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Spasticity and muscle contracture following stroke

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Summary

It has become increasingly recognized that the major functional deficits following brain damage are largely due to 'negative' features such as weakness and loss of dexterity rather than spasticity. A variety of studies suggest that spasticity is a distinct problem and separate from the loss of dexterity, but that it may be implicated in the formation of muscle contracture and even in the recovery of strength. In order to address these issues, we examined the relationship between spasticity, contracture, strength and dexterity in the affected upper limb following stroke. Spasticity was measured both as increased tonic stretch reflexes and increased resistance to passive stretch (hypertonia). Twenty-four patients were recruited non-selectively from three rehabilitation units within 13 months of their stroke. Few patients exhibited increased tonic reflexes but half were found to have muscle contracture, the earliest at 2 months following stroke. Hypertonia was associated with contracture but not with reflex hyperexcitability. Increased tonic stretch reflexes were observed only in a subgroup of those with contracture and where present could usually be elicited only at the end of muscle range. This finding suggests that instead of spasticity causing contracture, contracture may actually potentiate spasticity in some patients. However, the majority of patients with contracture did not have increased tonic stretch reflexes. In addition, we found no relationship between spasticity and either weakness or loss of dexterity. Therefore, while hypertonia remains an important problem following cerebral lesions, it would appear that the amount of attention directed to reflex hyperexcitability associated with spasticity is out of proportion with its effects. Consequently, hypertonia needs to be clearly distinguished from reflex hyperexcitability in patients with spasticity.

Keywords: spasticity; muscle contracture; stroke; reflex hyperexcitability; hypertonia

Abbreviations: IEMG = rectified and low-pass filtered EMG; MAS = Motor Assessment Scale; MCA = middle cerebral artery

Introduction

It has been traditional to characterize the signs of brain damage as either 'positive', i.e. those features that are not normally present such as spasticity and abnormal postures, or 'negative', i.e. those features that have been lost such as strength and dexterity. Over the past 20 years, it has been increasingly recognized that the major functional deficits following brain damage are largely due to the negative features (e.g. Landau, 1974, 1988; Burke, 1988). However, patients often develop secondary complications such as muscle contracture. These secondary complications may in turn interfere with the recovery of function. The present study examines the relationship between spasticity, muscle contracture, strength and dexterity across a population of patients during recovery of function following stroke.

Much effort has been directed at reducing spasticity as part of the treatment and rehabilitation of brain-damaged patients. This has stemmed from the historical view that spasticity was the major determinant of motor dysfunction. One of the first investigators to question this view was Landau (1974) and since then a variety of experiments have supported his position. The historical view would suggest that inhibition of spasticity should result in an improvement in function. However, when hyperactive reflexes have been suppressed with drugs, in people with either stroke (McLellan, 1977) or cerebral palsy (Nathan, 1969), there has been no parallel increase in movement control. Similarly, when adults and children with cerebral palsy have learnt to reduce spasticity following training, it has not lead to an improvement in voluntary control of movement (Neilson and McCaughey, 1982). In addition, Sahrmann and Norton (1977) demonstrated that impairment of movement following stroke is not primarily due to reflexes in the spastic antagonist muscles

but to abnormalities of agonist contraction. Abnormal motor unit firing patterns have also been documented in the muscles of spastic patients (e.g. Rosenfalck and Andreassen, 1980; Young and Wierzbicka, 1985; Farmer *et al.*, 1993). Notwithstanding these findings, the continued interest in mechanisms of and therapeutic interventions for spasticity suggests that it retains a focus that is out of step with its theoretical importance.

Some of the confusion about the role of spasticity in movement dysfunction has probably arisen because the clinical measurement of spasticity involves gauging the resistance of the limbs to passive movement. This procedure does not allow different causes of an increase in resistance to be identified. Historically, such an increase has been assumed to be due to exaggerated stretch reflexes, but Dietz *et al.* (1981) provided evidence that altered mechanical properties of muscle may contribute to hypertonia in spastic patients. Perry (1980) was one of the first researchers to document the clinical observation that spasticity usually presented in conjunction with muscle contracture. Halar *et al.* (1978) demonstrated muscle shortening in the lower limb in stroke patients with clinical contracture and this was accompanied by increased passive stiffness of the ankle. Other investigators also have demonstrated increased passive ankle stiffness in spastic patients (Gottlieb *et al.*, 1978; Dietz and Berger, 1983), both with (Hufschmidt and Mauritz, 1985) and without (Thilmann *et al.*, 1991*b*) clinical signs of contracture. A similar increase in joint stiffness, also attributable to passive soft tissue changes, has been observed in the lower limb of spastic cerebral-palsied children and adults with contracture (Tardieu *et al.*, 1982*a*; O'Dwyer *et al.*, 1994). Indirect evidence for altered mechanical properties of upper limb muscles in spastic patients has also been presented (Lee *et al.*, 1987; Dietz *et al.*, 1991). However, despite these clinical and experimental observations, the nature of the relationship between spasticity and contracture remains unresolved.

Spasticity has not always been seen in a purely negative light. Spastic hypertonia has been considered to be superior to a flaccid paresis (Hufschmidt and Mauritz, 1985) and Berger *et al.* (1984) suggested that the hypertonicity of leg extensor muscles enables hemiparetic patients to support their body during locomotion. According to Dietz *et al.* (1986), the mechanism underlying this ability may lie in the alterations of active biomechanical properties of muscle fibres implied by histochemical changes in spastic muscle (Edström, 1970; Dietz *et al.*, 1986). Both Twitchell (1951) and Brunnstrom (1970) have noted that, during recovery following hemiplegia, muscle stretch reflexes return before volitional movement and it has been shown in stroke patients that stretch-evoked reflex activity can augment voluntary muscle activity (Norton and Sahrmann, 1978). According to the 'servo-assistance' hypothesis of Matthews (1972), voluntary muscle activation is normally augmented by reflex afferents and this notion has been supported by recent studies in humans showing that muscle afferents provide a net facilitation to the motoneuron pool, reflexly increasing motor output at all levels of voluntary drive by approximately one-third (e.g. Gandevia *et al.*, 1990). Such reflex augmentation of voluntary muscle activity could be even greater in the presence of spasticity, whether due to lowered reflex threshold (Katz and Rymer, 1989) or increased reflex gain (Thilmann *et al.*, 1991*a*). It is possible, therefore, that spasticity may be positively related to strength during recovery of function following brain damage.

Taken together, these studies suggest that spasticity is a distinct and separate problem to the loss of dexterity which follows brain damage, but that it may be implicated in the formation of muscle contracture and even in the recovery of strength. It should be noted, however, that the findings outlined above include studies of congenital as well as acquired brain damage. In the present study we direct our attention to hemiparesis following stroke. We measured spasticity, contracture, strength and dexterity in order to examine the relations between them in 24 patients who were within 1 year of their stroke. It has recently been shown that the severity of motor impairment and the patterns of motor recovery are similar for the upper and lower limbs following stroke (Duncan *et al.*, 1994). The affected upper limb was studied here, specifically the elbow flexor muscles, because clinical (Ada and Canning, 1990) and experimental (Lee *et al.*, 1987; Dietz *et al.*, 1991; Thilmann *et al.*, 1991*a*) observations suggest that they are a common site for the development of both contracture and spasticity. Furthermore, contrary to earlier clinical impressions, the elbow flexor muscles have been found to be relatively more weakened than the extensors (Colebatch *et al.*, 1986).

Methods

Subjects

In earlier studies of stroke-induced hemiparesis, patients were selected on the basis of clinically detectable spasticity (e.g. Lee *et al.*, 1987; Powers *et al.*, 1988, 1989; Dietz *et al.*, 1991; Thilmann *et al.*, 1991*a*; Katz *et al.*, 1992; Ibrahim *et al.*, 1993*a*). In the present study, we wished to study the relationships between spasticity and several other variables in a group which was representative of the stroke population undergoing rehabilitation. Therefore, we tested all hemiparetic patients, both in-patients and out-patients, in three metropolitan rehabilitation units during a 1-month period. Subjects were only excluded if they had such severe language, perceptual or cognitive deficits that they were unable to follow the instructions required to participate in the study. Since most of the procedures did not involve active participation, this excluded very few patients. Spasticity is a secondary adaptation to upper motor neuron lesions (Burke, 1988) that requires time to develop (Brown, 1994) and similar considerations apply to muscle contracture. Therefore, we did not accept patients earlier than 1 month post-stroke. This process yielded 24 subjects

Table 1 *Subject characteristics*

Subject	Age (years)	Gender	Side of hemiparesis	Time since stroke (months)	Site of lesion (on CT scan)	MAS item 6 (0–6)
1	87	M	R	2	Brainstem	3
2*	74	F	L	2.5	(R) MCA territory	1
3	79	M	L	3	(R) Cerebral penduncle	6
4	51	M	L	1	(R) Frontoparietal area	1
5	58	F	R	2.5	(L) Frontal lobe	4
6	61	M	L	2.5	(R) Frontoparietal area	0
7*	58	F	R	3	(L) Frontal lobe	0
8	62	F	R	7	(L) Basal ganglia	0
9	78	M	L	6	(R) Basal ganglia	1
10	36	F	L	12	(R) Parietal and basal ganglia	2
11*	65	M	R	6.5	(L) Internal capsule	1
12	71	F	R	7.5	(L) Basal ganglia	1
13	42	F	R	2.5	(L) Basal ganglia	6
14*	78	F	R	2	(L) Internal capsule	0
15	74	F	L	5	(R) Internal capsule	6
16	54	M	R	3.5	(L) Basal ganglia haemorrhage	2
17	74	M	R	7	(L) Posterior external capsule	6
18	64	F	R	6	(L) Internal capsule	1
19	67	M	R	8	(L) Frontoparietal area	0
20	40	M	L	8	(R) Frontoparietal area	1
21	40	M	L	13	(R) MCA territory	0
22	72	F	R	7	(L) Pontomedullary area	5
23*	65	M	L	7.5	(R) Frontoparietal area	3
24	52	M	L	2	(R) MCA territory	0

Subjects were all found to be right-handed. MCA = middle cerebral artery. *Subjects with hyperexcitable tonic stretch reflexes.

with a wide range of characteristics (*see* Table 1), all of whom had suffered a stroke within the last year. Their functional ability is indicated in Table 1 by scores between 0 and 6 on the upper arm category (item 6) of a clinical scale, the Motor Assessment Scale (MAS) (Carr *et al.*, 1985), and it can be seen that the degree of impairment in the group ranged from mild to severe. The experimental procedures were approved by the relevant institutional ethics committee and all subjects gave informed consent before data collection was undertaken.

Experimental set-up

The equipment measured elbow joint displacement, torque and biceps muscle activity (Fig. 1). The subject sat at a table with the affected forearm securely supported by a horizontal frame. Rotation of the frame, whether by the experimenter or the subject, produced a change in elbow angle that was measured by a potentiometer aligned directly below the elbow joint. A load cell (capacity 250 N; linearity 97%) attached to the frame measured the resistance of the forearm to movement. Silver silver-chloride surface electrodes measured biceps muscle activity.

After amplification of the EMG activity (×5000) and torque (×1000), the elbow angle, torque and EMG activity were sampled by a 16-bit analog to digital converter at 1000 Hz and stored on computer. In order to remove any 50 Hz line frequency interference or low-frequency movement artefact, the EMG was high-pass filtered (digital 8th-order Butterworth) at 80 Hz. Subsequently, the EMG activity was full-wave rectified and then, along with the elbow angle and torque signals, low-pass filtered (digital 8th-order Butterworth) at 5 Hz to obtain a DC voltage. This rectified and low-pass filtered EMG (IEMG) was proportional to the contraction level of the muscle. The cut-off frequency was chosen because all frequencies of interest were <5 Hz.

Measurements of spasticity and one measure of dexterity were carried out using this set-up. The procedures for collection of EMG activity were standardized between subjects to promote reliability. For example, for each subject the electrode placement was the same relative to the muscle belly and the impedance was kept as low as possible by thorough abrading and cleaning of the skin. In addition, the same experimenters always carried out the same part of the procedures.

Definition and measurement of spasticity

The most widely accepted definition of spasticity describes it as 'a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes ('muscle tone') with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the upper motor neuron syndrome' (Lance, 1980). The increased stretch reflexes are assumed to cause hypertonia, i.e. increased resistance to passive movement. Following stroke, however, increased resistance to passive movement may be the result of altered passive mechanical properties of muscle tissue as

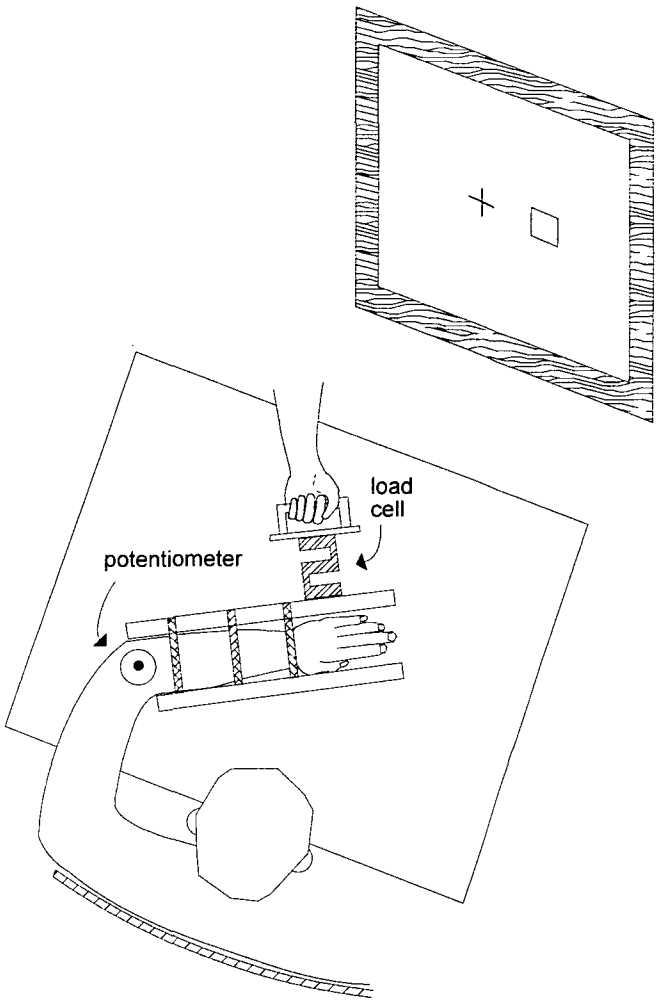

Fig. 1 Experimental set-up. The arm-frame could be clamped to measure isometric strength or could move freely so that either the subject could track the target (square) on the computer screen or the experimenter could stretch the biceps. A high-backed chair supported the subject so that when the arm was resting in the arm-frame, movement was confined to the elbow joint.

well as hyperexcitable reflexes. Therefore, we measured both the stretch-evoked muscle activity via EMG activity (i.e. reflex hyperexcitability) and the resistance to passive stretch via the load cell (i.e. hypertonia).

We chose to measure the excitability of the tonic rather than the phasic stretch reflex, since it is generally recognized that the tonic stretch reflex is of greater physiological and clinical significance (e.g. Lance *et al.*, 1966; Landau, 1974; Neilson, 1993). Reflex hyperexcitability was assessed at two muscle lengths and velocities since the gain of the tonic stretch reflex has been found to vary with changes in these characteristics of stretch (Neilson and McCaughey, 1981; Nash *et al.*, 1989). We stretched the muscles by performing small amplitude (10° peak-to-peak) quasi-sinusoidal stretching for 30 s at frequencies of 2 and 3.5 Hz, producing peak velocities of 60° s^{-1} and 110° s^{-1}, respectively. These frequencies are in the realm of normal movement but are too fast for consistent voluntary following (Neilson 1972), particularly for hemiparetic patients. The stretching was performed at two muscle lengths; the elbow was flexed at 90° or at 20° from full extension. The latter position near the end of the muscle range was designed to gauge the effect of any muscle contracture that might be present. Therefore, three conditions were tested: 90°±5° at 2 Hz, 90°±5° at 3.5 Hz, 20°±5° at 2 Hz.

Subjects relaxed, as confirmed by the absence of EMG activity, and then the forearm was manually rotated back and forth about the elbow. The rotation was synchronized with a metronome in order to control the frequency. The elbow angle was displayed on the computer monitor so that the amplitude of stretch could be controlled. The consistency of the imposed stretch, as measured by spectral analysis, was kept high both for frequency (mean±SD = 2±0.1 Hz and 3.5±0.1 Hz) and amplitude (13°±1°) (*see* top traces in Fig. 2). Normally, no EMG activity is observed when the relaxed biceps muscles of a neurologically-normal person are stretched in this manner and at these velocities (e.g. Burke, 1983; Ibrahim *et al.*, 1993*a*) and we have recently validated this observation in normal elderly subjects (W. Yeo, L. Ada, N. J. O'Dwyer and P. D. Neilson, unpublished observations). Therefore, any stretch-induced EMG activity observed was taken as evidence of reflex hyperexcitability. This procedure makes no assumption regarding whether such EMG activity is due to lowering of reflex threshold (Katz and Rymer, 1989), increase in reflex gain (Thilmann *et al.*, 1991*a*) or more complex changes in modulation of reflex threshold or gain (Neilson and Lance, 1978; Gottlieb and Myklebust, 1993).

The angle and IEMG signals were subjected to cross correlational and spectral analysis (McRuer and Krendel, 1959; Neilson, 1972) to quantify any tonic stretch reflexes present. This analysis allows stretch-evoked muscle activity at the stretching frequencies of 2 and 3.5 Hz to be distinguished from other activity unrelated to the stretch. The magnitude of these stretch reflexes reflects the degree of reflex hyperexcitability and was quantified by the gain of the tonic stretch reflex, i.e. by the magnitude of the stretch-evoked IEMG activity divided by the magnitude of stretch. If stretch reflexes of any reasonable magnitude are present, the mean level of IEMG activity would also be expected to increase during the stretching procedure. Therefore, in addition to the gain of the reflex, the mean level of IEMG activity during stretch was measured and compared with that during rest. The angle and torque signals were also subjected to cross correlational and spectral analysis and the resistance to passive movement was quantified by the gain of the torque-angle relationship, i.e. by the magnitude of the stretch-evoked torque divided by the magnitude of the stretch. Figure 2 provides examples of angle, torque, EMG and IEMG signals for subjects with and without tonic stretch reflexes.

Definition and measurement of contracture

The number of sarcomeres in a muscle is not fixed, being capable of either increasing or decreasing even in adult

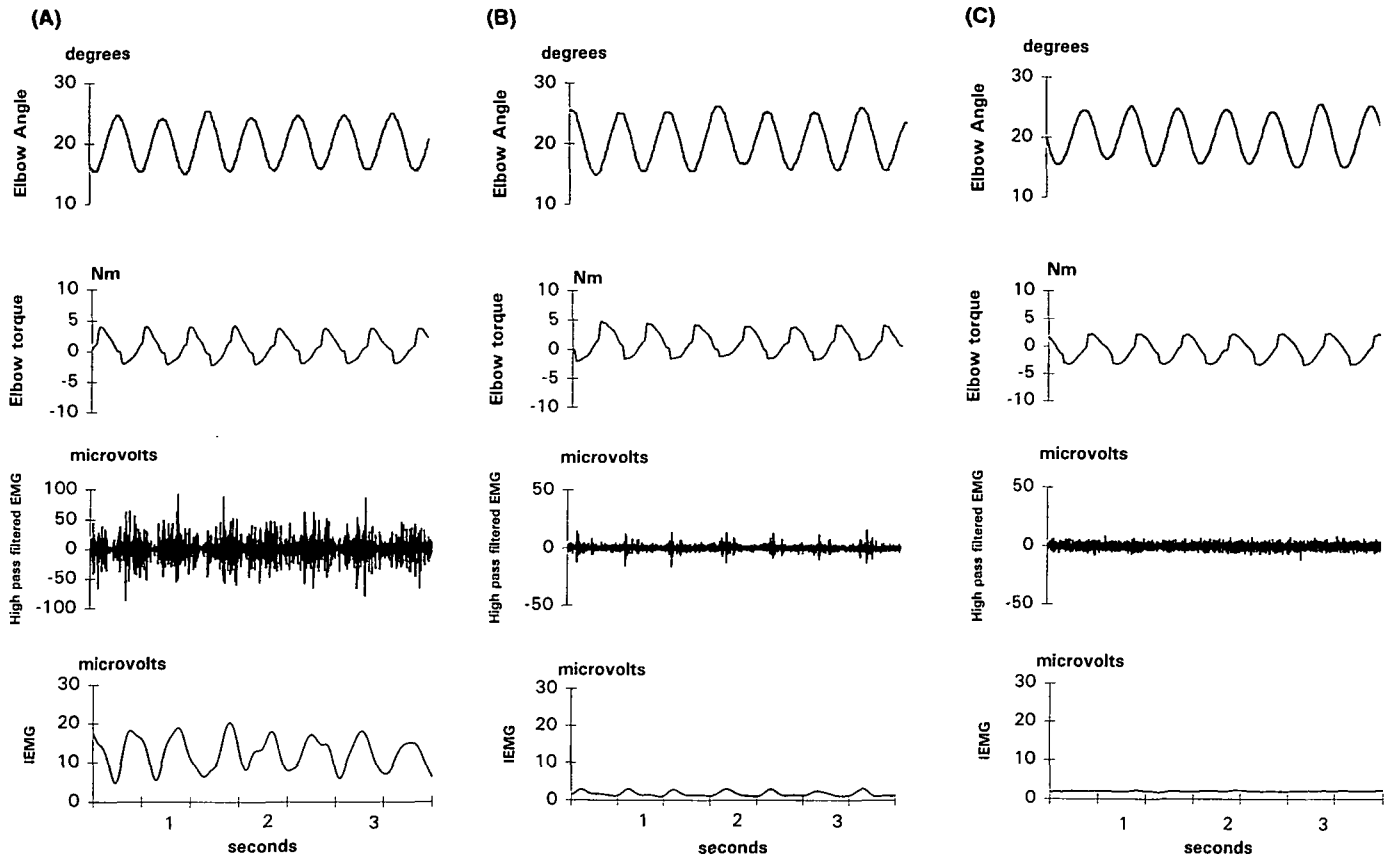

Fig. 2 Responses to passive stretch of the biceps at 20° at 2 Hz. Traces of elbow angle, torque, biceps EMG and IEMG. (**A**) Subject 14 who showed the largest reflex activity in response to stretch under all conditions. The bursts of muscle activity are coherent with the stretch (coherence 0.96) and have a phase lead (70°) showing their velocity dependence. (**B**) Subject 11 showing small but similar abnormal stretch reflex responses (coherence 0.99) and phase lead (88°). (**C**) Subject 6 showing no response to the stretch of biceps (coherence only 0.17). The distortion in the torque signal is due to a small amount of friction in the joint of the arm-frame. It contributed <1% of the power in the torque signal at the stretching frequency and so had negligible impact on the torque-angle gain.

muscle (Tabary *et al.*, 1972; Williams and Goldspink, 1973). Contracture consists of a shortening of muscle length due to a decrease in the number of sarcomeres in series along the myofibrils, accompanied by an increase in the resistance to passive stretch (Tardieu *et al.*, 1982*a*; Bax and Brown, 1985; O'Dwyer *et al.*, 1989). Muscle fibres are not lost or replaced by connective tissue, as is often assumed (e.g. Lee *et al.*, 1987). Tardieu *et al.* (1982*a*) reported that in contracture in cerebral palsy, muscle structure on light and electron microscopy was normal apart from reduction of fibre length and no excessive connective tissue was observed. The reduced compliance is probably attributable to remodelling of muscle connective tissue (O'Dwyer *et al.*, 1989; Goldspink and Williams, 1990). The range of joint motion, therefore, is reduced both by the shortening of the muscle fibres and by the loss of muscle compliance (Williams, 1988).

Despite the reduction in muscle compliance, if a contracture is minor in extent it may still be possible to achieve a normal range of motion by the application of sufficient force. For example, Halar *et al.* (1978) applied a force of 40 lbs (178 N) and achieved similar magnitudes of ankle dorsiflexion on the affected and unaffected sides of hemiplegic patients, even in the presence of clinical contracture. Consequently, in order to assess the magnitude of joint motion, it is important not only to standardize the force applied but also not to exceed the magnitude of force that is normally sufficient to stretch the muscles through the joint range. In addition, if a multi-joint muscle is being assessed (as is the case of the biceps brachii which crosses both the elbow and shoulder joints), it is important to standardize the position of the joint not being measured. It is not easy to apply these controls in the clinical assessment of muscle contracture, yet without them comparison cannot be made across subjects or with the normal population. In the present study, the subjects lay supine with their upper arm resting horizontally on a firm bed, thereby placing the shoulder in neutral. The elbow joint was extended firmly by the experimenter and held in this position for 30 s so as to allow time for relaxation in case muscle activity was elicited by the manoeuvre. Then the arm was released and maintained in extension solely by the weight of the forearm due to gravity. Selective EMG activity monitoring confirmed that the elbow flexor muscles were relaxed in this posture. Normally the forearm will lie flat on the bed under these conditions. The position of the arm was

photographed and contracture of the elbow flexor muscles was quantified by measuring the angle of the forearm relative to the bed from the photograph. The greater the degree of flexor contracture, the greater the angle. It should be noted, however, that the biceps brachii in this posture is still not fully lengthened across the shoulder joint, so that a forearm flexion measurement of 0° does not entirely rule out a small contracture. Therefore, this procedure underestimates the true extent and frequency of elbow flexor contracture.

Definition and measurement of strength

Since the elbow flexors were the muscles of major interest in this study, strength was measured during a maximal isometric flexor contraction of the elbow, with the arm-frame fixed at 90°. Both flexor torque and IEMG were collected and these two measures were subsequently found to be significantly correlated ($r = 0.56$; $P<0.01$). It is likely that the torque was influenced to a variable degree by cocontraction of the extensors and since the flexor IEMG provided an unambiguous estimate of the patients' ability to voluntarily activate the muscles, we chose to present this as the measure of strength.

The subjects were required to relax for 5 s, pull into flexion maximally for 5 s and relax again for 5 s. During this procedure the subject was provided with visual feedback from the display, since this has been shown to improve the achievement of maximal output (Jones *et al.*, 1979). The best of three attempts was taken to represent the subject's maximum. Flexor IEMG activity was averaged over the rest and contraction periods and the difference between them taken as maximal voluntary effort.

Definition and measurement of dexterity

Dexterity is adroitness or skill in using the body and it is therefore difficult to assess comprehensively. On the one hand, general measures of everyday tasks which require co-ordination of limb synergies tend to obscure the role of individual muscles. On the other hand, measures of specific muscles can be criticized for not being relevant to general function. We have, therefore, attempted to measure both levels of dexterity.

The overall ability to use the upper arm was measured using the MAS which provided a measure of motor function related to everyday tasks. Scores are assigned from 0 to 6, where 0 represents no activity and 6 is the highest score possible. Item 6 measures upper limb function and includes tasks such as 'raising the arm to shoulder height and holding for 10 s'. The scale has been shown to be reliable when used by a trained tester (Carr *et al.*, 1985), as it was during this study.

Specific dexterity of the elbow joint was assessed by requiring the subject to track the movements of a target on a computer screen using only elbow flexion and extension. The target moved irregularly back and forth across the computer screen. The subject sat at the table with the forearm supported in the arm-frame and controlled the response cursor via 10° of elbow flexion and extension (±5°) around a mean position of 90°, one of the positions at which reflex excitability was assessed. Following familiarization with the task, 1-min tests of a slow and fast target were recorded. The targets consisted of random numbers filtered (2nd-order Butterworth low-pass) at 0.25 Hz for the slow target and at 0.5 Hz for the fast target.

While performance of this task depended on coordinated control of the amplitude and timing of elbow flexor and extensor muscle activity, assessment of performance was based on the relationship between the target and the subject's response controlled by their elbow angle. A traditional measure of overall tracking performance is the root mean square value of the error, i.e. the difference between the target and the response signal (McRuer and Krendel, 1959). However, this becomes a less satisfactory measure of performance as the target moves faster and a significant time delay is introduced. Therefore, a more detailed cross-correlational and spectral analysis was carried out in order to assess the similarity of the target and response waveforms. This analysis provides a measure of the overall coherence between the target and response, i.e. the proportion of the response that is correlated with the target over the frequency bandwidth of the target. The coherence at each frequency is analogous to the r^2 measure in a regression analysis. For ideal tracking, the overall coherence would be one. Figure 3 provides examples of both slow and fast tracking from two subjects with different abilities.

Statistical analysis

The measurements of spasticity (reflex hyperexcitability and hypertonia), contracture, strength and dexterity yielded 11 variables for statistical analysis. Reflex hyperexcitability was represented by the gain of the tonic stretch reflex and hypertonia by the gain of the torque-angle relationship during stretching for the three conditions. Contracture was represented by the angle of elbow flexion. Assessment of strength yielded IEMG elbow flexor activity. General dexterity measured by the clinical scale yielded one variable, whereas specific dexterity was represented by the overall coherence in the slow and fast tracking conditions. The values of these variables for all subjects are presented in Table 2.

Most of the data were examined descriptively. Standard ANOVAs were used to examine (i) the difference in mean IEMG between rest and stretching, (ii) the difference in resistance to stretch between subjects with reflexes and those without, and (iii) the difference in resistance to stretch between subjects with contracture and those without. Finally, the relations between variables, including time since stroke, were analysed by Pearson's product moment correlation.

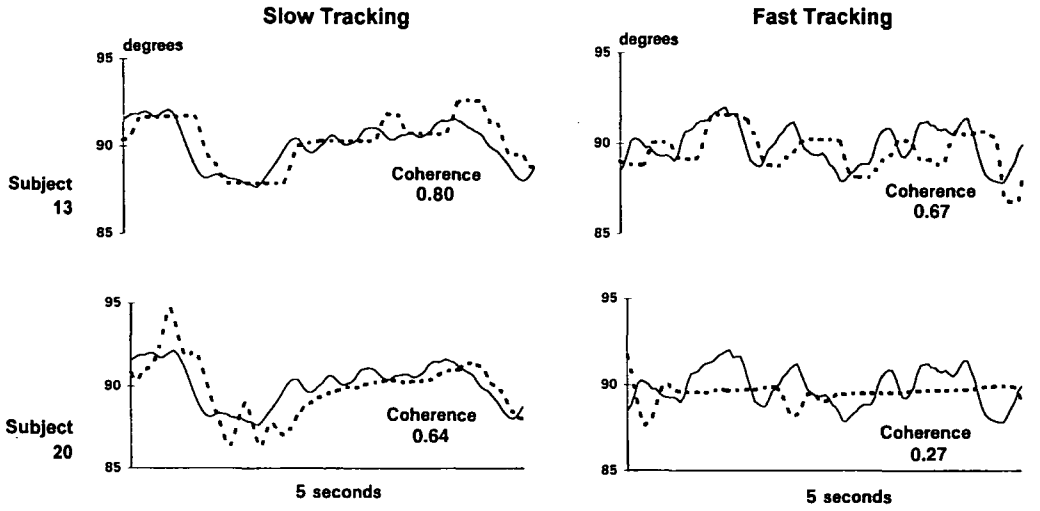

Fig. 3 Ten-second excerpts of tracking responses to the slow and fast targets by two subjects with differing levels of performance. The target is the solid line and the subject's response the dashed line. Both subjects track the slow target quite well, although Subject 20 (bottom traces) illustrates overshooting when trying to move the cursor back on target. Subject 13 (top traces) reproduces the faster target quite well but with a time delay, which is a normal feature, whereas Subject 20 reproduces very little of the fast target waveform, tending to 'freeze' or move with very little amplitude and a prolonged time delay.

Table 2 *Individual variables*

Subject	Time since stroke (months)	Contracture: elbow flexion (degrees)	Spasticity						Dexterity			Strength: elbow flexors IEMG (μV)
			Reflex excitability: gain of tonic stretch reflex			Hypertonia: resistance to passive movement			MAS: item 6 (0–6)	Slow track: overall coherence	Fast track: overall coherence	
			90°@2Hz (μV deg^{-1})	90°@3.5Hz (μV deg^{-1})	20°@2Hz (μV deg^{-1})	90°@2Hz (Nm deg^{-1})	90°@3.5Hz (Nm deg^{-1})	20°@2Hz (Nm deg^{-1})				
1	2.0	6	0.0	0.0	0.00	0.446	1.390	0.413	3	0.41	0.32	89
2	2.5	3	0.0	0.0	0.48	0.336	1.280	0.535	1	0.00	0.00	18
3	3.0	2	0.0	0.0	0.00	0.345	1.190	0.569	6	0.57	0.41	80
4	1.0	0	0.0	0.0	0.00	0.428	1.356	0.458	1	0.28	0.08	82
5	2.5	5	0.0	0.0	0.00	0.411	1.197	0.504	4	0.28	0.01	7
6	2.5	0	0.0	0.0	0.00	0.316	1.122	0.523	0	0.00	0.00	0
7	3.0	14	0.0	0.0	0.37	0.432	1.395	0.436	0	0.00	0.00	12
8	7.0	0	0.0	0.0	0.00	0.223	1.071	0.405	0	0.00	0.00	4
9	6.0	6	0.0	0.0	0.00	0.344	1.109	0.526	1	0.00	0.00	2
10	12.0	7	0.0	0.0	0.00	0.422	1.155	0.414	2	0.56	0.31	31
11	6.5	4	0.0	0.0	0.20	0.412	1.339	0.606	1	0.00	0.00	14
12	7.5	5	0.0	0.0	0.00	0.369	1.242	0.452	1	0.52	0.33	9
13	2.5	0	0.0	0.0	0.00	0.350	1.253	0.412	6	0.80	0.67	242
14	2.0	13	0.5	1.1	1.26	0.380	1.194	0.527	0	0.0	0.00	55
15	5.0	0	0.0	0.0	0.00	0.371	0.963	0.372	6	0.32	0.26	10
16	3.5	0	0.0	0.0	0.00	0.313	1.244	0.407	2	0.49	0.17	10
17	7.0	0	0.0	0.0	0.00	0.500	1.611	0.435	6	0.53	0.35	59
18	6.0	5	0.0	0.0	0.00	0.366	1.241	0.436	1	0.43	0.20	7
19	8.0	2	0.0	0.0	0.00	0.412	1.501	0.616	0	0.00	0.00	0
20	8.0	0	0.0	0.0	0.00	0.104	0.907	0.343	1	0.64	0.27	47
21	13.0	0	0.0	0.0	0.00	0.257	1.104	0.565	0	0.37	0.05	89
22	7.0	0	0.0	0.0	0.00	0.317	1.047	0.495	5	0.49	0.30	27
23	7.5	22	0.0	0.0	0.81	0.278	0.961	0.506	3	0.46	0.31	87
24	2.0	0	0.0	0.0	0.00	0.303	1.124	0.399	0	0.00	0.00	0
Mean	5.3	4				0.351	1.208	0.473		0.30	0.17	41
SD	3.1	5.4				0.083	0.17	0.074		0.25	0.18	53

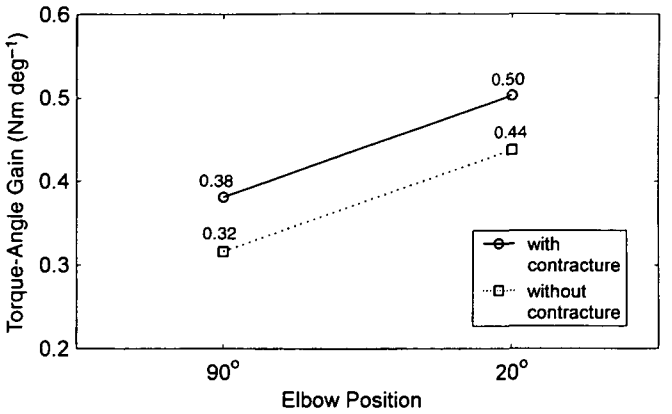

Fig. 4 Mean torque-angle gains (Nm deg^{-1}) at 2 Hz stretching at 90° and 20° elbow angle for subjects with and without contracture. Subjects with contracture show a higher resistance to passive stretch than subjects without contracture regardless of muscle length. As expected, for all subjects the resistance to passive movement increases at the 20° position near the end of range of joint excursion.

Results

Few subjects had demonstrable tonic stretch reflexes. Five subjects exhibited reflexes at 20°±5° at 2 Hz but only one exhibited reflexes in all three stretching conditions. Where present, the magnitude of this reflex activity was low; typically the bursts of EMG activity during stretching were in the range of ±50 μV amplitude. However, the change in mean IEMG from rest to stretching was significantly increased for those subjects with stretch reflex activity compared with those without [$F(1,22) = 7.8$; $P < 0.025$], although this was unlikely to be of functional significance since the increase was only from 4 to 8 μV of IEMG.

While very few subjects showed reflex activity, about half had a demonstrable contracture. Loss of range of elbow joint extension was observed in 13 of the 24 subjects, the size of the flexion contracture ranging from 2° to 22°. Furthermore, contracture was associated with an increased resistance to passive movement. As illustrated in Fig. 4, a significant increase in torque-angle gain at 2 Hz stretching was observed in subjects who had a contracture compared with those without [$F(1,22) = 9.03$; $P < 0.01$]. Not surprisingly, for all subjects, the resistance to stretch was significantly increased at 20° near the end of range of joint movement, compared with 90° close to the middle of the range [$F(1,22) = 33.85$; $P < 0.001$]. However, the size of this increase was not significantly different between subjects with and without contracture [$F(1,22) = 0.0005$; $P = 0.98$], i.e. in the presence of contracture, the increased stiffness was present throughout the joint range, both at 90° and 20°.

The increased resistance to passive stretch associated with contracture was independent of reflex hyperexcitability. Thus, the resistance was still significantly increased [$F(1,17) = 5.83$; $P < 0.05$] if the subjects with reflex hyperexcitability were excluded from the comparison. Furthermore, among the subjects with contracture, the presence of reflexes did not produce a significant increase in the resistance to stretch [$F(1,11) = 0.04$; $P = 0.84$]. Even though the stretch-evoked EMG activity was of a magnitude which increased the mean IEMG above that during relaxation, it did not increase the resistance beyond that due to contracture.

The correlations between the various measures are presented in Table 3. There were no significant correlations between the 'positive' and 'negative' features following stroke but there were significant correlations within these subgroups of features. Thus, the negative features, i.e. strength and the three measures of dexterity (MAS score and slow and fast tracking ability), were all significantly correlated. Only some of the positive features (contracture, reflex hyperexcitability and hypertonia) were correlated and most of these correlations were attributable to 'outlier' effects due to the fact that only one subject exhibited tonic stretch reflexes in the 90° stretching conditions. There was, however, one correlation of interest between tonic stretch reflexes at 20° and contracture ($r = 0.74$). This reflects the fact that all five subjects who had reflex hyperexcitability also had contracture. However, four of these subjects exhibited tonic stretch reflex activity only with the biceps in this lengthened position and not at 90°. Furthermore, another eight subjects had contracture but no reflex hyperexcitability. Finally, none of the variables measured correlated with the time since the stroke.

Discussion

Our original expectation that spasticity and contracture would be related was not supported by the findings of this study. Few tonic stretch reflexes were observed in response to passive stretch in this group of hemiparetic patients, even though half of them exhibited a contracture. Reflex activity was present in only seven out of 72 stretching trials and in only one patient under every stretching condition. This low occurrence of reflex hyperexcitability transpired despite the likely damage to corticofugal pathways in most patients (*see* Table 1) and the fact that many patients presented with the characteristic 'hemiplegic posture' of a slightly flexed elbow that is associated clinically with spasticity.

Other studies of hemiparetic stroke patients have reported reflex responses to relatively slow stretches comparable in duration (250 ms and 143 ms) and mean velocity (40°s^{-1} and 70°s^{-1}) with those employed in the present study (Powers *et al.*, 1988, 1989; Thilmann *et al.*, 1991*a*; Katz *et al.*, 1992). The only difference that might account for the discrepancy in findings would appear to be the amplitude of stretch, which was 10° in the present study compared with 30° or more in these earlier studies. Nevertheless, smaller stretches (12°), rapidly applied (60 ms, 200°s^{-1}), have been shown to elicit phasic reflexes in hemiparetic patients (Ibrahim *et al.*, 1993*a*). Perhaps more important than the parameters of stretch are the subject characteristics. The subjects in earlier studies usually had clinically manifest, chronic (usually >1 year)

Table 3 *Relationship between variables (Pearson product–moment correlations)*

	Time since stroke (months)	Contracture: elbow flexion (degrees)	Positive features						Negative features			
			Hyperreflexia: gain of tonic stretch reflex			Spasticity: Hypertonia: resistance to passive movement			Dexterity			Strength: elbow flexors IEMG (μV)
			90°@2Hz (μV deg^{-1})	90°@3.5Hz (μV deg^{-1})	20°@2Hz (μV deg^{-1})	90°@2Hz (Nm deg^{-1})	90°@3.5Hz (Nm deg^{-1})	20°@2Hz (Nm deg^{-1})	MAS: item 6 (0–6)	Slow track: overall coherence	Fast track: overall coherence	
	A	B	C	D	E	F	G	H	I	J	K	L
Time since stroke	A	0.01	−0.22	−0.22	−0.19	−0.10	−0.20	0.11	−0.11	0.25	0.09	−0.10
Contracture	B		0.35	0.35	0.74*	0.21	0.00	0.07	−0.15	−0.13	−0.06	0.02
Tonic stretch reflexes	C			1.00**	0.77**	0.15	−0.01	0.09	−0.20	−0.24	−0.20	0.05
	D				0.77**	0.15	−0.01	0.09	−0.20	−0.24	−0.20	0.05
	E					0.09	−0.03	0.15	−0.21	−0.30	−0.21	0.07
Resistance to passive movement	F						0.77*	0.20	0.25	−0.11	0.03	0.00
	G							0.24	0.00	−0.13	−0.05	0.06
	H								−0.18	−0.41*	−0.38	−0.10
Dexterity	I									0.65*	0.75*	0.46*
	J										0.90*	0.59*
	K											0.69*

*$P < 0.05$; **spurious correlations due to the fact that only one subject had reflex activity at 90° position.

spasticity (Lee *et al.*, 1987; Powers *et al.*, 1988, 1989; Thilmann *et al.*, 1991*a*; Katz *et al.*, 1992; Ibrahim *et al.*, 1993*a*), making it highly probable that they would exhibit the abnormal tonic or phasic stretch reflexes that were reported in these studies. These successive reports of abnormal reflex activity may have perpetuated a focus on spasticity in the clinic. In the present study, the subjects were drawn as non-selectively as possible from three standard rehabilitation units within 1 year following their stroke and they are therefore more representative of stroke patients undergoing rehabilitation than previous studies. We have found only a small proportion of these hemiparetic patients to have spasticity manifest as exaggerated tonic stretch reflexes.

A possible interpretation of this finding is that reflex hyperexcitability may have been present early following stroke, preceding our investigation, in some subjects. However, this is an unlikely possibility since spasticity appears to be an adaptation to, rather than a direct result of, cerebral damage (Chapman and Wiesendanger, 1982; Burke, 1988) and requires time to develop (Brown, 1994). For example, Thilmann *et al.* (1991*a*) found that spasticity was rarely apparent during the first month following stroke but that stretch reflex gain increased over the second and third month. Almost half (11) of our subjects were seen within 3.5 months after their stroke (Table 2) and only three of these had reflex hyperexcitability. Furthermore, this interpretation depends on the premise that early reflex hyperexcitability had disappeared in our subjects by the time of the study.

We have demonstrated a link between muscle contracture and increased resistance to passive stretch. However, the increased resistance was not dependent on the presence of tonic stretch reflexes and patients with both reflex hyperexcitability and contracture were no more stiff than those with contracture alone. Antagonist muscle activity, which was not measured in this study, would be important here only if hypertonia that was not attributable to biceps reflex hyperexcitability was instead attributable to triceps reflex hyperexcitability. This appears unlikely, especially since abnormalities of flexor muscles appear more pronounced than those of extensor muscles in spastic patients (Dietz *et al.*, 1991; Ibrahim *et al.*, 1993*a*). Therefore, the increased passive resistance appears to be attributable to the presence of contracture rather than reflex hyperexcitability.

The process of adaptive muscle change following cerebral lesions is a complex one. Contracture obviously affects the passive non-contractile properties of muscle, but the characteristics of the active muscle length–tension curve are also altered when fibre length is reduced by loss of sarcomeres (Williams and Goldspink, 1978). Atrophy of type II muscle fibres and fibre type transformation have been documented in spastic patients (Edström, 1970; Dietz *et al.*, 1986) and an increased torque output for a given level of EMG activity has been reported in a number of studies (e.g. Lee *et al.*, 1987; Dietz *et al.*, 1991; Ibrahim *et al.*, 1993*a*). Given such findings as well as those of the present study, it now seems likely that adaptive changes in muscle tissue are often responsible for the clinical impression of hypertonia. Clinical measures of spasticity measure hypertonia by gauging the resistance to passive displacement of the limb (e.g. Ashworth, 1964) but this method cannot distinguish between the peripheral contribution due to muscle adaptation and the neural contribution due to increased stretch reflexes. As noted

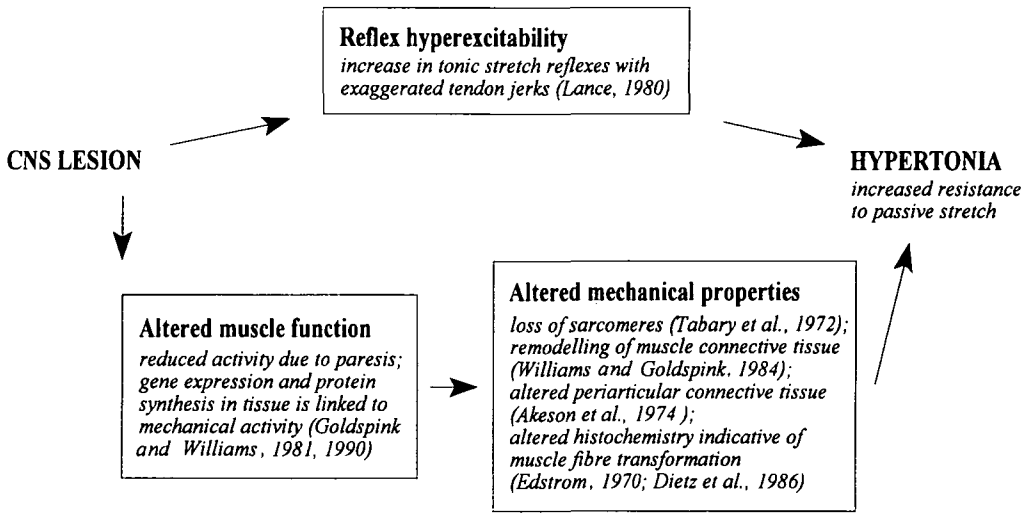

Fig. 5 Contributions of reflex and muscle adaptation to hypertonia following cerebral lesions. Resistance to passive movement can be increased via reflex hyperexcitability and/or via altered mechanical properties. Only altered mechanical properties due to contracture appeared to contribute to hypertonia in the patient group in the present study.

by Katz and Rymer (1989) and illustrated in Fig. 5 here, these are separate contributors to clinical hypertonia following cerebral lesions. In our present patient group, adaptation of the mechanical properties of muscle appeared to be the primary contributor to hypertonia. Therefore, it is necessary to recognize a clear distinction between reflex hyperexcitability and hypertonia in patients with spasticity.

It has been commonly assumed that the exaggerated reflexes of spasticity lead to muscle contracture. However, in the current study, the presence of tonic stretch reflexes in only five of the 13 patients with muscle contracture and principally when elicited with the muscles in a lengthened position, suggests instead that muscle contracture may potentiate the stretch reflex, at least in some patients. If a muscle and its spindles shorten due to contracture, the stretching effects of a given change in joint angle differ from a normal muscle in several ways, all of which may increase the size of the tonic stretch reflex. First, the muscle is brought closer to the end of its range, thereby increasing the effect of length-dependent facilitation (Ashby and Burke, 1971; Neilson and Lance, 1978; Nash *et al.*, 1989); secondly, a larger change in relative muscle length is imposed, thereby producing a larger amplitude stretch of the spindles; thirdly, as noted by Perry (1980), the forces accompanying the movement are transmitted more completely and more promptly because the tissues are stiffer as a consequence of the contracture. These three theoretical mechanisms indicate how contracture might potentiate the stretch reflex. In line with this, Perry (1980) has cited clinical experiences which suggest that correction of contracture may reduce spasticity. Nevertheless, the nature of the temporal relation between spasticity and muscle contracture would best be resolved by a longitudinal study of a group of patients beginning immediately after the stroke.

Our findings suggest that contracture was not caused by spasticity, and thus raise the question of the actual cause. Contracture of the elbow flexor muscles was identified in 13 of the 24 patients in the present study. All the patients were studied within 1 year of their stroke, but contracture was documented as early as 2 months. The likely mechanisms of development of contracture in hemiparesis following stroke can be found in studies on regulation of muscle length in experimental animals (e.g. Tabary *et al.*, 1972; Williams and Goldspink, 1971, 1978, 1984; Tardieu *et al.*, 1982*b*; *see* O'Dwyer *et al.*, 1989 for review). Such studies show that skeletal muscle is highly adaptable, so that its structural characteristics are determined by its conditions of use (a classic biological example of the relation between structure and function). When a muscle is immobilized in a shortened position, a shortening of muscle fibre length occurs due to loss of sarcomeres in series, accompanied by shortening of muscle connective tissue and an increase in stiffness of the muscle. The extent of these changes is illustrated by the study of Tabary *et al.* (1972) who produced a 40% reduction in number of sarcomeres in cat soleus muscle with immobilization in a shortened position for 4 weeks. In stroke patients, the arm may be effectively immobilized in the presence of paralysis or weakness and this predisposes the patients to rest their paretic arm in their lap (Ada and Canning, 1990), particularly if, due to difficulty with walking, they spend much of their time sitting. This posture results simply as a consequence of convenience and comfort but it subjects the elbow flexor muscles, among others, to immobilization in a shortened position, precisely the conditions shown to produce muscle contracture in experimental animals.

In line with earlier studies cited above, we found no relationship between spasticity and motor function. This was true regardless of whether spasticity was measured as hyperexcitable tonic stretch reflexes or increased resistance

to passive stretch and whether motor function was measured by a general clinical scale or a more specific tracking task. Similarly, there was no relationship between spasticity and strength. Since few of these stroke patients exhibited hyperactive tonic stretch reflexes, especially at the elbow position (90°) where strength was measured, a role for spasticity in recovery of either motor function or strength was unlikely in any event. These findings are consistent with the view, which is gaining increasing acceptance, that spasticity and the negative features of weakness and loss of dexterity following brain damage are separate entities (e.g. Carr and Shepherd, 1987; Katz *et al.*, 1992; Thilmann *et al.*, 1993). Indeed, none of the 'positive' and 'negative' features measured in this study were found to be correlated.

In the present study reflex excitability was investigated under passive conditions only, so we cannot comment on the stretch reflex behaviour of our patient group under active conditions. Just as in normal subjects, the response to muscle stretch in hemiparetic subjects changes between rest and activity (Ibrahim *et al.*, 1993*a*). While short-latency reflexes are exaggerated during activity, long-latency reflexes have been shown to be reduced in amplitude (e.g. Berger *et al.*, 1984; Cody *et al.*, 1987; Dietz *et al.*, 1991; Ibrahim *et al.*, 1993*a*). Furthermore, reflexes elicited under active conditions are more likely to be functionally relevant than those elicited under passive conditions. Thus, clinically identifiable stages of recovery of motor function have recently been shown to be related to the late EMG response to electrical stimulation and inversely related to the early response (Ibrahim *et al.*, 1993*b*).

In summary, spasticity does not appear to be related to the negative features of weakness or loss of dexterity following stroke. Furthermore, it does not seem to be the cause of the common secondary problem of muscle contracture. Although a longitudinal study is desirable to clarify the nature of the relationship between spasticity and contracture, the indications are that contracture may potentiate the stretch reflex, at least in some patients. Given the common occurrence of contracture in the patients in this study, the need to maintain muscle length following stroke seems paramount. Therefore, the amount of attention directed to reflex hyperexcitability associated with spasticity of cerebral origin would appear to be out of proportion with its effects. However, hypertonia following cerebral lesions remains an important problem requiring further investigation, especially because of its link with contracture.

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