J. Physiol. (1958) 143, 195–212

SPINAL REFLEX REGULATION OF FUSIMOTOR NEURONES

By C. C. HUNT and A. S. PAINTAL

From the Department of Physiology, Albert Einstein College of Medicine, New York, and the Department of Physiology, University of Utah College of Medicine, Salt Lake City

(*Received* 26 *March* 1958)

In mammals, muscle fibres within the muscle spindles receive a separate motor innervation from ventral root fibres of small diameter (Leksell, 1945; Kuffler, Hunt & Quilliam, 1951). Discharge in these efferent fibres causes excitation of spindle receptors, an effect synergic with that produced by muscle stretch. The muscle spindle is thus subject to reflex control by variations in number and frequency of impulses in its motor nerve fibres. The motor fibres to the spindle have been designated γ efferents by Leksell (1945) and small motor fibres by Kuffler *et al.* (1951). Both designations have possible ambiguity since motor fibres of this diameter to other than the hind limb muscles of the cat may have other functions. We therefore propose the term fusimotor with reference to neurones that are motor to the muscle spindle. This has functional connotation and bears similarity to such well established names as vasomotor and sudomotor. The designation motor neurone (or motoneurone) will be used for neurones with large diameter axons that are motor to extrafusal muscle fibres.

Following the demonstration of the function of fusimotor neurones, studies were made of the reflex activity of such neurones (Hunt, 1951; Kobayashi, Oshima & Tasaki, 1952). Several characteristic features were found. Many fusimotor neurones, in the decapitate or decerebrate cat, showed a background repetitive discharge in the absence of added stimulation. In a number of reflex patterns, such as the flexor and crossed extensor reflexes and tonic neck reflexes, excitation or inhibition of fusimotor neurones accompanied similar effects on motoneurones. In many reflex acts the threshold for excitation of some fusimotor neurones was lower than that required to cause excitation of motoneurones. Subsequently, the reflex activity of fusimotor neurones was investigated by Granit & Kaada (1952), Eldred, Granit & Merton (1953), and Granit, Job & Kaada (1952), who found that stimulation of various supraspinal structures excited or inhibited fusimotor activity. As had been found

in spinal fusimotor reflexes on stimulation of peripheral nerve, the last workers also noted that the threshold for eliciting changes in fusimotor activity was often lower than that required to cause changes in motoneurone discharge. In addition, Eldred & Hagbarth (1954) provided important information on the effect of cutaneous stimulation on fusimotor activity. Most of these studies used the changes in frequency of discharge in spindle afferent fibres as an index of fusimotor discharge.

Little information is available on the spinal segmental organization and properties of fusimotor neurones that determine their reflex behaviour to influences from primary afferent fibres or from supraspinal activity. The present study is concerned with this problem. Information will be presented to show that the reflex activation of fusimotor neurones differs in several respects from that of motoneurones, owing to differences in intraspinal connexions and in the properties of the neurones themselves.

METHODS

Spinal cats were used throughout. Under full ether anaesthesia both common carotid arteries were ligated, the trachea cannulated and the vertebral arteries permanently clamped. Anaemic destruction of the brain was shown to be complete by total cessation of respiratory movements, by dilatation of the pupils and obvious arrest of circulation to the skin and mucosa of the head. Following application of the vertebral clamp the spinal cord was divided at the atlanto-occipital membrane and respiration was artificially maintained. Ether was then discontinued. The lumbo-sacral cord was exposed by laminectomy and appropriate limb nerves were dissected. Stimulating electrodes were usually placed on the central ends of cut peripheral nerves, although in some cases they were placed on intact nerves so that the effects of natural stimulation as well as of nerve volleys could be studied. Exposed tissues were covered with pools of paraffin oil, usually equilibrated with 95% O_2 and 5% CO_2.

Two methods were used to record activity in efferent nerve fibres. The first was to place naturally occurring small filaments of ventral root (L7 or S1), distally cut, on recording electrodes. One electrode was placed on the cut end, the other several centimetres centrally, if possible, so as to avoid recording in the length of nerve occupied by demarcation current. In these circumstances relative amplitude of individual spike potentials served to differentiate large and small fibres (Hunt, 1951). The second method was to record from filaments of a muscle nerve dissected free after removal of the sheath. One ventral root was left intact (S1 or L7) and gently freed from surrounding structures so that a length could be looped over a pair of stimulating electrodes in the paraffin oil. Fibres emerging via this root to a filament under study could be identified as to conduction velocity by observing the stimulus-response latency of impulses evoked by a ventral root stimulus. The remainder of the ventral root outflow was divided. This restricted stimulation of the ventral root outflow reduced the amount of dissection required to isolate a single-fibre response in a muscle nerve filament. In many instances it was possible to establish the identity of an individual fusimotor fibre, in the presence of several other motor fibres in a filament. This was done by observing that an impulse failed to be evoked by ventral root stimulation when the fibre was refractory following a reflexly initiated impulse. The following muscle nerves have been examined: flexor digitorum longus, extensor digitorum longus, tibialis anterior, gastrocnemius medialis, biceps semitendinosus and quadriceps.

RESULTS

General features of fusimotor reflex response to nerve volleys

One of the striking differences between the reflex-response of motoneurones and of fusimotor neurones to single afferent volleys is the more frequent occurrence of repetitive discharge in the latter. The response of a fusimotor

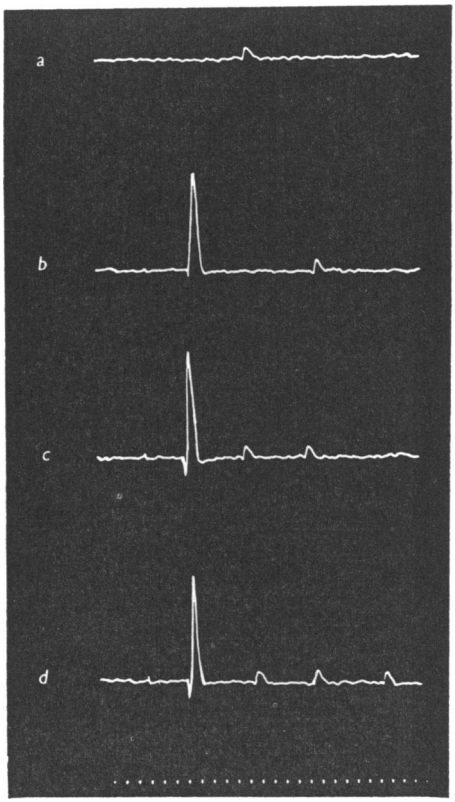

Fig. 1. Discharge in a fusimotor neurone recorded in a ventral root filament. *a*, base line; *b*, *c*, *d*, stimulation of flexor hamstring nerve at increasing strength. Note monosynaptic reflex response of motoneurone that precedes response of fusimotor neurone. Time marker 2000 c/s; large spike retouched.

neurone to single volleys in skin or muscle nerves was often repetitive, consisting of up to 3–4 impulses. A characteristic response is shown in Fig. 1, the discharge being recorded in a ventral root filament, the volley originating in the nerve to biceps posterior and semitendinosus. The large amplitude impulse is the monosynaptic reflex response of a biceps-semitendinosus motoneurone. The number of responses was dependent on the stimulus strength to the

afferent nerve; reduction in the number of afferent fibres stimulated caused the response to be reduced to one. The duration and frequency of discharge varied among different fusimotor neurones and with the particular afferent nerve stimulated (see below). One type of response to afferent volleys in skin or muscle nerves that was frequently encountered consisted of 3–4 impulses with a peak frequency of 300–500/sec. These reflex responses are clearly of higher frequency than those of motor neurones. In this feature the fusimotor response bears greater similarity to the discharge of internuncial neurones (Woodbury & Patton, 1952). A different type of fusimotor reflex response was a more protracted, lower frequency train of impulses produced by single afferent volleys.

In the presence of background discharge the response to nerve volleys was altered in that a 'spontaneous' impulse occurring at certain intervals before the first reflex discharge could delay the entire series of responses. The evoked responses were 're-set' in time by the spontaneous impulse, the intervals between evoked discharges remaining essentially the same. The occurrence of a spontaneous discharge within several milliseconds of the expected time of the first evoked discharge caused the latter to drop out, whereupon the initial discharge appeared earlier than the usual second discharge and the discharge train was reduced by one impulse.

The spontaneous discharge as well as other factors cause the latency of fusimotor response to show considerable variation. Typically the first impulse in a response series showed less variation in latency than subsequent impulses, the last of a train of evoked impulses usually being the most labile. Thalamic neurones exhibit similar variation in latency (Rose & Mountcastle, 1954). The latency variation is considerably greater than that found in monosynaptic reflex response of motoneurones. The central latency of fusimotor response to muscle nerve volleys is greater than the latency of monosynaptic reflex response of motoneurones. On the other hand, the earliest of a series of evoked fusimotor reflex responses usually preceded the polysynaptic reflex response of motoneurones to skin or muscle nerve volleys. Typical latencies for the earliest fusimotor response recorded in ventral root filaments to afferent nerve volleys were as follows (msec): sural 4·6, triceps surae 4·5–5·0, deep peroneal 5·6 (afferent conduction distances about 15 cm), and biceps semitendinosus 4–4·5 (afferent conduction of about 12 cm).

In fusimotor neurones showing spontaneous discharge a reflex response was followed by a period of silence or of diminished frequency of the background discharge. This pause often lasted for 20 msec or longer and its explanation is not obvious, for the period of silence of spontaneous discharge outlasts the recovery period of most fusimotor neurones and such neurones are capable of response to a repetition of the excitatory afferent volley during this period.

Central delay. Estimates of central delay were made in six fusimotor

neurones by measuring the latency of response to dorsal root volleys. In three of these reflex discharges were recorded in ventral root filaments; the values obtained for central delay were respectively, 2·95, 2·7 and 2·6 msec. Since the conduction velocities of these fibres were not measurable, no correction has been made for efferent conduction time. However, this may be estimated to be about 0·3–0·5 msec in the case of a modal fusimotor fibre with a diameter of 6μ (see Kuffler *et al.* 1951) and an efferent conduction distance of 1–1·5 cm.

In the other three experiments reflex discharges in fusimotor fibres were recorded in filaments of medial gastrocnemius nerve, and since the conduction velocities of these fibres were determined, the efferent conduction times were deducted from the stimulus–impulse latencies to yield the following values for central delay: 2·46, 2·03 and 2·04 msec. In both groups of experiments the afferent conduction distance in the dorsal root was 1 cm or less, so that a negligible fraction of the measured delay is due to the time taken for the afferent volleys to reach the cord.

The above experiments indicate that the central delays for the earliest reflex response of fusimotor neurones are in excess of 2 msec. By comparison, the latency of the dorsal-to-ventral root monosynaptic reflex response of motoneurones is about 1·0–1·5 msec (Eccles & Pritchard, 1937; Renshaw, 1940; Lloyd, 1943). There is, therefore, no evidence that fusimotor neurones receive monosynaptic excitatory connexions from primary afferent fibres.

Recovery following conduction of an antidromic impulse

Reflex response of a fusimotor neurone was recorded in a cut filament of ventral root of sufficient length for a pair of stimulating electrodes to be applied central to the recording electrodes on the same filament. Reflex response to a nerve volley of constant amplitude could be tested at various intervals after a stimulus to the ventral root filament. The latter initiated antidromic impulses in the fusimotor neurone as well as in the few other fibres which were contained in the filament. An alternative method was to record the reflex response of an individual fusimotor neurone isolated in a muscle nerve and to stimulate an entire intact ventral root. This procedure had the disadvantage of causing initiation of antidromic impulses in a large number of other ventral root fibres. However, antidromic impulses in other motor fibres were found to have little effect on the fusimotor neurone under study (see below).

The recovery of excitability was examined by obtaining an estimate of the firing probability of a fusimotor neurone to stimulation of an afferent nerve at constant strength and at a constant rate of 1/2 sec. In this connexion, the term firing index has been used to designate the $\frac{\text{number of responses}}{\text{number of trials}} \times 100$ (Lloyd & McIntyre, 1955). Usually 25 or 50 trials were used. This method of testing can reveal excitability changes only when the firing index is between

zero and 100. However, the very rapid recovery of a fusimotor neurone following conduction of an antidromic impulse can be easily demonstrated, as in the unit with a control firing index of 100 in Fig. 2. This recovery curve may be compared with the recovery of a population of motoneurones following conduction of antidromic impulses (Lloyd, 1951) and with the recovery of an individual motoneurone with a firing index of 100 shown in Fig. 2.

A total of six fusimotor neurones have been studied for recovery following conduction of an antidromic impulse; their recovery curves are shown in Fig. 3. Five of these had control firing indices of 100; four recovered to their control firing index within 10 msec after the antidromic impulse reached the cord. In one unit (□) the recovery curve is incomplete but it is evident that its recovery was slower. Another fusimotor neurone with a control firing index of 79 (●) had a recovery nearly complete by 30 msec. Since the responses in some fusimotor neurones were recorded in muscle nerves with significant time taken for efferent conduction and since the latency of response to afferent volleys was appreciable in comparison with the recovery time of the fusimotor neurones, the intervals in Figs. 2 and 3 are calculated as the time between arrival of the antidromic impulse in the fusimotor neurone at the cord and the time when the neurone normally discharged to the test volley.

The recovery of fusimotor neurones appears to be considerably more rapid than that of motoneurones. In populations of motoneurones recovery of excitability is not complete until about 120 msec after conduction of antidromic impulses (Lloyd, 1951). The absence of such prolonged subnormality in fusimotor neurones is probably related to their ability to discharge at considerably higher frequencies than motoneurones and to their greater tendency to repetitive response to single afferent volleys.

In several experiments in which fusimotor response was recorded from ventral root filaments, antidromic volleys in the remainder of the root had no detectable effect on the excitability of the fusimotor neurones. Thus the recurrent collaterals of motoneurones or the current flows coincident to the antidromic volleys appear to have little influence on fusimotor neurones, confirming the findings of Granit, Pascoe & Steg (1957).

Background activity

The occurrence of repetitive discharge in the absence of added stimulation is characteristic of some fusimotor neurones. Indeed, this feature has been used to detect fusimotor neurones in filaments of ventral root (Hunt, 1951). In the present study, in addition to active fibres, it was possible to isolate fusimotor fibres in which there was no background discharge by recording impulses initiated by stimulation of intact ventral root in muscle nerve filaments (see Methods). By serial examination of a succession of fusimotor fibres it would be possible to obtain a representative sample and to determine the

proportion of such fibres that exhibit background or 'spontaneous' discharge. This was not done systematically, as interest centred on fibres that were readily influenced by reflex actions and these were usually fibres showing a background discharge. However, the majority of fusimotor neurones isolated

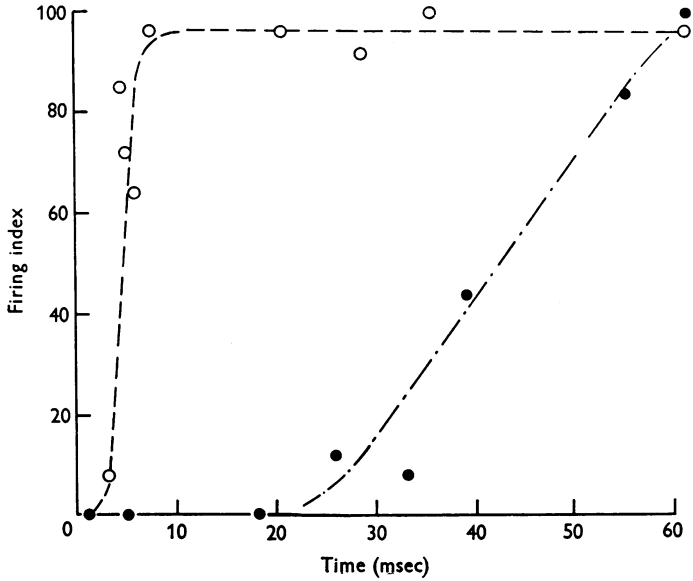

Fig. 2. Recovery following conduction of an antidromic impulse in a fusimotor neurone (○) and in a motoneurone (●). Abscissa, interval between arrival of antidromic impulse at cord and time of normal reflex response.

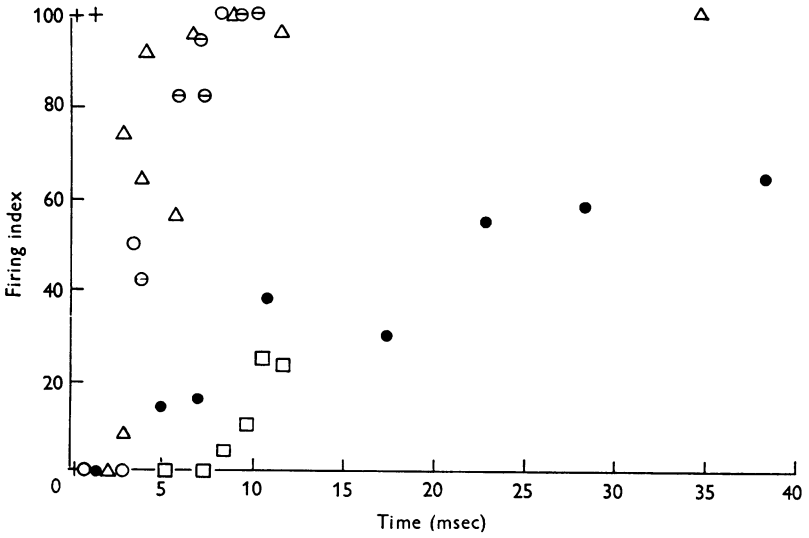

Fig. 3. Recovery following conduction of antidromic impulse in six fusimotor neurones. Abscissa as in Fig. 2.

did not display background discharge. Thus, 47 out of 118 units isolated, or 40%, showed background discharge. A reasonable estimate of the fraction of fusimotor neurones having background discharge in the acute spinal cat would be about $\frac{1}{4}$–$\frac{1}{3}$, with a more representative sample.

Interval between impulses is typically irregular in background discharge of fusimotor fibres. The frequency of spontaneous discharge ranged from a few to 60 impulses/sec, the usual range being 10–40/sec. The typical pattern of spontaneous discharge is seen in Figs. 4 and 5.

The term 'background discharge' should not be taken to mean that fusimotor neurones fire repetitively in the absence of an inflow of excitatory afferent impulses (see Hunt, 1951). As will be seen later, afferent discharge from cutaneous sources plays a major role in the initiation of fusimotor discharge. The conditions of the experiment must contribute to the background afferent inflow and hence to the production of background discharge in fusimotor fibres. Among the factors of importance in this regard are the presence of skin flaps about the dissected areas, pins used for fixation and contact of skin with various structures.

Background discharge in fusimotor neurones is abolished by interruption of afferent inflow to the spinal cord (Hunt, 1951). Furthermore, the present study indicates that reflex excitation of fusimotor neurones by impulses in primary afferent fibres occurs through polysynaptic pathways. It might be expected, therefore, that an agent which blocked polysynaptic reflex effects would reduce or abolish background discharge of fusimotor neurones. The drug myanesin has been used to test this premise. Fig. 4 shows the discharge of a fusimotor neurone in the absence of added stimulation and in response to sural nerve volleys before and after two injections of myanesin 20 mg/kg intravenously. Myanesin reduced the background discharge as well as the response to sural volleys, the duration of this effect being about 20 min. These findings alone do not indicate the mechanism of initiation of background discharge in fusimotor neurones, but are consistent with the fact that the background discharge results from excitatory effects mediated polysynaptically.

Responses of fusimotor neurones supplying particular muscles

The general features of the reflex response of fusimotor neurones have been considered above. That which follows is concerned with the reflex activation of fusimotor discharge to specific muscles. Isolation of fusimotor fibres has been accomplished in the nerves to a number of hind-limb muscles, representing flexors and extensors of the knee, ankle and toes. Responses to nerve volleys from various afferent sources and to natural stimuli have been sought in an attempt to explore further the functional organization of fusimotor neurones and their role in spinal reflex actions.

A total of 143 fusimotor neurones have been studied in the following muscle nerves: gastrocnemius medialis (31), tibialis anterior (21), extensor digitorum longus (13), flexor digitorum longus (28), semitendinosus (35) and quadriceps (5). Conduction velocities were determined in 119 of these.

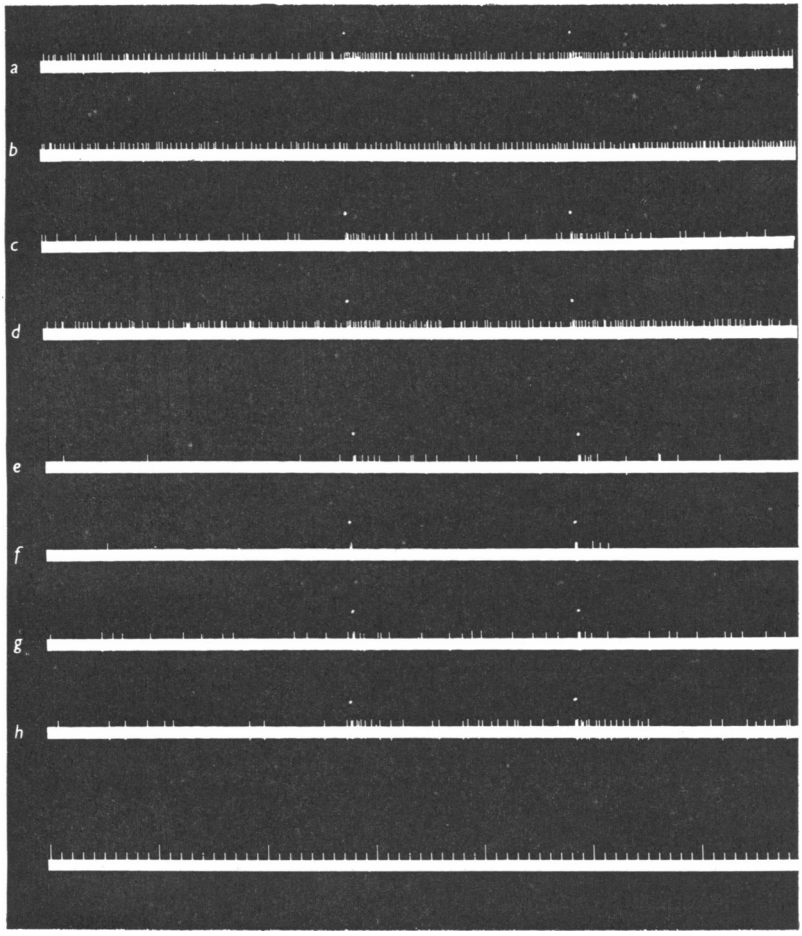

Fig. 4. The effect of myanesin on the discharge of a fusimotor neurone to medial gastrocnemius. *a*, shows base-line discharge and reflex response to sural volleys, position of stimuli indicated by dots; at time zero 20 mg/kg of myanesin injected intravenously. Record *b* begins 11·2 sec, *c* 58·3 sec, and *d* 2 min later. After 2 min 36 sec, another 20 mg/kg of myanesin was injected. Record *e* at 3 min 16 sec, *f* at 3 min 45 sec, *g* at 6 min 45 sec, and *h* at 23 min 45 sec. Time marker 1 and 0·1 sec. There is a transient increase in base-line discharge shortly after injection (*b*), followed by a decrease in background discharge as well as a decrease in response to sural volleys (*c–h*).

Responses to nerve volleys

Fusimotor neurones to the muscles specified were examined for their reflex responses to volleys in several peripheral nerves, usually sural, plantar, deep peroneal, superficial peroneal, flexor hamstring, and triceps surae. Stimuli were of a strength estimated to exceed threshold for all myelinated fibres. The principal effects are shown in Table 1. Those cases in which nerve volleys resulted in discharge of fusimotor neurones are shown as +; inhibition of spontaneous discharge is indicated by −; lack of a demonstrable effect is indicated by ○. The effects are often mixed; for example, sural volleys caused reflex discharge in some fusimotor neurones of flexor longus digitorum; in others inhibition was evident. Even in a particular fusimotor neurone both excitation and inhibition could be seen. Thus gastrocnemius medialis fusimotor

TABLE 1. Responses of fusimotor neurones to nerve volleys

	Muscle				
Nerve volley	Medial gastrocnemius	Tibialis ant.	Ext. dig. longus	Flex. dig. longus	Semi-tendinosus
Sural	+	+	− +	− +	+
Plantar	−	+	− +	− +	
Triceps surae	○	+	− +		○ +
Deep peroneal	+ −			− +	+
Sup. peroneal		+	+	− +	
Biceps-semitend.	+	○			

+ excitation; − inhibition; ○ no effect.

neurones showing background discharge often showed one impulse evoked by a plantar nerve volley following which the spontaneous discharge was inhibited for a period. From the response of fusimotor neurones to volleys in various peripheral nerves (Table 1) no meaningful pattern is evident. This is probably due to the mixed reflex effects associated with activation of functionally dissimilar afferent fibres. Further work is needed to relate the reflex effects to the diameter group of afferent fibres stimulated.

A number of the fusimotor neurones studied did not discharge in response to any of the nerve volleys tested; such units generally exhibited no background discharge and no response to natural stimulation. The responses of fusimotor neurones varied over a considerable range, indicating considerable differences in reflex excitability.

In general, cutaneous nerve volleys are more effective in influencing fusimotor neurones than are muscle nerve volleys. The reflex effects produced by afferent muscle nerve volleys of graded size were studied to a limited extent, the incoming volley size being measured by a volume lead at the root-cord junction. Afferent muscle nerve volleys producing less than 50% of maximal group I spike potential amplitude at the root-cord junction failed to produce reflex discharge of fusimotor neurones. However, volleys slightly larger than

this, particularly from flexor-hamstring nerves, evoked fusimotor reflex response. In view of the possibility of overlap in diameter distribution of functionally homogeneous afferent fibre groups, these results fail to provide clear evidence of excitatory effects on fusimotor neurones by impulses in large afferent fibres from muscle spindles or tendon organs, although such effects are not ruled out.

Responses to natural stimuli

The responses have been studied of fusimotor neurones from various muscles to natural stimulation of various parts of the hind limbs. Certain areas were routinely tested, in particular the foot pads, and the skin area innervated by the sural nerve of the ipsilateral and contralateral hind limbs. Touch or light or heavy pressure was employed in an attempt to elicit reflex effects in a fusimotor neurone under study. No particular attempt was made to determine the modality of sensation concerned with the production of a reflex effect nor was the area of skin involved carefully mapped. Rather, characteristic effects were sought which were produced on fusimotor neurones to specific muscles representing flexors and extensors of knee, ankle and toes by stimulation of a number of peripheral sites. The following muscles have been studied:

Quadriceps. Owing to the anatomical position of this muscle nerve, identification of fusimotor neurones was based on relative spike potential amplitude when recording from filaments of quadriceps nerve, and laminectomy was not performed. Fusimotor neurones to this muscle that exhibited spontaneous discharge were inhibited by touching or squeezing the foot pad on the area of sural innervation of the ipsilateral limb. Stimulation of similar areas of the contralateral hind limb produced an increase in discharge or initiated discharge in quiescent neurones. These reflex actions can be interpreted as concomitants of the flexor and crossed extensor reflex patterns (Hunt, 1951). It was also noted that stimulation of the skin overlying quadriceps increased discharge in fusimotor neurones to this muscle, whereas stimulation of the skin over the flexor hamstring muscles inhibited background discharge. The latter findings are similar to those reported by Eldred & Hagbarth (1954).

Semitendinosus. A total of twenty-five fusimotor fibres were isolated, ranging in conduction velocities from 16 to 38·5 m/sec. Of these five showed background discharge. Touching or squeezing the ipsilateral paw increased the discharge frequency in three of the fibres with background discharge and decreased it in one; in one previously quiescent unit discharge was initiated. Similar stimulation of the ipsilateral sural area caused inhibition in two units, excitation in one. Stimuli to the contralateral paw as well as to the contralateral sural area caused inhibition. These effects, as in the case of quadriceps, are in the direction that would accompany flexor and crossed extensor reflex patterns, with the exception of inhibition produced by ipsilateral sural area

stimulation. In one unit it was observed that touching the skin over quadriceps inhibited the background discharge, touching the skin over the flexor hamstring muscles increased discharge (cf. Eldred & Hagbarth, 1954).

Gastrocnemius medialis. A total of thirty-one fusimotor neurones were examined, fourteen showing background discharge. Conduction velocities ranged from 21 to 48 m/sec. In most of the fibres tested touching or squeezing the ipsilateral paw inhibited background discharge. Stimulation of the sural area uniformly initiated or increased discharge in those units affected. Stimulation of the contralateral paw caused an increase in discharge in five out of seven units tested; one unit showed inhibition and one was not influenced. The principal effects of ipsilateral and contralateral paw stimulation are those that might be expected as components of flexor and crossed extensor reflexes.

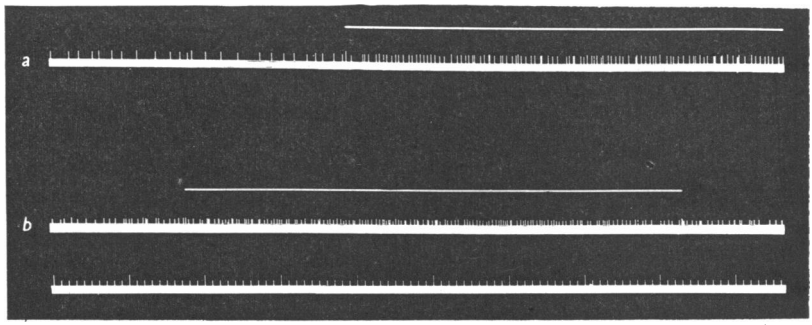

Fig. 5. Response of a fusimotor neurone to gastrocnemius medialis. *a*, base-line discharge and response to squeezing tendo Achillis; *b*, base-line discharge and response to stretch of tendo Achillis. Time marker, 1 and 0·1 sec; period of stimulation indicated by signals.

The facilitation produced by ipsilateral sural area stimulation is similar to the effect noted by Eldred & Hagbarth (1954), although inadvertent excitation of receptors in the tendo Achillis may have played a contributory role to the effects noted (see below). The predominantly excitatory effect of activation of cutaneous receptors in the sural region is in keeping with the excitatory effect of sural nerve volleys on gastrocnemius medialis fusimotor neurones (see above).

In several experiments on gastrocnemius medialis fusimotor neurones tension was applied to the tendo Achillis in an attempt to determine the reflex effect of activity from receptors in the muscle. It was found that merely grasping the tendo Achillis between the fingers caused a striking increase in reflex discharge of gastrocnemius medialis fusimotor neurones. Similar excitation sometimes occurred on pulling the tendon (Fig. 5). In some cases, however, pinching or squeezing the tendon caused marked excitation, whereas the application of moderately high degrees of tension to the tendon was

ineffective. The reflex fusimotor excitation was maintained as long as pressure was applied to the tendon, indicating that the receptors concerned were slowly adapting. Complete section of the triceps surae nerve did not alter this reflex effect, whereas subsequent section of the tibial nerve abolished it. The afferent fibres responsible for the reflex excitation, therefore, do not run in the nerve to the muscle but course by some other route to reach the tibial nerve. The nature of the receptors responsible for this fusimotor excitation is not known. They may be located in the tendo Achillis or in the surrounding fascia. The fact that the reflex effect persists when the skin is widely separated from the tendon and also after section of the sural nerve innervating this area shows that these receptors are not of cutaneous origin.

Tibialis anterior. Background discharge was present in nine of the twenty-one fusimotor neurones isolated to this muscle. Conduction velocities were between 16 and 43 m/sec. In seven out of nineteen units tested, touching or squeezing the ipsilateral paw increased the discharge; in twelve units there was no effect. Natural stimulation of the sural area of skin to the same hind limb was without detectable effect in all units examined, although sural volleys caused discharge in a number of units. In the contralateral limb, stimulation of the sural area caused increase in discharge in two out of ten neurones tested, and touching or squeezing the paw inhibited four and excited two out of nineteen units examined. The occasional excitatory effect of the contralateral sural and paw areas may represent a crossed flexor reflex pattern (Perl, 1957).

Flexor longus digitorum. Twenty-eight fusimotor neurones were isolated to this muscle, of which eleven showed background discharge. Conduction velocities ranged from 26 to 49 m/sec. Of twenty-two units examined eight were inhibited by touching or squeezing the ipsilateral foot, one was facilitated. Stimulation of the skin in the ipsilateral sural area caused inhibition in four units, excitation in four. Stimulation of the contralateral foot pad and sural area uniformly caused inhibition in those neurones showing a demonstrable effect. The predominant effect of natural stimuli to the ipsilateral foot pad was one of inhibition; frequently the base-line discharge was completely arrested merely by light touch to the pad.

Extensor longus digitorum. A total of thirteen fusimotor neurones were isolated with conduction velocities ranging between 21 and 45 m/sec. Of these ten showed background discharge. The principal action produced by touch or squeezing of the ipsilateral foot pad was one of inhibition (eight out of twelve units being inhibited and one excited). Stimulation of the ipsilateral sural area caused inhibition in three and excitation in one out of five units examined. Contralateral foot pad and sural area stimulation always produced excitation in those neurones showing a demonstrable effect. Since this muscle is classed as a physiological flexor, the principal reflex effects produced by natural stimuli to the ipsilateral and contralateral paw and sural areas are in a direction

opposite to that expected in flexor and crossed extensor reflexes. It is surprising to find that tactile stimulation of the ipsilateral foot pad causes a striking inhibition of fusimotor neurones to extensor longus and excitation of those to tibialis anterior, since these muscles are synergists linked by monosynaptic excitatory connexions (LaPorte & Lloyd, 1952). The fact that both flexor and extensor longi digitorum are inhibited by touching the ipsi-lateral foot pad may signify that both extensor and flexor of the digits are inhibited when the foot makes contact with the ground during walking. However, the behaviour of motoneurones to these muscles during comparable natural stimulation is not yet known.

Responses to muscle stretch

In a previous communication (Hunt, 1951) discharge in certain fusimotor neurones was found to be inhibited by muscle stretch. Fusimotor discharge, recorded in ventral root filaments, was inhibited on stretch of gastrocnemius in spinal preparations. In this case the peripheral destination of the fusimotor neurones was not known. Inhibition of fusimotor discharge was also detected in decerebrate preparations, when recording efferent activity in a small muscle nerve branch, on stretch of the same muscle (cf. Eldred *et al.* 1953). At that time no particular distinction was made between the inhibitory effect in spinal or decrebrate preparations, although autogenetic effects were only recorded in the latter.

In the present study we have reinvestigated the problem of autogenetic inhibition of gastrocnemius fusimotor discharge exclusively in spinal cats. Stretch was applied to the tendo Achillis while recording from individual fusimotor neurones to the gastrocnemius which exhibited background discharge. No significant changes in discharge frequency accompanied stretch in preparations in which nerves other than those to triceps surae had been divided. Repeated attempts failed to demonstrate the expected autogenetic inhibition. Furthermore, when recording from medial gastrocnemius fusimotor neurones, no reflex effects were observed following volleys in the heteronomous nerve, that to lateral gastrocnemius-soleus. We have, therefore, been unable to demonstrate a powerful reflex effect of afferent fibres from muscle stretch receptors on fusimotor neurones to the same muscle in the acute spinal cat. The previous results (Hunt, 1951) showing autogenetic inhibitory effects were performed in decerebrate cats. Inhibition of fusimotor discharge following muscle stretch was also observed in spinal cats, but in this case the effects may not have been autogenetic, since the destination of the fusimotor neurones was not known. Possibly supraspinal mechanisms present in the decerebrate preparation could account for the difference in the present and previous results. Under the conditions of the present experiments autogenetic inhibition has not been demonstrable. Further investigation will be needed to decide whether

there is a spinal mechanism for autogenetic inhibition of fusimotor neurones. We can only state that if such a mechanism is present, its potency is not sufficient for it to be manifest under the conditions of the present experiments. This should not be taken to imply that muscle stretch has no spinal reflex effect on the discharge of fusimotor neurones to other muscles.

DISCUSSION

Hunt (1951) noted a number of differences in the reflex behaviour of motor neurones and fusimotor neurones. Fusimotor neurones were found to discharge at higher frequencies and to have a lower threshold for reflex activation than motor neurones. Subsequent investigations by Granit and his collaborators have shown further differences between these two types of motor cells. Eldred *et al.* (1953) found that fusimotor neurones could be excited or inhibited by stimulation of specific supraspinal sites and the threshold for effects on fusimotor neurones was less than that required to affect motor neurones. They also noted that section of dorsal roots had a much greater effect on motor neurone response than on fusimotor response to supraspinal stimulation. The latter effect may be explained by the fact that discharge in group I*a* afferent fibres modulates excitation in motor neurones whereas fusimotor neurones lack such excitatory connexions (see below).

The present study provides further evidence of basic differences in the properties and functional organization of fusimotor neurones and motor neurones. Fusimotor neurones appear to have a much briefer subnormality after impulse conduction as revealed by the rapid recovery of such neurones following the conduction of an antidromic impulse. The reflex excitability of a fusimotor neurone may be largely restored within a few milliseconds after impulse conduction, in contrast to the prolonged subnormality of motor neurones. These differences undoubtedly play a role in the capacity of these two types of motor cells to discharge repetitively. The motoneurones characteristically discharge in reflex acts at comparatively low frequencies, often in the range of 10–50/sec (Adrian & Bronk, 1929). In contrast, fusimotor neurones may discharge at frequencies in excess of 100/second and may at times reach peak frequencies of 150/second to natural stimuli. Further, the difference in rate of recovery may play a part in the response to single afferent volleys. Fusimotor neurones show a much greater tendency to give repetitive discharge to a single afferent volley than do motoneurones. It is only in protracted reflex actions, caused by stimulation of slowly conducting afferent fibres, that motoneurones display repetitive discharge. On the other hand, fusimotor neurones often discharge repeatedly, following a single afferent volley confined to lower threshold afferent fibres.

Another basic difference between fusimotor neurones and motoneurones is the apparent lack of monosynaptic excitatory connexions from primary

afferent fibres to the former. The monosynaptic pathway to motoneurones, of which the afferent arc is provided by afferent fibres from muscle spindles, plays an important part in determining the reflex excitability of motoneurones. Measurements of central delay give no support for the presence of such connexions to fusimotor neurones. Rather these seem to be innervated only by polysynaptic pathways within the spinal cord. In this connexion it has not been possible to demonstrate reflex effects on fusimotor neurones that clearly arise from activity in group I*a* afferent fibres from muscle spindles. The myotatic, or stretch reflex, therefore, appears to be confined to motoneurones.

In contrast to previous studies by Hunt (1951) and by Eldred & Hagbarth (1954) the present experiments reveal that there is a considerable gradation in reflex excitability among fusimotor neurones. The technique employed herein of recording fusimotor discharge in filaments of muscle nerve has permitted identification of those units which do not show a background or spontaneous discharge. In the acute spinal preparation less than 50% of the fusimotor neurones have background discharge. In general such neurones are more readily excited by reflex action than motoneurones to the same muscles. However, other fusimotor neurones are less readily excited than motoneurones and there appears to be a gradation in reflex excitability between these two extremes. Previous studies have laid great emphasis on the fact that the threshold for reflex excitation of fusimotor neurones, both from structures in the periphery as well as sites of stimulation in the supraspinal structures, is frequently lower. This has led Eldred *et al.* (1953) to suggest that a major pathway for the initiation of discharge in motoneurones is indirect via the fusimotor neurones and group I*a* muscle spindle afferent fibres, their so-called 'gamma servo system'. It now becomes apparent that such a scheme could only be applied to a fraction of the fusimotor neurone population.

The reflex control of fusimotor discharge, at least from the periphery, is complex. Excitatory and inhibitory influences impinge from a number of structures upon one individual fusimotor neurone. The effects produced by cutaneous stimulation are the most striking and it appears likely that influences from this source play a major role in the regulation of activity in fusimotor neurones. The reflex patterns that have been found will require further study for their fuller interpretation. In addition, knowledge of the behaviour of motoneurones in similar circumstances will be necessary.

SUMMARY

Reflex responses of individual fusimotor neurones have been recorded in ventral root and in muscle nerve in acute spinal cats. The following characteristic features have been found:

1. The reflex discharge to single afferent volleys is often repetitive and at high frequency.

2. Recovery of reflex excitability following conduction of an antidromic impulse is rapid.

3. Measurements of minimal central reflex delay fail to provide evidence of monosynaptic excitation by primary afferent fibres.

4. Only a fraction of the population exhibits background or 'spontaneous' discharge. The background discharge and reflex response to nerve volleys are abolished by suitable doses of myanesin.

5. Fusimotor neurones to specific muscles have been examined for their reflex responses to nerve volleys and to natural stimuli. The patterns of response are described and the implications as to functional organization are discussed.

6. Previously reported autogenetic inhibition of fusimotor discharge by muscle stretch has not been confirmed in acute spinal preparations.

This investigation was supported by research grants (B1039, B1320) from the Institute for Neurological Disease and Blindness, Public Health Service, and by a grant from the United Cerebral Palsy Research and Educational Foundation. Certain preliminary experiments were performed by one of us (C.C.H.) at the Rockefeller Institute for Medical Research. We wish to thank Mr Harry Feintuch for help in instrumentation.

REFERENCES

ADRIAN, E. D. & BRONK, D. W. (1929). The discharge of impulses in motor nerve fibres. Part II. The frequency of discharge in reflex and voluntary contractions. *J. Physiol.* **67**, 119–151.

ECCLES, J. C. & PRITCHARD, J. J. (1937). The action potential of motoneurones. *J. Physiol.* **89**, 43*P*.

ELDRED, E., GRANIT, R. & MERTON, P. A. (1953). Supraspinal control of the muscle spindles and its significance. *J. Physiol.* **122**, 498–523.

ELDRED, E. & HAGBARTH, K. E. (1954). Facilitation and inhibition of gamma efferents by stimulation of certain skin areas. *J. Neurophysiol.* **17**, 59–65.

GRANIT, R., JOB, C. & KAADA, B. R. (1952). Activation of muscle spindles in pinna reflex. *Acta physiol. scand.* **27**, 161–168.

GRANIT, R. & KAADA, B. R. (1952). Influence of stimulation of central nervous structures on muscle spindles in cat. *Acta physiol. scand.* **27**, 130–160.

GRANIT, R., PASCOE, J. E. & STEG, G. (1957). The behaviour of tonic α and γ motoneurones during stimulation of recurrent collaterals. *J. Physiol.* **138**, 381–400.

HUNT, C. C. (1951). The reflex activity of mammalian small-nerve fibres. *J. Physiol.* **115**, 456–469.

HUNT, C. C. & KUFFLER, S. W. (1951). Further study of efferent small-nerve fibres to mammalian muscle spindles. Multiple spindle innervation and activity during contraction. *J. Physiol.* **113**, 283–297.

KOBAYASHI, Y., OSHIMA, K. & TASAKI, I. (1952). Analysis of afferent and efferent systems in the muscle nerve of the toad and cat. *J. Physiol.* **117**, 152–171.

KUFFLER, S. W., HUNT, C. C. & QUILLIAM, J. P. (1951). Function of medullated small-nerve fibres in mammalian ventral roots: efferent muscle spindle innervation. *J. Neurophysiol.* **14**, 29–54.

LAPORTE, Y. & LLOYD, D. P. C. (1952). Nature and significance of the reflex connexions established by large afferent fibres of muscular origin. *Amer. J. Physiol.* **169**, 609–621.

LEKSELL, L. (1945). The action potential and excitatory effects of the small ventral root fibres to skeletal muscle. *Acta physiol. scand.* **10**, Suppl. 31, 1–88.

LLOYD, D. P. C. (1943). The interaction of antidromic and orthodromic volleys in a segmental spinal motor nucleus. *J. Neurophysiol.* **6**, 143–151.

LLOYD, D. P. C. (1951). Electrical signs of impulse conduction in spinal motoneurones. *J. gen. Physiol.* **35**, 255–288.

LLOYD, D. P. C. & MCINTYRE, A. K. (1955). Monosynaptic reflex responses of individual motoneurones. *J. gen. Physiol.* **38**, 771–787.

PERL, E. R. (1957). Crossed reflexes of cutaneous origin. *Amer. J. Physiol.* **188**, 609–615.

RENSHAW, B. (1940). Activity in the simplest spinal reflex pathways. *J. Neurophysiol.* **3**, 373–387.

ROSE, J. E. & MOUNTCASTLE, V. B. (1954). Activity of single neurons in the tactile thalamic region of the cat in response to a transient peripheral stimulus. *Bull. Johns Hopkins Hosp.* **94**, 238–282.

WOODBURY, J. W. & PATTON, H. D. (1952). Electrical activity of single spinal cord elements. *Cold Spring Harbor Symp. Quant. Biol.* **17**, 185–188.