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I. Hammar · Z. Szabo Läckberg · E. Jankowska

New observations on input to spino-cervical tract neurons from muscle afferents

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Abstract Peripheral input to spino-cervical tract (SCT) neurons located in the L4 and L5 segments of the cat spinal cord was investigated using both extracellular and intracellular recording. The main aim was to find out whether midlumbar SCT neurons are excited monosynaptically not only by cutaneous afferents but also by group II muscle afferents, as in the sacral segments but apparently not in the caudal lumbar segments. Input from group II muscle afferents was found in 73% of investigated neurons; the latencies of excitation by group II afferents were compatible with a monosynaptic coupling between these afferents and 62% of neurons. The majority of the midlumbar SCT neurons were excited by group II afferents of the quadriceps and deep peroneal nerves. The predominant monosynaptic input from cutaneous afferents to the same neurons was from the saphenous nerve.

Key words Spinal cord · Ascending tracts
Spinocervical neurons · Group II muscle afferents
Cat

Introduction

The spinocervical tract (SCT) is one of the major spinal ascending tracts that forward information from skin receptors to the cerebral cortex, the information being relayed in the lateral cervical nucleus and the thalamus. The SCT neurons are generally thought to be primarily involved in processing information from the lowest threshold touch receptors (in particular hair follicle afferent fibres (Brown et al. 1980; for review see Brown 1981) which excite them monosynaptically (Hongo et al. 1968). Many of these neurons are, however, also excited by skin nociceptors (Brown and Franz 1969; Cervero et al. 1977), most likely polysynaptically (Hongo et al. 1968)). With respect to their input from muscle receptors, SCT neurons were first found to be affected by group III and IV muscle afferents (Hongo et al. 1968; Hamann et al. 1978), possibly via the same polysynaptic pathways that are activated by skin nociceptors. Subsequent studies have in addition revealed that some of these neurons are co-excited by relatively low threshold (group II) muscle afferents: via undefined pathways (Hamann et al. 1978), disynaptically (Harrison and Jankowska 1984) or monosynaptically (Riddell et al. 1994). However, the monosynaptic coupling between group II muscle afferents and SCT neurons has so far only been found for those located in the sacral segments (within a length of the spinal cord containing the pudendal motor nucleus and just rostral to it). The present study was therefore undertaken in order to investigate whether monosynaptic input from group II muscle afferents characterises only the sacral or also the more rostrally located SCT neurons. Since no indications for such an input were found within the lumbo-sacral enlargement (Hongo et al. 1968; Harrison and Jankowska 1984) the study focused on SCT neurons located in the L4–5 segments in which group II synaptic actions are particularly pronounced (Edgley and Jankowska 1987). Also taken into consideration was the possibility that SCT neurons might previously have been included in the sample of unidentified midlumbar dorsal horn neurons co-excited by group II and cutaneous afferents described by Edgley and Jankowska (1988).

I. Hammar · Z. S. Läckberg · E. Jankowska (✉)
Department of Physiology, University of Göteborg,
Medicinaregatan 11, S-413 90 Göteborg, Sweden,
FAX no: +46 −31-7733512,
e-mail: Elzbieta. Jankowska @ physiol. gu. se

Materials and methods

The experiments were performed on three cats weighing 2.5–4.4 kg. The cats were deeply anaesthetised. Anaesthesia was induced with a single dose of sodium pentobarbital (44 mg/kg i.p.) and maintained with several doses of chloralose (up to 40 mg/kg i.v.). The depth of anaesthesia was assessed by monitoring withdrawal, corneal and pupillary reflexes during surgery and the diameter of pupils and blood pressure during the experiments (when the animals were paralysed and artificially ventilated). The mean

blood pressure was kept above 90 (usually 110–130) mg Hg and the end tidal CO_2 was maintained close to 4% by adjusting the artificial respiration and the rate of infusion of a solution of 100 mM sodium bicarbonate containing 5% glucose. The core temperature was maintained at 37–38° C and the temperature of the paraffin pools at 35–37° C. The experiments were approved by the local ethical committe and the principles of laboratory animal care as specified by the NIH (1985) were followed.

The spinal cord was exposed from the fourth lumbar to the sacral segments and at the levels of the 13th thoracic (Th13) and 1st and 3rd cervical (C1 and C3) segments. The dura matter was left intact and only small holes were made in it at the sites of microelectrode penetrations at the lumbar level. Records from the cord dorsum were taken transdurally and the surface of the lateral funiculi at the Th12–13 and C1–3 levels was likewise stimulated transdurally (with 0.2 ms current pulses). For further details of the experimental procedures see Riddell et al. (1994)

The neurons were recorded by using microelectrodes filled with a 2 M solution of potassium citrate (tip diameter of 1–2 μm and resistance of 2.5–4.0 MΩ). They were identified taking into account that either all of their terminal axonal branches, or at least some of them, should terminate within the C2–3 segments (for references see Brown 1973, 1981). The following neurons were therefore classified as SCT neurons: (i) neurons that were antidromically activated by stimuli (usually less than 250 μA) applied at the surface of the lateral funiculus caudal to the C3 dorsal root but were not activated by stimuli (0.5–1 mA) applied rostral to the C1 dorsal root ($n=10$; Fig. 1A, B; Fig. 2C, D), or (ii) neurons that were activated by both C3 and C1 stimuli but at a higher threshold from the C1 level and appeared to conduct at a slower conduction velocity between the C3 and C1 segments ($n=23$). Constant latency (see Figs. 1–4) and collision with orthodromically evoked responses were used as criteria for the antidromic activation.

In order to increase the reliability of the identification of SCT neurons the following precautions were taken. Firstly, the dorsal columns were transected caudal to the C3 segment in order to avoid inclusion of any neurons with axons in the dorsal columns which might be activated by spread of current by stimuli applied to the lateral funiculus at the C3 segment. Secondly, it was verified that conditions of stimulation at the C1 level were satisfactory before considering a difference in threshold of the antidromic activation of a presumed SCT neuron by C3 and C1 stimuli was genuine. This was done by comparing thresholds of activation of dorsal spino-cerebellar tract (DSCT) neurons from the C3 and C1 segments, which were expected to be practically the same, as illustrated in Fig. 1A, B (bottom traces) and Fig. 1C, D. The DSCT neurons were usually recorded simultaneously with the SCT neurons, another microelectrode being placed in Clarke's column. Thirdly, SCT neurons were differentiated from other ascending tract neurons with axons in the lateral funiculus by C1 thresholds that were at least three times higher than C3 thresholds ($n=11$; with an example in top records in Fig. 1A, B) or by the latencies of their antidromic activation from the C1 segment that were at least 0.5 ms longer even when C1 thresholds were only 1.7–3.0 times higher ($n=12$; as in cells illustrated in Figs. 3, 4). These additional criteria were somewhat arbitrary, but they applied to distinct subpopulations of the tested neurones which were dichotomously distributed. In addition, these criteria may be justified as follows. Assumming that the C3 threshold stimuli were 150–200 μA, three times stronger stimuli would be 450–600 μA and, by extrapolating the data of Gustafsson and Jankowska (1976), effects of such stimuli may be attributed to the spread of current over distances of at least 20–30 mm, while distances between the rostral part of the C1 segment and the area of termination of SCT neurons in the lateral cervical nucleus would be less than that. Only marginal differences in the latency of activation of the neurons by C3 and C1 stimuli would support this contention. Latencies of the antidromic activation by C1 stimuli which were ≥0.5 ms longer, and were combined with 1.7–3.0 times higher thresholds appeared, on the other hand, to be more compatible with activation of smaller ascending axon collaterals of the same

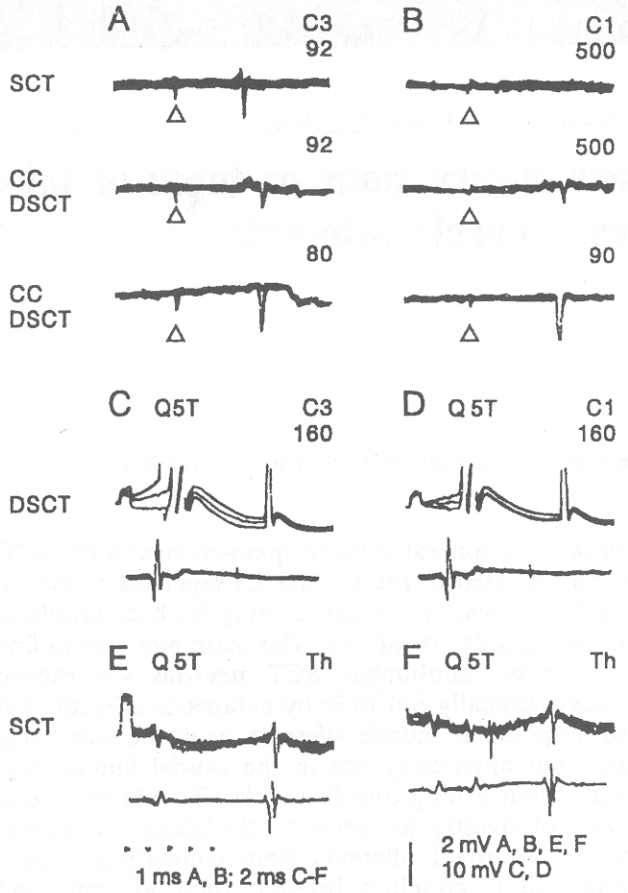

Fig. 1 Comparison of responses of a spinocervical tract neurone (SCT, *top records* in *A*, *B* and *E*, *F*), two Clarke's column dorsal spino-cerebellar neurons (CC-DSCT, *middle* and *bottom records* in *A*, *B*) and a dorsal horn dorsal spino-cerebellar tract neuron (DSCT, *top records* in *C*, *D*; the only intracellular records in this figure). Activation of the SCT neuron by group II afferents of the Q nerve (only following the second stimulus) is illustrated in *E*, *F* and of the dorsal horn DSCT neuron at the beginning of records *C* and *D*. Antidromic responses of the same neurons to stimuli applied at a thoracic (Th) or cervical (C) levels are shown for comparison in the *right parts* of the records. The *bottom records* in *C–F* are from the cord dorsum. Note that of the simultaneously recorded SCT and CC-DSCT neurons (*A*, *B*; *top* and *middle records*) only the latter was activated by C1 stimuli. *Arrow heads* indicate shock artefacts. In this and the following figures the intensity of the stimuli applied to the lateral funiculus at the C3 and C1 segments is in μA, while that applied to peripheral nerves is expressed in multiples of threshold for the most sensitive fibres in a given nerve. Voltage calibration is for the microelectrode records only

fibres that were stimulated by C3 stimuli, other collaterals having terminated within the lateral cervical nucleus. Differences in latencies greater than ≥0.5 ms were taken to indicate a lower conduction velocity between the C3 and C1 segments, since all neurons with similar C1 and C3 thresholds were activated from the C1 segment with only 0.2–0.4 ms longer latencies and conduction velocities of SCT and DSCT neurones greatly overlap. The 0.5 ms differences were in addition found when the distances at which the C1 stimuli activated the fibres must have been shorter because of the higher intensity of these stimuli and a more marked spread of current. In view of the unknown distances between the C1 electrodes and the branching sites of the fibres or the degree of the spread of current in these cases, no attempts were made to esti-

mate the conduction velocity between the C3 and C1 segments. Neurons which did not fulfil these criteria were considered as dorsal horn DSCT neurons (Edgley and Jankowska 1988) or other ascending tract neurons.

In order to investigate the peripheral input to the SCT neurons, a number of left hind limb muscle and skin nerves were dissected free. These included: the quadriceps (Q), sartorius (Sart), gracilis (Grac), posterior biceps and semitendinosus (PBST), anterior biceps and semimembranosus (ABSM), gastrocnemius and soleus (GS), plantaris (PL), deep peroneal (DP, i.e. tibialis anterior and extensor digitorum longus from which the mixed nerve branch to the extensor digitorum brevis was removed), cutaneous femoris (CF), saphenous (Saph), the caudal branch of sural (Sur) and the superficial peroneal (SP) nerves. The nerves were stimulated with 0.1 ms rectangular current pulses (single or double stimuli, 2.5 or 3.3 ms apart). Stimulus strengths are expressed relative to the threshold (T) for the most excitable afferents in the nerve. Effects of stimuli applied to muscle nerves with intensity exceeding 1.5 T were considered to be due to group II afferents since thresholds of the most excitable group II afferents are of about 1.5 T (Jack, 1978). However, since stimuli of 1.5–2.5 T may excite both group I and group II afferents, only effects of stimuli supramaximal for group I afferents (usually more than 1.7–2.5 T, depending on the nerve and defined in each experiment) were attributed to group II afferents.

Results

Thirty-three neurons recorded from in the dorsal horn of the L4 and L5 segments fulfilled the above outlined criteria and were classified as SCT neurons. All of these were recorded at locations where large focal field potentials were evoked both by skin and group II muscle afferents (mainly Saph, SP, Q and DP), at depths not exceeding 2 mm from the surface of the spinal cord. Twenty-one neurons were recorded from only extracellularly, four neurons only intracellularly and eight neurons both extra and intracellularly. Examples of records from the latter are in Figs. 2 and 3.

The neurons fell into three groups with regard to the input from skin and muscle afferents: a group of 20 (61%) neurons excited by both muscle group II afferents and cutaneous afferents, a group of 9 (27%) neurons excited only by stimulation of skin nerves and a group of 4 (12%) neurons excited only by stimulation of muscle nerves. The actual proportion of neurons with input from both the skin and muscle afferents might nevertheless have been larger, since effects of not all of the muscle and skin hind limb nerves were tested. Furthermore, in the cases with only extracellular records, weak synaptic actions might have been undetected. Only some of the neurons were activated by single stimuli applied to muscle nerves and apparently weaker input to other cells required temporal summation of actions of two stimuli to discharge them, as illustrated in Fig. 1E, F; even weaker input might only have been detected in intracellular records.

The main sources of input from muscle afferents were group II afferents of the Q and DP nerves. Stimulation of these two nerves at 5 T induced action potentials and/or EPSPs in 73% of neurons with input from muscle nerves, Q afferents exciting 61% and DP afferents

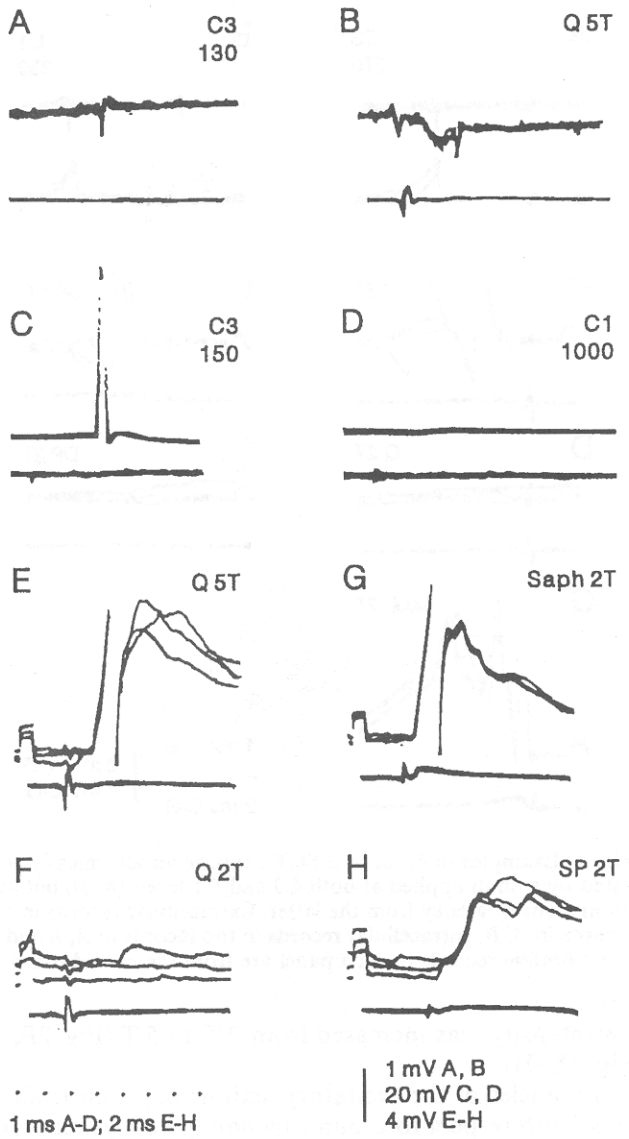

Fig. 2 Examples of input to a SCT neuron antidromically activated by stimuli applied at a C3 but not at a C1 level (**A, C, D**). Extracellular records in *A,B*; Intracellular records in *C–H*. *Bottom records* in each pair are from the cord dorsum. Note that the 2 T stimuli applied to the Q nerve (subthreshold for group II afferents) were not effective

45%. Smaller proportions of neurons were excited by stimulation of the GS (18%), PBST (9%), Grac (6%), P1(3%) and Sart (3%) nerves, individual neurons being co-excited by afferents of one to five of these nerves. Stimuli below threshold for group II afferents (about 1.5–2 T depending on the nerve; Jack 1978) were ineffective. Since the effective stimuli had in addition to be supramaximal for group I afferents (usually 2.0–2.5 T for Q and 1.7–2.0 T for other nerves) present observations are in agreement with previous reports that SCT neurons are not affected by group I muscle afferents (Hongo et al. 1968; Hamann et al. 1978; Harrison and Jankowska 1984; Riddell et al. 1994). This is illustrated with the appearence of the EPSPs only when the stimu-

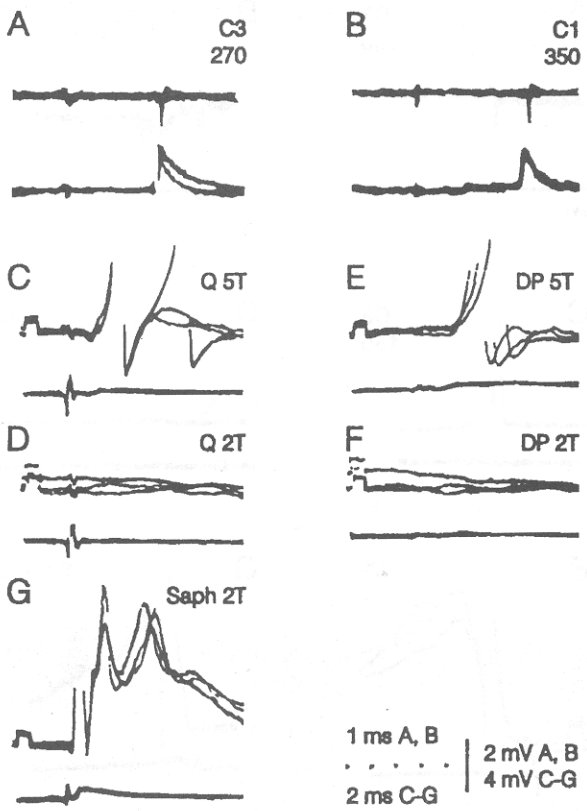

Fig. 3 Examples of input to a SCT neurone antidromically activated by stimuli applied at both C3 and C1 levels (**A, B**) but at a 0.9 ms longer latency from the latter. Extracellular records in top records in *A, B*; Intracellular records in top records in *A, B* and in *C–G*. *Bottom* records in each panel are from the cord dorsum

lus intensity was increased from 2 T to 5 T (Fig. 2E, F, Fig. 3E–H).

Latencies of the excitatory actions of group II afferents (with respect to group I incoming volleys recorded from the cord dorsum) varied depending on the peripheral nerve. However, considering that the majority of group muscle II afferents conduct at a slower velocity than group I muscle afferents, the following latencies were considered to be compatible with monosynaptically evoked actions: up to 2.1 ms for Q, Sart, Grac and PBST afferents and up to 2.5 ms for DP and GS afferents (see Edgley and Jankowska 1987). Totally, 62% of SCT neurons were excited at such latencies. Some of the longer latencies of the extracellularly recorded actions might likewise be compatible with monosynaptic actions, spike potentials often being induced with an additional delay of 0.2–0.5 ms or more (see Fig. 2B, E). The majority of the present sample of SCT cells excited by group II muscle afferents may thus be concluded to be excited monosynaptically.

The main sources of input from cutaneous afferents were afferents of the Saph nerve. These afferents excited all but one of the SCT neurons with input from skin nerves. Some neurons (27%) were excited only by cutaneous afferents of the Saph nerve, while other neurons were co-excited by Saph and SP (24%), Saph, Sur and SP (18%), Saph, Sur, SP and CF (15%) or Saph, Sur and CF (3%).

The latencies of synaptic actions evoked from the Saph nerve ($\leq$1 ms in intracellular records and $\leq$1.3 ms in extracellular records) were compatible with monosynaptically evoked responses in 58% of the SCT neurons. These are illustrated in Figs. 2G and 3G. Similar proportions of neurons co-excited by skin and muscle nerves, or excited only by skin nerves, showed such short latency responses. Synaptic actions evoked by stimulation of the SP nerve were sometimes (in two neurons) evoked at similarly short latencies, but both SP and other skin nerves most often evoked them at longer latencies (example in Fig. 2H).

The SCT neurons of the present sample were not only co-excited but also co-inhibited by muscle group II and cutaneous afferents. IPSPs were evoked in them only by stimuli supramaximal for group I muscle afferents (by more than 2 T stimuli), as illustrated in Fig. 4E–H, and by both low and higher threshold cutaneous afferents. The latencies of IPSPs evoked from the most effective nerves (Saph, Q, DP and Sart) were often only about 1.0 ms longer than of the earliest components of EPSPs evoked from the same nerves in the same (see Fig. 4C) or other neurons. These IPSPs were therefore likely to be evoked disynaptically and those from other nerves polysynaptically.

Discussion

Differentiation of input to SCT neurons at different locations

The results of the present study show that group II muscle afferents provide monosynaptic input to a high proportion of both midlumbar and sacral SCT neurons. By recording from axons of SCT neurons of unspecified location in the lateral funiculus, Hamann et al. (1978) found group II input in only 15% of their total sample (the synaptic coupling between group II afferents and these SCT neurons not having been specified, because they were activated by natural stimuli) and disynaptic input from group II afferents was reported to be found in less than one half of L6–7 SCT neurons by Harrison and Jankowska (1984). In contrast, monosynaptic input from group II afferents was found in two thirds of the extracellularly recorded and in all of the intracellularly recorded sacral SCT neurons (Riddell et al. 1994); similarly high proportions of midlumbar neurones were found in this study. The generally higher proportions of SCT neurons with group II input found by Harrison and Jankowska (1984), Riddell et al. (1994) and in the present study than those found by Hamann et al. (1978) might be explained by more synchronous actions of electrical than of natural stimuli and by a more extensive use of intracellular than of extracellular records. The use of not only single but also double and triple stimuli applied to group II afferents must also have as-

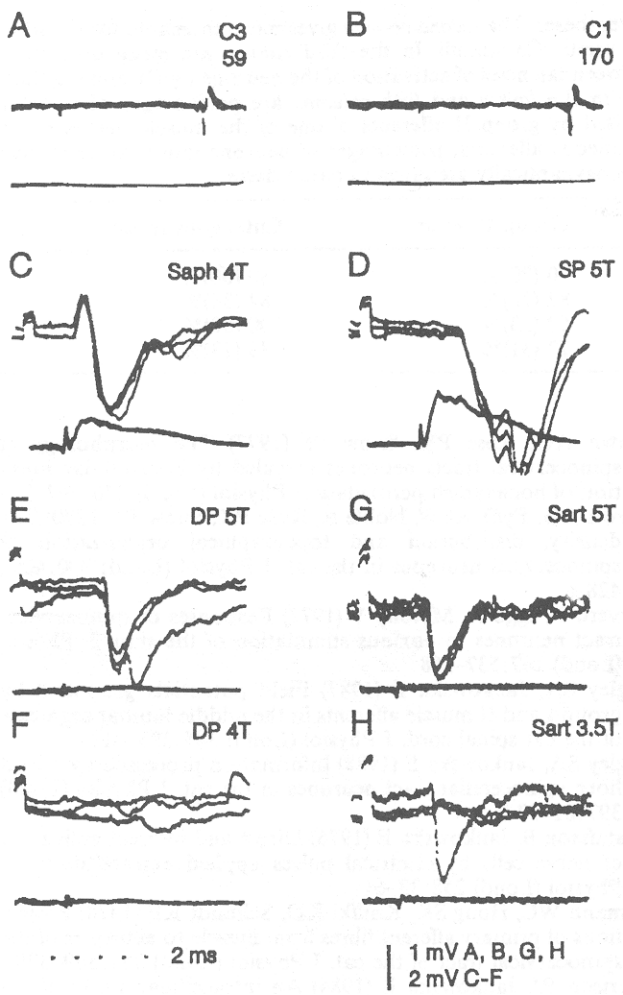

Fig. 4 Examples of IPSPs evoked in a SCT neuron antidromically activated by stimuli applied both at C3 and at C1 levels (at a higher threshold) which was apparently excited only by cutaneous afferents. **A, B** extracellular records of antidromic spikes (*top*), and records from the cord dorsum (*bottom*). **C–H** intracellular records of PSPs evoked by stimulation of peripheral nerves (*top*) and records from the cord dorsum of incoming volleys (*bottom*)

sisted in the disclosure of di- and trisynaptically evoked actions of these afferents. However, it is unclear why monosynaptic actions of group II afferents have not been found in SCT neurons located in the L6 and L7 segments. The most likely possibility to consider is that a smaller proportion of SCT neurons of these segments are specialised in integrating information provided by touch and muscle receptors than in either more caudal or more rostral segments, and that such neurons did not happen to be included in the previously investigated samples. One might also consider that the shortest latencies of synaptic actions of group II afferents found by Harrison and Jankowska (1984) would be compatible with monosynaptic actions of slower conducting group II afferents, as well as with disynaptic coupling between group II afferents and SCT neurons, which at that time appeared to be more likely. It is therefore proposed that the probability of monosynaptic actions of group II afferents on SCT neurons is higher within the sacral and midlumbar segments than within the L6 and L7 segments, but perhaps without sharp borderlines between neurons located within these segments.

Comparison between midlumbar SCT and DSCT neurons

One of the major problems of this study was the differentiation between the midlumbar SCT and the dorsal horn DSCT neurons, since the patterns of input to these two populations of neurons appear to be indistinguishable. As described above, the criteria for the identification of the SCT neurons were revised in order to allow us to investigate SCT neurons with and without axon collaterals projecting rostral to the C3 segment. The tacit assumption of both this and the previous studies was that DSCT neurons do not give off any major axon collaterals between the C3 and C1 segments and that both the conduction velocity and the excitability of their axons are the same caudal and rostral to the C3 segment. On the basis of this assumption, we pooled together the data for SCT neurons that were not activated by stimuli applied at the C1 level, for neurons that were activated by C1 stimuli at at least three times higher threshold and for neurons that were activated by C1 stimuli at 1.7–3.0 times higher threshold but at least 0.5 ms longer latency. The three sets of data have nevertheless been separated in Table 1 which shows that there are no major qualitative differences in the type of input to these neurons, and that a considerable proportion of all of them are co-excited by group II and cutaneous afferents. It should be noted in particular that the highest proportion of neurons with group II input – both monosynaptic and at a longer latency – was found in the group of neurons that were identified as SCT neurons with the greatest confidence (top row in the Table 1). The table also shows that no major differences were found between the three subpopulations of neurons classified as SCT neurons, and neurons that were activated at a similar threshold from both the C3 and C1 segments and at latencies of activation from the C1 segment which were longer by only 0.2–0.4 ms and, in view of the observations of Edgley and Jankowska (1988), are likely to be the dorsal horn DSCT neurons. The SCT neurons might thus be used in parallel with spino-cerebellar neurons to forward information on changes in muscle length and on the state of activity in spinal neuronal networks (see Lundberg 1971) to supraspinal neurons; the former primarily to the cerebral cortex and the latter to the cerebellum.

Comparison with midlumbar interneurons

Similarities in the input to SCT neurons and to dorsal horn interneurons with group II input were found in the sacral segments (Riddell et al. 1994) and the present

Table 1 Comparison of input from group II and cutaneous afferents to four groups of midlumbar neurons: SCT neurons that were antidromically activated from the C3 but not from the C1 segment; SCT neurons that were activated from the C1 segment at more than three times higher threshold; SCT neurons that were activated from the C1 segment at only 1.7–2.9 higher threshold but at longer latencies. Numbers of these neurons are given in parentheses. The *second column* gives mean threshold for C1 stimuli versus C3 stimuli. In the *third column* are mean differences between latencies of activation of the neurons by C1 and C3 stimuli. In the *fourth* and *fifth columns* are percentages of neurons excited by group II afferents of one of the muscle nerves or by cutaneous afferents; percentages of neurons found to be excited monosynaptically are given in parentheses

Neurons	C1/C3 thr	C1–C3 lat	Group II input	Cutaneous input
SCT ($n=10$)	–	–	90 (70)%	80 (60)%
SCT ($n=11$)	>3.0	0.55 ms	82 (36)%	82 (36)%
SCT ($n=12$)	1.7–2.9	0.60 ms	50 (33)%	100 (58)%
DSCT ($n=21$)	0.5–2.9	0.33 ms	77 (31)%	85 (23)%

study reveals just as great similarities in the midlumbar segments. Both the midlumbar SCT neurons and the midlumbar dorsal horn interneurons appear to a great extent to be co-excited by group II and cutaneous afferents, even though the cutaneous input might be stronger in the majority of the SCT neurons. This is indicated by larger and faster rising EPSPs evoked by cutaneous afferents (the records of Figs. 2E, G and 3C–G being representative in this respect). The SCT and the dorsal horn group II interneurons may thus be considered to process largely similar information. SCT neurons might act both at a spinal level – supplementing actions of group II interneurons via their initial axon collaterals (Brown et al. 1977; Rastad et al. 1977) – and via neurons of the lateral cervical nucleus and the medial lemniscal system. They may accordingly be used to ensure that responses mediated by spinal and supraspinal networks are properly coordinated.

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