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THE DORSAL COLUMN PROJECTION OF MUSCLE AFFERENT FIBRES FROM THE CAT HINDLIMB

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SUMMARY

1. The extent of the projection of hindlimb muscle afferent fibres ascending the dorsal columns has been studied in barbiturate-anaesthetized cats. This has been investigated using electrical stimulation of the dorsal columns at different spinal levels while recording from (i) peripheral muscle nerves, and (ii) single muscle afferent fibres within the dorsal columns. These two approaches have produced complementary results.

2. The conduction velocity of both group I and group II afferent fibres decreased progressively after entering the dorsal columns.

3. The majority of group I and group II fibres project at least as far as L2 but leave the dorsal columns at or before the lower thoracic segments.

4. By taking advantage of the lower electrical threshold of Ia compared to Ib fibres in the hamstring nerves, it could be shown that both Ia and Ib fibres leave the dorsal columns at similar locations.

5. A small number of afferent fibres were found to project to C1. On the basis of previous work it is likely that such fibres originate from Pacinian or paciniform corpuscles.

INTRODUCTION

There have been several detailed studies of the dorsal column projection of primary afferent fibres of cutaneous (Brown, 1968; Petit & Burgess, 1968; Horch, Burgess & Whitehorn, 1976) and of articular origin (Gardner, Latimer & Stilwell, 1949; McIntyre, 1962*a*; Burgess & Clark, 1969; Clark, 1972). However, similar investigations of the projection of muscle afferent fibres have not yet been performed. From the work of Lloyd & McIntyre (1950) it is known that some group I muscle afferent fibres from the cat hindlimb ascend the dorsal columns as far as the upper lumbar or lower thoracic segments. They observed that the group I volley recorded from the surface of the dorsal columns was reduced in amplitude as it travelled rostrally and could not be detected at mid-thoracic levels. With respect to the group II projection, two brief reports suggest that group II afferents undergo less slowing of conduction in the dorsal column compared to group I muscle afferent fibres (McIntyre, 1962*b*), and that the group II afferent fibres project beyond the group I fibres at least as far as the uppermost thoracic segments (McIntyre & Lloyd, 1948).

The aim of the study reported here was to investigate further the dorsal column projection of hindlimb muscle afferent fibres by stimulating the dorsal columns at various segmental levels and recording the antidromic activity in (a) fine filaments of muscle nerves in the periphery and (b) single muscle afferent fibres in the lumbar dorsal columns. With this combined approach we have confirmed and greatly extended the observations of Lloyd & McIntyre (1950) on the dorsal column projection of group I muscle afferent fibres and, in contrast to previous reports (McIntyre & Lloyd, 1948; McIntyre, 1962*b*), have provided evidence of a similar pattern of projection for group II fibres. An abstract of some of these results has been published (Fern, Harrison & Riddell, 1987).

METHODS

Preparation

Experiments were performed on nine cats (2·1–3·2 kg weight) anaesthetized with sodium pentobarbitone (40 mg kg^{-1}, I.P. initial dose; supplemented intravenously as required). Anaesthetic level was assessed by inspection of a continuous blood pressure recording and the diameters of the pupils of the eyes. The animals were intermittently paralysed with gallamine triethiodide and artificially respired. End-tidal CO_2 and body temperature were continuously monitored and maintained within physiological limits.

The experimental arrangement is illustrated in Fig. 1. The spinal cord was exposed by a laminectomy between L7 and Th12, and at the Th10 segment. In three experiments, an additional laminectomy was performed at C1/2. The dura was opened and seven or eight pairs of bipolar silver ball electrodes placed at various levels on the dorsal columns for stimulation; typically, at L6, L4, L3, L2, L1, Th13, Th10 and, when exposed, at the C1 level. The electrodes were positioned at the centre of each spinal segment, judged as the mid-point between the most rostral and caudal roots, and were orientated longitudinally along the mid-line of the cord with their cathodes facing caudally; their location, with respect to the spinal segments, was confirmed by post-mortem dissection.

The following muscle nerves were transected, dissected free and mounted for recording and/or stimulating on pairs of bipolar silver wire electrodes; posterior biceps–semitendinosus, anterior biceps–semimembranosus, medial gastrocnemius, lateral gastrocnemius–soleus and plantaris. A selection of the following nerves were also included for comparison: sural, superficial peroneal, interosseous and the posterior nerve to the knee joint. Some of the muscle nerves were dissected as far distally as possible in order that fine filaments could be separated and mounted individually on pairs of bipolar silver wire electrodes, which were used for recording. Where a whole nerve was divided into several filaments in this manner, the stimulating electrode was placed proximally on the whole nerve. Cord dorsum potentials were recorded through a monopolar silver ball electrode on the dorsal columns near the L7 dorsal root entry zone. This was used to monitor the effectiveness of stimuli applied to the dorsal columns and to determine the thresholds of afferent fibres in relation to the threshold of the most excitable fibres in peripheral nerves.

Recordings from peripheral nerves and nerve filaments

Recordings were made, of the antidromic volley evoked in peripheral nerves or their filaments, by stimulation of the collateral axons of muscle afferent fibres in the dorsal columns (0·1 ms duration, generally 100 μA though up to about 1 mA current strength). The proportion of fibres projecting to different segmental levels was assessed by comparing the antidromic volley evoked by stimulation at various rostrocaudal locations. The peripheral thresholds of the afferent fibres contributing to the antidromic discharges were determined by collision with orthodromic impulses evoked by stimulation of the nerve or filament. By relating the peripheral stimulus strength required to extinguish different components of the antidromic volley to threshold for the most excitable fibres in the nerve or filament, the thresholds of afferent fibres activated from the dorsal columns could be obtained.

Care was taken to ensure that the discharges recorded from the nerves, particularly those of

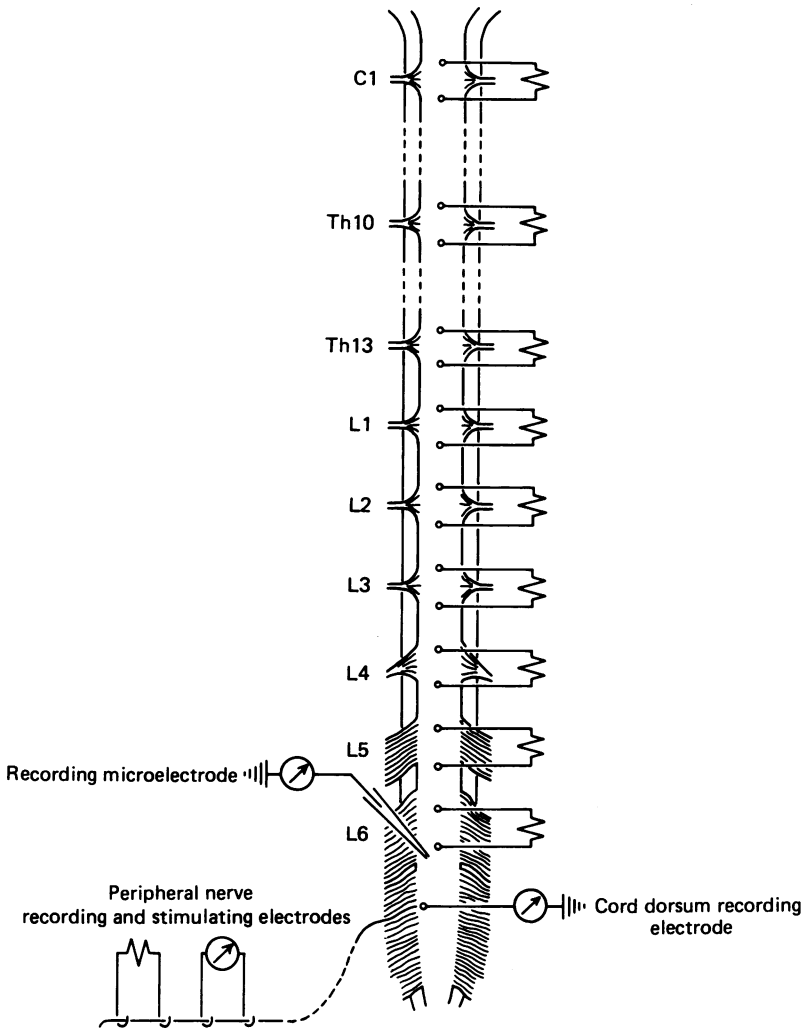

Fig. 1. Diagram of the experimental arrangement showing a plan view of the exposed spinal cord and the location of recording and stimulating electrodes. Stimulating electrodes were placed on peripheral nerves and at various segmental levels on the dorsal columns.

longer latency, were evoked by direct activation of the collateral axons of muscle afferent fibres. Such direct responses could generally be distinguished from dorsal root reflexes which were of long and variable latency and highly dependent on the rate and strength of stimulation of the cord. The L6 and L7 ventral roots were cut in the later experiments, in order to prevent any orthodromic discharges of motoneurones from reaching these nerves and hence contaminating the recordings of sensory activity.

Recordings from single afferent fibres in the dorsal columns

Search stimuli (0·1 ms duration, at least five times threshold (T)) were applied to each of the peripheral nerves in turn whilst tracking in the dorsal columns at L6 with glass micropipettes filled with 2–4 M-NaCl. Recordings were made from single muscle afferent fibres which were identified by

activation from a single muscle nerve at constant latency. These were made simultaneously with recordings from the cord dorsum electrode by which the peripheral threshold of the afferent fibre was determined. An attempt was made to antidromically activate afferent fibres by stimulation of the dorsal columns at various spinal levels. The stimulus intensity was gradually increased until threshold for an antidromic response was reached or up to an intensity of about 1 mA without response. The latencies of orthodromic and antidromic impulses were measured following stimuli of suprathreshold strength. At the end of each experiment the peripheral conduction distance was measured *in situ* by laying a thread along the exposed nerves and the peripheral conduction velocities of fibres calculated. The distance between each of the dorsal column electrodes was also measured and the conduction velocities of fibres in the dorsal columns calculated from the differences in latency between each spinal level.

RESULTS

Recordings from muscle nerves and filaments

Activation of muscle afferents from the lumbosacral cord

Stimulation of the dorsal columns at the lumbosacral enlargement evokes an antidromic discharge in the collateral axons of primary afferent fibres which can be recorded from peripheral muscle nerves or nerve filaments. The resulting antidromic volley consists of distinct early and late components which, on the basis of latency measurements and collision testing, can be shown to be associated with activity in group I and group II fibres respectively.

Latencies. The antidromic volley is composed of an early (1–2 ms), large-amplitude, synchronous compound action potential which is followed by a number of small, temporally dispersed deflections of longer latency (2–8 ms). Examples of such responses are shown in the lower records of Fig. 2. Taking into account the conduction distance, the peripheral conduction velocities of fibres contributing to the early component of the volley range from 70 to 100 m s^{-1}, whilst the activity producing the later deflections is conducted at between 20 and 70 m s^{-1}. These conduction velocity ranges are appropriate for activity in group I and group II afferent fibres respectively.

Collision test. The origins of the early and late components of the antidromic volley were confirmed by the use of a collision test. The stimulus to the dorsal columns was preceded by a stimulus to the muscle nerve, just proximal to the recording electrodes. The interval between the two stimuli was selected such that it never exceeded the conduction latency of the earliest component of the antidromic volley. Under these conditions, antidromically conducted activity would collide with orthodromic impulses before reaching the recording location. It was therefore possible, by adjusting the strength of the conditioning stimulus applied to the muscle nerve, to extinguish various components of the antidromic volley. A conditioning stimulus of $1{\cdot}4T$ was found to be sufficient to extinguish almost all of the early component of the volley whereas the later components remained virtually unaffected (Fig. 3, lower records). Since stimuli of this strength are known to be subthreshold for group II fibres in muscle nerves (Eccles & Lundberg, 1959; Jack, 1978; Ellaway, Murphy & Tripathi, 1982), most of the fibres contributing to the early component of the antidromic volley must be of group I origin while those responsible for the later deflections are mainly group II fibres.

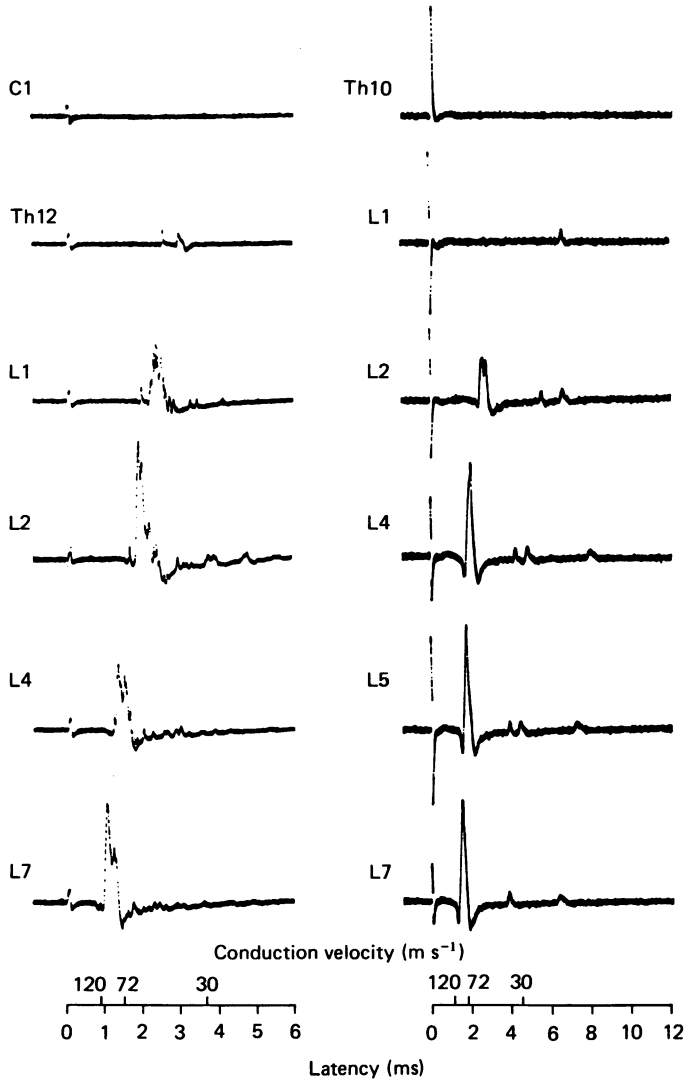

Fig. 2. Examples of antidromic activity evoked in filaments of muscle nerves by stimulation of the dorsal columns at the various segmental levels indicated. The recordings in the left-hand panel were made from a filament of the posterior biceps nerve. The recordings in the right-hand panel were made from a filament of the posterior biceps nerve in another experiment. The single units evident in the records of the right-hand panel were investigated using the collision test and their peripheral thresholds were, in order of latency, 2·4, 2·7 and 6·1*T*. Approximate conduction velocity ranges for group I (120–72 m s^{-1}) and group II fibres (72–30 m s^{-1}) are indicated but apply only to activity evoked from the L7 level. The latency scale is applicable to all records. Each of the records in this and subsequent Figures is composed of several superimposed sweeps.

Activation of muscle afferent fibres from different segmental levels

Afferent fibres supplying the hindlimb enter the spinal cord over the lumbosacral enlargement and stimulation at this level should therefore activate the majority of

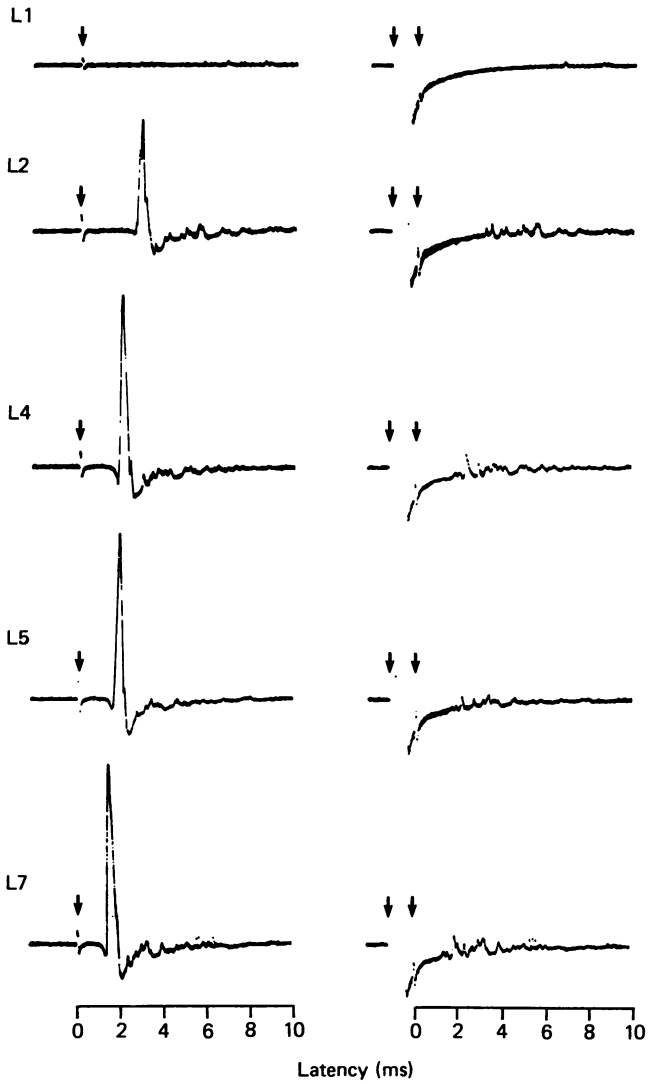

Fig. 3. Collision of the early synchronous component of the antidromic volley with impulses evoked by a conditioning stimulus to the muscle nerve at 1·4*T*, a strength subthreshold for the activation of group II fibres. The records on the left show the antidromic volleys recorded from a filament of the posterior biceps nerve following stimulation of the dorsal columns at the various spinal levels indicated. The records on the right show the result of colliding the antidromic volley with activity evoked by preceding the stimulus to the dorsal columns with a stimulus to the muscle nerve at 1·4*T*. The positions of the stimulus artifacts are indicated by the arrows.

muscle afferent fibres. Stimulation at more rostral levels, however, will activate only those afferent fibres with an axon collateral ascending in the dorsal columns to the stimulation site and should therefore reveal the extent to which muscle afferent fibres project rostrally in the dorsal columns.

Group I projection. Stimulation of the dorsal columns at progressively more rostral

spinal levels evokes antidromic volleys which, because of the lengthening conduction distance, are progressively longer in latency and increasingly dispersed. There is, nevertheless, no significant reduction in the amplitude of the group I compound action potential evoked from lumbar levels until the upper lumbar segments are reached (Figs 2 and 3, left). Stimulation of the dorsal columns at L1 or the lower thoracic segments, however, evoked antidromic volleys in which the amplitude of the group I component was substantially reduced such that few, if any, fibres were activated from these segments.

Group II projection. Because of slower conduction velocities, the antidromic volley associated with activity in group II fibres is more temporally dispersed and consists of a number of small deflections. The amplitude of the group II volley cannot therefore be readily assessed. Despite this temporal dispersion, considerable asynchronous activity is evident in the antidromic volleys evoked from the lumbar cord as far rostral as the L1/L2 region (Figs 2 and 3, left). With stimulation beyond this region, however, the group II component is abruptly diminished, little if any activity being evoked from the lower thoracic segments.

A consequence of the greater dispersion of group II activity was that with recordings from fine filaments it was sometimes possible to discern a number of unitary impulses within the antidromic volley (Fig. 2, right-hand panel). While such fibres could usually be traced in the activity evoked from the lumbar segments up to the L1 level, they could rarely be activated at lower thoracic segments (but see later).

Collision. Fibres supplying different cutaneous receptor types are known to undergo different degrees of slowing as they ascend the dorsal column (Brown, 1968; Petit & Burgess, 1968). Similarly, group I muscle afferent fibres are reported to demonstrate a greater reduction in conduction velocity than group II fibres (McIntyre & Lloyd, 1948; McIntyre, 1962*b*; but see later). This implies that the separation into distinct group I and group II components, which is evident in the antidromic volley evoked from lumbosacral levels, might not be maintained following stimulation at more rostral levels. This possibility was therefore investigated by colliding orthodromic impulses, with activity evoked by stimulation of each of the dorsal column stimulation sites in turn. As can be seen from the example in Fig. 3, conditioning stimuli of 1·4*T* were sufficient to extinguish the early compound action potential but longer latency asynchronous activity remained evident in the volleys evoked from all segmental levels. Thus, irrespective of the level of origin of the antidromic volley the group II activity always remained at a longer latency than the group I, with no tendency for the group I volley to slow down with respect to the group II component.

The collision test could also be used to investigate the projection of group Ia afferent fibres within the dorsal column, since on the whole the Ia afferents of the hamstring muscles have a lower electrical threshold than the Ib fibres (Bradley & Eccles, 1953; Coppin, Jack & McIntyre, 1969; Jack, 1978). In two experiments, conditioning stimuli were applied to a hamstring nerve at a strength (1·3*T*) known to selectively activate (Jack, 1978) only the Ia fibres. Such conditioning stimulation extinguished a proportion of the antidromic group I volley attributable to Ia fibres, whilst a later component, presumably due in large part to group Ib fibres, remained.

The proportion of activity remaining in comparison to that extinguished was similar following stimulation at all rostrocaudal levels. Thus, the projection of group Ia fibres parallels that of group I fibres as a whole, suggesting that the projection of both group Ia and Ib fibres is similar.

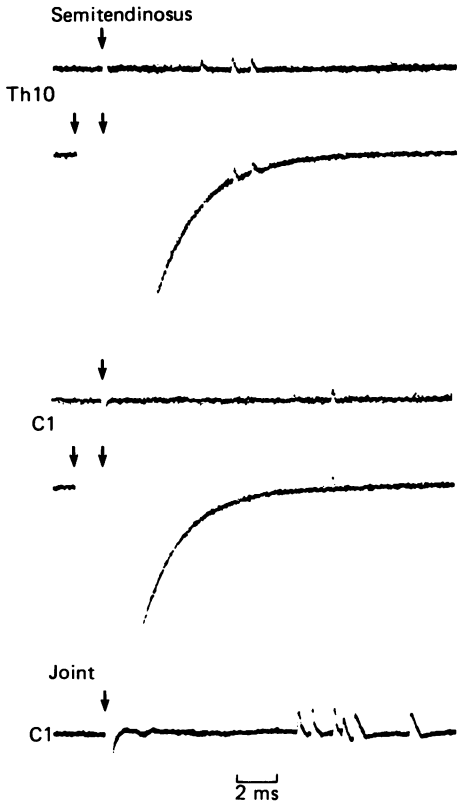

Fig. 4. Unitary activity evoked from the thoracic and cervical levels of the dorsal columns. The top and middle pairs of traces were recorded from a filament of the semitendinosus nerve after stimulation of the dorsal columns at Th10 and C1 respectively. The lower traces of each pair show the result of preceding the stimulus to the dorsal columns with a stimulus to the posterior biceps–semitendinosus nerve. The nerve stimulus is at a strength (1·6*T*) which is at threshold for collision of the earliest unit activated at Th10 and for the single unit activated at C1. The bottom trace is a recording from the joint nerve after stimulating the dorsal column at C1.

Fibres projecting to upper cervical levels

It was frequently possible to activate a small number of afferent fibres (typically one to four) by stimulation of the thoracic cord at the Th10 segment (Fig. 4, top records). In contrast, only rarely could fibres be activated by stimulation of the cervical cord. Of eighteen filaments and two whole nerves tested with stimuli applied to the dorsal columns at C1 only four (one whole nerve and three filaments) contained fibres that could be antidromically driven. In the one whole nerve recording (from lateral gastrocnemius–soleus), more than five fibres could be distinguished, but fewer

were present in the filaments; a medial gastrocnemius filament contained two projecting fibres, while a filament of the posterior biceps nerve and a filament of the semitendinosus nerve each contained a single projecting fibre (Fig. 4, middle records). Use of the collision test with carefully graded electrical stimuli showed that the peripheral thresholds of individual projecting fibres ranged from 1·0 to 1·7*T*. Overall latencies for conduction from the peripheral recording site to C1 ranged from 7 to 12 ms.

Recordings from non-muscular nerves

Recordings were also made from several non-muscular nerves, including cutaneous nerves, the posterior nerve to the knee joint and the interosseous nerve. Comparison of these recordings with those expected from the results of other studies (McIntyre, 1962*a*; Brown, 1968; Petit & Burgess, 1968; Clarke 1972) provided useful controls that our parameters of stimulation were adequate to excite dorsal column fibres at different spinal levels. Thus, since substantial responses were recorded from non-muscular nerves following stimulation of the dorsal columns at Th10 and C1, while in the same experiments comparatively little response could be obtained from muscular nerves, we are confident that very few muscle fibres reach these levels.

In addition, we have confirmed the results of the little known study by Clark (1972) concerning the projection of fibres of the posterior nerve to the knee joint within the dorsal columns. Our results are entirely in accordance with his findings. In particular, the majority of afferents were found to leave the dorsal columns in the upper lumbar or the lowest thoracic segments and ten fibres or less were found to reach the cervical levels. Figure 4 (bottom record) shows an example in which stimulation of the cervical dorsal columns evoked a volley of single unitary activity consisting of more than eight distinguishable impulses in the posterior nerve to the knee joint.

Recordings from single muscle afferent fibres in the dorsal columns

Useful data were derived from micropipette recordings made from 113 single muscle afferent fibres in the dorsal columns at L6/7 close to their level of entry. Of these, eighty-nine fibres conducted at greater than 72 m s^{-1} and were therefore considered to be group I afferent fibres while the remaining twenty-four units had conduction velocities in the range 22–72 m s^{-1} and were therefore considered to be group II fibres. The ascending projections of sixty-seven single units were traced by their response to stimuli applied to the dorsal columns at different spinal levels; fibres were presumed to have terminated when either the antidromic response failed, or an abrupt increase in threshold occurred. The remaining forty-six units in the sample were tested with stimuli applied at the Th10 and C1 levels only.

Group I afferent fibres

The data obtained from fifty-three group I fibres was analysed graphically by plotting conduction time against conduction distance for each stimulation site; plots for four posterior biceps–semitendinosus and four plantaris group I fibres are shown in Fig. 5. The slopes of the lines drawn between the points give the conduction velocities over various parts of the conduction path. All fibres underwent a reduction

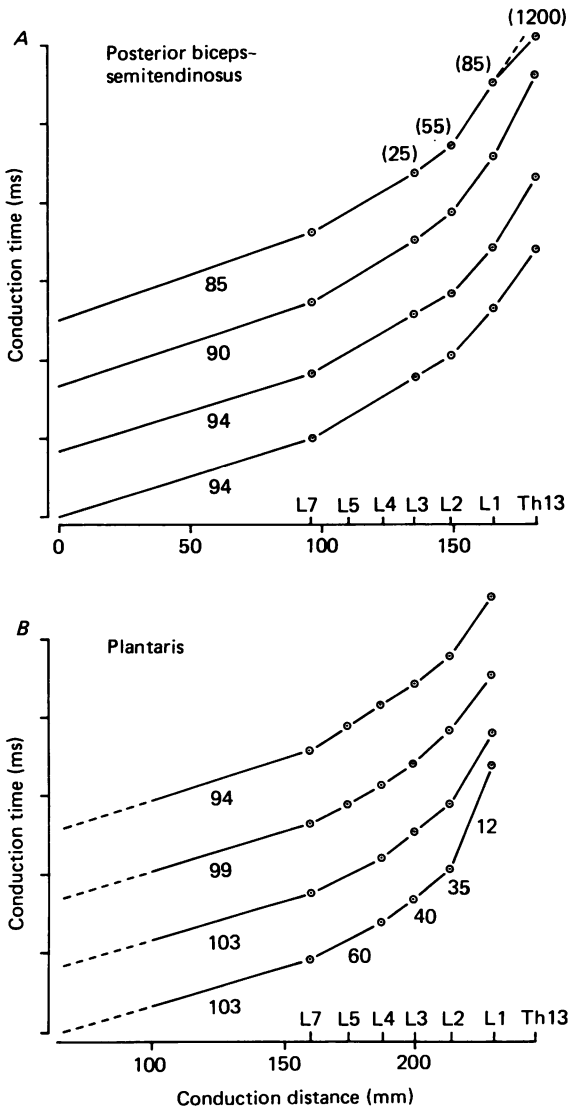

Fig. 5. Plots of conduction time *versus* conduction distance for a number of single group I muscle afferent fibres. *A* shows plots for afferents of posterior biceps–semitendinosus and *B*, plots for plantaris afferents. The numbers in parentheses indicate the current (in microamperes) required to activate the sample axon at the level indicated. The numbers without parentheses indicate the conduction velocity of the axon over the segment indicated (m s^{-1}). Note that the conduction velocity of the fibre illustrated in the top plot of *A* apparently increases at L1. Since, however, the current threshold at Th13 is very high (1·2 mA) this is interpreted as indicating that the fibre has terminated several millimetres caudal of the stimulation site and that the stimulus has spread caudally to the point of termination.

in conduction velocity upon entering the spinal cord of between 30 and 60% with a clear tendency for the fibres with the fastest peripheral conduction velocities to experience the greatest slowing (regression coefficient $-0{\cdot}74$; $P < 0{\cdot}001$). Further gradual reductions in conduction velocity occurred as the fibres travelled rostrally falling to between 12 and 20 m s^{-1} over the terminal portion of fibre.

Dorsal column fibres were most readily excited at the L3 region where thresholds varied between 15 and 80 μA. The majority of fibres could be activated at more rostral stimulation sites with only a moderate rise in threshold until a level was reached where the fibre failed to respond even at much increased stimulus strengths

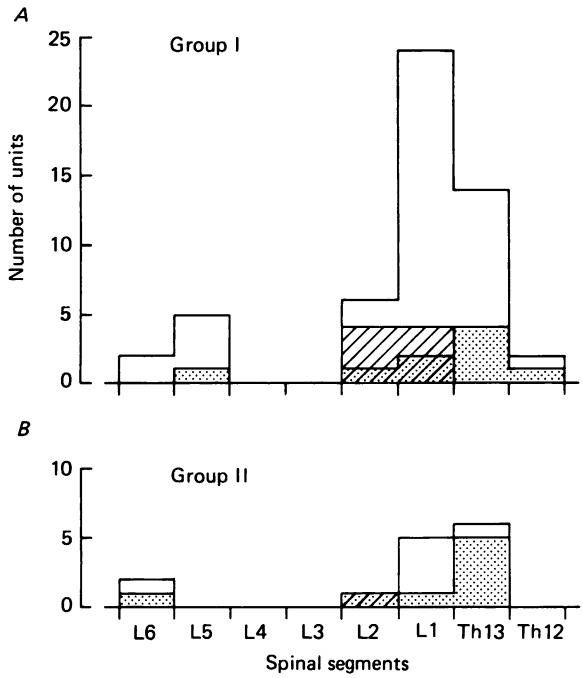

Fig. 6. Histograms summarizing the most rostral level of projection of fifty-three group I (*A*) and fourteen group II (*B*) afferent fibres. The histograms also show the most rostral level of projection of posterior biceps–semitendinosus afferents (stippled) and of plantaris afferents (hatched) for comparison.

(at least 1 mA; 15–80 times the threshold current at L3). A small number of units could be antidromically activated from above the presumed level of termination at high stimulus strengths but conduction latencies did not increase in accordance with the greater conduction distance; this was revealed by plots of conduction latency against distance in which conduction velocities over the terminal segments of fibres appeared to have increased (Fig. 5*A*, top curve). In these instances a spread of current caudal of the stimulation site seems most likely to have occurred. Single fibres were therefore considered to project to the most caudal level from which an antidromic response could be evoked without the requirement for an abrupt increase ($>$ 10 times) in stimulus strength.

The levels of projection of single group I muscle afferent fibres are summarized in Fig. 6*A*. The majority of fibres (46; 87%) were found to project to the upper lumbar or the lowest thoracic segments of the cord although a small number of fibres apparently terminated in more caudal lumbar segments (see Discussion). In addition to the fifty-three fibres illustrated in Fig. 6*A*, none of thirty-six group I fibres tested with stimuli applied only at the Th10 and C1 levels could be activated from either of these stimulation sites.

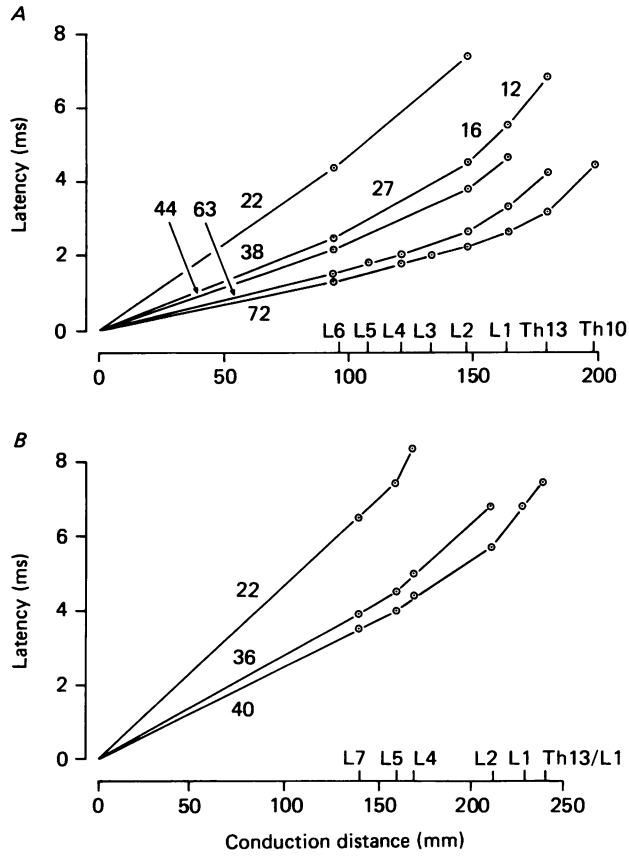

Fig. 7. Plots of conduction latency *versus* conduction distance for group II afferent fibres. Plots for single group II fibres of posterior biceps–semitendinosus are shown in *A*. The plots in *B* are for the single group II fibres shown in the nerve filament recording in the right-hand panel of Fig. 2. The numbers indicate the conduction velocity (m s^{-1}) of the axon over the segment indicated.

There was no apparent correlation between the conduction velocities of group I fibres and their levels of projection in the dorsal columns. In addition, the pattern of projection of hamstring fibres with peripheral thresholds in the group Ia range did not differ from that of fibres with thresholds in the higher group I, predominantly Ib range (Bradley & Eccles, 1953). There was however a tendency, apparent in Fig. 6, for plantaris afferent fibres to terminate at L2/L1 while in general fibres in the posterior biceps–semitendinosus nerve projected one segment more rostrally to L1/Th13.

Group II afferent fibres

Figure 7*A* shows plots of conduction latency against distance for five group II afferent fibres recorded in the dorsal columns and Fig. 7*B* shows similar plots for unitary group II potentials (illustrated in Fig. 2, right) recorded from a muscle nerve filament. The slopes of the lines indicate the progressive decrease in conduction velocity which accompanies the ascent of group II fibres in the dorsal columns. Figure 6*B* summarizes the levels of projection of fourteen group II fibres most of which reached the upper lumbar or the lowest thoracic levels. An additional ten group II fibres tested only with stimuli at the Th10 and C1 levels failed to respond at either location.

The patterns of conduction velocity decrease and dorsal column projection of group II fibres therefore parallel those observed within the larger sample of group I fibres. In contrast to group I fibres, however, there was a tendency for the peripheral conduction velocities of group II fibres to relate to the rostral extent of their dorsal column projection. This trend is apparent in Fig. 7*B* where the slowest fibre (22 m s^{-1}) terminates at L4, the intermediate fibre (36 m s^{-1}) at L2 and the fastest fibre (40 m s^{-1}) at Th13. This trend is also apparent (with one exception) for the fibres illustrated in Fig. 7*A*.

DISCUSSION

The present results show that group I and group II muscle afferent fibres undergo a reduction in conduction velocity upon entering the dorsal columns, with subsequent progressive reductions as the fibres travel rostrally, and that the great majority of fibres leave the dorsal columns at upper lumbar or lower thoracic segments.

Methodological considerations

In concluding that muscle afferents terminate at any given level, it was important to consider the progressive change in excitability the axons are expected to undergo in view of the reduction in diameter that they experience as they ascend the dorsal columns (Hongo, Kudo, Sasaki, Yamashita, Yoshida Ishizuka & Mannen, 1987). Hence, it is conceivable that, in the present experiments, the excitability of ascending axons might have decreased sufficiently to the point when they could not be activated by the electrical stimuli used.

We were encouraged to believe that we could, in fact, stimulate most fibres present by the following observations. First, even when stimuli of much higher strengths (1 mA) and longer durations were used, they only marginally increased the size of the antidromic volley recorded from peripheral nerves and any such increase could adequately be explained by stimulus spreading to activate fibres terminating at more caudal levels. Second, in general, when recording from single fibres, a stimulus of 15 μA was adequate to activate them at the L3 segment, where they are conducting at 20–30 m s^{-1}. Since the electrical thresholds of nerve fibres are approximately inversely related to their conduction velocities (Eccles & Lundberg, 1959; Ellaway *et al.* 1982), an increase of the stimulus strength to 20 times that required at L3 (i.e. 1 mA) should result in the activation of fibres with a conduction velocity of 20 times less, i.e. in the region of 1–2 m s^{-1}. Thus, as both group I and group II afferent fibres

underwent a *gradual* reduction in conduction velocity up to T13/L1 to about 15–20 m s^{-1} beyond which we could not activate them (with an appropriate increase in latency), we can be reasonably confident that the great majority terminate at the upper lumbar or the lower thoracic levels.

While recording from single fibres, a small number were found which appeared to terminate in the *caudal* lumbar segments. However, we do not feel that these observations contradict our main conclusions, since it is highly likely that some, and possibly all, of these apparent caudal terminations were the result of mechanical damage to the dorsal columns: some fibres must have been damaged in the course of making pial 'patches' and in making repeated electrode tracks through the dorsal columns. Consequently, if any of these 'terminations' are in fact real they would appear too few to be of great importance.

Comparison with previous results

The present experiments were an extension of those by Lloyd & McIntyre (1950), which largely consisted of observing the group I volley recorded from the surface of the dorsal columns at different levels. They noted that the volley was progressively reduced in amplitude as it travelled rostrally and could not be detected at mid-thoracic levels. They realised that greater sensitivity could be achieved by the reverse procedure of stimulating the dorsal columns and recording the antidromic discharge from muscle nerves, but did not pursue this to any great extent. The present work has employed this latter approach, together with the complementary approach of single-unit recording. The present findings that group I fibres terminate in the upper lumbar or lower thoracic segments of the spinal cord are entirely in accordance with those of Lloyd & McIntyre (1950).

With regard to group II afferents, however, there was little information available previous to this study regarding the extent to which they project through the dorsal columns. There are, in fact, only two previous reports which seem to address this issue. The first, an abstract (McIntyre & Lloyd, 1948), gives few details, though reports that group II muscle afferents can be traced as far rostral as the uppermost thoracic segments. Our findings are clearly in contrast to this, but without further details of their study we are unable to comment on this discrepancy. The second study (McIntyre, 1962*b*) reports that the conduction latencies from the thoracic dorsal columns to peripheral nerves are shorter for group II fibres than for group I and concludes from this that conduction in group II fibres is subject to less slowing in the dorsal columns than conduction in group I fibres. In the present experiments, group II activity evoked from different spinal segments, whether recorded from single fibres or muscle nerves, always occurred later than activity in group I fibres. In addition, as the stimulus strength applied to the dorsal columns at various levels was gradually increased, the group I component, recorded from muscle nerves, was always the first to be activated (though the group II volley began to be recruited before the group I volley was maximal). Thus, at different levels of the dorsal column, the group I fibres are generally more excitable and hence of larger diameter and faster conduction velocity than the group II fibres, even though both groups of fibre have undergone progressive reductions in conduction velocity.

Afferent fibres that project to the cervical cord

Stimulation of the cervical dorsal columns revealed that, in contrast to cutaneous afferents, only a very small number of muscle fibres project to this level. Indeed, because of the temporal dispersion and the unitary nature of the recordings, it is probable that we were able to distinguish all muscle afferents reaching cervical levels of the cord, in any particular filament tested. Furthermore, by colliding such antidromically evoked discharges with those orthodromically evoked, these fibres were found to have peripheral thresholds (1·0–1·7T) within the group I, or lowest group II, range.

Since only a very small proportion of afferent fibres in muscle nerves reach the cervical cord, one is tempted to speculate as to their receptor origin. Undoubtedly, they are greatly outnumbered by muscle spindle and Golgi tendon organ afferents and consequently, if these 'cervical' afferents are of spindle or tendon organ origin, it is not clear why these few fibres should project to cervical levels, while the majority terminate more caudally. Alternatively, and probably more likely, these fibres might be of different origin since other afferent species sometimes 'contaminate' muscle nerves. In particular, there are several reports that Pacinian or paciniform corpuscles are associated with muscles, though they are relatively scarce (Barker, 1962; Boyd & Davey, 1968; Jack, 1978). For example, Barker (1962) found six paciniform corpuscles in three soleus muscles, and ten paciniform corpuscles in three semitendinosus muscles. In addition, a few of the much larger Pacinian corpuscles are sometimes associated with muscles, though their intramuscular occurrence is quite exceptional (Barker, 1962).

It is known that Pacinian afferent fibres project the full length of the dorsal columns (McIntyre, 1962*a*; Brown, 1968; Petit & Burgess, 1968). In addition, Burgess & Clark (1969) have shown that those afferents in the joint nerve that project the full length of the dorsal columns are rapidly adapting and originate from the paciniform type of ending within the joint capsule. Furthermore, the antidromic discharges recorded from the interosseous nerve in McIntyre's (1962*a*) study and from the joint nerve in Clarke's (1972) study, following stimulation of cervical levels, were from 7 to 13 ms in latency, precisely the range of latencies observed in our sample of C1 projecting afferents in muscle nerves. Thus, the small numbers of Pacinian or paciniform corpuscles associated with muscles could provide an adequate explanation, of the nature of the small number of fibres that were observed to project to C1 in the present experiments.

Functional considerations

The present study provides information about whether or not at least one collateral of any particular afferent fibre continued up the dorsal columns. Presumably other branches leave the dorsal columns and terminate nearby in the spinal grey matter. Given that collaterals become thinner with successive branching (Hongo *et al.* 1987) and consequently have a reduced conduction velocity, it follows that the regions of conduction velocity decrease, as well as the level of termination of the ascending collateral, should be the regions over which second-order neurones activated by these afferent fibres are located. Indeed, it is now established that both

group I and group II muscle afferent fibres activate neurones within the lumbosacral enlargement, the middle lumbar segments (see for example Cavallari, Edgley & Jankowska, 1987; Edgley & Jankowska, 1987*a*, *b*) and Clarke's column. Thus, a *progressive* reduction in conduction velocity along the lumbar segments as far as the level of Clarke's column would be expected.

Clarke's column extends from the upper lumbar segments to the most rostral thoracic segments (Rexed, 1954). However, by successive lesioning of the dorsal columns it has been established that the hindlimb group I relay in Clarke's column is located at its most caudal tip (L3/L4; Oscarsson, 1957). Consequently, it has not been fully appreciated until now that group I afferents project rostral of the Clarke's column relay by two or three segments. It is therefore of particular interest to consider the possible regions of termination of this projection.

One possibility is that there is, in fact, a projection to Clarke's column at more rostral locations (L1/Th13) but that these neurones do not discharge in response to maximal group I volleys and did not therefore reveal themselves in the experiments of Oscarsson (1957), in which mass recordings were made from the dorsolateral funiculus. However, such a subliminal projection would be in contrast to the characteristic properties of Clarke's column neurones in L3/L4 which are securely activated (Eide, Fedina, Jansen, Lundberg & Vyklicky, 1969). Unfortunately, subsequent work recording directly from Clarke's column neurones has concentrated on investigating neurones at L3 or L4, and there is no evidence, for or against, a projection to Clarke's column neurones at L1/Th13.

An alternative possibility is that this projection is to a region outside Clarke's column. This seems likely in view of recent work showing that at L3 and L4 the termination of group I and group II afferents outside Clarke's column is quite substantial (Cavallari *et al.* 1987; Edgley & Jankowska, 1987*a*, *b*; Hongo *et al.* 1987), activating interneurones projecting to hindlimb motoneurones (Cavallari *et al.* 1987) and ascending tract neurones (Edgley & Jankowska, 1988). It is therefore quite possible that the most rostral lumbar segments are similarly involved. The projection to the rostral lumbar segments may well therefore be destined for neurones outside Clarke's column and is perhaps involved in the processing of hindlimb 'segmental' information and/or the activation of ascending tract neurones.

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