The Effects of Stretch Receptors on Motoneurons

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The stretch receptors are the muscle’s measuring instruments, and their effects on motoneurons have been studied by myography, electromyography, monosynaptic testing and, ultimately, by intracellular recording. A complete review of their anatomy, physiological properties including efferent control, and their central effects would require a whole book. Here we shall leave out, or regard as known, most of the historical development of our knowledge prior to 1955, and concentrate instead on prospects and notions for further advance as seen against the background of more recent research. Furthermore, we shall deal almost exclusively with the natural stimuli, contraction and stretch.

At the outset, we would like to emphasize that, as far as motoneurons are concerned, the essential problems no longer circle around reflexes, though reflexes are still the helpful tools they always have been in this field. The essential problems concern the biasing or setting of the various mechanisms by the aid of which motoneurons are made to operate, reflexively or otherwise. There is biasing of them by the neuromuscular intramuscular machinery of the gamma loop, by internuncial systems, and by neurohormones operating on the alpha and gamma motoneurons from higher stations, as recent work has shown. We shall come to this question at the end of this paper.

THE TENDON ORGANS

When the muscle spindles pause in the rising contraction of the muscle in which they are located, the tendon organs fire (Fig. 1). If this contraction is elicited from the muscle nerve, there will, of course, be a component of afterhyperpolarization from a number of motoneurons as well as a recurrent inhibition in many of them, particularly strong in the tonic ones. In order to avoid backfiring, it is therefore best to stimulate the muscle from the peripheral stump of a cut ventral root. In animals under Nembutal, there will not then occur any firing of the impaled motoneuron as long as the membrane potential is high, and so it is possible to demonstrate that both in extensors and flexors there is an autogenetic inhibitory postsynaptic potential during the rising phase of the contraction. This effect is genuine because it passes the chloride test.

There is a striking difference between the effect of size of contraction in extensors and flexors. In the latter, as tensile stress rose, Green and Kellerth
found the inhibitory potential soon annulled by a discharge filling out the silent period from both ends, and they could prove that this effect came from excitatory spindle afferents. In the extensors, on the contrary, the hyperpolarizing response to contraction increases when at constant extension the contraction itself increases, and this effect is also seen in many synergic motoneurons deprived of their own muscle innervation. Both these effects are illustrated in Fig. 2 by records taken from a synergic motoneuron (cf. also Fig. 12).

![Graphical representation of the effects on muscle tension and potential](image)

**Fig. 1.** De-efferented chloralose-pentobarbitone cat. Gastrocnemius spindle primary (large spike) and tendon organ (small spike) afferents studied at an extension of 4.5 mm. Brief submaximal tetanus.

**Fig. 2.** De-efferented pentobarbitone cat. Synergic popliteal motoneuron influenced by contraction of the gastrocnemius muscle. Upper trace, intracellular record, lower trace, muscle tension. Muscle at 5 mm extension. Stimulation of peripheral stump of cut ventral root L7 with single shocks (left) and 3 shocks at 330/sec (right). The silent motoneuron (above) responded by hyperpolarization that deepened with the larger contraction. The cell was then fired by injected current (below) to show that the larger contractions produced definite silent periods.
Thus there is autogenetic inhibition from the tendon organs contributing to the silent period, as seen in the lower record of Fig. 2, in which the motoneuron was fired by transmembrane stimulation. This is a good way of detecting the contribution from the tendon organs, because the cell is then depolarized artificially. If the same amount of depolarization were to be produced by taking care to activate muscle spindles, then the spindle pause would cause a disfacilitation which, of course, on mere inspection also is a hyperpolarization. It seems obvious enough that if the spindles had been allowed to produce the depolarization themselves, then cessation of their activity would remove it. Their contribution to the silent period has not been seriously in doubt since 1933, when Matthews demonstrated the pause of the spindle in contraction. What needed to be shown, however, was that Denny-Brown\textsuperscript{30} and Granit in 1950\textsuperscript{44} were right in assuming genuine inhibition during the silent period, autogenetic as well as synergic. Intracellular recording was needed to exclude presynaptic inhibition in this context. Transmembrane firing in this as in several other cases proved a good tool.

What role, apart from the informative one of marking and measuring contractile tension, can we assign the tendon organs in this connection? For the motoneurons of the large extensor muscles they serve as highly needed brakes on contraction. But they must also play a role in stepping\textsuperscript{46} because both Denny-Brown\textsuperscript{30} and Granit\textsuperscript{45} by somewhat different methods found flexor nuclei reciprocally facilitated during the rising contraction of their antagonist extensor, helping the flexor phase of the step to come off. It is more difficult to assess the role of the tendon organs in flexor contractions. As stated, their autogenetic inhibitory effect was soon overruled as tension rose.\textsuperscript{62} Granit\textsuperscript{45} never found a reciprocal excitation from flexor contractions on extensor motoneurons, merely inhibition.

Passing from contraction to stretch, the first point to remember is that pull leading to moderate passive tension is an inefficient way of stimulating tendon organs, even though a number of them measure the amount of tension in absolute terms. Most of them fire well only to contractile tension. For the old work we refer to Granit's summary of 1955,\textsuperscript{46} which emphasized the need for contractile tension. Recently, Jansen and Rudjord\textsuperscript{74} have made a systematic study of tendon organ impulses, showing very clearly that their most efficient adequate stimulus is contractile tension. Nevertheless, there is no reason to assume that the tendon organs capable of responding to passive tension fail to exert the effects obtained by using contractile tension and intracellular recording as indicators. By the monosynaptic index, stretch of extensors is known to produce autogenetic inhibition, flexors being less potent in this respect. Review of the literature will be found in Bianconi \textit{et al.},\textsuperscript{14,15} and we shall return to this point below.

At the moment the most interesting question with regard to the tendon organs seems to concern the circumstances under which they are biased into action by
internuncials, their synaptic apparatus being disynaptic or polysynaptic.\textsuperscript{35,80} There is hardly any doubt but that the tendon organs contribute heavily to the clasp-knife effect that occurs when extensors are forced to strong contractions by bending the leg of the decerebrate cat. This effect cannot be elicited in the cerebellar alpha rigidities, suggesting low excitatory or high inhibitory bias on the interneurons of the tendon organs. There is good evidence that in spinal cats the internuncial cells operating motoneurons are biased in favor of extensor inhibition,\textsuperscript{36,76} but when we speak of the problems of biasing that remain to be studied we do not necessarily mean pathological states but want to include natural movements exerted under inhibitory constraint from tendon organs. Here is an untitled field. We would not be surprised if the interneurons of the tendon organs were found to maintain a central station semi-exclusive for themselves. At least their descending control path differs from that of (what Lundberg calls) the “flexor reflex afferents.”\textsuperscript{36} In the decerebrate-decerebellate cat, release of the inhibitions from the tendon organs requires a section at the medullary level,\textsuperscript{67} which thus is below the pontine region that releases the flexor reflex afferents to which the spindle secondaries are likely to belong. This suggests that the tendon organs have an independent internuncial biasing system, Hufschmidt’s\textsuperscript{68} delta neurons, making it possible for contracting muscles to operate with controllable degrees of restraint.

In this brief review of what is known about the effects of tendon organs on motoneurons, we have left out many valuable papers based on fiber size and monosynaptic testing. The reader will find them reviewed in papers by Holmqvist and Lundberg\textsuperscript{67} and Bianconi et al.\textsuperscript{14,15} We have done this in order to have more time for the complex theme of spindle control.

**Muscle Spindles**

The recent results in the anatomy and physiology of the muscle spindles were summarized and discussed at the Nobel Symposium 1 in Stockholm dealing with muscular afferents and motor control\textsuperscript{47} and, while something must be said here in the way of an introduction which overlaps with that book, we shall soon go on to readjust our focus for points of view and general ideas that received less attention at the Nobel symposium.

Working mainly with rabbit muscles, Barker\textsuperscript{5} originally placed the primary and the secondary spindle endings on the same intrafusal nuclear bag fiber, making the former encircle the elastic bag and the latter innervate the myotube region nearest to it. His observations made Granit\textsuperscript{46} use the term myotube organs for the latter. This arrangement has been reconfirmed by Adal and Barker,\textsuperscript{1} but in the meantime conclusive evidence showed cat muscle spindles to be different and to contain two separate intrafusal fibers, the nuclear bag fiber known for
over a century and a shorter and thinner type, the nuclear chain fiber (Fig. 3),
of which there are, say, four in each spindle — one to eight, Barker and Gidumal.8
This knowledge we owe to Cooper and Daniel (human spindles),23,24 Boyd (sum-
mary),17 and Barker and his coworkers.6 The spindle secondaries arise from the

g nuclear chain fibers and only a minority is found in the myotube region of the
nuclear bag fibers. The spindle primaries, to be sure, are still found in their old
site, encircling the nuclear bag, but they also throw a branch to the nuclear chain
fibers and so can make use of the properties of both kinds of intrafusal muscle.
These disclosures focused attention on the mechanical properties of the two types
of intrafusal fiber and on the need for a reassessment of the problem of the peri-
pheral distribution of the terminals of the fusimotor gamma fibers. Close coopera-
tion among the physiologists and anatomists interested in these questions has led
to a considerable advance in understanding them.

We can profitably discuss the interconnections between all the aspects of spindle
innervation as a number of dualities of form and function that somehow must be
correlated. The first of these dualities is the existence of two types of spindle
organs, primaries and secondaries, to which the duality of two types of intrafusal
fibers was correlated (with a few exceptions). Unfortunately, the results did not
come out clean-cut because, as we saw, the rabbit has both organs on the same
intrafusal fiber, and when there are both types of intrafusals present, the primary
organs throw branches to both of them. Even for secondaries, restriction to
nuclear chain fibers is not absolute.

The functional duality on the afferent side is more satisfying: the spindle pri-
maries are sensitive to velocity of pull, as known from 1933 onward81 and there
is good agreement between histologists and physiologists that this depends on the mechanical arrangement of an elastic nuclear bag inserted between more viscous muscular poles. Sybil Cooper showed that this high sensitivity to rate of stretch is lacking in the secondaries which measure static length alone; her work has been confirmed many times. Bianconi and van der Meulen made an extensive study of the vibrational sensitivity of primaries and secondaries in the cat, using the technique of Granit and Henatsch, and on an average found it very high for primaries and low for secondaries. Bessou and Laporte had made similar observations on primaries and secondaries in the tenuissimus preparation. Thus, they laid the basis for an important technique of activating the primaries selectively in intact animals and in man (see below). This pair of functional dualities agrees with the location of primaries and secondaries to different kinds of intrafusal fibers. Primaries run in the large Group I fibers; secondaries, in the Group II fibers.

Boyd's idea, of two types of gamma fusimotor fibers, his gammas 1 and 2, separable by their diameter, was not accepted at the Nobel symposium because of Barker's well-founded criticism. It is not verifiable functionally, but the static gamma fibers spread over a wider caliber spectrum than the dynamic ones. Another efferent duality, supported by Cooper, Boyd, and Barker, survived this controversy, namely, the existence of two kinds of motor endings, gamma plates and gamma trails, the latter a diffusely spreading type. Again, unfortunately, on the question of their distribution the histologists were in two opposing camps, and we cannot at the moment say that either of these motor endings is restricted to just the one type of intrafusal fiber, nuclear bag or nuclear chain.

Functional dualities, as we have seen, are more satisfying on the whole. Peter Matthews found an interesting duality on the efferent side when he described dynamic and static fusimotor gamma efferents. The dynamic velocity-sensitive component of the response to stretch was enhanced when the dynamic efferents were simultaneously stimulated while the static portion of the discharge was less influenced; the static efferents merely enhanced the static part of the discharge, i.e., the one to maintain stretch. This duality has stood up well to various tests by Matthews and his coworkers (see, e.g., the summary by Jansen; cf. also Appelberg et al.) and shows differential tasks of fusimotor fibers. Thus, both Crowe and Matthews as well as Bessou et al. found that once a gamma efferent proved static or dynamic for one particular spindle, its branches innervating other spindles likewise were static or dynamic, respectively. Accordingly, there must be a corresponding duality at the motor end. Jansen and Matthews, confirmed by Bessou and Laporte, contributed to the analysis of this duality by showing that the dynamic behavior of the primaries could be altered over a wide range while this property in the secondaries varied but little. The secondaries were controlled by static fusimotor gamma fibers.
The phenomenon of "driving" spindles by efferent tetani has thrown some light on that question. The static fusimotor fibers\(^{18}\) are the ones that can drive spindles, even in the rabbit,\(^{41}\) while the dynamic ones build up a spindle discharge slowly. At least in the cat the majority of the secondaries derive from nuclear chain intrafusals, and thus driving seems connected with the motor properties of such intrafusals. The fact that long ago Kuffler et al.\(^{27}\) found that some spindle primaries in the cat also could be driven is then due to their endings on the nuclear chain intrafusals. The immediate inference drawn from this state of affairs is that the nuclear chain intrafusal muscles would be more inclined to twitch than the nuclear bag muscles. However, static gamma efferents in the rabbit not only exist but behave as do the static fibers in the cat,\(^{41}\) despite lack of nuclear chain fibers. Now, since in the rabbit the single nuclear bag fibers seem to do all the tricks, twitching as well as contracting slowly, driving or no driving may well depend on the properties transferred to the fiber by their dual motor end organ or the location of these organs near to or far from the sensory zone\(^{12,41}\) rather than being inherent in the differences between two types of intrafusal muscles.

In all this argumentation the properties of the two types of intrafusal fibers have until quite recently been the missing link. In our laboratory, Smith\(^{91}\) studied them in the rat by cinematography under the microscope and found the large fibers slowly build up their contraction to repetitive direct stimulation whereas the small ones were quick fibers responding quite well also to single shocks. This at least made intelligible that the small muscle fibers holding most of the secondaries could be "driven" while the large intrafusals were the ones requiring tetani in order to contract properly. Dieter-Spiff\(^{31,32}\) reported similar preliminary observations on the large nuclear bag fibers in the lumbrical muscles of the dog, and Takano,\(^{94}\) by an indirect method of calculating from the discharge rate of spindles, arrived at the conclusion that there must be slow and fast intrafusal fibers.\(^*\)

Finally, this problem has been illuminated by another duality of motor innervation described by Bessou and Laporte.\(^{11}\) Using the tenuissimus muscle of the cat, they found that stimulation of static gamma fibers induced conducted diphasic spikes of the kind previously noted by Eyzaguirre,\(^{43}\) while stimulation of dynamic gamma fibers produced local nonconducted negative potentials. At the moment this is a duality of action in good agreement with the notion of Peter Matthews that the dynamic fibers (in the cat) build up slow contractions in nuclear bag fibers at gamma trail endings while static gamma fibers produce quick contractions at gamma plate endings in nuclear chain fibers. As pointed out by Bessou and Laporte,\(^{12}\) there are some observations that do not fit well into this interpretation, above all, in our opinion, the finding by Barker, mentioned above, to the effect that both types of motor ending run to both types of intrafusal fiber,

* See Addendum.
as well as their own observation that the rabbit with only nuclear bag intrafusals has static and dynamic fibers that do not perceptibly differ in their action from their partners in the cat. In its final considerations, the Toulouse group\textsuperscript{12,41} lay the emphasis on the nature and location of the motor ending that would make it possible for the same intrafusal fiber to act in two ways. Its diagram, presented at the Nobel symposium,\textsuperscript{12} is shown in Fig. 4.

![Diagram of dynamic and static fusimotor fibers](image)

**Fig. 4.** Tentative description of the mechanisms of action of dynamic and static fusimotor fibers (see text).\textsuperscript{12}

\(P\): primary ending, \(S\): secondary ending.

A duality, unearthed by Granit and Holmgren,\textsuperscript{51} should be mentioned in this connection because it has now obviously risen to a new level of actuality. They found in stimulating with electrodes in the brain stem that some spindle primaries could be driven at modest rates and some not at all, the latter merely responding by a slowly rising and falling rate of discharge. Often, both effects were superimposed on the same spindle. The effect of driving could be stopped by a lateral deep section in the lower part of the spinal cord, whereas the slowly rising and falling effect withstood large criss-cross sections at several levels in the cord. Someone ought now to return to this problem; the armory seems replenished for a fresh attack.

There remains to say a few words about fusimotor alpha fibers, but we believe these had better be fitted in when we come to consider spindle function.

**Spindle Reflexes on Motoneurons**

It is very easy to be concise on spindle primaries. They uniformly excite their own and some synergic motoneurons. This being the case, why is it so difficult to elicit stretch reflexes in man? We know them chiefly as pathological phenomena. The answer from our recent intracellular experiments in the cat\textsuperscript{55,56} is definite on this point: Some bias is needed for stretch reflexes. In extensors, the spindle primaries in addition to some opposition from the tendon organs also
encounter antagonistic inhibition from the spindle secondaries. Pull on an extensor leads mainly to mixed hyperpolarizing and depolarizing synaptic activation noise (Fig. 5). It has long been known\(^7\) that if during pull on the muscle its motoneurons are tested by a half-maximal monosynaptic potential, there is inhibition in the sense that the monosynaptic response is greatly diminished and so, often, is also the monosynaptic postsynaptic potential recorded intracellularly (Fig. 6). In the latter case, however, one has the opportunity of testing the true average change in excitability by depolarizing the cell by current from the electrode tip, and then there is no inhibition. Firing or acceleration of established firing is then the result of stretch; in other words, there is a stretch reflex. One could say that the cell rejects mixed noise, unless it is itself sufficiently depolarized. Not until then does it become capable of interpreting the net effect of the activating noise. The same is actually true also for inhibition.\(^{55}\) Inhibition, otherwise ineffectual, emerges as a decrease of firing rate as soon as a cell is fired by injected current. At a recent symposium in Stockholm on inhibition (C. von Euler, editor, New York, Pergamon Press, 1967), Granit gave a full account of the value of

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**Fig. 5.** Anemically narcotized cat. Pentobarbitone 28 mg/kg. Popliteal motoneuron of about 75 mV spike height; membrane potential 65 mV. Activation noise recorded on sweep (AC) and standing spot (DC). The upper and middle records are controls with triceps (autogenetic) and semitendinosus (antagonistic). The lowermost record begins with the end of about 4 sec pull on triceps maintained throughout. At the arrow, semitendinosus pull begins. Weight of 500 g used. Note: In this interference record, hyperpolarizing activation noise of semitendinosus is more prominent than in the control. Note also the "spiky noise" to triceps pull as well as wavelets.\(^\text{55}\)
using a cell fired from the inside as indicator of excitation and inhibition and further important examples were given in a paper by Kellerth. We need not return to this problem here.

Of greater interest at the moment is the question of what is biasing the stretch reflexes and why this bias is so difficult to produce volitionally. There can hardly be any doubt today that in the normal life of man one major biasing instrument is the gamma-spindle loop, and we can see it brought into action by stimulating at gamma strength from the peripheral stump of a cut ventral root in the animal experiment, as in Fig. 7. Recording is intracellular.\textsuperscript{54a} At greater initial tensions (on the right), the tendon organs come in and suppress the firing rate.

Why is it so difficult to put on the gamma-spindle loop volitionally in imitation of the experiment just presented? We suggest this answer: In motor control most circuits are highly automatized, having developed to serve some sensible biological purpose, and no such purpose is served in biasing the loop for the benefit of the clinician pulling on a muscle. For the living organism, it is a pointless act. But the effect as such can be achieved, as it were, artificially also in man by vibrating the tendon of a muscle (occasionally the muscle belly) at a high rate, thereby stimulating the large spindle afferents, the small ones deriving from the secondaries not being able to follow such rates of stimulation.\textsuperscript{16} Such experiments have recently
been carried out by several workers independently on man \(^{29,38,64,65,88,96}\) and by Matthews \(^{84}\) on the soleus of the cat. Such an experiment on man is shown in Fig. 8.

These results could be discussed from many points of view, but space is limited, and we shall take up merely the further evidence they provide for a supporting polysynaptic pathway in the stretch reflex, which many workers seem to imagine is purely monosynaptic. Opposition to this point of view was raised by Granit \textit{et al.}\(^{59}\) when they had found that posttetanic potentiation of the tonic motoneurons\(^{50}\) also gave posttetanic potentiation for a number of reflexes that were polysynaptic in nature. Since posttetanic potentiation is restricted to the terminals actually tetanized, it was necessary to account for these results by a diagram of the kind shown in Fig. 9. Some polysynaptic EPSP's were then actually seen by Eccles \textit{et al.}\(^{34}\) stimulating Ia afferents. Finally, Tsukahara and Ohye\(^{95}\) demonstrated that by repetitive stimulation of the cut nerves to ankle extensors at strength 50 to 60\% of the Ia maximum and recording from thin filaments in the ventral roots, there were in addition to filaments with purely monosynaptic impulses others that had one or several polysynaptic responses 0.6 msec apart. Thus, at least the tonic motoneurons have polysynaptic backing. The disappearance of
FIG. 8. The speed of onset and the maintained strength of tonic vibration reflex (TVR) in relation to the frequency of the vibration during isometric (A) and isotonic (B) conditions. A, TVR induced in finger flexors by vibration (at three different frequencies) on the volar surface of fingers. With fingers extended the subject pushes the terminal phalanges of the three middle fingers against the vibrator (amplitude 1.8 mm), trying to maintain a slight, constant pressure. Arrows indicate the sudden start and end of the vibrations. The three superimposed records show how the tonic reflex response increases with increasing frequency of vibration. Up to 10 sec are omitted where the records are interrupted. B, TVR produced in the elbow flexors by vibration (at three different frequencies) of the biceps tendon. Subject standing erect with his arm hanging passively. Diagram shows how the speed of the elbow flexion movement induced by vibration increases with increasing frequency of vibration. Vibrator amplitude 1.8 mm.38

FIG. 9. Diagram illustrating convergence of three afferent sources on common tonic interneurons (IN), projecting with common path on motoneuron (Motor N) together with monosynaptic path. Posttetanic potentiation (Ptp) occurs at synapses of interneurons on motoneuron, explaining the long-lasting facilitations by which the three reflexes support one another.39
the tonic reflexes to vibration after giving barbiturates to the patient or the decerebrate cat tells the same story.

We cannot review here the extensive work of Hagbarth and Eklund on normal subjects and in patients with various pareses and spasticities (much of it still unpublished), but our own impression is that it opens up a whole new field for the clinical worker and in an interesting manner ties it to the results obtained with animals in the study of stretch reflexes, Magnus-De Kleijn reflexes, and the gamma-spindle loop. It does this also by demonstrating that the last-mentioned postural reflexes turn up in man during activation of the large spindle afferents, signifying the facilitation of an internuncial system that the reflexes to head posture share with spindle afferents.

The evidence we have now reviewed confirms in a definite manner the notion we have had for a long time, namely, that stretch reflexes require spindle primaries biased by an active gamma loop. We see this process overemphasized in the gamma-type rigidity of the decerebrate animal. But recent work has brought into focus the so-called alpha-gamma linkage, by which is meant that muscular contractions are supported by alpha-gamma coactivation, meaning that the firing of the alpha motoneurons is modulated by spindle activity linked to it neuronally and not merely across the loop. For this connection between alphas and gammas to carry some meaning it is necessary to demonstrate it with natural stimulation and, in fact, such linked “natural” reflexes have been reported by many authors. For these reasons, it was particularly satisfying to find alpha-gamma linkage so prominent in respiration, as demonstrated in Fig. 10 from the work of Critchlow and von Euler, in which the spindles are coactivated with the inspiratory muscles (A) and lose this property (C) when the gamma supply is inactivated by cocainization in the manner of Matthews and Rushworth. C. von Euler has recently given a review of the role of the spindles in respiration to which we refer (cf. also Sears), being unable to discuss this important and extensive field fully in the present context. Suffice it to mention that in addition to the rhythmically activated gamma fibers there are tonic ones that are independent of the rhythmic activity of the alpha motoneurons, as further analyzed by Eklund et al. The tonic gamma motoneurons are linked to cerebellar stimulation and passive movements of the chest wall, suggesting a postural regulatory function for them.

Contractions of muscles governed by alpha-gamma linkage employ the gamma loop in a manner more normal than in the stretch reflex of the decerebrate animal. Any increase of length of the muscle caused by an increase of load in contraction will be counteracted by a stretch reflex started by the spindles, which on account of the linked activity have added a good share to the depolarization of the motoneuron. In Fig. 3 of the paper by Granit et al. it can be seen that stimulation
at gamma strength of the cut ventral root has produced a motoneuron depolarization of the order of 11 mV just below the threshold for firing. The number of impulses this would have produced in a firing cell can easily be measured by actually firing the cell from the tip of the intracellular electrode. In the recent work by Granit et al.\textsuperscript{57} it proved possible in such experiments to show for an ensemble of large motoneurons that the amount of depolarizing potential produced in various
reflexes was linearly related to the increase in the frequency of discharge that it produced in motoneurons fired from the inside, the constant being 2.28 impulses/sec/mV. Thus, in this particular case loop activity in a firing cell at the threshold would have controlled a range of 25 (11 mV x 2.28) impulses/sec, which serves to demonstrate that the loop by no means is negligible when cooperating as a length-measuring device in contraction. When the latter, for instance, gradually alters from isotonic to isometric — as is common enough when lifting and holding weights — the coshorteden spindles will automatically produce the required amount of compensatory motoneuronal depolarization. Thus, by the simple device of linking alpha and gamma control — in respiration at the spinal-cord level, according to von Euler — the organism has obtained an automatic adjustment of output to load. Long ago we found the link broken when the cerebellum was interfered with and assumed this to be the cause of cerebellar dysmetrias. Recently, van der Meulen and Gilman not only confirmed this fact but also supported the theory by demonstrating that recovery after cerebellectomy ran parallel with reestablishment of alpha-gamma linkage.

Why need there be separate dynamic and static control? Matthews in his valuable review of muscle spindle problems has emphasized the servo aspect of this arrangement, pointing out that in a feedback circuit the velocity-sensitive component compensates for lag and prevents oscillation caused by lag in the system; and Henatsch (cf. also Bruggencate et al.) indeed has described tremor running parallel with an experimentally produced increase in the ratio of static to dynamic control. There are basically more static than dynamic fusimotor fibers, three times more in the cat (Matthews) and six times more in the rabbit. We should also remember that in muscular activity, rate of change of length is a variable just as important as change of length and thus need be indicated by the muscle’s measuring instrument. In the end it is a question of rate of change of depolarization in motoneurons, and this dynamism the dynamic spindle effectors can handle elegantly, especially when aided by fusimotor alpha fibers of which, at least in flexors, there is a definite supply.

There is evidence from the work of Jansen and Matthews, Alnaes et al., and Jansen and Rudjord, investigating fusimotor activity in flexors and extensors in both decerebrate and spinal animals, that dynamic and static fusimotor gamma fibers actually can be biased separately.

Coming now to spindle secondaries responding to stretch of extensors in which by polysynaptic inhibition they oppose the primaries, it is of considerable importance that Jansen and Matthews found both static and dynamic gamma activity of extensors increased in the decerebrate animal. The stretch reflex in the decerebrate animal thus fights concurrent inhibition, from the gamma-activated spindle secondaries, and in addition from the tendon organs, which act in pro-
portion to the amount of active tension developed. One of us has advanced the theory\textsuperscript{48} that Sherrington's lengthening reaction, which implies that the muscle can produce the same tension at different extensions, is a consequence of the action of the secondaries, which automatically adjust autogenetic inhibition of the motoneurons in proportion to extension. Whether one believes in or chooses to deny this theory it is imperative to assume an organ measuring length and producing adjustable inhibition to it in order to understand this wonderful feat of the striate muscle whose task, otherwise, is to produce tension in proportion to extension — the more, the greater the number of muscle fibers in action. There is, as stated, no lengthening reaction in the acutely decerebellated animal lacking alpha-gamma linkage. This preparation merely has excessive alpha activity.

So far, since inhibition of the secondaries is overpowered by the excitation of the primaries, it has not yet been possible to demonstrate the autogenetic inhibition in any other way than as a diminution of the half-maximum monosynaptic response in extensor stretch, known for a long time\textsuperscript{44} and often confirmed. The depression of the monosynaptic response is greater in extensors than in flexors, as was found in a recent repetition of such experiments with both protagonists and antagonists.\textsuperscript{14,15} This should be expected from the fact that the secondaries act in the flexor reflex pattern,\textsuperscript{70} leaving only the flexor tendon organs to counteract excitation from the primaries. However, as pointed out above, no sooner is the cell fired from the tip of an intracellular microelectrode than all these autogenetic inhibitions to stretch are supplanted by excitation.\textsuperscript{55,56}

Clearly, there is not yet the same amount of clarity in our views on the effects of spindle secondaries on motoneurons as has been reached with the primaries. And it may well be that their most important function is to deliver information on static length to upper stations, in particular to the cerebellum. As far as motoneurons are concerned, one would have to expect an inhibition in extensors that should be absent in flexors. Figure 11 shows the late or delayed inhibition after an extensor contraction, which actually fulfills this criterion\textsuperscript{14} because it has not been found in the antagonist flexor.\textsuperscript{15} The experiment is an analysis by monosynaptic testing of the excitability of the motoneuron pool during and after an extensor contraction caused by a brief tetanus from the peripheral stump of the cut ventral root. The excitability changes have been related to the activity of the three types of stretch receptors. The delayed inhibition is assumed to be a consequence of the differential recovery curves of primaries and secondaries after a contraction.\textsuperscript{10}

Occasionally, one encounters the same effect in individual extensor motoneurons studied intracellularly,\textsuperscript{54} mostly as inhibitory activating noise, as in Fig. 12. Green and Kellerth\textsuperscript{62} found no sign of delayed inhibition in their intracellular study of the effects of flexor contractions on flexor motoneurons. There is an
obvious function for delayed inhibition in, for instance, stepping in that the leg extensors which possess large cross sections compared with the flexors need be well inhibited to permit bending of the leg.

In discussing biasing we have so far restricted ourselves to the gamma loop and the internuncial systems, separate for control of tendon organs and spindle organs. But recently, biasing by humoral agents has come to the fore and is likely to become increasingly important in the next phase of development of our theme. Thus, Arvidsson et al.4 have shown that drugs inhibiting monoamine transmission (e.g., reserpine) or facilitating acetylcholine transmission (e.g., physostigmine) increase motor alpha activity and decrease gamma activity, while drugs facilitating monoamine transmission (l-dopa) or inhibiting acetylcholine transmission (e.g., atropine) have the reverse effect, facilitating the gamma motor neurons and depressing the alphas. This work was carried out with intact rats,
lightly anesthetized with viadril. It is a development of Steg's analysis of reserpine rigidity in isolated alpha and gamma fibers and seems to hold considerable promise, not in the least for the clinical approach.

**ADDENDUM**

Boyd's two recent communications confirm Smith's work on single, large intrafusal fibers, but he believes the twitches to be caused by local contractions better visible in the nuclear chain fibers because of their more rapid contractions.

**REFERENCES**


PROPERTIES OF STRETCH RECEPTORS


DISCUSSION

DR. NELSON: I would just like to add a comment to Dr. Kellerth’s discussion of the organization of afferent input of the spinal cord. There are two points: one deals with how much of the dendritic receptive area of the motoneuron is relevant for determining the excitability of the motoneuron. That is, how effective are the synapses distributed to different regions of the dendritic tree? A detailed analysis of morphologic and electrical data indicates that, in fact, even the most peripheral dendrites are electrically fairly close to the somatic or axonal trigger zone (J. Neurophysiol. 30: 1097–1112, 1967). The entire dendritic synaptic connections thus must be considered as important determinants of motoneuronal firing. The second point concerns the question of where the synapses responsible for the monosynaptic EPSP produced by Iα afferent fibers are distributed on the motoneuron. This has been approached by intracellular and theoretical studies done by Wilfrid Rall, Robert Burke, Thomas Smith, Karl Frank, and me in three ways (J. Neurophysiol. 30: 1169–1193, 1967).

The first method was to measure the change in membrane impedance that would be expected to accompany the EPSP. The second was to study the wave form of EPSP’s evoked by various means, and the third was to examine the effects of polarizing currents on EPSP wave form and amplitude. Synaptic potentials occurring close to the intracellular recording site would be expected to have a fast time course, be accompanied by a measurable impedance change, and be altered by polarizing currents. PSP’s generated in the distal dendrites would be
slower, not accompanied by a measurable impedance change, and little affected
by polarizing currents. The data indicate that, in fact, a wide spectrum of behavior
is seen, indicative of a wide dendritic distribution of Ia synapses. The interaction
between different PSP's likewise is consistent with a wide distribution of these
synapses. Thus, the entire dendritic tree is an effective target for synaptic input,
and interaction in the dendritic tree may be an important factor governing the
integration of information impinging on a given neuron.

Dr. Eccles: I have one or two points to make here. First, going back to quite
early in Dr. Kellerth's paper, I would like to raise a question concerning the Ib
afferents of flexors and extensors. Now, our work in Canberra showed that the
Ib afferents of flexors are very heavily concerned in presynaptic inhibition. Lund-
berg and ourselves agree with Dr. Granit that the Ib afferents from flexors are
not much concerned in the ordinary Ib inhibition of flexor motoneurons. In fact,
the Ib afferents from flexors give very little in the way either of EPSP's or IPSP's
on motoneurons. On the contrary, the Ib afferents from flexors are very effective
in producing presynaptic inhibition. I am sure that there is an important physio-
logical story in the presynaptic inhibition from flexors controlling, to a con-
siderable extent, the group Ia input to motoneurons.

Do you find this autogenetic inhibitory action of flexor muscles regularly with
flexor motoneurons? That's one question.

The other question is this: How do you account for, or do you not account for,
the results of Devanandan, Rosamond Eccles, and Yokota and of Devanandan,
Eccles, and Stenhouse who show that flexor muscle contractions or passive
stretches bring about powerful presynaptic inhibitions of the Ia afferent
pathway?

Dr. Kellerth: Dr. Green and I have recently been studying the intracellular
reflex effects produced by contraction of the ankle flexor muscles tibialis anterior
and extensor digitorum longus. We found, indeed, that these contractions in-
duced powerful inhibitory postsynaptic potentials in homonymous and synergic
flexor motoneurons. Furthermore, we were able to correlate these postsynaptic
flexor inhibitions only to the activation of Golgi tendon organs during muscle
contraction. A preliminary report of this study has been published (Green and
Kellerth^2) and the work will soon be reported in extenso. The discrepancy
between our results and those of, e.g., Eccles, Eccles, and Lundberg^35 may
possibly be due to the Golgi tendon organs discharging repetitively during a
muscle contraction and in this way giving rise to inhibitory effects which may be
difficult to reproduce with single-shock stimulation of muscle afferents.

In the present paper we decided to leave out the aspects of presynaptic in-
hilation, partly because of the need for space for other complex questions and partly because the problem of the indicators of presynaptic inhibition was scrutinized in September, 1966, at the Fourth International Meeting of Neurobiologists in Stockholm. At this symposium the case for and against the validity of the indicators used to establish presynaptic inhibition was critically discussed (Eccles, Granit, Kellerc, Lundberg, and others). We did not want to repeat this discussion here because the Stockholm symposium will be published in the Wenner-Gren International Symposia Series by the Pergamon Press (edited by Curt von Euler) and our criticism thus will be accessible within a reasonable time.

Contraction of the ankle flexor muscles induced postsynaptic inhibition in 40 out of the 44 flexor motoneurons investigated, which shows that the inhibition was quite a regular finding.

On magnifying the few intracellular records of Devanandan et al. (1965b, J. Physiol., 179: 430-441) referring to the diminution of the monosynaptic EPSP, alleged to take place with no influence on the membrane potential of the motoneurons, we found these EPSP's exhibit an accelerated fall to the baseline, suggesting a conductance change, and hence were not convinced that the cell membranes had been unaffected by the conditioning stimulus. Our group in Stockholm has found that stretch and contraction engage the postsynaptic membrane of motoneurons, while Devanandan et al. (1965, a and b; 1966) have shown that in addition dorsal root potentials are evoked, the physiological significance of which, however, in my opinion is still somewhat elusive.

DR. HENNEMAN: I'd just like to emphasize again what Dr. Kellerth has already mentioned, this matter of the threshold of the tendon organ being very low to muscular contractions but high to stretch. In fact, in some recent work now in press from our laboratory, we have demonstrated to our satisfaction that tendon organs do not respond to passive stretch at all, so long as the passive stretch does not exceed the physiological lengths of the muscle. It's quite true that if you stretch a muscle beyond the normal length it would ever reach in the body, tendon organs will respond. But if you keep your passive stretch within physiological limits, these endings are completely unresponsive.

Therefore, I think there is now a serious question as to whether a tendon organ should be called, in the old sense, a stretch receptor. Perhaps this designation should remain in some doubt.

On the other hand, the tendon organ is so sensitive to muscular contractions that we've been able to show that the contraction of a single motor unit in an otherwise completely quiescent muscle is quite sufficient to discharge a tendon organ.
Dr. Kellerth: I would like to ask Dr. Henneman whether he has tried to correlate the stretch sensitivity of tendon organs with the location of the receptors in the muscle. Jansen and Rudjord in Oslo showed that a number of tendon organs in the soleus muscle responded to stretch, and thus to tension as such, but that the great majority gave considerably greater responses to contractile tension. Recently, Ainaes in the same laboratory extended this work to the ankle flexors (1967, Acta Physiol. Scand., 70: 176–181), and the Oslo laboratory has kindly given me access to the data. The flexors had a very much larger number of tendon organs that responded to absolute tension, passive or active. The tendon organs that required contractile tension for giving their full-range responses were found to be located in the upper portion, and those sensitive to absolute tension, at the distal end of the muscle. This rule held for both extensors and flexors. Thus, the “peculiar” behavior of a tendon organ may depend upon the location of the receptor in the muscle.

Dr. Henneman: No, we’ve not correlated it with the position of the receptor in the muscle, but we’ve never found a tendon organ which would respond within this physiological length — and the length measured was the ankle, let’s say, fully extended.