J. Physiol. (1978). **282**, *pp.* 59–71
With 7 text-figures
Printed in Great Britain

SOME PROPERTIES OF SPINAL γ-MOTONEURONES IN THE CAT, DETERMINED BY MICRO-ELECTRODE RECORDING

BY R. E. KEMM* AND D. R. WESTBURY
From the Department of Physiology, the Medical School, Birmingham B15 2TJ

(*Received 8 February 1978*)

SUMMARY

1. Micro-electrode recordings were made from motoneurones in the lumbo-sacral region of the cat spinal cord whose axonal conduction velocities were 10–55 m/sec. Most of these may be presumed to be fusimotor in function.

2. Intracellular records from twelve γ-motoneurones revealed six with short (2–4 msec) and six with long (30–100 msec) duration after-hyperpolarizations following an antidromically conducted action potential.

3. Using extracellular recording, the excitability of eighty-nine other γ-motoneurones following an antidromic impulse was tested with a second antidromic action potential. In eighty-four of these neurones, the minimum antidromic response interval was short, 1·5–3·5 msec, implying that in most γ-motoneurones, after-hyperpolarization was of limited effectiveness and of short duration. In the remaining five neurones, the minimum response interval was longer, 20–80 msec.

4. There was a lack of monosynaptic excitation from group 1 afferent axons in the dorsal roots in eleven of the twelve motoneurones from which intracellular records were obtained. Polysynaptic excitation was commonly observed.

5. In these anaesthetized preparations, there was a lack of recurrent i.p.s.p.s even though such evidence of Renshaw inhibition could be found in the neighbouring α-motoneurones.

6. The mean input resistance of γ-motoneurones was shown to be 1·55 MΩ and the principal time constant 8·5 msec by passing hyperpolarizing current through the recording micro-electrode in a bridge circuit. These values are open to error because of the small numbers of neurones investigated, and of the use of the single micro-electrode method.

7. Depolarizing current passed through the recording micro-electrode caused a maintained discharge of action potentials at a high rate. After-hyperpolarization had little effect on discharge rate. The threshold for injected current to cause discharge was very low, and the discharge rate increased rapidly with the magnitude of the current.

8. These properties of γ-motoneurones are discussed in relation to their function.

* Present address: Department of Physiology, University of Melbourne, Parkville, Melbourne, Australia 3052.

INTRODUCTION

γ-Motoneurones discharge at higher rates than do α-motoneurones under similar circumstances and they have lower reflex thresholds. Their discharges are often maintained for long periods. Studies of the influence of these neurones on muscle spindles show that this increases with discharge rate up to 150 impulses/sec (Matthews, 1972), and discharge rates of up to 100/sec have been recorded from γ-motoneurones in decerebrate cat preparations (Hunt, 1951). This suggests that most γ-motoneurones are different from α-motoneurones, particularly from the small, tonic α-motoneurones, in their properties and in the factors which control their discharge rate.

In experiments presented here, intracellular and extracellular micro-electrode recording techniques have been used to provide information about the properties of γ-motoneurones which influence their discharge. The results have shown that γ-motoneurones are much less influenced by after-hyperpolarization than are α-motoneurones. A preliminary account of some of these results has been published (Kemm & Westbury, 1976).

METHODS

Experiments were performed upon young cats of either sex, weighing between 2·2 and 3·9 kg. The animals were anaesthetized with sodium pentobarbitone (Nembutal, Abbot Laboratories). The lumbo-sacral spinal cord was exposed by laminectomy. The dura mater was opened and the dorsal roots (L6, L7 and S1) were divided and the central parts of these were mounted on bipolar electrodes for stimulation. Arrangements were made to stimulate either the sciatic nerve or its branches in the hind limb. Bilateral pneumothorax was performed, and positive pressure ventilation employed. The animals were paralysed with gallamine triethiodide (Flaxedil, May & Baker Ltd). End-tidal CO_2 was measured (Beckmann gas analyser, LBI) and maintained at 4·5 %. Arterial blood pressure was maintained at 80 mmHg or above. The temperature of the animal and of the pool of liquid paraffin covering the spinal cord was kept at 37 °C.

Electrical recordings were made from γ-motoneurones in the spinal cord using conventional micro-electrode techniques. Glass micropipettes filled with 2M-potassium citrate and having resistance of 10–30 MΩ were employed. An electrometer with high input impedance was used as the first stage of the recording system and this incorporated compensation for capacitance in the input circuit. A bridge circuit was employed for passing current through the recording electrode to polarize the neurones.

Both intracellular and extracellular records were obtained from γ-motoneurones. The neurones were identified by their antidromic response to electrical stimulation of the sciatic nerve or of its branches, and by the conduction velocity of their axons. In some cases the neurones discharged repetitively, when collision of orthodromic and antidromic impulses could be observed.

RESULTS

Motoneurones with axonal conduction velocities between 10 and 55 m/sec were studied. Fig. 1 shows the distribution of axonal conduction velocities of 228 motoneurones observed by micro-electrode recording. These span the range of group Aγ and seem to form a single group on the basis of conduction velocity. Most of these neurones may be presumed to be fusimotor in function (Kuffler, Hunt & Quilliam, 1951) but those conducting faster than 50 m/sec may have included skeletomotor neurones. It was not usually possible in these experiments to identify the motoneurones functionally. Only extracellular recordings were obtained from most of these motoneurones, but intracellular recordings were obtained from twelve neurones

with stable membrane potentials of 50 mV or more and no signs of injury. The results of these intracellular recordings were supported by twenty-two more recordings in which the membrane potential was lower than 50 mV or was not stable.

Intracellular records

In the twelve intracellular records, the changes in membrane potential following stimulation of the axons of the motoneurones could be measured directly. In six of the motoneurones, the action potential was followed by an after-hyperpolarization of

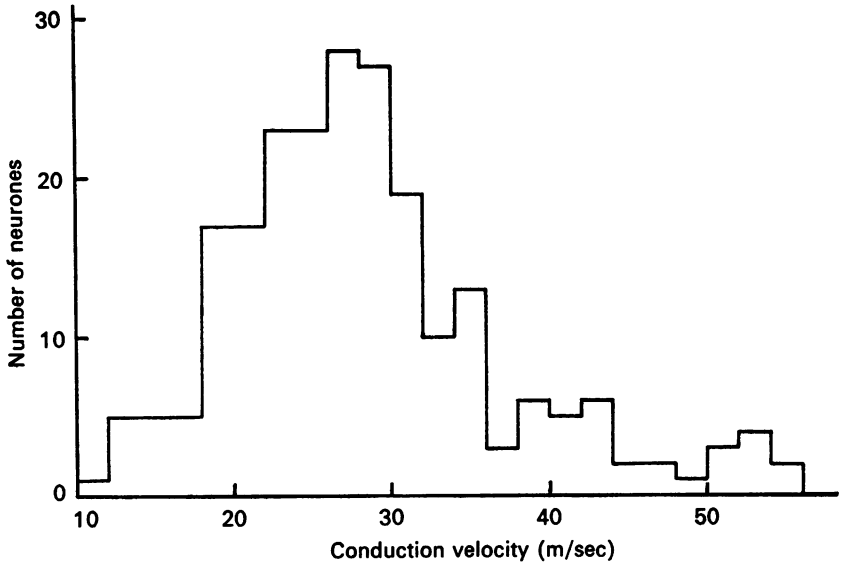

Fig. 1. Histogram showing the distribution of axonal conduction velocities of lumbosacral motoneurones between 10 and 55 m/sec recorded by micro-electrode. The velocities were calculated from the conduction time of antidromic action potentials to the neurone soma following stimulation of the sciatic nerve or one of its branches. The histogram represents 228 motoneurones; most records were obtained extracellularly.

small amplitude (1–4 mV) and short duration (2–4 msec). These neurones had conduction velocities of 22–42 m/sec, mean 31 m/sec. In the other six motoneurones, the after-hyperpolarization was larger (5–7 mV), and of long duration (30–100 msec). The axonal conduction velocities of these neurones were 28–55 m/sec, mean 46 m/sec. Although four of these motoneurones had conduction velocities of about 50 m/sec, two were seen with slower conducting axons, 38 and 28 m/sec.

Only one of these motoneurones (in the second group; conduction velocity, 51 m/sec) received monosynaptic excitation following electrical stimulation of group 1 afferent axons in the dorsal root, the others did not have such excitation (see below).

Fig. 2. *A* illustrates the antidromic action potential of a motoneurone with a small after-hyperpolarization. This record shows clearly that the lack of significant after-hyperpolarization was not the result of damage to the neurone as the membrane potential of 62 mV was stable and the action potential invaded both the initial segment and the soma, as was shown by the subdivision of the action potential into

initial segment and soma-dendritic fractions (Brock, Coombs & Eccles, 1953). Records of synaptic potentials could be obtained from these neurones showing that the records demonstrating short after-hyperpolarizations were not from axons.

Fig. 3. illustrates the after-hyperpolarization following an antidromic action potential in a motoneurone in which the after-hyperpolarization was much larger and of long duration, as in the second group of neurones. This record is similar to those

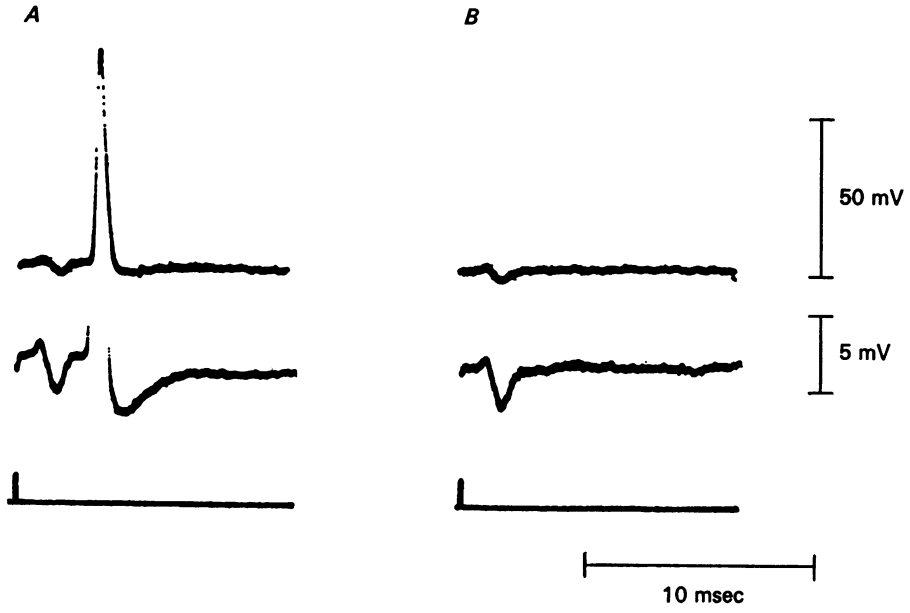

Fig. 2. Intracellular record from a motoneurone with an axonal conduction velocity of 27 m/sec. The membrane potential was 62 mV, and the amplitude of the action potential was 67 mV. The record shows the changes in membrane potential which occurred following electrical stimulation of the sciatic nerve in the upper leg. The records were obtained with DC amplifiers, the upper traces are at low gain, the lower traces are at higher gain. In *A*, the stimulus to the nerve was above the threshold for the axon of the gamma motoneurone, and in *B*, the stimulus was just below that strength. In both records, neighbouring alpha motoneurones were excited and this is shown in both recordings by an evoked potential at 1 msec which could also be recorded outside the γ-motoneurone.

obtained from motoneurones with faster axonal conduction velocities (Eccles, Eccles & Lundberg, 1958) and the negative after-hyperpolarization was preceded by a brief delayed depolarization.

Among the twenty-two motoneurones in which there were signs of injury, for example, low membrane potential or small action potential amplitude, there was a preponderance of neurones with small, short after-hyperpolarizations. Although these results must be regarded with suspicion, they indicated the possibility that the selection of motoneurones from which intracellular records were obtained was not representative of the whole population of γ-motoneurones.

Of the twelve motoneurones from which reliable intracellular records were obtained, only one received a monosynaptic input from group 1 afferent axons when these were stimulated electrically in the divided dorsal roots. The remaining eleven

motoneurones showed e.p.s.p.s which began with longer latency, suggesting that polysynaptic pathways were involved. Fig. 4 shows a record from a motoneurone with a conduction velocity of 22 m/sec following electrical stimulation of the dorsal roots at a strength just above the threshold for group 1 axons. In this neurone a complex e.p.s.p. was observed which began after 5·5 msec and which was often large enough to discharge the motoneurone orthodromically, but no synaptic input was

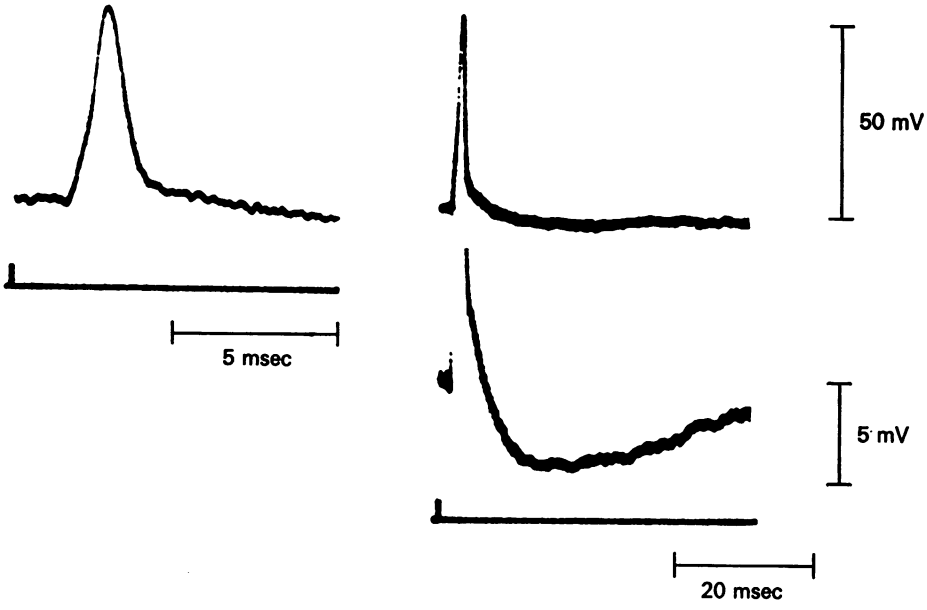

Fig. 3. Intracellular record from a motoneurone with a conduction velocity of 52 m/sec. The membrane potential was 53 mV, and the action potential was 50 mV in amplitude. The record shows the response following stimulation of the sciatic nerve in the upper leg at a strength above the threshold for the axon of the motoneurone. The records were obtained with DC coupled amplifiers, the upper trace being at low gain, and the lower at higher gain. This motoneurone was not excited monosynaptically from group 1 afferent axons in the dorsal root.

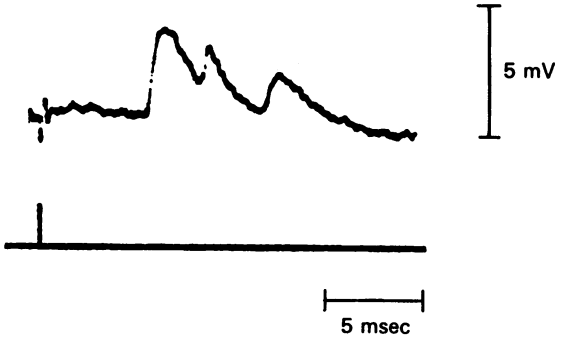

Fig. 4. Intracellular record from a motoneurone with an axonal conduction velocity of 22 m/sec. The membrane potential was 60 mV and the amplitude of the antidromic action potential was 61 mV. The record shows the response to a single electrical stimulus delivered to the central end of the divided ipsilateral dorsal roots at a strength just above the threshold for group 1 axons.

seen at latencies which would correspond to a monosynaptic connexion. A monosynaptic post-synaptic potential would have been expected to begin at about 0·75 msec, and this delay was confirmed in intracellular recordings from α-motoneurones in the same experiments where large monosynaptic e.p.s.p.s were seen.

Inhibitory post-synaptic potentials arising from the activation of recurrent collaterals and Renshaw cells were not found in any of the twelve γ-motoneurones although such inhibitory potentials were observed in α-motoneurones in the same experiments. Evidence for this lack of Renshaw inhibition is shown in Fig. 2. Electrical stimulation of the sciatic nerve excited a large number of α-motoneurone axons and orthodromic influences were prevented by section of the dorsal roots. Activity of the neighbouring α-motoneurones is shown by the deflexion of the trace at about 1 msec. This evoked potential could also be recorded when the micro-electrode was positioned outside the γ-motoneurone. Despite the α-motoneurone activity, no i.p.s.p.s can be in the higher gain records from the γ-motoneurone (lower traces). This can be seen more clearly in Fig. 2*B* where the stimulus has been reduced so as to be just below the threshold for the axon of the γ-motoneurone, but still above that of the α-motoneurones. Recurrent inhibition would have been expected to begin at about 3 msec in these experiments.

Input resistance, time constant and discharge

It was possible in four of the γ-motoneurones with membrane potentials of more than 50 mV to pass current through the recording micro-electrode using a bridge circuit. Small hyperpolarizations of the neurones allowed an estimate to be made of their input resistances and time constants. The mean values for these parameters for four motoneurones with conduction velocities between 22 and 55 m/sec were, for input resistance 1·55 MΩ, and for time constant, 8·5 msec, using the method of Rall (1960).

These values may be subject to considerable error, in addition to being few in number. The use of the single micro-electrode method for the measurement of these parameters is open to error. These errors may be minimized by the use of specially prepared micro-electrodes (Burke & ten Bruggencate, 1971) but such large micropipettes could not be employed in the present experiments. The use of the Rall (1960) method to determine the principal time constant for the neurones involves acceptance of several assumptions about the geometry of the neurones and of the distribution of the injected current within them which may not be justified. The method has been used for α-motoneurones (e.g Burke & ten Bruggencate, 1971) but it is not possible to decide at present on whether it is appropriate for other neurones.

The passage of a depolarizing current through the recording micro-electrode elicited a maintained discharge of action potentials from the neurones. Fig. 5 shows an example of the discharge produced by a current of 3 nA in one of the γ-motoneurones. In this neurone, the discharge was maintained at 40 impulses/sec for the duration of the injected current, up to 1 sec in this case. The motoneurone was silent before the passage of current. The first two or three action potentials were discharged at a much higher rate than subsequent impulses. This is clearly illustrated in Fig. 5*A* where the first interspike interval was less than 4 msec. Fig. 5*B* shows that during a maintained discharge, a hyperpolarization developed which was associated

with the slowing of the discharge. This hyperpolarization was more than could be accounted for by the summation of individual after-hyperpolarizations, as these were of short duration, about 2 msec in this motoneurone.

Extracellular records

Because of the technical difficulty of obtaining satisfactory intracellular records from γ-motoneurones, there is doubt as to how representative these records are, both in the present study and in previous work (Eccles, Eccles, Iggo & Lundberg, 1960). However, a large number of extracellular records may be obtained, and these may

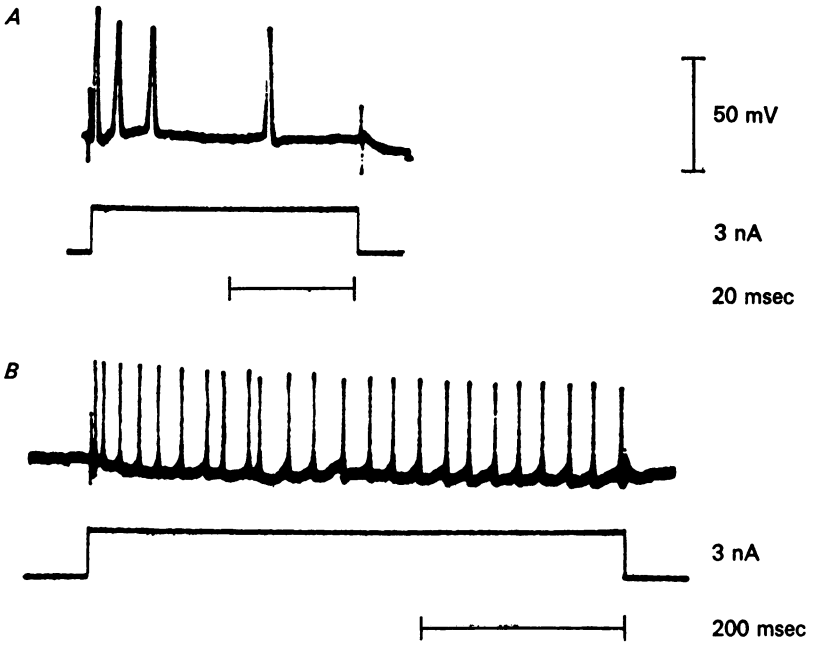

Fig. 5. Intracellular record from the same motoneurone as in Fig. 4 showing the changes in membrane potential during the passage of a depolarizing current of 3 nA through the recording electrode. The records of current have been drawn. The input resistance of this neurone was 1·7 MΩ and the principal time constant was 9·4 msec (see text).

provide a more comprehensive view of the properties of the population of γ-motoneurones in the lumbo-sacral spinal cord. Extracellular records were obtained from eighty-nine γ-motoneurones in which two strong stimuli were applied to the sciatic nerve to induce antidromic action potentials in their axons. The minimal interval at which these impulses would invade the soma of the motoneurones was measured. This was taken as an approximate measure of the duration of the reduced excitability of the neurone following an action potential. If the effects of recurrent inhibition are minimal or non-existent in these preparations, as the intracellular records would suggest, then this measure largely reflects the extent of the after-hyperpolarization that follows the first action potential.

In a few γ-motoneurones, and in a sample of α-motoneurones in which the method could be assessed by intracellular recording, the minimum response interval gave a useful estimate of the

duration of after-hyperpolarization, but for most of the neurones tested the method provides an indirect estimate only.

The minimum response interval was taken as the time between antidromic action potentials recorded in the spinal cord when the time between the stimuli delivered to the sciatic nerve was reduced to the minimum interval at which the neurone soma discharged in all trials. The time between responses was usually greater than the time between stimuli because the conduction velocity for the second impulse was less than that for the first. When the minimum response interval for the neurone was very short, it could not be shown to be different from the refractory period of its axon.

As far as could be determined, short response intervals were not the result of delayed depolarizations interposed between the action potential and a subsequent after-hyperpolarization. In an attempt to avoid this possibility, the stimulus interval was reduced from a large value, often 100 msec, to determine the interval at which the neuronal response failed.

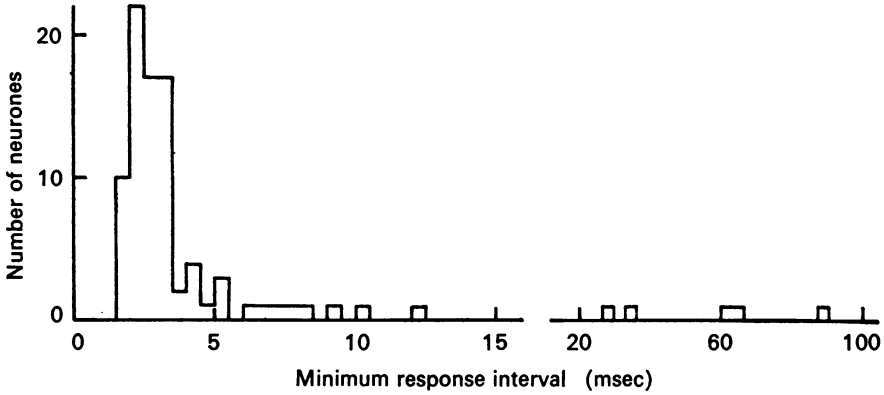

Fig. 6. Histogram showing the distribution of minimum response intervals following paired stimuli delivered to the sciatic nerve. The responses were obtained by extracellular recording from eighty-nine motoneurones with axonal conduction velocities in the range 10–55 m/sec. Note the break in the abscissa and the change in scale at that point.

The distribution of these intervals between antidromic responses is plotted in Fig. 6 as a histogram. Eighty-four of the motoneurones had short minimum response intervals (1·5–12 msec, sixty-six in the range of 1·5–3·5 msec). The axonal conduction velocities of these were 11–51 m/sec. In the remaining five motoneurones, the minimum response interval was longer (20–80 msec) and the axonal conduction velocities of these neurones were in the range 24–52 m/sec.

There was no clear relationship between the minimum response interval and conduction velocity. This lack of relationship is shown in the scatter diagram in Fig. 7. Those motoneurones with long minimum response times were not found among the slowest conducting neurones. Although three of them were among the fastest conducting neurones the remaining two had conduction velocities of less than 30 m/sec. While most of the motoneurones with short response intervals had conduction velocities of 40 m/sec or less, one had a value of 51 m/sec.

DISCUSSION

In these experiments, records were obtained from lumbo-sacral motoneurones with axonal conduction velocities of between 10 and 55 m/sec. It was not possible to determine with certainty the function of the neurones. Motoneurones subserve

skeletomotor, fusimotor, or mixed skeleto-fusimotor roles. The division into alpha and gamma ranges was originally based on conduction velocity (or axon diameter) with the transition between the ranges at about 55 m/sec. It is clear that the functional subgroupings are not as separate as this, but the degree of overlap is unknown. It has been generally accepted that motoneurones with axonal conduction velocities of 50 m/sec or less are fusimotor in function (Kuffler, Hunt & Quilliam, 1951; Hunt, 1951). The exhaustive study of Ellaway, Emonet-Dénand, Joffroy & Laporte, (1972) has shown that motoneurones with conduction velocities above 50 m/sec have a

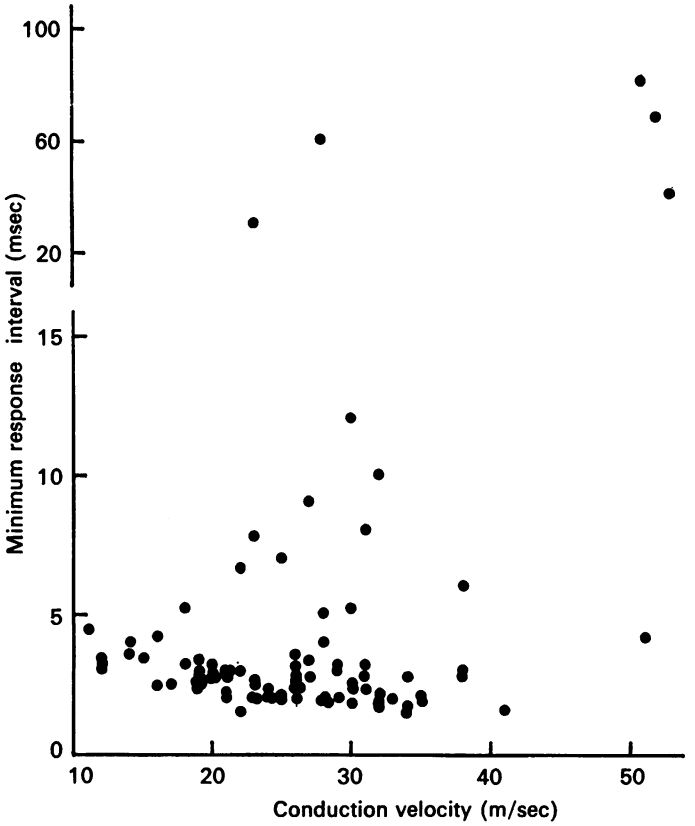

Fig. 7. Scatter diagram showing the relationship between minimum antidromic response interval following stimulation of the sciatic nerve with paired shocks, and the axonal conduction velocity of the motoneurones. Responses from eighty-nine neurones are included. Note the break in the ordinate and the change in scale at that point.

skeletomotor function and are not exclusively fusimotor. This question is clouded by the existence of skeleto-fusimotor motoneurones which innervate both extra- and intrafusal muscle fibres. Such axons have so far been identified in the cat with conduction velocities of between 39 and 100 m/sec (McWilliam, 1975; Emonet-Dénand, Jami & Laporte, 1975). The properties of these are largely unknown, although Burke & Tsairis (1977) have described one such motoneurone which closely resembled a small, tonic alpha motoneurone (type S, Burke, 1967).

There is strong evidence that gamma motoneurones, presumed to be fusimotor, do

not receive monosynaptic excitation from group 1 afferent axons (Hunt & Paintal, 1958; Eccles *et al.* 1960), and the present results would support this view. It seems likely that the lack of a monosynaptic group 1 input, along with axonal conduction velocity form an adequate criterion for the functional identification of fusimotor motoneurones, but the question will remain in doubt until these presumptive data are correlated with direct functional identification. On the basis of these criteria, it is likely, though not proven, that all but one of the intracellular records obtained in these experiments were from fusimotor motoneurones. This is also likely to be true for all of the extracellular records, although it is not possible to be as sure of a lack of monosynaptic excitation in these.

Recurrent inhibition

The present results have revealed no sign of inhibitory post-synaptic potentials resembling recurrent inhibition in gamma motoneurones. Previous work has identified recurrent inhibition acting on some gamma motoneurones, but these experiments have all been carried out on decerebrate or spinal preparations in which gamma motoneurones were discharging (Grillner, 1969; Ellaway, 1971; Noth, 1971), and the effect was weak. Renshaw inhibition is more pronounced in the decerebrate preparation than in the present anaesthetized preparations. However, in the experiments presented here, recurrent inhibitory post-synaptic potentials could be clearly demonstrated in α-motoneurones, although they were not apparent in adjacent gamma motoneurones, suggesting that the mechanism of recurrent inhibition occurring in the latter is different from the well known disynaptic connexion. Such a possibility was raised by Noth (1971) who found evidence of convergence of afferent input from contralateral dorsal roots which might have modulated the recurrent inhibition to gamma motoneurones, suggesting an additional interneurone in the pathway. The division of ipsilateral dorsal roots in the present experiments might have deprived the recurrent inhibitory mechanism for gamma motoneurones of facilitation.

Sjöström & Zangger (1976) have made simultaneous recordings from α- and γ-motoneurones in the spinal cat during stepping movements. γ-Motoneurones discharged before alpha motoneurones in the movement, but there was no reduction in the gamma discharge when α-discharge (and presumably Renshaw cell discharge) began, again implying little effective recurrent inhibition under those circumstances also.

After-hyperpolarization

Eccles *et al.* (1960) suggested, on the basis of intracellular recordings, that the after-hyperpolarizations of many gamma motoneurones were of long duration, not dissimilar to those of the small alpha motoneurones. In their records neurones with both long and short after-hyperpolarizations were seen, but the latter were regarded with suspicion because of injury. The results of the present experiments show clearly that many γ-motoneurones do have short after-hyperpolarizations and that this is not the result of injury following penetration by the micro-electrode.

If the minimum response interval to paired electrical stimuli delivered to the sciatic nerve is a reasonable measure of after-hyperpolarization, as seems likely, then

neurones with short, ineffective after-hyperpolarization make up the majority of γ-motoneurones. From this point of view, the twelve motoneurones from which intracellular records were obtained may not have been representative of the whole population.

Hunt & Paintal (1958) investigated the recovery of reflex excitability of γ-motoneurones following an action potential and found it to be rapid. Their result is similar to the present findings using paired antidromic responses of the motoneurones, and also lends support for the view that after-hyperpolarization and recurrent inhibition are not major influences in these motoneurones. However, they only investigated six motoneurones and the present results extend the findings considerably. In addition, the present experiments have shown that there are some motoneurones in the γ-range with long, more effective after-hyperpolarizations, although these would seem to be few in number. The functional significance of these is not known, but only one of them received monosynaptic excitation from group 1 afferent axons.

Discharge characteristics

The values of input resistance measured in the present experiments are lower than would have been expected from measurements of input resistance of α-motoneurones (Burke & ten Bruggencate, 1971). The present results are open to question because of the difficulties involved in the use of the single electrode for both recording and current passing. Neither is it certain how representative are the values of principal time constant.

The γ-motoneurones were very sensitive to injected depolarizing current. 2 or 3 nA was usually sufficient to elicit a maintained rapid discharge, whereas such a current would be below the threshold for α-motoneurones in these preparations. The maintained discharge was at a higher rate than is seen in α-motoneurones with depolarizing current of such low amplitude. These findings are consistent with the high reflex excitability of γ-motoneurones which has been observed (Hunt, 1951). The discharge of most α-motoneurones is regulated by the after-hyperpolarization conductance (Baldissera & Gustafsson, 1971) but this would not seem to be a sufficient explanation for the regulation of discharge in γ-motoneurones where the after-hyperpolarization was short and apparently ineffective. The progressive, slow hyperpolarization seen during discharge, for example, in Fig. 5*B*, will have contributed to the slowing of discharge, but the mechanism of this is not certain.

Functional implications of the properties

Autogenetic excitation of fusimotor neurones implies an element of positive feedback, an arrangement which is probably not appropriate for many situations (Houk, 1972; Westbury & Kemm, 1978). One of the advantages that might be gained, therefore, from the separation of skeletomotor and fusimotor functions is the ability to employ an effective stretch reflex without an inevitable component of positive feedback. Autogenetic excitation of γ-motoneurones by afferent fibres from the primary endings of muscle spindles has been described by Trott (1976) and by Fromm & Noth (1976) but the influence is not very strong and is presumably not monosynaptic, in view of the consistent finding that γ-motoneurones lack such a connexion.

The reflex threshold of gamma motoneurones is much lower and their responsiveness is much greater than that of α-motoneurones (Hunt, 1951; Hunt & Paintal, 1958). The properties underlying their discharge characteristics are consistent with this. γ-Motoneurones can produce maintained discharges of action potentials at much higher rates than can α-motoneurones. This must presumably reflect differences in membrane properties and in particular in after-hyperpolarization. A second advantage which may be gained by the separation of skeletomotor and fusimotor functions is the ability to employ different patterns of efferent discharge, each perhaps more appropriate to the muscle fibres innervated.

Recurrent inhibition of gamma motoneurones is much weaker than that operating on α-motoneurones, and seems to be mediated by a different mechanism. Such flexibility would seem to offer advantage, for it would not be appropriate for α-motoneurone discharge to bring about, inevitably, a decrease in γ-motoneurone activity. Those records which have been obtained showing concurrent α- and γ-motoneurone activity (Sjöström & Zangger, 1976) contain no sign of recurrent inhibitory influence on the γ-motoneurones.

Fusimotor neurones can be clearly divided into static and dynamic subgroups on the basis of their influence on muscle spindle response (Matthews, 1962; Emonet-Dénand, Laporte, Matthews & Petit, 1977). There are differences in descending input to the two types of neurone (Appelberg & Jeneskog, 1972) and Bergmans & Grillner (1969) have shown that dynamic fusimotor neurones are active in the spinal preparation and static neurones are silent, and that the situation is reversed by an injection of DOPA, at least in flexor muscles. No clear subdivision of the γ-motoneurones so far studied is apparent from their properties. The relationship between the roles of the motoneurones and their properties is unlikely to be resolved until direct identification of the peripheral connexions of each motoneurone is possible.

We wish to thank Mr P. Walker for technical assistance. The work is supported by a grant from the Wellcome Trust.

REFERENCES

Appelberg, B. & Jeneskog, T. (1972). Mesencephalic fusimotor control. *Brain Res.* **15**, 97–112.

Baldissera, F. & Gustafsson, B. (1971). Regulation of repetitive firing in motoneurons by the after-hyperpolarization conductance. *Brain Res.* **30**, 431–434.

Bergmans, J. & Grillner, S. (1969). Reciprocal control of spontaneous activity and reflex effects in static and dynamic flexor γ-motoneurones revealed by an injection of DOPA. *Acta physiol. scand.* **77**, 106–124.

Brock, L. G., Coombs, J. S. & Eccles, J. C. (1953). Intracellular recording from antidromically activated motoneurones. *J. Physiol.* **122**, 429–461.

Burke, R. E. (1967). Motor unit types of cat triceps surae muscle. *J. Physiol.* **193**, 141–160.

Burke, R. E. & Tsairis, P. (1977). Histochemical and physiological profile of a skeletofusimotor (β) unit in cat soleus muscle. *Brain Res.* **129**, 341–345.

Burke, R. E. & ten Bruggencate, G. (1971). Electronic characteristics of alpha motoneurones of varying size. *J. Physiol.* **212**, 1–20.

Eccles, J. C., Eccles, R. M., Iggo, A. & Lundberg, A. (1960). Electrophysiological studies on gamma motoneurones. *Acta physiol. scand.* **50**, 32–40.

Eccles, J. C., Eccles, R. M. & Lundberg, A. (1958). The action potentials of the alpha motoneurones supplying fast and slow muscles. *J. Physiol.* **142**, 275–291.

Ellaway, P. H. (1971). Recurrent inhibition of fusimotor neurones exhibiting background discharges in the decerebrate and the spinal cat. *J. Physiol.* **216**, 419–439.

ELLAWAY, P. H., EMONET-DÉNAND, F., JOFFROY, M. & LAPORTE, Y. (1972). Lack of exclusively fusimotor α-axons in flexor and extensor leg muscles of the cat. *J. Neurophysiol.* **35**, 149–153.

EMONET-DÉNAND, F., JAMI, L. & LAPORTE, Y. (1975). Skeleto-fusimotor axons in hind-limb muscles of the cat. *J. Physiol.* **249**, 153–166.

EMONET-DÉNAND, F., LAPORTE, Y., MATTHEWS, P. B. C. & PETIT, J. (1977). On the subdivision of static and dynamic fusimotor actions on the primary ending of the cat muscle spindle. *J. Physiol.* **268**, 827–861.

FROMM, C. & NOTH, J. (1976). Reflex responses of gamma motoneurones to vibration of the muscle they innervate. *J. Physiol.* **256**, 117–136.

GRILLNER, S. (1969). Supraspinal and segmental control of static and dynamic γ-motoneurones in the cat. *Acta physiol. scand.* suppl. 327, 1–34.

HOUK, J. C. (1972). The phylogeny of muscular control configurations. In: *Biocybernetics* **4**, ed. DRISCHEL, H., & DELLMAR, P., pp. 125–144. Jena: Fischer.

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

HUNT, C. C. & PAINTAL, A. S. (1958). Spinal reflex regulation of fusimotor neurones. *J. Physiol.* **143**, 195–212.

KEMM, R. E. & WESTBURY, D. R. (1976). The after-hyperpolarization of gamma motoneurones. *J. Physiol.* **263**, 124–125*P*.

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

MCWILLIAM, P. N. (1975). The incidence and properties of β axons to muscle spindles in the cat hind limb. *Q. Jl exp. Physiol.* **60**, 25–36.

MATTHEWS, P. B. C. (1962). The differentiation of the two types of fusimotor fibre by their effects on the dynamic response of muscle spindle primary endings. *Q. Jl exp. Physiol.* **47**, 324–333.

MATTHEWS, P. B. C. (1972). *Mammalian Muscle Receptors and Their Central Actions.* London: Edward Arnold.

NOTH, J. (1971). Recurrente Hummung der Extensor-Fusimotoneurone? *Pflügers Arch.* **329**, 23–33.

RALL, W. (1960). Membrane potential transients and membrane time constant of motoneurons. *Expl Neurol.* **2**, 503–532.

SJÖSTRÖM, A. & ZANGGER, P. (1976). Muscle spindle control during movements generated by the deafferented spinal cord. *Acta physiol. scand.* **97**, 281–291.

TROTT, J. R. (1976). The effect of low amplitude muscle vibration on the discharge of fusimotor neurones in the decerebrate cat. *J. Physiol.* **255**, 635–649.

WESTBURY, D. R. & KEMM, R. E. (1978). Some implications of the properties of spinal gamma motoneurones. In *Progress in Clinical Neurophysiology* chap. 8, ed. DESMEDT, J. E. (In the Press).