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ORIGINAL ARTICLE

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Muscle and tendon stiffness in patients with upper motor neuron lesion following a stroke

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Abstract The objective of this study was to investigate muscle and tendon stiffness in the triceps surae muscles in patients who had previously had a stroke. The participants were 12 men showing slight to moderate degrees of muscle tonus in the affected leg. All patients showed minimal or no overt clinical motor symptoms, and all walked without mechanical aid. Muscle strengths in isometric and isokinetic activities were measured, as was passive resistance during plantarflexion in each leg. Walking speed was also measured. Evaluations of physical performance and muscle tone were made. Muscle and tendon stiffness was calculated from measurements whilst passively stretching during electrical stimulation, separately for each leg. Muscle strength was significantly higher in the non-affected than in the affected leg. Muscle stiffness was significantly higher in the affected leg than in the non-affected leg. Tendon stiffness was significantly higher in the non-affected than in the affected leg. The higher muscle stiffness in the affected leg might enhance the possibility for storing elastic energy during preactivation. Lower tendon stiffness in the affected leg might reduce the development of fatigue in movements at low velocities.

Key words Eccentric-concentric actions · Electromyography · Gastrocnemius muscle · Soleus muscle · Human

Introduction

The elasticity of muscle and tendon has been shown to play an important role in using muscle energy during locomotion (Shadwick 1990). Repeated eccentric-concentric muscle actions occur in many normal activities in daily living, i.e. during plantar flexion when walking and running. In a stretch-shortening cycle (SSC), an eccentric muscle action is immediately followed by a concentric action. The torque output of this concentric action has been found to be greater than that of a corresponding pure concentric action (Cavagna et al. 1968; Asmussen and Bonde-Petersen 1974; Komi 1984; Svantesson et al. 1994). It has been suggested that this special effect might be attributable to the combined effects of the use of elastic energy and myoelectrical potentiation of muscle activation (Bosco et al. 1982). Use of elastic energy in SSC in combination with muscle and tendon stiffness has been discussed by Goubel (1987) and Shorten (1987). A study of SSC using a dynamometer during plantar flexion in patients with an upper motor neuron lesion has shown higher concentric torque values after preceding muscle actions in both legs, the percentage increase being significantly higher in the affected (hemiparetic) leg as compared to the non-affected leg (Svantesson and Sunnerhagen 1997). We suggested that the higher performance of the affected leg was a result of better use of elastic energy in that leg owing to differences in stiffness of the muscle-tendon complex.

A model of the muscle-tendon complex has been developed and defined by Morgan (1977) as two springs connected in series. One spring with a constant stiffness (tendon stiffness, S_t) was assigned to the passive structures of both muscles and tendon. The other spring (muscle stiffness, S_m), with a stiffness proportional to force, was assigned to the cross-bridges of the muscle. Using this model S_m and S_t can be calculated separately.

After a stroke, there has been found to be a reorganization of the central nervous system to compensate for the loss of cells with normal function (Bach-y-Rita 1981). In patients with hemiparesis after a stroke, changes in muscle structures and metabolism have been reported (Sjöström et al. 1980). Changes in passive stiffness of the affected ankle muscle-tendon structure in hemiparetic subjects have been reported by Sinkjaer and

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Magnussen (1994) who also demonstrated no changes in intrinsic stiffness (properties of contracting fibres). They considered this to be an indication that the contractile properties of the muscles were unaffected.

The purpose of the present study was to investigate S_t and S_m in patients who had previously suffered a stroke, who showed minimal or no overt clinical motor symptoms. The stiffness was to be compared during walking, and in muscle strength and passive resistance tests.

Methods

Subjects

The criteria for including the patients in the study were: having an upper motor neuron disorder following one incident of cerebrovascular disease more than 6 months earlier, the ability to walk without a mechanical aid, an age between 40 and 65 years and having an active range of motion of at least 40° of the ankle joint (so that they could perform the test). Criteria for exclusion were: having had a previous stroke or having orthopaedic problems.

A group of 12 male patients with hemiparesis (Table 1) in the lower extremities (six right and six left), who fulfilled the inclusion criteria, were selected from the Department of Rehabilitation Medicine at Sahlgrenska University Hospital, where they had been in-patients. They had a mean age of 55 (SD 8, range 41–65) years, a mean height of 177 (SD 6, range 162–185) cm and a mean body mass of 81 (SD 10, range 56–97) kg. All participants gave their informed consent according to the declaration of Helsinki before taking part in the study, which had been approved by the Ethics Committee at the Faculty of Medicine, Göteborg University.

The patients were examined both by a physician and a physiotherapist. On a modified Ashworth scale for assessment of spasticity between 0 and 4, where 0 has been described as indicating normal tone (Bohannon and Smith 1987), all patients scored between 0 and 3 with a median of 1. To evaluate the physical activity level of the patients, the physical activity scale for the elderly (PASE;Washburn et al. 1993)) was used. The PASE scores may range from 0 to 400 or more. Our patients scored a mean of 139 (SD 23). The motor function of the affected leg was evaluated according to Fugl-Meyer et al. (1975). The Fugl-Meyer score has been described as ranging from 0 to 34, where a higher score indicates better motor function. The patients received scores of between 14 to 33 with a median of 30. The sensory function of the affected lower leg was also evaluated according to Fugl-Meyer et al. (1975), and 7 patients were judged to have impaired function (see Table 1).

Protocol

A 30-m walking test was performed twice (Lundgren-Lindquist et al. 1983). The person was first told to walk at a self-chosen speed and then, after a 2-min rest, to walk as fast as he could. The time was measured and the velocity calculated.

A Kinetic-Communicator II dynamometer (Kin-Com; Chattanooga Group, Inc., Hixson, Tenn., USA) was used for measurements of torque production. The subject lay in a prone position during the tests, with knees and hips fully extended and with the test foot hanging free of the bench, allowing free movements of the ankle joint. The lower part of the leg was firmly secured to a pad attached to the bench. The other leg lay straight on the bench, parallel to the leg being tested. The elbows were flexed, and the hands were placed under the shoulders or the head. To minimize horizontal sliding during the tests, two extra pads were attached to the bench to support and secure the shoulders as has been described by Svantesson et al. (1994). The axis of the lever arm of the Kin-Com was carefully adjusted to the axis of rotation of the ankle joint. The lever arm was preset to a range of motion of approximately 80–115° for all subjects. The total range of motion of the ankle joint was not examined to minimize the risk for injury. A 90° angle of the ankle joint was defined as the point at which the sole of the foot was perpendicular to the axis of the lower leg and 80° was in the direction of dorsiflexion. Footwear was standardized. The same shoe model in appropriate sizes was used by all participants. A foot device, a 110-mm-long pedal covered by hard rubber, was used. The pedal was carefully adjusted under the metatarsophalangeal joints. Torque and angle on the Kin-Com were sampled with a frequency of 100 Hz.

The test of passive resistance was performed in the same position on the dynamometer. The patient was instructed to relax while the actuator arm was passively moved from 105° of plantarflexion to 85° of dorsiflexion. Three trials were performed, and the second trial was used for further analysis. The results of the first trial could be variable, while those of the second and third trials were always identical.

The stiffness test was performed in the same position on the dynamometer. The calf muscle was stimulated tetanically by a specially developed electrical stimulator monitored by PC software (AB Detektor, Göteborg, Sweden). Two electrodes (dimensions 50×100 mm) were attached to the surface of calf muscles. The pulse frequency was set at 50 Hz, pulse width at 50 μm and duration at about 3 s. Immediately when the muscle was stimulated and a plateau of isometric tension was obtained, a short fast stretch was given to the muscle by the dynamometer (Fig. 1). The stretch was preset from 100° to 80° of the ankle joint at a preset isokinetic velocity of 200° s^{-1} on the dynamometer, leading to a stretch of the calf muscle that Grieve et al. (1978) have shown can be calculated. As the length of the gastrocnemius muscle has been shown to be 18% of the body length (Grieve et al. 1978), the calculated stretch length was 6.0 mm (SEM 0.1 mm, ranging from 5.5 to 6.3) for the

Table 1 Characteristics of the patients. *R* Right, *L* left, *PASE* physical activity scale for the elderly

Age (years)	Location of lesion	Time since lesion (months)	Physical activity (PASE)	Muscle tone (modified Ashworth scale)	Motor function (Fugl-Meyer score)	Sensory function affected leg (Fugl-Meyer score)
58	R Parietal	46	162	0	32	Impaired
58	R Basal ganglia	47	99	1	30	Not impaired
53	L Parietal	22	152	3	25	Impaired
55	R Parietal	33	227	1	30	Impaired
62	R Temporal	68	161	0	30	Not impaired
63	R Parietal	52	280	0	33	Impaired
59	R Parietal	17	70	1	32	Not impaired
41	L Parietal	20		0	32	Not impaired
65	L Basal ganglia	10	48	1	28	Not impaired
54	L Parietal	16	46	2	14	Impaired
41	R Parietal	18	187	2	28	Impaired
54	L Parietal	6	97	1	29	Impaired

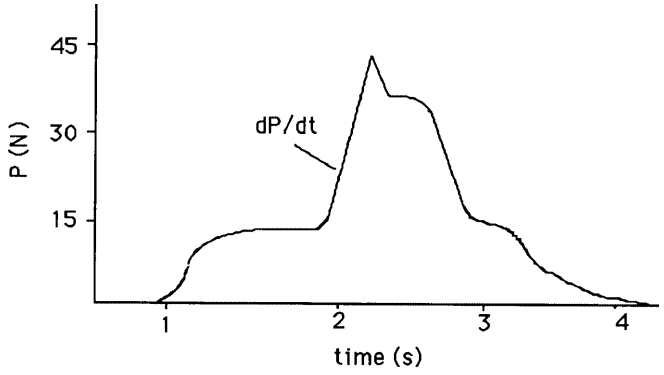

Fig. 1 Once a steady level of force (P) had been reached by electrical stimulation, a rapid stretch was applied. The slope of the line (dP/dt) during the stretch was recorded and calculated using single regression analysis for each level of stimulation. The intensity of the stimulation was varied to produce the force levels from 15% to 30% maximal voluntary contraction

10° interval included in the calculation. The level of force was controlled by the stimulation current intensity.

There was one patient who found the electrical stimulation too painful and was unable to perform the stiffness test. He was therefore excluded from the calculations of stiffness.

Test procedure

Before the tests, the subject warmed up for 5 min by cycling at submaximal intensity. The subjects then made three pure concentric plantar flexions with maximal effort on the dynamometer at a preset velocity of 60° s^{-1}. After 2 min of rest, a combined activity consisting of an eccentric muscle action immediately followed by a concentric muscle action was performed with maximal effort at a preset velocity of 60° s^{-1}. The highest concentric peak torque values of the three trials of the two tests were used.

Before the stiffness test, three voluntary isometric muscle actions (MVC) were performed with maximal effort at a 90° angle of the ankle joint. The highest force output of the three trials was extracted and used as a reference value for the stimulation intensity during the stiffness test. The test of passive resistance was then performed.

The surface electrodes, covered by electrically conductive gels, were secured on the calf muscle by tape. The proximal electrode was placed 12 cm and the distal electrode 22 cm below the popliteal line. The voltage intensity produced by the stimulator was gradually increased. When it reached the level at which a muscle contraction was recognized, the force measurement of the plantar flexors started using the stretch. The stiffness test was performed at least three times using a different stimulation level each time. The stimulation level produced force values between 15% and 30% of MVC. The torque curve for each stimulation level was stored for further analysis. During stimulation we checked the muscle contraction so that the force produced was linearly increasing with increasing stimulation. However, if the relationship between the increasing force and intensity of the stimulation was broken, we stopped increasing the stimulation intensity because we thought antagonist muscles would be stimulated.

When the first leg had been examined, the subject rested for 10 min, after which the same procedure was repeated for the other leg. The test order of the two legs was randomized. The subjects were tested on two separate occasions.

Analysis

The effect of a preceding muscle action (SSC) was taken to be the percentage difference between the concentric peak torque values of the pure concentric and the combined eccentric-concentric muscle action.

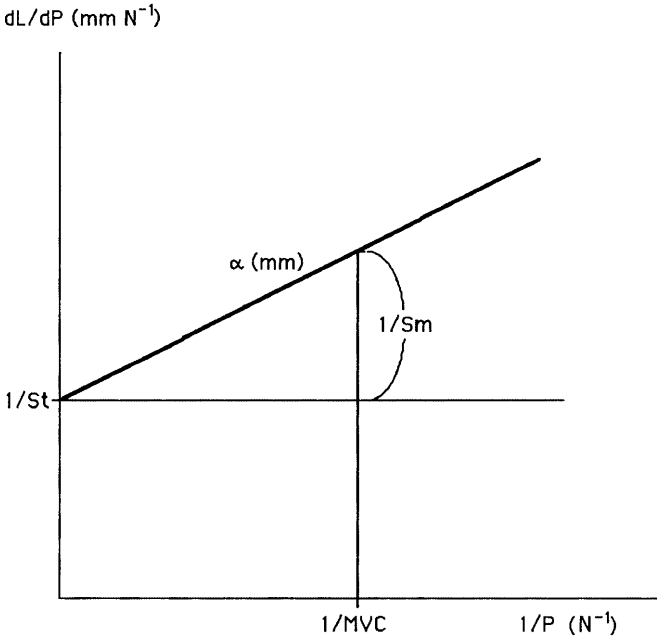

Fig. 2 According to the theory of Morgan (1977), the linear regression line has the equation $\frac{dL}{dP_\alpha} = \frac{\alpha}{P} + \frac{1}{S_t}$, where the intercept of the ordinate gives the value of $1/S_t$ (L length, P force, S_t tendon stiffness). The slope gives the value of α. Maximal muscle stiffness (S_m)is given by MVC/α, where MVC is maximal voluntary contraction

The force curves seen in Fig. 1 from the stiffness test were analysed using methods that have been described by Cook et al. (1996). The dP/dt from the stretch was calculated using simple regression analysis. From the calculated change in length of 6 mm of the total muscle-tendon complex during a time course of 0.1 s, the inverted force ($1/P$) and dL/dP were plotted for each level of stimulus intensity (Fig. 2). According to the theory of Morgan (1977), S_t is constant with increasing stretch force. On the other hand, S_m increases linearly with the force applied: $S_m = P/\alpha$ where α is a constant with the dimension of length (Morgan 1977). From the plot (Fig. 2), the α was calculated using simple regression analysis and S_t was read. Maximal S_m is dependent on the MVC (maximal voluntary isometric contraction). In this group of patients, it might have been difficult to obtain a true MVC, and α was thus used to express S_m.

The passive resistance at each angle of the ankle joint was plotted, and a simple regression analysis was made. The coefficient of the slope was calculated. For greater detail, see Broberg and Grimby (1983).

Statistics

The Wilcoxon one-sample non-parametric test was used for differences between paired observations. A significance level of 0.05 was used.

Results

Mean walking velocity for the self-chosen speed was 1.15 (SEM 0.05) m s^{-1} and for maximal speed 1.58 (SEM 0.10) m s^{-1}.

Mean peak torques measured on the dynamometer for isometric and pure concentric actions and concentric

Table 2 Torque values for the isometric, the pure concentric, the concentric after eccentric and the percentage increase in torque due to eccentric preactivation for the affected and the non-affected legs

Action	Torque, affected leg (N · m)		Torque, non-affected leg (N · m)		Significance *P*
	Mean	SEM	Mean	SEM	
Isometric	74	9	110	8	0.002
Pure concentric	40	8	68	6	0.008
Concentric after eccentric	83	9	101	8	0.011
Increase (%)	106	15	69	10	0.008

The *P*-value gives the significance of the difference between legs

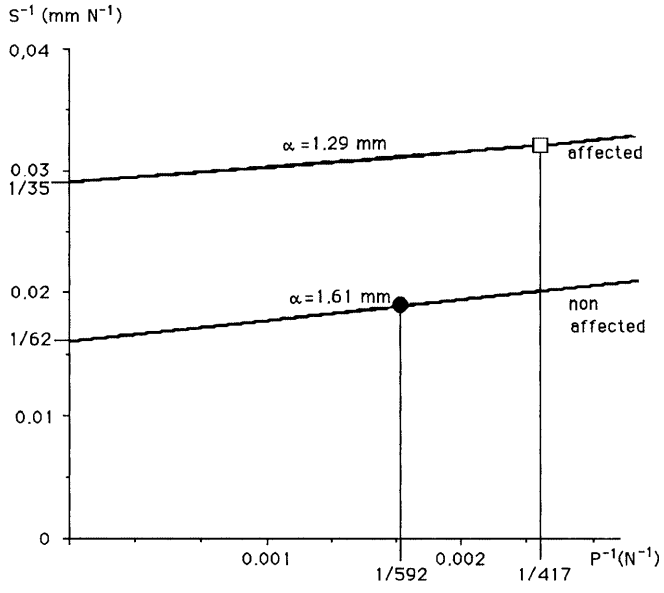

Fig. 3 Inverted total stiffness ($1/S$) as a function of inverted force (P; $1/N$) for the affected and non-affected legs. Tendon stiffness, slope coefficient (α) and maximal voluntary isometric contraction are shown for both legs

actions preceded by eccentric actions of the plantar flexion are shown for both the affected and the non-affected legs in Table 2. This table also shows the percentage increase in torque between a pure concentric action and a concentric action preceded by an eccentric action for each leg.

The slope coefficient for passive resistance was 0.63 (SEM 0.06) · N^{-1} for the affected leg and 0.48 (SEM 0.04) · $N^{-1}$for the non-affected leg. There was a significant difference ($P = 0.004$) between legs.

The S_t were 35 (SEM 4) N mm^{-1} for the affected leg and 62 (SEM 8) N mm^{-1} for the non-affected leg. There was a significant ($P = 0.010$) difference between legs. The S_m, expressed as α, was 1.29 (SEM 0.16) mm for the affected leg and 1.61 (SEM 0.18) mm for the non-affected leg (Fig. 3). There was a significant ($P = 0.009$) difference between legs.

Discussion

The present study revealed that S_m was significantly higher in the affected leg compared to the non-affected leg. The S_t, however, was significantly higher in the non-affected than in the affected leg. This was an indication that changes may occur in the mechanical properties of the muscle and tendon complex following a stroke.

As has also been shown in a study by Svantesson and Sunnerhagen (1997), the percentage increase in concentric torque after a preceding eccentric muscle action was significantly larger in the affected leg as compared to the non-affected leg among stroke patients. However, all patients in that study scored 0 on the modified Ashworth scale, indicating minimal changes in tonus. It was assumed that the higher performance in the affected leg was caused by better use of elastic energy due to increased muscle stiffness. Accordingly, in the present study, we found increased muscle stiffness in the affected leg. Dietz and Berger (1983) suggested that altered mechanical properties in the muscle contributed to changes in muscle stiffness during locomotion in stroke patients. However, Sinkjaer and Magnussen (1994) published a study in which intrinsic stiffness did not differ between legs, indicating that the contractile properties of the muscle were unaffected.

Komi (1984) emphasized that it is difficult to separate elastic and myo-electrical components in SSC. There is obviously an integration of the two systems as has been described by Bosco et al. (1982). An increase in myo-electrical potentiation would normally result in more elastic energy. The stretching of muscles in eccentric actions has an effect on muscle spindles as well as on Golgi tendon organs. These reflexes – facilitatory or inhibitory – influence the outcome. In a paretic leg, one could expect a reduced inhibitory activity, which could lead to an increase in tonus, resulting in an enhanced muscle stiffness in the affected leg. This can also be seen in the results in the present study, where the affected leg showed increased passive resistance and a higher score on the modified Ashworth scale, indicating decreased inhibition. As elastic energy is partly stored in the contractile elements, any increase in the number of cross-bridges being activated would result in a larger capacity for elastic energy storage. In the present study, the affected leg was weaker than the non-affected leg. It has been shown that this difference in concentric muscle actions between the affected and the non-affected legs can be reduced by adding preceding eccentric actions without changes of the electromyogram (EMG; Svantesson and Sunnerhagen 1997) owing to the use of stored elastic energy. Lee et al. (1987) also showed, in voluntarily

activated spastic elbow muscles, smaller EMG recordings along with greater stiffness in the affected side.

In contrast to the results by Sinkjaer and Magnussen (1994), a lower S_t was found in the present study. One explanation for this discrepancy might be differences in the experimental design. A lower S_t can result in a reduced demand for energy to stretch and shorten the muscle tendon complex during eccentric-concentric muscle actions at lower velocities and might therefore reduce the development of fatigue during repeated eccentric-concentric actions in low speed motions. This can be seen from a study by Svantesson et al. (1998) where, despite a lower muscle strength in the affected leg, the same number of heel-rises could be performed in both legs.

The changes in the mechanical properties in the affected leg shown in the present study might explain the differences in use of preceding muscle actions between the affected and non-affected leg. This may be of importance in the physical training of hemiparetic patients. Training activities emphasizing eccentric-concentric exercises may result in a more normal function in the affected leg and may be useful for enhancing the recovery of motor function.

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