Journal of Physiology (1988) **398**, *pp.* 233–257
With 11 *text-figures*
Printed in Great Britain

SENSORY INPUT TO CELLS OF ORIGIN OF UNCROSSED SPINOCEREBELLAR TRACT LOCATED BELOW CLARKE'S COLUMN IN THE CAT

BY M. AOYAMA*, T. HONGO AND N. KUDO

From the Department of Physiology, Institute of Basic Medical Sciences, University of Tsukuba, Niihari-gun, Ibaraki 350, Japan, and Department of Neurophysiology, Institute of Brain Research, Faculty of Medicine, University of Tokyo, Bunkyo-ku, Tokyo 113, Japan

(*Received 9 June 1987*)

SUMMARY

1. Sensory inputs to and locations of uncrossed spinocerebellar tract neurones in the lower lumbar cord were studied in chloralose-anaesthetized cats.

2. Neurones with axons ascending in the ipsilateral thoracic funiculi and projecting to the cerebellum were found mainly dorsal to the central canal (laminae V and VI) in the L5–L6 segments, i.e. at levels caudal to Clarke's column. Axons considered to originate from these cells were located in the dorsal half of the lateral funiculus at the level of L2, intermingled with axons of the dorsal spinocerebellar tract originating at the levels of Clarke's column.

3. Synaptic actions of primary afferents on neurones with antidromic invasion following stimuli applied to ipsilateral thoracic funiculi or to the cerebellum were investigated using intracellular or extracellular recording in the caudal lumbar segments.

4. Monosynaptic excitatory effects were evoked by electrical stimulation of group I muscle afferents of the hindlimb ipsilateral to the cell body. The majority of neurones received monosynaptic excitation from two or more muscles, predominantly extensors. They were frequently co-excited by group Ia muscle spindle and group Ib tendon organ afferents.

5. Volleys in cutaneous afferents produced excitation with short central latencies. In addition to the monosynaptic and disynaptic excitation from low-threshold cutaneous afferents, there were indications of monosynaptic effects from slightly slower conducting fibres. The majority of these neurones also received monosynaptic excitation from group I muscle afferents. Neurones with cutaneous input tended to be located more dorsally compared with those responding only to muscle afferents.

6. Volleys in joint afferents produced monosynaptic excitatory postsynaptic potentials (EPSPs) in the neurones with EPSPs from group I or group I and cutaneous afferents.

* Present address: Omiya Municipal General Center for the Physically, Mentally and Auditorily Handicapped, Omiya, Saitama 331, Japan.

7. Some neurones were disynaptically inhibited from group I muscle afferents. Convergence of monosynaptic group I excitation and disynaptic group I inhibition occurred in varieties of patterns.

8. Polysynaptic excitation, inhibition or mixed effects of both were evoked from ipsilateral cutaneous afferents and high-threshold muscle and joint afferents, whereas effects from the controlateral afferents were feeble.

9. The patterns of convergence from various species of primary afferents onto the neurones are discussed in comparison with those to other spinocerebellar tract neurones and interneurones in the lumbosacral cord.

INTRODUCTION

Dorsal and ventral spinocerebellar tracts (DSCT and VSCT) are well known respectively as uncrossed and crossed paths carrying information from the hindlimb to the cerebellum (Oscarsson, 1973). It was assumed for a long time that DSCT originates from Clarke's column (see references in Mann, 1973) which extends from the upper thoracic segment to the third or the fourth lumbar segment (Rexed, 1954). Physiological studies have extensively been performed on the nature of sensory inputs to Clarke's column neurones and demonstrated that group Ia and II muscle spindle afferents or group Ib tendon organ afferents contribute to monosynaptic excitation (Lundberg & Oscarsson, 1960; Eccles, Oscarsson & Willis, 1961*b*; Knox, Kubota & Poppele, 1977) and disynaptic inhibition (Eccles *et al.* 1961*b*; Hongo, Jankowska, Ohno, Sasaki, Yamashita & Yoshida, 1983*a*, *b*) and also volleys in joint or cutaneous afferents evoked excitation to these neurones (Lundberg & Oscarsson, 1960; Mann, 1971; Lindström & Takata, 1972; Kuno, Muñoz-Martinez & Randic, 1973). In general, individual Clarke's column neurones have been considered to receive modality-specific information from a relatively small area of the ipsilateral hindlimb (Oscarsson, 1973).

Some spinocerebellar neurones with uncrossed axons, on the other hand, have been found outside Clarke's column: ventral or lateral to the column (Kuno *et al.* 1973) or below the caudal level of the column (Hungo, Okada & Sato, 1967; Aoyama, Hongo & Kudo, 1973; Tapper, Mann, Brown & Codgell, 1975). Recent anatomical investigations using retrograde transport of horseradish peroxidase (HRP) revealed that there exist a few groups of spinocerebellar tract neurones with ipsilaterally ascending axons in the lumbar cord caudal to Clarke's column (Matsushita, Hosoya & Ikeda, 1979). The question will be raised, therefore, whether the uncrossed spinocerebellar tract originating from neurones outside Clarke's column is functionally different from that originating in Clarke's column.

The main purpose of the present study was to elucidate the nature of synaptic actions from various afferents of the hindlimb on uncrossed spinocerebellar tract neurones below the caudal level of Clarke's column, especially those in laminae V and VI of the lumber enlargement (Matsushita *et al.* 1979). It will be shown that group I afferents of different muscle nerves converge extensively onto individual neurones. Co-excitation of these neurones by muscle, cutaneous and joint afferents will also be demonstrated.

Some of the present results have been reported in a preliminary communication (Aoyama, Hongo & Kudo, 1973).

METHODS

Preparation. Experiments were performed on twenty-three cats operated on under ether and subsequently anaesthetized with α-chloralose (initial dose, 50 mg/kg). The animals were immobilized with gallamine triethiodide (Flaxedil, Specia) and artificially ventilated during experiments. Blood pressure and heart rate were continuously monitored to determine the level of anaesthesia and conditions of the animal. Additional doses of α-chloralose (5–10 mg) were given, when blood pressure was elevated above 130–140 mmHg in response to stimulation or spontaneously, or when pupils were not properly constricted. Mean arterial blood pressure was maintained above 100 mmHg throughout the experiment by intravenous infusion of Ringer solution, Dextran or phenylephrine hydrochloride (Neosynesin, KOWA), if necessary.

Laminectomies were made to expose L4–L7 and T12–L1 spinal segments. The dorsal column and right spinal funiculi (contralateral to the recording side) were removed from T12–L1. In eleven cats, laminectomies were also performed at C1–C2 segments and the occipital bone was removed to expose the cerebellum.

The following nerves of the left hindlimb were dissected and mounted on stimulation electrodes: quadriceps (Q), gracilis (Grac), anterior biceps–semimembranosus (ABSM), posterior biceps–semitendinosus (PBST), gastrocnemius–soleus (GS), plantaris (Pl), flexor digitorum longus and flexor hallucis longus without posterior knee joint and interosseus nerves (FDL), the remaining part of tibial nerve (Tib), tibialis anterior and extensor digitorum longus, i.e. deep peroneal (DP), superficial peroneal (SP), and posterior knee joint (Joint, only in ten cats).

In eight animals hamstring, sural and superficial peroneal nerves of the right hindlimb were also dissected for stimulation.

Recording and stimulation. Intracellular and extracellular recording was made from neurones in the left spinal cord at the fifth, sixth and seventh lumbar segments. Glass micropipettes filled with 2 M-potassium citrate solution, resistance of 2–10 MΩ, were used. In one cat intracellular recording was made from axons in the lateral funiculus at L2. Arriving volleys from the peripheral nerve were recorded with a silver ball electrode placed on the L6 or L7 dorsal root entry zone. The potentials displayed on the oscilloscope (Tektronix, 565) were recorded photographically. An averaging computer (Nihon Kohden, ATAC 501) was used in some cases.

The neurone with the ipsilaterally ascending axon was identified by antidromic invasion from the left dorsolateral funiculus stimulated at the low-thoracic level rostral to the cord transection (dorsal column and right spinal funiculi) and also at the first cervical segment (eleven cats). Antidromic invasion from the cerebellum was also tested in thirteen cats. In the initial series of experiments we stimulated the surface of the cerebellum with a silver ball electrode, but often failed to reveal distinct low-threshold foci of antidromic activation of these neurones. Therefore, different sites in the depth of the anterior lobe and/or the ipsilateral inferior peduncle were stimulated with a tungsten electrode in nine cats. The tungsten electrode with a tip diameter of about 5 μm was inserted at angle of 20 deg from the vertical plane into the cerebellum, and one to fourteen tracks were tested for each neurone. Negative pulses of 0·2 ms duration were applied to the electrode against the reference electrode (silver plate) placed on the temporal muscle. The stimulation currents were always measured and limited to below 1 mA, usually below 300 μA. Each track of mapping was histologically reconstructed to determine stimulating points after the experiments.

Locating neurones and axons. In each region of the spinal cord where the neurones were recorded, the microelectrode was left in the last track. After the cord was fixed in 10% formalin, serial frozen sections (50 μm thick) were prepared and the electrode track was defined in the transverse section. The location of individual neurones was determined by the angle of the manipulator and the actual depth from the cord dorsum which was measured for each neurone recorded from. Serial transverse sections were subsequently stained with cresyl violet. No characteristic structure of Clarke's column (Rexed, 1952) was verified under the microscope. The site of each axon recorded from in the lateral funiculus was identified in the same way. In this case serial sections were made of L3 through to L5 to verify the caudal end of Clarke's column.

Abbreviations. The abbreviations of peripheral nerves were described above. The other following abbreviations were used: excitatory postsynaptic potential (EPSP), inhibitory postsynaptic potential (IPSP), postsynaptic potential (PSP), dorsal spinocerebellar tract (DSCT), ventral spinocerebellar tract (VSCT), flexor reflex afferents (FRA).

RESULTS

Identification of neurones

Intracellular and extracellular records were obtained from 127 neurones which were located in the fifth, sixth and seventh lumbar segments, that is, caudal to Clarke's column. In the first series of experiments the neurones were identified by their antidromic invasion from the ipsilateral funiculi (except the dorsal column) at the lowest thoracic level and monosynaptic excitation from group I muscle afferents (sixty-four neurones). These neurones were located in the intermediate region where monosynaptic focal potentials were evoked by group I volleys in muscle afferents as shown in Fig. 1. The neurones were differentiated from the spinocervical tract neurones, which have monosynaptic excitation from cutaneous afferents, but not muscle afferents, and are similarly located in the lower lumbar cord (Lundberg & Oscarsson, 1961; Hongo, Jankowska & Lundberg, 1968; Bryan, Trevino, Coulter & Willis, 1973; Brown, Rose & Snow, 1977; Harrison & Jankowska, 1984) and from neurones with ipsilaterally ascending axons which respond polysynaptically to stimulation of cutaneous and high-threshold muscle afferents (Lundberg & Oscarsson, 1961). Cells with axons ascending in the dorsal column (Uddenberg, 1968; Angaut-Petit, 1975; Brown & Fyffe, 1981) which received monosynaptic excitation from group I muscle afferents and cutaneous afferents (Jankowska, Rastad & Zarzecki, 1979) were also excluded from the present study, since the dorsal column was lesioned below stimulation (see Methods).

In order to test if neurones thus identified constituted another spinocerebellar tract, their antidromic invasion following stimuli applied to the cerebellum was examined in thirteen experiments. The cortical surface, various sites within the anterior lobe (different depths in one to fourteen tracks), or the ipsilateral inferior peduncle were stimulated. Out of forty-four neurones tested, forty-three neurones were antidromically activated from at least one site of stimulation in the cerebellum at below 300 μA or on the surface at below 2·8 mA. Only one track was made, without producing a response, in the remaining one neurone. Failure of antidromic invasion from the cerebellum could be due to the long distances between stimulating points and the axonal trajectory or due to damage of the axons by previous tracking. We conclude therefore that essentially all such neurones that are activated antidromically from the ipsilateral thoracic cord and monosynaptically from group I afferents project to the cerebellum. Lowest-threshold foci in the cerebellum at which stimuli below 100 μA were effective were within the ipsilateral vermis and the medial intermediate zone of the anterior lobe.

In the experiments with cerebellar stimulation, fifty-eight neurones identified by antidromic invasion from the ipsilateral spinal funiculi and the cerebellum were sampled. The sample included fourteen cells that were not excited by group I muscle afferents; eight of them were exclusively excited by cutaneous afferents (e.g. Fig. 5)

and the remaining six cells had no monosynaptic input from the tested nerves. The material also included another five cells that were excited only from cutaneous afferents. They were not tested with the cerebellar stimulation, but were antidromicallv invaded from rostral C1 which is rostral to the lateral cervical nucleus

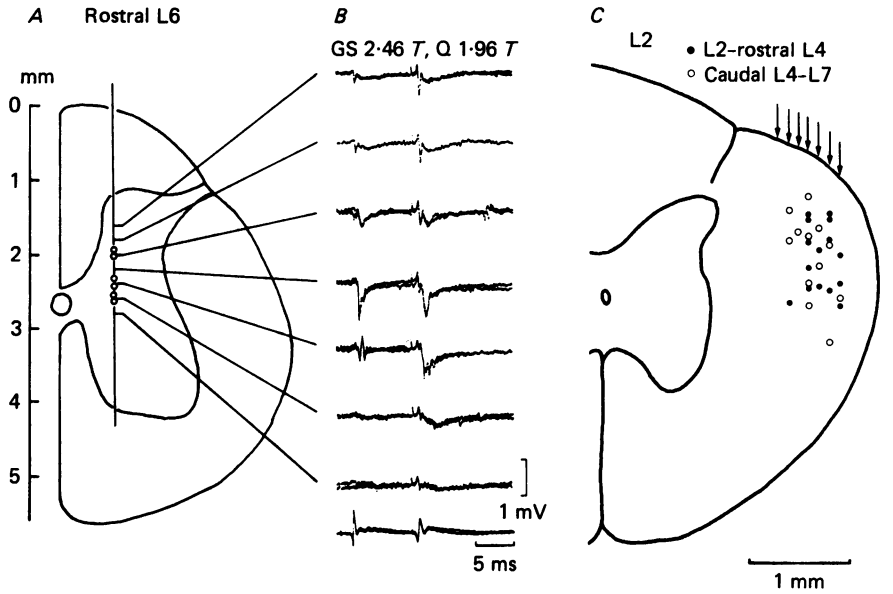

Fig. 1. Locations of spinocerebellar tract neurones (*A* and *B*) and their axons (*C*). *A*, drawing of a transverse section of the rostral part of L6 with an electrode track. *B*, field potentials evoked by stimulation with supramaximal intensities for group I afferents of gastrocnemius–soleus (GS) and quadriceps (Q) nerves (2·46 and 1·96 times threshold) and recorded along the track in *A*. Unit activities of several neurones appear upon the group I focal synaptic potentials at the depth of 2·2 and 2·4 mm. Some of these neurones were antidromically activated following stimulation of the ipsilateral spinal funiculus at T13. Open circles in *A* represent the location of such neurones collected at three different rostro-caudal planes within 400 μm. The lowest traces in *B* show incoming volleys recorded at the L7 dorsal root entry zone. *C*, locations of spinocerebellar axons originating from levels between L2 and rostral L4 (●) and between L4 caudal and L7 (○), sampled in the same cat. All the spinocerebellar axons encountered through systematic tracking (arrows) at L2 were sampled.

where the spinocervical tract terminates (Lundberg, 1964). The five cells were also considered to be spinocerebellar, because all other such cells were antidromically invaded from the cerebellum (seven cells examined). Some of them might, however, be the origin of fibres ascending in the dorsolateral funiculus and terminating in the dorsal column nuclei (Gordon & Grant, 1982).

The conduction velocities of the axons of the cells monosynaptically activated by stimulation of group I fibres were calculated from the latency of the antidromic spike evoked by stimulation of the low-thoracic funiculus and the conduction distance. They ranged from 26·9 to 114·5 m/s with the mean of 75·0 $\pm$ 16·3 (S.D.) m/s (n = 51). The distribution of conduction velocities was approximately the same for cells with input from skin and those without.

Figure 1*C* shows the locations in the lateral funiculus at L2 of individual spinocerebellar axons activated monosynaptically from group I afferents, all sampled in the same cat. The rostro-caudal level of the cell of origin was determined for each axon by means of the method used by Hongo *et al.* (1967). The axons arising from the levels between caudal L4 and L7 (open circles) were distributed similarly to those originating from the levels between L2 and rostral L4 where Clarke's column exists (filled circles).

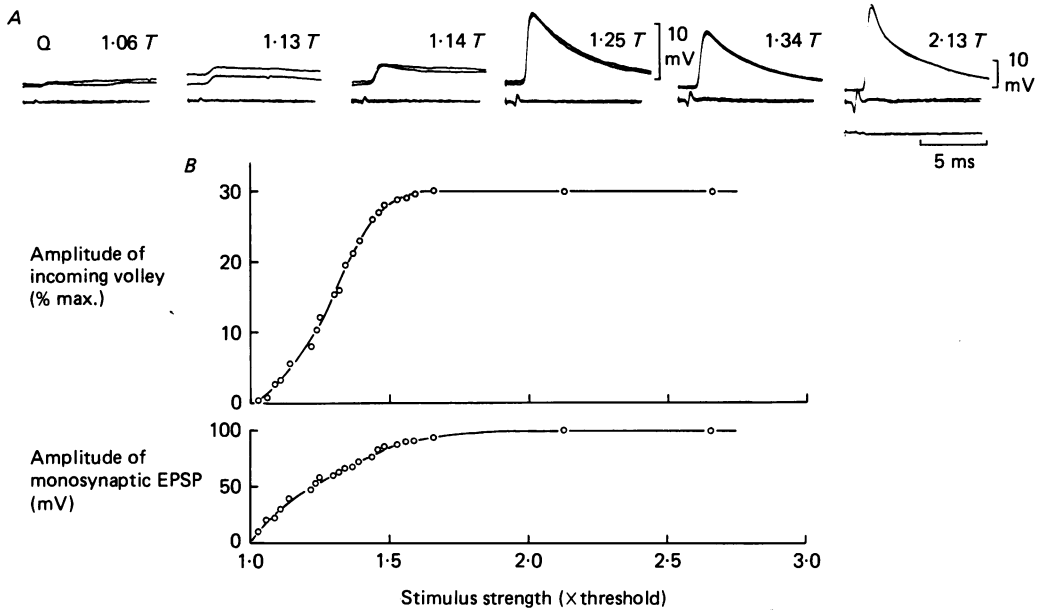

Fig. 2. EPSPs evoked by stimulation of group I afferents of quadriceps (Q) nerve. *A*, upper traces are intracellular records (positivity upward) from a neurone in L6 identified by antidromic invasion from ipsilateral thoracic funiculi. Right: two records (1·34 and 2·13 times threshold) are taken with a smaller amplification. Lower traces show records (negativity upward) from the dorsal root entry zone indicating the arrival of afferent volleys. Lowermost traces, when present, show extracellular records obtained after withdrawal of the microelectrode from the cell. Stimulus strengths are indicated in multiples of threshold of the nerve. *B*, amplitudes of monosynaptic EPSPs and those of group I incoming volleys are plotted against strengths of stimulation to Q nerve in upper and lower graphs respectively.

Monosynaptic excitatory effects from primary afferents

The following observations are based on intracellular (thirty-six cells) and extracellular (ninety-one cells) records from neurones in which effects from all or almost all dissected nerves were examined systematically. The origin of monosynaptic input was identified in principle by intracellular recording. The frequency of occurrence of monosynaptic input from different sources was, however, obtained from both intracellular and extracellular records.

The origin of the monosynaptic excitation

Muscle afferents. Monosynaptic excitatory effects from group I muscle afferents are illustrated in Fig. 2. As seen in *A*, volleys in quadriceps nerve evoked excitatory

postsynaptic potentials (EPSPs) the size of which gradually increased as the stimulus strength increased. A monosynaptic connection between the group I fibres and the neurone is evidenced by the central delay of these EPSPs (0·6 ms) estimated from the interval between the arrival of the incoming volleys to the L7 dorsal root entry zone and the onset of the EPSPs. The central latencies of EPSPs evoked from group I

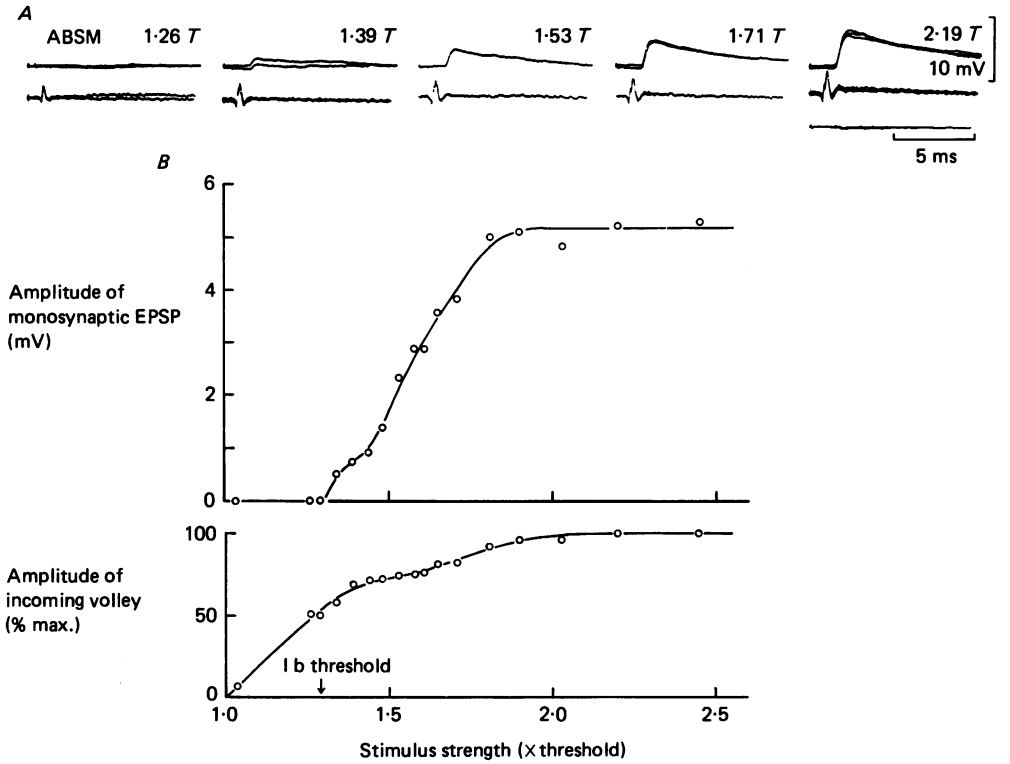

Fig. 3. EPSPs evoked by stimulation of group I afferents of ABSM nerves. Intracellular records (upper traces) were obtained from the same neurone as in Fig. 2. The arrow in the lower graph of *B* indicates the threshold for Ib fibres. For further explanation, see text.

muscle afferents in twenty-three neurones ranged from 0·5 to 1·0 ms with the mean of 0·8 ms.

Figure 2*B* shows amplitudes of monosynaptic EPSPs (upper graph) and of group I incoming volleys (lower graph) at various intensities of stimulus strength (stimulus strengths are given as multiples of the threshold for the incoming volley). Their parallel increase indicates that both Ia and Ib afferents in quadriceps nerve contributed to these EPSPs. In the case of the neurone illustrated in Fig. 3, Ia afferents of anterior biceps–semimembranosus (ABSM) nerve did not evoke any postsynaptic potentials; group Ib volleys of ABSM were differentiated from Ia volleys by a notch seen in records from the dorsal root entry zone (lower trace in *A*; cf. Bradley & Eccles, 1953). The monosynaptic EPSPs appeared only when the stimulus strength was increased over the threshold of Ib fibres (arrow in *B*) and became maximal when all group I fibres were excited; only group Ib fibres thus

appeared to contribute to these EPSPs. In some neurones monosynaptic EPSPs were evoked by only group Ia afferents (e.g. Fig. 10*A*). Such a systematic analysis to separate effects of volleys in group Ia and Ib fibres as exemplified in Figs 2 and 3 was done for thirty-six EPSPs obtained from twenty neurones. In twenty-one cases both Ia and Ib afferents of muscle nerves contributed to their monosynaptic excitation.

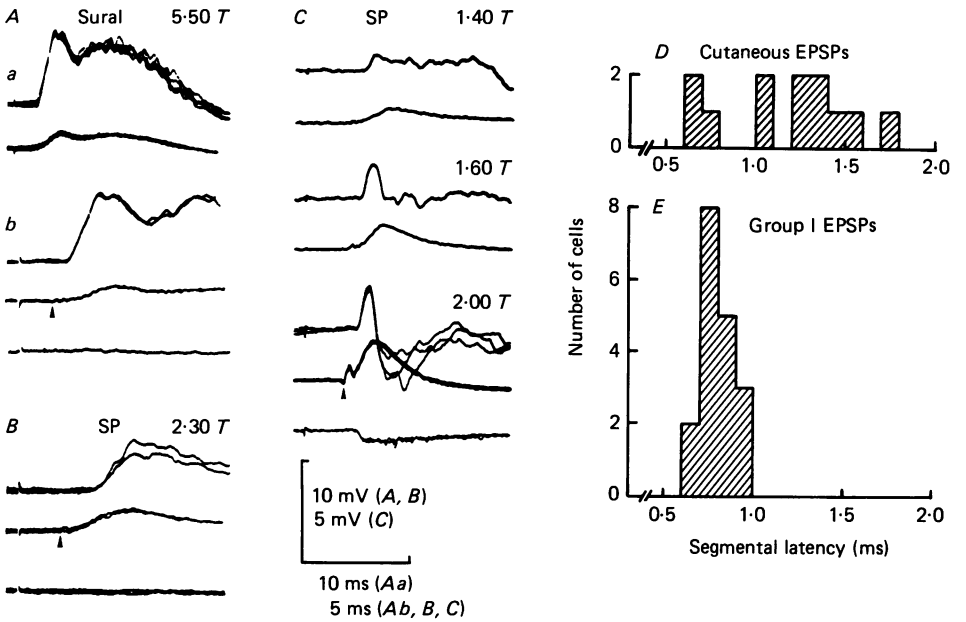

Fig. 4. EPSPs evoked by stimulation of cutaneous afferents and their latencies. *A* and *B*, EPSPs (top traces) evoked from sural (*A*) and superficial peroneal (SP) (*B*) nerve in a neurone recorded in rostral L5. The second traces show cord dorsum potentials and the third, when present, show extracellular field potentials recorded just outside the cell. *C*, PSPs (top traces) evoked by stimulation of SP at different strengths in another cell located in middle L5. Other traces are as in *A* and *B*. Arrow-heads in *A–C* indicate the arrival of incoming volleys. *D*, histogram showing segmental latencies of EPSPs evoked from cutaneous nerves. *E*, histogram showing segmental latencies of group I EPSPs evoked in the same cells as in *D*.

In others either Ia or Ib fibres were effective; exclusively Ia EPSPs and Ib EPSPs were evoked in ten and five cases respectively.

In addition to the excitation from group I afferents, there were in several neurones indications of monosynaptic effects from group II afferents. However, since it was sometimes difficult to differentiate between group II monosynaptic and group I disynaptic excitation, even with intracellular recording, the group II actions were not systematically analysed.

Cutaneous afferents. Stimulation of pure cutaneous nerves (sural, superficial peroneal) evoked EPSPs with short latencies as exemplified in Figs 4*A–C*, 5*B–C* and 10*D–E*. The central latencies of twelve such EPSPs (nine neurones) ranged between

0·6 and 1·8 ms (mean, 1·2 ms), and were distributed as shown in Fig. 4*D*. The three EPSPs with latencies below 1·0 ms (0·7 ms in two, Fig. 4*A* and *C*, and 0·6 ms in one, not illustrated) must have been evoked monosynaptically from the fastest-conducting fibres. The latencies of group I-evoked monosynaptic EPSPs in the nine

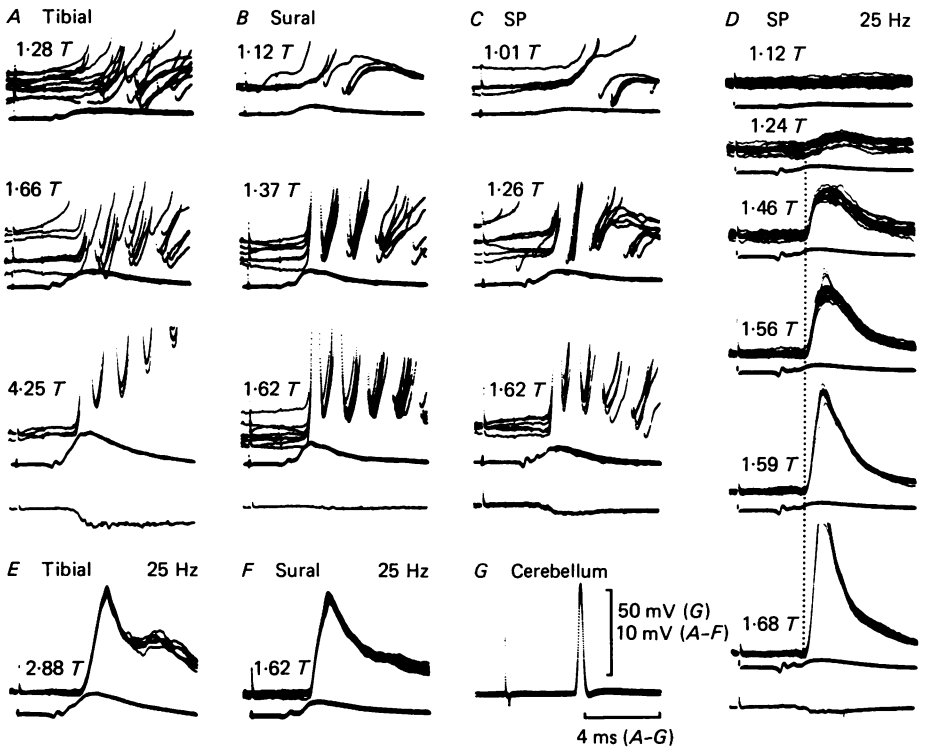

Fig. 5. EPSPs evoked by stimulation of cutaneous afferents in a spinocerebellar tract neurone of the rostral part of L5. Upper traces show intracellular records from the tract cell, and lower and lowermost traces show cord surface potentials and extracellular control records respectively. *G*, antidromic spike potentials evoked from the cerebellum. *A–C*, EPSPs evoked by stimulation with various intensities of tibial (*A*), sural (*B*) and SP (*C*) nerves. *D–F*: EPSPs evoked by repetitive stimuli (25 Hz) of SP at different strengths (*D*), and of tibial (*E*) and sural (*F*).

cells where those of skin EPSPs were measured are shown in Fig. 4*E*, for comparison.

The remaining EPSPs with somewhat longer central latencies could have been produced via two possible pathways, disynaptic from fastest-conducting fibres and monosynaptic from slightly slower fibres. The EPSPs from pure cutaneous nerves (sural, superficial peroneal) in Fig. 5 had central latencies of 1·3 ms (*B*, sural) and 1·1 ms (*C*, superficial peroneal), augmented with stimuli of increasing strength, and caused repetitive firing. The excitatory response to stimulation of the mixed tibial

nerve (*A*) showed similar properties, a latency of 1·1 ms and repetitive firing, and hence was probably produced by cutaneous afferents in the nerve. All these EPSPs survived well under high-frequency stimulation (*D–F*, verified up to 50 Hz), and their onset remained unchanged when recruited with increasing stimulus strengths

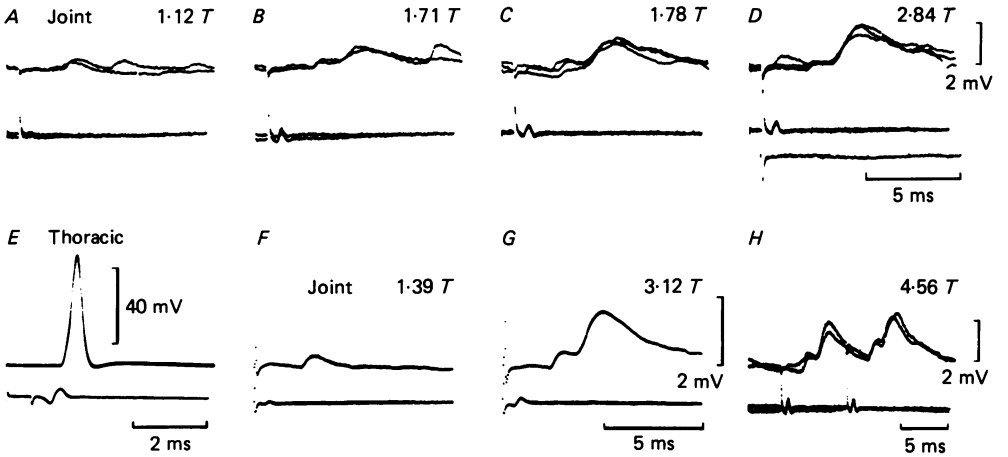

Fig. 6. EPSPs evoked by stimulation of posterior knee joint nerve. Upper traces show intracellular records from a neurone in the caudal part of L5, and lower traces indicate compound action potentials recorded from the sciatic nerve in *A–D* and *F–H* and cord dorsum potential in *E*. *E*, antidromic spike potentials evoked by stimulation of ipsilateral thoracic funiculi. *A–D* and *F–H*: EPSPs evoked by stimulation of joint nerve at different intensities. The threshold of the joint nerves was determined by recording compound action potentials in the sciatic nerve. Potentials in *F* and *G* are averaged. In *H* double shocks are applied at an interval of 6·5 ms.

(*D*). This indicates that monosynaptic effects from slightly slower-conducting fibres probably contributed to the early component of the response. On the other hand, EPSPs with longer and variable latencies were also observed, especially when stimuli were weak (Fig. 5*A*, 1·28–1·66 times threshold; *C*, 1·01; also Fig. 10*D*, 1·11–1·20; *E*, 1·02–1·10; *F*, 1·04–1·22), indicating clearly that lowest-threshold fibres can produce EPSPs through interneurones. Shortening of the latencies with increase of stimulus strength in Fig. 10*D–F* may be due to earlier firing of interneurones in the disynaptic pathway but may also be due to addition of monosynaptic effects from higher-threshold fibres. It was usually difficult to distinguish the two effects in such cases. The EPSPs evoked from skin afferents with short central latencies, below 1·8 ms, were therefore all grouped together and denoted 'oligosynaptic effects' in the following descriptions. Short-latency effects from the mixed nerve (tibial) were judged to originate from skin afferents, when single shock stimuli evoked repetitive firing and the central latency was longer than those from group I afferents, i.e. 1·0–1·8 ms for EPSPs (e.g. Fig. 5*A*, Fig. 10*F*).

Joint afferents. EPSPs from the posterior knee joint nerve were evoked in three out of nineteen neurones examined intracellularly. There were indications of a monosynaptic coupling as illustrated in Fig. 6*A–D* and *F–H* in which the upper and

lower traces represent the intracellular records and the compound action potential recorded from the sciatic nerve. Two components of responses were evoked by stimulation of the joint nerve. The first component appeared at a stimulus strength just above threshold for the nerve and was maximal at about 1·8 times threshold. The second component required a stimulus strength exceeding 1·6 times threshold and became maximal at 2·8 times threshold. Their latencies measured from the shock artifacts were 2·4 and 3·7 ms respectively; the former was similar to the latency of group I-evoked EPSPs. Similar responses to two successive shocks (*H*) suggested monosynaptic coupling of both faster- and slower-conducting fibres with the neurone being recorded from. Only one component of responses was evoked from joint afferents in other two neurones (another cat) with latencies of 3·2 ms (similar latencies of group I EPSPs from plantaris and FDL) and 5·2 ms, which corresponded to the early and late component of Fig. 6, respectively. The thresholds and the latencies of the early and late components of EPSPs were comparable to those of EPSPs from joint afferents with fast and slow conduction velocities which were previously found in Clarke's column neurones and were interpreted as being evoked monosynaptically (Lindström & Takata, 1972).

The frequency of occurrence

The presence of monosynaptic input from group I muscle and skin afferents was examined in both intra- and extracellularly recorded neurones. In extracellular records the monosynaptic excitatory effects too weak to generate spike potentials were tested by using the spatial facilitation technique. Since at least one peripheral nerve usually had an excitatory effect strong enough to evoke an action potential in the cell, a subthreshold stimulation of this nerve was applied synchronously with stimulation of other tested nerves. When no nerves were potent enough to excite, combined stimulation of two or three nerves was used as the test stimulus. The high sensitivity of the technique was confirmed by intracellular records subsequent to extracellular records from the same neurones as shown in Fig. 7. Figure 7*A–E* shows extracellular records from the neurone identified antidromically by stimulation of the ipsilateral cervical cord (*A*). Stimulation of plantaris nerve with supramaximal intensity for group I fibres failed to evoke spike potentials (*D*), but when combined with subliminal stimulation of FDL (*C*) action potentials were evoked as in *E*. Weak monosynaptic excitation from plantaris afferents was confirmed by subsequent intracellular records from the neurone (*I–K*).

In Fig. 7*E* central delays of action potentials fluctuated between 0·9 and 1·5 ms. Subsequent intracellular records revealed that volleys in both FDL and plantaris afferents evoked only monosynaptic EPSPs without additional polysynaptic effects on the tested neurone. Consequently all action potentials in *E* were monosynaptic responses. Similar observations in six other cases (three cells) showed that the central latencies of monosynaptic excitation from group I afferents were 0·9, 1·1, 1·1, 1·2, 1·4 and 1·5 ms. In extracellular records, therefore, the responses to stimulation of group I afferents with central delays of up to 1·5 ms were considered as evoked monosynaptically. From similar comparisons a central delay of up to 2·6 ms was used as the criterion of oligosynaptic excitation from skin afferents (see above). The central latency of cutaneous, oligosynaptic excitation thus identified

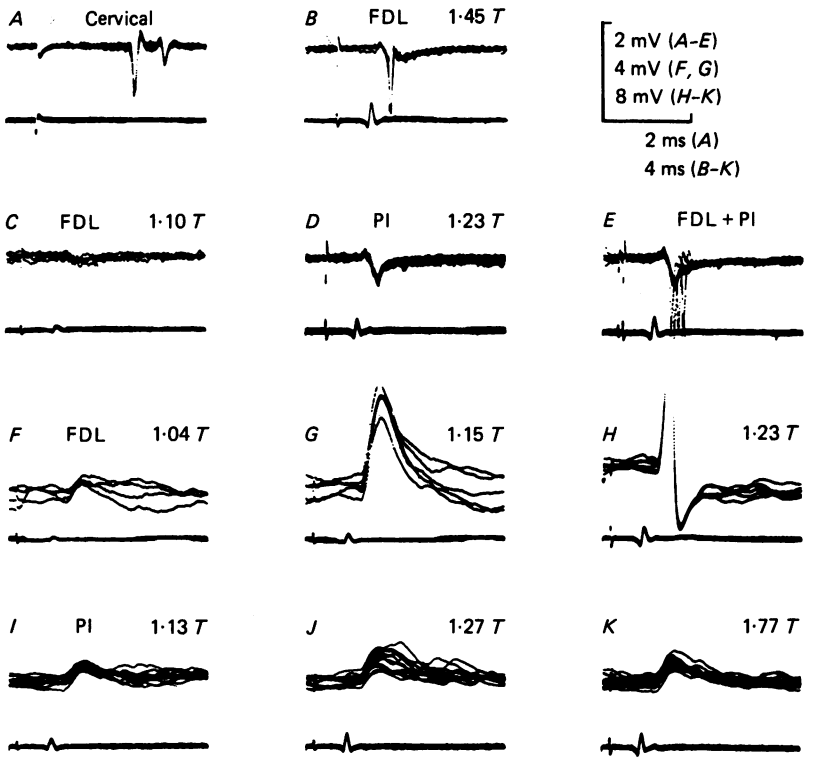

Fig. 7. Comparison of extracellular and intracellular responses of monosynaptic excitation from group I muscle afferents. *A–E*, upper traces show extracellular records in the middle part of L5 segment. Antidromic spike potentials of two neurones were evoked by stimulation of ipsilateral cervical cord with different latencies (*A*). The neurone with the larger spike was orthodromically activated from FDL group I fibres (*B*). In *C* and *D* with no spike potentials, stimulation of FDL nerve at subthreshold intensity for exciting the neurone (1·10 times threshold) and stimulation of plantaris (Pl) nerve (1·23 times threshold) were applied separately. When both were stimulated together at an appropriate interval so that group I volleys arrived at the dorsal root entry zone at the same time, spike potentials occurred (*E*). *F–K*, upper traces show subsequent intracellular records from the same neurone. Stimulation of FDL nerves evoked monosynaptic EPSPs (*F* and *G*) and action potentials (*H*). *I–K* show monosynaptic EPSPs from Pl nerve. Even group I supramaximal stimulation of Pl nerve did not produce action potentials (*K*). Lower traces in all show cord surface potentials.

was 0·8–2·6 ms in range ($1{\cdot}4 \pm 0{\cdot}4$ (S.D.) ms, $n = 27$), and was fixed in each case when strong stimuli (above 3 times threshold) were used.

Table 1 summarizes the results thus obtained from neurones in which all the dissected nerves were examined. In the intracellular recordings the monosynaptic input from group I muscle afferents was found in most and the oligosynaptic input from cutaneous afferents in about half of the neurones examined. The figures were approximately the same for 'All cells' (see footnote of Table 1) and for the group of cells identified as spinocerebellar by antidromic invasion from the cerebellum, though the input from skin was more frequently seen in the latter sample. The

extracellular recordings also show that the group I input occurred more frequently than the skin input, though the probabilities were less than in intracellular data. The lower values are undoubtedly due to the method being less sensitive than intracellular recording.

TABLE 1. Frequency of occurrence of various inputs. Percentages and ratios (in parentheses) of numbers of cells with the input *versus* numbers of cells examined

Input	Intracellularly recorded cells	Extracellularly recorded cells
	All cells	
Group I monosynaptic	97 (35/36)	80 (73/91)
Skin mono-oligosynaptic	44 (16/36)	25 (23/91)
Joint* monosynaptic	15 (3/19)	0 (0/16)
No input	0 (0/36)	7 (6/91)
	Cells identified by cerebellar stimulation	
Group I monosynaptic	85 (6/7)	75 (38/51)
Skin mono-oligosynaptic	71 (5/7)	29 (15/51)
Joint* monosynaptic	0 (0/2)	0 (0/3)
No input	0 (0/7)	12 (6/51)

* Only posterior knee joint nerve tested. 'All cells' indicates cells identified by thoracic stimulation plus cells identified by cerebellar stimulation.

Polysynaptic effects

Inhibition with short latencies

Group I muscle afferents. Inhibitory postsynaptic potentials (IPSPs) as in Fig. 8*A* and *B* were evoked from group I muscle afferents in eight out of the thirty-six intracellularly recorded neurones. The central latencies of these IPSPs ranged between 1·1 and 1·7 ms (mean 1·4 ms, $n = 8$). These values were about 0·6 ms longer than the latencies of monosynaptic EPSPs from group I afferents (see above, Fig. 4*E*), indicating that the IPSPs were evoked disynaptically.

Tests on group I IPSPs evoked from thigh muscle nerves suggested that both Ia and Ib fibres could be the origin of the disynaptic IPSPs. In two cases as in Fig. 8*A*, IPSPs began to appear and were augumented when the stimulus strength was increased to group Ib range, while in another case stimulation to the group Ia range did evoke disynaptic IPSPs (not illustrated).

Cutaneous afferents. Distinct IPSPs with a short latency were evoked from low-threshold cutaneous afferents only in one cell (Fig. 4*C*) out of thirty-six cells examined. An IPSP was revealed by intracellular passage of current in another cell. All the other IPSPs (eighteen cases) evoked from cutaneous afferents (sural, superficial peroneal) had long latencies (above 3·7 ms), and are included in the descriptions of the FRA effects (next section).

Effects from the FRA

The effects of stimulation of high-threshold muscle and cutaneous afferents (flexor reflex afferents, FRA; cf. Eccles & Lundberg, 1959) were tested in twenty-nine intracellularly recorded neurones. The effects from joint afferents were also examined

in fourteen of them. Some FRA actions were evoked polysynaptically from the ipsilateral nerves in all these neurones, though not necessarily from all the tested nerves. In fourteen neurones effects from different nerves were similar, predominantly excitation in ten neurones and predominantly inhibition in four. In

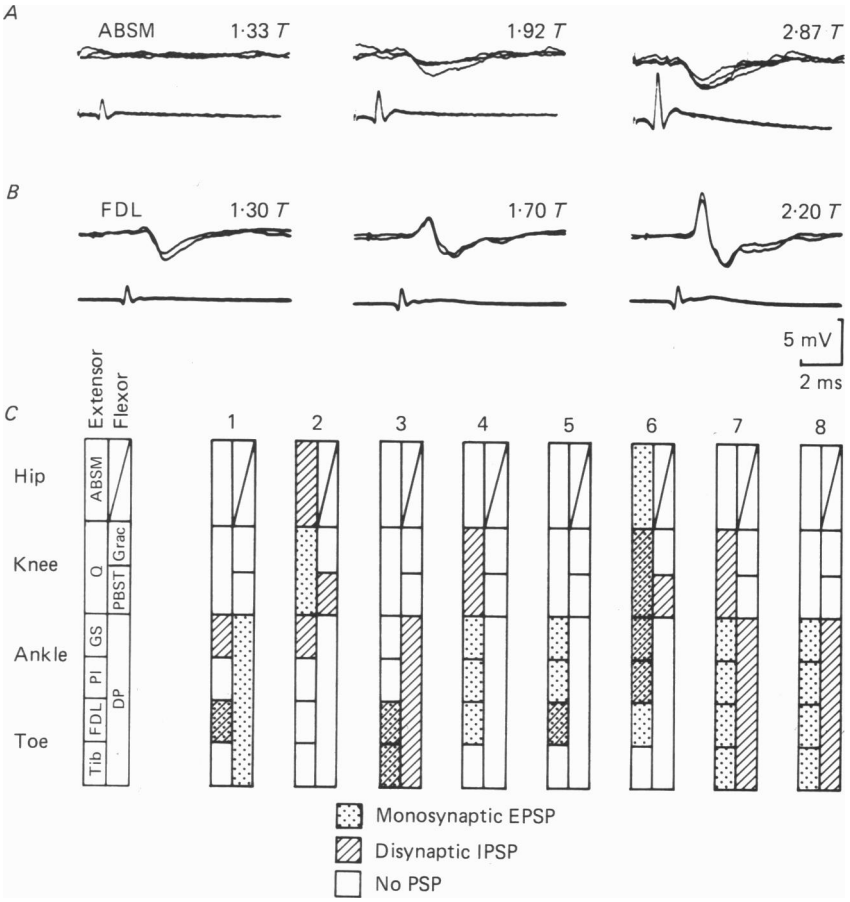

Fig. 8. Disynaptic IPSPs evoked by stimulation of group I afferents. *A* and *B*, upper and lower traces are intracellular records from two neurones, one in middle L5 (*A*) and the other in caudal L5 (*B*), and cord surface potential, respectively. The neurone in *B* is the same as in Fig. 6. *C*, the pattern of convergence of monosynaptic EPSPs and disynaptic IPSPs evoked from group I muscle afferents in eight neurones. Each of the columns 1–8 represents one cell, and shows the effects of stimulation of nerves as indicated in the leftmost column. Effects from hip flexors were not tested. Monosynaptic excitation is indicated by dots and disynaptic inhibition by hatching. Open columns show no such PSPs.

fifteen other neurones excitation was evoked from some nerves and inhibition from others. There was no clear relation between the pattern of the FRA effects and that of monosynaptic input in individual neurones.

In contrast to effects of the ipsilateral FRA, stimulation of the contralateral FRA did not produce any effects in seven out of eleven neurones tested. Some PSPs were

evoked in the other four neurones, but they were mixed excitatory and inhibitory and of much smaller amplitude than those from ipsilateral nerves.

The patterns of convergence

This section describes the pattern of convergence of mono- and oligosynaptic excitation from group I, cutaneous and joint afferents and disynaptic inhibition from group I muscle afferents.

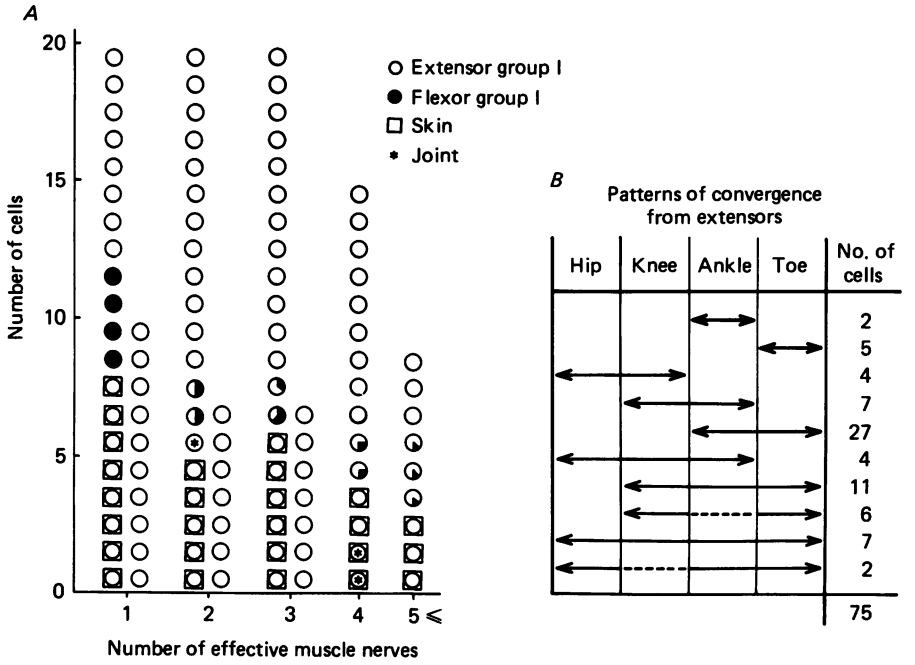

Fig. 9. The degree and the pattern of convergence of monosynaptic excitation from group I muscle and joint fibres and short-latency excitation from cutaneous fibres. Intracellularly and extracellularly recorded neurones are both included. *A*, frequency distribution of the number of effective muscle nerves. Ordinate, number of neurones. Abscissa, the number of muscle nerves evoking monosynaptic excitation. Each circle represents one neurone. Open and filled circles represent neurones that responded only to extensor and flexor muscle nerves respectively. Neurones excited from both extensors and flexors are indicated by circles with partly filled areas, where the proportion of the open and filled areas indicates the proportion in the number of effective extensor and flexor nerves. Squares and asterisks show convergence of additional excitation from cutaneous and joint nerves, respectively, on the neurone. *B*, the patterns of convergence of group I excitation from extensor muscle nerves. Rightmost column shows the number of neurones of each type. For further explanation, see text.

Convergence of effects from group I afferents of different muscles

Figure 9*A* shows the frequency histogram of neurones receiving monosynaptic excitatory effects from various numbers of muscle nerves. Thirty out of 107 neurones (28%) received monosynaptic excitation from only one muscle nerve, and the remaining seventy-seven neurones (72%) were excited from two or more different

muscle nerves. The maximum convergence of group I afferents from six different muscle nerves was observed in three neurones.

Monosynaptic excitation by group I afferents from flexors was much less frequent than excitation from extensors; only thirteen neurones (12%) responded monosynaptically to stimulation of flexors (circles with filled area in Fig. 9*A*) and all but four were also excited by volleys in group I afferents from extensors. In the remaining ninety-five neurones the extensor muscle nerves were the only source of group I monosynaptic excitation (open circles). Seventy-five of them were monosynaptically excited from more than one extensor muscle, and were classified into ten types according to the muscles of the receptive field as shown in Fig. 9*B*, where hip, knee, ankle and toe extensors were grouped together. For each type the receptive field is indicated by a continuous line, and the number of cells of each type is given in the right-hand column. In only seven of the seventy-five neurones was the convergence from within the same muscle group, ankle or toe extensors, while in the remaining sixty-eight neurones (91%) the inputs converged from muscles of different joints. Out of forty-four neurones with monosynaptic inputs from muscles of two different joints, thirty-eight neurones (86%) were excited by volleys in afferents from extensors of the adjacent joints, that is, hip and knee, knee and ankle or ankle and toe. The convergence of muscle afferents from three joints also had similar patterns; 88% (fifteen out of seventeen cells) of neurones received excitation from hip, knee and ankle or knee, ankle and toe muscle afferents. Hence there was a tendency for individual neurones to be excited predominantly by volleys in afferents from extensors of the same or the adjacent joints.

Monosynaptic excitatory and disynaptic inhibitory PSPs were evoked from different combinations of muscle nerves as shown in Fig. 8*C* for all the eight cells that received the inhibition. Note that disynaptic IPSPs originated from various and often multiple (neurones 1, 2, 3, 6, 7) muscles. The combinations of the inhibitory and excitatory inputs were complex, but some main features may be pointed out as follows. First, the excitation and inhibition were frequently evoked from antagonists (neurones 1, 2, 3, 6, 7, 8). These were of both patterns, flexor excitation–extensor inhibition (neurone one) and extensor excitation–flexor inhibition (neurones, 2, 3, 6, 7, 8). Second, combined excitation and inhibition from flexors never occurred but that from extensors was found in all cells except one (neurone 8). The same extensor nerve even gave rise to both EPSPs and IPSPs (neurones 1, 3, 5, 6).

Convergence of excitation from muscle, cutaneous and joint afferents

Table 2 shows neurone groups classified according to the species of afferents of their excitatory inputs, examined intracellularly. Approximately half the cells (twenty of thirty-six) received mono- or oligosynaptic inputs only from group I afferents or from cutaneous afferents, among which cells with group I inputs predominated. The remaining cells (sixteen of thirty-six) received inputs converging from different species of afferents. Figure 10 illustrates co-excitation of a neurone by muscle and cutaneous afferents, as an example. Both low- and high-threshold group I (most likely Ia and Ib) afferents from GS and plantaris produced monosynaptic EPSPs as shown in *B* and *C*, while EPSPs from ABSM seemed to be evoked mainly by Ia volleys (*A*) since they reached the maximal amplitude below the threshold for

the second component of the incoming volleys. EPSPs from cutaneous afferents (*D–F*) appeared with somewhat longer latencies as described before. Such skin EPSPs were evoked in addition to EPSPs of muscle origin in 42% (fifteen of thirty-six) of neurones (Table 2). They were found in neurones with EPSPs from one or

TABLE 2. Types of cells

Input	All cells	Cells identified by cerebellar stimulation
Group I only	53 (19/36)	29 (2/7)
Skin only	3 (1/36)	14 (1/7)
Joint only	0 (0/19)	0 (0/2)
Group I+skin	36 (13/36)	57 (4/7)
Group I+joint	5 (1/19)	0 (0/2)
Group I+skin+joint	11 (2/19)	0 (0/2)
Undetected	0 (0/36)	0 (0/7)

The data derive from intracellularly recorded cells only. Values given as percentages and numbers in parentheses are those of cells with an identified input as a proportion of cells examined. 'All cells', as in Table 1.

several muscle nerves, but only in those excited by group I afferents from extensors (see Fig. 9*A*, where extracellular data are included). Convergence of excitation from two or three cutaneous nerves (as in Figs 5 and 10) was seen in 38% (six out of sixteen) of intracellularly recorded cells with skin input.

EPSPs from joint afferents were seen in three out of nineteen tested neurones (16%). They were evoked together with EPSPs from extensor group I or extensor group I and cutaneous afferents (Table 2).

Correlation between location of the neurones and their excitatory input

The vast majority of the neurones examined were found in L5 and in the rostral part of L6; only one cell was recorded in L7.

Figure 11*A–C* shows the locations of forty-four neurones with monosynaptic input from group I muscle afferents which were identified by antidromic activation from ipsilateral thoracic funiculi in six cats. The locations are plotted in representative planes of three rostro-caudal levels, in rostral (*A*), middle (*B*) and caudal (*C*) L5; among neurones in *C* are included four neurones from the rostral part of L6. Most of the neurones were found dorsal to the central canal at locations corresponding to middle to medial parts of Rexed's laminae V and VI. Neurones receiving monosynaptic input from one, two or more muscles did not show marked difference in their location, but there was some tendency for those with a single input (×) to be more lateral.

Figure 11*D* shows the location of neurones found in seven other cats and identified by their antidromic activation from the cerebellum. These neurones were classified into three groups depending on their input: open circles and open triangles represent the neurones responding only to muscle or cutaneous afferents, respectively, while filled triangles represent neurones with muscle plus cutaneous input. There is a certain tendency for more dorsal location of neurones with cutaneous input, whether they were or were not co-excited by muscle afferents.

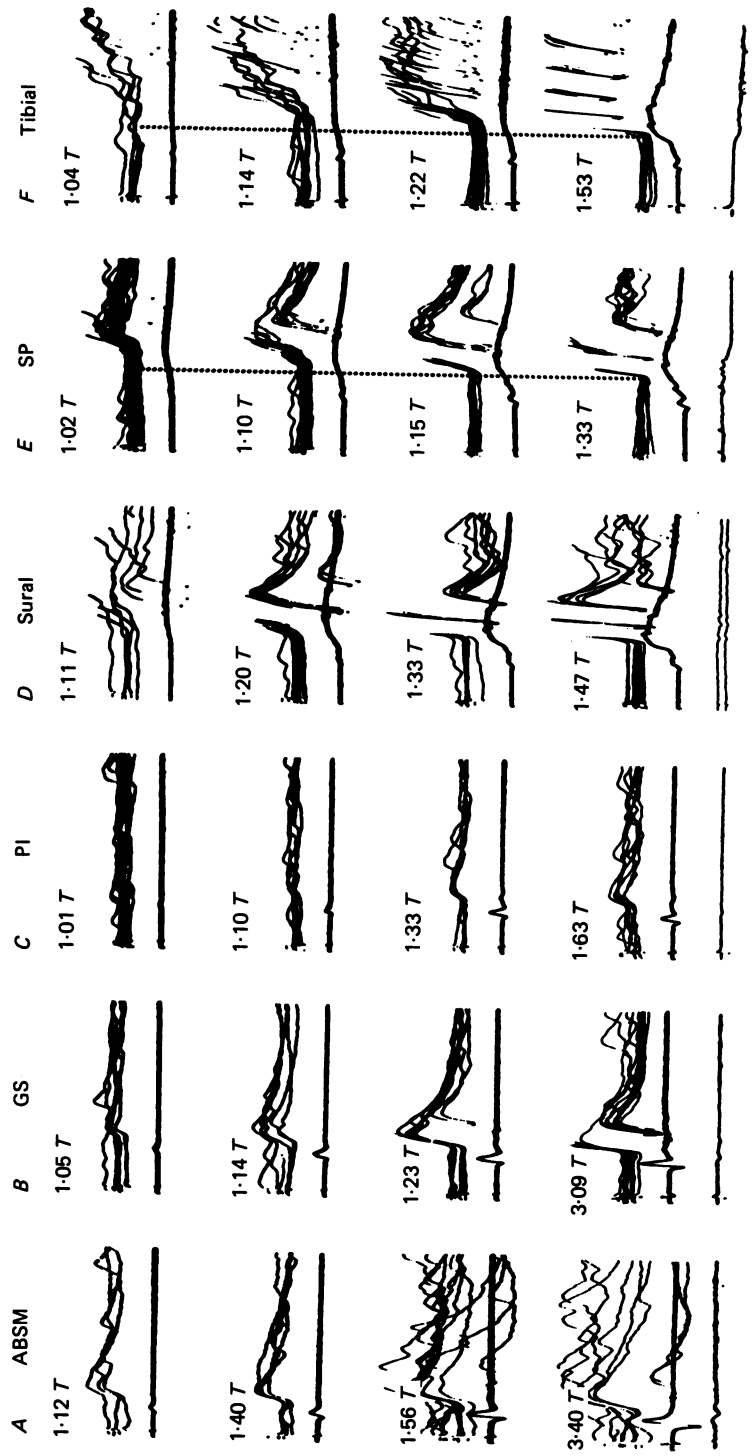

Fig. 10. Convergence of inputs from group I muscle and skin afferents. Upper traces show intracellular potentials recorded from a neurone in the rostral part of L5, and lower traces cord dorsum potentials. Bottom traces of each column are extracellular control records. *A–C*, monosynaptic EPSPs evoked from group I muscle afferents from ABSM (*A*), GS (*B*) and PI (*C*). *D–F*, EPSPs evoked from sural (*D*), SP (*E*) and tibial (*F*) cutaneous afferents. *G*, antidromic action potentials following stimulation of the cerebellum.

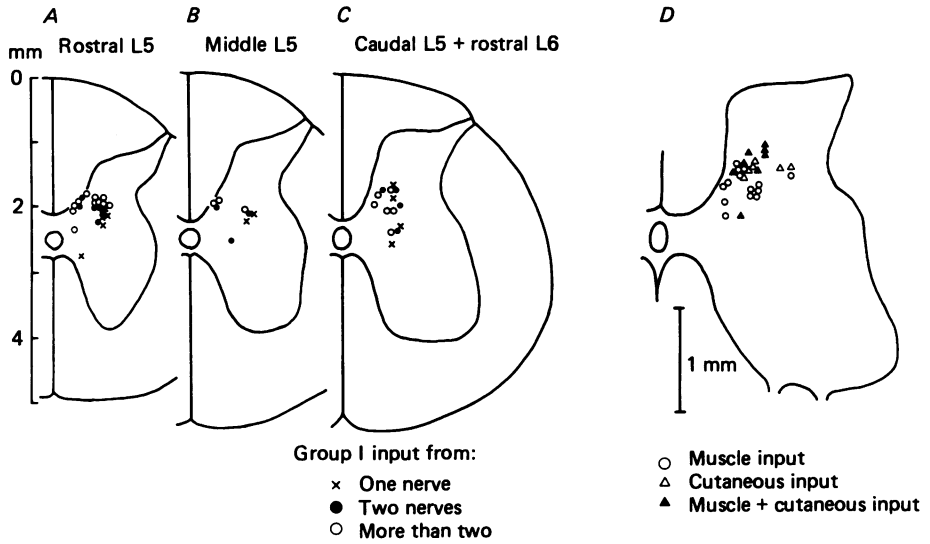

Fig. 11. Locations of neurones with different inputs. The drawings show the locations of neurones identified by antidromic invasion following stimulation of the ipsilateral thoracic funiculi (*A–C*) and the cerebellum (*D*), plotted on representative transverse sections of L5. Each neurone was located as described in Methods. *A–C*, positions of forty-four neurones from six different experiments are superimposed in the drawings from the rostral L5 (*A*), middle L5 (*B*) and caudal L5 and rostral L6 (*C*). Crosses, filled circles and open circles represent neurones that responded to stimulation of one, two and more muscle nerves, respectively. *D*, locations of twenty-eight cerebellar-identified neurones recorded in L5 in seven other experiments are superimposed in the drawing. Open circles and open triangles represent neurones receiving their input from muscle and cutaneous afferents respectively. Filled triangles represent neurones that responded to stimulation of both muscle and cutaneous nerves.

DISCUSSION

Uncrossed spinocerebellar tract originating in caudal lumbar segments

The present study has shown that there exist ipsilaterally ascending neurones with monosynaptic input from muscle, joint and/or cutaneous afferents below the caudal level of Clarke's column, i.e. below L3 or L4 (cf. Rexed, 1954; Matsushita *et al.* 1979). These neurones were most numerous in L5 and the rostral part of L6. We have confirmed histologically that there was no cell cluster characterizing Clarke's column at these levels. The results of the analysis of projections of the investigated cells to the cerebellum indicate that their axons constitute the uncrossed spinocerebellar tract (Fig. 1*C*).

The investigated neurones are situated in the medial part of the dorsal horn (see Fig. 11) where group I volleys produce monosynaptic focal synaptic potentials (see Fig. 1; also Eccles, Fatt, Landgren & Winsbury, 1954), but have long been overlooked in previous electrophysiological studies (Eccles, Eccles & Lundberg, 1960; Hongo, Jankowska & Lundberg, 1966, 1972). Hongo *et al.* (1967) found that some spinocerebellar axons in the lateral funiculus at the L1 level may have their cells of origin at the L5 and L6 level, by systematically comparing the latencies of direct

responses of these axons to stimulation of different levels of the lumbar cord. Only a small number of these fibres have been penetrated in the experiments with intra-axonal recording, but this may be explained by the smaller population of their cells of origin in comparison with Clarke's column neurones. It is recalled in this context that part of the mass ascending discharges evoked in the ipsilateral lateral funiculus by maximal group I volleys remained after transection of the dorsal column in the lower part of the L4 segment (Oscarsson, 1957*a*). These discharges could well be in the axons of the now-described spinocerebellar tract originating below the level of transection, since such dorsal column lesions interrupt all the monosynaptic excitatory input to Clarke's column neurones (Hongo *et al.* 1983*a*).

The recent anatomical study utilizing retrograde transport of HRP has disclosed spinocerebellar tract neurones at several locations previously not known, and among them are uncrossed spinocerebellar tract neurones in the medial part of laminae V–VI of L5 and L6 (classified as the lumbar medial lamina VI–SCT) which also differ from Clarke's column neurones in their shape and size (Matsushita *et al.* 1979). Another group of uncrossed spinocerebellar tract neurones disclosed by Matsushita *et al.* (1979) (the lamina V–SCT neurones) are similarly located in the lower lumbar cord but more dorsally and extending more laterally, i.e. in the whole extent of lamina V. The segmental level and the laminar location of the spinocerebellar tract neurones in the present study (Fig. 11) correspond to those of the above two groups described by Matsushita *et al.* (1979). Among all the neurones studied, those with only muscle input correspond rather well to the medial lamina VI–SCT. Those with cutaneous input, whether or not activated from muscle also, tend to distribute more dorsally and appear to correspond to lamina V–SCT and dorsal cell groups of medial lamina VI–SCT. Cells with input from both muscle and skin among them distribute more medially within the region. In the medial part of laminae V and VI, therefore, cells of different input types are located together with a tendency for cells with cutaneous input to be distributed more dorsally.

Comparison with other spinocerebellar tracts from the lumbosacral cord

A characteristic feature of the neurones under study is a high degree of convergence onto them of the same species of afferent from different nerves and even of different species of afferents. The features of convergence can be compared with those of Clarke's column neurones as follows. More than 70% of the now-investigated neurones with group I input were found to receive monosynaptic excitation from two or more muscles, operating at the same and at different joints (Fig. 9). This appears to be in contrast to monosynaptic excitation from group I fibres of Clarke's column neurones, the majority of which have been considered to be activated primarily from a single muscle or a few synergists (Holmqvist, Lundberg & Oscarsson, 1956; Eccles, *et al.* 1961*b*; Mann, 1971; Kuno *et al.* 1973; Knox *et al.* 1977); Mann (1971) and Knox *et al.* (1977) reported convergence of monosynaptic actions from more than one muscle or muscle group in about 20% of DSCT axons, but the sampled axons in these studies could originate not only from Clarke's column but also from more caudal levels. For comparing the degree of convergence in the caudal spinocerebellar neurones now under study with that in Clarke's column neurones, only some of the previously reported observations can be used, because some studies used only intra-

axonal recording from DSCT fibres which discloses only supraliminal effect (Holmqvist *et al.* 1956) and other studies used a much smaller number of peripheral nerves for testing (Kuno *et al.* 1973; Knox *et al.* 1977). In the intracellular study of Clarke's column neurones by Eccles *et al.* (1961*b*) in which various nerves of the hindlimb were extensively tested, convergence of afferents from different muscles was found in 36% (twenty-one out of fifty-eight) of neurones. Convergence from two or more muscles thus appears to be significantly more frequent (74%), and the receptive field (up to six muscles) appears to be wider in the present spinocerebellar tract neurones. The same holds true for the neurones with cutaneous input. Clarke's column neurones were reported to respond to stimulation of only one cutaneous nerve (Kuno *et al.* 1973), while 38% of the present spinocerebellar neurones with skin input were excited by cutaneous afferents from two or three nerves.

Convergence from group I muscle and skin afferents has been reported to be rather rare in Clarke's column neurones, which hence have been subdivided into two main functional groups, the proprioceptive and exteroceptive component (Oscarsson, 1973). Only a small proportion of neurones (6%, Kuno *et al.* 1973) were found to be activated by both muscle and cutaneous afferents. Mann (1971) gave the value of 8%, but axons originating below Clarke's column could have been also included. In the case of the caudal spinocerebellar neurones, in contrast, the convergence of muscle and cutaneous afferents occurred to a much higher degree, 42% (fifteen of thirty-six cells, cf. Table 2) as revealed by intracellular recording.

The ventral spinocerebellar tract (VSCT), another major pathway carrying information from the lumbar cord to the cerebellum, originates caudal to Clarke's column and ascends after crossing (Oscarsson, 1957*b*; Hubbard & Oscarsson, 1962; Burke, Lundberg & Weight, 1971). Of the two divisions of the VSCT, Ib-VSCT neurones show an extensive convergence of group I afferents from various muscles of the hindlimb (Oscarsson, 1957*b*; Eccles, Hubbard & Oscarsson, 1961*a*), and the spinal border cells of another division of the VSCT show occasional occurrence of the convergence of group Ia and Ib afferents from the same muscle (Lundberg & Weight, 1971), similar to the neurones under study. However, the convergence of oligosynaptic input from cutaneous nerves has not been observed in VSCT neurones (Eccles *et al.* 1961*a*; Lundberg & Weight, 1971). The most distinct difference between the VSCT neurones and the investigated neurones is with respect to polysynaptically induced effects from the FRA. In the former a pronounced inhibition from the FRA is one of their characteristic features (Oscarsson, 1957*b*; Eccles *et al.* 1961*a*; Lundberg & Weight, 1971), while inhibition was less frequent and weaker in the majority of the neurones studied.

The potent excitatory inputs from the periphery as well as the weakness of the FRA effects on the uncrossed spinocerebellar neurones now reported may indicate that they belong to a category similar to Clarke's column neurones and not to VSCT neurones. Anatomically too, they can be combined with Clarke's column neurones to form the DSCT, as indicated by the axonal courses (Fig. 1).

Comparison of peripheral input between the present neurones and laminae V–VI interneurones

The area of location of investigated neurones overlaps with the area in L5 and L6 where interneurones with monosynaptic input from group I afferents are most common (Eccles *et al.* 1960; Hongo *et al.* 1966, 1972; Jankowska, Johannisson & Lipski, 1981). In addition to the similarity in location, these two groups of neurones appeared to have several features of their input in common, as compared with previous reports on the laminae V–VI interneurones (Eccles *et al.* 1960; Hongo *et al.* 1966, 1972; Jankowska *et al.* 1981; Harrison & Jankowska, 1985*a*, *b*). A large population of both of them are co-excited by group Ia muscle spindle and group Ib tendon organ afferents with an extensive convergence of group I afferents from different muscles. The convergence of afferents from extensors appeared to be a frequent feature for both these groups of neurones although perhaps more dominant in the spinocerebellar tract neurones. In addition to the excitation by group I afferents both groups were often di- or polysynaptically excited from cutaneous afferents, the possibility of monosynaptic excitation of the tract neurones by slower-conducting cutaneous afferents being discussed in the Results. Both were also often inhibited from group I afferents of the same or different nerves from which the monosynaptic excitation is evoked. The inhibition seemed, however, to be less frequent and polysynaptic effects from the FRA tended to be weaker in the tract neurones, but these may be due to differences in conditions of preparations.

The similarities of peripheral inputs in the two groups of neurones are rather difficult to interpret at the present stage of our knowledge but deserve comments. Harrison & Jankowska (1985*a*, *b*) have recently discussed the possible importance of topographic factors stating that the proportions of neurones with input from various fibre systems are similar for different types of neurones in the same area, which may be accounted for by the dense arborizations of primary afferent fibres in laminae V–VI (Brown & Fyffe, 1978, 1979; Hongo, Ishizuka, Mannen & Sasaki, 1978; Ishizuka, Hongo, Mannen & Sasaki, 1979). If this applies to the tract neurones now under study, it would mean that the peripheral information supplied to laminae V–VI interneurones mediating reflex actions (Czarkowska, Jankowska & Sybirska, 1981; Jankowska *et al.* 1981; Brink, Harrison, Jankowska, McCrea & Skoog, 1983) is duplicated and given to the spinocerebellar tract neurones. If so, the tract cells could convey information to the cerebellum concerning inputs from periphery to laminae V–VI interneurones of the segmental pathways nearby. Another possibility may be that the spinocerebellar tract neurones are interposed in segmental reflex chains as are the laminae V–VI interneurones, if they have segmental axon collaterals like the spinocervical tract cells (Brown *et al.* 1977; Rastad, Jankowska & Westman, 1977). In this case the neurones would provide the cerebellum with the same signals that are used in the reflex pathways to motoneurones. One must also consider the possibility that the laminae V–VI interneurones project to tract neurones such as Clarke's column neurones (Hongo *et al.* 1983*b*). The function of the spinocerebellar tract cells now described would in any case be better understood by elucidating their relationship with laminae V–VI interneurones.

We wish to thank Dr E. Jankowska for her valuable comments on the manuscript, and Mrs A. Ohgami and Mrs K. Katagiri for excellent technical and secretarial assistance. The experiment was undertaken in part at the Department of Physiology, Tokyo Medical and Dental University. The study was supported by grants from the Ministry of Education, Science and Culture of Japan.

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.

AOYAMA, M., HONGO, T. & KUDO, N. (1973). An uncrossed ascending tract originating from below Clarke's column and conveying group I impulses from the hindlimb muscles in the cat. *Brain Research* **62**, 237–241.

BRADLEY, K. & ECCLES, J. C. (1953). Analysis of the fast afferent impulses from thigh muscles. *Journal of Physiology* **122**, 462–473.

BRINK, E., HARRISON, P. J., JANKOWSKA, E., MCCREA, D. A. & SKOOG, B. (1983). Post-synaptic potentials in a population of motoneurones following activity of single interneurones in the cat. *Journal of Physiology* **343**, 341–359.

BROWN, A. G. & FYFFE, R. E. G. (1978). Morphology of group Ia afferent fibre collaterals in the spinal cord of the cat. *Journal of Physiology* **274**, 111–127.

BROWN, A. G. & FYFFE, R. E. G. (1979). The morphology of group Ib afferent fibre collaterals in the spinal cord of the cat. *Journal of Physiology* **296**, 215–228.

BROWN, A. G. & FYFFE, R. E. G. (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., 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.

BRYAN, R. N., TREVINO, D. L., COULTER, J. K. & WILLIS, W. D. (1973). Location and somatotopic organization of the cells of origin of the spino-cervical tract. *Experimental Brain Research* **17**, 177–189.

BURKE, R., LUNDBERG, A. & WEIGHT, F. (1971). Spinal border cell origin of the ventral spinocerebellar tract. *Experimental Brain Research* **12**, 283–294.

CZARKOWSKA, J., JANKOWSKA, E. & SYBIRSKA, E. (1981). Common interneurones in reflex pathways from group Ia and Ib afferents of knee flexors and extensors in the cat. *Journal of Physiology* **310**, 367–380.

ECCLES, J. C., ECCLES, R. M. & LUNDBERG, A. (1960). Types of neurone in and around the intermediate nucleus of the lumbosacral cord. *Journal of Physiology* **154**, 89–114.

ECCLES, J. C., FATT, P., LANDGREN, S. & WINSBURY, G. J. (1954). Spinal cord potentials generated by volleys in the large muscle afferents. *Journal of Physiology* **125**, 590–606.

ECCLES, J. C., HUBBARD, J. I. & OSCARSSON, O. (1961*a*). Intracellular recording from cells of the ventral spinocerebellar tract. *Journal of Physiology* **158**, 486–516.

ECCLES, J. C., OSCARSSON, O. & WILLIS, W. D. (1961*b*). Synaptic action of group I and II afferent fibres of muscle on the cells of the dorsal spinocerebellar tract. *Journal of Physiology* **158**, 517–543.

ECCLES, R. M. & LUNDBERG, A. (1959). Synaptic actions in motoneurones by afferents which may evoke the flexor reflex. *Archives italiennes de biologie* **97**, 199–221.

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.

HARRISON, P. J. & JANKOWSKA, E. (1984). An intracellular study of descending and non-cutaneous afferent input to spinocervical tract neurones in the cat. *Journal of Physiology* **356**, 245–261.

HARRISON, P. J. & JANKOWSKA, E. (1985*a*). Sources of input to interneurones mediating group I non-reciprocal inhibition of motoneurones in the cat. *Journal of Physiology* **361**, 379–401.

HARRISON, P. J. & JANKOWSKA, E. (1985*b*). Organization of input to the interneurones mediating group I non-reciprocal inhibition of motoneurones in the cat. *Journal of Physiology* **361**, 403–418.

HOLMQVIST, B., LUNDBERG, A. & OSCARSSON, O. (1956). Functional organization of the dorsal spinocerebellar tract in the cat. V. Further experiments on convergence of excitatory and inhibitory actions. *Acta physiologica scandinavica* **38**, 76–90.

HONGO, T., ISHIZUKA, N., MANNEN, H. & SASAKI, S. (1978). Axonal trajectory of single group Ia and Ib fibers in the cat spinal cord. *Neuroscience Letters* **8**, 321–328.

HONGO, T., JANKOWSKA, E. & LUNDBERG, A. (1966). Convergence of excitatory and inhibitory action on interneurones in the lumbosacral cord. *Experimental Brain Research* **1**, 338–358.

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.

HONGO, T., JANKOWSKA, E. & LUNDBERG, A. (1972). The rubrospinal tract. IV. Effects on interneurones. *Experimental Brain Research* **15**, 54–78.

HONGO, T., JANKOWSKA, E., OHNO, T., SASAKI, S., YAMASHITA, M. & YOSHIDA, K. (1983*a*). Inhibition of dorsal spinocerebellar tract cells by interneurones in upper and lower lumbar segments in the cat. *Journal of Physiology* **342**, 145–159.

HONGO, T., JANKOWSKA, E., OHNO, T., SASAKI, S., YAMASHITA, M. & YOSHIDA, K. (1983*b*). The same interneurones mediate inhibition of dorsal spinocerebellar tract cells and lumbar motoneurones in the cat. *Journal of Physiology* **342**, 161–180.

HONGO, T., OKADA, Y. & SATO, M. (1967). Corticofugal influences on transmission to the dorsal spinocerebellar tract from hindlimb primary afferents. *Experimental Brain Research* **3**, 135–149.

HUBBARD, J. I. & OSCARSSON, O. (1962). Localization of the cell bodies of the ventral spinocerebellar tract in lumbar segments of the cat. *Journal of Comparative Neurology* **118**, 199–204.

ISHIZUKA, N., HONGO, T., MANNEN, H. & SASAKI, S. (1979). Trajectory of group Ia afferent fibers stained with horseradish peroxidase in the lumbosacral spinal cord of the cat: three dimensional reconstructions from serial sections. *Journal of Comparative Neurology* **186**, 189–212.

JANKOWSKA, E., JOHANNISSON, T. & LIPSKI, J. (1981). Common interneurones in reflex pathways from group Ia and Ib afferents of ankle extensors in the cat. *Journal of Physiology* **310**, 381–402.

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

KNOX, C. K., KUBOTA, S. & POPPELE, R. E. (1977). A determination of excitability changes in dorsal spinocerebellar tract neurons from spike-train analysis. *Journal of Neurophysiology* **40**, 626–646.

KUNO, M., MUÑOZ-MARTINEZ, E. J. & RANDIC, M. (1973). Sensory inputs to neurones in Clarke's column from muscle, cutaneous and joint receptors. *Journal of Physiology* **228**, 327–342.

LINDSTRÖM, S. & TAKATA, M. (1972). Monosynaptic excitation of dorsal spinocerebellar tract neurones from low threshold joint afferents. *Acta physiologica scandinavica* **84**, 430–432.

LUNDBERG, A. (1964). Ascending spinal hindlimb pathways in the cat. In *Progress in Brain Research*, ed. ECCLES, J. C. & SCHADE, J. P., vol. 12, pp. 135–163. Amsterdam: Elsevier.

LUNDBERG, A. & OSCARSSON, O. (1960). Functional organization of the dorsal spino-cerebellar tract in the cat. VII. Identification of units by antidromic activation from the cerebellar cortex with recognition of five functional subdivisions. *Acta physiologica scandinavica* **50**, 356–374.

LUNDBERG, A. & OSCARSSON, O. (1961). Three ascending spinal pathways in the dorsal part of the lateral funiculus. *Acta physiologica scandinavica* **51**, 1–16.

LUNDBERG, A. & WEIGHT, F. (1971). Functional organization of connections to the ventral spinocerebellar tract. *Experimental Brain Research* **12**, 295–316.

MANN, M. D. (1971). Axons of dorsal spinocerebellar tract which respond to activity in cutaneous receptors. *Journal of Neurophysiology* **34**, 1035–1050.

MANN, M. D. (1973). Clarke's column and the dorsal spinocerebellar tract: A review. *Brain, Behavior and Evolution* **7**, 34–83.

MATSUSHITA, M., HOSOYA, Y. & IKEDA, M. (1979). Anatomical organization of the spinocerebellar system in the cat, as studied by retrograde transport of horseradish peroxidase. *Journal of Comparative Neurology* **184**, 81–106.

OSCARSSON, O. (1957*a*). Primary afferent collaterals and spinal relays of the dorsal and ventral spinocerebellar tracts. *Acta physiologica scandinavica* **40**, 222–231.

OSCARSSON, O. (1957*b*). Functional organization of the ventral spinocerebellar tract in the cat. II. *Acta physiologica scandinavica* **42**, suppl., 146.

OSCARSSON, O. (1973). Functional organization of spinocerebellar paths. In *Handbook of Sensory Physiology*, ed. IGGO, A., vol. 2, pp. 339–380. Berlin, Heidelberg, New York: Springer.

RASTAD, J., JANKOWSKA, E. & WESTMAN, J. (1977). Arborization of initial axon collaterals of spinocervical tract cells stained intracellularly with horseradish peroxidase. *Brain Research* **135**, 1–10.

REXED, B. (1952). The cytoarchitectonic organization of the spinal cord in the cat. *Journal of Comparative Neurology* **96**, 415–496.

REXED, B. (1954). A cytoarchitectonic atlas of the spinal cord in the cat. *Journal of Comparative Neurology* **100**, 297–379.

TAPPER, D. N., MANN, M. D., BROWN, P. B. & COGDELL, B. (1975). Cells of origin of the cutaneous subdivision of the dorsal spino-cerebellar tract. *Brain Research* **85**, 59–63.

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