# Interneurones in pathways from group II muscle afferents in sacral segments of the feline spinal cord

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1. Properties of dorsal horn interneurones that process information from group II muscle afferents in the sacral segments of the spinal cord have been investigated in the cat using both intracellular and extracellular recording.
2. The interneurones were excited by group II muscle afferents and cutaneous afferents but not by group I muscle afferents. They were most effectively excited by group II afferents of the posterior biceps, semitendinosus, triceps surae and quadriceps muscle nerves and by cutaneous afferents running in the cutaneous femoris, pudendal and sural nerves. The earliest synaptic actions were evoked monosynaptically and were very tightly locked to the stimuli.
3. EPSPs evoked monosynaptically by group II muscle afferents and cutaneous afferents of the most effective nerves were often cut short by disynaptic IPSPs. As a consequence of this negative feedback the EPSPs gave rise to single or double spike potentials and only a minority of interneurones responded with repetitive discharges. However, the neurones that did respond repetitively did so at a very high frequency of discharges (0·8–1·2 ms intervals between the first 2–3 spikes).
4. Sacral dorsal horn group II interneurones do not appear to act directly upon motoneurones because: (i) these interneurones are located outside the area within which last order interneurones have previously been found and (ii) the latencies of PSPs evoked in motoneurones by stimulation of the posterior biceps and semitendinosus, cutaneous femoris and pudendal nerves (i.e. the main nerves providing input to sacral interneurones) are compatible with a tri- but not with a disynaptic coupling. Spatial facilitation of EPSPs and IPSPs following synchronous stimulation of group II and cutaneous afferents of these nerves shows, however, that sacral interneurones may induce excitation or inhibition of motoneurones via other interneurones.
5. Comparison of the properties of group II interneurones in the sacral segments with those of previously studied group II interneurones in the midlumbar segments leads to the conclusion that these two populations of neurones are specialized for the processing of information from different muscles and skin areas. In addition, equivalents of only one of the two subpopulations of midlumbar interneurones have been found at the level of the pudendal nucleus: neurones with input from group II but not from group I muscle afferents. Neurones integrating information from group I and II muscle afferents and in direct contact with motoneurones thus seem to be scarce in the sacral segments.
6. On the basis of the patterns of input to sacral group II interneurones it is hypothesized that these interneurones are involved in postural adjustments associated with the defecation and grooming reflexes, as well as some labyrinthine reflexes.

We have recently reported that neurones relaying information from group II muscle afferents are located not only rostral, as previously found by Edgley & Jankowska (1987*a*), but also caudal to the lumbosacral enlargement (Jankowska & Riddell, 1993*b*), i.e. both in midlumbar and in sacral segments of the spinal cord. In the midlumbar segments, several types of neurones have input from group II afferents; these include last order interneurones with excitatory and inhibitory actions on motoneurones (Cavallari, Edgley & Jankowska, 1987), other interneurones (Edgley & Jankowska, 1987*b*) and ascending tract neurones projecting to the cerebellum (Edgley & Jankowska, 1988).

The types and properties of neurones with input from group II afferents terminating in the sacral segments have not yet been investigated. The studies reported here and in the accompanying paper (Riddell, Jankowska, Hammar & Szabo-Läckberg, 1994) were therefore designed to answer the following questions. (i) What kinds of neurones are contacted by group II afferents in the sacral segments: only interneurones, only ascending tract neurones or both types of neurones? (ii) What kind of sensory information is processed by these neurones together with that provided by group II muscle afferents? (iii) To what extent do the properties of group II-activated neurones in the sacral segments resemble or differ from those of midlumbar group II neurones? Preliminary observations have been published in abstract form (Jankowska & Riddell, 1992, 1993*a*).

## METHODS

### Preparation

Experiments were performed on fifteen cats under deep anaesthesia. The anaesthesia was induced with one dose of pentobarbitone (40 mg $kg^{-1}$ I.P.) and was maintained with several doses of chloralose (up to 50 mg $kg^{-1}$ I.V.). The adequacy of the anaesthesia was verified by monitoring withdrawal and corneal reflexes during the surgery and the diameter of the pupils and blood pressure during the experiments when the animals were paralysed with gallamine triethiodide and artificially ventilated. The mean blood pressure was kept above 90 (usually 110–130) mmHg and the end-tidal $CO_2$ was maintained close to 4% by adjusting the parameters of the artificial respiration and the rate of infusion of a solution of 100 mM sodium bicarbonate containing 5% glucose. The animals' core temperature was maintained at 37–38 °C and that in the paraffin pools at 35–37 °C. The same cats were also used for the studies reported by Jankowska & Riddell (1993*b*).

A number of left hindlimb peripheral nerves were dissected and mounted on electrodes. These routinely included: the quadriceps (Q), sartorius (Sart), posterior biceps (PB) and semitendinosus (ST) (which when dissected together are referred to as PBST), anterior biceps and semimembranosus (ABSM), medial gastrocnemius (MG), lateral gastrocnemius (LG) and soleus (S) (which when dissected together are referred to as 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), the caudal branch of sural (Sur), superficial peroneal (SP) and the posterior knee joint (J) nerves. Additional nerves dissected in some experiments included: the gracilis (Grac), adductor femoris (Add), popliteus (Popl), flexor digitorum longus (FDL, dissected free from the interosseous nerve), peroneus longus, tertius and brevis (Per), the remaining part of the tibial (Tib), saphenous (Saph), pudendal (Pud), cutaneous femoris (CF) and the right PBST, GS, Sur and Pud nerves.

The spinal cord was exposed by laminectomy from the fourth lumbar to sacral segments and at the level of the lowest thoracic (Th) segments. The dura was either left intact, except for small holes, or opened over all the exposed segments. Electrode penetrations were made between blood vessels covering the surface of the dorsal columns on the left side. Selection of the region in which to make electrode penetrations was guided by the rostrocaudal distribution of the largest cord dorsum potentials evoked by group II afferents of the PBST nerve which are associated with the largest group II field potentials at the level of Onuf's nucleus (Jankowska & Riddell, 1993*b*).

The locations of neurones from which recordings were made were determined from the distance of the recording site from the surface along the electrode track and by defining the electrode trajectories with respect to the position of marking electrodes left in each of the regions explored. The positions of the marking electrodes were defined histologically with respect to Onuf's nucleus (for further details see Jankowska & Riddell, 1993*b*).

### Recording and stimulation procedures

Neurones selected for testing were those responding to stimulation of the PBST and/or GS nerves at an intensity near maximal for group II afferents but not antidromically activated by stimuli applied above the lumbosacral enlargement, i.e. excluding ascending tract neurones which are described in the accompanying paper (Riddell *et al.* 1994). Neurones were selected while tracking through the dorsal horn and the intermediate zone within regions in which the largest monosynaptic field potentials were evoked by group II afferents of the PBST and GS nerves (at depths of about 1·2–2·5 mm). Recordings from the neurones were usually first made extracellularly to establish which of the dissected peripheral nerves evoked discharges and whether the neurones were antidromically activated by stimuli applied to the left and right lateral funiculi at the Th13 level. In some experiments responses to stimuli applied to the ipsilateral lateral funiculus at L4 were also investigated. An attempt was then made to penetrate the neurones, either in the same electrode track or in subsequent tracks 20–100 $\mu$m away. After a successful penetration the absence of antidromic activation by thoracic stimuli was confirmed and PSPs evoked from peripheral nerves were recorded intracellularly. Glass microelectrodes filled with 2 M potassium citrate solution (1–1·5 $\mu$m tip diameter, 3–5 M$\Omega$ resistance) were used for recording from the interneurones and a silver ball electrode placed about 5–10 mm more rostrally was used for recording afferent volleys from the surface of the spinal cord. Records were photographed directly from the oscilloscope screen and stored on videotape. Recordings which were stable over a sufficiently long period of time were also averaged (Nicolet Instrument Corporation (Madison, WI, USA) averager, type 1170, 5 $\mu$s per address time resolution, averages of 16–64 individual potentials).

Peripheral nerves were stimulated with 0·1 ms rectangular current pulses, applied singly, in pairs or as a train of a few stimuli at 200, 300 or 400 Hz. Stimulus strengths are expressed relative to threshold ($T$) for the most excitable afferents in the nerve, as detected by recording from the dorsal root entry zone, or from the sciatic nerve. The lateral funiculi were stimulated transdurally with 0·2 ms current pulses of up to 1 mA.

In a supplementary series of experiments intracellular recordings were made from fifty motoneurones in the L5–S2 segments to find out if any of the PSPs evoked in them might be mediated by sacral dorsal horn group II interneurones. Details of procedures used in these experiments are described in Results.

# RESULTS

## Sample of interneurones

The sample of interneurones investigated included 148 neurones that did not project beyond the lumbosacral enlargement. Most of these neurones (50 of 52 tested) did not project even as far rostrally as the L4 segment. The neurones were located in a length of spinal cord between 1 mm rostral and 1 mm caudal to Onuf's nucleus as shown in Fig. 1*E*. Since the majority of these interneurones were located in the sacral segments, they will be referred to as sacral group II interneurones. The interneurones were located in the dorsal horn, predominantly in the lateral half of laminae IV and V of Rexed (1954), as indicated in Fig. 1*A–C* on outlines of the spinal cord in the transverse plane. The interneurones were recorded from only extracellularly ($n = 78$), only intracellularly ($n = 52$) or both extra- and intracellularly ($n = 18$).

## Excitatory input

### Excitatory input from muscle nerves

Investigation of field potentials within the region in which the interneurones of the present study were recorded has shown that group II afferents of PBST evoked larger potentials than afferents of other muscle nerves (Jankowska & Riddell, 1993*b*). In accordance with these observations, when several (7–10) different ipsilateral muscle nerves were investigated for their effects on sacral interneurones, group II afferents of the PBST nerve appeared to be the main source of input. In a number of the sacral interneurones these were in fact the only muscle afferents with detectable actions (69 % of extracellularly recorded neurones and 13 % of intracellularly recorded neurones).

Both extracellularly recorded discharges and intracellularly recorded EPSPs evoked from muscle nerves

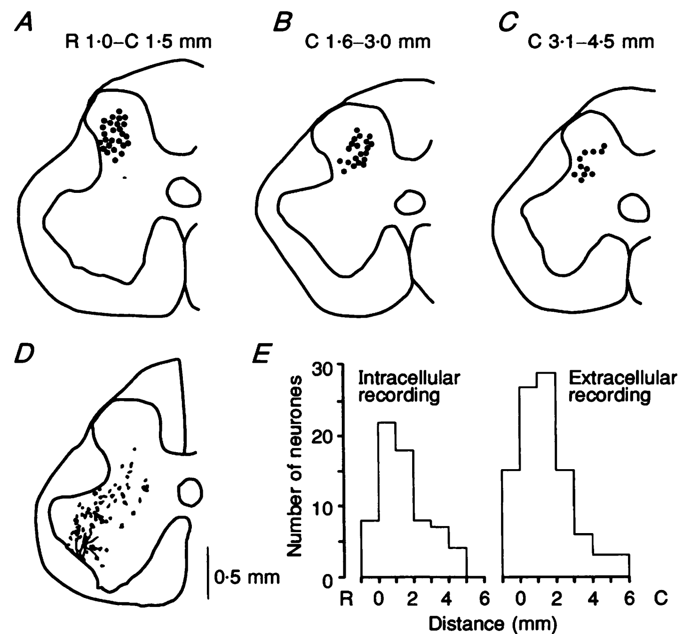


**Figure 1. Location of the sample of sacral interneurones in relation to Onuf's nucleus**

*A–C*, the locations of 58 intracellularly recorded interneurones within regions corresponding to the rostral (*A*), middle (*B*) and caudal (*C*) parts of Onuf's nucleus. The regions are defined by their distances in millimetres rostral (R) and caudal (C) to the rostral border of Onuf's nucleus. The location of individual interneurones was reconstructed from the distance from the surface along the recording electrode track (see Methods). Plots from different experiments were superimposed by alignment of the dorsal and lateral borders of the dorsal horn and the lateral borders of the ventral horn. *D*, distribution of last order interneurones within a region 1–2 mm caudal of the rostral border of Onuf's nucleus (S. A. Edgley & E. Jankowska, unpublished data). The neurones were labelled by retrograde transneuronal transport of wheat germ agglutinin-conjugated horseradish peroxidase (WGA–HRP) from the MG motor nuclei and are from two cats in which transneuronal transport was particularly effective. Note that practically all labelled neurones were outside the area in which interneurones with group II input were encountered in this study. *E*, rostrocaudal distribution of interneurones investigated in this study with respect to the rostral border of Onuf's nucleus (0 on the abscissa). The extent of this nucleus was defined as described by Jankowska & Riddell (1993*b*). The histograms show data for 67 interneurones recorded from intracellularly (left) and 96 interneurones recorded from extracellularly (right).

appeared only when the intensity of stimuli was increased above threshold for group II afferents; that is above 1·7–2·5*T* depending on the nerve (see Jack, 1978). Such tests were made for all of the neurones, the probability of discharging the extracellularly recorded neurones being increased by use of double stimuli 3·3 ms apart. The proportions of interneurones in which group II afferents of PBST and of other muscle nerves produced extracellularly recorded discharges and/or evoked intracellularly recorded EPSPs are compared in Fig. 2. Figure 2*A* shows that afferents of PBST produced EPSPs in virtually all interneurones, while afferents of Q, GS and Grac produced them in 50–69 % and afferents of Pl, ABSM, DP and Sart in less than 30 % of interneurones. Differences in the proportions of extracellularly recorded neurones discharged by these afferents were even more pronounced (Fig. 2*C*). Group II afferents of other nerves (ipsilateral Popl, Add and FDL and contralateral PBST and GS) produced either only occasional effects, or no effects at all. Input from group II afferents of toe muscles could not be investigated because these afferents could not be stimulated in isolation from cutaneous afferents in the Tib nerve.

When the amplitudes of EPSPs were compared, those evoked by PBST afferents were usually found to be larger than those evoked by other muscle afferents. These differences were more marked in neurones overlying the caudal half of Onuf's nucleus (see Fig. 3*G–P*) than in neurones overlying the rostral half of this nucleus (see Figs 3*A–F* and 4*F–I*). This may explain why a greater proportion of caudally located neurones (72 %) than of rostrally located interneurones (50 %) were discharged only by group II afferents of PBST.

Figures 4*K–N* and 8*N–O* show that stimuli near threshold or subthreshold for group II afferents were ineffective, even when they were near maximal or supramaximal for group I afferents. Only in one neurone were synaptic actions attributable to group I afferents (of quadriceps) seen. EPSPs from group I afferents were, however, evoked in the same experiments in more rostrally located interneurones; the latter were located 2–5 mm rostral to Onuf's nucleus.

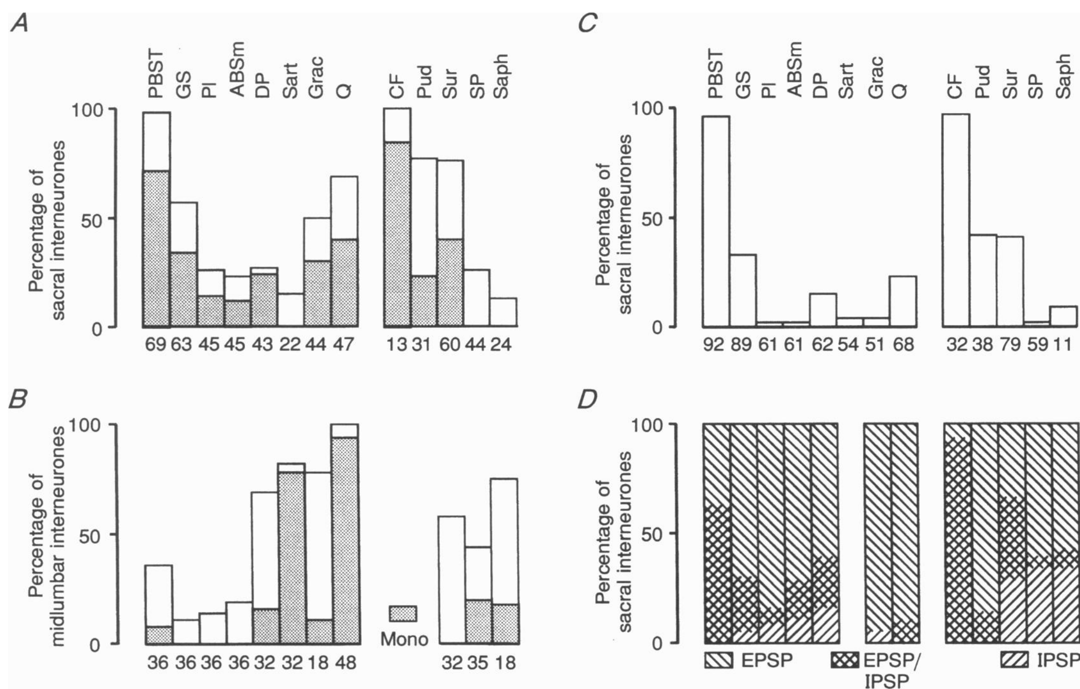


**Figure 2. Proportions of interneurones with input from group II afferents of 8 muscle nerves and from afferents of 5 cutaneous nerves**

*A*, histogram showing the proportion of intracellularly recorded interneurones in which EPSPs were evoked (at latencies not exceeding 4 ms). The shaded parts of these bars indicate the percentage of neurones in which the EPSPs are considered likely to have been evoked monosynaptically (see Fig. 5 and text). *B* shows, for comparison, the proportions of intracellularly recorded dorsal horn midlumbar interneurones with excitatory input from the same nerves (S. A. Edgley & E. Jankowska, unpublished data). *C*, proportions of extracellularly recorded interneurones with excitatory input (< 5 ms latency). In *A*, *B* and *C* numbers of neurones tested are indicated beneath each bar. *D*, proportions of intracellularly recorded interneurones in which different nerves produced predominently EPSPs (shading from top to bottom), predominatly IPSPs (shading from bottom to top) and EPSPs followed by IPSPs (overlapping regions of shading).

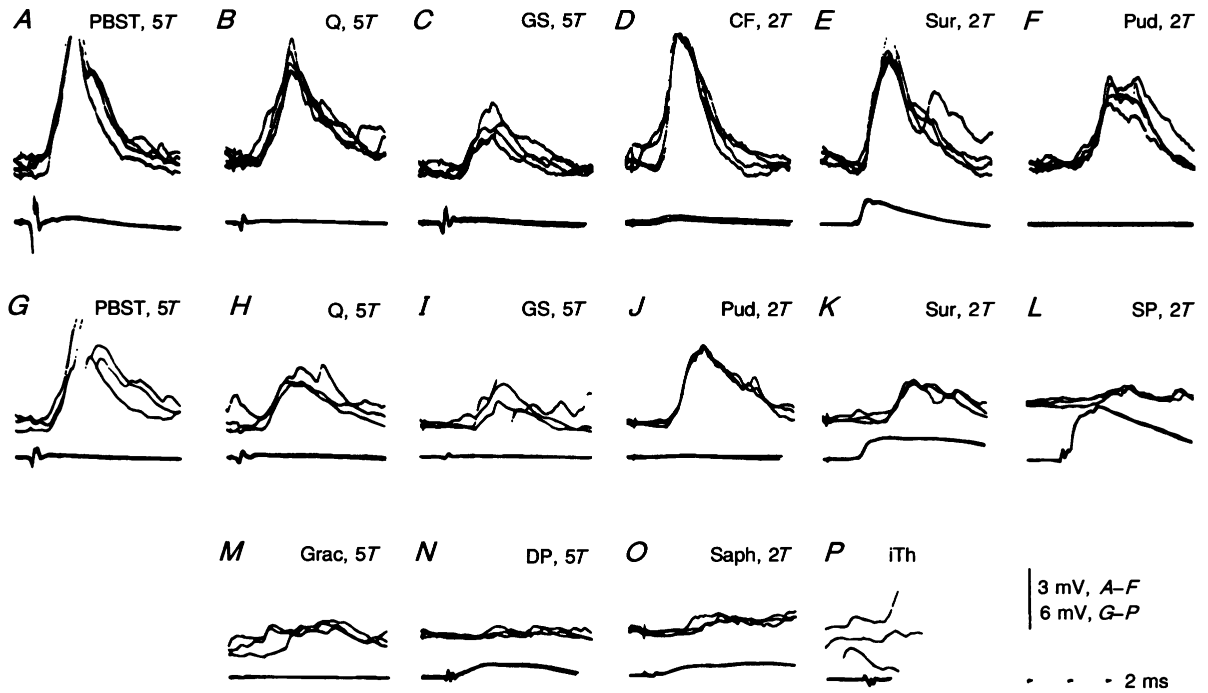


**Figure 3. Examples of EPSPs (upper traces) evoked from muscle and cutaneous nerves in 2 sacral interneurones (*A–F* and *G–P*)**

The interneurones were located about 0·2 and 2·4 mm caudal to the rostral border of Onuf's nucleus. Note that the largest EPSPs were evoked from the PBST and CF nerves in the more rostrally located interneurone and from the PBST and Pud nerves (CF was not tested) in the more caudally located interneurone. In this and the following figures lower traces are from the surface of the spinal cord; negativity is downwards in the microelectrode records and upwards in surface records. Stimulus intensities are indicated above each record in multiples of threshold ($T$) of the nerve stimulated.

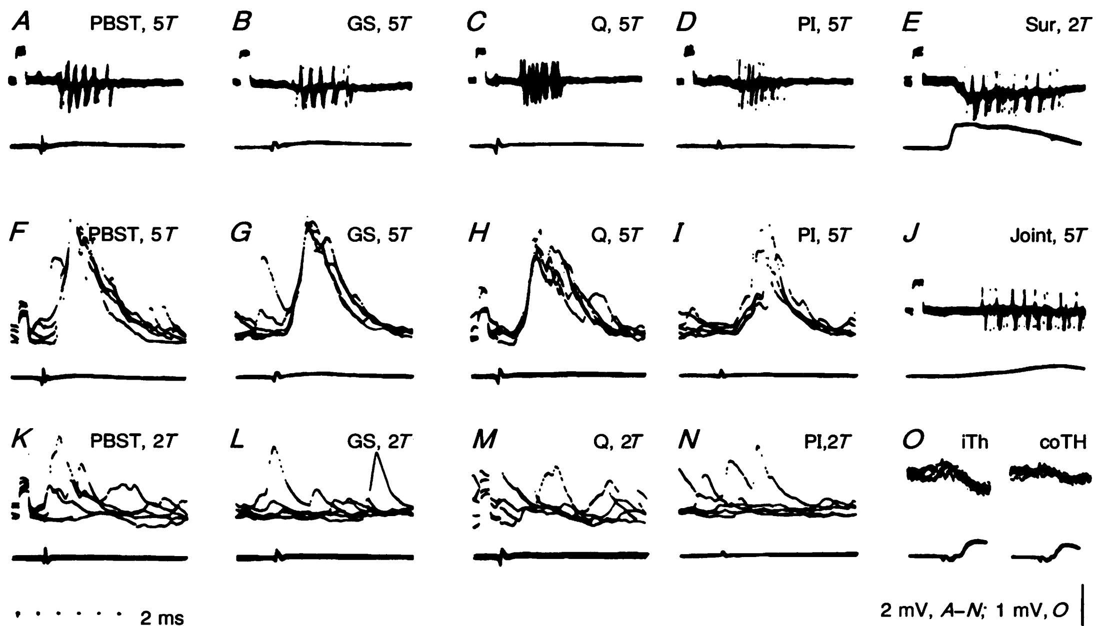


**Figure 4. Examples of excitatory actions evoked by group II muscle afferents and cutaneous and joint afferents in a sacral interneurone**

The interneurone was located about 0·5 mm caudal to the rostral border of Onuf's nucleus. The upper traces show extracellular (*A–E* and *J*) and intracellular (*F–I* and *K–O*) recordings from the same neurone. Note the lack of antidromic responses to stimulation of either the ipsilateral (iTh) or contralateral (coTh) lateral funiculi at the Th13 level (*O*) and the lack of responses to stimuli $2T$ applied to muscle nerves (*K–N*). Note also that cutaneous (*E*) and joint (*J*) afferents were as effective as muscle nerves in producing extracellularly recorded discharges.

The great majority of interneurones included in this analysis were located in the dorsal horn and the lack of input from group I afferents is therefore consistent with observations that the field potentials evoked there on stimulation of muscle nerves are produced exclusively by group II afferents (Jankowska & Riddell, 1993*b*). The reasons for the lack of interneurones with group I input in the deeper regions of the grey matter, where some group I potentials can be recorded, are not clear. One possibility is that lateral parts of the sacral grey matter were explored more extensively than the medial parts where group I field potentials are largest (see Fig. 5 in Jankowska & Riddell, 1993*b*). Alternatively, our results may reflect a lower density of neurones with group I input in the sacral segments than in more rostral segments of the spinal cord.

The influence of higher threshold (group III and IV) muscle afferents was not systematically investigated. However, additional EPSPs or spike potentials evoked by raising the stimulus intensity from 5 to 20*T* were seen in half of the fourteen interneurones with group II input which were tested in this manner.

## Excitatory input from cutaneous and joint nerves

Cutaneous afferents were another main source of input to interneurones excited by group II afferents. As shown in the histograms of Fig. 2*A* and *B*, as large a proportion of these interneurones were excited by afferents of the CF nerve as by afferents of PBST. Furthermore, as illustrated in the records of Figs 3*A*–*F* and 8*A* and *B*, amplitudes of EPSPs evoked by CF stimulation were often (in 50 % of cells) similar or larger than those evoked from PBST. Afferents of Pud and Sur excited smaller proportions of sacral interneurones but EPSPs evoked by these afferents were often (in 47 and 26 % respectively) similar or larger than EPSPs evoked from PBST afferents. As illustrated in Fig. 3, Pud afferents had stronger actions on interneurones overlying the caudal part of Onuf's nucleus and Sur had stronger actions on interneurones located more rostrally. SP and Saph afferents had weaker and much less frequent actions. Afferents of the contralateral Sur nerve excited 2 of 13 interneurones (2 of 4 intracellularly and 0 of 9 extracellularly recorded) and those of the contralateral Pud nerve 3 of 13 interneurones (2 of 4 intracellularly and 1 of 9 extracellularly recorded). Afferents of the posterior knee joint nerve evoked excitation of sacral interneurones only occasionally.

## Latencies of excitation evoked by group II muscle and cutaneous afferents

Segmental delays of the actions of group II afferents were measured with respect to group I afferent volleys because discrete group II volleys could only exceptionally be distinguished (see also Edgley & Jankowska, 1987*a*). The precise time of arrival of nerve impulses at the terminals of group II afferents is also difficult to calculate because of considerable overlap in the conduction velocities of

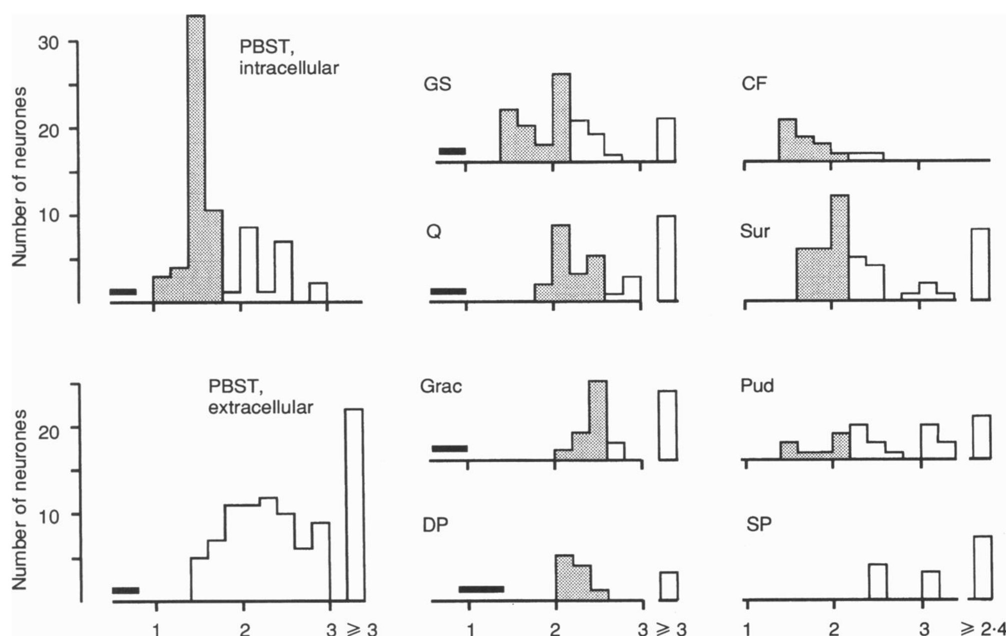


**Figure 5. Histograms of latencies of EPSPs evoked from various nerves in sacral group II interneurones**

The latencies were measured between the onset of group I afferent volleys (i.e. the peak of the first positive deflection), or volleys in the fastest conducting cutaneous afferents, and the onset of EPSPs. The latencies are grouped in 0·2 ms ranges. The thick horizontal bars indicate the expected time ranges for the arrival of the most synchronous group II volleys and the shaded bars indicate those EPSPs considered likely to be evoked monosynaptically (see text for details).

different functional categories of muscle afferents (see Jankowska, 1992, for references) and the unknown degree of slowing of conduction along descending axon collaterals. PSPs evoked at latencies considered compatible with a monosynaptic coupling are indicated in Fig. 5 by the shaded parts of the histograms. As shown here and in Fig. 2, EPSPs probably evoked monosynaptically were induced by all but one (Sart) of the muscle nerves tested and in a high proportion of the interneurones. The premises on which these estimates are based are described in the small print section below.

Impulses in the fastest conducting fraction of group II afferents of the *PBST nerve* would be expected to arrive at the spinal cord between 0·5 and 0·8 ms later than those in group I afferents (as indicated by the thick horizontal bars in Fig. 5; see also Fig. 7 in Jankowska & Riddell, 1993*b*) and the intraspinal conduction time of group II afferents would account for a further 0·5–1·0 ms delay (Fu & Schomburg, 1974; Lundberg, Malmgren & Schomburg, 1987*a*). Latencies of EPSPs of between 1·1 and 1·7 ms with respect to group I volleys are therefore considered to be compatible with a monosynaptic linkage between group II afferents of PBST and the interneurones. Frequency following tests showed that when evoked by trains of stimuli at 200–400 Hz these EPSPs were induced without temporal facilitation, as required for monosynaptically evoked actions. Such tests are illustrated in Fig. 6*A–F*. Minimal segmental latencies of extracellularly recorded responses were 1·4 ms, i.e. only 0·3 ms longer than those of EPSPs, although most were some 0·5–1·0 ms longer.

For *GS afferents*, EPSPs evoked at segmental latencies of 1·4–2·1 ms are considered to be evoked monosynaptically in view of the longer conduction path of GS group II afferents. The peripheral conduction times of these afferents are 0·9 ms longer than those of group I afferents of GS (Fu, Santini & Schomburg, 1974) and 0·4 ms longer than those of group II afferents of PBST. The central conduction times of GS and PBST group II afferents should, on the other hand, be similar since both sets of fibres enter the spinal cord at the same segmental level.

For *Q, Sart and Grac afferents* segmental latencies of up to 2·5 ms, i.e. 0·8 ms longer than those of PBST afferents, are considered to be compatible with monosynaptically evoked actions. Although impulses in group II afferents of these nerves are less delayed than those of group I afferents because of the shorter peripheral conduction path (see Table 1, far right column, of Edgley & Jankowska, 1987*a*), they enter the spinal cord about 20 mm more rostrally than afferents of PBST. The additional conduction time between the L6 and sacral segments may amount to about 0·6 ms since the intraspinal conduction velocity of ascending collaterals of group II afferents is 30–60 % of that in peripheral nerves (Munson, Fleshman & Sypert, 1980; Sypert, Fleshman & Munson, 1980; Fern, Harrison & Riddell, 1988) and may be even lower in descending collaterals. Three further observations are also of relevance to the estimates of the ranges of monosynaptic actions of Q afferents in the sacral segments. Firstly, the earliest actions of Q group II afferents were evoked only about 0·6 ms later in the sacral than in the midlumbar segments (see Fig. 8 of Edgley & Jankowska, 1987*a*) and this difference is too short for the actions on sacral neurones to involve an additional interneurone. Secondly, the minimal latencies of disynaptically evoked EPSPs and IPSPs in L7–S1 motoneurones (both those expected on the basis of the data of Edgley & Jankowska (1987*b*) and those actually recorded in the study of Lundberg *et al.* (1987*a*), 2·6–2·9 ms from group I volleys, are at least 0·8 ms longer than the minimal latencies of EPSPs evoked by Q group II afferents in sacral interneurones. Thirdly, EPSPs evoked by trains of stimuli at 200–400 Hz were usually induced without temporal facilitation, as illustrated in Fig. 6*G* and *H*. For the *DP nerve*, based on the same arguments, latencies of up to 2·6 ms are considered monosynaptic.

Minimal segmental latencies of EPSPs evoked by stimulation of cutaneous nerves were 0·5 ms and those of spike potentials in extracellular records were 0·8 ms; they were thus fully compatible with a monosynaptic coupling between cutaneous afferents and the interneurones. If latencies of less than 1·1 ms are considered to be compatible with monosynaptic coupling, then monosynaptically evoked EPSPs were produced by the faster conducting afferents of CF, Sur and Pud nerves but not of the SP and Saph nerves.

### Types of excitatory responses

Responses recorded extracellularly consisted of single spikes, double spikes or a series of spikes, as illustrated in Fig. 7. When responses to both electrical and natural stimuli were investigated, the type of response evoked showed a striking similarity. The proportions of neurones responding with single or double spikes were 80 % for GS, 78 % for PBST, 66 % for Q, 55 % for CF, 54 % for Sur and 23 % for Pud (calculated for samples of at least 15 neurones). Both single and double spikes and the first few spikes of a series were often very tightly locked to the stimuli, as can be seen from the synchronized responses in both the superimposed single records and averaged records shown in Fig. 7*C*. Many sacral interneurones therefore appear to be very securely activated. The earliest discharges usually appeared during the rising phase of EPSPs subsequently recorded (see Figs 3*A–D* and 8*K* and *L*). The responses of interneurones could follow repetitive stimuli up to at least 300 Hz (see Fig. 6*M*) and intervals between the first two to three spikes of repetitive responses to single stimuli were often as short as 0·8–1·2 ms (see Figs 4 and 7). Such short intervals were seen in fifteen neurones activated by electrical stimulation of muscle nerves at 5*T* and/or stimulation of skin nerves at 2*T*. High frequencies of activation were also seen during activation by natural stimuli (touch or light pressure on skin of the upper part of the thigh) as illustrated in Fig. 7*D*. The shortest intervals between spikes in a burst induced by such stimuli were of 1·0–1·5 ms.

## Inhibitory input

### Nerve origin

The records of Fig. 8*F*, *L* and *M* show that in some cells EPSPs evoked by group II muscle afferents were followed by IPSPs. These IPSPs appeared at similar stimulus intensities to EPSPs (see Fig. 8*L–O*), and are therefore also attributed to group II afferents. EPSPs followed by IPSPs were also evoked by cutaneous afferents, as shown in

Fig. 8*G* and *H*. IPSPs were sometimes seen directly after the penetration of a neurone, while in other cases, such as that illustrated in Fig. 8*F–H*, a depolarization of the neurone was required to reveal them. An interesting feature of the records of Fig. 8 is that IPSPs revealed by depolarization were evoked from only some of the nerves (PBST, CF and Sur but not Grac or Pud). The diagram of Fig. 2*D* summarizes this kind of data for the whole sample of neurones and shows that EPSP/IPSP sequences were more frequently evoked from some nerves, in particular PBST (70 %), GS (33 %), CF (90 %) and Sur (53 %), than from others. Group II afferents of other muscle nerves evoked EPSP/IPSP sequences in only 8–25 % of neurones and in some neurones only IPSPs appeared to be evoked. Of other cutaneous afferents, those of Pud had dominant excitatory actions while stimulation of SP and Saph most often evoked IPSPs.

Sequences of EPSPs/IPSPs were correlated with single or double spike responses in fifteen of eighteen interneurones recorded both intra- and extracellularly (as in Fig. 8*K*). The high proportions of neurones responding with single or double spikes to stimuli applied to PBST, GS, Q, CF and Sur nerves (54–80 %; see above) suggest, therefore, that EPSPs evoked from these nerves are often cut short by IPSPs. In the remaining three neurones the EPSPs were not followed by IPSPs and the neurones responded with a series of extracellularly recorded discharges (as illustrated in Fig. 4). This does not imply, however, that in neurones responding with repetitive discharges only EPSPs are evoked. In some of these neurones a break in the discharge occurred after the first one or two spikes (as in the third and fourth records from the bottom in Fig. 7*A* and *B*) and such a break might be an indication of combined excitatory and inhibitory actions in strongly excited neurones. Negative feedback therefore appears to be a very common feature of the actions of group II muscle and cutaneous afferents in the sacral segments.

### Latencies of IPSPs

The onset of IPSPs was more difficult to define than that of EPSPs because they were usually superimposed on EPSPs.

Measurements of the latencies of IPSPs were therefore subject to greater error and the values obtained will tend to be over- rather than underestimates of the latencies. Nevertheless, measurements made under optimal conditions (as in the records of Fig. 8) suggest that the latencies of IPSPs are most probably less than 1 ms longer than the latencies of EPSPs evoked from the same nerves. If the EPSPs are evoked monosynaptically, then the IPSPs must often have been evoked disynaptically. Generally, the shortest latencies of IPSPs evoked from the PBST (1·8 ms), Q (2·6 ms), DP (2·5 ms), CF (1·0 ms) and Sur (1·1 ms) nerves were only 0·6–0·7 ms longer than the latencies of the earliest EPSPs and at least one-third of IPSPs were less than 1·5 ms longer. Most of these latencies would therefore

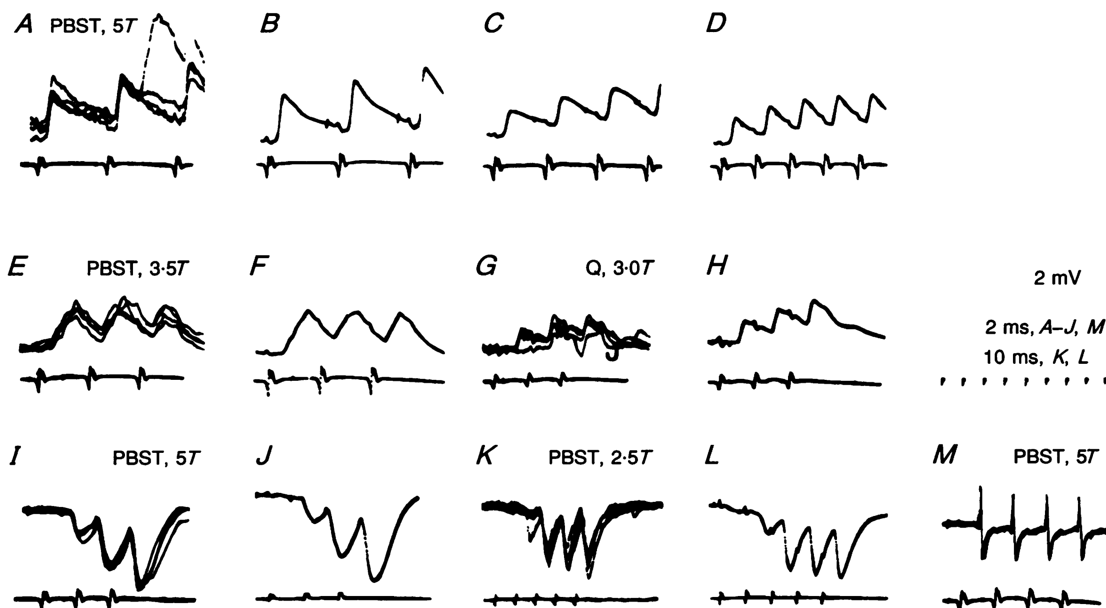


**Figure 6. Examples of EPSPs and IPSPs evoked by repetitive stimuli in five neurones, *A–D*, *E–F*, *G–H*, *I–J* and *K–L***

Note that both shorter latency (1·2 ms, *A–B*; 1·5 ms, *E–F*) and longer latency (2·0 ms, *G–H*) EPSPs followed successive stimuli without increment. This is in contrast to the clear temporal facilitation of IPSPs shown in *I–L*. The records in *A*, *E*, *G*, *I*, *K* and *M* consist of 4 superimposed individual traces. The records in *B–D*, *F*, *H*, *J* and *L* are averages of 16 or 32 responses. All PSPs required stimuli of at least 2*T*. Records in *M* illustrate that extracellularly recorded action potentials followed the same stimulus frequencies.

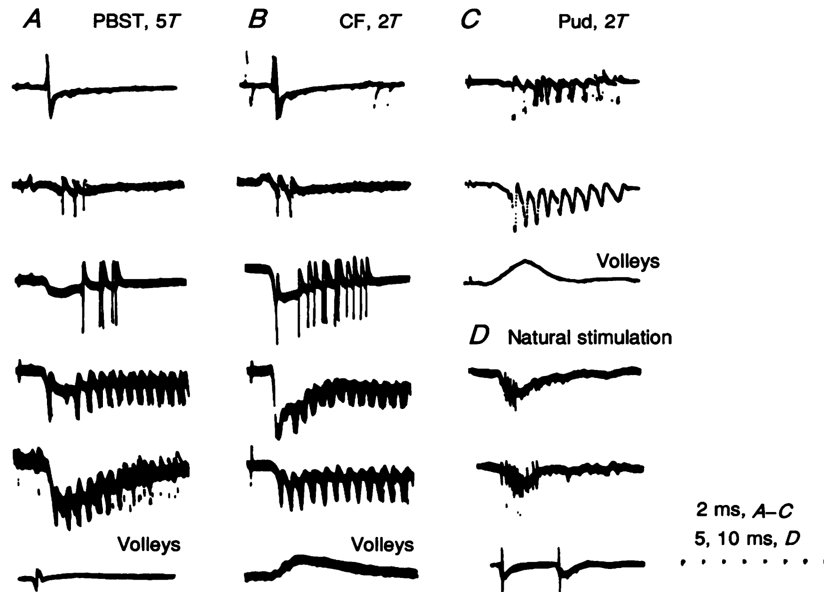


**Figure 7. Examples of different types of responses of extracellularly recorded sacral interneurones**

The records in *A* show responses to stimulation of the PBST nerve and the records in *B* responses evoked in the same neurones by cutaneous afferents of CF. The lowermost records show incoming volleys. *C*, responses evoked by stimulation of the Pud nerve in another neurone (superimposed records of 3 single responses and averages of 16 responses). *D*, responses to touching (top and middle) and tapping twice (bottom) the skin of the upper–medial part of the thigh. Note that even light natural stimuli evoked quite marked field potentials.

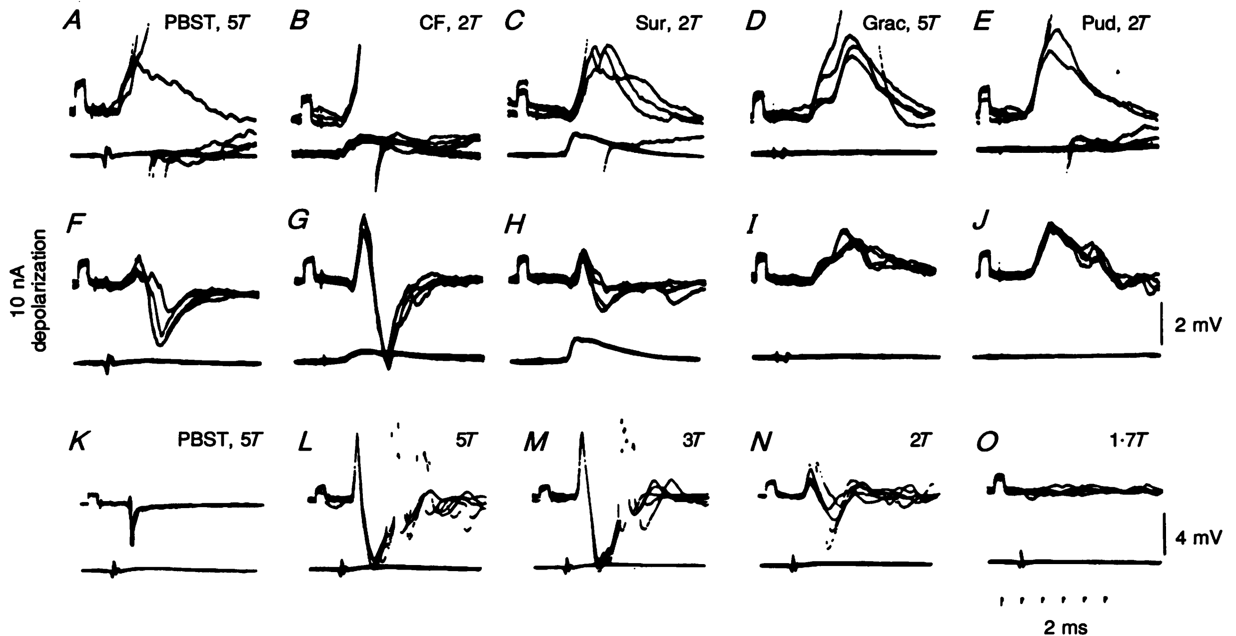


**Figure 8. Examples of sequences of excitatory and inhibitory responses evoked in two interneurones (*A–J* and *K–O*) located 0·8 and 4 mm caudal to the rostral border of Onuf's nucleus, respectively**

All but one (*K*) of the upper traces are intracellular records. Note that in the first neurone depolarization (10 nA) disclosed IPSPs superimposed on di- or polysynaptic EPSPs evoked by PBST, CF and Sur but not by Grac and Pud. Records *L–O* illustrate that both IPSPs and EPSPs were evoked by stimuli of the same intensity, i.e. supramaximal for group I afferents.

be compatible with a disynaptic coupling in inhibitory pathways from both group II and cutaneous afferents to sacral interneurones. Distinct waves of IPSPs of group II origin following successive stimuli in a train (as in Fig. 6*I–L*) are in keeping with these conclusions.

Since interneurones monosynaptically activated by group II afferents of PBST are much less frequently encountered in more rostral segments, disynaptic IPSPs induced by group II afferents of PBST would most probably be mediated by sacral interneurones. IPSPs seen in sacral interneurones may therefore reflect inhibitory interactions between subpopulations of these neurones. The same interneurones might also mediate the disynaptic inhibition evoked from the nerves which co-excited them (in particular CF, Sur, GS and Q) while inhibitory actions of the nerves which only rarely excited sacral interneurones are more likely to be mediated by more rostrally located interneurones. The longer latencies of IPSPs evoked by these nerves (1·5–3·5 ms longer than those of EPSPs) strengthen this conclusion.

## Do sacral group II interneurones have direct actions upon motoneurones?

Three approaches were used to evaluate this possibility. The first approach was to compare the locations at which sacral group II interneurones were recorded with the locations of last-order sacral interneurones labelled by retrograde transneuronal transport of HRP from hindlimb motoneurones (S. A. Edgley and E. Jankowska, unpublished data). As shown in Fig. 1 there was hardly any overlap in the areas of location of the two samples of interneurones, indicating that direct contacts between dorsal horn group II interneurones and the motoneurones (MG and ST) used in the transneuronal transport study are rather unlikely. However, these observations do not exclude the possibility that sacral group II interneurones synapse on other motoneurones.

The second approach was to investigate whether sacral interneurones have direct actions on motoneurones by looking for disynaptically evoked PSPs following

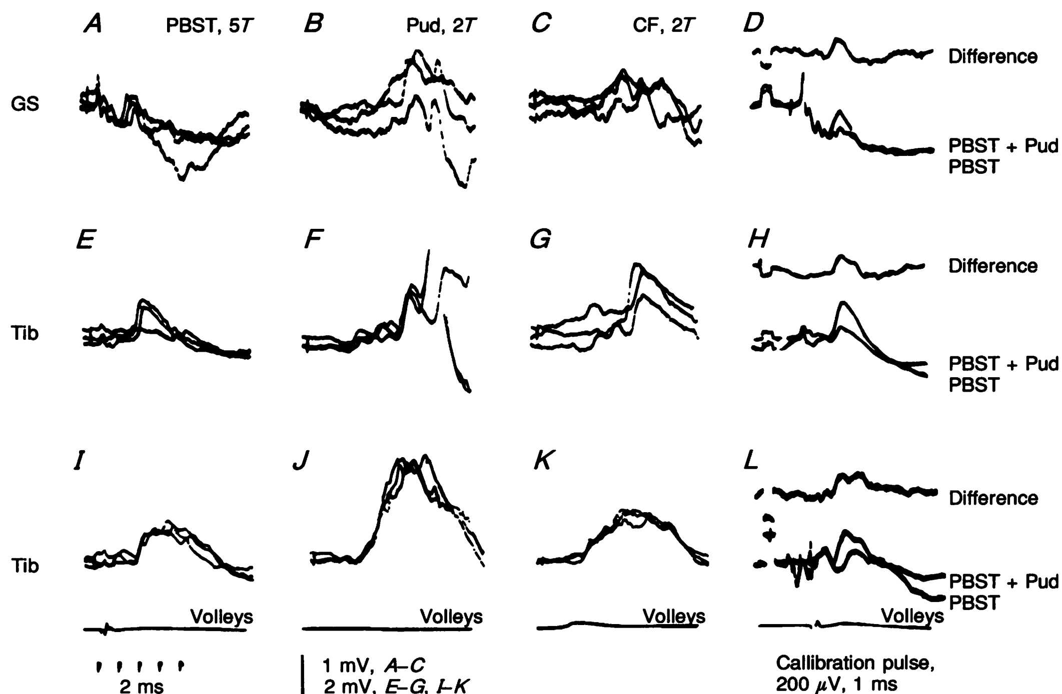


**Figure 9. Examples of EPSPs evoked by afferents of the PBST, Pud and CF nerves in motoneurones and of mutual facilitation of the actions of Pud and PBST afferents**

The records in the 3 horizontal rows are from one GS (*A–D*) and two Tib (*E–H* and *I–L*) motoneurones. The first 3 vertical columns show PSPs (3 superimposed records) evoked by stimuli applied to the PBST, Pud or CF nerves, respectively. The bottom traces are corresponding records from the surface of the spinal cord (volleys). All EPSPs evoked from the PBST nerve appeared only at stimulus intensities supramaximal for group I afferents. The averaged records in the fourth vertical column show, from the bottom to the top: afferent volleys, effects of stimulation of PBST alone (PBST), effects of nearly simultaneous stimulation of PBST and Pud (PBST + Pud) and the differences between the latter and the sum of the effects of stimulation of either of these nerves separately. The difference records represent PSPs mediated by interneurones co-excited by PBST and Pud afferents. Note that facilitated PSPs were evoked with similar delays with respect to stimulus artifacts and incoming volleys.

stimulation of nerves which provide monosynaptic input to these interneurones. Recordings from fifty motoneurones (6 Pud, 14 Tib, 3 PBST, 7 GS, 8 DP, 10 Q and 2 Grac) were used for this purpose. Disynaptic PSPs evoked by stimulation of the CF and, in particular, the Pud nerve were considered to provide the most decisive test, since the actions of these nerves are restricted primarily to neurones in the sacral segments (see Riddell & Jankowska, 1993*b*) while disynaptic PSPs of PBST origin might also be evoked by more rostrally located interneurones. It was considered that if PSPs were evoked disynaptically by CF and Pud nerves then they should appear at minimal latencies of not more than 1·6 ms from the afferent volley (allowing 0·8–1·0 ms for the shortest segmental latencies of activation of sacral interneurones, 0·5 ms for conduction time between the interneurones and motoneurones located within the same or neighbouring segments and 0·3 ms for a synaptic delay between them). However, although stimulation of Pud and CF did produce PSPs in motoneurones, no such short latency PSPs were found. The shortest latency PSPs (2·0–2·5 ms) were seen in Tib, GS and Pud motoneurones (see Fig. 9*B* and *C*) while PSPs in other motoneurones were evoked at latencies of more than 3·0 ms. The latter included PBST motoneurones located in the same or adjacent (L7) segment, and Q and DP motoneurones located one or two segments more rostrally. No indications were therefore found for any direct actions of sacral interneurones upon motoneurones.

The third approach was to investigate whether shorter latency PSPs might be evoked under conditions where the actions of individual nerves were facilitated, considering that those interneurones which synapse on motoneurones might be only weakly excited by either Pud or CF afferents. In view of the convergence of PBST and Pud or CF afferents on a great proportion of sacral interneurones, actions evoked by these interneurones were expected to be more easily detected following stimulation of two of these nerves together. Stimuli were therefore applied to the PBST and Pud or CF nerves, either separately or nearly synchronously, and responses exceeding the sum of responses evoked by the separately applied stimuli were attributed to interneurones co-excited by PBST and Pud or CF afferents. This procedure (see Lundberg, 1975) was applied while recording from ten motoneurones (4 GS and 6 Tib). Facilitation was observed in six of these for combinations of PBST and Pud stimuli and in three for combinations of PBST and CF stimuli; they are illustrated in the right-most records of Fig. 9. Stimuli applied to the Pud and CF nerves were always much weaker than those evoking responses in the motoneurones while stimuli applied to PBST were adjusted to evoke weak responses. The facilitated responses (see traces labelled 'difference') were therefore related to the PBST afferent volleys. The facilitated responses of shortest latecy appeared within 2·5–3·3 ms (in 5 motoneurones). These latencies are longer than those that would be expected for disynaptically evoked PSPs, which should not exceed 2·2 ms (allowing 1·4 ms for minimal segmental latencies of activation of sacral interneurones, 0·5 ms for the conduction time between the interneurones and motoneurones located within the same or neighbouring segments and 0·3 ms for a synaptic delay). These observations support the observations reported above that sacral interneurones have no direct actions upon motoneurones. The presence of facilitation shows, however, that sacral interneurones may act upon motoneurones via other interneurones as part of tri- or oligosynaptic pathways.

## DISCUSSION

One of the aims of the experiments reported in this and in the companion paper (Riddell *et al.* 1994) was to find out what kinds of neurones are contacted by group II afferents in sacral segments. The results show that such neurones include both interneurones and ascending tract neurones. Properties of these neurones will be compared in the companion paper (Riddell *et al.* 1994); the following discussion will therefore be restricted to the interneurones.

### Comparison of sacral and midlumbar interneurones with group II input

One of the features shared by the sacral interneurones investigated in the present study and the previously investigated midlumbar interneurones (see Edgley & Jankowska, 1987*b*; Cavallari, Edgley & Jankowska, 1987; Edgley, Jankowska & Shefchyk, 1988; Bras, Cavallari, Jankowska & Kubin, 1989) is that both integrate information on muscle length (via group II muscle spindle afferents) with that provided by skin receptors. Nevertheless, neurones located at the level of Onuf's nucleus differ from those located in the L3, L4 and the rostral part of the L5 segment in four main respects.

The *first difference* is that two distinct subpopulations have been found in the population of midlumbar interneurones: dorsal horn interneurones with input from only group II muscle afferents and intermediate zone interneurones with input from both group I and group II muscle afferents; only equivalents of the dorsal horn midlumbar interneurones have been found at the level of Onuf's nucleus. Since some field potentials from group I afferents can be recorded at the level of Onuf's nucleus (Jankowska & Riddell, 1993*b*) we do not exclude the possibility that neurones co-excited by group I and II afferents are present in sacral segments, but consider that they are much scarcer than in the midlumbar segments and that the probability of encountering them is therefore much smaller. Judging from the distribution of field potentials evoked by group I afferents we would expect such neurones to be located predominantly rostral to Onuf's nucleus.

The *second difference* between the midlumbar and sacral interneurones is in the muscle origin of their group II input. As shown in Fig. 2, the PBST nerve is the main

source of group II input to the sacral interneurones. In intracellularly recorded interneurones, group II PBST afferents were found to evoke EPSPs in all sacral but in only about one-third of the midlumbar dorsal horn group II interneurones and in an even smaller proportion (1/10) of the midlumbar intermediate zone interneurones (see Fig. 6 in Edgley & Jankowska, 1987*b*). In contrast, group II afferents of the Q, Sart and DP nerves are the main source of input to the midlumbar interneurones. Synaptic actions of Sart and DP afferents on sacral interneurones were seen only very rarely and although those of Q afferents were somewhat more frequent, all were usually weaker than the synaptic actions of PBST afferents. Accordingly, the extracellularly recorded sacral interneurones were activated most often only by PBST afferents and the midlumbar interneurones by Q, Sart and DP afferents.

The *third difference* between the midlumbar and sacral neurones is in their extramuscular sources of input. Cutaneous afferents appeared to contribute to the excitation of a higher proportion of sacral than of midlumbar interneurones. In addition cutaneous afferents of different nerves varied in their effectiveness; sacral neurones were excited mainly by afferents of CF, Sur and Pud, and midlumbar interneurones mainly by afferents of Sur, Saph and SP. Although afferents of CF and Pud have not been tested for their actions on individual midlumbar interneurones, the conclusion that they have only weak actions on midlumbar interneurones is based on much smaller field potentials evoked from CF in the midlumbar compared to sacral segments and on the failure to find field potentials evoked by Pud afferents in midlumbar segments (J. S. Riddell & E. Jankowska, unpublished observations).

The *fourth difference* is in the timing of the actions and/or in the coupling in pathways from the various group II and cutaneous afferents to sacral and midlumbar interneurones. Most muscle nerves excited neurones of the two populations with a longer delay at one or other of the sites. For example, the latencies of EPSPs evoked by Q were longer in sacral than midlumbar segments (cf. Fig. 6 of the present paper with Table 1 of Edgley & Jankowska, 1987*b*). The differences in the minimal latencies of the actions of these afferents were only some 0·6 ms and are attributable to differences in the proximity of the sacral and midlumbar segments to the level of entry of the afferents. Such differences are therefore compatible with a monosynaptic coupling at both sites. Differences exceeding 1·0–1·5 ms, on the other hand, indicate an oligosynaptic coupling. The larger proportions of neurones responding at these longer latencies to stimulation of the Q and Sart nerves in the sacral segments and to stimulation of the PBST nerve in midlumbar segments therefore suggest that the actions of group II afferents outside their specific midlumbar and sacral relay regions are to a great extent indirect.

With respect to cutaneous afferents, no differences were found in the minimal latencies of EPSPs evoked from the SP and Saph nerves in midlumbar segments and from the CF, Pud and Sur nerves in sacral segments; both were compatible with monosynaptic actions of these afferents. In contrast, the minimal latencies of EPSPs evoked from the SP or Saph nerves in sacral segments were at least 1 ms longer than in the midlumbar segments. This is more than can be accounted for simply by a longer intraspinal conduction time (at least for SP afferents that enter only a few millimetres more rostrally than CF or Sur afferents). Only afferents of the CF, Sur and Pud nerves therefore appear to provide a direct input from cutaneous afferents to the sacral group II interneurones.

## On the likely function and organization of sacral group II interneurones

Neither previous nor the present observations provide any indication that sacral group II interneurones might have direct actions upon motoneurones. Dorsal horn interneurones at the level of Onuf's nucleus were not labelled by retrograde transneuronal transport of WGA–HRP from the ST and MG motor nuclei. It is therefore unlikely that such interneurones make synaptic contacts with these motoneurones. A survey of the synaptic actions of CF and Pud afferents on motoneurones of both these and other motor nuclei failed to reveal any that could be evoked disynaptically. All of the PSPs induced in motoneurones by stimulation of the CF and Pud nerves appeared at latencies too long to be compatible with direct actions of sacral interneurones. Those components of PSPs which appeared after spatial facilitation of the actions of the PBST and Pud or of the PBST and CF nerves likewise appeared with minimal latencies exceeding those expected of disynaptically evoked responses.

We therefore propose that other interneurones are primarily mediating the disynaptic actions of group II afferents in the PBST and GS nerves upon motoneurones (Lundberg, Malmgren & Schomburg, 1987*a*). Such interneurones are unlikely to be located in the midlumbar segments, since interneurones with group I and II monosynaptic input from the PBST and GS nerves are only exceptionally found in the intermediate zone of L3–L4 segments (Edgley & Jankowska, 1987*b*). However, interneurones monosynaptically excited by group I and II muscle afferents of the PBST or GS nerves were found within the lumbosacral enlargement (Harrison & Jankowska, 1985; Lundberg, Malmgren & Schomburg, 1987*b*). Interneurones excited by group II afferents of the GS and PBST nerves at a short latency and with axons ascending in the lateral funiculus up to the L4 segment (Harrison & Riddell, 1989) would also be likely to have somata within the lumbosacral enlargement, since sacral interneurones only exceptionally projected as far rostrally.

If sacral group II interneurones do not have direct actions upon motoneurones they may also be eliminated as candidates for interneurones mediating disynaptic EPSPs from the Sur nerve (Pinter, Burke, O'Donovan & Dum,

1982; Labella, Kehler & McCrea, 1989). However, only small samples of motoneurones were used in both the morphological and physiological experiments in which the possibility of direct contacts between sacral group II interneurones and motoneurones were investigated. We cannot therefore exclude this possibility, but consider that first-order sacral dorsal horn interneurones are more likely to mediate oligosynaptic or polysynaptic rather than disynaptic actions of group II muscle afferents and cutaneous afferents upon motoneurones.

Sacral interneurones might contribute to polysynaptically evoked excitation or inhibition of motoneurones via shared pathways of flexor reflexes which are evoked by group II as well as other flexor reflex afferents (Eccles & Lundberg, 1959). They might also contribute to a number of other reflexes which are induced by nerve impulses from both muscle spindle secondaries and skin afferents. The characteristic input from muscle, skin and joint afferents to midlumbar intermediate zone group II interneurones has been related to steering of locomotion and to postural reflexes induced from the vestibular apparatus and neck receptors (see Edgley *et al.* 1988; Yates, Kasper & Wilson, 1989; Jankowska & Edgley, 1993). The dominating input from the PBST and GS muscles to sacral neurones might thus, by analogy, associate these neurones with other postural reflexes. In man the most important of these might be responses induced by a forward sway, since such responses involve activation of biceps femoris and soleus muscles, while a backward sway induces activation of quadriceps and tibialis anterior muscles (see Nashner, 1976; Dietz, Quintern & Sillem, 1987; Allum, Honegger & Pfaltz, 1989; Iles & Pisini, 1992; see also Macpherson, 1988). Other functions of sacral interneurones might be associated with their input from the skin around the anus. Recent observations that external anal sphincter, but not the external urethral sphincter motoneurones, have excitatory input from PBST as well as pudendal and sural nerves (Fedirchuk, Hochman & Shefchyk, 1992) are particularly interesting in this context. They suggest that sacral interneurones co-excited by afferents in the PBST, Sur and Pud nerves are involved in the control of defecation and in postural reactions associated with defecation and with grooming of the anal region.

If some sacral group II interneurones are first-order interneurones in oligosynaptic or polysynaptic pathways to motoneurones then these interneurones must have other interneurones as their target cells and be excitatory. There is evidence, however, that some group II interneurones are likely to be inhibitory, since not only EPSPs but also IPSPs are induced in many sacral interneurones. As argued in the Results section, the latency of the earliest of these IPSPs is strongly indicative of a disynaptic coupling and their origin suggests that they are mediated primarily by sacral interneurones monosynaptically excited by group II PBST afferents, since such interneurones are only rarely encountered in more rostral segments. Some of the first- or later-order sacral inhibitory interneurones will also be needed to induce inhibition of sacral ventral horn spinocerebellar tract neurones (Grottel, Huber & Kowalski, 1991) and spinocervical tract neurones (Riddell *et al.* 1994). In both these kinds of neurone IPSPs are most readily evoked from the PBST nerve and in the latter also from the CF nerve. Interneurones co-excited by afferents in the pudendal nerve are likely to be predominantly excitatory, since Pud afferents only rarely contribute to the inhibition of sacral interneurones.

The frequent occurrence of EPSP/IPSP sequences in sacral interneurones following stimulation of the PBST, CF and Sur nerves shows that the synaptic actions of these nerves are under strong control of negative feedback networks activated by the same stimuli. However, it is difficult to decide whether, under normal conditions of activation, IPSPs cutting short EPSPs operate primarily to reduce the duration of excitatory actions or to limit the discharge rate of sacral interneurones, as the recurrent IPSPs do in motoneurones. Such IPSPs might also reflect inhibitory interactions between subpopulations of sacral interneurones as in other interneuronal systems (for references see Jankowska, 1992); activation of some subpopulations could then be combined with the depression of activity of other subpopulations as a part of the process of selecting the most suitable of alternative neuronal networks.

Similarities in the origin of primary afferent depolarization of the terminals of group II fibres in the sacral segments (Riddell, Jankowska & Huber, 1993) and the pattern of input to sacral interneurones (in particular from group II afferents of the BPST nerve and from low-threshold cutaneous afferents of the CF and Pud nerves) suggest in addition that some sacral interneurones are involved in presynaptic inhibition of transmission from group II afferents.

We may thus conclude that the population of sacral interneurones ought to include at least three distinct categories of neurones, those mediating postsynaptic excitation, postsynaptic inhibition and presynaptic inhibition.

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