

# Stretch reflexes in the upper limb of spastic man

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**SUMMARY** The reflex response to stretch has been studied in the upper limb of 20 spastic patients. The amplitude of reflex EMG was found to be closely related to the velocity of stretch. Reflex EMG in biceps and triceps muscles was augmented by increasing the length of the muscle, although minimal inhibition of reflex EMG in biceps was obtained in two patients by extreme stretch. The clasp-knife sensation in the upper limb cannot be related to autogenic inhibition but may be explained by the characteristics of a velocity-dependent reflex in which limb movement is braked by the mechanical effect of increasing muscle tension. It is suggested that this be called the pseudo-clasp-knife reaction to distinguish it from the clasp-knife phenomenon of the quadriceps muscle, since the underlying neurophysiological mechanism is quite different.

The build-up of resistance as a muscle is stretched and the subsequent melting of resistance as the muscle is stretched further is known as the clasp-knife phenomenon, and is considered an important, if not essential, feature in the diagnosis of spasticity (Landau, 1969). While the clasp-knife phenomenon can be found most readily in the extensor muscles of the lower limb, it is also accepted that it occurs in the upper limb of spastic patients (Walshe, 1963; Denny-Brown, 1966). The clasp-knife phenomenon has been considered to depend on the presence of autogenic inhibition of the stretch reflex, generally attributed to the reflex effects of group Ib fibres from the Golgi tendon organs. Recently it has been shown that the inhibition of the reflex activity responsible for the clasp-knife phenomenon in the quadriceps muscle of spastic patients can be related to the activation of secondary endings which are inhibitory to extensor muscles and facilitatory to flexor muscles in the lower limb (Burke, Gillies, and Lance, 1970; Burke, Andrews, and Ashby, 1971), effects that may be released from brain-stem inhibitory control in spasticity (Holmqvist and Lundberg, 1959; Burke, Knowles, Andrews, and Ashby, 1971).

This study attempts to clarify the clasp-knife sensation as it is found in the flexor and extensor muscles of the upper limb of hemiplegic patients and

to compare the underlying neurophysiological mechanisms with those found in the lower limb in spasticity.

## METHODS

The study involved 18 patients with cerebral spasticity, one patient with a high cervical spinal cord lesion, and one suffering from motor neurone disease. Of the patients with cerebral spasticity, the cause was cerebral thrombosis or embolism in 10, cerebral haemorrhage in two, subarachnoid haemorrhage in three, and head injury in three.

The patients were examined from three weeks to two years after the onset of the neurological deficit and all showed a typical 'pyramidal' pattern of weakness, increased stretch reflexes, exaggerated tendon jerks, and extensor plantar responses. The patients were examined lying supine with the shoulder girdle relaxed while stretch reflexes were induced by manual movements of the limb. Surface electrodes separated by 10 cm were placed over the biceps and triceps muscles to record the electromyogram (EMG) induced by muscle stretch. These potentials were also integrated (time constant 0.2 sec). The EMG was monitored on an oscilloscope to detect artefact and to assess any spread of electrical activity from antagonistic muscles.

The distances between the origins and insertions of the biceps and triceps muscles were measured from radiographs of the limb in various degrees of flexion of the elbow joint. A linear relationship was found between the length of the muscles and elbow joint angle (biceps 0.8 mm/degree, triceps 0.3 mm/degree). Changes in muscle length may therefore be measured in degrees of joint

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movement. Elbow joint angle was measured by a goniometer, the output of which was differentiated (time constant 5 msec) to provide a voltage proportional to angular velocity.

Direct EMG, integrated EMG, elbow joint angle, and angular velocity were recorded simultaneously using a four channel Offner Dynograph for the biceps and a Grass polygraph for the triceps muscles. The joint angle trace was also displayed on a second oscilloscope so that a constant excursion of the limb could be reproduced. The resistance developed during the stretching movement was measured by a force transducer, consisting of a DC-activated four arm strain-gauge bridge bonded to a metal bar, which was attached to the handle by which the limb was moved.

Four types of stretching movement were employed. Full range linear movements of differing velocities were used to define the relationship between reflex EMG and velocity. The effect of muscle length on reflex EMG was tested by moving the limb in three sequential steps of equal amplitude and velocity and by performing small oscillations of the limb (20°-30° amplitude) at different centres of oscillation. In addition, sinusoidal movements of the limb through its full range were employed to examine the relationship of reflex EMG to the phase of a sinusoidal cycle. A smooth sinusoidal excursion of the muscle could be obtained by matching the movement of the limb with the sine wave produced by a Hewlett Packard function generator displayed on an oscilloscope. Cycles in which the recorded joint angle movement was not truly sinusoidal were rejected. The relationship of the peak EMG of each muscle to the phase of the sinusoidal movement could be obtained from the Dynograph record. Where the reflex activity was small, the EMG produced by 10 to 20 stretching cycles was rectified and averaged by a fixed-programme averaging computer, and the readout photographed with a Polaroid Land camera.

The interpretation of the results of sinusoidal stretching has been discussed elsewhere (Burke, Andrews, and Gillies, 1971) and is summarized in Fig. 1. Briefly, if reflex EMG were directly proportional only to the velocity of stretch the peak of EMG would appear in the middle of the stretching phase where velocity is greatest. If reflex EMG were directly proportional only to muscle length the peak would be expected at the end of the stretching phase where muscle length is greatest. Reflex activity related to both velocity and length would have its peak somewhere between these points depending on the proportion of each component. If reflex EMG were inhibited by increasing muscle length (as in the quadriceps muscle) the peak of EMG would be expected in the first half of the stretching phase.

The line of best fit of the EMG-velocity relationship was calculated by regression analysis and the significance of the relationship assessed by determining the correlation coefficient. Student's *t* test was used to compare the means of the intercepts on the *x*-axis and the slopes of the regression lines of the two muscles.

#### RESULTS

**RELATIONSHIP BETWEEN REFLEX EMG AND VELOCITY OF STRETCH** The reflex EMG of biceps and triceps

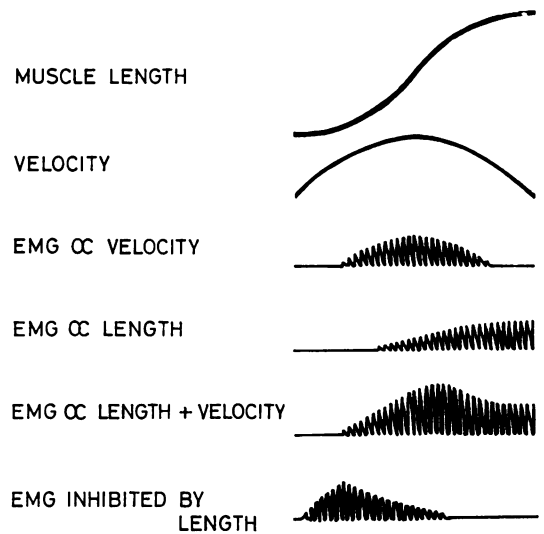


FIG. 1. Diagrammatic illustration of the relationship between reflex EMG and the phases of a sinusoidal stretching movement in four theoretical situations.

muscles evoked by linear stretching movement was studied over a range of velocities in 15 patients. In all patients the reflex EMG of both muscles increased in amplitude with increasing velocity of stretch (Figs. 2, 3, 4). The relationship between reflex EMG and velocity of stretch was linear over the range employed (0°-500°/sec) and the degree of scatter was small, the correlation coefficients being significant in all but one instance.

A threshold velocity below which no reflex EMG was evoked could be established in mildly spastic patients. In the more severely spastic patients reflex EMG occurred even with the slowest movements. There was no significant difference between the means of the threshold velocities of the two muscles but reflex EMG augmented to a greater extent with velocity in the triceps ( $t = 3.88$ ,  $P < 0.001$ ).

**RELATIONSHIP BETWEEN REFLEX EMG AND MUSCLE LENGTH** Maintained stretch of the biceps muscle produced a sustained static reflex in seven patients. This was always greatest in amplitude when the muscle was at its greatest length (Fig. 5). In the other eight patients no static stretch reflex was recorded in the biceps muscle. A static stretch reflex was not recorded from the triceps muscle in any of the 15 patients.

The effect of muscle length on reflex EMG was also examined by performing small oscillating

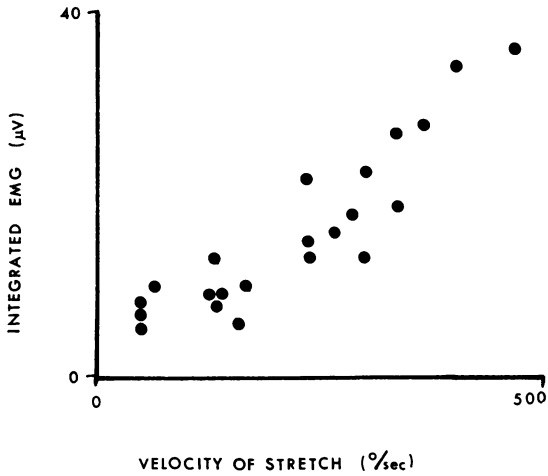


FIG. 2. Velocity dependence of the stretch reflex in biceps muscle. Each point represents the integrated EMG recorded for a linear movement through the full range of stretch.

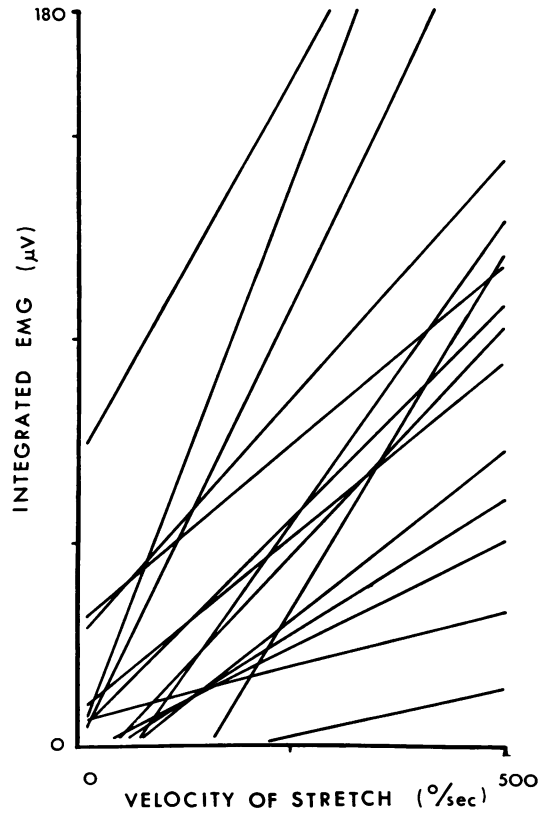


FIG. 4. Velocity dependence of the stretch reflex in the triceps muscle of 15 spastic patients. The lines of best fit were calculated from multiple recordings.

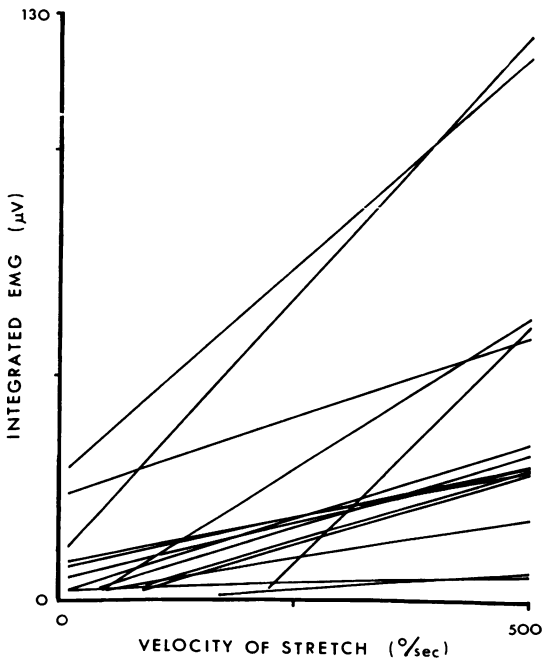


FIG. 3. Velocity dependence of the stretch reflex in the biceps muscle of 15 spastic patients. The lines of best fit were calculated from multiple recordings.

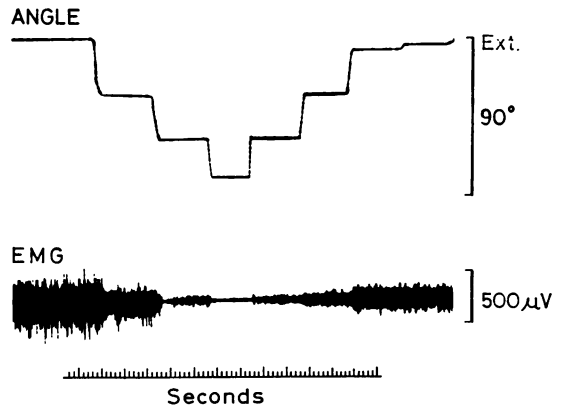


FIG. 5. Facilitation of the static stretch reflex of biceps muscle with increase in muscle length. In this, and subsequent illustrations, stretch of the muscle is indicated by upward displacement of the angle trace.

movements of constant amplitude and frequency at different centres of oscillation. In the biceps muscle of eight patients the reflex EMG produced by such stretching reached its height when the muscle was oscillated at its greatest length (Fig. 6). In two patients the reflex EMG was facilitated by increasing length until the position of maximum stretch at which it diminished slightly. This diminution in reflex response occurred only in the last few degrees of extension, well beyond that position at which the clasp-knife sensation was felt. In five patients muscle length had no effect on the amplitude of the EMG.

The reflex EMG produced by such stretching of the triceps muscle, was greatest when the muscle was oscillated at its greatest length in seven patients and was unaffected by muscle length in four. In the

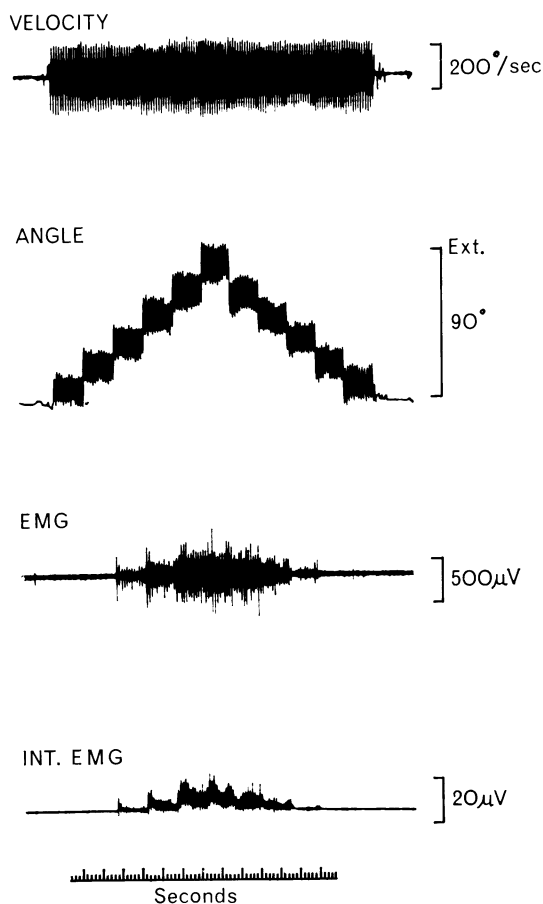


FIG. 6. Facilitation of the stretch reflex of biceps muscle with increase in muscle length demonstrated with oscillating movements.

remaining four patients insufficient EMG was produced by movements of such small amplitude for an assessment to be made.

The effect of muscle length on reflex EMG was also tested in some patients using step-wise linear stretching movements of equal velocity and amplitude. In both biceps and triceps muscles reflex EMG became maximal in the last step of the stretching movement (Fig. 7).

It may be concluded that reflex EMG is facilitated by increasing muscle length in the majority of patients in both biceps and triceps muscles.

**RELATIONSHIP OF REFLEX EMG TO PHASES OF A SINUSOIDAL MOVEMENT** The relationship of the peak of the reflex EMG to the phase of a sinusoidal movement was examined in 13 patients. The findings were the same for both biceps and triceps muscles. In every case, the peak of the reflex EMG appeared in the second half of the stretching phase (Fig. 8). When the frequency of the sinusoidal movement was increased the peak of the reflex EMG moved nearer to the middle of the stretching phase, approximating the position of maximum velocity of stretch. These findings suggest that the reflex EMG is proportional both to the velocity of stretch and to the length of the muscle, the earlier peak at higher frequencies being due to an increase in the velocity component of the reflex EMG. The peak of the reflex EMG never appeared in the first half of the stretching phase which would be the expected position of reflex EMG if it were inhibited by increasing muscle length.

**RESISTANCE TO STRETCH DURING ELICITATION OF CLASP-KNIFE SENSATION** In five patients resistance to stretch, elbow joint angle, angular velocity, and rectified EMG were recorded during the clinical elicitation of the clasp-knife sensation in the upper limb. The movement was interrupted by the clasp-knife reaction and was never linear (Fig. 9). In every case there was an initial rise in velocity accompanied by an increased amplitude of reflex EMG and followed by an increase in the resistance to stretch. The velocity then fell and the movement proceeded at lower velocity with correspondingly lower resistance to passive stretch, the EMG continuing throughout. An analysis of the relationship between velocity and reflex EMG shows that EMG per unit velocity actually increases throughout the movement suggesting that the facilitatory effect of increasing muscle length is greater than any inhibitory effect related to muscle length or tension.

#### DISCUSSION

The amplitude of reflex EMG produced by stretching

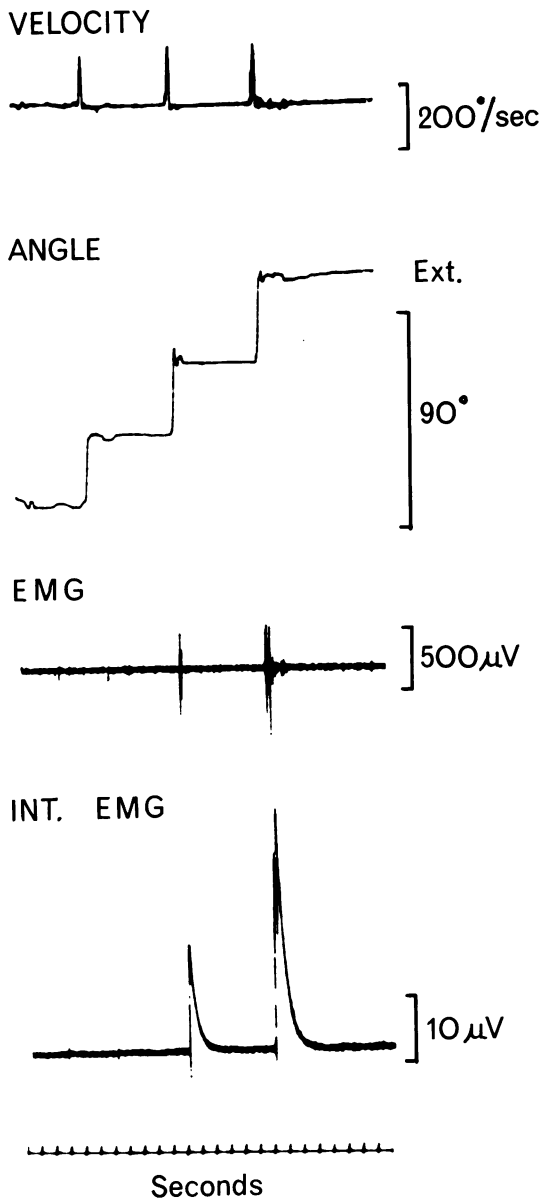


FIG. 7. Facilitation of the stretch reflex of biceps muscle with increase in muscle length demonstrated with sequential stretching movements of approximately equal velocity.

movements of biceps and triceps muscles in spastic patients is shown to be closely related to the velocity of the stretching movement. A similar relationship has been demonstrated in other muscles of spastic patients (Rushworth, 1960; Shimazu, Hongo,

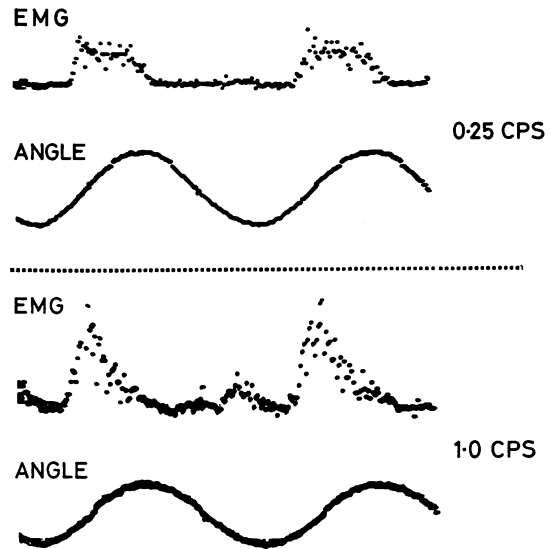


FIG. 8. Relationship between reflex EMG of biceps muscle and the phases of a sinusoidal stretching movement shown by average of 10 cycles of rectified EMG and joint angle. Stretch of the muscle is indicated by upward displacement of the angle trace. The EMG occurs in the second half of the stretching phase suggesting that it is facilitated by increasing muscle length. At the faster frequency there is an increase in the component related to the velocity of stretch.

Kubota, and Narabayashi, 1962; Leavitt and Beasley, 1964; Burke, Gillies and Lance, 1970, 1971), and is compatible with the concept of increased sensitivity of the velocity-responsive primary spindle endings related to dynamic fusimotor drive (Jansen, 1962).

Reflex EMG of biceps and triceps muscles in spastic man is facilitated by increasing muscle length in the majority of patients. This can be demonstrated by step-wise linear stretches, by oscillatory movements about different limb positions, and by the relationship of the peaks of reflex EMG to the phases of a sinusoidal movement. In addition, where EMG is found in response to static stretch of the biceps muscle, it is always maximal when the muscle is fully stretched. These findings in the upper limb of spastic man are quite different from those in the lower limb where reflex EMG is facilitated by increasing muscle length in the hamstring muscles (Burke *et al.*, 1971) but inhibited by increasing muscle length in the quadriceps muscle (Burke *et al.*, 1970) and must reflect different underlying neurological mechanisms. The length-dependent inhibition in the quadriceps muscle of spastic man can be satisfactorily explained by the reflex action of second-

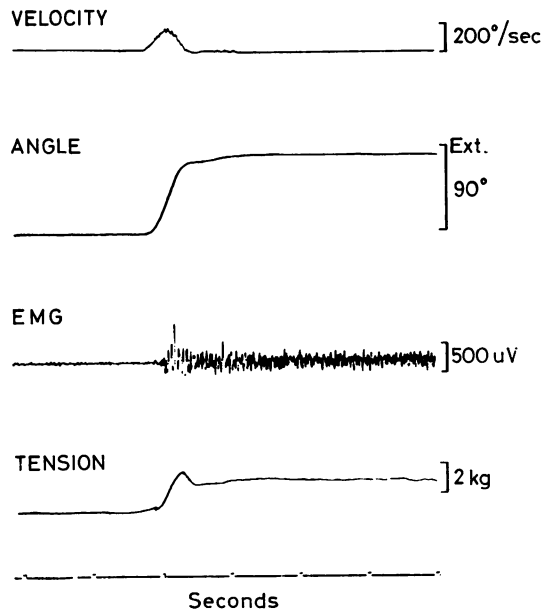


FIG. 9. Relationship of velocity of movement, reflex EMG and resistance to passive stretch during the elicitation of the clasp-knife reaction in the upper limb. The clasp-knife sensation coincides with the peak in the tension trace.

dary spindle endings which are inhibitory to extensor and facilitatory to flexor motoneurons in the lower limb of spastic man (Burke *et al.*, 1970; Burke *et al.*, 1971), an effect that may be released from brainstem control in spasticity (Holmqvist and Lundberg, 1959; Burke *et al.*, 1971). It is not known whether the secondary endings have similar reciprocal effects in the upper limbs of man but these studies suggest that, if secondary endings are inhibitory to the motoneurons of biceps (antigravity muscle) or of triceps (limb extensor muscle), the inhibition of reflex EMG is negligible in comparison with that seen in the lower limbs, and that the changes in reflex EMG with length are dominated by the facilitatory effects from the primary ending.

Autogenic inhibition might also be expected from the Ib fibre input from Golgi tendon organs (Granit, 1950) and has been considered capable of causing almost total inhibition of reflex EMG in spastic states if the muscle develops sufficient tension as it is stretched (Jansen, 1962; Rushworth, 1964; Patton, 1965). The failure to demonstrate autogenic inhibition supports the concept that Golgi tendon organs respond more readily to active contraction of muscle fibres than to passive tension (Jansen and Rudjord, 1964; Houk and Henneman, 1967; Houk,

Singer, and Goldman, 1970) and are thus able to modulate reflex EMG but are unable to abolish it. The smaller overall response of Golgi tendon organs to passive tension (Stuart, Goslow, Mosher, and Reinking, 1970) may account for the small decrement in reflex EMG seen in extreme stretch of biceps muscles in two patients.

The clasp-knife phenomenon of spastic man has been considered to depend upon the presence of autogenic inhibition of reflex EMG (Patton, 1965). In the quadriceps of spastic man autogenic inhibition has been clearly demonstrated and fully accounts for the clasp-knife phenomenon. In the upper limb, however, a clasp-knife sensation can be obtained when autogenic inhibition cannot be demonstrated. Clearly this clasp-knife sensation must have a different explanation.

The amplitude of the reflex EMG of both biceps and triceps muscles has been shown to be closely related to the velocity of a stretching movement. The tension that develops during a dynamic stretch is largely determined by this reflex contraction (Herman, 1970) and a close relationship, in spasticity, between the velocity of stretch and the developed muscle tension has been established by several authors (Rondot, Dalloz, and Tardieu, 1958; Foley, 1961; Herman and Schaumberg, 1968; Herman, 1970). Thus at any instant the velocity of the movement will dictate the amount of reflex EMG and the subsequent muscle tension.

A detailed examination of the temporal relationship of tension, EMG, and velocity as the clasp-knife sensation is elicited in the upper limb offers an alternative explanation for the sensation of resistance which later gives way. As the velocity of movement builds up, reflex EMG increases in parallel. After a brief delay the tension also increases and the velocity of the movement is thus retarded. The reduction in velocity is accompanied by reduced EMG and tension and the movement then proceeds more slowly. The reflex EMG is not inhibited in this final phase but continues until, or even beyond, the end of the movement.

It is therefore possible to explain the clasp-knife sensation found in the biceps and triceps muscles of spastic patients solely on the basis of a velocity dependent reflex. The presence of an additional inhibitory factor from the Golgi tendon organ cannot be excluded but an examination of the relationship between EMG and velocity shows that the EMG per unit velocity increases throughout the movement so that the facilitatory effect of length predominates over any inhibition related to passive tension.

This form of clasp-knife sensation must be clearly distinguished from that found in the quadriceps muscle since it implies a different underlying neuro-

logical mechanism. When autogenic inhibition can be demonstrated (as in the quadriceps muscle) it appears appropriate to use the descriptive term 'clasp-knife phenomenon' but when autogenic inhibition cannot be demonstrated and the sensation can be related simply to the velocity of movement it would be preferable to use an alternative term such as 'pseudo-clasp-knife reaction' (Herman, 1970). Such a distinction can be made at the bedside: the hypotonia and pendular knee-jerk encountered in a spastic patient when the lower limb has been flexed to 90° has no counterpart in the upper limb.

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