J. Physiol. (1986), **381**, *pp.* 333–349
With 8 text-figures
Printed in Great Britain

RELATIONS BETWEEN SPINOCERVICAL AND POST-SYNAPTIC DORSAL COLUMN NEURONES IN THE CAT

BY A. G. BROWN, R. NOBLE AND J. S. RIDDELL*
From the Department of Veterinary Physiology, University of Edinburgh, Summerhall, Edinburgh EH9 1QH

(*Received 24 March 1986*)

SUMMARY

1. In chloralose-anaesthetized cats single-unit micro-electrode recordings were made at the lumbosacral level either from axons in the dorsolateral funiculus and dorsal columns, identified as belonging to the spinocervical tract (s.c.t.) or post-synaptic dorsal column (p.s.d.c.) pathway respectively, or from neurones in the dorsal horn similarly identified.

2. Attempts were made to show that s.c.t. and p.s.d.c. neurones had axons that bifurcated, so that they sent branches into both the ipsilateral dorsolateral funiculus and the dorsal columns. That is, that some, or all, of the presumed s.c.t. or p.s.d.c. axons were common to both populations. In addition, the effects of stimuli applied to the ipsilateral dorsolateral funiculus at C3 and C1 on the resting discharges of p.s.d.c. neurones were examined in order to determine the effectiveness of the link between the s.c.t. and the p.s.d.c. pathway.

3. Thirty-three s.c.t. units (twenty-six axonal recordings and seven soma–dendritic recordings) and thirty p.s.d.c. units (twenty-four axonal and six soma–dendritic recordings) were examined for bifurcating axons by electrically stimulating the dorsolateral funiculus at C3 and the dorsal columns at C4. None of the p.s.d.c. units could be antidromically activated from the ipsilateral dorsolateral funiculus with stimulus strengths up to 40 V or seventy times threshold for antidromic activation from the dorsal columns. Similarly, twenty s.c.t. units could not be activated antidromically from the dorsal columns at stimulus strengths up to 30 V or thirty times threshold for their antidromic excitation from the dorsolateral funiculus. Thirteen s.c.t. units were antidromically activated from the cervical dorsal columns, eight at seventeen or more times threshold for their activation from the dorsolateral funiculus and five at between two and nine times threshold. All s.c.t. units that were activated antidromically from both the cervical dorsal columns and the dorsolateral funiculus showed similar latencies for the two responses.

4. Twenty-five p.s.d.c. units were examined for the effects of ipsilateral dorsolateral funiculus stimulation on their resting activity. In thirteen, clear evidence of facilitatory effects from C3 were observed, whereas similar results were seen in only six of these units when C1 was stimulated and the effects were less. The facilitation had a latency of 3–16 ms and lasted for 6–22 ms. In all but one of the twenty-five

* J. S. R. was a M.R.C. Research Student.

units, stimulation at both C1 and C3 produced profound inhibition of the resting discharge that began at between 8 and 26 ms and lasted for up to 300 ms. There were no obvious differences in either the duration or degree of inhibition evoked from the two sites.

5. We conclude that (a) when the present results are considered together with previous results on the receptive field properties, the dendritic tree anatomy and the ultrastructure of synapses on presumed s.c.t. and p.s.d.c. neurones, it is reasonable to believe that the two ascending somaesthetic pathways arise from different populations of neurones in the dorsal horn, (b) the excitatory effects on p.s.d.c. units elicited from the dorsolateral funiculus at C3 are largely due to antidromic activation of s.c.t. neurones, which are known to have collateral axons that terminate on p.s.d.c. neurones, the excitation from C1 could conceivably be due to activation of the s.c.t. but seems more likely to be due to activation of other neuronal systems. The inhibitory effects are largely, if not entirely, due to activation of descending systems.

INTRODUCTION

The spinocervical tract (s.c.t.) and the post-synaptic dorsal column (p.s.d.c) pathway are generally considered to be two separate somaesthetic pathways at the level of the spinal cord, arising from different populations of dorsal horn neurones, having axons travelling in the ipsilateral dorsolateral funiculus and the dorsal columns respectively, and terminating in the lateral cervical nucleus (l.c.n.) and the dorsal column nuclei (d.c.n.) respectively (see Brown, 1981). Indeed, reports of the receptive field properties of the two sets of neurones (Hongo, Jankowska & Lundberg, 1968; Uddenberg, 1968; Brown & Franz, 1969; Brown, 1971; Angaut-Petit, 1975; Brown & Fyffe, 1981; Brown, Brown, Fyffe & Pubols, 1983; Noble & Riddell, 1984), of the anatomy of their dendritic trees (Brown, Rose & Snow, 1977; Brown & Fyffe, 1981; Enevoldson, 1982) and of the ultrastructure of their synaptic inputs (Bannatyne, Brown, Fyffe & Maxwell, 1982; Maxwell, Fyffe & Brown, 1982, 1984; Bannatyne, 1984) establish that there are distinct differences between them.

There are, however, close relationships between the two systems. Thus s.c.t. neurones may have collateral axons that terminate on p.s.d.c. cells (Maxwell & Koerber, 1986) and Jankowska, Rastad & Zarzecki (1979) have shown that electrical stimulation of the ipsilateral dorsolateral funiculus at C3–4 (below the l.c.n.) may evoke excitatory post-synaptic potentials in p.s.d.c. neurones, whereas stimulation at C1 (above the l.c.n.) is much less effective. The excitatory potentials had latencies commensurate with a monosynaptic link and Jankowska *et al.* (1979) suggested that the effects from C3 were due to antidromic activation of s.c.t. axons with subsequent orthodromic invasion of their collateral axons at the lumbosacral level. Also, some p.s.d.c. neurones have axons that initially enter the ipsilateral dorsolateral funiculus before turning to project in the dorsal columns (Brown & Fyffe, 1981). Furthermore, some neurones, identified as belonging to the p.s.d.c. system, have axons that enter the dorsolateral funiculus and do not cross into the dorsal columns, at least not for several millimetres which is the length of axonal staining following intracellular horseradish peroxidase (HRP) injection (Brown & Fyffe, 1981). In addition, some axons with cell bodies in the dorsal horn are known to ascend the ipsilateral

dorsolateral funiculus but to terminate in the dorsal column nuclei (Dart & Gordon, 1973; Gordon & Grant, 1982). Whether or not this latter set of neurones gives collateral axons to the l.c.n. is not known, although Craig & Tapper (1978) provide some evidence suggesting that they do.

Recently, the concept of separate s.c.t. and p.s.d.c. neurones has been challenged. Bennett, Nishikawa, Lu, Hoffert & Dubner (1984) claimed, from a sample of intracellularly stained p.s.d.c. neurones, that there is little, if any, morphological difference between the dendritic tree organization of these neurones and s.c.t. cells. Furthermore, they stressed the similarities between receptive field properties of the two sets of neurones and suggested that the two pathways are not completely independent. Following this, Lu, Bennett, Nishikawa & Dubner (1985) searched for evidence that dorsal horn neurones might have branched axons, with one branch ascending the dorsal columns and the other ascending the ipsilateral dorsolateral funiculus. They found that twenty-three of fifty-six neurones could be antidromically activated from both the dorsal columns and dorsolateral funiculus at cervical levels, with twenty-two only from the dorsal columns and eleven only from the dorsolateral funiculus. They concluded that many neurones contribute axons to both the p.s.d.c and s.c.t. systems. Lu *et al.* (1985), however, used search stimuli of 30 V and observed that the antidromic latencies for the two presumed axons were always similar, differing on average by only 0·26 ms. This suggested to us that their stimuli might have spread between the ascending tracts even though they dissected the dorsal columns and dorsolateral funiculus away from the cord. Finally, in a short note, Jiao, Zhang, Liu, Wang & Lu (1984) described double retrograde labelling of dorsal horn neurones after fluorescent dye injections into the l.c.n. and d.c.n.

The experiments to be reported in the present paper were performed (1) to examine, by electrophysiological means, the possibility that s.c.t. and p.s.d.c neurones might, in part at least, comprise a common population, and (2) to further examine the actions of s.c.t. neurones on the discharges of p.s.d.c. cells.

METHODS

Experiments were performed on eight cats (2·2–3·1 kg wt.) anaesthetized with chloralose (70 mg/kg) after induction with halothane in a nitrous oxide:oxygen mixture and paralysed with gallamine triethiodide. End-tidal CO_2, arterial blood pressure and rectal temperature were monitored and maintained within normal limits. Anaesthetic level was assessed by inspection of a continuous blood pressure record and the diameters of the pupils of the eyes.

The experimental arrangement is shown diagrammatically in Fig. 1. Laminectomies were performed at C1–C4 and L3–L7 inclusive. The dorsal columns were sectioned, by dissection with watchmakers' forceps, at caudal C3. While the dissection was being performed the cord dorsum potential, at L7–S1 (see below), was monitored in response to electrical stimulation of the dorsal columns above the site of the transection. The dissection was continued until the initial negative component of the cord dorsum potential had disappeared. In one animal, at the end of the experiment, the spinal cord at the lesion was sectioned transversely and examined for completeness of the lesion. Pairs of silver-ball stimulating electrodes were placed on the dorsolateral funiculus of the left side at rostral C1 and at C3, i.e. above and below the l.c.n. for identification of s.c.t. units, and on the dorsal columns at C3 and at C4, i.e. above and below the dorsal column section for identification of p.s.d.c. units. Cerebrospinal fluid that accumulated around the cervical cord was removed repeatedly during an experiment. The spinal cord at both laminectomy sites was covered with warm liquid paraffin. This preparation, in which the dorsal columns and dorsolateral

funiculus were not physically separated should have biased the experiment in favour of stimulus spread between the two parts of the cord.

In six cats, micro-electrode recordings were made from single axons in both the dorsal columns and left dorsolateral funiculus (at L4–L6) using micropipettes filled with 4 M-NaCl. In the remaining two cats recordings were made, using similar electrodes with lower impedances, from

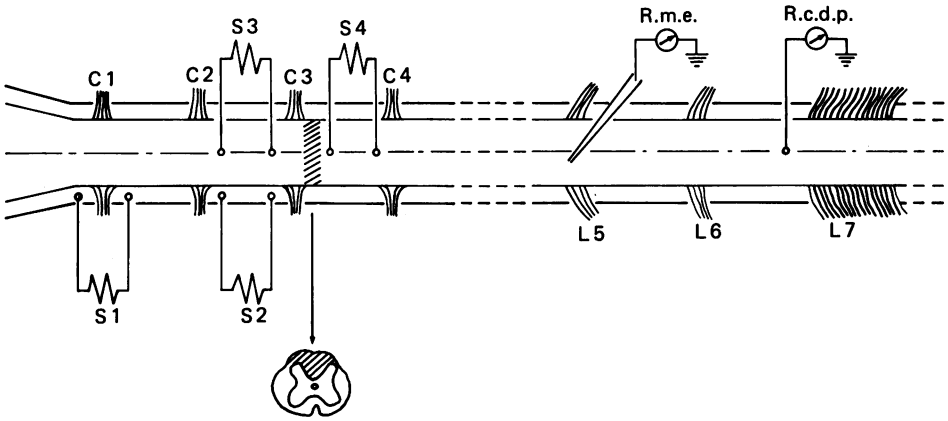

Fig. 1. Diagrammatic representation of the experimental arrangement. The drawing represents a dorsal plan view of the spinal cord at the cervical (C1–C4) and lumbar (L5–L7) enlargements. S1 and S2 represent stimulation sites on the dorsolateral funiculus at C1 and between C2 and C3. S3 and S4 are stimulation sites above and below a lesion of the dorsal columns at C3 (indicated by hatching). R.m.e., recording micro-electrode; r.c.d.p., cord dorsum recording electrode.

neurones in the left dorsal horn (at L6–S1). For axonal recordings search stimuli (3 V, approx. 250 μA, 0·1 ms, once every 600 ms) were applied to either the dorsolateral funiculus at C3 or the dorsal columns at C4 according to the location of the recording site. Stimuli of this strength applied to the d.l.f. at C3 are known to be capable of antidromically exciting all s.c.t. neurones (Brown, Fyffe, Noble, Rose & Snow, 1980). For neuronal recording from the dorsal horn the search stimuli were applied to the dorsal columns at C4 since most units could be excited from there.

S.c.t. units were identified by antidromic excitation from the dorsolateral funiculus at C3 and either failure to respond from C1 (thirteen of thirty-three units) or an antidromic response of longer latency (indicating a conduction velocity drop of at least one-third, seventeen of thirty-three units) or with a much higher stimulus threshold (three of thirty-three units). P.s.d.c. units were identified by their antidromic response from the dorsal columns at C4 and absence of response from above the dorsal column section at C3. All antidromic responses were identified by the collision test between orthodromic and antidromic impulses, and by frequency following (five shocks at 500 Hz minimum). All s.c.t. and p.s.d.c. units so identified had convergent excitatory inputs and resting discharges.

When units were tested for branching axons, stimuli (0·1 ms in duration) were applied through each pair of electrodes in turn (on the dorsolateral funiculi at C3 and C1 and on the dorsal columns at C4 and C3) and the voltage increased until a response was observed or to some maximal voltage with no responses (see Results). Threshold voltages necessary to elicit antidromic responses were carefully noted. For s.c.t. units, which were nearly all (thirty-one of thirty-three) excited orthodromically from the dorsal columns at C4, the stimulus voltage was raised beyond that eliciting orthodromic responses and either the stimulus repetition rate was increased from the standard rate of 1·67 Hz to 500 Hz or a train of five stimuli at rates up to 500 Hz were used to determine if antidromic responses were present.

When p.s.d.c units were tested for orthodromic effects from the dorsolateral funiculus, stimuli in trains of five shocks at 333 Hz, 0·1 ms in duration and 3 V (about 250 μA) in amplitude, were

applied at C3 and C1. All latency and duration measurements were taken from the time of the first shock in the train. All responses were recorded on tape and both on-line and off-line analysis was carried out with a CED 1401 interface (Cambridge Electronic Design, U.K.) and BBC B microcomputer.

In all experiments the cord dorsum potential was recorded through a monopolar silver-ball electrode on the dorsal columns, near the dorsal root entrance zone, at L7 or S1. At the end of each experiment the conduction distances from the stimulating electrodes (cathodes) to the recording sites were measured.

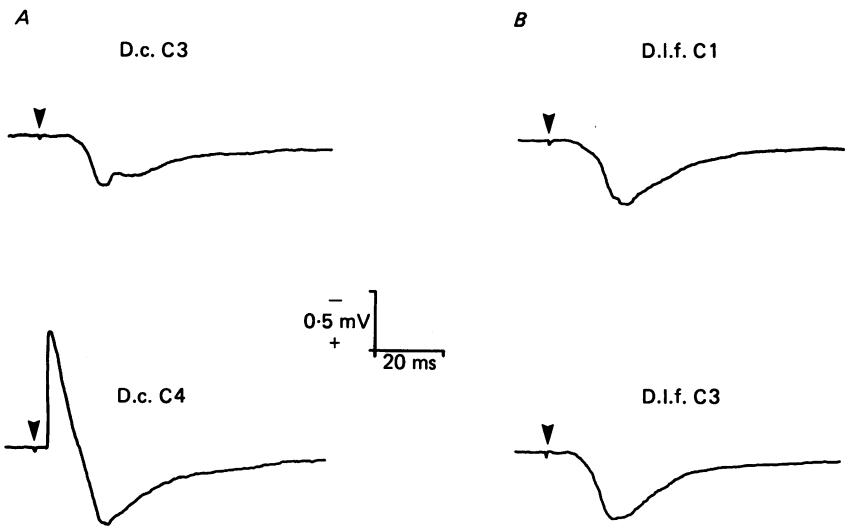

Fig. 2. Cord dorsum potentials evoked at the L7 segment by electrical stimulation of the cervical spinal cord. *A*, stimulation of the dorsal columns (d.c.) at C3, above the dorsal column transection and at C4 below the transection. *B*, stimulation of the dorsolateral funiculus (d.l.f.) at C1 and at C3, above and below the lateral cervical nucleus. All records are single traces, negativity upwards, and all stimuli were of 3 V strength. Note the large negative wave evoked from the dorsal columns at C4 and its absence in all other records. The positive wave evoked from the dorsal columns at C3 is due to stimulus spread to the dorsolateral funiculus at this strength of stimulation.

RESULTS

Activation of s.c.t. and p.s.d.c. neurones from the dorsolateral funiculus and dorsal columns

Cord dorsum potentials

Lu, Bennett, Nishikawa, Hoffert & Dubner (1983) and Lu *et al.* (1985) claim that electrical stimulation of the dorsolateral funiculus at upper cervical levels does not evoke a field potential in the ipsilateral lumbosacral dorsal horn unless there is stimulus spread to the dorsal columns. In our experience, cord dorsum and dorsal horn field potentials can be observed in the lumbosacral cord following electrical stimulation of the dorsolateral funiculus at stimulus strengths close to threshold for the fastest s.c.t. axons.

Fig. 2 shows typical cord dorsum potentials recorded at the lumbosacral cord upon stimulating (*A*) the dorsal columns above and below a dorsal column section at C3,

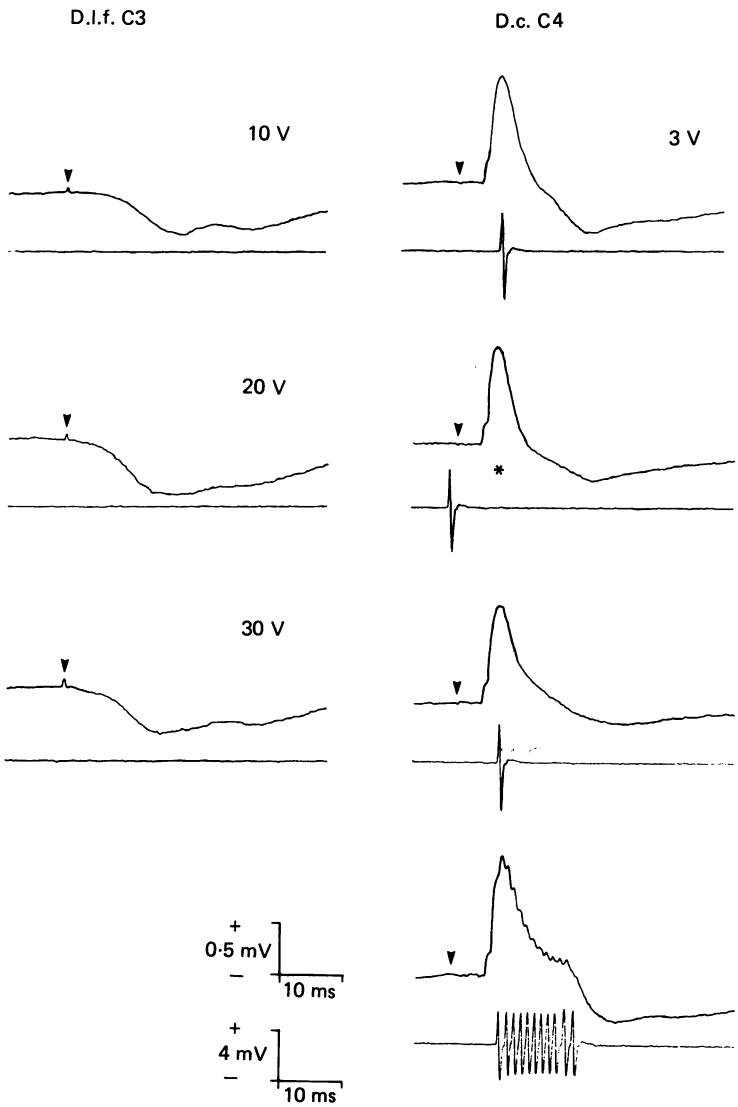

Fig. 3. Identification of a p.s.d.c. unit. Each pair of traces shows the cord dorsum potential recorded from L7 (upper traces) and an extracellular single-unit recording from an axon in the dorsal columns (lower trace). Stimulation of the ipsilateral dorsolateral funiculus (d.l.f.) with shocks of 10, 20 and 30 V (left-hand panel) failed to excite the unit, whereas stimulation of the dorsal columns (d.c.) at C4 excited the unit antidromically at 3·0 V (right-hand panel, threshold for activation was 1·1 V). Collision between an orthodromic and the antidromic potential is shown in the second pair of traces in the right-hand panel (the asterisk marks the position where the antidromic action potential should have appeared). The third trace shows its reappearance in the absence of an orthodromic potential. The bottom pair of traces in the right-hand panel shows the unit following a stimulation frequency of 1000 Hz.

and (*B*) the dorsolateral funiculus at C1 and C3. Stimulation of the dorsal columns at C4 produces a potential similar to the well known negative–positive potential described over many years that results from excitation of cutaneous nerves. Stimulation of the dorsolateral funiculus at both C3 and C1 and stimulation of the dorsal columns at C3 above the dorsal column section, all produce similar lumbosacral cord dorsum potentials that are almost completely positive going and which peak at about the same time as the positive component of the potential evoked from the dorsal columns at C4 below the lesion. The potential evoked from the dorsal columns at C3 is presumably due to stimulus spread to the dorsolateral funiculus.

Unit recordings

Recordings were made from sixty-three single units. The sample consisted of fifty axonal recordings, twenty-six from units identified as belonging to the s.c.t. and twenty-four identified as belonging to the p.s.d.c system, together with thirteen neuronal recordings from the dorsal horn, seven s.c.t and six p.s.d.c. (two of the dorsal horn recordings were intracellular, the rest extracellular).

P.s.d.c. units. The thirty p.s.d.c units could all be excited antidromically from the C4 dorsal columns at stimulus strengths between 0·05 and 2·6 V. None of them could be excited antidromically from either the dorsal columns at C3 or from the ipsilateral dorsolateral funiculus at C3. Both of these positions were above the level of the dorsal column section (Fig. 3). All but two of the units were tested with dorsolateral funicular stimulation of at least 20 V, with eighteen tested at 30 V and one at 40 V. Two units were tested at only 6 and 10 V. In the same experiments, however, s.c.t. units were antidromically activated from the dorsolateral funiculus at low stimulus strengths (see below).

S.c.t. units. The thirty-three s.c.t units were all excited antidromically from the ipsilateral dorsolateral funiculus at C3 with stimuli of 0·3–3·3 V. Of this sample, thirteen could not be antidromically fired from the C4 dorsal columns with stimuli of at least 20 V (four units were tested at 30 V). A further seven units were not activated with stimuli up to 5 V (they were not tested at greater stimuli). Thirteen units, however, including four of the seven recorded from the dorsal horn, could be excited antidromically from the dorsal columns at C4 (Fig. 4), and ten of these thirteen could also be antidromically activated from the dorsal columns at C3 above the dorsal column section. Eight of these thirteen units required a dorsal column stimulus of at least 17 times the strength of that required to activate them from the dorsolateral funiculus, three required stimuli of 6–9 times the dorsolateral funiculus threshold and two required only 2·3 and 3·5 times threshold.

All of the thirteen units that could be excited antidromically from both the dorsolateral funiculus at C3 and the dorsal columns at C4 had antidromic latencies from the two sites of within 0·3 ms of each other. The latency from the C4 stimulus was always less than that from the C3 stimulus.

Seventeen of the units identified as belonging to the s.c.t., including ten of those antidromically excited from the dorsal columns at C4, could be excited antidromically from the dorsal columns at C3 (above the dorsal column section). The C3 dorsal column electrodes were placed rostral to the C3 dorsolateral funicular electrodes and the antidromic latencies from the dorsal columns were either the same (seven units)

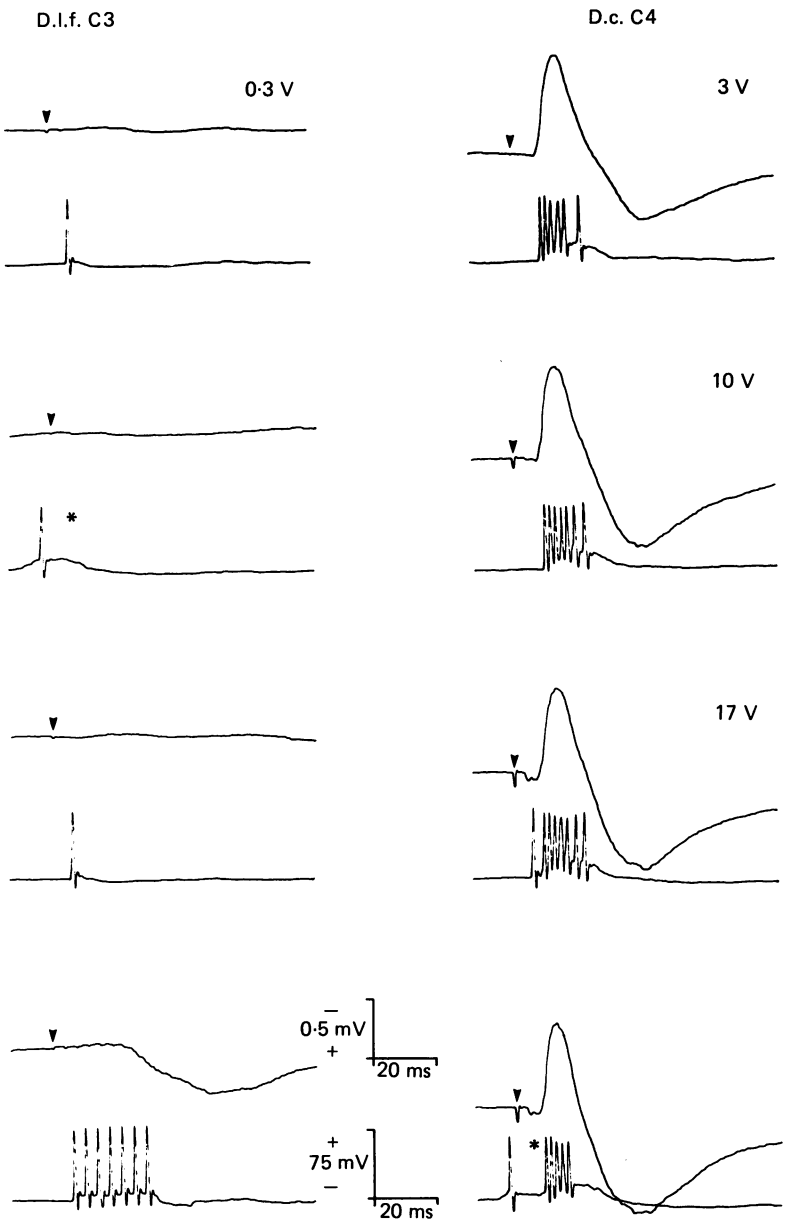

Fig. 4. For legend see opposite.

or up to 0·3 ms longer (nine units). One unit had a latency from the C3 dorsal columns 0·8 ms longer than from the C3 dorsolateral funiculus.

The sample of units

The present sample of units covered the range of both s.c.t. and p.s.d.c. axonal conduction velocities. Fig. 5. shows histograms for the two samples, including those

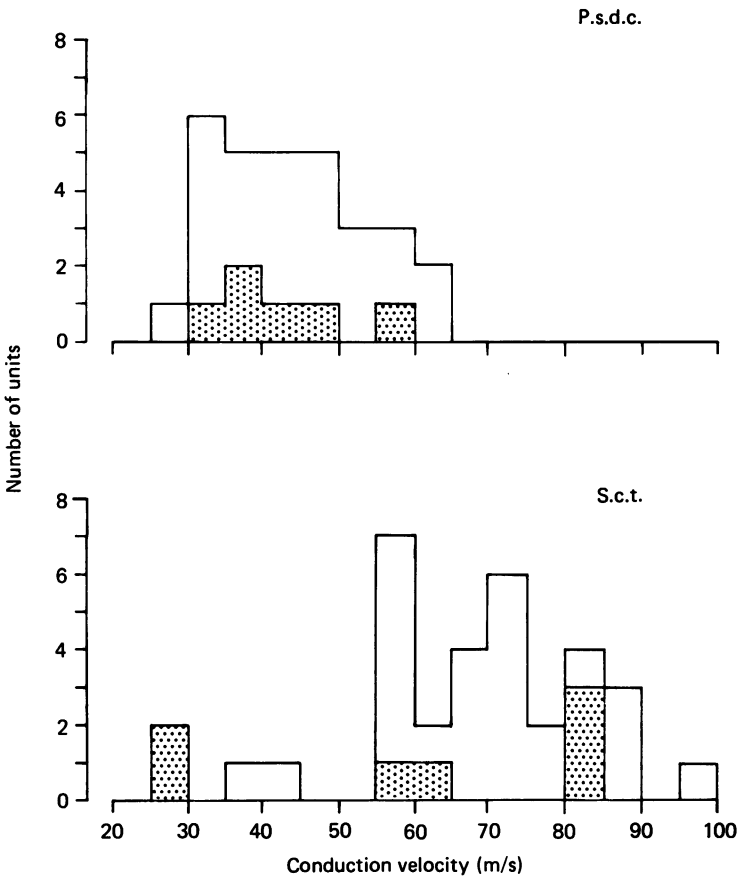

Fig. 5. Conduction velocities of the samples of p.s.d.c. and s.c.t. units. The total samples are shown as open histograms which include the units recorded from the dorsal horn (stippled).

Fig. 4. Identification of a s.c.t. unit. The Figure is similar to Fig. 3 except that the unit was recorded intracellularly in the dorsal horn. The unit had a threshold to ipsilateral dorsolateral funiculus (d.l.f.) stimulation at C3 of 0·3 V (note that no cord dorsum potential was recorded at this stimulus strength). The second pair of traces in the left-hand panel shows collision between antidromic and orthodromic impulses (the expected position of the antidromic impulse is marked with an asterisk), the third pair of traces shows the reappearance of the antidromic impulse in the absence of an orthodromic impulse at the critical interval. The bottom pair of records shows the unit following a stimulation frequency of 500 Hz. The right-hand panel shows responses to stimulation of the dorsal columns (d.c.) at C4 (at 3, 10 and 17 V respectively). At 3 and 10 V orthodromic responses were recorded. At 17 V an antidromic impulse was also evoked which could be shown to collide with an orthodromic action potential in the bottom pair of traces (the asterisk marks the position where the antidromic impulse was expected had collision not occurred).

units recorded from the dorsal horn. The conduction velocities of s.c.t. units ranged from 27 to 95 m/s and those of p.s.d.c. units from 26 to 62 m/s.

Intracellular recordings from dorsal horn neurones

It is possible that neurones with bifurcating axons are present in the dorsal horn but the antidromic impulse travelling down one branch fails to invade the other; in this case axonal recording may not reveal the branching. As described above, thirteen units in the present sample were recorded extracellularly (and two of them intracellularly) from the dorsal horn. The results obtained, however, were no different to those recorded from axons, but these constituted a minority of the sample. Nevertheless, in previous experiments in which intracellular recordings were made in the dorsal horn (Brown, Fyffe, Noble & Rowe, 1984) neurones were tested for antidromic responses from both the cervical dorsal columns and ipsilateral dorsolateral funiculus in the same way as described in the present paper. Of twelve s.c.t. neurones none were excited antidromically from the dorsal columns, although in ten, the dorsal column stimulus produced excitatory post-synaptic potentials. Of eight p.s.d.c. neurones, none were excited antidromically from the ipsilateral dorsolateral funiculus although two showed excitatory post-synaptic potentials.

Effects from the dorsolateral funiculus on p.s.d.c. neurones

Twenty-five p.s.d.c. units were examined for the effects of stimulating the ipsilateral dorsolateral funiculus, nineteen were recorded from axons in the dorsal columns and six from neurones in the dorsal horn.

Excitatory effects

In thirteen units peri-stimulus time histograms revealed a clear excitatory response to electrical stimulation of the dorsolateral funiculus at C3 (five shocks at 333 Hz, 0·1 ms duration and 3 V amplitude). This effect could be seen against both the resting and raised level of discharge evoked by a sustained pinch to the receptive field and had a latency of from 3 to 16 ms (from the first stimulus in the train) and lasted for 6–22 ms (Figs. 6 and 7). The effectiveness of the dorsolateral funiculus stimulation was variable between cells, causing a small effect in some cells but a much larger effect in others in the same experiment. Six of these thirteen units also showed similar excitatory effects from stimulating the funiculus at C1 (Fig. 6). This excitation was always less than that evoked from C3. No obvious differences were found between those p.s.d.c. units excited and those apparently not excited from C3. Excited units included those with receptive fields on either hairy or glabrous skin and all had both low-threshold and high-threshold excitatory components in their receptive fields.

Inhibitory effects

In addition to the excitatory actions, stimulation of the ipsilateral dorsolateral funiculus at both C3 and C1 produced profound inhibition in twenty-four of the twenty-five units (Figs. 6, 7 and 8). The inhibition began at 8–26 ms in those units that were not excited by the stimulation and virtually silenced the resting or pinch-evoked discharge for 93–145 ms with a full recovery of activity not occurring

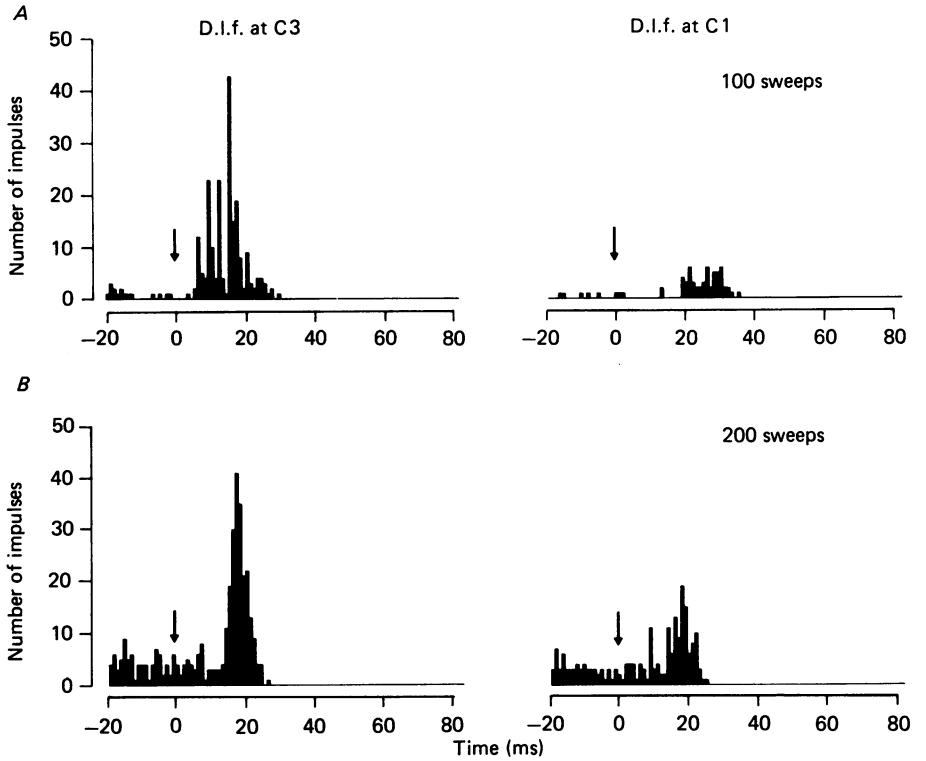

Fig. 6. Peri-stimulus time histograms showing the effects on two p.s.d.c. neurones (*A* and *B*) of stimulating the dorsolateral funiculus (d.l.f.) at C3 (left) and C1 (right). Onset of stimulus train (five stimuli, 333 Hz) is indicated by arrows. Impulses are accumulated in 1 ms time bins. In *B* background activity was elevated by a sustained pinch to the excitatory receptive field.

until after about 250–300 ms (see Fig. 8). There were no differences between the inhibitory effects produced from C3 or C1.

DISCUSSION

Do some dorsal horn neurones have bifurcating axons with one branch contributing to the s.c.t. and the other to the p.s.d.c. pathway?

Evidence for dorsal horn neurones with bifurcating axons ascending each of these somaesthetic pathways comes largely from the report of Lu *et al.* (1985). In their experiments the upper cervical dorsal columns and dorsolateral funiculus were each dissected free from the rest of the cord, and separated from it with a sheet of plastic. Each dissected component was stimulated electrically, using a pair of needle electrodes, with shocks of 0·2–0·5 ms width and 30 V amplitude in order to search for dorsal horn units excited antidromically. Stimulus spread between the dorsal columns and dorsolateral funiculus was assessed by field potential recording in the dorsal horn at lumbosacral levels. Field potential recording as performed by Lu *et al.* (1983, 1985), however, appears to be insensitive. They claim that stimulation of

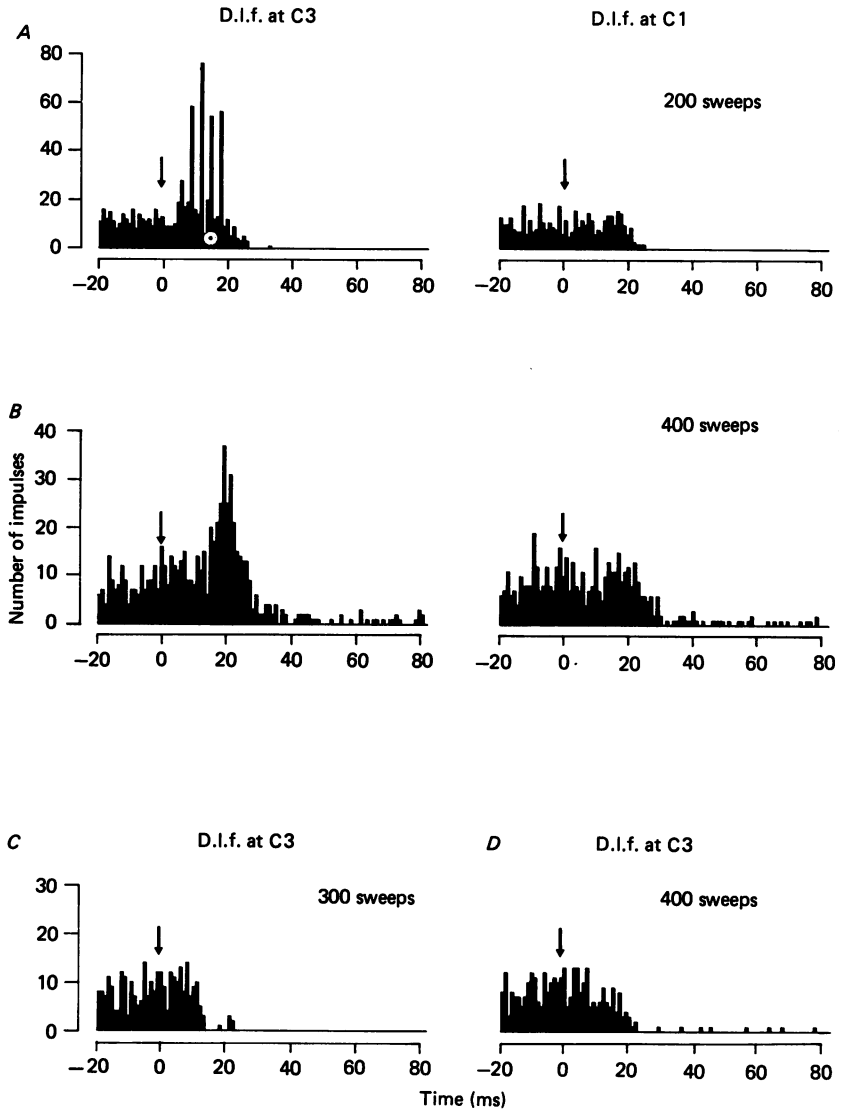

Fig. 7. Peri-stimulus time histograms showing the effects on single p.s.d.c. units of stimulating the ipsilateral dorsolateral funiculus (d.l.f.). *A* and *B* show the responses of two different units to stimulation at C3 (left) and C1 (right). In both cases a clear excitatory response was recorded to stimulation at C3 but not C1. *C* and *D* show the responses to stimulation at C3 of two units which were not excited.

the dorsolateral funiculus does not evoke a field potential in the lumbosacral dorsal horn unless the stimulus spreads to the dorsal columns. But, as shown in the present paper, dorsolateral funiculus stimulation will produce a very obvious cord dorsum potential in the lumbosacral cord that is clearly different from that evoked from the dorsal columns. Thus the cord dorsum potential evoked from the dorsolateral funiculus consists largely of a positive potential whereas that evoked from the dorsal

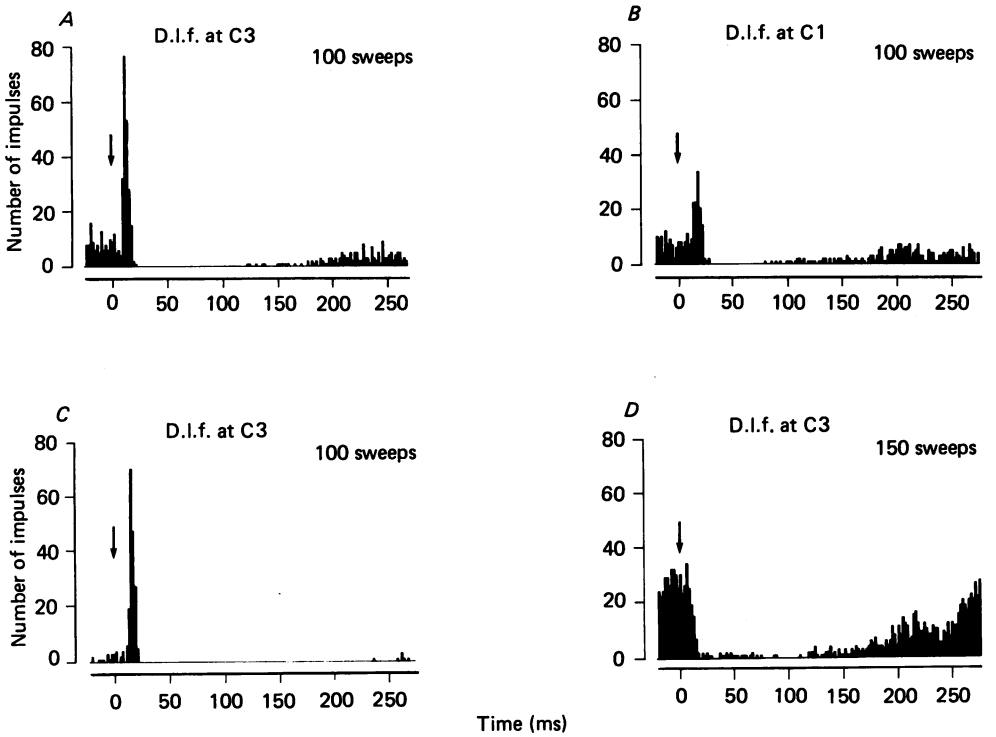

Fig. 8. Time course of inhibitory actions on p.s.d.c. units revealed by peri-stimulus time histograms accumulated in response to stimulating the dorsolateral funiculus (d.l.f.) at C3 (*A*, *C* and *D*) and at C1 (*B*). *A*, *B* and *C* are responses from the same unit; *A* and *B* with background discharge elevated with a sustained pinch and *C* without. *D* shows the time course of inhibition in a p.s.d.c. unit which showed no facilitatory response. Impulses accumulated in 2 ms time bins.

columns has a large negative component followed by a positive one, the latter being similar to the positive wave evoked from the dorsolateral funiculus.

The field potentials illustrated by Lu *et al.* (1983) are unusual in that, although the initial negativity reverses at about 2 mm (as would be expected) the following positivity, which is obvious in our records as it is in records produced by stimulating peripheral cutaneous or mixed nerves, is inconspicuous in surface records and only apparent at depths of 1·0 and 1·5 mm whereas a clear reversal at between 0·4 and 0·8 mm would be expected (see, for example, Coombs, Curtis & Landgren, 1956; Eccles, Kostyuk & Schmidt, 1962). We conclude that the test for stimulus spread, used by Lu *et al.* (1983, 1985) was insensitive and may have missed such spread.

In the present experiments, with conditions chosen such that there was a bias in favour of stimulus spread between the dorsal columns and dorsolateral funiculus, we could find no evidence that neurones belonging to the p.s.d.c. or s.c.t. systems had bifurcating axons with branches ascending both the ipsilateral dorsolateral funiculus and the dorsal columns and reaching the stimulation site at C3. For our sample of p.s.d.c. cells the results were unequivocal. None of the units identified as belonging to the p.s.d.c. system could be antidromically excited from the dorsolateral funiculus

at C3 even at voltages some 70 times the threshold for activation from the dorsal columns. For our sample of s.c.t. neurones, however, about one-half could be antidromically excited from both the dorsolateral funiculus and dorsal columns, but, for the following reasons, we believe this can be accounted for by stimulus spread: (1) all but two of these units required stimuli to the dorsal columns that were considerably greater than the threshold required to activate them from the dorsolateral funiculus. (2) All but three of these units could also be activated by stimuli to the dorsal columns at a location above the level of the dorsal column lesion. Once again this required levels of stimuli far greater than the threshold for antidromic activation from the dorsolateral tract, and in this case could only be due to stimulus spread. (3) None of the units identified as belonging to the p.s.d.c., and recorded either in the dorsal columns or dorsal horn, could be antidromically activated by stimuli applied to the dorsolateral funiculus at C3. This was probably because this stimulus location was also above the level of the dorsal column lesion, and the effects of stimulus spread from the dorsolateral funiculus to the dorsal columns would have been contained above the transection. (4) When units could be antidromically excited from both the dorsal columns and the dorsolateral funiculus it was a striking fact that the antidromic latencies from each site were almost identical; this result is similar to that reported by Lu *et al.* (1985). This would mean that if there are neurones with bifurcating axons then each axonal branch would be of the same diameter as the other. There are, therefore, no obvious reasons why one branch should be any less excitable than the other and yet s.c.t. units that were antidromically activated from the dorsal columns required stimuli considerably greater than the threshold to activate them from the dorsolateral funiculus. (5) In their experiments, Lu *et al.* (1985) used search stimuli of at least 0·2 ms in duration and 30 V in amplitude. In the present experiments, however, we used stimuli of around 3 V (0·1 ms duration) to search systematically for units belonging to the spinocervical tract. This level of stimulation was chosen because it had been previously shown by combined electrophysiological and retrograde labelling with HRP (Brown *et al.* 1980) that it was capable of activating all s.c.t. cells in the lumbosacral dorsal horn. We do not believe, therefore, that a population of units was missed by not using search stimuli of greater strength.

From the present results we conclude that the s.c.t. and p.s.d.c. neurones form in substantial part, if not entirely, separate populations of neurones. The cell bodies of these two sets of neurones are, however, located in the same region of the dorsal horn (Craig, 1976; Rustioni & Kaufman, 1977; Brown *et al.* 1980; Enevoldson, 1982; Bennett, Seltzer, Lu, Nishikawa & Dubner, 1983). Furthermore, Jiao *et al.* (1984) reported that injection of different fluorescent dyes into the lateral cervical nucleus and dorsal column nuclei led to double staining of dorsal horn neurones, but their results are not conclusive. It is known that there are neurones with axons projecting in the dorsolateral funiculus which nevertheless terminate in the dorsal column nuclei (Dart & Gordon, 1973; Gordon & Grant, 1982). If these axons give off collateral branches to the lateral cervical nucleus, as suggested by Craig & Tapper (1978), then such double labelling would be expected.

Effects on p.s.d.c. neurones from the cervical dorsolateral funiculus

The present results confirm and extend previous observations. Jankowska *et al.* (1979) showed that stimulation of the ipsilateral dorsolateral funiculus at C3 may produce excitatory post-synaptic potentials in p.s.d.c. neurones and that stimulation at C1 is much less effective. Jankowska *et al.* (1979) concluded that these potentials were, in all probability, being evoked via spinal collateral branches of axons of s.c.t. neurones which terminate in the l.c.n. at C1–2. Maxwell & Koerber (1986) have provided direct evidence for connexions between the two sets of neurones as required by the hypothesis of Jankowska *et al.* (1979) but Svensson, Westman & Rastad (1985) described neurones in the l.c.n. with descending axons (as far as L6 at least). Obviously the effects described by Jankowska *et al.* (1979) and those described in the present paper may have been via either the s.c.t. or the newly described descending pathway. Since the direct connexions do exist it seems reasonable to suppose that some of the effects from the dorsolateral funiculus at C3 are evoked via the spinocervical tract.

The excitatory effects evoked from the dorsolateral funiculus at C3 may be powerful enough to cause p.s.d.c. cells to fire and certainly produce a facilitation strong enough to be observed against the resting discharge. Not all p.s.d.c. cells show this excitation (about one-third of the sample recorded by Jankowska *et al.* and about one-half of the present sample) and there were no obvious differences in the receptive fields between those that showed the effect and those that did not, although there was a suggestion that those showing the facilitation had faster conducting axons.

Not all s.c.t. cells can be involved in this action on p.s.d.c. neurones. About 30% of s.c.t. cells do not have collateral axons near the segmental level of their cell bodies (Brown *et al.* 1977). This group includes those s.c.t. cells excited by hair movement alone, whereas those cells responding in addition to noxious mechanical stimuli have collateral axons that are given off near the cell body. In the present experiments all those p.s.d.c. cells excited from the dorsolateral funiculus were also excited by heavy pressure and pinch of the skin. It cannot be concluded, however, that their noxious mechanical input arrived solely via the s.c.t., such input may not be powerful enough to drive the observed responses. The results emphasize, however, that there is close interaction between these two major ascending pathways at the earliest opportunity.

The results also confirm that p.s.d.c. neurones may be excited from the ipsilateral dorsolateral funiculus at C1. Jankowska *et al.* (1979) showed that stimulation of the pyramids produced excitatory post-synaptic potentials in some p.s.d.c. cells. There are, however, a number of possible mechanisms for the present observations, these include stimulation of descending axons in addition to those of the corticospinal tract and antidromic activation of ascending axons with collateral branches terminating either directly on p.s.d.c. cells or on interneurones in the pathways afferent to them.

Finally, we have shown that p.s.d.c. neurones may be profoundly inhibited by stimulation of the ipsilateral dorsolateral funiculus at C3 and C1. This inhibition lasted for up to about 300 ms. We have no evidence about the possible mechanisms of this inhibition but Jankowska *et al.* (1979) did not describe inhibitory post-synaptic potentials in response to similar stimulation. The time course of the effect is similar to that of presynaptic inhibitory actions in the spinal cord. Furthermore, since

similar effects were evoked from C3 and C1 it is most unlikely that the effects were elicited either via the s.c.t. or through the system of descending axons originating in the l.c.n. (Svensson *et al.* 1985). It is known that the modality and receptive field characteristics of p.s.d.c. cells can be profoundly modified by descending inhibitory mechanisms (Noble & Riddell, 1985) and it seems most likely that a set of descending axons were being stimulated in the present experiments. Nishikawa, Bennett, Ruda, Lu & Dubner (1983) have provided some evidence for a direct serotoninergic innervation of p.s.d.c. cells. Whether or not such innervation is responsible for the inhibitory actions observed in the present experiments remains to be seen.

Conclusions

When the present results are considered together with previous results on the receptive field properties, the dendritic tree anatomy and the ultrastructure of synapses on presumed s.c.t. and p.s.d.c. neurones, it is reasonable to conclude that these two ascending pathways arise, in substantial part, from different populations of cells in the dorsal horn. If there are any cell bodies common to the two projections then these can only comprise a relatively small number of the total population of s.c.t. or p.s.d.c. neurones and could not be clearly demonstrated in the present study. Nevertheless the two projections are not independent of each other at the level of the spinal cord. The present study does provide evidence to support the suggestion that some s.c.t. cells make effective excitatory synapses with p.s.d.c. neurones.

This work was supported by a Programme Grant from the M.R.C. We wish to thank Miss S. Brass, Mr J. A. Campbell, Mrs A. Corbett and Mrs J. Wood for assistance. The animals were held in the Wellcome Animal Research Laboratories, Faculty of Veterinary Medicine.

REFERENCES

ANGAUT-PETIT, D. (1975). The dorsal column system. II. Functional properties and bulbar relay of the postsynaptic fibres of the cat's fasciculus gracilis. *Experimental Brain Research* **22**, 471–493.

BANNATYNE, B. A. (1984). The fine structure of the postsynaptic dorsal column system in the cat. Ph.D. Thesis, University of Edinburgh.

BANNATYNE, B. A., BROWN, A. G., FYFFE, R. E. W. & MAXWELL, D. J. (1982). Ultrastructure of post-synaptic dorsal column neurones in the cat. *Journal of Physiology* **332**, 58–59*P*.

BENNETT, G. J., NISHIKAWA, N., LU, G.-W., HOFFERT, M. J. & DUBNER, R. (1984). The morphology of dorsal column postsynaptic spinomedullary neurons in the cat. *Journal of Comparative Neurology* **224**, 568–578.

BENNETT, G. J., SELTZER, Z., LU, G.-W., NISHIKAWA, N. & DUBNER, R. (1983). The cells of origin of the dorsal column postsynaptic projection in the lumbosacral enlargements of cats and monkeys. *Somatosensory Research* **1**, 131–149.

BROWN, A. G. (1971). Effects of descending impulses on transmission through the spinocervical tract. *Journal of Physiology* **219**, 103–125.

BROWN, A. G. (1981). *Organization in the Spinal Cord. The Anatomy and Physiology of Identified Neurones*. Berlin, Heidelberg, New York: Springer-Verlag.

BROWN, A. G., BROWN, P. B., FYFFE, R. E. W. & PUBOLS, L. M. (1983). Receptive field organization and response properties of spinal neurones with axons ascending the dorsal columns in the cat. *Journal of Physiology* **337**, 575–588.

BROWN, A. G. & FRANZ, D. N. (1969). Responses of spinocervical tract neurones to natural stimulation of identified receptors. *Experimental Brain Research* **7**, 231–249.

BROWN, A. G. & FYFFE, R. E. W. (1981). Form and function of dorsal horn neurones with axons ascending the dorsal columns in cat. *Journal of Physiology* **321**, 31–47.

BROWN, A. G., FYFFE, R. E. W., NOBLE, R., ROSE, P. K. & SNOW, P. J. (1980). The density, distribution and topographical organization of spinocervical tract neurones in cat. *Journal of Physiology* **300**, 409–428.

BROWN, A. G., FYFFE, R. E. W., NOBLE, R. & ROWE, M. J. (1984). Effects of hind limb nerve section on lumbosacral dorsal horn neurones in the cat. *Journal of Physiology* **354**, 375–394.

BROWN, A. G., ROSE, P. K. & SNOW, P. J. (1977). The morphology of spinocervical tract neurones revealed by intracellular injection of horseradish peroxidase. *Journal of Physiology* **270**, 747–764.

COOMBS, J. S., CURTIS, D. R. & LANDGREN, S. (1956). Spinal cord potentials generated by impulses in muscle and cutaneous afferent fibres. *Journal of Neurophysiology* **19**, 452–467.

CRAIG, A. D. (1976). Spinocervical tract cells in cat and dog labelled by the retrograde transport of horseradish peroxidase. *Neuroscience Letters* **3**, 173–177.

CRAIG, A. D. & TAPPER, D. N. (1978). Lateral cervical nucleus in the cat: fundamental organization and characteristics. *Journal of Neurophysiology* **41**, 1511–1534.

DART, A. M. & GORDON, G. (1973). Some properties of spinal connections of the cat's dorsal column nuclei which do not involve the dorsal columns. *Brain Research* **58**, 61–68.

ECCLES, J. C., KOSTYUK, P. G. & SCHMIDT, R. F. (1962). Central pathways responsible for depolarization of primary afferent fibres. *Journal of Physiology* **161**, 237–257.

ENEVOLDSON, T. P. (1982). Structural and functional inter-relationships of the dorsal column nuclei with other somaesthetic mechanisms in the cat. D.Phil. Thesis, University of Oxford.

GORDON, G. & GRANT, G. (1982) Dorsolateral spinal afferents to some medullary sensory nuclei. An anatomical study in the cat. *Experimental Brain Research* **46**, 12–23.

HONGO, T., JANKOWSKA, E. & LUNDBERG, A. (1968). Post-synaptic excitation and inhibition from primary afferents in neurones of the spinocervical tract. *Journal of Physiology* **199**, 569–592.

JANKOWSKA, E., RASTAD, J. & ZARZECKI, P. (1979). Segmental and supraspinal input to cells of origin of non-primary fibres in the feline dorsal column. *Journal of Physiology* **290**, 185–200.

JIAO, S. S., ZHANG, G. F., LIU, Y. J., WANG, Y. S. & LU, G.-W. (1984). Double labelling of cat spinal dorsal horn neurons with fluorescent substances. *Pain* **2**, suppl. S130.

LU, G.-W., BENNETT, G. J., NISHIKAWA, N. & DUBNER, R. (1985). Spinal neurons with branched axons traveling in both the dorsal and dorsolateral funiculi. *Experimental Neurology* **87**, 571–577.

LU, G.-W., BENNETT, G. J., NISHIKAWA, N., HOFFERT, M. J. & DUBNER, R. (1983). Extra- and intracellular recordings from dorsal column postsynaptic spinomedullary neurons in the cat. *Experimental Neurology* **82**, 456–477.

MAXWELL, D. J., FYFFE, R. E. W. & BROWN, A. G. (1982). Fine structure of spinocervical tract neurones and the synaptic boutons in contact with them. *Brain Research* **233**, 394–399.

MAXWELL, D. J., FYFFE, R. E. W. & BROWN, A. G. (1984). Fine structure of normal and degenerating primary afferent boutons associated with characterized spinocervical tract neurons in the cat. *Neuroscience* **12**, 151–163.

MAXWELL, D. J. & KOEBER, H. R. (1986). Fine structure of collateral axons originating from feline spinocervical tract neurons. *Brain Research* **363**, 199–203.

NISHIKAWA, N., BENNETT, G. J., RUDA, M. A., LU, G.-W. & DUBNER, R. (1983). Immunocytochemical evidence for a serotoninergic innervation of dorsal column postsynaptic neurons in the cat and monkey: light- and electron-microscopic observations. *Neuroscience* **10**, 1333–1340.

NOBLE, R. & RIDDELL, J. S. (1984). Inhibitory fields in the post-synaptic dorsal column system of the chloralose-anaesthetized cat. *Journal of Physiology* **357**, 32*P*.

NOBLE, R. & RIDDELL, J. S. (1985). Effects of descending influences on the high-threshold excitatory fields of post-synaptic dorsal column neurones in the chloralose-anaesthetized cat. *Journal of Physiology* **364**, 43*P*.

RUSTIONI, A. & KAUFMAN, A. B. (1977). Identification of cells of origin of non-primary afferents to the dorsal column nuclei of the cat. *Experimental Brain Research* **27**, 1–14.

SVENSSON, B., WESTMAN, J. & RASTAD, J. (1985). Light and electron microscopic study of neurones in the feline lateral cervical nucleus with a descending projection. *Brain Research* **361**, 114–124.

UDDENBERG, N. (1968). Functional organization of long, second-order afferents in the dorsal funiculus. *Experimental Brain Research* **4**, 377–382.