Does spasticity contribute to walking dysfunction after stroke?

Louise Ada, Wantana Vattanasilp, Nicholas J O'Dwyer, Jack Crosbie

Abstract

***Objectives*—Clinically, it is assumed that spasticity of the calf muscles interferes with walking after stroke. The aim was to examine this assumption by evaluating the contribution of spasticity in the gastrocnemius muscle to walking dysfunction in an ambulant stroke population several months after stroke.**

***Methods*—Fourteen stroke patients who were able to walk independently and 15 neurologically normal control subjects were recruited. Both resting and action stretch reflexes of the gastrocnemius muscle were investigated under conditions that simulated walking. Resting tonic stretch reflexes were measured to assess spasticity whereas action tonic stretch reflexes were measured to assess the possible contribution of spasticity to gait dysfunction.**

***Results*—Two thirds of the stroke patients exhibited resting tonic stretch reflexes which indicate spasticity, whereas none of the control subjects did. However, the stroke patients exhibited action tonic stretch reflexes that were of similar magnitude to the control subjects, suggesting that their reflex activity during walking was not different from that of control subjects. Furthermore, there was no evidence that the action stretch reflex in the stroke patients contributed a higher resistance to stretch than the control subjects.**

***Conclusions*—Whereas most of the stroke patients exhibited spasticity when measured both clinically and physiologically, they did not exhibit an increase in resistance to dorsiflexion due to exaggerated action tonic stretch reflexes. It is concluded that it is unlikely that spasticity causes problems in walking after stroke in ambulant patients. Therefore, it seems inappropriate to routinely reduce or inhibit the reflex response to improve functional movement in stroke rehabilitation. Factors other than spasticity should be considered when analysing walking after stroke, so that appropriate treatment is provided to patients.**

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Problems in walking after stroke have often been attributed to spasticity by both medical and physiotherapy practitioners.[1 2] Observation of the so called "spastic gait" discloses common abnormalities such as the toes not clearing the ground during the swing phase.[3 4] The assumption has been that spasticity, in the form of hyperactive reflexes, produces overactivity in the calf muscles and the resulting hypertonia resists dorsiflexion of the ankle joint.[5] The clinical sequel of this assumption has been to inhibit the calf muscles after brain damage—for example, with drugs[6] or by rehabilitation strategies.[2] The purpose of this study was to revisit these assumptions by determining the contribution of spasticity in the calf muscles to walking dysfunction in a population of patients at least six months after stroke.

Clinically, spasticity is a term commonly applied to a wide range of motor impairment. However, it has been defined as an increase or exaggeration of the stretch reflexes—both the tonic and phasic components.[7] The gastrocnemius is the multijoint calf muscle which flexes the knee and plantarflexes the ankle. During the single support phase of stance, the foot remains flat on the ground and the shank rotates forward. This results in the gastrocnemius muscle being stretched at the end of its range because the knee is extended and the ankle achieves maximum dorsiflexion. It is possible, therefore, that any abnormal stretch reflex would produce overactivity in the muscle thereby interfering with dorsiflexion. It is difficult to measure stretch reflexes during walking. Normally, during the period of stance when the foot is flat on the ground, there is activity in the gastrocnemius muscle which increases from mid-stance to reach a peak at heel off.[8 9] It is impossible to distinguish the contribution of the stretch reflex to this activity from voluntary activation. This study, therefore, investigated the nature of the stretch reflex in the gastrocnemius muscle after stroke under conditions that simulated the ankle movement during walking as far as possible.

Reflexes were measured under both passive and active conditions. When a relaxed muscle is stretched slowly (resting tonic stretch reflex), there is normally no electrical response.[10–14] After neurological impairment, however, there is often an abnormal response which is labelled as spasticity. On the other hand, when an active muscle is stretched the muscle activity is modulated by the stretch and this phenomenon has been termed an action tonic stretch.[15 16] Resting stretch reflexes were, therefore, measured to assess spasticity and action stretch reflexes were measured to assess the possible contribution of spasticity to gait dysfunction.

Table 1 Characteristics of subjects

Subjects	Age (y)	Sex	Time after stroke (months)	MAS* walking (1–6)	Side tested	Ashworth scale† (0–4)	Tendon jerk‡ (+ to ++++)
Stroke:							
1	58	M	11	5	L	0	+++
2	56	M	9	5	L	3	+++
3	69	F	5	5	R	1	+++
4	71	F	18	5	L	3	++++
5¶	71	M	20	3	L	0	+++
6	56	F	13	5	L	2	+++
7	59	M	7	4	R	1	+++
8	64	F	5	5	L	1	+
9	76	M	8	5	R	2	++
10¶	65	F	16	3	L	1	+++
11	64	M	18	4	R	1	++
12	45	F	14	4	L	2	+++
13	72	F	18	4	L	1	++
14¶	78	F	7	3	L	0	+++
Control:							
15	49	F	—§	—§	L	0	++
16	46	F	—	—	R	0	++
17	63	F	—	—	L	0	++
18	62	F	—	—	R	0	++
19	53	F	—	—	L	0	++
20	52	M	—	—	L	0	++
21	46	M	—	—	R	0	++
22	57	F	—	—	R	0	++
23	48	M	—	—	L	0	++
24	48	F	—	—	R	0	++
25	47	F	—	—	L	0	++
26	51	F	—	—	R	0	++
27	46	F	—	—	L	0	++
28	56	M	—	—	R	0	++
29	54	M	—	—	R	0	++

*Motor assessment scale (MAS)[17]; grade 1=stands on affected leg and steps forward with other leg. (weight bearing hip must be extended); therapist may give standby help; grade 2=walks with standby help from one person; grade 3=walks 3 metres alone or using any aid but no standby help; grade 4=walks 5 metres with no aid in 15 seconds; grade 5=walks 10 metres with no aid, turns around, picks up a small sandbag from the floor, and walks back in 25 seconds (may use either hand); grade 6=walks up and down 4 steps with or without an aid but without holding the rail 3 times in 35 seconds.
†Ashworth scale[18]; grade 0=no increase in muscle tone; grade 1=slight increase in muscle tone, manifested by a catch and release or by minimal resistance at the end of the range of motion when the affected part is moved in flexion or extension; grade 2=more marked increase in muscle tone through most of the range of motion, but affected part easily moved; grade 3=considerable increase in muscle tone, passive movement difficult; grade 4=affected part rigid in flexion or extension.
‡='Tendon jerk'[19]; 0=absent; +=hyporeflexia; ++=normal; +++=slight hyperreflexia; ++++=marked hyperreflexia.
§Not applicable.
¶These patients walk with a one point cane.

Methods

SUBJECTS

Fourteen stroke patients were recruited for this study (table 1). They presented clinically as hemiparetic and were within five to 20 (mean 12 (SD 5))months of their first stroke. The mean age of the subjects was 65 (SD 9) years. The only inclusion criteria were that subjects who could walk independently with or without an aid—that is, they scored 3 or above on the walking item of the motor assessment scale (MAS),[17] and had enough cognitive ability to participate in the measurement procedures. Clinical measures of lower limb muscle tone[18] and Achilles tendon jerks[19] were performed on all subjects (table). Stroke patients ranged evenly between 0 to 3 on the Ashworth scale with most demonstrating an increase in muscle tone and exaggerated tendon jerks compared with control subjects. Fifteen neurologically normal subjects were recruited to act as control subjects. Their mean age was 52 (SD 6) years. The experimental procedures were approved by the relevant institutional ethics committee and all subjects gave informed consent before data collection was undertaken.

MEASUREMENT OF TONIC STRETCH REFLEXES

Tonic stretch reflexes were measured under conditions that simulated the single support phase of walking as much as possible. During single support, the knee extends and the ankle joint rotates from 10° of plantarflexion to 10° of dorsiflexion[20] which effectively stretches the gastrocnemius muscle. Perry[21] has shown that there is no difference in the reflex response of the gastrocnemius muscle when tested in a sitting position compared with a standing position. Therefore, the subject sat with the knee extended while the ankle was rotated through 20° at frequencies of 0.5, 1, 1.5, and 2 Hz, which were chosen to cover the frequency range of ankle movement in normal walking as well as the slower walking of stroke patients. The subject sat with the foot securely strapped to the movable arm of an instrumented foot frame (fig 1). A potentiometer aligned with the lateral malleolus measured ankle angular displacement. A load cell (capacity: 450N; linearity 97%) measured the resistance of the foot to movement to quantify hypertonia, and silver-silver chloride surface electrodes measured medial gastrocnemius EMG activity to quantify reflex hyperexcitability. Manual sinusoidal stretching was performed (1) while the subjects were relaxed (resting tonic stretch reflex) and (2) while they were contracting their gastrocnemius muscles as they would be during walking (action tonic stretch reflex).

In the first condition, subjects relaxed, as confirmed by the absence of EMG, and then the foot was manually rotated back and forth about the ankle at the four frequencies. Each

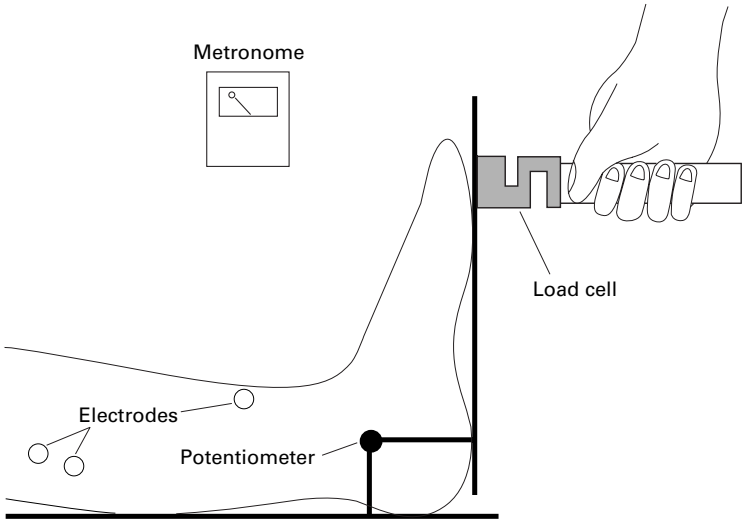

Figure 1 Set up for measurement of the tonic stretch reflex. Diagram of the equipment used for measuring and recording of the EMG, ankle angle, and force signals. The foot was positioned so that the lateral malleolus was aligned with the potentiometer.

trial of stretching lasted 25 seconds. The movement was timed to a metronome to control the frequency and the ankle angle was displayed on the computer monitor so that the amplitude of stretch could be controlled. Usually, no EMG activity is seen when the relaxed muscles of a neurologically normal person are slowly stretched in this manner.[11 22] Therefore, any stretch induced EMG activity (resting tonic stretch reflex) seen in the stroke patients was taken as evidence of spasticity.

In the second condition, subjects contracted their gastrocnemius muscle during stretching. The background contraction was achieved in the following way. Subjects stood on the ball of one foot as a way of standardising a procedure which would elicit a near maximal contraction of the gastrocnemius muscle. Then, in long sitting they were required to maintain a background contraction of 10% of this activity and feedback of the average level of muscle activity was displayed on the computer monitor to help subjects sustain this contraction while the foot was rotated back and forth. When the contracting muscles of a neurologically normal person are stretched in this manner, the voluntary activity is modulated up and down by the stretch.[16 23 24] Therefore, the stretch induced EMG activity (action tonic stretch reflex) seen in the control subjects was used as a frame of reference against which to compare the reflex activity of the stroke patients.

After amplification of the EMG (×5000) and force (×1000), the three signals—ankle angle, force, and EMG—were sampled by a 16-bit A-D converter at 1600 Hz and stored on a computer. Angle torque was calculated as the product of the load output and the perpendicular distance to the ankle joint axis. To remove any 50 Hz line frequency interference or low frequency movement artefact, the EMG was high pass filtered (digital, zero phase eighth order Butterworth) at 80 Hz. Subsequently, the EMG was full wave rectified and, along with the ankle angle and torque signals, low pass filtered (digital, zero phase eighth order Butterworth) at 4 Hz to obtain a DC voltage (IEMG) proportional to the contraction level of the muscle. This cut off frequency was chosen because the frequencies of interest were well below 4 Hz.

The magnitude of reflex activity and resistance were quantified as follows. The angle and IEMG signals were subjected to cross correlational and spectral analysis[25 26] to quantify the resting and action tonic stretch reflexes. This analysis allows stretch evoked muscle activity at the stretching frequencies to be distinguished from other activity unrelated to the stretch. The magnitude of these stretch reflexes was quantified by the gain of the tonic stretch reflex—that is, the magnitude of the stretch evoked IEMG activity divided by the magnitude of stretch. The angle and torque values were also subjected to cross correlational and spectral analysis and the resistance to passive movement was quantified by the gain of the angle-torque relation—that is, the magnitude of the stretch evoked torque divided by the magnitude of the stretch. Figure 2 presents the angle, EMG, and IEMG signals of resting and action tonic stretch reflexes for a stroke patient and a control subject.

MEASUREMENT OF WALKING

Subjects were asked to walk barefooted over flat ground at their preferred walking speed. A flexible electrogoniometer (Penny and Giles twin axis, M110) was used to measure the angular movement of the ankle joint during walking. The electrogoniometer was oriented along a line from the head of the fibula, through the lateral malleolus, to the head of the fifth metatarsal. Pressure sensitive foot switches were attached under the big toe and the heel so that they indicated heel contact, foot flat, heel off, and toe off in the gait cycle. Subjects were required to stand with the lower leg vertical to record the neutral position of the ankle joint and this was defined as 0°. Then they were asked to walk along a walkway three times, which yielded five to nine strides for each subject. The signals from the pressure sensors and the electrogoniometer were sampled by an A-D converter at 100 Hz and low pass filtered (digital, zero phase, eighth order Butterworth at 20 Hz). The frequency spectrum of ankle movement during walking and the range of the ankle joint during the foot flat period (the range during which the gastrocnemius muscle was stretched) were computed.

STATISTICAL ANALYSIS

Measurement during stretching yielded four outcome variables—reflex gain and torque-angle gain (with repeated measures at four different frequencies) under both relaxed and active conditions. Measurement during walking yielded three variables— ankle dorsiflexion at the end of foot flat period, ankle plantarflexion at the beginning of foot flat period, and the stride time. The data were examined descriptively, and mean and SD are presented. The measures of walking were examined to confirm that the conditions of stretch did mimic the movement of the ankle joint during walking.

The necessary assumptions of the analyses were tested and were satisfactory. Analyses of variance (or covariance) were used to compare outcome variables: (1) the gain of the action tonic stretch reflex (across frequency) between control and stroke patients, (2) torque-angle gain (across frequency) under active conditions between control subjects and stroke patients, (3) the torque-angle gain (across frequency) under relaxed conditions for the control subjects, stroke patients without reflexes, and stroke patients with reflexes. Each analysis had one group factor and one repeated measures factor.[27] Data from two stroke patients were excluded from these analyses of variance due to their inability to perform the tests satisfactorily. Finally, multiple regression was used to examine the relative contributions of the gain of the action tonic stretch reflex and the background muscle contraction to the torque-angle gain. In general, the degrees of freedom in the error term reflects the number of subjects included in that analysis.

Results

WALKING MEASUREMENT

Ankle joint movement during the single support phase of walking was measured for each cycle. During this period, the stroke patients exhibited a range of 13.5° of ankle movement (from 5 (SD 5)° of plantarflexion to 8.5 (SD 5.3)° of dorsiflexion) which was significantly less (t=3.6, df=27, p<0.01) than the control subjects who exhibited a range of 20.7° (from 11.5 (SD 3.5)° of plantarflexion to 9.2 (SD 2.7)° of dorsiflexion). The stroke patients walked more slowly than the control subjects as reflected in their increased stride times, 1.8 (SD 0.6) seconds for stroke and 1.3 (SD 0.1) seconds for the control subjects. In turn, a fast Fourier transform of the ankle angle for the whole gait cycle (fig 3) showed that the periodicity of the walking cycle for control subjects was 0.8 Hz, which was significantly higher (t=4.32, df=27, p<0.001) than that of the stroke patients at 0.6 Hz. In the stroke patients, 75% of the accumulated power was located below 2.3 Hz, whereas the corresponding frequency of 2.8 Hz in the

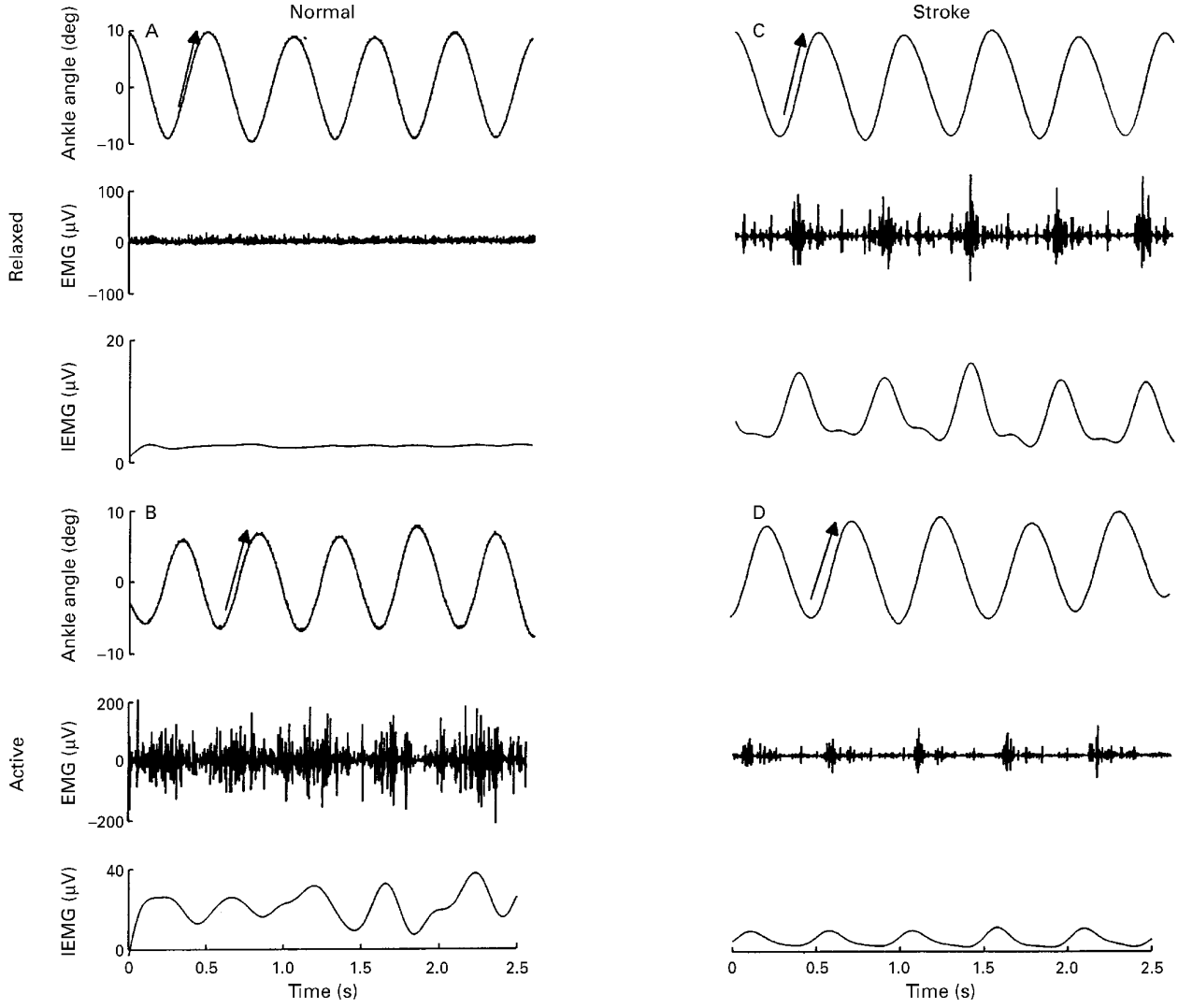

Figure 2 Ankle angle, EMG, and IEMG under relaxed and active stretch at 2 Hz for a control subject and a stroke patient. (A) Control subject during relaxed stretch showing no EMG activity. (B) Control subject during active stretch showing normal action tonic stretch reflex. (C) Stroke patient during relaxed stretch showing abnormal resting tonic stretch reflex. (D) Stroke patient during active stretch showing a smaller action tonic stretch reflex than the control subjects. Arrows indicate direction of stretch.

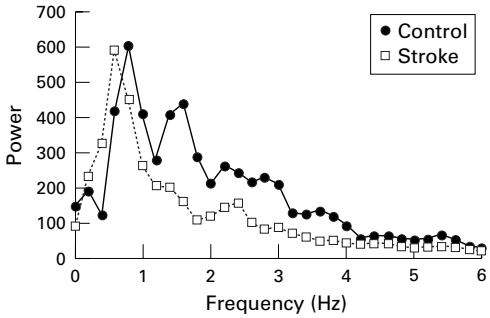

Figure 3 Mean fast Fourier transformation of ankle joint angle during walking for control subjects compared with stroke patients.

control subjects was significantly higher ($F(30,810)=5.4$, $p<0.001$). It can also be seen from figure 3 that control subjects demonstrate a second peak at about 1.6 Hz which is the first harmonic of the fundamental frequency of 0.8 Hz. This second peak is not present in the mean spectrum of the stroke patients due to variability across the subjects in the pattern of dorsiflexion/plantarflexion movements of the ankle.

TONIC STRETCH REFLEXES

The control subjects showed no evidence of resting tonic stretch reflexes at any frequency (fig 2A and 4A). There was no muscle activity time locked to the stretching cycle and the mean IEMG during relaxation did not vary significantly across the five conditions (no stretch and stretch at 0.5, 1.0, 1.5, and 2.0 Hz; $F(4,56)=0.53$, $p=0.71$). The stroke patients, on the other hand, often exhibited resting tonic stretch reflexes (fig 2C and 4A) especially, at the higher frequencies. At the lower frequencies, very few subjects exhibited reflexes (two at 0.5 Hz, five at 1 Hz) and the gain was very small (0.02 μV/° at 0.5 Hz, 0.09 μV/° at 1 Hz) whereas more subjects exhibited reflexes at the higher frequencies (eight at 1.5 Hz, nine at 2 Hz) and the gain was larger (0.19 μV/° at 1.5 Hz, 0.22 μV/° at 2 Hz).

The control subjects exhibited clear action tonic stretch reflexes (fig 2B and 4B) in 49 of 60 trials (82%) with a gain in the order of 0.63 μV/°. Similar to the control subjects, the stroke patients exhibited action tonic stretch reflexes (fig 2D and 4B) in 41 of 48 trials (85%) with a gain in the order of 0.4 μV/° which was smaller than the control subjects but not significantly so ($F(1,25)=3.10$, $p=0.09$, fig 4B). It is known that the gain of the action tonic stretch reflex is related to the magnitude of background contraction.[23] The subjects were required to maintain a background contraction of 10% of IEMG of the gastrocnemius activity produced when balancing on the ball of one foot. When the stroke patients balanced on the ball of one foot, they exhibited significantly ($t=3.23$, df=27, $p<0.05$) less muscle activity (68 (SD 33) μV IEMG) than the control subjects (127 (SD 60) μV IEMG). Therefore, they were required to maintain less background muscle activity during the active stretch. However, when an analysis of covariance with "IEMG when balancing on the ball of one foot" as a covariate was performed, no significant difference was found between the gain of the action tonic stretch reflexes in stroke and control subjects ($F(1,24)=0.46$, $p=0.51$, fig 4C).

RESISTANCE TO STRETCH

When stretch was applied to the relaxed gastrocnemius muscle, stroke patients exhibited significantly ($F(1,25)=9.08$, $p<0.05$) higher torque-angle gains (0.55 (SD 0.04) Nm/°) than the control subjects (0.36 (SD 0.03) Nm/°, fig 5A). The higher torque-angle gain after stroke may be due to the contribution of reflex responses to the resistance. However,

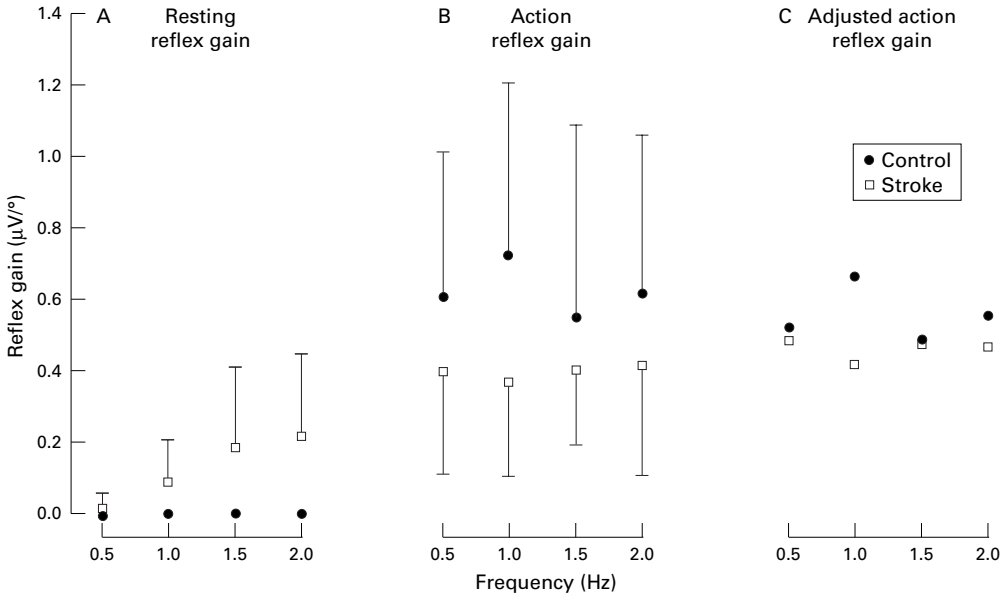

Figure 4 Mean reflex gain (SD) of tonic stretch reflexes during relaxed and active stretch for the control subjects compared with stroke patients. (A) Reflex gain during relaxed stretch showing small responses by stroke patients and no response by the control subjects. (B) Reflex gain during active stretch showing larger responses by the control subjects compared with stroke patients. (C) Reflex gain during active stretch adjusted by "IEMG when balancing on the ball of one foot" showing no significant difference in responses between control subjects and stroke patients.

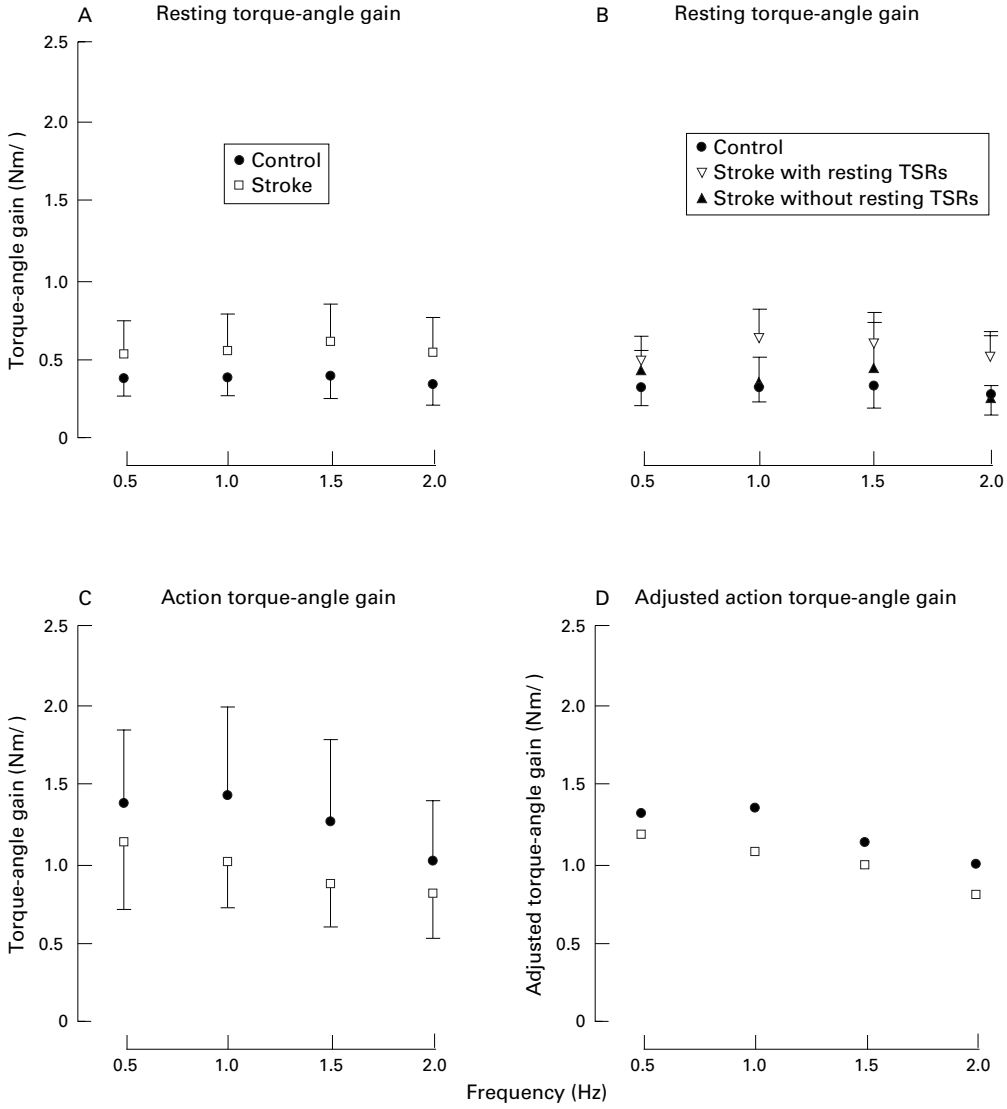

Figure 5 Mean torque-angle gain (SD) during relaxed and active stretch for control subjects compared with that for stroke patients. (A) Torque-angle gain during relaxed stretch for all stroke patients showing higher responses compared with that for the control subjects. (B) Torque-angle gain during relaxed stretch for stroke patients with reflexes showing higher responses than that for patients without reflexes or the control subjects. (C) Torque-angle gain during active stretch showing higher responses for the control subjects than for stroke patients. (D) Torque-angle gain during active stretch adjusted by the "gain of the action tonic stretch reflex" (TSR) showing no significant difference between stroke patients and control subjects.

not all of the stroke patients exhibited abnormal tonic stretch reflexes. Therefore, for each frequency the stroke patients were separated into those with and those without resting tonic stretch reflexes. The torque-angle gain for the stroke patients without abnormal reflex responses was not different from the control subjects ($F(1,13)=0.01$, $p=0.92$) whereas those with abnormal reflexes had significantly higher torque-angle gains ($F(1,13)=9.97$, $p<0.01$, fig 5B). Therefore, it is possible to conclude that the abnormal reflex responses contributed to an increase in resistance under relaxed conditions.

When stretch was applied to the contracting gastrocnemius muscle, stroke patients exhibited significantly ($F(1,25)=4.72$, $p<0.05$) lower torque-angle gains (1 (SD 0.1) Nm/°) than the control subjects (1.3 (SD 0.2) Nm/°, fig 5C). However, the neural contributions to the torque-angle gain are the voluntary background activity and the reflex response. When the torque-angle gain was adjusted by performing an analysis of covariance with "mean IEMG" as a covariate, the action torque-angle gain in stroke patients was no different from that in control subjects ($F(1,24)=1.26$, $p=0.27$, fig 5 D). Multiple regression analysis indicated that, in the control subjects, reflex responses contributed 28% to the torque-angle gain whereas in stroke patients, reflex responses contributed only 4% to the torque-angle gain.

Discussion

The aim of this study was to investigate the contribution of tonic stretch reflexes in the gastrocnemius muscle to walking after stroke. Muscles were stretched under conditions that simulated walking as closely as possible but ankle movement was measured during walking to verify these conditions. The range of stretch was 20°, from 10° of plantarflexion to 10° of

dorsiflexion, which approximated the range of the control subjects very well (range 20.7° from 11.5° of plantarflexion to 9.2° of dorsiflexion). The stroke patients showed slightly less range of ankle movement during the single support phase of stance (13.5°). This reduction in range was not at the expense of dorsiflexion (control subjects=9.2°, stroke patients=8.5°), so that the maximum stretch of the gastrocnemius during walking (9.2° of dorsiflexion in control subjects, 8.5° of dorsiflexion in stroke patients) (at 9.2° or 8.5°) was much the same as during the experimental condition (at 10°). As expected, the stroke patients walked more slowly than the control subjects. This was reflected in a longer stride time (1.8 *v* 1.3 seconds), a lower periodicity (0.6 Hz *v* 0.8 Hz) and a narrower frequency range of ankle joint movement during walking (2.3 Hz v 2.8 Hz). The magnitude of stretch and the range of frequencies exhibited by the stroke patients during walking were adequately reproduced during measurement of the tonic stretch reflexes. Therefore, it is unlikely that abnormal reflex activity would have been elicited during walking by movement range or frequencies that were not tested in the experimental condition.

We found that during slow stretch of the relaxed gastrocnemius muscle, about two thirds of the stroke patients exhibited reflex activity, suggesting the presence of spasticity, and in line with previous studies, none of the control subjects did.[11 12 28–29] This supports the suggestion of Ibrahim *et al*[30] that patients with spastic paresis have a poor ability to switch off reflex responses under passive conditions. Furthermore, these reflexes contributed to a higher passive torque-angle gain suggesting that at least some of the clinical impression of an increased resistance to movement as measured by the Ashworth scale (table) was a result of abnormal reflex activity.

When stretch was applied to the contracting gastrocnemius muscle, both groups exhibited action tonic stretch reflexes over 80% of the time. Importantly, on average, stroke patients exhibited smaller rather than exaggerated reflex responses although this difference was not significant. Studies of the elbow flexors have also shown that under active contraction, reflex activity is reduced on the spastic side compared with the normal side.[30–32] Furthermore, as the stroke patients walked more slowly than the control subjects, the speed of rotation of the shank over the foot would have been reduced. In combination with the lack of increase of amplitude of action tonic stretch reflexes, this suggests that the likelihood of eliciting abnormal velocity sensitive stretch reflexes during walking would be decreased after stroke, not increased.

The clinical picture of spasticity in stroke patients is of increased resistance to passive movement caused by exaggerated tonic stretch reflex activity. Although clinical assessment is usually performed with the muscle relaxed, there is an assumption that any abnormal reflex activity will be exaggerated during the effort of active movement. However, our findings were that even when stroke patients exhibited abnormal tonic stretch reflex activity under relaxed conditions, their action tonic stretch reflex was not different from control subjects. We found that the reflex seemed to be "on" a small amount, regardless of test conditions. Rather than an abnormal, "out of control" reflex, this profile suggests that the reflex is not being modulated This is in agreement with the conclusion drawn by Berger *et al*[33] regarding the contribution of reflex activity to walking dysfunction after a stroke where the spastic leg was perturbed by applying a quick stretch to the calf muscles just after heel strike. Although this perturbation produced a large monosynaptic reflex, the polysynaptic reflex was reduced compared to normal. Furthermore, the authors concluded that even the exaggerated monosynaptic reflex response played a negligible part in the production of calf muscle tension. This is consistent with a study of phasic stretch reflexes in the upper limb which also found that spastic stroke patients displayed a "defective modulation" of the stretch reflex.[30] An inability to modulate the stretch reflex during walking has also been found in other neurological conditions such as head and spinal cord injury[34] and multiple sclerosis.[35 36]

It is possible that, even though the reflexes exhibited after stroke were small, they may have had an exaggerated ability to produce muscle stiffness. It has been suggested, for example, that a change in the active properties of the muscle after stroke results in an increased torque output for a given level of EMG activity.[30–32] In support of this, we found that the stroke patients exhibited half the amount of EMG activity compared with the control subjects when performing a task with the same force requirements (in this case, balancing on the ball of one foot). However, analysis showed that the contribution of the reflex to active stiffness was negligible in the stroke patients compared with the control subjects—that is, the reflexes of the stroke patients were ineffective at producing stiffness. In this population of patients, which included people from normal stiffness to high stiffness (table), there was no indication of the stretch reflex contributing to an increased resistance to stretch under active conditions that simulated those of walking.

Clearly, the most important methodological limitation of this study concerns the question of whether walking is mimicked adequately by our seated test conditions to draw conclusions about the behaviour of the tonic stretch reflex during walking. The seated position allows a stretch stimulus which is almost identical in range and frequency to that of the shank rotating over the foot in walking. Most importantly, repeated stretching allows the contribution of reflex muscle contraction to the stiffness of the ankle joint to be separated from the voluntary contribution. On the other hand, stretch reflexes have been shown to be modified for different tasks such as standing, walking, and running,[34] which suggests that there are neurophysiological events that are different in walking than in long sitting. The main finding about the behaviour of the tonic stretch reflex

from the present study is that, rather than a profound difference in tonic stretch reflex amplitude compared with control subjects, stroke patients cannot modulate their tonic stretch reflex differently under passive versus active conditions. It is, therefore, not unreasonable to assume that this impaired ability to modulate the tonic stretch reflex will also exist during walking.

In conclusion, during walking, when the ankle is dorsiflexing during single support and the gastrocnemius muscle is contracting eccentrically, it seems unlikely that there is an increase in resistance to dorsiflexion due to exaggerated reflex activity after stroke. The subjects in this study had had a stroke several months before testing and could walk independently. There was no specific inclusion criterion that the patients had to be clinically hypertonic and so the sample represents a typical group of ambulant stroke patients. Therefore, it seems inappropriate to routinely reduce or inhibit the reflex response, which may not be exaggerated in stroke patients, to improve functional movement in stroke rehabilitation. There is a growing body of evidence that problems in resistance to movement after stroke may be caused by factors other than spasticity. For example, recently both Davies *et al*[37] and O'Dwyer *et al*[38] have found a mechanical rather than a reflex cause of increased resistance after stroke. Therefore, factors such as contracture need to be considered so that appropriate treatment is provided to stroke patients.

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1 Kottke FT. Neurophysiologic therapy for stroke. In: Licht S, ed. *Stroke and its rehabilitation*. Baltimore: Waverl, 1975:317.
2 Bobath R. *Adult hemiplegia: evaluation and treatment.* 3rd ed. London: Heinemann, 1993
3 Chin P-L, Rosie A, Irving M, *et al*. Studies in hemiplegic gait. In: Rose FC, ed. *Advances in stroke therapy*. New York: Raven Press, 1982:197–211.
4 Olney SJ, Richards C. Hemiparetic gait following stroke. Part I: characteristics. *Gait and Posture* 1996;**4**:136–48.
5 Brunnstrom S. Recording gait patterns of adult hemiplegic patients. *Journal of the American Physical Therapy Association* 1964;**44**:11–8.
6 Hesse S, Krajnik J, Luecke D, *et al*. Ankle muscle activity before and after botulinum toxin therapy for lower limb extensor spasticity in chronic hemiparetic patients. *Stroke* 1996;**27**:455–60.
7 Lance JW. Symposium synopsis. In: Feldman RG, Young RR, Koella WP, eds. *Spasticity: disordered motor control*. Miami: Symposia Specialists, 1980:485–94.
8 Winter DA, Yack HJ. EMG profiles during normal human walking: stride-to-stride and inter-subjects variability. *Electroencephalogr Clin Neurophysiol* 1987;**67**:402–11.
9 Dietz V, Berger W. Normal and impaired regulation of muscle stiffness in gait. A new hypothesis about muscle hypertonia. *Exp Neurol* 1983;**79**:680–7.
10 Neilson PD, Lance JW. Reflex transmission characteristics during voluntary activity in normal man and patients with movement disorders. In: Desmedt J, ed. *Cerebral motor control in man: long loop mechanisms. Progress in clinical neurophysiology.* Vol 4. Basel: Karger, 1978:263–99.
11 Rack PMH, Ross HF, Thilmann AF. The ankle stretch reflexes in normal and spastic subjects. *Brain* 1984;**107**: 637–54.
12 Hufschmidt A, Mauritz K-H. Chronic transformation of muscle in spasticity: a peripheral contribution to increased tone. *J Neurol Neurosurg Psychiatry* 1985;**48**:676–85.
13 Burke D. Spasticity as an adaptation to pyramidal tract injury. In: Waxman SG, ed. *Functional recovery in neurological disease. Advances in neurology*. New York: Raven Press, 1988;**47**:401–23.
14 Neilson PD. Tonic stretch reflex in normal subjects and in cerebral palsy. In: Gandevia S, Burke D, Anthony M, eds. *Science and practice in clinical neurology*. Cambridge: Cambridge University Press, 1993:169–90.
15 Neilson PD. Frequency-response characteristics of the tonic stretch reflexes of biceps brachii muscle in intact man. *Medical and Biological Engineering* 1972;**10**:460–72.
16 Neilson PD, Andrews CJ. Comparison of the tonic stretch reflex in athetotic patients during rest and voluntary activity. *J Neurol Neurosurg Psychiatry* 1973;**36**:547–54.
17 Carr JH, Shepherd RB, Nordholm L, *et al*. Investigation of a new motor assessment scale for stroke. *Phys Ther* 1985;**65**: 175–80.
18 Ashworth B. Preliminary trial of carisoprodal in multiple sclerosis. *Practitioner* 1964;**192**:540–2.
19 DeJong RN. Case taking and the neurologic examination. In: Baker AB, Baker LH, eds. *Clinical neurology.* Vol 1. Philadelphia: Haeper and Row, 1984:49.
20 Perry J. *Gait analysis. Normal and pathological function*. New York: McGraw-Hill, 1992.
21 Perry J. Determinants of muscle function in the spastic lower extremity. *Clin Orthop* 1993;**188**:10–26.
22 Gottlieb GL, Agarwal GC, Penn R. Sinusoidal oscillation of the ankle as a means of evaluating the spastic patient. *J Neurol Neurosurg Psychiatry* 1978;**41**:32–9.
23 Neilson PD, McCaughey J. Effect of contraction level and magnitude of stretch on tonic stretch reflex transmission characteristics. *J Neurol Neurosurg Psychiatry* 1981;**44**: 1007–12.
24 Bennett DJ. Stretch reflex responses in the human elbow joint during a voluntary movement. *J Physiol (Lond)* 1994; **474**:339–51.
25 McRuer DT, Krendel ES. The human operator as a servo element. *Journal of the Franklin Institute* 1959;**267**:381–403,511–36.
26 Neilson PD. Speed of response or bandwidth of voluntary system controlling eblow position in intact man. *Medical and Biological Engineering* 1972;**10**:450–9.
27 Winer BJ. *Statistical principle in experimental design.* 2nd ed. New York: McGraw-Hill, 1971.
28 Evans CM, Fellows SJ, Rack PMH, *et al*. Response of the normal human ankle joint to imposed sinusoidal movements. *J Physiol (Lond)* 1983;**344**:483–502.
29 ReberŠek S, Stefanovska A, Vodovnik L, *et al*. Some properties of spastic ankle joint muscles in hemiplegia. *Med Biol Eng Comput* 1986;**24**:19–26.
30 Ibrahim IK, Berger W, Trippel M, *et al*. Stretch-induced electromyographic activity and torque in spastic elbow muscles. Differential modulation of reflex activity in passive and active motor tasks. *Brain* 1993;**116**:971–89.
31 Lee WA, Boughton A, Rymer WZ. Absence of stretch reflex gain enhancement in voluntarily activated spastic muscle. *Exp Neurol* 1987;**98**:317–35.
32 Dietz V, Trippel M, Berger W. Reflex activity and muscle tone during elbow movements in patients with spastic paresis. *Ann Neurol* 1991;**30**:767–78.
33 Berger W, Horstmann G, Dietz V. Tension development and muscle activation in the leg during gait in spastic hemiparesis: independence of muscle hypertonia and exaggerated stretch reflexes. *J Neurol Neurosurg Psychiatry* 1984;**47**:1029–33.
34 Stein RB, Yang JF, Bélanger M, Pearson KG. Modification of reflexes in normal and abnormal movements. In: Allum JHJ, Allum-Mecklenburg DJ, Harris FP, *et al*, eds. *Progress in brain research*. Vol 97. Amsterdam: Elsevier, 1993:189–96.
35 Sinkjær T, Toft E, Hansen HJ. H-reflex modulation during gait in multiple sclerosis patients with spasticity. *Acta Neurol Scand* 1995;**91**:239–46.
36 Sinkjær T, Andersen JB, Nielsen JF. Impaired stretch reflex and joint torque modulation during spastic gait in multiple sclerosis patients. *J Neurol* 1996;**243**:566–74.
37 Davies JM, Mayston MJ, Newham DJ. Electrical and mechanical output of the knee muscles during isometric and isokinetic activity in stroke and healthy adults. *Disabil Rehabil* 1996;**18**:83–90.
38 O'Dwyer NJ, Ada L, Neilson PD. Spasticity and muscle contracture following stroke. *Brain* 1996;**119**:1737–49.