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FIELD POTENTIALS GENERATED BY GROUP II MUSCLE AFFERENTS IN THE MIDDLE LUMBAR SEGMENTS OF THE CAT SPINAL CORD

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SUMMARY

1. A powerful projection from group II muscle afferents of hind-limb muscles to the 3rd, 4th and 5th segments of the lumbar spinal cord has been demonstrated by focal synaptic field potential recording.

2. Field potentials were found at two locations: one in the dorsal horn (Rexed's laminae IV and V) and the other in the intermediate zone and ventral horn (Rexed's laminae VII and VIII). In the dorsal horn the field potentials were exceptionally large and were evoked only by group II afferents. At more ventral locations, they were smaller and were sometimes preceded by small field potentials evoked by group I afferents.

3. At both locations field potentials could be evoked by stimulation of a number of hind-limb muscle nerves at strengths sufficient to activate group II afferents. However, some nerves consistently evoked more powerful effects than others and the largest potentials were from the nerves to quadriceps, sartorius and to the pretibial flexor muscles (tibialis anterior and extensor digitorum longus). Activation of articular afferents (from the knee joint nerve) or Pacinian corpuscle afferents (from the interosseous nerve) evoked small field potentials at some locations.

4. In the dorsal horn the latency of the field potentials was so short that they must have been generated monosynaptically. Field potentials in the ventral horn had longer latencies, by 0·5–1·0 ms, but they also appear to have been monosynaptically evoked by slowly conducting intraspinal collaterals. This conclusion is based primarily on the effects of intraspinal stimulation which was found to antidromically activate afferents with the appropriate latencies and thresholds.

5. Evidence is presented that the dorsal and ventral field potentials are generated by afferents whose receptors can be activated by small (less than 100 μm) muscle stretches.

INTRODUCTION

Relatively little is known of the central projection and actions of muscle afferents in the group II range (4–12 μm diameter, conduction velocity 24–72 m/s; see Matthews, 1972). The majority of these are from the secondary endings of muscle

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spindle receptors but other types of receptor are also innervated by fibres in the same range. These include axons of receptors located intramuscularly, as in the case of Paciniform corpuscles or free nerve endings, or extramuscularly in joints, periosteum or other tissues (see Boyd & Davey, 1968; Matthews, 1972; see also the Discussion). Generally group II afferents are known to have direct actions upon motoneurones (Kirkwood & Sears, 1975; Stauffer, Watt, Taylor, Reinking & Stuart, 1976; Lundberg, Malmgren & Schomburg, 1977; Lüscher, Ruenzel, Fetz & Henneman, 1979; Munson, Fleshman & Sypert, 1980), some spinal interneurones (Lundberg, Malmgren & Schomburg, 1986*b*) and some ascending tract neurones (Eccles, Oscarsson & Willis, 1961). However, only in the case of motoneurones have these actions been convincingly attributed to secondary muscle spindle afferents. It is not known whether group II afferents which innervate different types of receptor have the same or different target cells or how widespread the actions of single afferents are.

In previous studies attention has focused primarily on projection of group II afferents to the grey matter of the caudal lumbar and the first sacral segments. A small number of group II afferents, which were functionally identified as secondary muscle spindle afferent axons, have been investigated morphologically following injection of horseradish peroxidase (Fyffe, 1979; N. Ishizuka, T. Hongo, N. Kudo, S. Sasaki, M. Yamashita & H. Mannen, personal communication). The collaterals of these afferents had dense arborizations in the dorsal horn (Rexed's laminae IV and V) and also more ventrally in lamina VII and among the motoneurones of lamina IX. Intraspinal stimulation at the same locations has been shown to activate terminal branches of similarly identified single secondary spindle afferent axons (Fu & Schomburg, 1974). Focal synaptic field potentials could also be recorded at these locations following electrical stimulation of group II afferents in the gastrocnemius and soleus nerves (Fu, Santini & Schomburg, 1974) reflecting monosynaptic actions of group II afferents on their target neurones (see Eccles, Fatt, Landgren & Winsbury, 1954; Coombs, Curtis & Landgren, 1956; see also Foreman, Kenshalo, Schmidt & Willis, 1979, in the monkey). However, these field potentials were small, and were superimposed on much larger field potentials evoked by group I afferents, whether they are evoked in the ventral horn or in the dorsal horn. This suggests that group II afferents either have relatively weak actions on neurones in the caudal lumbar segments, as has been demonstrated for motoneurones (Stauffer *et al.* 1976; Lüscher *et al.* 1979; Munson *et al.* 1980), or that the neurones they influence are distributed over a wide area and are mixed with other neurones.

We now demonstrate that much larger field potentials are evoked more rostrally, in the 3rd, 4th and 5th lumbar segments (L3, L4 and L5), by group II afferents in many hind-limb muscle nerves. Furthermore, these potentials are largely free from 'contamination' by group I field potentials, indicating that the middle lumbar segments are to a greater extent specialized for processing information from group II afferents. An essential contribution of group II muscle spindle afferents to these potentials is suggested by the fact that they are evoked by small muscle stretches as well as by electrical stimuli. A preliminary report of some of these results has been published (Edgley & Jankowska, 1985).

METHODS

Preparation

The observations described in this report were made during experiments on twenty-five cats weighing between 2·5 and 4 kg, which were also used for other experimental series (Edgley & Jankowska, 1986, 1987; Edgley & Gallimore, 1986; S. A. Edgley, E. Jankowska & S. Shefchyk, in preparation). In all cases the initial surgical procedures were carried out under ether anaesthesia, after which anaesthesia was maintained using chloralose (60–80 mg/kg initial dose, with supplementary doses of 20 mg/kg to a total dose of 5–7 mg/(kg h) in twenty-three experiments. In the other two experiments the cats were decerebrated at a pre-collicular level after which anaesthesia was discontinued. No differences between the results obtained in the two preparations have been found.

During the preliminary surgery, most of the nerves of the left hind-limb were transected and dissected free. Of these, nerve branches to quadriceps (q.), sartorius (sart.), gracilis (grac.) and adductor femoris (add.) were mounted in tunnel electrodes. In two experiments the quadriceps nerve was further subdivided into the nerves to vastus lateralis, vastus medialis and rectus femoris which were stimulated individually. Branches of the sciatic nerve were dissected free for stimulation via pairs of silver wire electrodes in a paraffin oil pool. The muscle nerves most commonly used were: posterior biceps and semitendinosus (p.b.s.t.); anterior biceps and semimembranosus (a.b.s.m.); the pretibial flexor muscles, tibialis anterior (t.a.) and extensor digitorum longus (e.d.l.) which were usually stimulated together; lateral gastrocnemius, medial gastrocnemius and soleus (g.s.); plantaris (pl.); and the nerve to the two heads of flexor digitorum longus (f.d.l.), tibialis posterior (t.p.), popliteus (popl.) and the interosseus (io.) nerve all of which were stimulated together as one nerve (f.d.l. *et al.*) in twelve experiments and separately in seven experiments. The cutaneous nerves routinely tested were the sural (sur.), superficial peroneal (s.p.) and saphenous (saph.) nerves. In fifteen experiments the posterior knee joint (joint) nerve was also tested.

A laminectomy was made to expose the spinal cord from the second lumbar to the sacral segments and at a low thoracic level. The dura was left intact to increase the stability of recording (see Eide, Fedina, Jansen, Lundberg & Vyklicki, 1969); only small holes were made in the dura and the pia to allow insertion of the recording and stimulating micro-electrodes. In seven experiments the cord was transected at a low thoracic level. The results from these experiments did not differ from those from experiments where the spinal cord was not lesioned.

During recordings the cats were paralysed with gallamine triethiodide (Flaxedil, May and Baker; about 3 mg/(kg h)) and artificially ventilated. The blood pressure and the end-tidal P_{CO_2} were kept above 90 mmHg and at about 4 % respectively. A bicarbonate solution (100 mM-$NaHCO_3$ solution with 5 % glucose) was infused intravenously, throughout both the dissection and the experiment, at a rate of 1–2 ml/(kg h) (Haglund & Lundgren, 1972) and urine was collected via an in-dwelling catheter. Occasional tests have shown that under these conditions both the arterial pH and P_{CO_2} were within, or close to, physiological values, even after many hours of anaesthesia and artificial ventilation; only exceptional cats of this series showed heart failure or a drop in blood pressure. The volume of urine collected roughly corresponded to that of the infused fluid.

Recording and stimulation

Field potential recordings were made using micropipettes filled with 1 or 2 M-KCl or 2 M-NaCl which were broken to tip diameters of 1–2·5 μm and had impedances of 1–5 MΩ. These recordings were made simultaneously with recordings from a silver ball electrode on the cord dorsum, which was used to deterime the timing and size of volleys in peripheral afferents as they reached the spinal cord. These volleys were taken from the middle of the 5th lumbar segment. Single-sweep recordings were used routinely, so that only field potentials which exceeded 50 μV could be clearly delineated; any potentials smaller than 50 μV were disregarded. At some locations an averager (Nicolet, model 1170) was used to measure the timing of the onset of field potentials.

In three experiments the nerve to the quadriceps muscles was not transected, but was freed from the surrounding connective tissue and placed in a cuff electrode. The common tendon of the muscles was then separated from the knee joint below the patella and attached via a wire to a stretching device. In one experiment the nerve to the pretibial flexors was similarly dissected in continuity and the tibialis anterior and extensor digitorum longus muscles were stretched. Triangular stretches of up to 120 μm with rising phases of 2–3 ms duration were applied, from initial tensions of

approximately 5 N (see Fetz, Jankowska, Johannisson & Lipski, 1979; Brink, Jankowska & Skoog, 1984).

Intraspinal electrical stimuli were delivered at some locations, in order to antidromically activate peripheral afferent fibres. Either glass or tungsten-in-glass electrodes were used; these could pass current pulses of up to 25 or 50 μA respectively. In these cases recordings were taken from fine peripheral-nerve filaments.

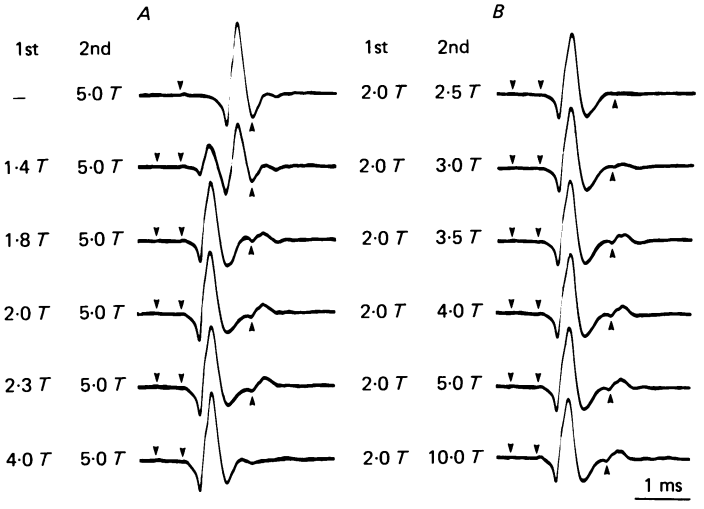

Fig. 1. Components of incoming afferent volleys attributable to group II afferents, demonstrated by a double-volley technique. The records show volleys recorded at the border between the L5 and L6 segments following stimulation of the quadriceps nerve with two shocks (downward arrows). In the series of records in *A*, the second stimulus was kept supramaximal for group II afferents at 5 *T* and was preceded by another, 0·5 ms earlier, of increasing strengths. Late components of the volleys evoked by the second stimulus (upward arrows) became clearly visible when the first stimulus was sufficiently large to maximally activate group I afferents (2 and 2·3 *T*), thereby making them refractory at the time of the second stimulus. Note that when the first stimulus was increased to activate a majority of group II afferents (4 *T*, bottom trace) the late volleys disappeared. The series in *B* illustrates the threshold characteristics of the late volleys. The first stimulus was kept constant at maximum for group I afferents. Late incoming volleys were evoked when the second stimulus was above 3 *T*. They were maximal at 5 *T*, with no additional growth when the second stimulus was increased to 10 *T*. In this and the following Figures stimulus intensities are expressed in multiples of the threshold for the most excitable fibres in the nerve. In surface recordings negativity is upward and in micro-electrode recordings it is downward.

The locations of recording and stimulating electrodes were determined by leaving electrode tips in the spinal cord. The cords were fixed by perfusion or immersion and subsequently 100 μm sections were cut in the plane of the electrode tracks.

Activation of group II afferents by electrical stimulation

It is not possible to selectively activate muscle afferents in the group II conduction velocity range using conventional electrical stimulation without also activating group I afferents unless transmission in group I afferents is blocked, which is not a straightforward procedure. Furthermore, there is considerable overlap between the electrical thresholds of the least-excitable fibres in the group I range and the most-excitable fibres in the group II range: the former may require stimuli more than twice threshold for the most excitable fibres (*T*), especially in large nerves, whereas the latter begin to be recruited at 1·5–1·6 *T* (Matthews, 1972; Fu *et al.* 1974; Jack, 1978; Lundberg, Malmgren & Schomburg, 1986*a*). Any effects evoked by electrical stimulation of nerves in the region

of overlap may thus not be assigned to group I or group II afferents with certainty. However, effects appearing with stimuli between 2·5 and 5 *T* should be safely attributed to afferents in the group II range (Eccles & Lundberg, 1959; Ellaway, Murphy & Tripathi, 1982; Lundberg *et al.* 1986*a*, *b*) and the conclusions of this study are based on the use of such stimuli.

Another major problem with the investigation of the actions of group II afferents is the determination of their central latencies and therefore their possible synaptic linkage (Fu *et al.* 1974; Lundberg *et al.* 1986*a*). One approach to this problem is to estimate the time of arrival of afferent volleys in the fastest-conducting group II afferents from the lengths of individual nerves, assuming that the conduction velocity of these afferents is 72 m/s or less. We have also used direct recordings of volleys in group II afferents at the dorsal root entry zone. These are superimposed on much larger group I volleys but distinct new components of these volleys can sometimes be seen with stimuli sufficient to activate group II afferents (Figs. 2 and 4, arrows). Furthermore, these volleys can be recorded separately by making the group I afferents refractory at the time of the stimulus, in a manner analogous to that used to investigate the I a and I b components of group I volleys (Bradley & Eccles, 1953). The results of this procedure are illustrated in Fig. 1 for volleys evoked from the quadriceps nerve. Stimulation of the nerve at 5 *T* evoked a maximal group I volley with some small late components (Fig. 1 *A*, top). When this stimulus was preceded by another which was maximal for group I afferents (2·0–2·3 *T*), but submaximal for the majority of afferents in the group II range, the late incoming volley appeared in isolation (upward arrows). This volley represents activity in all but the most excitable afferents in the group II range, since those with thresholds which overlap with the thresholds of group I afferents will be refractory at this time. Fig. 1 *B* shows that in the illustrated case it only appeared with stimuli which exceeded 2·5 *T* and was maximal at 4–5 *T*. These thresholds are in agreement with the previously found values for group II afferents (see above). Distinct group II volleys could be found for the nerves to quadriceps, sartorius and the pretibial flexors and their latencies are given in Table 1 (last column). It should be noted that these latencies fully agree with the expected latencies of group II volleys: e.g. for quadriceps afferents they were 1·2–1·85 ms and since the conduction distance was approximately 100 mm, activity in fibres conducting at 72 m/s would reach the spinal cord about 1·4 ms after the stimulus.

RESULTS

Examples of field potentials evoked by graded stimulation of the quadriceps and pretibial flexors nerves are shown in Fig. 2. It will be noted that they appeared above threshold for group II afferents, between 2 and 2·5 *T*, in parallel with an additional component of the incoming volleys (arrows, see Methods). At these stimulus strengths the volleys in group I afferents were maximal or near-maximal, as can be seen from records in the right-hand column of Fig. 2. At least a major component of these potentials was therefore evoked by afferents in the group II range. When field potentials were evoked from thigh muscle nerves (quadriceps or posterior biceps–semitendinosus) recordings from the cord dorsum often showed a separation of the group I afferent volley into I a and I b components (Bradley & Eccles, 1953). In these cases the longer-latency field potentials did not appear until the I b component was well developed (Fig. 2). If group I afferents do contribute to these field potentials at all, their contribution must thus be small and be primarily from the highest threshold group I b afferents.

At some locations field potentials were also evoked by stimuli subthreshold for group II afferents and had shorter latencies (Fig. 2, early potentials at the depth of 2·45 mm). In these cases the early component of the field potentials grew to maximum in parallel with the first component of the incoming volleys and could therefore be classified as of group I origin.

Distribution of field potentials in the transverse plane

The distribution of the field potentials evoked by group I and II afferents in various parts of the grey matter was investigated by making serial electrode penetrations in the transverse plane. Fig. 3 shows that the largest negative field potentials evoked

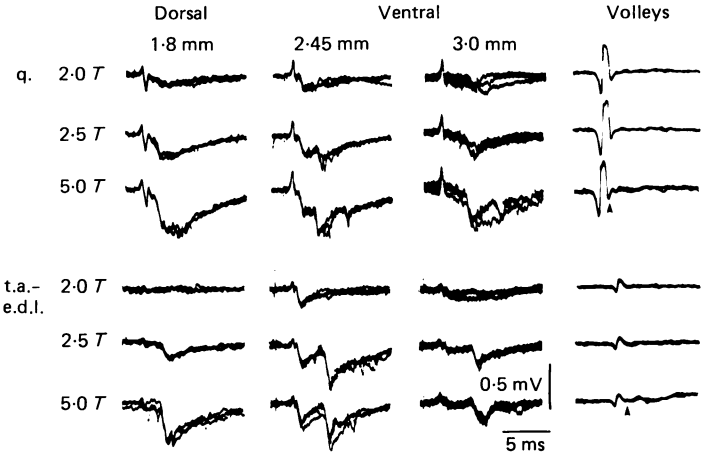

Fig. 2. Field potentials evoked by group II afferents in the quadriceps and deep peroneal nerves at three different locations in the grey matter of the L4 segment. Field potentials evoked by stimuli of increasing intensity are shown in the three left-hand columns and the corresponding incoming volleys are in the right-hand column. Note that the field potentials evoked by group II afferents appear with stimuli of 2 *T* to the quadriceps nerve (q.) and above 2 *T* for the pretibial flexor nerve (t.a.–e.d.l.) and that at a depth of 2·45 mm they are preceded by field potentials evoked by group I afferents. The incoming group II afferent volleys are marked in the right-hand column by arrows. These just appear at 2·5 *T* and are clearly visible at 5 *T*. The voltage calibration is only for the micro-electrode records.

by group II afferents were found at two locations: in the dorsal horn and deeper in the intermediate zone and ventral horn. These will be referred to as *dorsal* and *ventral* group II field potentials, respectively. Field potentials evoked by group I afferents were found in the intermediate zone and close to Clarke's column.

Dorsal field potentials. Although field potentials could be found throughout much of the dorsal horn, they appeared to have clear foci in laminae IV and V where they often had amplitudes of 0·5–1·0 mV (Fig. 2, depth 1·8 mm; Fig. 3, depth 1·4 mm; Fig. 9*B*). As the electrode was withdrawn from a focus the potentials declined and disappeared. However, in some penetrations which passed through the superficial laminae of the dorsal horn, dorsolateral to the focus, a reversed potential (positivity) was seen which had the same latency and threshold characteristics as the large negative field potential (Fig. 3, depth 0·8–1·2 mm). Such positivities were never seen in recordings taken from locations directly dorsal or dorso-medial to the foci, or in the dorsal columns. Below the foci the size of the field potentials declined rapidly and the onset became asynchronous.

Large negative potentials were also seen in recordings taken from the surface of the spinal cord in the middle lumbar segments (Fig. 3, surface). Fig. 4 shows that these potentials had thresholds and latencies similar to those of the intraspinally

recorded dorsal field potentials, suggesting that they reflect the same events. It will be noted that the positive field potentials recorded just above the foci of the negative field potentials had a much shorter time-to-peak and declined much faster than either the negative field potentials or their surface counterparts. We have not tried to relate these positive potentials to any particular pre- or post-synaptic group II actions but

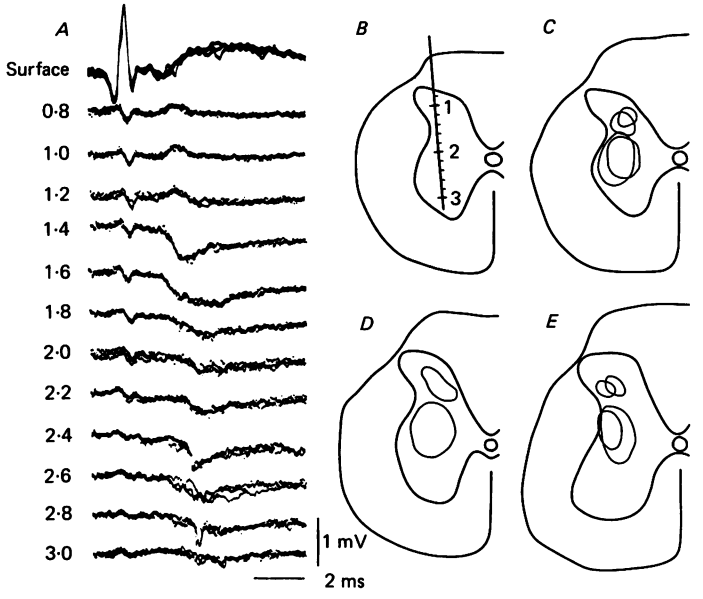

Fig. 3. Distribution of group II field potentials in the transverse plane. *A*, field potentials evoked by stimulation of the quadriceps nerve at different depths (in millimetres) along an electrode track shown in *B* and the corresponding cord-dorsum potential taken from the surface of the spinal cord. *C*, *D* and *E* show regions where the dorsal field potentials exceeded 250 μV and where the ventral field potentials exceeded 200 μV, at two planes close to the border between the L3 and L4 segments, in the middle of the L4 segment, and in two planes at the border between the L4 and L5 segments. Data in *C–E* are based on records taken along serial electrode penetrations, 100 or 200 μm apart, covering the whole width of the grey matter. The voltage calibration is for the micro-electrode recordings.

they could represent the difference between simultaneously developing faster-rising reversed positive (source) potentials and slower-rising negative cord-dorsum potentials. An analysis of the mechanism of the latter has likewise been beyond the scope of this study, but they might be analogous to the negative cord-dorsum potentials evoked in parallel with large laminae III–IV negative field potentials by low-threshold cutaneous afferents (see Bernhard, 1953; Coombs *et al.* 1956).

Intermediate zone and ventral horn field potentials. Field potentials evoked by group II afferents seen ventral to the dorsal horn (Fig. 2, depths 2·45 and 3 mm; Fig. 3, depths 1·8–2·8 mm; Fig. 9*E*) were always smaller than the dorsal ones and only exceptionally exceeded 200 μV. In addition, they were often of similar amplitude throughout laminae VII and VIII and lacked clear foci. Field potentials evoked by group I afferents were found only at some locations in laminae VI and VII (Fig. 2, depth 2·45 mm; Fig. 9*E*).

Distribution of field potentials in the longitudinal plane

The large dorsal group II field potentials were found in a region extending from the mid L5 segment caudally to the mid L3 segment rostrally. Caudal to this region the dominating field potentials, particularly in laminae V and VI, were evoked from

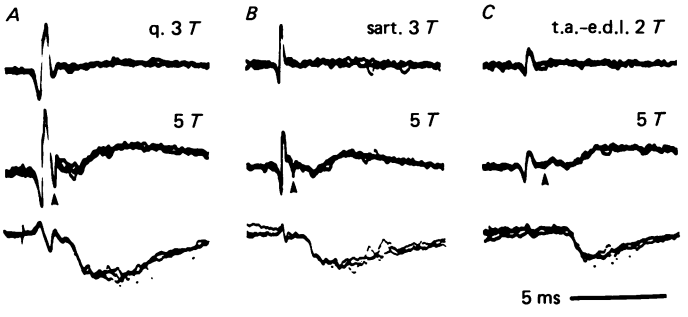

Fig. 4. Comparison of cord-dorsum and dorsal field potentials. The two top records in each row are from the surface of the spinal cord in the L4 segment (negativity upwards) while the bottom ones are field potentials (negativity downwards) at the depth 1·3 mm from the surface. These were evoked by stimuli just at threshold (3 or 2 *T*) and maximal (5 *T*) for group II afferents of quadriceps, sartorius and pretibial flexors. The arrows show group II components of incoming volleys. Note similar latencies of the cord-dorsum and the dorsal field potentials from these volleys.

group I afferents. Increasing the stimulus strength to activate group II afferents produced only small additions to the group I field potentials at these locations, as has been described for the 7th lumbar and 1st sacral segments (Fu *et al.* 1974). Rostral to the middle of the L3 segment the only obvious field potentials were evoked from group I afferents and were in the region of Clarke's column.

There was evidence for some somatotopic organization of the field potentials evoked from group II afferents between the middle of the L5 and L3 segments, in that different nerves evoked their most powerful effects at different locations. Fig. 5*B* and *C* show that caudally, in L5 and at the border between the L5 and L4 segments, the largest field potentials were evoked from the pretibial flexor nerves. In the caudal and middle parts of L4 segment the quadriceps nerve was most effective and more rostrally the largest potentials were from sartorius. A similar tendency was found for both dorsal and ventral field potentials and for the corresponding late components of the cord-dorsum potentials recorded at these levels (Fig. 5*A*).

Nerves which evoke group II field potentials

Dorsal field potentials. All of the nerves tested evoked group II field potentials at some locations, but certain nerves were more effective than others. The nerves to quadriceps, sartorius, pretibial flexors and flexor digitorum longus *et al.* consistently produced the largest potentials, and at many locations. The proportion of electrode penetrations in which field potentials from each nerve were found is shown in Fig. 6. The least-effective nerves were plantaris, gastrocnemius–soleus and anterior biceps–semimembranosus, each of which evoked potentials at less than 10% of the tested

locations. Furthermore, when potentials were evoked from these nerves (in the caudal part of the L4 segment) they were small.

In four series of electrode tracks the separate branches of the quadriceps nerve were tested. At all locations the largest potentials were evoked from the nerve to vastus lateralis; however, at three of the locations large potentials were also evoked by the nerve to vastus medialis. The nerve to rectus femoris evoked potentials at one of the locations. It therefore seems that the afferents responsible for the field potentials are found in all three branches of the quadriceps nerve.

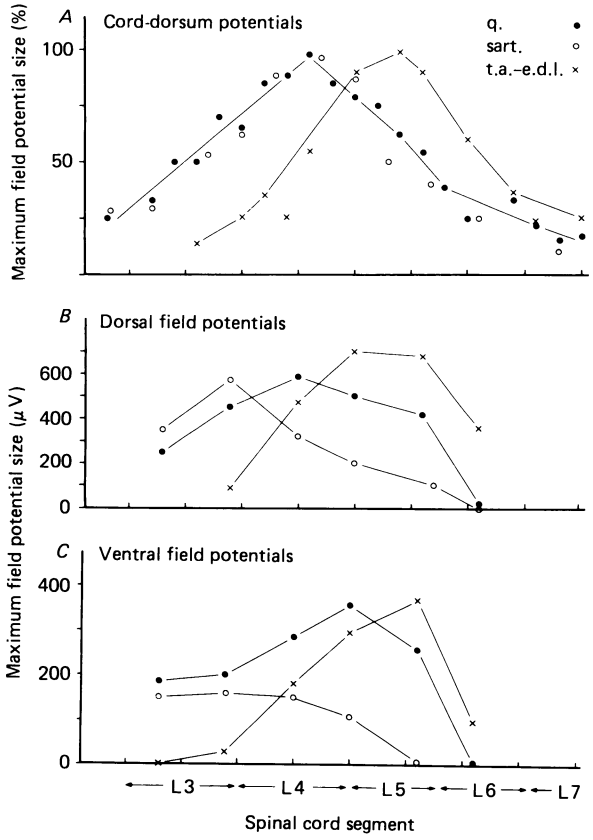

Fig. 5. Distribution of group II field potentials along the spinal cord. The graphs show the maximum amplitudes of the cord-surface (*A*), dorsal (*B*) and ventral (*C*) field potentials from recordings at different segmental levels. The data in *A* include pooled results from four different experiments, the size of the potentials being plotted as proportions of the maximum. Those in *B* and *C* are from serial electrode tracks in a single experiment. The locations of different spinal cord segments are shown on the abscissa which is to scale, the upward ticks indicating 5 mm intervals.

In a similar manner, the nerve to pretibial flexors was separated into the branches to tibialis anterior and extensor digitorum longus and these were tested separately at nine different locations. Field potentials were evoked by the tibialis anterior nerve at all of the electrode penetrations, whereas they were evoked from the extensor digitorum longus nerve at two of them.

When taken together the nerves to flexor digitorum longus, tibialis posterior and

popliteus were very effective in evoking field potentials (at *ca.* 70% of locations). However, when these nerves were stimulated individually not all were equally effective: the nerves to flexor digitorum longus proper did not evoke field potentials at any of the fourteen locations where they were tested. The interosseous nerve (which mainly innervates Pacinian corpuscles which lie in the interosseous membrane between the tibia and fibula) was tested at fourteen locations and did evoke small field potentials at three of them. It therefore seems that the most-effective nerves were those to tibialis posterior and popliteus: these were tested at four locations and field potentials were found at all of them.

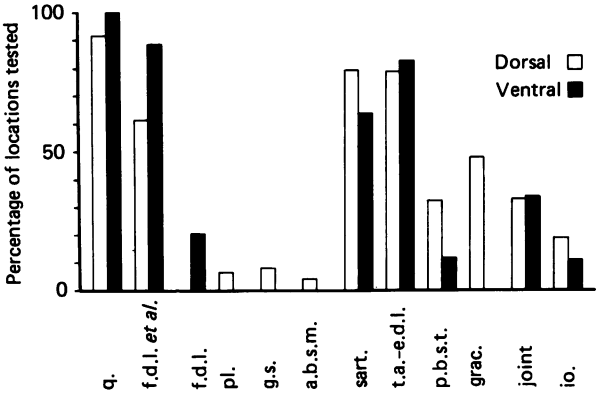

Fig. 6. The relative frequency of occurrence of field potentials from group II afferents of different nerves. The columns show the number of locations where each nerve evoked field potentials, as a proportion of the number of tracks where any nerves evoked field potentials. Open columns: dorsal field potentials; filled columns: ventral field potentials. Abbreviations: q., quadriceps; f.d.l. *et al.*, the nerves to both heads of flexor digitorum longus including interosseous, popliteus and tibialis posterior nerve branches; f.d.l., the nerves to the two heads of flexor digitorum longus alone; g.s., gastrocnemius and soleus; pl., plantaris; a.b.s.m., anterior biceps and semimembranosus; sart., sartorius; t.a.–e.d.l., the pretibial flexors tibialis anterior and extensor digitorum longus; p.b.s.t., posterior biceps and semitendinosus; grac., gracilis; joint, posterior knee joint nerve; io. interosseous nerve.

In order to determine the possible contribution of joint afferents 'contaminating' muscle nerves to the field potentials, the posterior knee joint nerve was stimulated in fifteen experiments. Of twenty-eight electrode tracks in which stimulation of the quadriceps nerve evoked large potentials, the joint nerve only evoked small potentials and only at eight locations. Furthermore, at five of these locations the largest field potentials evoked from the joint nerve were deeper than the foci of the group II field potentials and at locations where group I field potentials were encountered.

Ventral field potentials. As stated above, the group II field potentials in laminae VII and VIII were much smaller than those seen in the dorsal horn and they were evoked from fewer nerves. However, the same nerves which had the largest and most frequent effects in the dorsal horn were most effective in these more ventral regions, as can be seen from Fig. 6. Field potentials from gastrocnemius–soleus, plantaris, anterior biceps–semimembranosus and gracilis were never encountered in laminae VII and VIII and the hamstring nerves only evoked field potentials at two (of eighteen tested) locations. The interosseous nerve evoked a small field potential in

one of ten electrode tracks. The joint nerve evoked potentials at six of sixteen tested locations.

In contrast, where group I afferents evoked field potentials (close to Clarke's column or in laminae V, VI and VII caudally) they were evoked from many nerves at any one location.

Latency and synaptic linkage of the field potentials

The sharp onset of the field potentials evoked by single stimuli of group II strength, particularly in the dorsal horn, suggests that they reflect monosynaptic actions of the afferents. Further support for this possibility comes from the observation that the field potentials (both dorsal and ventral) followed repetitive stimuli at frequencies up to 100/s without temporal facilitation as illustrated in Fig. 7, and from the following analysis of their latencies.

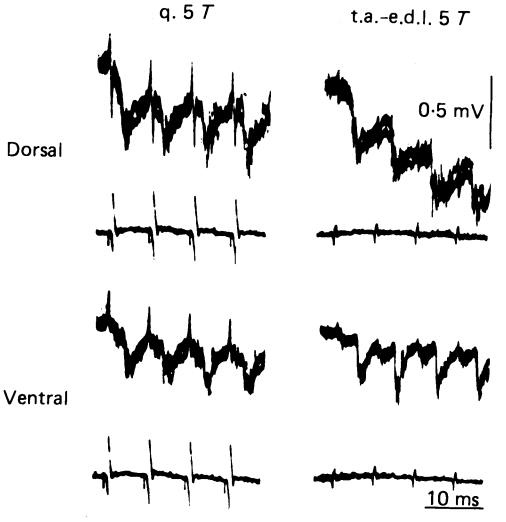

Fig. 7. Frequency-following characteristic of group II field potentials. Dorsal (upper) and ventral (lower) field potentials evoked by repetitive stimulation of the quadriceps and pretibial flexor nerves at 100 Hz are illustrated (top traces). Lower traces are the corresponding incoming volleys. Note that the field potentials followed the stimuli without temporal facilitation.

The central latencies of the onsets of field potentials evoked from branches of the femoral nerve (quadriceps and sartorius), the pretibial flexor nerves, and muscle branches of the tibial nerve in different tracks are plotted in Fig. 8. Where available, the latencies have been measured from the group II component of the incoming volleys (see Methods). These values are plotted in Fig. 8*A* and *B* for dorsal and ventral field potentials respectively. Latencies of field potentials from experiments where clear group II components of the incoming volleys could not be found (and from nerves which did not produce clear group II volleys) have been measured with respect to the onset of the group I afferent volley and are included in Fig. 8*C–F*. The mean latencies are given in Table 1.

Dorsal field potentials. Those evoked from the quadriceps, sartorius and from the pretibial flexor nerves usually had latencies less than 1 ms with respect to the group

II volleys. These allow for only a single synapse and therefore indicate that at least the earliest components of the field potentials are generated monosynaptically. In fact, some of these latencies are over-estimates, since they rely on measurements of the arrival of the action potentials in group II afferents at the L5 segment and the field potentials were recorded some distance rostrally. The field potentials evoked from other nerves, and in the experiments where group II volleys were not seen, also had latencies compatible with their being evoked monosynaptically. Field potentials

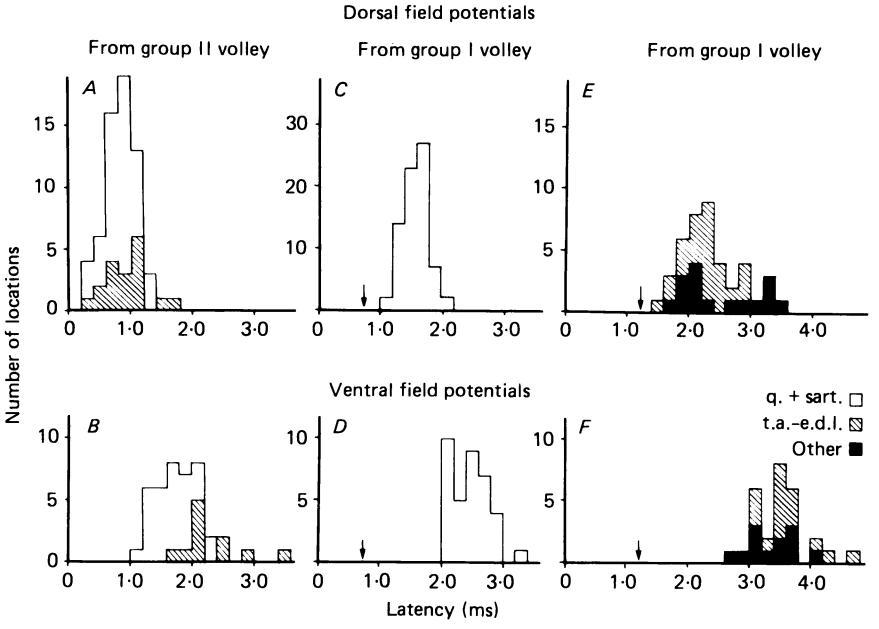

Fig. 8. Latencies of group II field potentials. *A* and *B*, respectively, show the latencies of dorsal and ventral field potentials from incoming volleys in group II afferents. Open areas are for field potentials evoked from quadriceps or sartorius; hatched areas are for field potentials evoked from the pretibial flexors. *C–F* give the latencies of group II field potentials from group I incoming volleys. *C* and *D* show the latencies of dorsal and ventral group II field potentials, respectively, from the quadriceps and sartorius nerves. The arrows indicate the expected time of arrival of volleys in group II afferents (means derived from the data in Table 1). *E* and *F* show field potentials evoked from the pretibial flexor nerve (hatched areas) and from branches of the common tibial nerve which were stimulated distal to the popliteal fossa (filled areas). The arrows mark the expected time of arrival of volleys in group II afferents of the pretibial flexor nerve, as derived from the data in Table 1.

from quadriceps or sartorius had latencies from the group I volley of 1·0–2·1 ms (Fig. 8*C*). Considering that the group II volleys arrived at the L5 segment 0·5–1·0 ms after the group I volleys, it may be concluded that these field potentials were also monosynaptically generated. The latencies of field potentials from the pretibial flexor nerves (taken from the group I volleys) were longer than those from quadriceps (1·5–2·9 ms; see Table 1 and Fig. 8*E*), in agreement with the greater separation between the arrival of activity in group I and group II afferents expected for longer nerves. However, since the group II volleys from these nerves reached the spinal cord 1·0–1·7 ms later than the group I volleys, in many cases the remaining time permits only a single synapse. No latencies for the arrival of nerve impulses in group II

afferents in the spinal cord following stimulation of the branches of the tibial nerve below the popliteal fossa were obtained. However, whenever field potentials were evoked from those nerves, they had latencies directly comparable to those evoked from the nerve to the pretibial flexors, which is of a similar length (Fig. 8*E*). The synaptic linkage of these field potentials is therefore also likely to be monosynaptic.

TABLE 1. Latencies of field potentials in the L4 segment

	Dorsal group II			Ventral group II			Group I			Shock-volley		
Nerve	*n*	Mean	(S.D.)	*n*	Mean	(S.D.)	*n*	Mean	(S.D.)	*n*	Mean	(S.D.)
					From group II volleys							
q.	37	0·77	(0·24)	23	1·65	(0·30)		—		29	1·44	(0·19)
sart.	8	0·81	(0·24)	5	2·0	(0·20)		—		5	1·52	(0·22)
t.a.–e.d.l.	18	0·87	(0·35)	11	2·38	(0·49)		—		11	3·19	(0·18)
					From group I volleys							
q., sart.	69	1·54	(0·19)	35	2·46	(0·31)	24	0·68	(0·13)	72	0·79	(0·11)
p.b.s.t., a.b.s.m.	11	1·97	(0·52)	2	3·6	(—)	4	0·63	(0·11)	11	1·15	(0·05)
g.s., pl., f.d.l. *et al.*	14	2·30	(0·54)	9	3·29	(0·42)	15	0·67	(0·20)	13	1·82	(0·16)
t.a.–e.d.l.	22	2·31	(0·30)	17	3·57	(0·40)	14	0·62	(0·10)	56	1·89	(0·15)

Latencies of field potentials in the L4 segment. The first column indicates the origin of the field potentials, with abbreviations as in Fig. 6. Columns 2–4 give the number of electrode tracks in which each type of field potential was encountered (*n*) and their mean latencies in ms, with standard deviations in parentheses. Latencies of field potentials from the incoming volleys in group II afferents are in the three top rows; the latencies of these volleys from the stimulus are given in the last column. Latencies of field potentials from incoming volleys in group I afferents are in the lower four rows; the latencies of these volleys from the stimulus are given in the last column.

Ventral field potentials. These had considerably longer latencies than the dorsal field potentials, usually by about 1 ms (see Table 1). Their latencies from the arrival of the group II afferent volleys were greater than 1·1 ms. Theoretically, these would permit an additional interneurone in the pathway and might suggest that the minimal linkage is disynaptic. However, as will be shown, these potentials may also be generated monosynaptically, the long latency being caused by a dramatic slowing of conduction velocity in the terminal branches of the afferents.

The latencies of the group I field potentials were similar for all nerves: since they were measured from the arrival of the group I volley, this was expected. These were 0·62–0·68 ms (Table 1) and comprise a brief conduction delay from the cord-dorsum electrode to the recording site and a single synaptic delay. It will be noted that these latencies are comparable to the latencies of the group II field potentials from the group II incoming volleys.

The latencies of field potentials evoked from the joint and interosseous nerves usually had similar latencies to the group II field potentials evoked from nerves of similar length. It appears therefore that afferents of approximately group II conduction velocities were responsible for some of the field potentials. However, at some locations field potentials were found with much shorter latencies (less than 1 ms) which were comparable with latencies of group I field potentials.

To test the possibility of a direct projection of group II afferents to the grey matter

of the middle lumbar segments, we have peformed intraspinal stimulation in electrode tracks where the largest field potentials were found, while recording from peripheral nerves. Results from such an experiment are illustrated in Fig. 9*A* and *D* with recordings from small filaments of the nerves to tibialis anterior or extensor digitorum longus. Proximal to the filaments the nerves were mounted together on silver wire

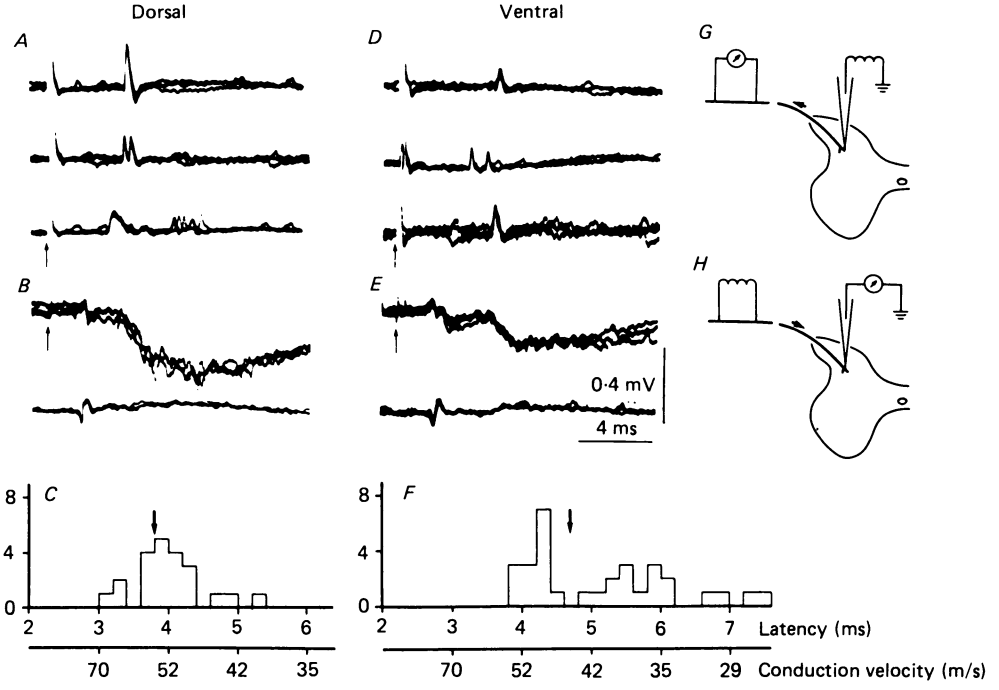

Fig. 9. Comparison of the latencies of field potentials and of peripheral nerve volleys evoked by intraspinal stimuli. *A*, action potentials in sensory fibres of the t.a. or e.d.l. nerves antidromically activated by intraspinal stimulation at the location of the dorsal field potential shown in *B*. *C* is a histogram of the latencies of responses of all of the fibres antidromically activated from the region of dorsal field potentials which had thresholds above 2 *T*. The downward arrow indicates the latency of the dorsal field potentials. The upper abscissa gives latencies from the stimuli and the lower one gives an estimate of the corresponding conduction velocities. *D* shows responses of fibres antidromically activated from the region of the ventral field potential illustrated in *E*. The histogram in *F* is as the one in *C*. The experimental arrangement for *A* and *D* is shown in *G*, and that for *B* and *E* is shown in *H*.

electrodes to allow stimulation. In this manner, the threshold of the fibres activated antidromically by intraspinal stimulation could be determined by collision with activity in fibres excited by different stimulus intensities (Eccles, Schmidt & Willis, 1963; Lundberg *et al.* 1986*a*). Relatively large stimuli (more than 20 μA) were often required to activate the afferent terminals, either because of their small size or because of a non-optimum location for the stimulating electrode.

In the region of the dorsal field potentials, intraspinal stimulation antidromically activated many fibres, at latencies from 3·1 ms (Fig. 9*A* and *C*), which were not collided by stimuli below 2 *T*. Field potentials evoked at this location by stimulation of the whole pretibial flexor nerve (Fig. 9*B*) had onsets 3·8 ms from the stimulus, i.e.

0·7 ms longer, which would permit only a single synaptic delay. An estimate of the conduction velocities of the fibres is given on the abscissa; these fall within the group II range.

Stimuli applied more ventrally activated afferent fibres with longer latencies (from 3·8 ms, see Fig. 9*D* and *F*) but the latencies of the ventral group II field potentials (Fig. 9*E*) were also longer (onset 4·7 ms from the stimulus). Furthermore,

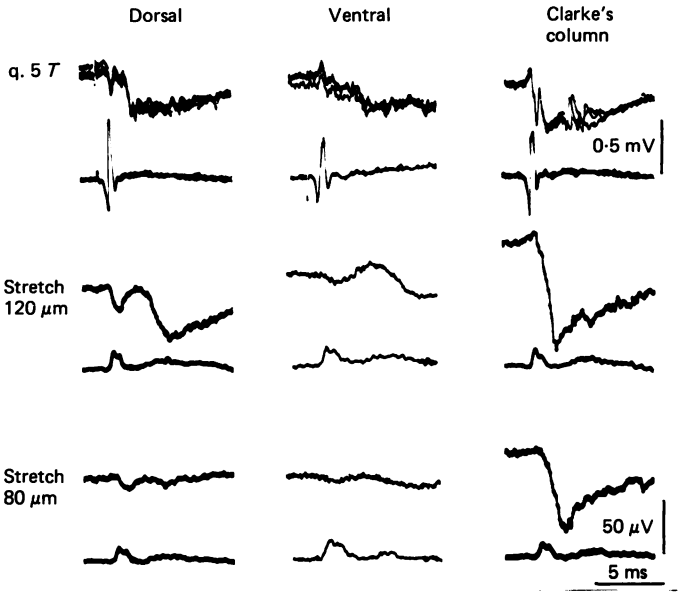

Fig. 10. Field potentials evoked by stretch of the quadriceps muscles. The top row illustrates dorsal and ventral group II field potentials, together with a group I field potential recorded in Clarke's column, evoked by electrical stimulation of the quadriceps nerve at 5 *T*. The lower two rows are averaged field potentials evoked by 120 and 80 μm stretches of the quadriceps muscles, recorded at the same locations. Note that only stretches greater than 80 μm evoked field potentials in the region of the electrically evoked dorsal and ventral group II field potentials, whereas smaller stretches evoked large field potentials in Clarke's column. The lower traces in all three rows are simultaneous records of incoming volleys. The lower calibration applies to all stretch-evoked field potentials.

it is probable that some of these fibres were activated via main collateral branches rather than from the terminals. Some of the latencies may therefore be underestimates. Again the antidromically activated fibres all had collision thresholds in the group II range. Many fibres with the appropriate threshold characteristics therefore project to the region of the ventral field potentials and nerve impulses evoked in them by peripheral stimuli would arrive only shortly (less than 1 ms) before the onset of the field potentials. An intervening neurone in the generation of the ventral field potentials is therefore not required.

Field potentials evoked by muscle stretch

As a preliminary analysis of the properties of the afferents responsible for the field potentials recorded in the mid-lumbar segments, we have investigated whether field potentials might be evoked by stretches of the quadriceps or pretibial flexors muscles.

In order to detect the very small potentials evoked by the stretches used, which were less than 120 μm, a number (64 or 128) of responses were averaged. Stretch-evoked field potentials were found both dorsally and ventrally (Fig. 10). In the dorsal horn field potentials evoked by stretches of the quadriceps muscles had latencies of 3·0–4·5 ms from the group I volleys (mean $3{\cdot}6 \pm 0{\cdot}45$ ms, $n = 10$) while those in the ventral horn had latencies ranging from 4·5 to 6·0 ms (mean $5{\cdot}1 \pm 0{\cdot}63$ ms, $n = 5$). These are considerably longer than the latencies of field potentials evoked by electrical stimulation. One reason for this is the additional conduction distance from afferent terminals in the muscle to the point where the electrical stimuli were delivered (where the femoral nerve enters the ilipsoas muscle), which was at least 50 mm, and for distantly located receptors could exceed 100 mm. By itself this could account for an additional delay of up to 1 ms. Less-effective activation of the receptors is likely to be another factor, as the amplitudes of the field potentials were small. At their largest the field potentials evoked by stretch had amplitudes of 80 μV, compared to 1 mV or more following electrical stimulation. Stretches of less than 80 μm did not evoke field potentials. Thus it seems unlikely that the largest stretches used, which were about 120 μm, would activate more than a small fraction of the afferents which evoke the field potentials. Field potentials evoked by stretches of pretibial flexors appeared with similar latencies but with less-sharp onsets and were therefore more difficult to measure.

The effectiveness of stretches of the quadriceps tendon was also tested by recording in the region of Clarke's column, where electrical stimulation of the quadriceps nerve evoked large group I field potentials. At this location 120 μm stretches evoked much larger field potentials which only decreased slightly in amplitude when the stretch was reduced to 80 μm (Fig. 10). These field potentials had latencies of 0·5–1·5 ms from the cord-dorsum volleys.

DISCUSSION

The classification of the field potentials analysed in this study as of group II origin is based primarily on observations of the effects of finely graded electrical stimulation. An absolute differentiation between group I and group II afferents cannot be made using this method because of the overlap in their thresholds (Matthews, 1972; Jack, 1978; see Methods). However, since the field potentials we have described appeared and/or grew most as the stimulus was increased from 2·5 to 5 T, we may conclude that the largest contribution to them is from group II afferents, even though we cannot exclude a contribution from the highest-threshold group I afferents. Furthermore, the long latency of the field potential onsets precludes a large monosynaptic contribution from afferents with a conduction velocity in excess of 70 m/s, whereas, both in the regions of the dorsal and ventral field potentials, we have strong evidence for a direct projection of afferents with lower conduction velocities.

Distribution of field potentials

Fu *et al.* (1974) found monosynaptic field potentials evoked by group II afferents at two locations in the L6 and L7 segments: one dorsally and the other ventrally. They also described longer-latency potentials, in the ventral part of the grey matter (the late field potentials). The field potentials which we have found are qualitatively

similar to their dorsal and ventral field potentials. Field potentials resembling their late potentials were seen infrequently and were not systematically analysed. In the dorsal horn, the field potentials found by Fu *et al.* (1974) were largest in laminae IV and V, where they reached amplitudes of 100 μV. Those in the mid-lumbar segments have a similar distribution but are much larger, sometimes exceeding 1 mV. At more ventral locations, Fu *et al.* (1974) found field potentials evoked by group II afferents at restricted locations within lamina VII. Again the field potentials in the mid-lumbar segments are larger and much more widespread. These locations are in good agreement with the morphology of secondary muscle spindle afferents in the L6 and L7 segments (Fyffe, 1979; T. Hongo, personal communication). The collaterals of the investigated afferents branched and terminated extensively in laminae IV and V and also had deep branches which terminated in lamina VII and among the motoneurones. Since profuse collateral branching was found in the L6 and L7 segments, it is difficult to understand why only small field potentials (Fu *et al.* 1974) and so few interneurones with direct group II input (Hongo, Jankowska & Lundberg, 1966; Harrison & Jankowska, 1985; Lundberg *et al.* 1986*b*) have been found in them. Collaterals of group II afferents in the mid-lumbar segments have not been investigated morphologically, but on the basis of our findings an even heavier projection to these same parts of the grey matter would be predicted.

In Fu *et al.*'s (1974) investigation severe problems were encountered because of the presence of large field potentials evoked by group I afferents. Throughout most of the grey matter of the mid-lumbar segments this is not a problem since group I afferents evoke no field potentials in the dorsal horn (except close to Clarke's column) and only occasional small field potentials in the ventral horn. This is in excellent agreement with the morphology of group I afferent collaterals in the mid-lumbar segments: these enter the grey matter through the ventromedial border of the dorsal columns and ramify extensively in the region of Clarke's column, but project only sparsely to other parts of the grey matter (T. Hongo, personal communication).

Longitudinally, the group II field potentials seem to be organized in parallel with the motoneurone pools but to be shifted rostrally by approximately one and a half segments. The pretibial flexor motoneurones are located in L7 and the caudal part of the L6 segment but group II afferents of these muscles evoke large field potentials in the rostral part of the L5 segment. Quadriceps motoneurones are in the L5 and L6 segments, but stimulation of the quadriceps nerve evokes the largest field potentials in the L4 segment. Finally, sartorius motoneurones are in the L5 segment but the sartorius nerve evokes the largest field potentials in the rostral part of the L4 and L3 segments. Since both the dorsal and ventral field potentials followed this pattern, neurones at different segmental levels may be specialized to process information from somewhat different receptive fields.

Synaptic linkage of the field potentials

The properties of the dorsal group II field potentials are fully compatible with a monosynaptic linkage. Moreover, their segmental latencies are usually less than 1·0 ms and therefore do not allow an intervening neurone. A disynaptic linkage is theoretically possible for the ventral field potentials, since they have segmental latencies which are greater than 1·1 ms. However, similar differences in latency were

also found by Fu *et al.* (1974) in the caudal segments where the ventral field potentials can be concluded to be at least in part monosynaptically generated. This conclusion is based on the demonstration of a direct projection of group II afferents to the region of the ventral field potentials, and on the finding that the conduction velocity in fine collateral branches rapidly falls, so that the intraspinal conduction time can amount to well over 1 ms (Fu & Schomburg, 1974; Lundberg *et al.* 1986*a*). The monosynaptic excitation of motoneurones by group II afferents also requires a direct projection of these afferents to the ventral parts of the grey matter. The segmental delays of the motoneuronal e.p.s.p.s evoked by secondary muscle spindle afferents (up to 1–2 ms; Kirkwood & Sears, 1975; Stauffer *et al.* 1976; Lüscher *et al.* 1979; Munson *et al.* 1980) were in good agreement with the intraspinal conduction times of these afferents. In the present series of experiments we have also demonstrated a direct projection of afferents with the correct activation thresholds to the region of the ventral field potentials. In many cases the conduction delays were such that activity in these afferents arrived in lamina VII less than 1·0 ms before the onset of the field potentials. We therefore extend the conclusions of Fu *et al.* (1974) that the ventral field potentials are also generated monosynaptically. In further support of this conclusion, in many neurones in the area where this ventral field potential was recorded only excitation was seen at latencies comparable to those of the field potentials, whereas the i.p.s.p.s had latencies 0·5–1·0 ms longer than the e.p.s.p.s (Edgley & Jankowska, 1987).

Possible receptor origin of the group II afferents which evoke field potentials in the mid-lumbar segments

Electrical stimulation of muscle nerves at 5 *T* will activate almost all of the afferent axons in the group II range (Eccles & Lundberg, 1959; Ellaway *et al.* 1982; Lundberg *et al.* 1986*a*). Although most of these can be assumed to originate from secondary endings of muscle spindles, other receptors may also have fibres in this range (Boyd & Davey, 1968; Matthews, 1972) and may have contributed to the field potentials we have described.

One of the most common types of 'contaminating' group II axons are joint afferents. For example, knee joint afferents can be found in the quadriceps and sartorius nerves (Freeman & Wyke, 1967). However, electrical stimulation of the posterior knee joint nerve, which always contains a large number of articular afferents, evoked only small field potentials, and at few locations. This does not exclude the involvement of articular afferents in the potentials, especially those from the hip joint, but suggests that their contribution is not large. Moreover, the posterior knee joint nerve is known to contain some muscle afferents (McIntyre, Proske & Tracey, 1978), which may include secondary muscle spindle afferents and may have been responsible for its effects.

Axons innervating Pacinian corpuscles may be another type of 'contaminating' group II fibre. However, about 35% of the myelinated afferent fibres of the interosseous nerve innervate Pacinian corpuscles (Hunt & McIntyre, 1960; Boyd & Davey, 1968) and since only occasional small field potentials were evoked by stimulation of this nerve a major contribution from Pacinian corpuscles afferents to the field potentials evoked from muscle nerves is unlikely.

Other types of axon in the group II range include a small number of free endings

(Stacey, 1969; Cleland, Rymer & Edwards, 1982) and undefined mechanoreceptors (MacLennan, 1972). The latter are particularly common in the tibialis anterior nerve and may be present in other muscle nerves. They may therefore have contributed to the field potentials we have described. However, since nothing is known of their central projections and the likelihood of activation of these receptors by small muscle stretches cannot be assessed, more definite conclusions about their involvement cannot be drawn.

The properties of the group II afferents which originate from the secondary endings of muscle spindles make them the most likely source of the group II field potentials in the mid-lumbar segments. They constitute the majority of group II afferents in muscle nerves and stimuli adequate for their activation may evoke field potentials; as shown in Results, small muscle stretches were effective in evoking field potentials, even though they were not particularly large. Rapid stretches of the size used are very effective in driving the primary muscle spindle afferents, as can be seen from the large field potentials they evoke in Clarke's column (Fig. 10*D*). They are much less effective in activating the secondary endings synchronously because of the lower dynamic sensitivity of the receptors (Matthews, 1972). However, they do provoke a discharge in some (Lundberg & Winsbury, 1960; Stuart, Mosher, Gerlach & Reinking, 1970; Fetz *et al.* 1979; Brink *et al.* 1984). Furthermore, although the projection of identified secondary muscle spindle afferents to the mid-lumbar segments has not been directly investigated, the distribution of group II field potentials in these segments fits precisely with the distribution of terminal branches of muscle spindle secondary afferents in more caudal segments.

The results presented in this paper suggest that the mid-lumbar segments are to a great extent specialized to process information from group II muscle afferents. The analysis of field potentials of group II origin described in this paper has therefore been combined with studies of neurones found at the same locations, which will be reported in forthcoming papers. Results from these studies have revealed a previously unknown group of dorsal spinocerebellar tract cells with a dominant group II input at the location of the dorsal group II field potentials (see Edgley & Gallimore, 1986; Edgley & Jankowska, 1986) and several types of interneurones at the location of both the dorsal and ventral group II field potentials (Edgley & Jankowska, 1987). Of the latter some have been found to have direct excitatory or inhibitory actions upon motoneurones (Cavallari, Edgley & Jankowska, 1987). To understand the functions of these various neurones we have paid particular attention to the possibility that they are directly activated by secondary muscle spindle afferents. This is strongly indicated by the results of this study and will be further supported by intracellular records from L4 neurones.

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