-to-test variations in the responses of individual patients and by the spread between the subthreshold and threshold rates including when the values overlapped due to patient variation. The six flexion threshold rate determinations which showed no overlap of rates and for which both subthreshold and suprathreshold rates were measured showed a mean spread of $34 \pm 6^\circ$/sec. The four corresponding extension values showed a mean spread of $23 \pm 15^\circ$/sec. The two flexion threshold rate determinations which overlapped by one and $11^\circ$/sec. Five overlapping extension rate threshold determinations showed a mean overlap of $27^\circ$/sec. The test procedure, therefore, discriminated threshold values with a resolution of about $30^\circ$/sec. Although this is a course measure, it serves to distinguish clearly patients with low threshold values around two or three degrees per second from patients with high values above $100^\circ$/sec. This resolution could be improved considerably by conducting more tests at intermediate rate values. Some patients, however, are limited in their tolerance to prolonged testing.

The resolution obtained from the tendon reflex measure was limited by two major factors. This response generally fluctuates by a factor of 5:1 or 10:1 during a series of ten or 20 taps because of physiological variations in the sensitivity of the reflex response. A minimum of ten taps were averaged for each test to reduce the effect of this variation. Another factor was the variation in manually delivered tap intensity. The effect of this variation was reduced by using the reflex sensitivity ratio rather than the measured reflex response force.

In conclusion, a conventional tendon tap test for hyperreflexia provides evidence of abnormal organization of upper motor neuron
control. This monosynaptic reflex arises almost exclusively from intramuscular stretch receptors in response to a nonphysiologically rapid stimulus. On the other hand, by flexing and extending the joint, first very slowly and then at increasingly rapid rates, one can identify the rate at which passive resistance first occurs. This passive motion test elicits responses which can involve both intramuscular and articular sensory systems. In hemiplegic and hemiparetic patients with spasticity this physiological stimulus identifies, in more functional terms, the abnormal organization of motor control especially at the spinal reflex level. These considerations suggest that in patients with spastic hemiparesis or hemiplegia a clinical test for rate threshold can provide additional information more directly related to functional features of spasticity than can the usual tests for hyperreflexia.

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20. Dodge R: A systematic exploration of a normal knee jerk, its technique, the form of the muscle contraction, its amplitude, its latent time and its theory. Z Allg Physiol 12: 1-58, 1911
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