Quantitative Assessment of Intrathecally Administered Baclofen in Spasticity

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Objective: To quantitatively assess the antispastic effect of intrathecally administered baclofen on muscle stiffness in spastic patients.

Design: Case-control study.

Setting: Clinical laboratory in a university hospital of a city of more than 1,000,000 inhabitants.

Participants: Eighteen healthy adult volunteers (9 men, 9 women) were recruited for establishing the normal values. Eleven spastic patients (8 men, 3 women) comprised the study group.

Main Outcome Measures: The resistance to passive sinusoidal displacement of 5° imposed to the ankle joint was measured at frequencies from 3 to 12Hz. Torque and displacement signals were subjected to a Fourier analysis to isolate the elastic and viscous components of the total muscle stiffness.

Results: In comparison with the period before intrathecal injection, and with the control group, it was shown that at 4 hours after injection, stretch reflex activity was abolished and elastic and viscous muscle stiffness approached control values. The abnormal residual stiffness concerned only the elastic component due to chronic transformations of the spastic muscle and/or due to changes in joints and periarticular connective tissue. This antispastic effect was completely reversed 36 hours after injection.

Conclusion: The present study shows that the antispastic effect of intrathecally administered baclofen in spastic patients can be quantitatively assessed by a sensitive method allowing measurement of elastic and viscous components of muscle stiffness.

Key Words: Spasticity; Intrathecal baclofen; Stiffness; Measurement; Rehabilitation.

SPASTICITY IS DEFINED as a hyperexcitability of the myotatic reflex pathway, leading to a velocity-related increase in the monosynaptic stretch reflex and an exaggeration of the tendon reflexes.[1] This hyperexcitability is often associated with increased muscle tone, which, among other signs, can be expressed by increased resistance to passive mobilization of the joint. This increased muscle tone is in part responsible for abnormal postures. The pathophysiologic mechanisms involved are only partially understood, mainly due to our lack of knowledge of basic neurobiologic mechanisms and the lack of objective quantitative measurements. Intrathecally administered baclofen is presently one of the most effective methods for treatment of spasticity.[2-7] In a recent review, Campbell and colleagues[2] examined 24 studies assessing the therapeutic effect of intrathecal injections of baclofen in 348 spastic patients. All of these studies demonstrated a reduction in spasticity, evaluated both qualitatively or semiquantitatively. However, other studies have shown that these qualitative or semiquantitative methods lack objectivity, and that there is considerable inter- and intrainvestigator variability.[8-11] Among the 24 studies, 4 also demonstrated the therapeutic effect using quantitative electrophysiologic methods based on the Hmax/Mmax ratio or the level of spontaneous electromyographic (EMG) activity. Although these methods reduce investigator-dependent variability, they raise the problem of a low inter- and intrasubject reproducibility.[12] In addition, these methods did not objectively measure resistance to passive stretch, and thus they provide only indirect information on muscle tone.[12,13]

Lehmann and coworkers[14] developed a quantitative method for directly assessing muscle tone in normal and spastic subjects. This method, inspired by Rack's work,[15] measures muscle resistance to passive low-amplitude sinusoidal displacements of the ankle joint at different frequencies of oscillation and was the method used in this study. The relaxed, passively displaced calf-ankle-foot-system can be crudely modeled, in mechanical terms, as a torsional spring, a torsional viscous damper, and a rotary mass connected in parallel. The torsional elements are appropriate, because the system is being rotated about the ankle joint. The torsional spring represents the elasticity of the gastrocnemius-soleus-Achilles' tendon unit. The torsional viscous damper represents the viscous characteristics of the same tissues. The rotary mass represents the mass of the foot rotating about an idealized ankle pivot joint. The application of sinusoidal displacement to such a passive viscoelastic system will produce a characteristic torque response, which is dependent on the particular mechanical properties of the model's elements. Assuming linear properties for spring, damper, and mass, the total torque will be the sum of the torque generated by each of the 3 mechanical elements. The torsional spring will contribute a torque response in phase with the displacement. Indeed, the magnitude of the torque response of the spring is independent of the frequency of the sinusoidal displacement.

The second element of the model, a torsional viscous damper, produces a resisting force in proportion to the velocity applied to it and the viscosity of the damper. The torque response of the damper (viscous stiffness) is linearly related to the frequency of the sinusoidal displacement. The torque contribution of the rotating mass is proportional to the acceleration applied to it and its rotational inertia. For a sinusoidal displacement, the acceleration is proportional to the square of the frequency of the displacement. The torque contribution of the mass can be readily computed and subtracted from the total response,

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thereby leaving only the net muscle stiffness composed of the elastic and viscous responses of the system.[16]

Finally, based on the fact that elastic stiffness is in phase with the displacement and viscous stiffness is out of phase by 90°, the 2 components of net muscle stiffness may be computed separately using Fourier analysis, ie, decomposing the signals of displacement and torque into their sinusoidal components.[14,15] Using this method, Seib and coworkers[17] demonstrated a temporary beneficial therapeutic effect of cryotherapy, and Lehmann and associates,[18] transcutaneous electrical nerve stimulation (TENS), in spastic patients. During cryotherapy (placing a plastic freezer bag containing a mixture of water and ice on the calf during 20 min), they observed a statistically significant reduction in total muscle stiffness, although, in some cases, it did rebound within 1 hour after treatment. With TENS, total muscle stiffness immediately decreased and remained depressed for up to 24 hours. Yet these authors were unable to reveal a change in viscoelastic stiffness after orally administered baclofen (40mg/day and 80mg/day) in 5 adult males with traumatic spinal cord injuries, in whom the levels were 40mg/day and 80mg/day of baclofen versus placebo treatment.[16-18]

The aim of the present study is to provide objective and quantitative evidence of the beneficial effect of intrathecally administered baclofen in spastic patients using the method described by Lehmann.[14]

PATIENTS AND METHODS

Subjects. Eighteen healthy adult volunteers (controls: 9 men and 9 women; median height, 1.72m [range, 1.62-1.77m]; median weight, 69kg [range, 60.8-74.6kg]; median age, 25yrs [range, 22-56yrs] were recruited for establishing the normal values. To take into account differences in stiffness related to age and sex with the population of patients, the controls were broken into 2 age groups: 12 younger subjects (20 to 40yrs of age) and 6 older subjects (60 to 70yrs of age). Eleven successive referrals for intrathecal baclofen therapy comprised the study group. These patients (8 men and 3 women) suffered from spasticity that was functionally detrimental according to their own opinion and clinical assessment. They were selected for a trial with intrathecal baclofen, because high-dose oral baclofen therapy (at least 75mg/day) was without adequate relief of spasticity and/or induced, unmanageable side effects (drowsiness, dizziness, and fatigue). The main characteristics of the 11 patients are presented in table 1; the median age was 54yrs (range, 35-57.5yrs), and the median duration of the disease was 6yrs (range, 1.5-16.5yrs). Controls and patients participated in the study after giving their informed consent in accordance with the recommendations of the Ethics Committee of the Faculty of Medicine and the Saint-Luc University Hospital.

Table 1: Patients' Biometric Data and Clinical Features

Patient	Sex	Age (yrs)	Height (m)	Weight (kg)	Duration of Disease (yrs)	Etiology
1	M	35	1.65	65	32	Cerebral palsy–diplegia
2	M	54	1.60	70	18	Transverse myelitis
3	M	65	1.75	79	9	Spinal cord injury
4	F	54	1.63	64	2	Spinal cord injury
5	F	69	1.65	70	6	Multiple sclerosis
6	M	29	1.75	60	1	Spinal cord injury
7	M	42	1.80	85	42	Familial spastic paraparesis
8	M	24	1.70	75	2	Traumatic brain injury
9	M	35	1.68	70	15	Traumatic brain injury
10	M	55	1.85	85	1	Stroke
11	F	60	1.60	65	1	Stroke

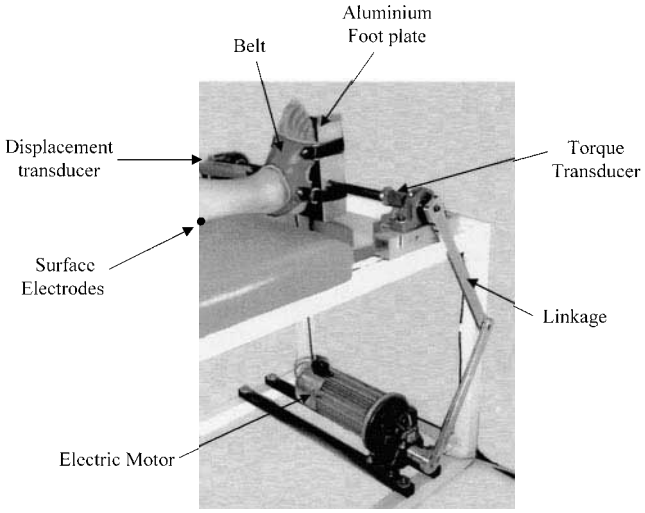

Fig 1. Spasticity measurement system.

Experimental apparatus The experimental apparatus (fig 1) was similar to that described by Lehmann,[14] the only difference being the subject's position on the examination table. In the present study, subjects were placed in a supine position with the knees extended, instead of the prone position; this change in position resulted in different values of stiffness from those reported by Lehmann.[14] Like Freedman and Herman,[19] we preferred the supine position, because the prone position was judged uncomfortable by all subjects. In addition, the supine position allowed better patient surveillance for possible side effects such as orthostatic hypotension or drowsiness[20] related to the intrathecal injection. All subjects were instructed to relax. Relaxation of the triceps surae was monitored via a telemetric EMG apparatus[a] with disposable surface electrodes[b] placed over the muscle belly, ie, one handbreadth below the popliteal crease on the mass of the calf.[21] Records contaminated by unintentional muscle activity were discarded.

The subject's foot was firmly belted to an aluminum plate mounted on a horizontal rotational axis. The foot was adjusted to optimize alignment of the ankle's axis of rotation with that of the footplate. An electric motor[c] produced 5° amplitude ($\pm$2.5° around the neutral position) sinusoidal displacements of the plate at a frequency of 3 to 12Hz in 1-Hz increments. A potentiometer[d] mounted on the axle of the footplate recorded angular displacements, while 4 strain gauges[e] in a Wheatstone bridge mounted on the axle measured the torsion force. The angle, force, and EMG signals were digitized[f] at 1,000Hz and recorded on an IBM-PC.

Experimental procedure Each subject underwent 3 measurement sessions. Each session consisted of 30 trials consisting of 10 different frequencies of oscillation (from 3 to 12Hz) repeated 3 times in random order. For each session, results were expressed as the means computed from the average of the 3 measurements for each frequency of oscillation. In the control group, the second and third measurement sessions were performed after 1 week and 1 month to assess the reproducibility of the measurements. In the control group, results were expressed as the medians, computed from the mean of the 3 consecutive sessions irrespective of age and sex. In the experimental group, the first session was performed before administration of ba-

clofen (session 1). The second session (session 2) was performed 4 hours after intrathecal administration, by direct lumbar puncture, of a bolus of 50µg baclofen. (This dose has been established as the first step in the screening procedure for chronic infusion of intrathecal baclofen through an implanted delivery system.[20,22] The third session was performed 36 hours after the injection (session 3). The testing times were defined on the basis of the drug's clinical effects and pharmacokinetics. The antispastic effect of a single 50-µg bolus decreases after 8 to 12 hours, after which the spasms and rigidity gradually increase to their previous level. The half-life of baclofen in the cerebrospinal fluid is approximately 4 to 5 hours.[23] The effect of intrathecal baclofen administration was assessed with a Friedman Repeated Measure Analysis of Variance on Ranks with Dunett's method.[g]

Signal analysis Angular displacement and torque were analyzed by Fourier transformation[h] to decompose the signals into their sinusoidal components. Only the fundamental frequency, corresponding to the footplate oscillation frequency, was retained. Total stiffness was drawn on the phase diagram and corresponds to the vector sum of the elastic and viscous components. Variations in the total stiffness ($N \cdot m \cdot rad^{-1}$) at the 10 different frequencies of oscillation were calculated from the total length (L) of the path formed by the apex of the 10 total stiffness vectors according to the following equation[14]:

$$L = \sum_{i=3}^{i=11} \sqrt{(v_i - v_{i+1})^2 + (e_i - e_{i+1})^2} \qquad \text{Eq. 1}$$

where *v* denotes viscous stiffness, *e* denotes elastic stiffness, and *i* indexes the frequencies of oscillation. In other words, the path length (L) is a measure of the variation in total viscoelastic stiffness over a range of frequencies of ankle oscillation.

RESULTS

Elastic and viscous stiffness are shown as a function of oscillation frequency for both the controls and the patients in figure 2A and 2B. Measurement reproducibility for elastic and viscous stiffness in the controls was good, because the 3 sessions were not statistically different ($F = .63$; $p = .54$). In controls, as expected, elastic stiffness was independent of frequency (average $R^2 = .04$). Median value of elastic stiffness was $41N \cdot m \cdot rad^{-1}$ (range, 32.0 = 49.5). Viscous stiffness increased linearly with oscillation frequency (intercept, $-1.4 \pm 7.6N \cdot m \cdot rad^{-1}$; slope $4.0 \pm 2.0N \cdot m \cdot s \cdot rad^{-1}$; average $R^2 = .88 \pm .08$) and rose from $15N \cdot m \cdot rad^{-1}$ at 3Hz to $55N \cdot m \cdot rad^{-1}$ at 12Hz. The apexes of the total stiffness vectors in the phase diagram (fig 3) showed a nearly vertical path with a length (L) estimated at $46N \cdot m \cdot rad^{-1}$ (range, 39.8 to 61.2). The pathlengths (L_c) vary with age and sex as shown in table 2. Older subjects had higher total stiffness values as compared with younger subjects, mainly because of an increase in elastic stiffness with age. Males also had significantly greater pathlengths than females because of greater stiffness in males.

For the 11 patients, the elastic and viscous components of the stiffness are also shown as a function of oscillation frequency in fig 2A and 2B. Before treatment (session 1), elastic stiffness (fig 2A) followed a V-shaped curve, attaining values of $135N \cdot m \cdot rad^{-1}$ at a low frequency and $120N \cdot m \cdot rad^{-1}$ at a high frequency of oscillation, while at intermediary frequencies (7-8Hz), the elastic stiffness values were minimal ($\pm 85N \cdot m \cdot rad^{-1}$). Viscous stiffness (fig 2B) followed an S-shaped path, crossing the curve for the controls at 7Hz, showing a minimum of about $4N \cdot m \cdot rad^{-1}$ at 4Hz, and a maximum of about $100N \cdot m \cdot rad^{-1}$ at 12Hz. In the phase diagram (fig 3), in the first session (session 1), the apexes of the total stiffness vectors follow a C-shaped path. The lengths (L_P-session 1) of the paths are shown in table 3 for each patient. Pathlengths were 2.8 to 12.6 times greater (median, 6.5) than those of controls, of comparable age and sex.

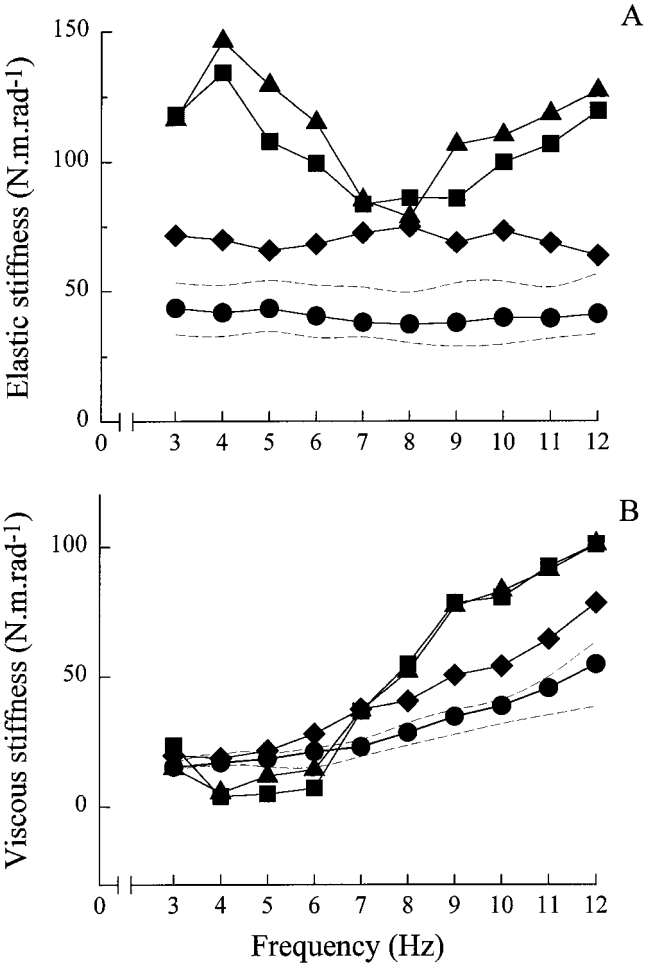

Fig 2. Elastic (A) and viscous (B) stiffness (in $N \cdot m \cdot rad^{-1}$) as a function of frequency of oscillation (in Hz). (●, the median values for stiffness in the controls; ■, the median values for stiffness in patients before intrathecal administration of baclofen [session 1]; ◆, the median values for stiffness in patients 4 hours after intrathecal injection of a 50-µg bolus of baclofen [session 2]; ▲, the median values for stiffness in patients 36 hours after the intrathecal injection of baclofen [session 3]. The *dashed lines* indicate values calculated for the 25% and 75% quartiles.

Four hours after intrathecal administration of baclofen (session 2; fig 2A), elastic stiffness became independent of oscillation frequency (average $R^2 = .11$) and was dramatically reduced (median, $71N \cdot m \cdot rad^{-1}$; range, 52.8-88.1), but it was still about twice the value in the controls. Viscous stiffness increased much faster with oscillation frequency as compared with the controls and rose from $20N \cdot m \cdot rad^{-1}$ at 3Hz to $80N \cdot m \cdot rad^{-1}$ at 12Hz (intercept, $-7.7 \pm 7.8N \cdot m \cdot rad^{-1}$; slope, $6.7 \pm 3.6N \cdot m \cdot s \cdot rad^{-1}$; average $R^2 = .89 \pm .08$). These changes are better illustrated in the phase diagram (fig 3), in which the apexes of the total stiffness vectors show a nearly vertical path, different from the C-shaped path of session 1 (fig 3). The individual pathlengths of patients treated with intrathecal baclofen (L_P-session 2) are shown in table 3. The median of the pathlengths in session 2 ($98N \cdot m \cdot rad^{-1}$; range, 80-111) was significantly reduced as compared with session 1; however,

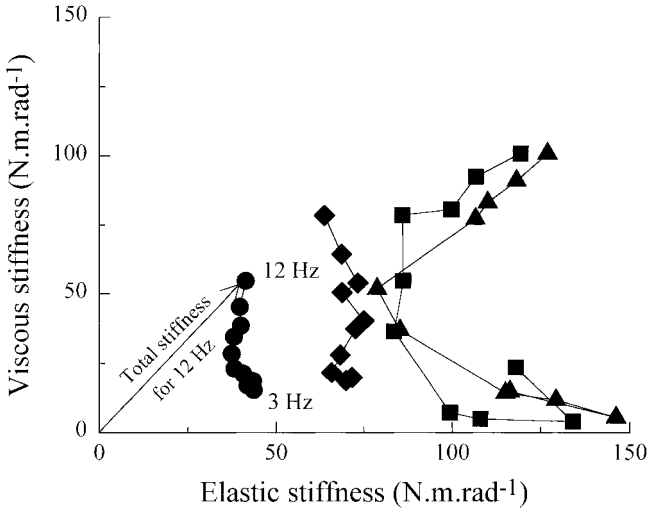

Fig 3. Phase diagram of viscous stiffness (in $N \cdot m \cdot rad^{-1}$) versus elastic stiffness (in $N \cdot m \cdot rad^{-1}$). The (●, the median values for stiffness in the controls; ■, median values for stiffness in patients before intrathecal administration of baclofen [session 1]; ◆, median values for stiffness in patients 4 hours after intrathecal injection of a 50-μg bolus of baclofen [session 2]; ▲, median values for stiffness in patients 36 hours after the intrathecal injection of baclofen [session 3]. The *arrow* represents the total stiffness vector at 12Hz in the controls.

the value remained significantly larger than the controls (Mann-Whitney rank sum test: $T = 66.0$; $p < .001$).

Thirty-six hours after intrathecal administration of baclofen (session 3), the curves of the elastic and viscous stiffness components (fig 2A-2B) were similar to those observed in the patients before the injection (session 1). In the phase diagram (fig 3), the apexes of the total stiffness vectors again had a C-shaped path as at session 1 (fig 3). The pathlengths at session 3 (table 3) were not significantly different from those obtained before the injection of baclofen, ie, with a median value 7.9 times greater than that in controls. These results clearly indicate that there is no significant residual effect of intrathecally administered baclofen. At the same time, they also show that the experimental method displays high reproducibility for spastic patients.

DISCUSSION

To better understand the pathophysiologic mechanisms of spasticity, and to assess the beneficial effect of therapeutic interventions, objective and quantitative tools are needed. Until now, the antispastic action of intrathecally injected baclofen was evaluated on the basis of qualitative clinical scales or semiquantitative methods.[2] To our knowledge, no former study has provided objective and quantitative evidence of the dramatic effect of intrathecally injected baclofen in reducing the increased resistance to passive motion that characterizes spasticity. The method developed by Lehmann,[14] based on work by Rack,[15] consists of measurements of the resistance of the ankle muscles to low-amplitude sinusoidal movements imposed at different frequencies of oscillation. The analysis of the resistance to displacement allows 2 components of muscle stiffness to be distinguished: a frequency-independent component, the elastic stiffness, and a frequency-dependent component, the viscous stiffness. It is shown that spasticity was associated with a characteristic rise in these 2 components. As compared with control subjects, elastic stiffness was 2 to 3 times greater, except at intermediary frequencies of oscillation (fig 2A), and the slope of the viscosity versus frequency of oscillation curve is much steeper (fig 2B). Four hours after the intrathecal injection of baclofen (session 2), both elastic and viscous stiffness approached the control values. These changes were associated with a reduction in reflex activity as shown by the absence of EMG activity in the triceps surae during ankle movements at low, intermediate, and high frequencies of oscillation (fig 4, traces G, H, and I). This effect was reversible, because 36 hours later, the measures of elastic and viscous stiffness returned to their initial levels. These changes in stiffness are readily expressed as variations in the length of the path (L) formed by the apex of the 10 total stiffness vectors (fig 3, table 3).

Table 2: Pathlengths in the Phase Diagrams of the Controls (L_C)

	Male L_C ($N \cdot m \cdot rad^{-1}$)	Female L_C ($N \cdot m \cdot rad^{-1}$)
Younger subjects (20-40 years of age)	45 (45.7-60.3)	27 (25.5-43.5)
Older subjects (60-70 years of age)	67 (66.1-76.9)	43 (44.8-50.8)

Median values (25%-75% quartiles) are expressed in $N \cdot m \cdot rad^{-1}$.

Table 3: Pathlengths in the Phase Diagrams for Spastic Patients (L_P), and the Ratio (L_P/L_C) of the Pathlengths of the Patients (L_P) to That of the Controls (L_C) in the 3 Consecutive Sessions

	Session 1		Session 2		Session 3	
Patient	L_P ($N \cdot m \cdot rad^{-1}$)	L_P/L_C	L_P ($N \cdot m \cdot rad^{-1}$)	L_P/L_C	L_P ($N \cdot m \cdot rad^{-1}$)	L_P/L_C
1	275	6.11	98	2.17	291	6.46
2	402	8.91	77	1.71	356	7.91
3	188	2.81	103	1.53	214	3.19
4	194	7.18	79	2.92	259	8.86
5	279	6.48	144	3.34	364	8.46
6	546	12.13	141	3.11	600	13.3
7	569	12.64	82	1.82	492	10.9
8	241	5.35	120	2.66	251	6.46
9	346	7.68	100	2.22	389	8.64
10	135	3.00	75	1.66	149	3.31
11	150	3.48	82	1.82	124	2.88
Median	275	6.5	98	2.2	291	7.9
25%-75% quartiles	191-374	4.4-8.3	80-111	1.8-2.8	232-376	4.9-8.8

C-shaped path in the phase plane and timing of the reflex response. In the 11 spastic patients, both before the intrathecal bolus of baclofen and 36 hours after, the total stiffness vectors changed with oscillation frequency in a characteristic way (fig 3). With increasing oscillation frequency, the vectors moved in a clockwise direction describing a C-shaped path. When such a phenomenon is observed, it may be assumed (see below) that the resistance includes a component that is a result of stretch reflex activity.[24]

In the spastic patients, low frequencies of oscillation (3-6Hz) generate EMG reflex activity in the triceps surae during dorsal flexion of the ankle (fig 4, traces D). Because the active stiffness (ie, the stiffness generated by stretch reflex activity) is in phase with the passive stiffness (ie, the stiffness generated by a muscle deprived of spinal influences), they add up, and consequently, the elastic stiffness is increased (fig 2A). However, the viscous stiffness is lowered (fig 2B). This can be explained as follows: A viscous component, like a damper, induces a time lag, which, in turn, is responsible for a phase delay in the torque-vs-displacement relationship. This phenomenon causes the muscle force to assist rather than resist the ankle displacement. As a consequence, the total stiffness vector in the phase diagram (fig 3) approaches the horizontal axis, and in some cases, can even

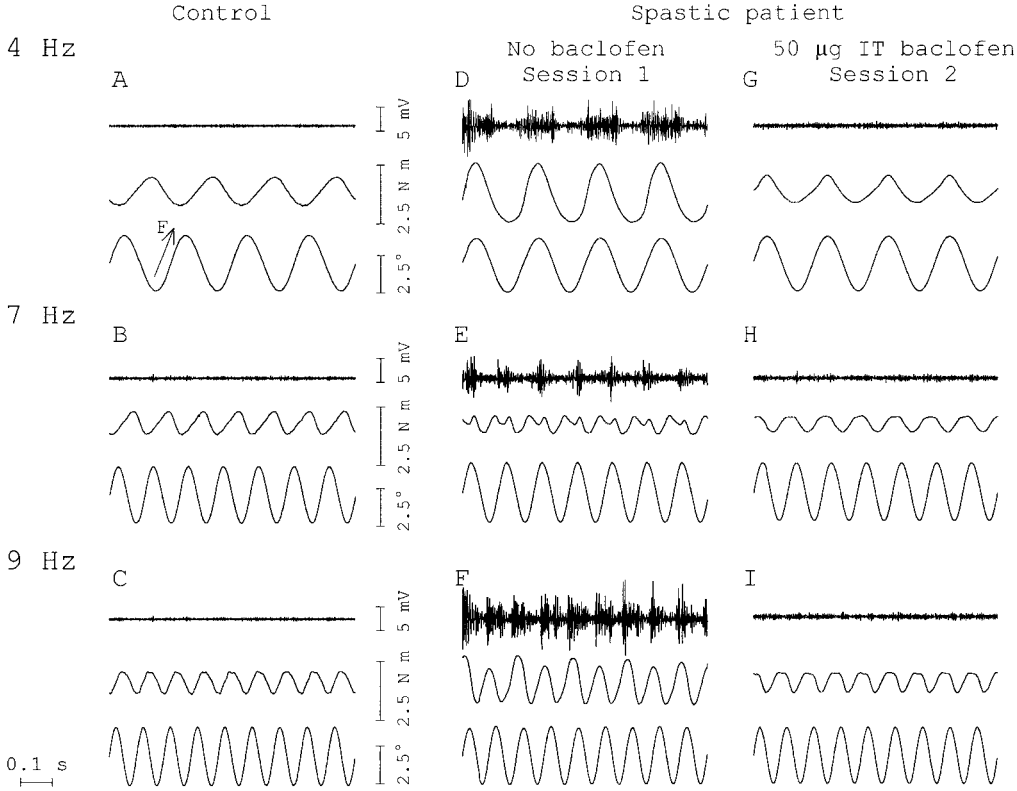

Fig 4. Typical records from a control subject (35-year-old male; records A to C) and from a patient (patient 2) before (records D to F) and 4 hours after a 50-µg intrathecal bolus injection of baclofen (records G to I). Each record consists of 3 traces showing, from top to bottom: surface EMG of the triceps surae, torque response at the ankle, and 5° sinusoidal displacement of the ankle joint (±2.5° around neutral position). The upward displacement is the direction of dorsal flexion of the ankle (F and upward *arrow*). The 3 rows represent, from top to bottom, records obtained at different frequencies of oscillation: respectively, 4Hz (records A, D, and G), 7Hz (records B, E, and H), and 9Hz (records C, F, and I).

produce a ''negative viscous'' resistance, indicating that the flexor plantar muscles exert more force while they are shortening than when they are being forcibly extended. The lowering of viscous stiffness at low frequencies of oscillation in spastic patients may thus be regarded as proof of the induction of stretch reflex activity by the movement imposed to the ankle.[25]

At intermediate frequencies (7-8Hz), and in contrast with the controls (fig 4, compare traces B and E), the torque response displayed two distinct peaks per cycle. The first peak occurs during dorsal flexion of the ankle. It is generated for the most part by the passive rheologic properties of the triceps surae, as a result of the delay due to the electromechanical coupling and the stretch reflex activity that occurs only at the end of the stretch movement. The second peak occurs at the beginning of the plantar flexion of the ankle approximately 50msec after the first EMG burst of stretch reflex activity. This mechanical action of the triceps surae has a tendency to assist the extension, reducing the resistance to the opposing movement. As reported by Rack and colleagues,[25] this reduced muscle stiffness was also observed in normal subjects when their ankle was submitted to prolonged (30-min) sinusoidal movements. However, that reduction was observed at higher frequencies (8-10Hz) as compared with spastic patients. It would occur at lower frequencies of oscillation in spastic patients because their motoneurons are triggered at lower frequencies, due to greater and more prolonged recurrent inhibition.[26] This reduction in muscle stiffness is clearly shown by the upper left point of the C-shaped path in the phase diagram (fig 3). This point is of special interest, because it indicates the frequency at which the contractile force, which originates from stretch reflex activity, gives maximal assistance to the ongoing movement. This point is known as the ''resonant'' frequency.[27]

With higher frequencies of oscillation (9Hz; fig 4, traces F), the torque response displays only one peak that appears toward the middle of the plantar flexion movement of the ankle. The shorter period of the cycle movement and the transmission time of the stretch reflex pathway (30-50msec for the Achilles' tendon reflex) explains why the corresponding EMG reflex activity is manifested on the following cycle. Under these circumstances, total muscle stiffness was greater because the active stiffness was again in phase with the passive stiffness. In spastic patients, stretch reflex activity modulates both components of muscle stiffness as a function of frequencies of oscillation.

C-shaped path in phase plane and influence of intrathecally administered baclofen. Intrathecal administration of baclofen suppressed the stretch reflex activity (fig 4, traces G, H, and I) as shown by the flat EMG records. More specifically, it quickly changed the total stiffness vectors from a C-shaped path to a nearly vertical line in the phase diagram (fig 3). Nevertheless, the pathlength (L) in the phase diagram remained significantly longer as compared with the controls. This cannot be explained on the basis of changes in viscous stiffness (fig 2B), because it was not different from the control value (Mann-Whitney rank sum test: $T = 87.00$; $p = .186$). Thus, the residual lengthening of L under intrathecally administered baclofen can only be attributed to the increase in passive elastic stiffness. Indeed, it is twice as high as in the controls (table 3). This increase in elastic stiffness may result from chronic transformations of the spastic muscle and/or from changes in the joints and periarticular connectives tissues that are expressed clinically by fixed contractures.[28,29] Similar results were reported and the same conclusions were drawn by Lehmann[14] in spastic patients after anesthetic block of the tibial and fibular nerves.

CONCLUSIONS

To study the effectiveness of therapeutic intervention, it is essential to first have an objective, quantifiable method of measuring spasticity. A quantitative method was developed by Lehmann[14] to measure the degree of spasticity by determining

the resistance to a sinusoidal displacement of the ankle. The present study shows that the antispastic effect of intrathecally administered baclofen in spastic patients can be quantitatively assessed. In addition, this sensitive method allows differentiation between the effects on elastic and viscous components of muscle stiffness.

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