Influences of muscle stretch reflexes on voluntary, velocity-controlled movements in spastic paraparesis

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Summary

We studied voluntary, velocity-controlled knee movements in velocity. The antagonist EMG activity was reduced in the same proportion as the agonist EMG activity in eccentric actions when stretch is imposed upon antagonists, the antagonist EMG activity increased with the velocity of stretch, indicating stretch reflex activation. In parallel with the stretch reflex activation of antagonists, there was reduced activation of the agonists compatible with Ia reciprocal inhibition of agonist motoneurons. When agonists were stretched in eccentric actions, stretch reflexes appeared to support the voluntary, agonist activation of knee flexors but not knee extensors.

Keywords: spasticity; voluntary contraction; stretch reflexes; reciprocal inhibition

Abbreviation: MANOVA = multivariate analysis of variance

Introduction

In spastic paresis, the weakness of voluntary movements can be caused by different mechanisms. First, the generation or transmission of descending voluntary commands may be disturbed. Secondly, spastic antagonistic muscles, when stretched by a movement may become activated by exaggerated muscle stretch reflexes which may then result in larger restraint than that normally given by antagonistic muscles. A third mechanism implying a segmental inhibition of agonist motoneurons emanating from the Ia inflow from muscle spindles of spastic antagonists may further decrease muscle strength. This mechanism was suggested to explain a marked increase in agonist EMG activity in maximal voluntary movements seen during medication with antispastic drugs (Knutsson et al., 1982; Knutsson and Mårtensson, 1984).

There are several other features of spastic paresis that can compromise the production of strong movements indirectly. Spasms and dystonias may reduce the strength of movements when they involve antagonistic muscles, though their main effect seems more related to transitory reduction of motor control. An inability to direct voluntary commands exclusively to muscles needed for a movement causes an overflow of activation to widely spread inappropriate muscles resulting in associated movements which severely disturb motor control. Finally, the muscle and tendon contractures that commonly develop in severe spasticity give restraint to movement in the outer parts of movement ranges.

The influence of spasms, dystonias, associated movements and contractures on voluntary movements can usually be assessed quite well in a clinical analysis of the individual patient. The strength of voluntary movements can also be assessed and, in some movements, quantitatively determined. However, it is seldom possible to estimate the relation between prime mover dysfunction and spastic restraint, since
the spastic restraint in passive movements is not the same as that in voluntary movements (McLellan, 1977; Knutsson and Mårtensson, 1980).

With the development of active isokinetic dynamometers it has become possible to study voluntary movements of different velocities both in concentric and eccentric actions. In concentric actions, the agonists shorten while the antagonists are stretched. In eccentric actions, the agonists are stretched while the antagonists are shortening. Thus, the activation of antagonists during voluntary movements can be studied both when stretch is imposed upon them and when there is no stretch imposed upon them. The activation of the agonists in voluntary movements can similarly be studied during stretch and shortening. It means that the influences of exaggerated stretch reflexes on voluntary movements can be studied in spastic patients by comparing the activation of muscles when they are stretched and not stretched. Thus, we measured torque and EMG activity in isokinetic movements of different velocities during concentric and eccentric actions of knee extensor and flexor muscles at maximal voluntary effort in patients with spastic paraparesis and used similar measurements in healthy subjects for control.

Material and methods

Subjects

The study was performed on 22 patients with spastic paraparesis (mean age 45 years; range 27–63 years) and in 22 healthy subjects (mean age 51 years; range 39–69 years). In each group there were 11 men and 11 women. Table 1 gives clinical data of relevance for the study. Seven patients suffered from hereditary spastic paraparesis. In the remaining 15 cases the paraparesis was secondary to multiple sclerosis (six cases), chronic myelopathy (four cases), fracture of the thoracic spine (two cases), intramedullary cyst (one case), myelitis (one case) and cerebral spastic diplegia (one case). The duration of disease ranged from 2 to 39 years. Inter-individual differences in walking ability is indicated in the table by the aids used in ambulation. Estimations of the strength of extension and flexion (Ditunno et al., 1994) and of spasticity in extensors and flexors (Ashworth, 1964) as found in clinical examinations are given for the left knee joint selected for the study. The strength in knee extensions had to be at least of Grade 3 in the rating scale used for the patient to be selected to participate in the study. A strength of Grade 2 was sufficient for the knee flexions since they gained support by gravitation in the sitting body posture used. In three patients, signs of spasticity were found only in the extensor muscles; in one patient spasticity was found only in the flexor muscles. In the remaining 18 patients, exaggerated stretch reflexes were seen both in extensor and flexor muscles of the left knee. Ankle clonus was seen in all but three of the patients. In 19 of the 22 patients, the muscular hypertonus had been considered severe enough to warrant a trial period of antispastic medication. Antispastic medication, when present, was withdrawn at least 1 week before entering the study. The study was approved by the Ethical Committee of the Karolinska Hospital, Stockholm, and all subjects gave informed consent to participate.

Dynamic dynamometry

Voluntary, isokinetic knee extensions and flexions were performed under the control of an active, dynamic dynamometer using a hydraulic servo-system under microcomputer control (KINCOM H500, Chattanooga Corp. Chattanooga, Tenn., USA). The dynamometer has a hydraulic actuator that rotates a lever arm with preset, constant velocities in selected angular ranges for control of movements and two couches for fixation of the subject in well defined body postures for measurement on the left or right side. The subject sat on a couch with the left thigh strapped in a horizontal position and the pelvis fixed and supported against tilt. The trunk was kept in a vertical position without fixation. The rotation axis of the lever arm was aligned to the mid position of the slightly changing axis of flexion-extension of the knee joint and the lower leg attached to the lever arm at the ankle through a strain-gauge transducer. After a few movements at submaximal effort to warm up, the experimental measurements at maximal voluntary effort were made.

Before the start of the study, the movement control by the dynamometer was redesigned to diminish disturbances from frequency-modulated torque signals that made concomitant records of torque and EMG difficult. Procedures were also included in the system for checking of reproducibility during examinations, correction of force recordings for gravitation and averaging of data. The strain gauge transducer between leg and lever arm recorded force with an accuracy within 3% of the load. Angular position was monitored with a high precision rotational potentiometer and velocity with a tachometer. Average velocities were within 1.5% of target velocities.

To synchronize lever arm movements with voluntary effort movements are started first when the force applied against the lever arm reaches a selected level. Concentric movements are started at forces in the direction of the movement while eccentric movements are started at forces in the direction opposite to the movement. If the force during motion falls below a minimal accepted level, commonly 20 N, the movement decelerates. During deceleration the force may increase. If it reaches above the minimal level, the deceleration is substituted with acceleration toward the target velocity. Otherwise the movement stops but will restart if the force again reaches above the minimal level. In consequence, velocity oscillations and stops are typical when voluntary strength is low. In patients with spastic paresis, these phenomena often disturb voluntary movements, thus limiting the possibility of studying interaction of agonists and antagonists during voluntary effort especially at high velocities. The control was therefore modified so that movements continued at the selected velocity even if the
voluntary strength was too low to execute the full movement; a negative force signal then indicated insufficient strength and showed the additional force needed to complete the actual movement.

In repeated isokinetic movements made with submaximal voluntary effort the force varies highly. In contrast, the force does not vary appreciably in isokinetic movements at maximal effort since the force is set by the upper limit of the voluntary strength (Knutsson, 1983; Knutsson and Mårtensson, 1985). Sapega et al., (1982). The torque of the voluntary movements was calculated from the force recordings and the length of the lever arm, and then corrected for gravitation.

**Electromyography**

The EMG was recorded with pairs of surface electrodes. They were placed over the quadriceps muscle at a distance of one-third of the thigh length from the patella, and with an inter-electrode distance equal to one-fifth of the thigh circumference, and over the hamstring muscles at a distance from fossa poplitea of 40% of the thigh length, on lines between the tuber ossis ischii and the insertions of the biceps femoris and semitendinosus muscles. The electrodes were connected to miniature preamplifiers close to the electrodes where the signals were amplified (~1000-fold) to decrease the sensitivity to noise from the dynamometer, and to allow signal transfer to an EMG-processing device without appreciable artefacts caused by the movements in the connecting leads. In the EMG-processing device included in

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**Table 1 Clinical details of individual patients**

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<th>Duration (years)</th>
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<th>Strength (0–5)*</th>
<th>Spasticity (0–4)†</th>
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the dynamometer system, the signals were rectified and passed through a band-pass Paynter filter (20–1000 Hz) and then through a low-pass filter (100 Hz) (Gottlieb and Agarwal, 1970). The integrated EMG was calibrated against a 200-Hz sinusoidal signal of 1-mV peak-to-peak amplitude rectified and filtered in the same way as the EMG signals. It was sampled at 100 Hz with a 12 bit A/D (analogue to digital) converter.

Statistical analysis of data
Differences were tested with Student’s t test or multivariate analysis of variance (MANOVA). When significant group differences occurred in multivariate analyses, tests with contrast were carried out with probability levels adjusted according to the Bonferroni procedure, to identify the variables that significantly contributed to the group differences.

Results
The results were based upon records of torque and EMG in voluntary, isokinetic movements of the type illustrated in Figs 1 and 2. They show computer processed torque and EMG records in concentric and eccentric knee joint movements of four velocities at maximal voluntary action of the knee extensor muscles in a healthy male volunteer (Fig. 1) and a male patient with spastic paraparesis (Fig. 2). Torque curves comprise mean (uninterrupted lines), and maximum and minimum (dotted lines) torque for consecutive angular positions from three repeated tests of each of the eight movements. EMG activity in the agonist (quadriceps) and the antagonists (hamstring) are given as mean of rectified and filtered surface EMG signals. Concentric movements started with the knee joint flexed 90–95° and eccentric movements with the knee joint flexed 20–30°.

In the healthy subject (Fig. 1), the action of the knee extensor muscles extended the knee joint to an angular position of 20°. The velocity of the movement was controlled by the dynamometer that kept the velocity constant at 30, 60, 120 or 180°/s except during the acceleration at movement onset and the deceleration at the end. The torque reached peak levels between 64° and 75° and then fell linearly to the end of each movement while the levels of EMG activity was relatively constant. The decrease of the torque during a concentric knee extension is largely due to the general length–tension relationship in muscle that leads to lowered contractile tension when a muscle shortens. Shortening of the lever arm of the quadriceps muscle from its maximum at about 60° causes a minor part of the decrease. Antagonist muscles, when activated during the movement, will increase their restraints successively during the movements as their contractile tension increases with muscle length and this may contribute slightly to the fall of the torque.

Eccentric movements started with the leg extended to 20°. Enforced by the dynamometer, the knee joint was flexed to an angular position of 90° with the velocity of movements kept constant at the same levels as in the concentric movements. The eccentric movements were resisted by maximal voluntary action of the knee extensor muscles. In contrast to the concentric movement, the torque increased during the movements in consequence to increasing length of agonists and decreasing length of antagonists.

Peak torque in slow concentric movements
Values of peak torque in concentric movements of a low velocity are commonly used for comparisons of muscle strength in isokinetic movements. In the healthy volunteer of Fig. 1, the mean peak torque in three tests at a velocity of 30°/s was 231 Nm (range 223–244 Nm). In the patient of Fig. 2, the mean peak torque was 90 Nm (range 88–91 Nm). Table 2 gives the means ± SDs of peak torque in concentric knee extensions and flexions of the male and female patients and healthy controls. In the female controls, mean peak torque values were 55% of corresponding values for the male controls in both extensions and flexions. In male and female patients, mean peak torque in knee extensions was 55% and 49%, respectively, and in knee flexions 37% and 46%, respectively, compared with controls.

Changes of torque with movement type and movement velocity
As seen in Figs 1 and 2, the torque was larger in eccentric than in concentric movements both in the healthy subject and in the patient. The differences were larger in the patient than in the healthy subject. The figures also show how the torque in concentric movements decreased with increasing velocity of movement. In the healthy subject, the mean peak torque decreased successively from 231 Nm at 30°/s to 157 Nm at the highest velocity, 180°/s. In the patient, the mean peak torque decreased from 90 Nm at the slowest velocity to 26 Nm at the highest velocity. These decreases in peak torque correspond to relative falls of 32% and 71% in the healthy subject and patient, respectively. Thus, the fall was more pronounced in the patient.

Since the torque is corrected for the gravitational force caused by the weight of the leg, the torque curves give the muscle force generated by the agonists reduced by the opposing force generated by the antagonists. Forces produced by the agonists are considered positive and those produced by their antagonists are considered negative. Thus, in the concentric and eccentric movements of Figs 1 and 2, the torque recorded is the torque produced by the agonists, the knee extensor muscles, minus the torque produced by their antagonists. In the concentric movements, the force produced by the agonists acts in the direction of the movements. In contrast, it acts in the direction opposite to the movements in eccentric voluntary actions.

In Fig. 2, at the end of the concentric movement at the
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Fig. 1 Maximum voluntary concentric and eccentric actions of knee extensor muscles at four different constant velocities (30, 60, 120 and 180°/s) in a healthy male volunteer. Torque curves give mean torque (uninterrupted lines), and maximum and minimum torque (dotted lines) from three repeated tests at each velocity. EMG activity is given as mean of rectified and filtered surface EMG recorded from the quadriceps and the hamstring muscle groups. Torque corrected for gravitation. Arrows indicate direction of movements. The highest velocity, the torque became negative. A negative torque means that the torque produced by the antagonists overpowers the torque produced by the agonist. Thus, the patient would not have been able by himself, to complete the concentric knee extension at the highest velocity. The fact that the movements were completed in the tests was due to the action of the dynamometer that was set to complete movements at the preset speed independent of whether they were supported or resisted by forces acting on the dynamometer lever arm.

To compare torque and EMG activity in concentric and eccentric movements of different velocities, data averaged from a selected movement range were used. The selection was made to give a common range in which the velocity of movement was kept constant in all the movements to be compared. The acceleration of a movement up to a high velocity requires a larger angular displacement than the acceleration up to a low velocity. Thus, a common range to all movements will be set by the constant velocity range at the highest velocity tested. With inter-individual variations in movement range and the differences seen in concentric and eccentric movements, the common constant velocity range was found to be 47–75°. Thus, averaged torque and EMG activity in this range were used in comparisons of torque and EMG activity.

Figure 3 gives torque and EMG activity in agonistic and antagonistic muscles in maximal voluntary knee extensor and flexor actions of the male patients and male healthy controls at different constant velocities in eccentric and concentric movements. The torque was lower in the patients than in the controls in extensor and flexor actions (MANOVA, \( P < 0.0001 \)). The agonist EMG activity was also lower in
patients than in controls in extensor (MANOVA, $P < 0.0001$) and flexor (MANOVA, $P < 0.001$) actions. The changes of torque with movement type and velocity were similar in patients and controls. Thus, the torque was generally higher in eccentric than in concentric movements and decreased with velocity in concentric movements. The low torque levels in the patients were combined with low agonist EMG activity. In the patients, the agonist EMG activity decreased with increasing velocity in concentric movements. In the controls, agonist EMG activity increased in concentric knee extensions of high velocity (Komi et al., 1987; Westing et al., 1991) and was higher in concentric than eccentric knee flexor actions.

The antagonist EMG activity in extensor actions was lower in the patients than in the controls in eccentric actions, but the difference was not statistically significant (MANOVA, $P > 0.05$). In concentric extensor actions, the antagonist
EMG in patients and controls was almost the same. In flexor actions, the antagonist EMG activity was lower in patients than in controls (MANOVA, $P < 0.01$). The differences were significant in all of the eccentric movements (at $-180^\circ$/s, $P < 0.0001$; at $-120^\circ$/s and $-60^\circ$/s, $P < 0.001$; at $-30^\circ$/s, $P < 0.01$) and in the slowest concentric movement (at $30^\circ$/s, $P < 0.01$) but not in the faster concentric movements.

In female patients and controls the torque–velocity curves followed patterns similar to those in the male subjects though the torque values were lower in all movements (MANOVA, $P < 0.0001$). On average, in female subjects the torque was ~60% of corresponding values for male subjects in all movements except concentric knee flexions of the patients in which mean torque approached zero and became negative at the highest velocity.

The mean levels of agonist EMG activity in female subjects were 50–70% of corresponding levels in the male subjects. The differences were statistically significant in both extensor ($P < 0.001$) and flexor ($P < 0.01$) actions. In female patients, the mean antagonist EMG was 65–80% of that in male patients; the differences were not statistically significant.

Though the absolute levels of torque and EMG activity differed highly in male and female subjects the relative differences in torque and in EMG activity between eccentric and concentric movements and between movements of different velocity were quite similar. Therefore, data from male and female controls, like those from male and female
Reproducibility in repeated determinations

It is well known that the motor functions vary considerably in patients with spastic pareses due to variation in spastic restraint, spasms and degree of muscle co-ordination. To assess reproducibility in repeated determinations of mean torque and EMG activity in the patients, identical measurements were made on three different days within a period of 2 weeks. Each test included maximal voluntary extensor and flexor actions in eccentric and concentric movements at four velocities. These repeated tests were performed in 15 of the 22 patients (cases 1–7, 10, 11, 15, 17–20 and 22 in Table 1). Figure 4 gives means and standard errors of torque and EMG activity in agonist and antagonists from the three series of measurements in knee extensor actions. None of the differences between individual variables were statistically significant (MANOVA, repeated measures design). A test of the reproducibility in repeated measures in maximal voluntary knee flexor actions gave the same result.

Changes in relative torque

Figure 5 gives plots of relative torque (mean ± 1.0 SEM) as compared with the torque in concentric movement at 30°/s for all patients (n = 22) and controls (n = 22) in the voluntary knee extensor and flexor actions. The relative differences between patients and controls were tested for statistical significance with MANOVA for (i) eccentric action at low velocity (−30°/s) relative to concentric action at the same velocity (30°/s) with Student’s t tests, (ii) torque at −180, −120 and −60°/s relative to torque at −30°/s (eccentric actions), and (iii) torque at 60, 120 and 180°/s relative to torque at 30°/s (concentric actions).

The mean torque generated by knee extensor muscles in slow eccentric actions (−30°/s) compared with slow concentric (30°/s) actions was 85% larger in patients and 28% larger in controls; the difference between the groups was statistically significant (P < 0.0001). The relative changes of the torque with increasing velocity of eccentric actions was slightly different in patients and controls (MANOVA, P < 0.05) due to a small increase of the torque at −180°/s in the patients.

At increasing velocity in concentric extensor action, the torque decreased more in patients than in the controls (MANOVA, P < 0.0001) resulting in significant differences in mean torque at 60°/s (P < 0.01), 120°/s and 180°/s (P < 0.0001). In three patients, negative torque, as illustrated in Fig. 2, was seen at the end of the fast concentric movements but not within the (47–75°) movement range used in the analysis.

In knee flexor actions, the mean torque increased more in patients than in controls (P < 0.05) when changing from slow concentric (30°/s) to slow eccentric (−30°/s) action. The torque was not changed with velocity in eccentric movements in patients or controls. In concentric actions, the torque fell more in patients than in controls with increasing velocity (MANOVA, P < 0.001), giving significant differences in relative torque between patients and controls at 60°/s (P < 0.01), 120°/s and 180°/s (P < 0.0001). In many patients the torque became negative during concentric knee flexions. At 180°/s, torque became negative in 14 patients within the movement range used for analysis (47–75°). It became negative in 12 patients at 120°/s, in four patients at 60°/s, and in two patients at 30°/s.

Changes of relative agonist EMG activity

In Fig. 6, the mean agonist EMG activity is given relative to the agonist EMG in concentric actions at 30°/s, in analogy with the display in Fig. 5. In knee extensor actions, the mean agonist EMG activity in slow eccentric action compared with
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Fig. 5 Torque in eccentric and concentric knee movements of patients with spastic paraparesis and healthy controls at different constant velocities relative to the torque in slow (30°/s) concentric movement. Mean torque determined in a movement range of 47–75° at constant velocities of 30, 60, 120 and 180°/s of eccentric and concentric movements at maximal voluntary effort in knee extensor and knee flexor actions, respectively. Circles and bars give means ± 1 SEM for healthy controls (male and female, n = 22) and spastic patients (male and female, n = 22).

Fig. 6 Agonist EMG activity in isokinetic knee movements at different constant velocities relative to the agonist EMG activity in slow concentric movement (30°/s) in patients with spastic paraparesis and in healthy controls. Eccentric and concentric movements during maximal voluntary action of knee extensor and flexor muscles, respectively. Velocities, movement range, means and SEM as in Fig. 5.

slow concentric action was slightly increased in the patients but unchanged in the controls (P < 0.05). In eccentric actions of different velocities, the agonist EMG did not change in patients or controls. In contrast, the agonist EMG activity in concentric actions differed highly between patients and controls (MANOVA, P < 0.0001). In the controls, it increased with increasing velocity in agreement with previous findings (Komi et al., 1987; Westing et al., 1991); between 60 and 120°/s it increased 15% (P < 0.001). In the patients, it decreased 25% between 30 and 180°/s with significant changes between 60 and 120°/s (P < 0.05) and between 120 and 180°/s (P < 0.01).

In flexor actions, the agonist EMG activity in patients was larger (P < 0.01) in slow eccentric (−30°/s) than in slow concentric (30°/s) movements. In eccentric actions, the agonist EMG activity increased with velocity in the patients but not in the controls (MANOVA, P < 0.05). The largest increase occurred between 120 and 180°/s (P < 0.001). In concentric actions, the agonist EMG activity changed differently in patients and controls (MANOVA, P < 0.05). In the patients it fell slightly between 60 and 120°/s (P < 0.05) and insignificantly between 120 and 180°/s (P = 0.06) while it increased slightly but insignificantly in the controls.

In concentric knee flexions, as mentioned above, the torque fell below zero in many of the patients at the end of the movement range used for the analysis (47–75°). When the torque became negative, the movement continued by action of the dynamometer. Although some agonist action continued to the ends of movements, as judged from the EMG recordings, the movement with negative torque can then be regarded as mechanically passive. Therefore the part of the movement range with negative torque at any velocity was removed and the relative changes of agonist EMG in each individual determined for the part of the range with positive torque at all velocities. Figure 7 gives the relative changes of the agonist EMG recalculated in this way, together with the corresponding changes shown in Fig. 6. The differences between the relative changes in the controls...
and the recalculated ones for the patients were significant (MANOVA, $P < 0.01$). The relative changes determined from phases with positive torque and the full range did not differ significantly (MANOVA, $P > 0.05$). Neither did the relative changes of mean agonist EMG change significantly in the controls after cutting off the movement range to approximately the same degree as in the patients with negative torque.

**Ratio between antagonist and agonist EMG activity**

The mean ratio between antagonist and agonist EMG activity in voluntary knee extensor actions are shown in Fig. 8. In the controls, the ratio was virtually the same in eccentric and concentric actions at different velocities. It indicates that the antagonist EMG activity was proportional to the varying levels of agonist EMG activity in the controls at different velocities and to the low level of agonist EMG in eccentric actions in the patients (Fig. 3).

The EMG activity recorded over the antagonists is due, in part, to cross talk from the agonists that are more strongly activated than the antagonists. In part, it is due to active muscle restraint by the antagonists stabilizing the joint and limiting sliding displacement of the tibia (Baratta et al., 1988; Hirokawa et al., 1991). Cross talk from agonists can be expected to be proportional to the degree of agonist activity, and there should also be a trend to proportionality between antagonist and agonist activation due to the changing demand of active antagonist restraint with changing contractile tension in the agonist muscles. In the patients, the ratio between antagonist and agonist EMG activity was larger in concentric than eccentric actions and it increased with velocity, unlike that in the controls (MANOVA, $P < 0.0001$).

Also, in flexor actions, the ratio was virtually the same in the controls at different velocities of eccentric and concentric actions. In the patients, the ratio increased in concentric actions at 120 and 180°/s, unlike that in the controls (MANOVA, $P < 0.0001$). In the eccentric flexor actions, the mean ratios were slightly lower in the patients than in the controls, but the differences were not significant. The velocity-dependent increases of the ratio between antagonist and agonist EMG seen in Fig. 8 appeared in parallel with decreased levels of agonist EMG (Figs 6 and 7).

**Discussion**

The results of the present study show that the dynamic motor capacity in patients with spastic paraparesis is severely compromised in concentric movements, especially at high movement velocities. In contrast, voluntary strength may be relatively well preserved in eccentric movements. By definition the agonist contraction in an eccentric action...
opposes but does not overpower external pulling forces imposed upon the agonistic muscles by potential or kinetic energy. If the opposing muscle force becomes equal to the external forces, the movement comes to a stop and the contraction becomes isometric. If the muscle force becomes so large that it overpowers the external forces, the muscle will shorten and its action becomes concentric. In concentric movements there are shortening contractions in the agonists while they produce movements, and at the same time stretch is imposed upon the opposing antagonists. In eccentric movements there are lengthening contractions in the agonists while they oppose movements produced by external forces that give pull on the agonists, and there is no stretch of the antagonists, which are shortened by the movement.

In the movements of daily life, the maximal motor capacity determined in the present study is not used more than occasionally in normal man. The intensity of contractions in both eccentric and concentric actions is commonly submaximal and graded to fit functional demands. In consequence, a slight or moderate decrease of the maximal capacity may not severely compromise the motor functions of daily life. With increasing weakness, a successively larger part of the maximal capacity will have to be used until the capacity is no longer sufficient for the functions. Since concentric muscle actions at high velocity are the ones that are most severely incapacitated in spastic paresis, they will be compromised first. This will lead to slowing down of movements, as often seen in patients with spastic paresis. With advancing deterioration there will be a successively more pronounced reliance upon eccentric actions since they are not so severely affected. In eccentric knee flexor actions the voluntary agonist activation may actually get some support by exaggerated stretch reflexes in the flexor muscles. A higher agonist EMG activity was seen in eccentric actions than in concentric actions in spastic patients. It indicates that stretch reflexes in flexor muscles may have been added to the voluntary activation in eccentric flexor actions of the patients. There were no signs of a similar support by stretch reflexes in eccentric knee extensor actions. The dissimilar influence of stretch on eccentric actions of knee flexor and extensor muscles is most probably due to the differential reflex effects of group II afferent fibres from secondary spindle endings that facilitate flexor motoneurons but inhibit extensor motoneurons (Burke et al., 1970, 1971). The inflow from primary and secondary endings in flexor muscles will thus act in synergy while the inflow from these endings in extensor muscles will have opposing effects.

The co-contraction of antagonistic muscles in knee joint movements are used for stabilizing the joint and limiting sliding displacement of the tibia (Baratta et al., 1988, Hirokawa et al., 1991), as well as for the control of pressure distribution on the joint surfaces (An et al., 1990). The higher the contractile force of the agonist muscles, the larger the need of antagonist co-contraction will be. However, it was not possible to judge the degree of co-contraction from the EMG recordings. In recordings with surface electrodes during maximal voluntary contractions, estimations of the true activity in the relatively inactive antagonist muscles in the vicinity of the highly active agonist muscles are uncertain due to cross talk from the agonists proportional to their activity (Koh and Grabner, 1992). The ratio between antagonist and agonist EMG activity was virtually the same in eccentric and concentric movements of different velocities in the healthy controls. In the eccentric actions of the patients, in spite of much lowered contractile tension as compared with the controls, the ratio was virtually the same as in the healthy controls. Thus, it may be assumed that not only the cross talk but also the antagonist co-contractions were roughly proportional to the agonist activity in all the different movements of the controls and in the eccentric movements of the patients in which there was no stretch imposed upon the antagonists.

In contrast, the ratio between antagonist and agonist EMG activity increased markedly in concentric movements of the patients since the agonist EMG activity decreased while the antagonist EMG increased. The lowered activity in the agonist muscles can be expected to decrease the cross talk from them to the antagonist muscles. Thus, the increase in the recording of EMG over the antagonist muscles must be due to an increase of the activation of the antagonist muscles. Since the increase appeared during stretch of the antagonist muscles, and showed a clear velocity dependence, it seems very likely that the increase was due to activation by muscle stretch reflexes.

The understanding of the role of the fusimotor system in normal motor control in man has advanced greatly from studies with direct recording of the afferent inflow from muscle stretch receptors (Valbo et al., 1979). Studies with recordings from muscle spindle afferents in spasticity are sparse, and those made have given conflicting results. Thus, Hagbarth et al. (1973) could not find any signs of increased sensitivity to ramp stretch in two spastic patients and they suggested that the exaggerated stretch reflexes were due to abnormal spinal processing of a relatively normal afferent input in response to stretch. Szumski et al. (1974), in studies of the spindle responses in the relaxation phase of twitch contractions in seven spastic patients, found an increased muscle spindle sensitivity. This finding supports an old concept that the exaggerated stretch reflexes are related to an excessive input from the muscle spindle although this does not exclude abnormal central processing. It is, however, not possible to draw any safe conclusions from the limited materials considering the large inter-individual differences in spasticity. Besides, the findings in the spastic patients only showed reactions at rest. As pointed out by Burke (1980), the reactions in the fusimotor system at rest are relatively uninteresting since it is a motor system. Its reactions at rest are not applicable during active movements, and no defects of fusimotor activity in voluntary movements of spastic patients have yet been defined.

The fact that muscle stretch reflexes were activated in the spastic patients, but not in the healthy subjects, during
concentric voluntary movements of precisely the same range and velocity is therefore assumed to be due to enhanced inflow from the muscle stretch receptors or to abnormal central processing of a virtually normal inflow from the receptors, or possibly of a combination of these two mechanisms.

In healthy subjects, the level of activation of the quadriceps muscle in concentric knee extensions increases with velocity (Komi et al., 1987; Westing et al., 1991). This increase has been assumed to be a consequence of a lower maximal muscle tension in fast compared with slow concentric movements. The lower tension may cause a smaller afferent inflow from tendon organs. Thus, their inhibitory effects upon homonymous motoneurones will be less pronounced. In the spastic patients, there was no increase of the quadriceps EMG activity in concentric knee extensions at fast speeds. Instead, the agonist EMG activity decreased with increasing movement velocity. Also in concentric knee flexions at fast speeds there was a fall of agonist EMG activity not seen in the normal controls.

This fall of agonist EMG activity during concentric movements may be explained by the inflow in Ia afferents from spastic antagonist muscles that inhibit the voluntarily activated agonist motoneurons via Ia inhibitory interneurons. A preserved or even enhanced reciprocal inhibitory influence of this type has been shown after lesions of the spinal cord in man (Ashby and Wiens, 1989) and in cerebral palsy (Berbrayer and Ashby, 1990).

The reciprocal inhibition from antagonist muscles to motoneurones of the agonist muscles discussed above has to be distinguished from the normal reciprocal inhibition of antagonist motoneurones associated with voluntary agonist contraction. The latter is controlled in parallel by Ia afferents from the agonist muscles and descending supraspinal commands activating Ia inhibitory interneurons projecting to the motoneurones of the antagonists muscles (Tanaka, 1974; Iles, 1983; Crone et al., 1985). It limits the reflex activation of the antagonist muscles when they are stretched during active movements. In studies of reciprocal inhibition in patients with spasticity, using H-reflex conditioning from antagonist muscle afferents, the results have indicated reduced or absent inhibition of the H reflex (Yanagisawa et al., 1976; Yanagisawa, 1980; Gottlieb et al., 1982; Artieda et al., 1991; Calancie et al., 1993).

The findings of the present study are compatible with the idea that the normal reciprocal inhibition from agonist to antagonist motoneurons is decreased in spastic paresis during voluntary dynamic actions of high effort. In contrast, a reciprocal inhibition in the opposite direction seemed to be present in the spastic patients, i.e. from antagonist to voluntarily activated agonist motor neurons.

The latter mechanism is very likely the basis for an antiparetic effect seen in response to therapy with two antispastic drugs, tizanidine (Knutsson et al., 1982) and baclofen (Knutsson and Mårtensson, 1984). Besides reduction of spastic stretch reflexes in passive movements, a prominent increase of maximal voluntary strength, i.e. reduced paresis, was seen in concentric movements. It was combined with markedly increased levels of agonist EMG activity, while the reduction of the antagonist EMG was too small to explain the increase in strength as an effect of reduced antagonist restraint. Assuming that the reduced voluntary strength in concentric movements of spastic patients is due, in part, to a reciprocal inhibition of agonist motoneurons emanating from stretch reflex afferents in the antagonists, a depression of the stretch reflexes may also depress this inhibition of agonist motoneurons. A depressed reciprocal inhibition of this type would be the result, independent of whether the antispastic effect was a depression of the fusimotor activity or caused by presynaptic inhibition at the terminals of primary stretch afferents. The demonstration of reciprocal inhibition of voluntarily, submaximally activated agonist motoneurons by electrical activation of low threshold afferents from an antagonist muscle in patients with spinal lesions (Ashby and Wiens, 1989) gives strong support for the concept that reciprocal inhibition of agonist motoneurons from antagonists can suppress voluntary activation in spinal spasticity. The demonstration in the present study of reduced agonist EMG activity in parallel with stretch-related activation in the antagonists during maximal voluntary activation gives further support for such a segmental mechanism in the pathogenesis of muscle weakness in concentric actions.

Acknowledgements
We wish to thank Åsa Brötell for her excellent technical assistance and Elisabeth Berg for useful discussions relating to the statistical analysis. This study was supported by grants from the Karolinska Institute Foundations and from Torsten and Ragnar Söderbergs Foundation.

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Received February 6, 1997. Revised April 14, 1997. Accepted May 3, 1997