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# THE EARLY DISCHARGE OF MAMMALIAN MUSCLE SPINDLES AT ONSET OF CONTRACTION

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There are several papers in which an early mass discharge in the undivided sensory root has been observed upon stimulation of the peripheral stump of severed motor roots (Dun & Feng, 1940; Masland & Wigton, 1940; Lloyd, 1941, 1942; Leksell, 1945; Granit & Skoglund, 1945). Beyond mentioning that such early discharges may come from artificial synapses at cut nerve ends, from ephaptic activation of sensory terminals by the muscle action potential and from muscle receptors, these papers will not now be discussed. Analysis at the single-fibre level, as we shall see, resolves many of the complexities encountered with mass discharges, but it is necessary to be aware of the sources of error elucidated in the papers mentioned.

In the previous paper (Granit, Pompeiano & Waltman, 1959) we showed that co-activation of extrafusal muscle and muscle spindles occurs in response to stimulation of, for example, the medullary pyramids or Deiters's nucleus, so that spikes from large spindle afferents can be picked up in dorsal root filaments early enough to make them coincide in time with the incipient contraction of their muscles of origin. Hunt & Kuffler (1951) had seen an early discharge from spindles when stimulating muscle nerve or ventral root, in fact too early to be caused by $\gamma$ fibres, and hence raised the issue whether there might not also be more rapidly conducting fibres for the spindles. This they found improbable. Our results with brain-stem stimulation provided evidence for a fast physiological mechanism of spindle activation and thus suggested that the early discharge deserved to be reinvestigated.

This conclusion is underlined by the considerable amount of histological evidence in favour of thicker fibres than those of the $\gamma$ system running to muscle spindles (see quotations in Granit, Pompeiano & Waltman, 1959). Barker (1948, p. 157), working on leg muscles of cats and rabbits, even concluded that 'in all probability the two polar halves of an intrafusal muscle-fibre function as independent contractile units', the reason being that he often

found one, sometimes two large fibres occupying one pole by themselves. These were 6–7 $\mu$, the smaller ones 3–4 $\mu$ in gold chloride stain. Recently Cooper & Daniel (1956) have seen large fibres in the poles of spindles in human hand muscles and have reopened the question as to what extent spindles may receive $\alpha$ motor fibres.

Hunt & Kuffler (1951) found that the early discharge depended upon muscle tension and that its latency (to be called in some contexts 'loop time') could be prolonged up to 6 msec when muscle tension was reduced. It could also be elicited by excitation of motor units, each of which could not be expected to cause potential changes sufficient for excitation by the muscle action potential. Their experiments on root division gave equivocal results (see full quotation below, p. 412). Their final conclusion (p. 310) was that the 'early discharge is caused by tension changes which arise within the muscle before they are recorded myographically'. As a serious argument against concluding that the early discharge is an $\alpha$ activation they also regarded the fact that it was seen in both the *A* (spindles) and *B* (Golgi tendon) organs of Matthews (1933). We feel, on the contrary, that this would be a consequence of genuine intrafusal fast activation. The spindles, though in parallel with muscle, have their afferent endings in series with intrafusal muscle, just as the tendon organs are in series with extrafusal muscle. Therefore both should be activated by contractions in their respective muscular elements. However, the extrafusal contraction may well delay the moment at which an intrafusal contraction can activate its sense organ, as was emphasized by Granit, Pompeiano & Waltman (1959) and shown to hold good for $\gamma$ activation (their Fig. 1). In their case the onset of $\gamma$ activation, as measured by a spindle discharge, was accelerated by selective removal of the extrafusal contraction with Flaxedil (gallamine triethiodide; May and Baker, Ltd). In preferring a tension theory to what might be called an 'innervation theory' Hunt & Kuffler's chief support came from experiments on root division (to be discussed below).

Spindles are length receptors inserted so as to make their two poles approach each other in extrafusal contraction. They will thereby unload the equatorial region containing the sense organ on the nuclear bag. This should now be well known. Therefore the evidence for quite the opposite mechanism, implied in the 'tension theory' of Hunt & Kuffler (1951), has to be particularly compelling to be conclusive, even when it is granted that some spindles may be displaced and pulled upon by contraction in adjacent extrafusal bundles. In order to exclude freakish effects it is necessary to have some idea of the distribution of latencies and the regularity with which the early discharge occurs at different extensions in muscles of different twitch times. In the present work we have studied 168 spindles in both slowly and rapidly contracting muscles, extensors as well as flexors of cats, and made a number of supplementary observations on rabbit spindles which behave similarly. In addition we have studied 19 Golgi tendon organs.

It should at the outset be realized that increased extension of a muscle may cause two antagonistic effects. (1) It will improve the phasic sensitivity of the spindles (Granit & Henatsch, 1956; Lippold, Redfearn & Vučo, 1958), which is important in fast activation. (2) It will cause larger contractions likely to unload spindles more effectively than contractions from zero extension. This would be expected to delay activation. The balance point of these two antagonistic effects may be very different in different spindles.

## METHODS

This is a continuation of the previous work on fast supraspinal activation of spindles (Granit, Pompeiano & Waltman, 1959), in part even making use of the same animals. A few were decerebrated, the others were anaesthetized with 20 mg chloralose and 10 mg pentobarbitone per kilogram. In both types of experiments a stimulating electrode was placed on the appropriate muscle nerve in the popliteal space, at knee level, both in order to determine conduction velocity in the spindle afferent isolated in a dorsal root filament and to study the response to contraction. The term 'slack muscle' means that the muscle is unhooked from the myograph. Another pair of stimulating electrodes, when used at all, was placed on the peripheral stump of the cut ventral root L7 or S1. In many experiments root stimulation was not employed; the ventral roots were then intact.

## RESULTS

*Comparisons of different muscles. Loop time.* The first question concerns the percentage of spindles capable of discharging early spikes when the shock is delivered to the nerve at knee level. This was found to be for soleus 77% out of a total of 31, for gastrocnemius 50% of 52, for tibialis anterior + extensor digitorum longus 40% of 85. Now, time to peak of twitch is around 60 msec for soleus, around 30 msec for gastrocnemius and below 20 msec for tibialis anterior (Cooper & Eccles, 1930; Gordon & Phillips, 1953). Extensor digitorum longus is also a fast muscle with contraction times of the order of those found in tibialis anterior. Thus, what our results show is that in the slow soleus the early discharge is far more regularly obtained than in the fast flexors. This, of course, is quite the opposite of what a tension theory would require. Early in contraction there is far more tension produced in fast muscles.

Figure 1A serves as a first introduction to what proved to be the major obstacle to an understanding of this problem. Records 1 and 2 show the early spike in a slack soleus at two shock strengths. The first spike is the direct spike (DS), slightly interfered with by some other spikes from the gastrocnemius nerves, the electrode being at the knee. In 1 the latency of the earliest spike from muscle (ES), is long. The stronger shock in 2 brings in a still earlier spike that is now our ES, but the other one is found in its former temporal position. In 3 and 4 the same experiment is repeated with the muscle hooked to the myograph. Our original ES in 1 is now doubled and has a slightly shorter latent period. With the stronger shock in 4 we again obtain the ES of record 2. There were thus, in this experiment, two types of ES, one isolated in record 1 being of low threshold and being doubled in 3 and 4 when the muscle is extended, while the other, seen only with the slightly stronger shocks in 2 and 4, has a very brief latent period, and in this particular case requires the number of $\alpha$ fibres excited by the stronger shock, but neither any extension nor the tension produced by the shock.

Figure 1B has been put in merely to confirm the finding by Hunt & Kuffler (1951) that the very early spike can also occur in a Golgi tendon organ. After

a pause this organ responds to tension in accordance with the definition of its properties (Matthews, 1933). Figure 1 also shows that the first type of ES occurs before visible contraction in a myogram, the second at the foot of the contraction. Both types are seen in extensors, but practically the latter type

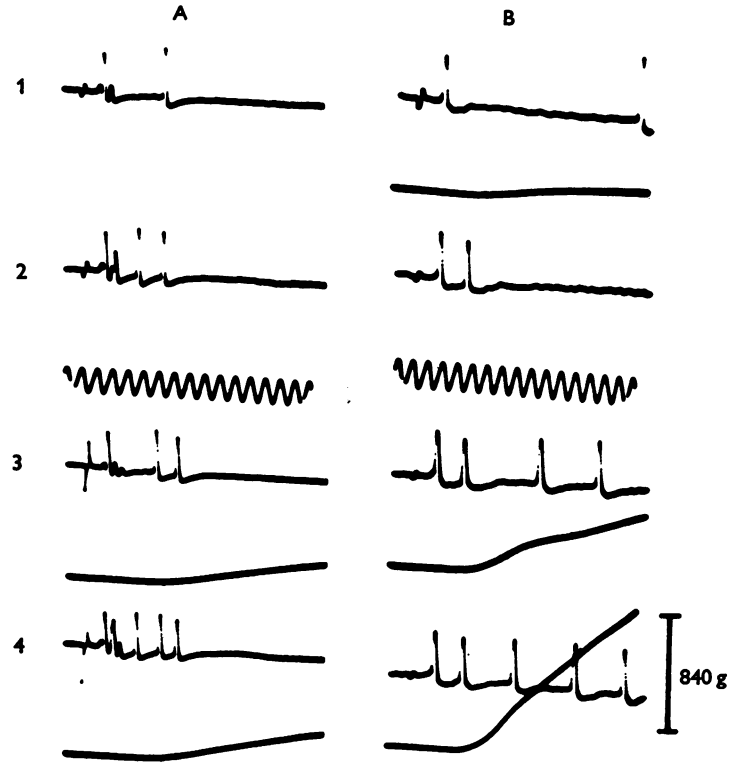


Fig. 1. A: Soleus spindle afferent in anaesthetized cat. Nerve stimulation. 1, shock strength 1·3 times threshold for direct spike, slack muscle; 2, same at 1·6 times threshold. Earliest spike in 1 at latency 5·2, in 2 at 3·4 msec; second spike in 2 at 5·2 msec. 3 and 4 at 15 mm extension and shock strengths 1·3 and 2·0 times threshold respectively. Conduction time of direct spike is 1·4 msec. B: Gastrocnemius tendon organ afferent in anaesthetized cat. Nerve stimulation. 1, at threshold for direct spike, conduction time 1·3 msec; 2, at 1·6 times threshold, slack muscle; 3, same strength at 4 mm extension; 4, same at 10 mm extension. Earliest spike at 3·2 msec. Time 1000 c/s. Calibration of myograph in this and all successive records marked for 1 cm on original film.

alone in flexors. For this reason it was of considerable interest to study the distribution of ES latencies in our material.

Within the early discharge we have measured the time to the ES. From this value has been subtracted the conduction time from knee level to dorsal root of the direct spike (DS). In the histogram of Fig. 2 loop time is ES–DS which, accordingly, refers to the loop traversed from nerve electrode at the upper

edge of the gastrocnemius muscle, round the muscle and back to nerve electrode. Only five flexor spindle responses were of the type conducted in slow afferents (40–60 m/sec). Three of them had no ES, the other two had loop times of 5·2 and 6·5 msec respectively. The rest, 163 spindles, were of the nuclear bag variety. Figure 2, of course, only includes the spindles that had an ES. No differences were noted in the conduction velocities between spindles and tendon organ afferents, which agrees with the results of Hunt & Kuffler (1951).

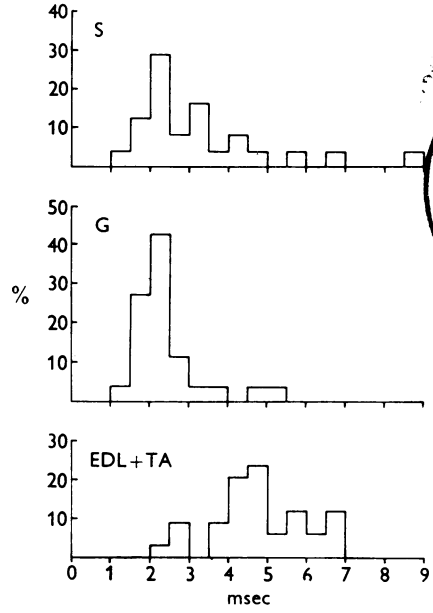




Fig. 2. Percentage distribution of loop time. From above downwards: 24 spindles in soleus, 26 spindles in gastrocnemius, 34 spindles in tibialis anterior and extensor digitorum longus.

The histogram shows that loop times in gastrocnemius were short and well concentrated within a narrow range. Some in soleus occupy the same range but others spread out into longer values. In the flexors, only five ES had short loop time, the others had long ones, but no longer than the longest in soleus. It is impossible to ascribe all these differences to variation in conduction times.

Gastrocnemius and tibialis anterior have very similar efferent calibre spectra for large fibres (Rexed & Therman, 1948). Extensor digitorum longus is likewise very similar to gastrocnemius (Eccles & Sherrington, 1930). Only soleus has efferent $\alpha$ fibres considerably thinner than the others (Eccles & Sherrington, 1930). Even if the long loop times in soleus were attributed to this factor, there remain to be explained the equally long loop times in the flexors. In our experiments with electrodes at the upper edge of gastrocnemius there is a further 3 cm to this muscle and soleus, and 6–8 cm to

the flexors to account for. This, at 100 m/sec, would add around 1·0 msec to the flexor loop time (8 − 3 = 5 cm, twice over) which is insufficient to explain the fundamental differences between flexors and extensors. Below it will be shown that the distribution of loop times hinges upon other factors.

Figure 1 has already given clear indication of the basic disclosure of our analysis, namely that the early discharge is a heterogeneous event within which two components clearly stand out. One is the spike of very short loop time, visible before contraction, the other is the spike at the foot of the contraction. The diagram of Fig. 2 bears out our conclusion that the former spike is rare in flexors. Hunt & Kuffler's tension theory can only refer to the former type of spike, because they speak of it as 'caused by tension changes which arise within the muscle before they are recorded myographically'. However they picture both types in a figure (their Fig. 10) which is not unlike our Fig. 1.

Figure 2 adds substance to our criticism of the tension theory. Why is the very early spike so rare in the flexors which, in fact, produce visible tension a good millisecond before anything is seen in the soleus myogram and, furthermore, do so at a very much faster rate? As a matter of fact, we do not find this very early spike to require any tension. It is, when present at all, regularly present in slack muscle, as in Fig. 1, and only requires a sufficiently strong synchronous shock to appear. It has the same property in the flexors in the rare instances when they possess it. The group of slightly later spikes behaves very differently. It will clarify presentation if the early component is dealt with first, and if, in doing so, extension, tension and number of efferent fibres activated by a shock (stimulus strength) are considered separately, whenever possible.

*Ephaptic nature of first component.* The first component, if elicited at near-threshold strength, is sensitive to extension. It may disappear when the muscle is pulled out. When the shock is strong the spike generally resists this effect of extension. However, sometimes no amount of stimulus strength can prevent the disappearance of the first component of ES.

In Fig. 3 are presented two such extreme cases of the effect of extension. It should be noted that there is only one ES and no later burst. A is a gastrocnemius spindle stimulated at knee level. In 1, at zero extension, the DS is shown. Some increase of stimulus strength in 2 adds the ES, shown in 3 at 5 mm extension. In 4, at 8 mm extension, it is gone but returns in 5 when the muscle is made slack. This experiment was next repeated with maximal shocks and the electrodes lifted by hand so as to pull slightly on the nerve and make good contact. The same result was obtained. In B 1 is the DS from another gastrocnemius spindle. B 2 is excitation from the ventral root for calculation of conduction velocity (see legend and below). In B 3 a strong stimulus is used, as is also seen by the fact that other spikes add themselves to the DS functionally isolated. Records B 3 and B 4 are at zero and 5 mm

extension respectively. With 11 mm extension, in B5, the ES is gone. Thus, contrary to the tension theory, an ES may disappear when the muscle is extended and produces more tension. Again it should be noted that the early

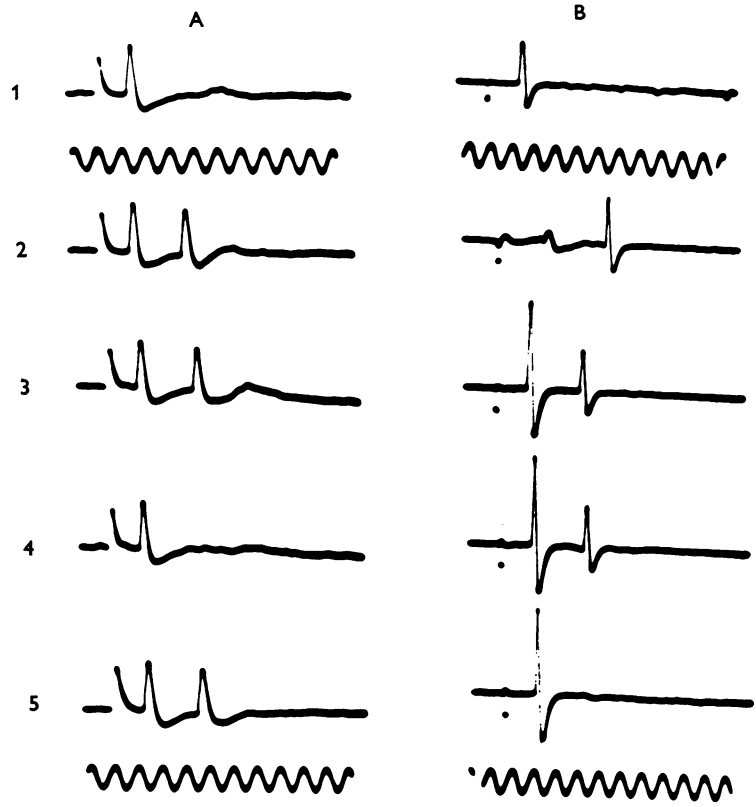


Fig. 3. Two spindle afferents from gastrocnemius of anaesthetized animals. Stimulating nerve. A. 1, direct spike, conduction time 1·2 msec; 2, strong shock elicits early spike at latency 3·5 msec. Zero tension; 3, same at 5 mm extension; 4, at 8 mm extension early spike disappears; 5, return of early spike in slack muscle. Loop time, which is ES–DS, equals 2·3 msec. B, another similar experiment. 1, direct spike, conduction time 1·2 msec: 2, stimulation from ventral root, spike after 5·0 msec; 3 and 4, strong shock to nerve elicits early spike from slack muscle with perfect regularity and augments direct spike by component of unknown origin. Latency 3·7 msec. 5, disappearance of early spike when muscle extended 11 mm. Loop time 2·5 msec. Nerve latency subtracted from root latency gives 1·3 msec or 115 m/sec, conduction distance being 15 cm. Time 1000 c/s.

spikes studied in Fig. 3 were extensor spikes of very brief loop time not followed by a burst at any stimulus strength or extension. It is pertinent to ask: why no later burst despite a great deal of tension? We shall provide good evidence for our preliminary conclusion that this is because the second component of ES is absent in these particular spindles.

Since the first component (see Fig. 1) also occurs in Golgi tendon organs, its

properties are next (Fig. 4) analysed in two such preparations. A 1 shows the ES in slack muscle. A burst follows when the muscle picks up tension (A 2). In A 4 the ES is absent at 10 mm extension and is but irregularly present at 9 mm extension in A 3. In B, with slack muscle, a 'frequency test' is carried out. This is generally done with slack muscle to avoid interference with the genuine effects of contraction. The tendon organ ES follows rate of stimulation to around 160/sec. C and D show a similar experiment with another tendon organ (see legend). It followed 200/sec in the frequency test with slack muscle

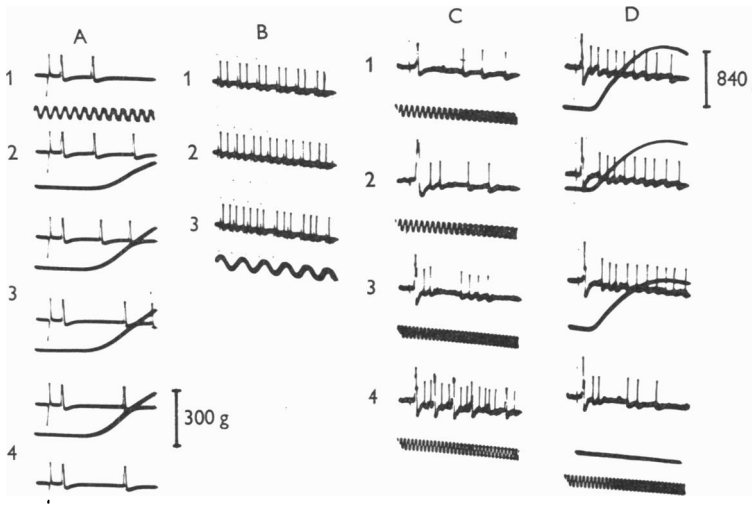


Fig. 4. Golgi tendon organs in tibialis anterior from two anaesthetized animals. Stimulating nerve. A and B, first experiment. A: 1, slack muscle, strong stimulus; direct spike with conduction time 1·5 msec. 2, at zero tension the early spike of latency 4·5 msec and loop time 3·0 msec is succeeded by spike caused by contraction. 3, at 9 mm extension the earliest spike becomes irregular. 4, at 10 mm extension the earliest spike is gone and the next in turn has its briefest latency, 7·6 msec (loop time 6·1). Records in B are frequency tests with slack muscle. 1, at 110/sec; 2, at 135/sec; 3, at 167/sec where following becomes irregular. Time 1000 c/s for A, 100 c/s for B. C and D, second experiment. C: 1, slack muscle, weak stimulus. 2, same with strong stimulus. 3, same with shrunk time base. 4, frequency test at 200/sec, where following still occurs, loop time (4·0–1·7) being 2·3 msec. Records in D, 1–3, to show disappearance of earliest response at higher extensions. 1, 4 mm; 2, 15 mm (spike gone); 3, 17 mm; 4, control with slack muscle. Early spike again present. Time 1000 c/s.

(C 4). The ES, so far from being favoured by tension, disappeared when tension increased as a consequence of applied extension (D 2 and 3).

The frequency test with slack muscle always succeeds and does so equally well with the similar ES component in muscle spindle afferents (see below, Fig. 6). Tendon organs in a flexor have been chosen for the particular reason that the frequency test fails with ES in flexor spindles except in the rare cases when the ES occurs before visible contraction (see below). All Golgi tendon organs, by definition (Matthews, 1933), will have the secondary burst in

response to tension and so from them one may obtain the loop time for a possible genuine effect on sense organs by contracting muscular tissue. It is, for instance, 6·1 msec in Fig. 4 A.

It is evident that genuine activation by contraction is dependent upon two factors, threshold of end organ and contraction itself. If spindles have a genuine mechanism, based on fast contraction, of the order of velocity seen in the surrounding extrafusal fibres from which they ontogenetically originate (Cuajunco, 1927), loop times may be slightly shorter because spindles tend to have lower thresholds than tendon organs (Matthews, 1933; Hunt & Kuffler, 1951). With Golgi organs in flexors we have had loop times for the secondary discharge from 4·2 to 8·5 msec. A glance at Fig. 2 will show that this includes the range of loop times in flexor spindles, excluding five spindles (6 %) of very brief loop times belonging to spikes preceding contraction and responding positively to the frequency test. For this test values of the order of 130/sec tend to be a minimum. The maximum runs up to values around 250/sec.

We have called these spikes ephaptic because their most likely mechanism of origin is direct activation by the muscle action potential, as was well elucidated by Lloyd (1942) when he showed that the mass discharge of dorsal root spikes follows the fast stimulus frequencies used above. To this we have only added the criterion that our first component of ES should do this in slack muscle. Leksell's (1945) 'back response' is identical with Lloyd's secondary centripetal discharge and Leksell gives one illustration (Fig. 28, p. 56) of this response diminishing when the muscle is pulled upon. The efferent conduction velocity, obtained from a comparison of root with nerve stimulation, is always in the upper $\alpha$ range (see legend of Fig. 3).

*Comparison with muscle action potential.* Our conditions have not been favourable for recording the muscle action potential, but Eccles & O'Connor (1939) have devoted a careful study to this very problem in two of the muscles we have used, soleus and tibialis anterior. Their electrode distance was 1 cm, ours averaged 3 cm for the extensors and 7 cm for the flexors (see above). In soleus the muscle action potential occurred after a delay that was usually 0·6, occasionally 1·0 msec. In our case, at 3 cm, the longer value is likely to be the better choice. The negative potential wave at the end-plate region lasted 2 msec in soleus, 1·5 msec in tibialis anterior. The longer efferent conduction distance in the flexors we assume to be roughly compensated for by the faster conduction velocity in comparison with soleus. The muscle action potential will stimulate on the rising phase, in favourable cases a second time on the falling phase (double spikes are seen from time to time). Since loop times are calculated with reference to the nerve electrodes it is necessary to add afferent conduction times of approximately 0·2 msec for soleus and 0·5 msec for the flexors. Thus loop times below 2·5 msec must be ephaptic and very likely, with our electrode arrangements, 3·0 msec is a critical duration in extensors, 3·5 msec in flexors. For practical purposes the frequency test, when positive, means that such spikes cannot be considered relevant to our problem, which concerns the possibility of genuine activation. This problem presents itself in pure form in most flexor spikes for which, also, the briefest loop time of Golgi tendon organs, 4·2 msec, is in good agreement with our measurements for spindles (Fig. 2), if 0·5–1·0 msec is added for initiation of the contraction, be it then intra- or extrafusal. The sense organs themselves have extremely high phasic sensitivity when extended, or under $\gamma$ bias (Granit & Henatsch, 1956) and will respond to elongations at the rate of a few microns per millisecond.

*Early spikes activated by contraction.* The genuine early discharge is seen in both extensors and flexors, though in the former more often than not complicated by the ephaptic spike. Figure 5 shows the separation of the two components for a soleus spindle afferent, stimulated from the root in order to exclude reflex interference and pull upon the muscle nerve (for anatomical reasons less of a complication with flexors). In this case the records at different extensions are at threshold, (1), at 1·7 times threshold to show the range of variation in 2 and 3, and at 3·3 times threshold in 4. The earliest spike cluster,

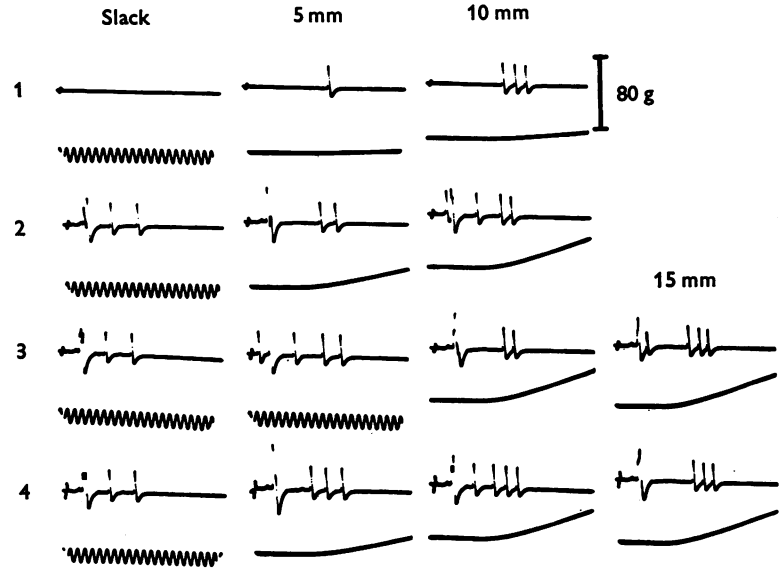


Fig. 5. Soleus spindle afferent in anaesthetized animal. The direct spike from nerve had conduction velocity 100 m/sec or 1·5 msec conduction time. Stimulation of ventral root L7. The first response seen with stronger shocks is from an unknown source near spine; its latency was only 2·5 msec. The earliest spike seen in slack muscle has latency 5·6 msec. The 'burst' is seen at the foot of the contraction. Separation of earliest spike and burst by variation of shock strength and extension. 1, at threshold, 0·03 V; 2 and 3, 0·05 V; 4, 0·5 V. Shortest latency of definite burst in 4 at 5 mm is 8·0 msec. At 15 mm it is 10 msec. Time 1000 c/s. Note that the earliest spike is irregularly present at 10 mm extension and disappears at 15 mm extension. Latency of burst lengthened at 15 mm extension.

whose pattern is rather variable, probably comes from an unidentified muscle near the spine; less probably, from an artificial synapse in the cut hamstring nerve (cf. p. 399). However, the regular spikes are clearly defined. A shift of loop time for the ES is seen when the muscle is lengthened, and though occasionally at 5–10 mm loop time reverts to short values, it never does at 15 mm. This means that for this particular figure we have chosen a spindle of the type analysed above, characterized by an ephaptic spike sensitive to extension. The ephaptic spike has disappeared at 15 mm extension. As stated,

spindles have this property if studied at shock strength near threshold, in confirmation of which the genuine burst is seen alone also at 10 mm in the uppermost line of Fig. 5. Its latency varies a little with stimulus strength. Extension also is important. Latency is shortest at 5 and 10 mm (8·0 msec), line 4. Tension rises faster and to much greater values at 15 mm where the latency is 10 msec, lines 3 and 4, in agreement with our conclusion that the rising tension, so far from favouring the appearance of the spike, actually delays it. This need not, however, always be the case because, as already pointed out (p. 400), the equilibrium point between the favourable effect of extension on phasic sensitivity and the unloading of the spindle by the increase in extrafusal tension is unpredictable. In the present case the favourable effect

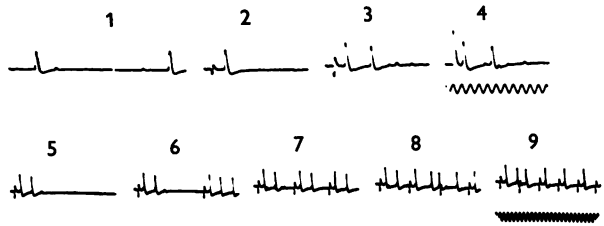


Fig. 6. Stimulation of nerve to study response of same spindle as in Fig. 5 to shock frequencies. Slack muscle. 1, spontaneous activity; 2, direct spike of conduction time 1·5 msec at threshold strength, 0·63 V; 3, at 1·0 V; 4, at 6·3 V with time 1000 c/s. Latency of earliest spike in slack muscle was between 4·2 and 4·5 msec, hence loop time 2·7 msec; 5–9, time base shrunk for frequency test at 1·0 V. 5, very slow frequency; 6, 60/sec; 7, 110/sec; 8, 155/sec; 9, 200/sec and time 1000 c/s. Note lengthening of latency and failure of driving beginning in 8. (Fig. 7, A4, shows failure of frequency test with flexor spindle.)

of extension is on the number and frequency of the spikes in the burst. Tension delays the process. This is commonly seen.

Figure 6 completes the argument by showing the result of the frequency test with the ephaptic spike of this particular spindle. The figure is fully explained in its legend.

While Figs. 5 and 6 serve to show how it is possible in extensors to separate the ephaptic spike from the genuine effect, our own procedure in studying the latter has been to take advantage of the natural isolation of the genuine process in flexors. In them it is not very common to find the early second component of the discharge in slack muscle. Some flexor spindles show it at zero initial length, others require some extension. Having noticed that the 'burst' of early spikes tended to be more prominent in the active spindles of extensors in the decerebrate preparation, we tried to raise the $\gamma$ bias of flexor spindles which failed to give early spikes in slack muscle. Such experiments are shown in Fig. 7. A1 illustrates the DS in slack muscle. There was no ES. Then some manipulation of the limb started activity from the spindle and if the shock was fitted into this discharge, the spindle responded with an ES (A2).

Record A 3 was put in to demonstrate that this ES belonged to the characteristic flexor type, discharging at the foot of the contraction; record 4 shows that it did not follow frequency 67/sec. The records B begin with slack

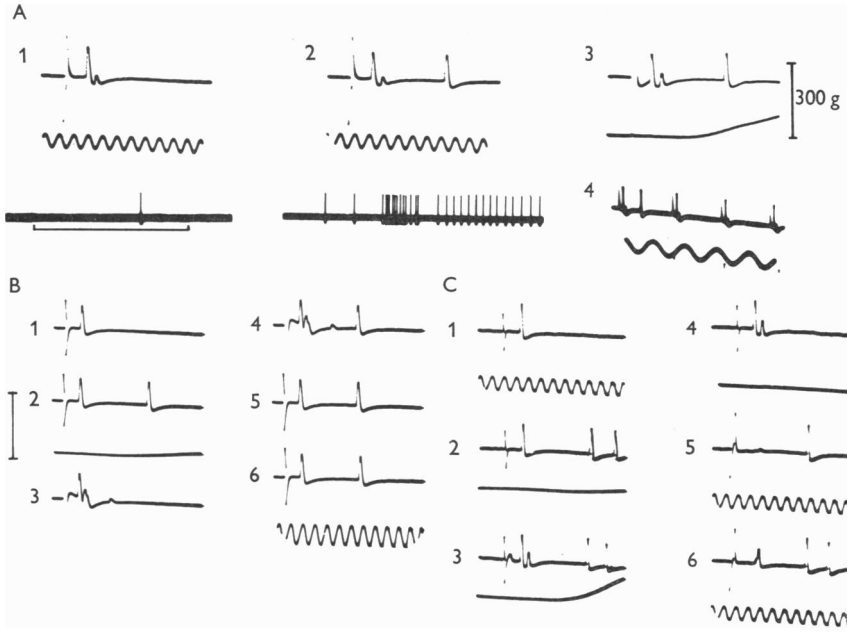


Fig. 7. Spindle afferents from flexors. Anaesthetized cats. Stimulating nerve. A, extensor digitorum longus. *Above*, records on sweep circuit and time 1000 c/s; *below*, simultaneous records on standing spot, 0·5 sec marked below middle record. 1, only direct spike of conduction time 1·5 msec when spontaneous activity low in slack muscle; 2, an early spike of latency 6·5 msec could be elicited at same shock strength after starting spindle activity by slight manipulation of leg; 3, contraction at zero extension to show position of spike relative to myogram—spike always present at zero; 4, only direct spike followed frequency 67/sec at this extension, nor did early spike in slack muscle, even when activated, follow frequencies between 25 and 50/sec. B, extensor digitorum longus; calibration of myograph as in A. 1, slack muscle, direct spike of conduction time 1·3 msec. Shock strength 0·32 V; 2, same at zero extension elicits early spike of latency 7·0 msec; 3, back to slack muscle but now shock strength 1·0 V, in spite of which no early spike; 4, after a 1·5 sec tetanus at 67/sec to stimulate $\gamma$ fibres there is now an early spike in slack muscle at 1·0 V; 5–6, cutting down stimulus strength to original 0·32 V; this spike was seen in slack muscle for some time after the tetanus. Latency now 6·0 msec. Time 1000 c/s. C, tibialis anterior. 1, slack muscle, direct spike of conduction time 1·4 msec; 2, zero extension, shock strength 0·1 V, early spike at latency 7·0 msec; 3, shock strength now 0·25 V; 4, same in slack muscle, no early spike, nor could one be elicited by increasing shock strength; 5, very weak stimulus below strength for DS through another stimulator, then tetanus of popliteal nerve for flexor reflex on separate stimulator; records taken after tetanization during reflex in $\gamma$ motoneurones; 6, repetition of 5 to demonstrate occasional double responses. Early spike in 5 and 6 has latent period of 6·0 msec and loop time of 4·6 msec, thus shorter than at zero extension in spite of muscle being kept slack from 4 to 6. Neither by flexor reflex nor by any amount of extension could latency be pushed below 6·0 msec.

muscle in which (in 1) only a DS is seen, but at zero extension in B 2 an ES can be obtained at time 7·0 msec from shock artifact. Yet in B 3, when the muscle was again unhooked from the myograph, a 3·3 times stronger shock (eliciting another component spike in the filament) failed to raise an ES. The nerve was therefore briefly tetanized at this strength (suprathreshold for $\gamma$ fibres) and immediately afterwards, in 4, an ES was obtained which in 5 and 6 was shown to require only the weaker shock of record 1, yet had a shorter latency, 6·0 msec. Activation could not in this case be obtained by tetani just maximal for extrafusal contraction. Finally, records C illustrate a case of spindle activation by a (popliteal) flexor reflex. Record 1 is the DS; in 2, at zero extension, a weak shock elicits a burst; in 3 a stronger shock shortens the interval between the pair of spikes in the burst but fails to have any effect in 4 when muscle is made slack. Then the spindle was activated reflexly to increase its $\gamma$ bias. Afterwards, in 5, an ES occurred in the slack state, sometimes double (6).

From experiments of this type it is concluded that intrafusal pull on the spindle's equatorial region containing the sense organ may replace extension of the muscle and, hence, that extrafusal tension cannot be regarded as the decisive factor whenever it is automatically increased by an increase of stimulus strength or extension. Sufficient activation of the spindle itself may often make it discharge an early spike or even a burst of spikes in slack muscle. These results were confirmed in a number of experiments in which the early spike was lost after de-efferentation (preceding root stimulation) but could be re-activated by post-tetanic potentiation at sufficient strength. It is clear that, since spindles differ widely in their sensitivity to $\gamma$ stimulation, activation by various means does not always succeed. Occasionally a tetanus may also silence a spindle (Kuffler, Hunt, & Quilliam, 1951; Granit, Homma & Matthews, 1959).

Flaxedil has been used in this laboratory with mammals (Granit, Homma & Matthews, 1959; Granit, Pompeiano & Waltman, 1959) to suppress extrafusal contraction with preservation of good $\gamma$ reflexes, and the same result has been obtained independently by Henatsch & Schulte (1958) with frog muscle spindles. Since in frogs $\alpha$ activation of spindles also is spared by Flaxedil (Henatsch & Schulte) it was used here at the end of several experiments with the early discharge. This generally disappeared before extrafusal contraction was fully gone, but could be restored by post-tetanic potentiation and then, on account of the smaller and more slowly rising extrafusal contraction, sometimes with shorter latency than in the controls before Flaxedil. The ephaptic spike was more sensitive to Flaxedil and less so to post-tetanic potentiation than the physiological response at the foot of the contraction (cf. Lloyd's (1942) results with curare on his ephaptic mass response). However, the arguments against the tension theory and for the differentiation of the early discharge into two components, neither of which depends on tension, are cogent enough without burdening this paper with additional illustrations from the Flaxedil experiments.

*Root division.* The experiments of Hunt & Kuffler (1951) on root division sometimes showed that none of the individual parts gave an ES when several put together produced one, suggesting to us that they studied an ephaptic

spike requiring a great many synchronously activated fibres. 'In some experiments, on the other hand, an early discharge was obtained on stimulation of a whole ventral root and of several of its subdivisions' (p. 310). It is difficult to interpret root records without clearly separating the two kinds of mechanism that are responsible for early spikes and without considering what $\gamma$ activation might do to subdivisions provided with a large number of $\gamma$ fibres as compared with filaments lacking them. To use flexors and apply post-tetanic potentiation is to play for safety.

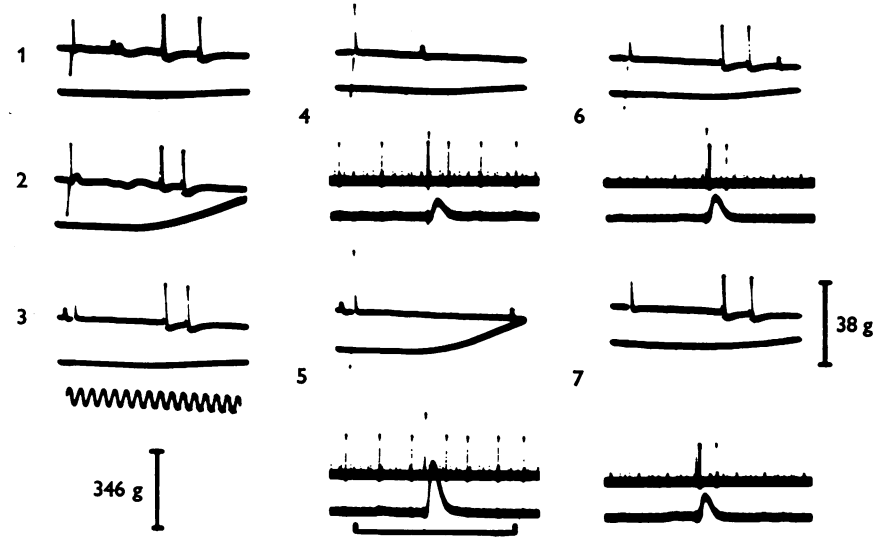


Fig. 8. Spindle afferent from tibialis anterior of anaesthetized animal. Effect of subdivision of ventral root L7. 1, extension 16 mm, weak shock to whole root, 0·1 V; 2, extension 15 mm, shock at 0·13 V, latencies in both cases 7·5 msec; 3, shock at 0·4 V to 1/12 of the root, the other portions without ES, latency now 8·0 msec, extension 13 mm. Note calibration of myograph and the much increased sensitivity used for records 4–7, which refer to stimulation of further subdivisions of 1/36 part of whole root, all at extension 13 mm and shock strength 0·1 V. These are simultaneous records on sweep and stationary spot. 4, this 1/36 part fails to respond with an ES, even after post-tetanic potentiation in 5 which greatly increased the muscle contraction; 6, this 1/36 part gave an ES; 7, same after post-tetanic potentiation. Note that in spite of considerably greater tension developed in 5, the ES only occurred in 6 and 7, latency 8·0 msec. Time 1000 c/s for sweep and 0·5 sec marked for standing spot.

With some patience one may succeed in finding a small fraction of a root such as the 1/12th used in Fig. 8, record 3. This was the only one that had the ES, which was also seen after the whole root was stimulated (1 and 2). This 1/12 part was divided into three roughly equal parts, each of which therefore represented about 1/36 part of the original root (L7). Two of these 1/36 parts failed to produce an ES, despite potentiation at maximal strength, after which they were tested at all strengths short of values producing stimulus spread. In

the figure the one 1/36 part has been picked out that gave a contraction (records 4 and 5). The third 1/36 part produced an ES (6 and 7) whose latent period, after post-tetanic potentiation (7) was reduced to 8·0 msec. The important point of this experiment is that the potentiated contraction not only was very small but considerably smaller than in the 1/36 part that failed to produce an ES. Therefore tension as such cannot explain the result. The ES is due to the action of a specific fibre or set of fibres.

The most general result of root division in flexors was that increase of stimulus strength or adding up thin filaments can reduce the latent period of the ES (after those parts of the root had been set aside which failed to produce an ES). With a suitable fraction of the root this result could be obtained without significant increase of contraction, whereas in other cases contraction did increase. Such experiments only show that number of fibres activated plays a part and hence support the notion of some overlap of the fibres concerned. If increase of tension is singled out as the only possible explanation of the effect of increased shock strength or addition of fibres from other filaments, this conclusion is open to the criticism that the connexion may be fortuitous (cf. Fig. 8), due merely to the fact that increase of number of efferent fibres is often equivalent to an increase of tension. For this reason it is held that the clarity which is supposed to be the reward of experiments on root division often is more apparent than real. It should be noted that the first and major part of Hunt & Kuffler's (1951) experiments on root division deals with spikes much later than the early discharge.

*Conduction velocity.* Figures 1 and 5 are instances of many experiments which have shown that the physiological early discharge, our second component of ES, can be obtained at shock strengths a good deal below those necessary for maximal extrafusal contraction, which in turn is a good deal below those necessary for the activation of $\gamma$ fibres (Leksell, 1945; Kuffler *et al.* 1951); not to mention that the large majority of $\gamma$ fibres require to be repetitively stimulated in order to exert any effect at all on the muscle spindles as studied in the afferent discharge, particularly when the muscle is slack (Hunt & Kuffler, 1951; Kuffler *et al.* 1951).

It would seem a simple matter to supplement these observations, convincing in themselves though they be, with direct measurements of the increase of latency when the shock is shifted from nerve to root. However, the previous sections have shown that the latent period of the ES is influenced by extension, is often delayed by the development of tension and may be shortened by intrafusal activation, etc., factors which our results show to be differently balanced when root and nerve stimulation are compared. The error introduced by measuring conduction velocity across the loop is of the order of 1 msec but, if at the available conduction distance the velocity be 100 m/sec, an error of 1 msec lengthening of latency will reduce the measured conduction velocity to

60 m/sec! One of our best experiments is reproduced in Fig. 9, and is explained in its legend. From the root the average latency of a large number of such records was 8·0 msec, from the nerve 6·8 msec. The difference of 1·2 msec corresponds to a conduction velocity of 125 m/sec. This value seems too large, but no possible error could bring it down to $\gamma$ conduction velocity.

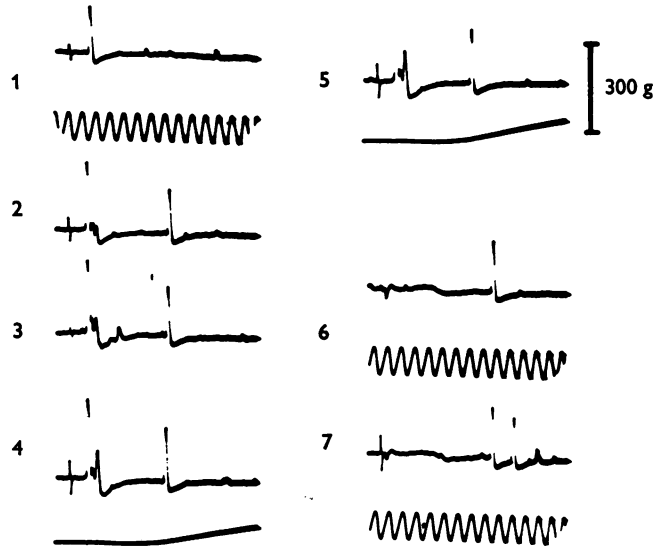


Fig. 9. Spindle afferent from tibialis anterior in anaesthetized animal. Comparison of root and nerve stimulation to estimate conduction velocity. *Nerve.* 1, muscle slack, shock at 0·12 V; 2 ,same at 0·2 V; 3, same at 1·0 V; 4, 8 mm extension, shock at 0·25 V; 5, same at 11 mm extension. *Root.* 6, shock to L7 at 0·32 V, muscle slack; 7, same at 8 mm extension. Difference in latency, root − nerve, is 7·98 − 6·78 = 1·2 msec, based on measurements from a large number of similar sweeps. Conduction velocity 125 m/sec.

In another set of three experiments the conditions were improved by using two electrodes 5 cm apart on the nerve and taking alternate readings, in one of them alternating rapidly by means of a switch. These values averaged 101 m/sec, with 143 and 73 m/sec as the extremes. All these observations refer to flexors. The ephaptic spike which can be measured in slack muscle was found to be activated by fibres in the upper range of $\alpha$ conduction velocities (cf. legend of Fig. 3).

## DISCUSSION

Lloyd's (1942) work and some observations of Leksell (1945) clearly showed that the ephaptic theory cannot be neglected; indeed, ephaptic spikes even turn up in ventral roots. By the frequency test with slack muscle, supplemented with measurements of loop time, we have proceeded to eliminate early spikes which can be explained in this manner, and so they can also be dismissed

from this discussion. As far as the present work is concerned, these ephaptic spikes may well contain an unknown fraction of 'physiological' spikes which our criteria fail to differentiate. Also the border line between the two components of early discharge need not always be readily defined. In the flexors the nerve fibres enter fanwise over a considerable distance, while in the two extensors their mode of entrance suggests precise foci, as were proved to exist for the soleus by Eccles & O'Connor (1939), who for this very reason used soleus in their study of the muscle action potential. This may provide one explanation of the differences between flexors and extensors, possibly not the only one.

The remaining theories are (i) the tension theory of Hunt & Kuffler (1951), (ii) the innervation theory and (iii) what one might call an organization theory. The tension theory of Hunt & Kuffler was developed to account for the spikes occurring before visible contraction, which were dismissed for the reasons mentioned above. This does not exclude another tension theory to explain our results with the 'physiological' second component in the early discharge. However, section by section the tension theory has been put to a test and always failed to explain our findings. It may well hold for exceptional spindles but for these no theory is required.

The organization theory is an attempt to rescue the tension theory by the additional assumption that a few extrafusal fibres are specifically connected to spindles in such a manner as to pull upon them when contracting. It thereby avoids the difficulty that, as a rule, tension is of little importance compared with extension and intrafusal state of tonic contraction. The theory postulates a specific anatomical mechanism of organization to explain what otherwise would be a deficiency on a pathological scale (40 % in flexors) in a measuring instrument designed to maintain a great measure of independence from extrafusal contraction. All our results, however, go to show (cf. Granit, Pompeiano & Waltman, 1959) that the second component of the early discharge is part of a physiological mechanism. The organization theory would require further specifications to account for the fact that these spindles actually are unloaded, just as those lacking the early discharge, when tension reaches full development. There is no definite evidence in favour of the organization theory. In this regard the innervation theory has the advantage of anatomical support (cf. p. 399).

Our previous experiments on stimulation from central stations such as the medullary pyramids and Deiters's nucleus showed that the mechanism of fast activation gave spikes arising with the muscle contraction. Most of them were too late to be ephaptic. Besides, central stimulation produces a burst spread out further in time by the large conduction distance, and so will not regularly be able to create the electrical fields that synchronous shocks to the nerve set up. The contractions from the pyramids, as a rule, were very small.

Nevertheless, since in no instance could fast activation be obtained from places that did not also potentially excite large extrafusal fibres, it is possible that the fast fibres for the spindles are extrafusal ones which send a tapering branch to a spindle. This alternative is suggested by some anatomical observations of Cooper & Daniel (1956).

Cooper & Daniel (1956) have also suggested other types of nerve ending on intrafusal muscle which by degeneration experiments (Boyd, 1959) have been shown to be motor. There are thus two types of motor endings on intrafusal muscle. Pascoe (1958) has described a spindle efferent, stimulated by violent reflex movements but stretch-insensitive, whose conduction velocity is not given in the paper but which (personal communication) appears to exceed that of $\gamma$ fibres. At the moment it is impossible to say more than that these observations will eventually have to be evaluated relative to the findings in this paper.

Finally Boyd (1959) has differentiated slow and twitch-like contractions in spindles by direct observations under the microscope on tenuissimus in cats. The effects we have described do behave as if they were elicited by brief intrafusal twitches. The slow contraction suggests the slow mechanism of $\gamma$ activation. Adding all these observations to the histological demonstrations of spindle motor fibres of two calibres (p. 399) it is clear that our highly specific effects are best explained by an innervation theory. This implies that a considerable number of spindles possess $\alpha$ innervation, often in combination with $\gamma$ innervation. The functional significance of fast spindle activation has been discussed in our previous paper (Granit, Pompeiano & Waltman, 1959) and, within limits, is independent of whether the $\alpha$ fibres concerned go to spindles alone, to extra- and intrafusal muscle, or only to nearby extrafusal fibres to which spindles are inserted so as to receive a brief twitch while the full force of the extrafusal contraction later is neutralized.

SUMMARY

1. Muscle spindle afferents, coming from ankle flexors or extensors (cat) and isolated in thin dorsal root filaments, discharge early in contraction to a shock to the muscle nerve or the severed ventral root.

2. This early discharge was found to consist of two components, one appearing in advance of visible contraction, the other one at the foot of the contraction. The properties of the two components have been analysed in 168 spindles and 19 tendon organs.

3. The first component requires a sufficiently strong shock to the muscle nerve, but neither tension nor extension of the muscle. It follows frequencies of the order of 130–200/ sec in slack muscle and originates at the time of the muscle action potential, similarly in spindles and tendon organs. It is held to be an ephaptic stimulation of sensory terminals or nerve fibres.

4. The second group of early spikes at the foot of the contraction is conveniently studied in flexors because in them the ephaptic component is rare. About 40% of the flexor spindles discharge such spikes. In extensors they are preceded by ephaptic spikes.

5. These spikes are activated by fibres conducting at $\alpha$ velocity and do not depend upon tension but upon the phasic sensitivity of the spindles as determined by $\gamma$ bias or equivalent extension of the equatorial region by pull on the muscle.

6. Flaxedil removes them before it removes $\gamma$ activity and even in advance of the last remnant of extrafusal contraction, but re-activation follows after tetani to the muscle nerve.

7. The second group of early spikes is identified with the similarly located early spikes elicitable from various central stations (Granit, Pompeiano & Waltman, 1959).

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