Increased sensory outflow from atrophying muscles and muscles after functional activity

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I would like to mention briefly some of our results dealing with phenomena which, although not directly included in the subject of this Symposium, may have pertinent bearing on a number of questions concerning muscle afferents and central nervous system functions. This concerns the 25—33 % of myelinated sensory fibres not allotted to proprioceptive end-organs, but ending in the skeletal muscle as free sensory nerve endings (Barker, Ip & Adal, 1962; Zelená & Hník, 1963).

The increased sensory outflow from muscles atrophying after tenotomy (Kozak & Westerman, 1961; Hník, Beránek, Vyklický & Zelená, 1963) and chronic ventral root section (Hník, 1964*c*) indicated that some change, or changes, occurring in the atrophied muscles cause an increase in the spontaneous discharge of sensory endings in the muscle. We have shown recently that this increased sensory activity is apparently due to the appearance of spontaneous activity in a group of non-proprioceptive fibres, which are not discharging spontaneously under analogous conditions in control muscles (Hník & Payne, 1965*a*). It seems unlikely that these spontaneously active fibres come in fact from proprioceptive endings which had ceased to respond to stretch, since the increase in overall activity in the muscle nerve per mm stretch after chronic ventral root section is considerably higher than that of control muscles (Hník, 1964*c*). Furthermore, it is possible to obtain an increase in sensory outflow from muscles without spindles (Zelená & Hník, 1960) by sectioning ventral roots and allowing sufficient time for muscle atrophy to develop (Hník, 1964*c*). It thus appears that the sensory outflow from muscles can be increased, even when they do not contain proprioceptor end-organs, but only free sensory nerve endings not discharging spontaneously prior to ventral root section (Hník, 1964*b*).

Presuming that there is a system in the muscle which is activated by metabolic, and perhaps circulatory changes taking place during atrophy, it became of obvious interest to see whether there would be an increase in sensory outflow after muscle activity. After some preliminary experiments (Hník, Hudlická &

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Štulcová, 1964) further evidence was obtained showing that even a 5 sec isometric tetanus leads to a very marked increase in the sensory outflow from normal muscles, which lasts for several minutes (Hník & Payne, 1965*b*). Isotonic contraction performed under otherwise identical conditions leads to a very much smaller increase.

Although it is tempting to ascribe the increase in sensory outflow in muscle atrophy and that after muscle activity to the same group of fibres, namely the surplus non-proprioceptive myelinated sensory nerve fibres, it is too early to draw any definite conclusions. We are at the present time trying to identify the group of fibres responsible for this increase. It is, however, perhaps worthwhile to consider the general implications of these findings.

From the pathophysiological point of view, a pathological process (namely muscle atrophy) has actually been shown to cause an increased sensory outflow from the affected region. Such nervous activity has previously been assumed in various disorders (e.g. gastric ulcers), but never actually been demonstrated. In fact, there is some indication even of a 'vicious circle' mechanism being involved in muscle atrophy due to tenotomy, since it is possible to slow down the rate of muscle atrophy by dorsal root section (Hník, 1964*a*). Furthermore, such a longlasting increase in sensory outflow from the periphery has evidently an effect on synaptic functions even at the spinal level, as it has been shown that after tenotomy the monosynaptic reflex response (Beránek & Hník, 1959; Kozak & Westerman, 1961; Beránek, Hník, Vyklický & Zelená, 1961), is considerably enhanced.

On the other hand, the transient increase in sensory outflow after muscle activity, especially that performed under isometric conditions, might play an important role in various regulatory mechanisms (affecting circulation, respiration and even, perhaps development of muscle hypertrophy). But these points are, of course, as yet purely hypothetical.

References

Barker, D., Ip, M. C. & Adal, M. N. (1962). A correlation between the receptor population of the cat's soleus muscle and the afferent fibre-diameter spectrum of the nerve supplying it. In *Symposium on muscle receptors*, ed. Barker, D. pp. 257–261. Hong Kong University Press.

Beránek, R. & Hník, P. (1959). Long-term effects of tenotomy on spinal monosynaptic response in the cat. *Science*, *130*, 981–982.

Beránek, R., Hník, P., Vyklický, L. & Zelená, J. (1961). Facilitation of the monosynaptic reflex due to long-term tenotomy. *Physiol. bohemoslov.*, *10*, 543–552.

Hník, P. (1964*a*). The effect of deafferentation upon muscle atrophy due to tenotomy in rats. *Physiol. bohemoslov.*, *13*, 209–215.

Hník, P. (1964*b*). Functional characteristics of free nerve endings and atypical spindles after muscle reinnervation in very young rats. *Physiol. bohemoslov.*, *13*, 216–219.

Hník, P. (1964*c*). Increased sensory outflow from de-efferented muscles. *Physiol. bohemoslov.*, *13*, 405–410.

Hník, P., Beránek, R., Vyklický, L. & Zelená, J. (1963). Sensory outflow from chronically tenotomized muscles. *Physiol. bohemoslov.*, *12*, 23–29.

Hník, P., Hudlická, O. & Štulcová, B. (1964). Activité afférente du muscle strié en rapport avec des changements circulatoires intramusculaires. *J. Physiol., Paris*, *56*, 569.

Hník, P. & Payne, R. (1965*a*). Spontaneous activity in non-proprioceptive sensory fibres from de-efferented muscles. *J. Physiol.*, *180*, 25–26 P.

Hník, P. & Payne, R. (1965*b*). Increased sensory outflow following muscle activity. *J. Physiol.*, in the Press.

Kozak, W. & Westerman, R. A. (1961). Plastic changes of spinal monosynaptic responses from tenotomized muscles in cats. *Nature, Lond.*, *189*, 753–755.

Zelená, J. & Hník, P. (1960). Absence of spindles in muscles of rats reinnervated during development. *Physiol. bohemoslov.*, *9*, 373–381.

Zelená, J. & Hník, P. (1963). Motor and receptor units in the soleus muscle after nerve regeneration in very young animals. *Physiol. bohemoslov.*, *12*, 277–290.

Additional communications by extra participants

Motoneurone excitability in patients with abnormal reflex activity

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Introduction

The excitability of spinal motoneurones was determined by the recovery of the H-reflex to preceding electrical stimulation in patients with spasticity, rigidity and spinal shock.

Method

The H-reflex was elicited from the tibial nerve and led off from the calf muscles. The recovery was determined with pairs of stimuli of equal strength to produce H-reflexes just above threshold and maximum H-reflexes and the recovery was followed to 4 seconds.

Results

The recovery of excitability in *normals* (Fig. 1, upper) showed an early return of the test reflex 5–20 msec after the conditioning stimulus followed by a period in which the reflex could not be evoked. The rising phase of the second facilitation showed a "hump". A similar hump has been found by Bianconi, Granit & Reis (1964) in cats and was attributed to facilitation of motoneurones by the phasic response of primary afferents at the start of relaxation of the muscle after the conditioning reflex contraction.

The second period of facilitation was followed by a depression which in analogy to the findings of Bianconi *et al.* (1964) in cats is attributed to the inhibitory effects of secondary afferents upon motoneurones to extensor muscles. Four seconds after the conditioning stimulus the test reflex had approached 90 per cent of the amplitude of the conditioning reflex.

The patients with *spasticity* showed enhanced recovery from intervals of 80 msec to 2 seconds. With stimulus strengths to produce H-reflexes near threshold, the increase of excitability was more pronounced. It comprised the intervals from 3 msec to 2 seconds. The early facilitation was more pronounced and the second return of excitability came earlier (at 55 ± 7 msec as compared with 80 msec in normals).

Similarly in twelve patients with *parkinsonian rigidity* the recovery was enhanced. In 9 patients re-examined after electrocoagulation of the ventro-lateral nucleus of the thalamus, when rigidity was much reduced or absent, the increase of excitability was less pronounced.

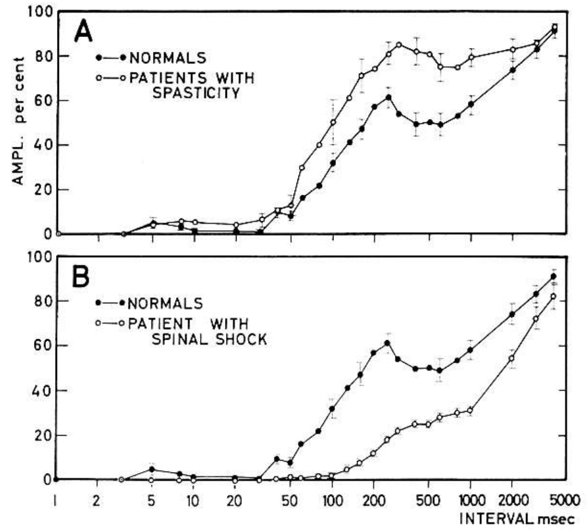

Fig. 1. Recovery of the H-reflex to paired stimuli of equal strength to produce reflexes of maximum amplitudes. Above: Mean from 10 patients with spasticity and 19 normals. Below: Patient L. M. with spinal shock and mean from 19 normals. Abscissa: Time intervals between stimuli of the pair. Ordinate: Amplitudes of test reflexes in per cent of conditioning reflexes. The vertical bars indicate mean errors.

To what extent the changes in recovery in patients with spasticity or rigidity were due to suprasegmental influences directly on the spinal motoneurones or *via* the fusimotor system remains unsettled.

In 10 patients with *acute spinal shock* an attempt was made to differentiate between a depression of motoneurone excitability and of spindle mechanisms.

Early in spinal shock the electrical response associated with a maximal ankle jerk was profoundly depressed or absent whereas the single maximal H-reflex was normal. With paired stimuli recovery deviated from normal. This is illustrated by findings in a 21 year old patient investigated 24 hours after a traffic accident which caused fracture of the cervical spine (Fig. 1, lower). The patient had spinal shock with flaccid paralysis, complete sensory loss, absent tendon reflexes but single maximal H-reflexes of normal amplitude. The recovery of the test reflex showed that the early facilitation was absent, the test reflex returned later than normal, the second facilitation was absent and the late depression was more pronounced than normal.

The normal amplitude of the single maximal H-reflex early in spinal shock indicates normal motoneurone excitability to single stimuli. The absence of the maximal ankle jerk then indicates decreased sensitivity of the muscle receptors, presumably caused by a depression of fusimotor activity. The profound suppression of the recovery of the test reflex is assumed to be due largely to the altered pattern of background activity from the muscle proprioceptors.

Reference

Bianconi, R., Granit, R. & Reis, D. J. (1964). The effects of extensor muscle spindles and tendon organs on homonymous motoneurones in relation to gamma-bias and curarization. *Acta physiol. scand.*, *61*, 331–347.

Spontaneous intrafusal contractions and spontaneous afferent discharges as a tool in the analysis of muscle spindles

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Spontaneous intrafusal contractions in isolated muscle spindles from *Rana temporaria* were reported by Buchthal & Jahn (1957). The spontaneous contractions were abolished by curare and reappeared when curare was replaced by Ringer's solution. In *Xenopus* Smith (1964) observed spontaneous contractions only when the $CaCl_2$ concentration of the Ringer's solution was lowered from 2.0 mM to 0.5 mM or when the KCl-concentration was raised from 2.5 mM to 5.0 mM. This spontaneous activity was not abolished by curare.

A re-examination of our observations in the frog was therefore desirable. I have used the same composition of Ringer's solution as Smith employed and the effect of curare was studied by recording action potentials from the equatorial zone of the spindle as well as by observing the intrafusal contractions microscopically. To minimize movements of the microelectrode, solutions with and without curare were exchanged slowly.

Spontaneous activity of intrafusal muscle fibres was sensitive to injury during isolation of the spindle, a spindle without spontaneous discharges of intrafusal muscle fibres still responding to electrical stimuli of the efferent nerve. In undamaged spindle preparations the spontaneous intrafusal contractions persisted for 24 hours with almost constant frequency.

After infusion of d-Tubocurarine (50 μg/ml) the frequency of spontaneous firing of the intrafusal muscle fibres decreased gradually from 0.4 per second to zero (Fig. 1, from experiments carried out with R. Pritchard, research student, Cornell University, New York). After washing out with Ringer's solution the action potentials of the intrafusal muscle fibres reappeared and gradually attained their original frequency of

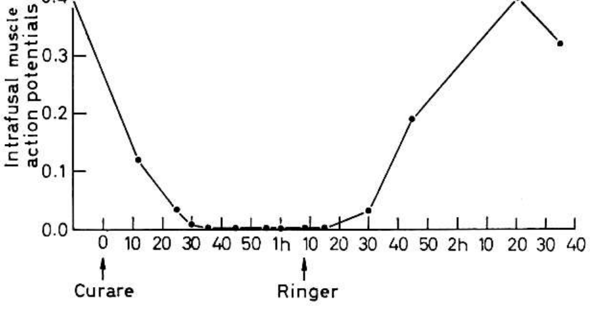

Fig. 1. Reversible abolition of the spontaneous discharges of intrafusal muscle fibres by d-Tubocurarine (50 μg/ml). Abscissa: time in min.

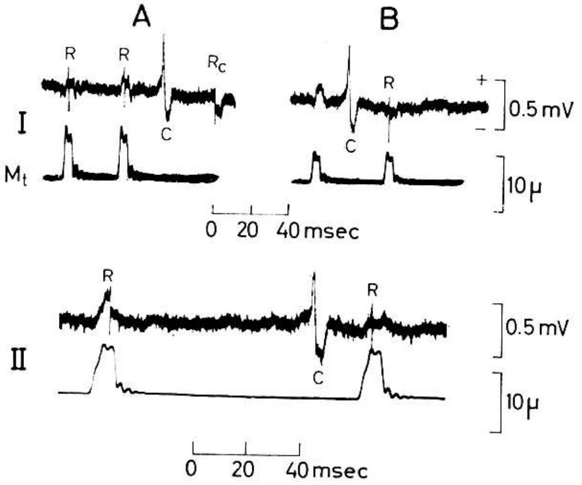

Fig. 2. Afferent discharges of an isolated muscle spindle evoked by stretch. To illustrate the decrease in latency and threshold caused by the spontaneous contraction of a single intrafusal muscle fibre preceding the stretch. I *A*: Evoked responses (R) and spontaneous extracellular action potential from an intrafusal muscle fibre (C). R_c: afferent response to the contraction. M_t: mechanical transients, rise time 2 msec, deformation at threshold. I *B*: response to a subthreshold stretch after a preceding intrafusal contraction. II: reduction of the latency of the second response (R), after a preceding intrafusal contraction; deformation: 1μ above threshold.

discharge. The rate of spontaneous afferent discharges decreased after curare infusion to about half.

The effect of curare on the spontaneous activity of intrafusal muscle fibres is consistent with the assumption that this activity originates at the motor nerve endings in a way analogous to the origin of the miniature end-plate potentials in extrafusal muscle fibres (Fatt & Katz, 1952). The diameter of intrafusal muscle fibres is 1/4–1/5 that of extrafusal fibres. Therefore, a spontaneous release of transmitter at the end-plate, which causes a local depolarization in extrafusal muscle fibres, may cause a conducted response in intrafusal fibres. Measurements of the conduction velocity of spontaneous discharges of intrafusal muscle fibres (Buchthal & Jahn, 1957) indicated that the spontaneously contracting intrafusal fibres of *Rana temporaria* behave like twitch fibres (pp. 426–7 in Buchthal & Sten-Knudsen, 1959).

The occurrence of these spontaneous intrafusal contractions offered the opportunity to study the effect of the contraction of a single intrafusal twitch fibre on subsequent responses to stretch. When stimulated by a mechanical transient the isolated frog spindle responded with a single afferent discharge to a threshold deformation of about 8 μ (Jahn, 1963). A preceding intrafusal contraction facilitated the response to single stretches in that subthreshold stretches produced an afferent response (Fig. 2*B*). When the deformation was above threshold a preceding intrafusal contraction reduced the latency. With a stretch of 1 μ above threshold a preceding intrafusal contraction reduced the latency of the response by 25% (Fig. 2); without a preceding intrafusal contraction the same latency occurred with a 1.75 to 2 times greater deformation (14–16 μ).

In addition to the immediate effect of an intrafusal contraction on latency and threshold of a single evoked response, there was a more protracted effect when the

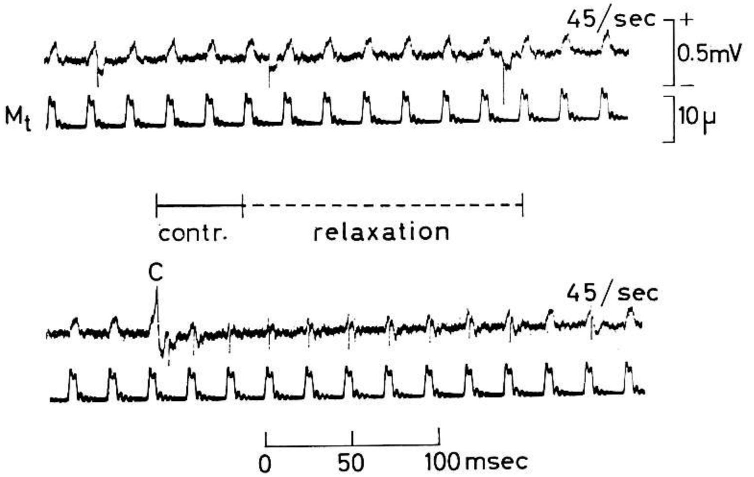

Fig. 3. Facilitating effect of an intrafusal contraction on the afferent responses to the stretches of a train. *Upper beams:* action potentials from the equatorial zone of the muscle spindle. *Lower beams:* mechanical transients, rise time: 2 msec, deformation: threshold for single transient stretch (45 stretches/sec). *Upper set of records:* Only one afferent response to the stretches of the train, the two other afferent discharges occurred independently of the stretches. (Record starts ca. 3 sec after the beginning of the train.) *Lower set of records:* Afferent response to stretches after an intrafusal contraction, the duration of the facilitation corresponds to the duration of the mechanical changes accompanying an intrafusal contraction (cf. diagram above the records). C: spontaneous action potential from an intrafusal muscle fibre.

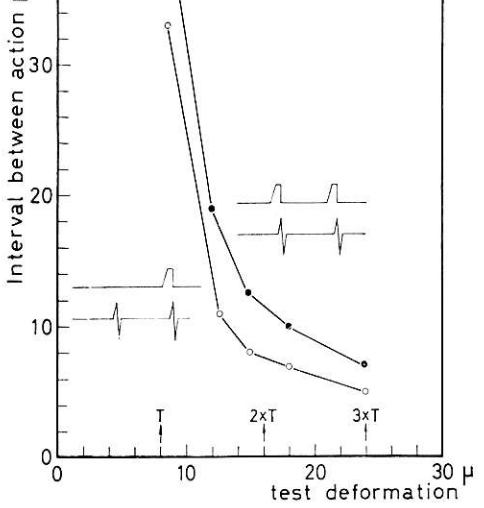

Fig. 4. The depression of excitability of the muscle spindle by preceding mechanical changes. Rise time of the stretches: 2 msec. *Open circles:* minimum intervals between a spontaneous afferent discharge and a response to a test stretch as a function of the test deformation. (Left inset: the spontaneous afferent discharge triggered the test stretch.) *Full circles:* minimum interval between responses to a conditioning stretch and a test stretch as a function of the test deformation. (Right inset: stimulation by paired stretches of equal size.) T: threshold for a single transient stretch.

spindle was stimulated by trains of stretches (Fig. 3). Trains of deformations which were threshold for single stretches evoked only a few responses if applied at a rate of more than 11 stretches per second. After an intrafusal contraction every stretch evoked a response even if applied at a 6 times greater frequency. The duration of the facilitation after an action potential of an intrafusal muscle fibre corresponded well to the duration of the mechanical changes in a contracting intrafusal fibre (Fig. 3). The degree of the facilitation caused by the intrafusal contraction is illustrated by the fact that a deformation of about twice threshold was required to obtain responses to every stretch. That the duration of the facilitation coincided with the mechanical response of an intrafusal contraction makes it likely that this facilitation is due to an additional deformation of the spindle corresponding in degree to the deformation which was threshold for a single stretch.

The spontaneous afferent firing of muscle spindles allowed a comparison of the excitability after a spontaneous afferent discharge, not associated with changes in the mechanical state of the spindle, with that after a stretch. In 8 spindles the average minimum interval between a spontaneous afferent discharge and a regular response to stretch of threshold deformation was more than 30 msec. With a deformation of twice threshold the minimum interval was 8 msec and with three times threshold 5 msec (Fig. 4). When the conditioning afferent action potential was evoked by stretch the minimum intervals were longer. With twice threshold deformation the minimum intervals were 50% longer (12 msec) and with three times threshold deformation 40% longer (7 msec). These experiments show the prolonged depression of excitability when a test stretch was preceded by changes in the mechanical state of the muscle spindle.

References

Buchthal, F. & Jahn, U. (1957). Spontaneous activity in isolated muscle spindles. *Acta physiol. scand.*, *42*, suppl. 145, 25–27.

Buchthal, F. & Sten-Knudsen, O. (1959). Impulse propagation in striated muscle fibres and the role of the internal currents in activation. *Ann. N.Y. Acad. Sci.*, *81*, 422–445.

Fatt, P. & Katz, B. (1952). Spontaneous subthreshold activity at motor nerve endings. *J. Physiol.*, *117*, 109–128.

Jahn, S. A. A. (1963). Physiological properties of isolated neuromuscular spindles. *Acta physiol. scand.*, *59*, suppl. 213, 67–68.

Smith, R. S. (1964). Contraction in intrafusal muscle fibres of *Xenopus laevis* following stimulation of their motor nerves. *Acta physiol. scand.*, *62*, 195–209.

Separation of the effects on the muscle servo of the alpha-, gamma- and Renshaw-control pathways

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Sinusoidally-fluctuating tensions are applied to the soleus muscle of the decerebrate cat by a method described elsewhere (Roberts, 1963). The firing-frequency of single motor units in the muscle is plotted against applied tension, to produce a roughly triangular figure (Roberts, 1958) with a steep rise of firing-frequency at threshold, a plateau at a 'preferred firing-frequency', and a return slope (Fig. 1) which sometimes crosses over the rising portion.

A corresponding plot for a single spindle afferent recorded in the dorsal root shows a rounded portion in place of the plateau (Fig. 2). This curve resembles that expected on the basis of a mixture of static and dynamic components each of which has its own threshold (Fig. 3).

The similarities between the curves for input and output signals suggest that the reflex centre may have a fairly simple transfer-function, producing a change of scale of firing-frequency. Feedback over the Renshaw circuit would provide such frequency-reduction (Taylor, 1960) but, owing to the nature of the characteristic discharge of Renshaw cells, it is to be expected that output would be proportional to input only up to a particular value of output frequency. The consequence of such a 'saturation effect' is indicated by the broken line in Fig. 3, and it is clear that this may be the reason for the plateau in the graph for motor unit response.

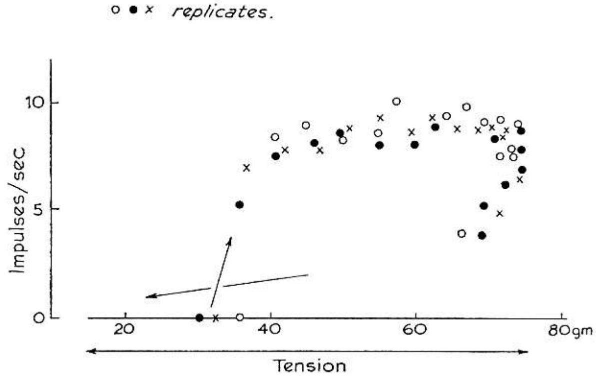

Fig. 1. Plot of firing-frequency against applied tension for a single motor unit in the soleus muscle of a decerebrate cat. Each point indicates a single impulse: the abscissa is the applied tension at the moment of firing, the ordinate is the reciprocal of the interval since the previous impulse. Three cycles, taken from a run, show the reproducibility of the response. Note that a curve through these points must cover the whole range of applied tensions (arrow) although the unit did not fire at the lower tensions.

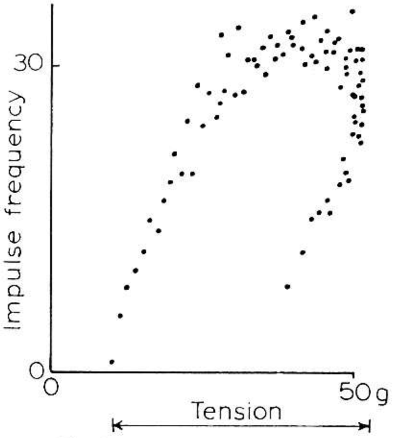

Fig. 2.

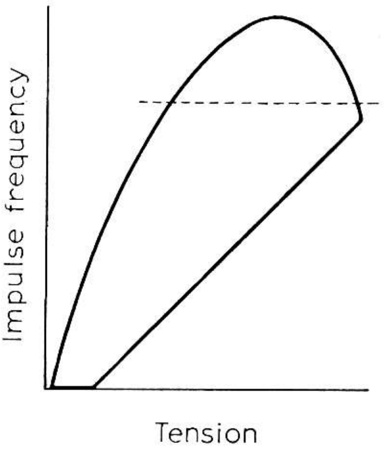

Fig. 3.

Fig. 2. Plot obtained as in Fig. 1, but for a single dorsal-root fibre carrying impulses from a spindle in soleus.

Fig. 3. Diagram to indicate the expected form of the plot in Fig. 2, on the basis of a mixture of static and dynamic components, each with its own threshold. The static component gives the linear return slope because the dynamic component makes no contribution when the rate-of-change of tension is negative. During the period of rising tensions, the 'velocity-dependent' dynamic component introduces phase-advance. The broken line indicates the frequency-limitation introduced during the passage of the signal through the reflex centre in consequence of the characteristics of Renshaw-cell discharges.

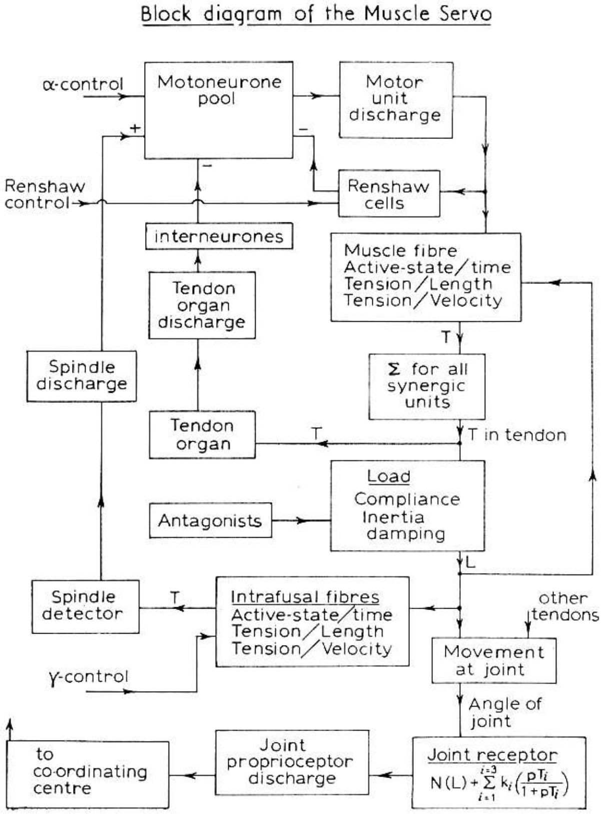

Fig. 4. Block diagram to indicate the interactions between some of the factors that must be taken into consideration in predicting the mechanical response to nervous motor commands.

In different conditions of converging reflex drive (cerebellar stimulation, head-tilting after neck denervation, crossed extensor reflex), the three sides of the triangle are shifted, sometimes independently.

Of the three pathways over which the muscle servo may be controlled (Fig. 4), only the gamma-pathway can produce effects that are different at different tensions. It follows that, if the slopes of parts of the triangle are altered, there must have been a change in gamma drive.

At a particular level of Renshaw-cell excitability, the saturation should occur at the same motoneurone firing-frequency, whatever the afferent drive to the motoneurone. A change in the position of the plateau therefore indicates a change in the background excitation of the Renshaw cells, such as has been seen by Haase & Van Der Meulen (1961).

Convergent direct alpha drive would be expected to alter the threshold at which a particular motoneurone responds to a given amount of reflex afferent drive from the spindles.

References

Haase, J. & Van Der Meulen, J. P. (1961). Effects of supraspinal stimulation on Renshaw cells belonging to extensor motoneurones. *J. Neurophysiol.*, *24*, 510–20.

Roberts, T. D. M. (1958). The assessment of changes in the sensitivity of a stretch reflex. *J. Physiol.*, *144*, 2–4 *P.*

Roberts, T. D. M. (1963). Rhythmic excitation of a stretch reflex, revealing (a) hysteresis and (b) a difference between the responses to pulling and to stretching. *Quart. J. exp. Physiol.*, *48*, 328–45.

Taylor, W. K. (1960). Computers and the nervous system. *Symp. Soc. exp. Biol.*, *14*, 152–168. and personal communication.

Phasic, tonic and static components of the reflex tension obtained by stretch at different rates

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This study aims at establishing the significance of rate of stretch as an input component of the stretch reflex by analyzing the output tension appearing in the responding muscles.

1. During stretch, tension develops not only owing to the reflex contraction but also because of passive elasticity and viscosity of the muscle. Passive tension shows a different course at different rates of stretch. To eliminate passive tension from our records, a compensation method was used (Takano & Henatsch, 1964): A pair of homonymous muscles from both sides was stretched simultaneously, one side being denervated and the other having intact innervation. In a bridge circuit, the total tension developing in the intact muscle was reduced by the passive tension of its denervated fellow muscle. Since steps were taken to obtain the same passive tension to stretch on both sides, it follows that the remainder of tension is the active reflex tension of the intact muscle. Hind limb muscles, i.e. gastrocnemius, soleus, and tibialis anterior, of precollicularly decerebrate cats were used. The muscle was stretched 14 mm at rates of 0–400 mm/sec with good linearity. The final extension was maintained for some time.

2. Direct records of active tension-extension-curves of gastrocnemius muscle at stretch rates of 1–100 mm/sec were analysed. The maximum tension and the gain constant (Granit, 1958) showed a relatively good linearly-rising relation to the logarithm of the stretch. The relation between response time of tension and stretch rate appeared to be linear if plotted in double-logarithmic scales. From these results it may be concluded that the rate of stretch is as important as its amplitude for the reflex response.

3. For a further analysis of the output tension in the stretch reflex, records were taken using the X-axis as a time base. At the slow stretch rate of 0.5 mm/sec there was no tension fall after the end of stretch. This tension will be named the "*static component*" of the stretch reflex. The static component can only be seen in isolation when the muscle is very slowly stretched. In the precollicularly decerebrate cat the critical rate seems to be 0.5 mm/sec for gastrocnemius and less than 0.2 mm/sec for soleus.

When the muscle is stretched at a faster rate, the tension develops not only to a higher peak but also falls more and more after the end of stretch. At rates exceeding 70 mm/sec two components of tension can be observed: first, tension developing during the rising phase of stretch; second, a slower rise of tension after the end of extension.

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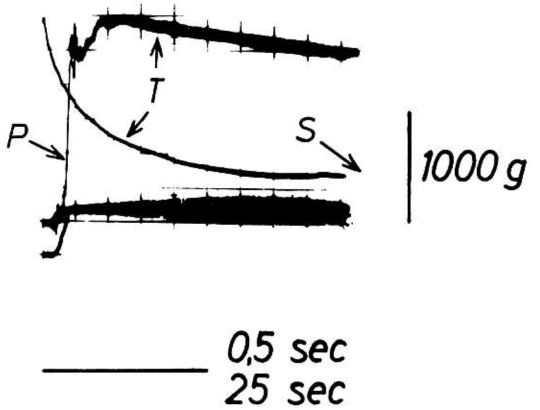

Fig. 1. Phasic (P), tonic (T), and static (S) components of the reflex tension in gastrocnemius muscle of precollicular decerebrate cat. Upper two records are tension at fast and slow sweep speeds. Reflex tension was directly recorded with a compensation method. The lowest record shows the length change of muscle. Stretch rate, 280 mm/sec.

The second one always lasts more than 1 sec, sometime 20–30 sec or even 1 min. This slow component will be named the '*tonic component*' of the stretch reflex. The first component, appearing with some latency after the beginning of extension, is named the '*phasic component*'. The higher the stretch rate, the clearer the distinction between these two components. Both phasic and tonic components are highly dependent upon the rate of stretch. The phasic component is created by the high rate of extension at the initial stage of stretch. It is due to an almost synchronous firing of the motor units, the actual tension development depending mainly upon the changing muscle extension. Progressive recruitment of new motor units may be rare during this phase. Some kind of circulating activity may be established at spinal and/or supraspinal levels, which is triggered by the initial phasic burst of spindle discharges. This probably causes the tonic component. Fig. 1 illustrates the phasic (P), and tonic (T), and static (S) components of the reflex tension appearing in gastrocnemius at the stretch rate of 280 mm/sec. The two tension records are taken at fast and slow sweep speeds, respectively.

4. The three components described above were compared in the slow soleus and the fast tibialis anterior muscles. The phasic component is smaller in soleus than in gastrocnemius, but the tonic component is highly dominant in soleus, both in amplitude and duration. The phasic component becomes smaller, the tonic one greater, at high rates of stretch of soleus. The static component must be looked for long after the end of stretch (after 1 min). Even at a stretch rate of 0.23 mm/sec the tonic component is still to be seen. The sensitivity to rate of stretch is greater in soleus than in gastrocnemius. On the other hand, the tonic component in the reflex tension of a phasic muscle like tibialis anterior is very small and lasts for a short time only. Here, a distinct relation between tension and rate was noted at rates of 10–100 mm/sec. At lower rates this muscle showed no tension response at all of any of the three components of the stretch reflex. The development of tension in tibialis anterior was very poor by comparison with soleus, although the mean weight of the former muscle is 1.6 times that of the latter. The reflex response of soleus per gram is about 10 times greater than that of tibialis anterior.

Details of this study, including a discussion of supraspinal influences upon the three components of the stretch reflex, will appear elsewhere (Takano, Henatsch & Heinich and Takano, unpublished).

Reference

Granit, R. (1958). Neuromuscular interaction in postural tone of the cat's isometric soleus muscle. *J. Physiol.*, *143*, 387–402.

Takano, K. & Henatsch, H.-D. (1964). Direkte Aufnahme von Längen-Reflexspannungs-Diagrammen einzelner Muskeln in situ. *Pflüg. Arch. ges. Physiol.*, *281*, 105.

Pyramidal influences on gamma- and alpha-motoneurones in the cat

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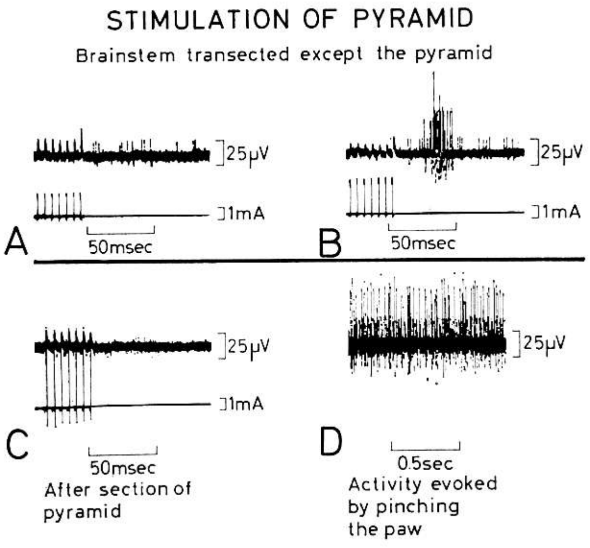
Repetitive stimuli applied to the bulbar pyramids of decerebrated cats evoked complex movements (Landau, 1952); short trains of stimuli produced twitch-like movements of the limbs, predominantly in the flexors (Brookhart, 1952). However, even threshold stimuli applied to the bulbar pyramids may spread to other descending tracts. Therefore we dissected the pyramid away from the brainstem before stimulation (Towe & Jabbur, 1959) or prevented impulses descending outside the pyramidal tract from reaching the spinal cord (Lloyd, 1941). We have studied evoked discharges of alpha- and gamma-motoneurones with the gamma loop interrupted. Stimulation of pyramidal fibres elicited a burst of action potentials in the anterior tibial muscle and the flexor activity was maintained during repetitive stimulation of one second's duration. Spontaneous or reflex activity in the gastrocnemius muscle was inhibited. Action potentials were recorded in the nerve close to the tenuissimus muscle and the two types of motoneurones were distinguished by the amplitude of their potentials (Fig. 1 *D*), the reflex behaviour, the conduction velocity and the sensitivity to procaine. Co-activation of gamma- and alpha-motoneurones occurred in response to repetitive stimulation of pyramidal fibres. The minimal train duration for gamma motoneurones was three stimuli of 0.1 msec duration at 200/sec. The threshold current decreased with longer trains and remained constant if more than five pulses of 0.1 msec at



200/sec were applied. Alpha motoneurones discharged at 1.2 to 2 times higher current intensities than the threshold of gamma motoneurones (Fig. 1 *A*, *B*). The latency of evoked gamma discharges was 29.5 ±4.5 msec ($n = 11$), of evoked alpha discharges 40.0 ±7 msec ($n = 11$). These latencies are about 25% longer than when the brainstem was intact. A 25% to 150% decrease in the latency of gamma discharges was observed when maximal stimuli were used instead of threshold stimuli. The duration of the evoked gamma discharges was longer (96.5 ±42 msec, $n = 10$) than the duration of the alpha discharges (34.0 ±17.2 msec, $n = 10$). Transection of the pyramid caudal to the point of stimulation abolished the evoked response (Fig. 1 *C*).

These results are in agreement with the results of chronic experiments on cats supported in a sling: flexion reflexes and tonic flexor activity were diminished for several weeks after section of the medullary pyramids (Wiesendanger & Laursen, unpublished). With respect to the alpha motoneurones our results agree with those of Kato, Takamura & Fujimori (1964), except that we regularly observed co-activation of gamma and alpha motoneurones in a flexor nerve whereas Kato *et al.*'s figure indicates a reciprocal effect of pyramidal stimulation, i.e. inhibition of gamma discharges and initiation of alpha discharges. Kato *et al.* recorded from ventral root filaments and gamma and alpha spikes in their records may be from motoneuronses of different muscles.

References

Brookhart, J. M. (1952). A study of cortico-spinal activation of motor neurons. *Res. Publ. Ass. Nerv. Men. Dis., 30*, 157–173.

Kato, M., Takamura, H. & Fujimori, B. (1964). Studies on effects of pyramid stimulation upon flexor and extensor motoneurones and gamma motoneurones. *Jap. J. Physiol., 14*, 34–44.

Landau, W. M. (1952). Patterns of movement elicited by medullary pyramidal stimulation in the cat. *Electroenceph. clin. Neurophysiol., 4*, 527–546.

Lloyd, D. P. (1941). The spinal mechanism of the pyramidal system in cats. *J. Neurophysiol., 4*, 525–546.

Towe, A. L. & Jabbur, S. J. (1959). Antidromic cortical response to stimulation of isolated pyramidal tract. *Science, 129*, 1676–1677.