Journal of Physiology (1992), **448**, *pp.* 565–578
With 5 figures
Printed in Great Britain

PROJECTIONS OF GROUP II-ACTIVATED MIDLUMBAR SPINOCEREBELLAR TRACT NEURONES TO THE REGION OF NUCLEUS Z IN THE CAT

BY M. ASIF* AND S. A. EDGLEY

From the Department of Anatomy, Downing Street, Cambridge CB2 3DY

(*Received 13 February 1991*)

SUMMARY

1. The possibility that dorsal horn spinocerebellar tract neurones in the midlumbar segments of the cat spinal cord which convey information from hindlimb group II muscle afferents to the cerebellum send collateral projections to medulla in the region of nucleus Z has been examined.

2. Dorsal horn spinocerebellar tract neurones ($n = 25$) were identified by antidromic activation from the cerebellum and by synaptic activation following stimulation of hindlimb group II afferents. A high proportion (21/25) were also antidromically activated by stimuli delivered to the region of nucleus Z.

3. The results of collision tests between antidromic spikes evoked from the cerebellum and the medulla and the fact that the latency for antidromic activation from nucleus Z at threshold was greater than from the cerebellum indicates that at least 11/25 (44%) of the neurones had collateral projections to the medulla.

4. Antidromic threshold mapping revealed that some of the neurones could be activated from parts of the dorsal medulla adjacent to, but not directly overlying, nucleus Z. The possible relevance of these data with regard to sensation of lower limb position and motion is discussed.

INTRODUCTION

The axons of most lower limb muscle proprioceptive afferents do not ascend in the dorsal columns beyond the upper thoracic segments of the spinal cord (Lloyd & McIntyre, 1950; Fern, Harrison & Riddell, 1988). However, there is ample evidence that muscle proprioceptors, in particular primary muscle spindle afferents, can contribute to conscious sensation (see Matthews, 1982). Information from group I muscle afferents does ascend to the cerebellum, via a relay in Clarke's column, the neurones of which project to the cerebellum through the dorsal spinocerebellar tract (DSCT). A pathway via which information from lower limb group I muscle afferents can be conveyed to the somatosensory cortex has been revealed by the demonstration that some Clarke's column cells send an axon collateral projection to the dorsal medulla, to the region of nucleus Z (Landgren & Silfvenius, 1971) or to nucleus X and

* Present address: St John's College, Oxford.

the rostral part of the cuneate nucleus (Johansson & Silfvenius, 1977*c*). In turn relay neurones at these locations project to the thalamus (Landgren & Silfvenius, 1971; Johansson & Silfvenius, 1977*a*, *b*). This pathway conveys information from Golgi tendon organ afferents (McIntyre, Proske & Rawson, 1985) as well as muscle spindle primary afferents. Hence the dorsal spinocerebellar tract (DSCT) makes up part of the pathway through which proprioceptive information from lower limb group I afferents ascends to the cerebral cortex. The situation for group II muscle afferents is not clear-cut. Some Clarke's column cells are co-excited by group Ia and group II afferents (e.g. Eccles, Oscarsson & Willis, 1961), but the group II EPSPs are small in comparison to those evoked by group Ia afferents.

A group of spinocerebellar tract neurones which are powerfully excited by group II afferents but not influenced by group I afferents have recently been described (Edgley & Jankowska, 1988; Edgley & Gallimore, 1988). These neurones differ from Clarke's column neurones in their input and in the location of their somata, which are in laminae IV and V of the dorsal horn. In many other respects they resemble Clarke's column neurones; both are large neurones with fast-conducting axons, both have extensive dendritic trees with similar morphology, the axons of both occupy similar locations in the lateral funiculus and terminate in the same lobules of the cerebellar cortex, after entering through the inferior cerebellar peduncle (Edgley & Gallimore, 1988; Grant & Xu, 1989). The present experiments were designed to test the hypothesis that dorsal horn spinocerebellar tract neurones, like Clarke's column neurones, send collateral projections to the dorsal medulla and therefore contribute to a pathway via which group II afferent information can reach the cerebral cortex.

An abstract of some of this work has been published (Asif & Edgley, 1990).

METHODS

The experiments were performed on six adult cats (2·5–4·5 kg). After induction of anaesthesia with ketamine (28–30 mg kg^{-1} I.M.), deep halothane anaesthesia (1·5–2·5%, in a 25% oxygen and 75% nitrous oxide gas mixture) was induced for surgery. A tracheal cannula was introduced to allow artificial ventilation, two intravenous cannulae were inserted into a femoral and a cephalic vein and one femoral artery was cannulated in order to monitor blood pressure. Nerves of the left hindlimb were dissected free to allow activation of group II afferents. These were the nerves to quadriceps, sartorius, the nerve to the hamstring muscles, the nerves to tibialis anterior and extensor digitorum longus and the nerves to flexors digitorum and hallucis longus (including branches to popliteus, tibialis posterior and the interosseous nerve). The spinal cord was exposed from L1 to the cauda equina and immersed in a paraffin oil pool at 37·5 °C. Small holes were made in the dura to allow access for recording electrodes as required. The spinal cord was fixed using clamps and vertebral hip pins and a stereotaxic headholder was used to fix the skull, which was moderately ventroflexed, to improve access to the dorsal medulla. An occipital craniotomy was performed to expose the posterior lobe of the cerebellum, including on the left-hand side the fissura prima 2–4 mm lateral to the midline.

When surgery was complete the halothane anaesthesia was discontinued and α-chloralose given intravenously to maintain deep anaesthesia (70–80 mg initially, supplemented as required). Before beginning recording the animals were paralysed with gallamine triethiodide(16 mg initial dose, subsequently 8 mg single doses) and artificially ventilated. During periods of paralysis deep anaesthesia was ensured by checking that blood pressure and heart rate were stable and did not alter in response to noxious stimulation. The pupils were also checked for complete constriction at regular intervals.

Neurones projecting to the cerebellum and dorsal medulla were identified by antidromic activation by stimuli delivered through tungsten needle electrodes. Recordings were made using

electrodes filled with 1 or 2 M-potassium citrate, 3–15 MΩ impedance. Dorsal horn spinocerebellar tract neurones were sought in the L4 and the rostral part of the L5 segments, using the large field potentials evoked by group II afferents in the dorsal horn in the region of the dendrites of these neurones (Edgley & Gallimore, 1988) as a guide. At the end of each experiment the electrodes were left in place to provide a histological reference. Signals were recorded on a digital audio tape-recorder for off-line analysis. At the end of the experiments the animals were killed with a large dose of barbiturate.

Determination of the cerebellar and dorsal medulla projections of Clarke's column cells

Before searching for dorsal horn spinocerebellar tract neurones effective stimulation sites for antidromic activation of Clarke's column cells were determined. Many Clarke's column and dorsal horn spinocerebellar tract neurones have projections to both posterior and anterior lobes of the cerebellum (see Edgley & Jankowska, 1988). In view of the proximity of the posterior lobe to the dorsal medulla and the need to push aside the posterior lobe in order to gain access to the dorsal medulla, cerebellar stimulating electrodes were placed in the anterior lobe. Tungsten needle electrodes were introduced into the cerebellum at the level of the fissura prima, with their tips angled rostrally by 25–40 deg. In each experiment the most effective locations for the activation of two to seven Clarke's column neurones were determined by constructing depth–threshold curves. The most effective locations were 9–12 mm deep to the cerebellar surface, in the white matter at the base of lobule IV. Similarly, in each experiment locations on the surface of the dorsal medulla from which at least one Clarke's column neurone could be antidromically activated with a current of 300 μA or less were determined. The dorsal surface of the medulla was exposed after opening the dura by raising and holding aside the posterior lobe of the cerebellum using a small spatula. Stimuli (0·2 ms pulses at rates of 1–1·5 s^{-1}) were delivered via tungsten needle electrodes placed on the medullary surface, for each neurone tested the stimulus was gradually increased from close to zero to a point where the neurone was driven. Precise localization of the electrodes on the medullary surface was achieved by using the obex as a reference point. Initially these were placed 3·0 mm lateral and anterior to the obex (see Landgren & Silfvenius, 1971). When making measurements of stimulus intensity care was taken to keep the dorsal medulla free from cerebrospinal fluid which greatly increased the thresholds of the neurones.

RESULTS

Identification of dorsal horn spinocerebellar tract neurones

Extracellular recordings were obtained from twenty-five group II-activated dorsal horn spinocerebellar tract neurones. All were activated from the cerebellar anterior lobe and the discharges were shown to be antidromic by their ability to follow brief trains of stimuli (300 s^{-1}; Fig. 1) and collision by spontaneous or evoked orthodromic spikes (Fig. 2). The cerebellar stimulating electrodes were fixed so the lowest threshold points in the anterior lobe could not be determined, but previous data indicate that the axons run close to those of Clarke's column cells in the cerebellar white matter (Edgley & Gallimore, 1988). Activation of one neurone by group II muscle afferents from three different hindlimb muscle nerves is illustrated in Fig. 1; stimuli of strengths sufficient to activate group I and group II afferents (4–5 times the threshold (T) of the most excitable fibres) provoked discharges whereas stimuli which activated the majority of group I afferents but few group II afferents ($< 2\ T$) were ineffective.

Evidence for collateral projections to the medulla

The majority (21/25) dorsal horn spinocerebellar tract neurones could also be antidromically activated from the dorsal medulla at sites in the region of nucleus Z. As for the cerebellar-evoked spikes, the spikes appeared at fixed latencies and

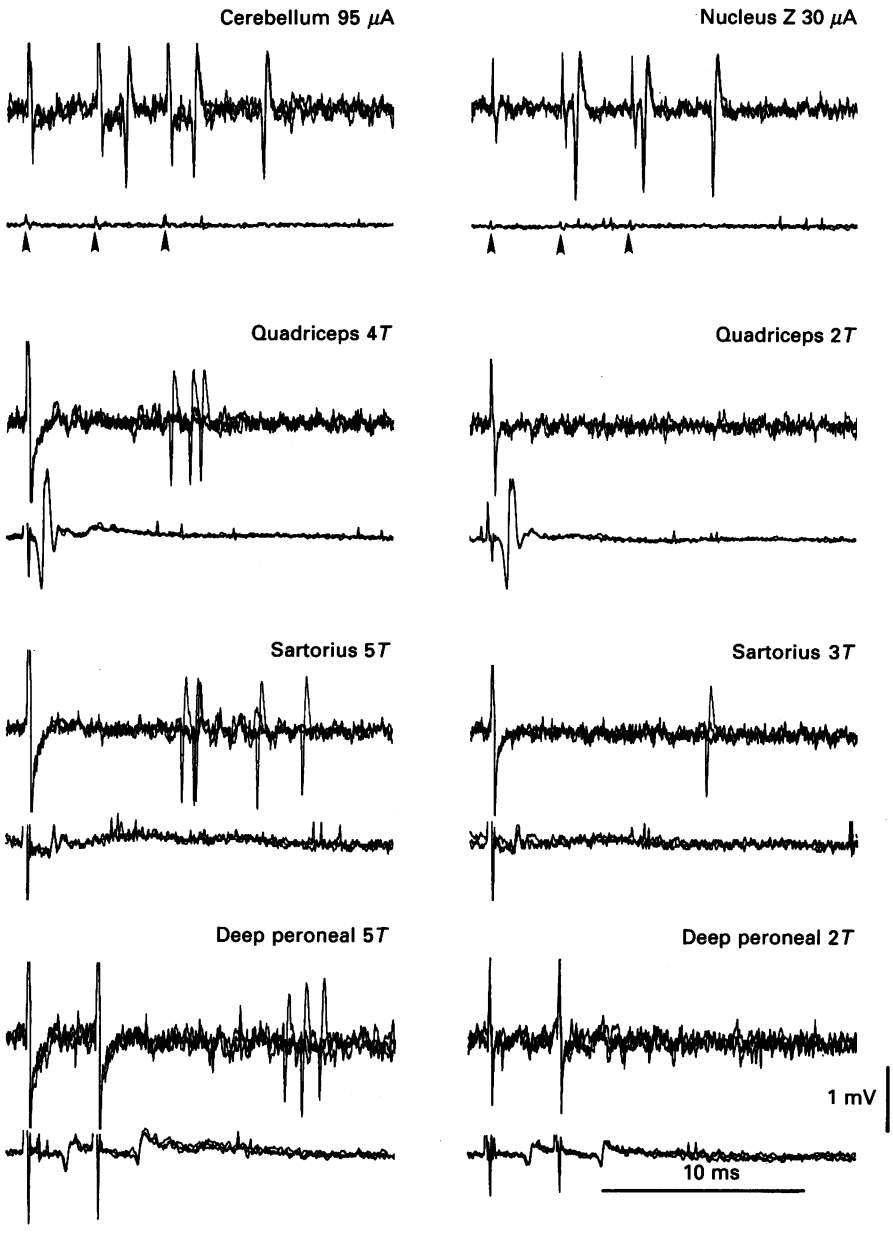

Fig. 1. Extracellular recordings from a dorsal horn spinocerebellar tract neurone. In each trace the upper records show the unit discharges and the lower traces are recordings taken from the dorsal surface of the spinal cord close to the recording site. The top records show antidromic discharges following trains of stimuli (arrow-heads) to the cerebellum and to the region of nucleus Z, at 95 and 30 μA respectively. The lower traces show responses to nerve stimulation, on the left at strengths sufficient to activate group I and group II afferents and on the right at strengths which would activate almost all group I but only a small proportion of group II afferents.

faithfully followed trains of stimuli at 300 s^{-1} (Fig. 1). Collision tests were used to confirm that the spikes were evoked antidromically. Few of the neurones discharged spontaneously, so spikes evoked orthodromically by stimulation of group II muscle afferents were used to collide with the antidromic spikes from the cerebellum or

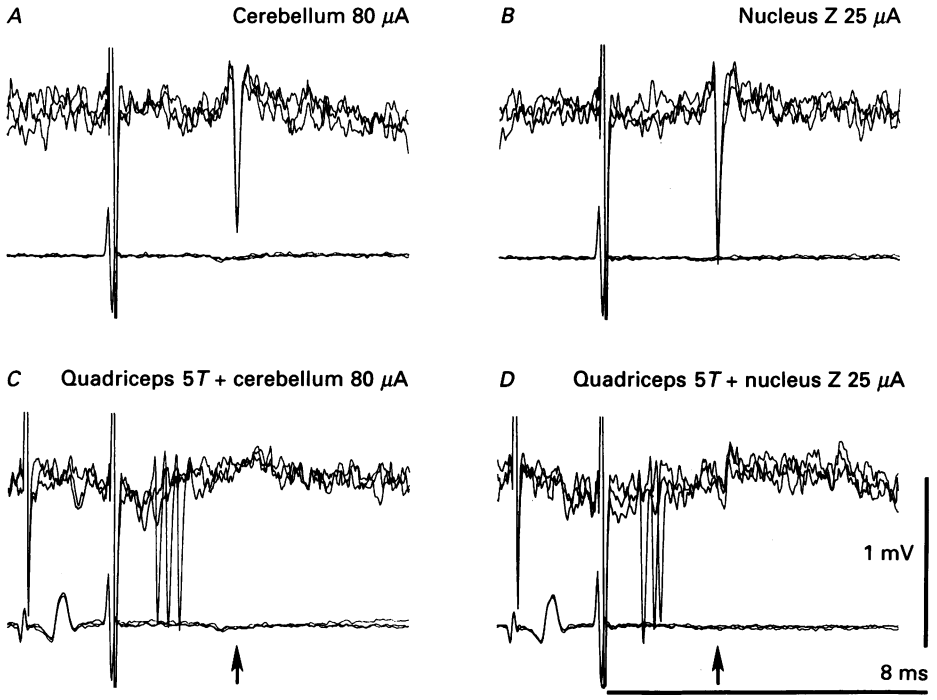

Fig. 2. Extracellular recordings from a neurone showing antidromic activation from the cerebellum and the region of nucleus Z (upper traces) and collision of these spikes with spikes evoked orthodromically by stimulation of quadriceps group II afferents. The arrows indicate the expected times of occurrence of the antidromic spikes.

medulla (Fig. 2). In these tests the orthodromically evoked spikes were timed to occur before the expected time of occurrence of the antidromic spike by an interval greater than the refractory period of the cell. The four cells which could not be antidromically driven from the medulla were recorded in three different experiments and were not driven with the maximum stimuli used (700–1500 μA). Thresholds for antidromic activation of the different cells ranged from 3 to 450 μA. As the DSCT ascends in the medulla it lies only 2–4 mm lateral to nucleus Z so that a major problem with the activation of neurones from the region of nucleus Z is to exclude the possibility of current spread to stem axons in the DSCT. This is unlikely to have occurred in the case of the units with low thresholds for electrical activation (nine units had thresholds of 3–75 μA) but may have been the case where stronger stimuli were used.

The most important evidence for the activation of neurones from collaterals to the region of nucleus Z rather than by current spread to stem axons in the DSCT is that

the latency of antidromic activation from the medulla was greater than or equal to the antidromic latency from the cerebellum in seven neurones, both being measured at threshold. Two of these neurones are illustrated in Fig. 3*A* and *B*. The cerebellar stimulating sites were some distance rostral to the medullary electrodes and therefore the conduction distance from the cerebellum was longer. In these cases the conclusion that the stimulus to the region of nucleus Z excited collateral branches is inescapable. Latencies of antidromic activation from the cerebellum and the medulla are compared in Fig. 3*C*. Since the conduction distance from the midlumbar spinal segments to the cerebellar anterior lobe is longer than the distance from midlumbar segments to the dorsal medulla (by a minimum of 15 mm, assuming a straight-line projection) some difference in the antidromic latencies would be predicted. The expected difference is shown by the dashed line in Fig. 3*C* (see legend for details as to its derivation). In total eleven neurones fall above this line indicating an antidromic latency from the medulla longer than expected for direct activation of the axons in the DSCT.

An obvious demonstration that the antidromic spikes evoked from the region of nucleus Z were due to the activation of collaterals of dorsal horn spinocerebellar tract neurones rather than stem axons would be to collide the cerebellar-evoked spikes with spikes evoked from nucleus Z, or vice versa. This was difficult in these experiments because of the high conduction velocities of the neurones (antidromic latencies were short; 3·0–4·8 ms from the cerebellum; 2·7–4·5 ms from the dorsal medulla) and the short conduction distances. The intervals at which collision should occur were short and likely to be close to the refractory periods of the neurones. Collision of this type was attempted for six neurones where the antidromic latencies from the medulla were shorter than those from the cerebellum, using stimuli fixed at 1·2 or 1·5 times the threshold for antidromic activation. In each case the critical intervals at which spikes evoked by stimuli to the medulla could collide with spikes evoked from the cerebellum, and vice versa, were determined. Estimates of refractory periods at the cerebellar and medullary stimulation sites were obtained by delivering paired stimuli to each site for five neurones; one neurone was lost before a refractory period from the medulla could be determined. Refractory periods obtained in this way ranged from 0·5 to 1·8 ms and were different from the two stimulating sites in four of the five neurones. In collision tests from two branches of an axon the critical interval for collision should equal the conduction time between stimulation sites (antidromically from the first stimulation site to the branch point and orthodromically from the branch point to the second stimulation site), plus the refractory period at the second stimulation site (see Shinoda, Arnold & Asanuma, 1976). In two of the six neurones tested the refractory periods are likely to have been overestimated since they were greater than or equal to the critical intervals for collision between the two sites. This situation could arise if both stimuli activated the axon at the same site, but this was not the case since the antidromic latencies from the two sites were different. The data obtained from collision tests for these two neurones could therefore not be used.

There is evidence for the existence of collaterals in the four remaining cases. If the two stimuli had activated the same DSCT axon at two different points then the critical interval for collision should equal the conduction time between the two sites

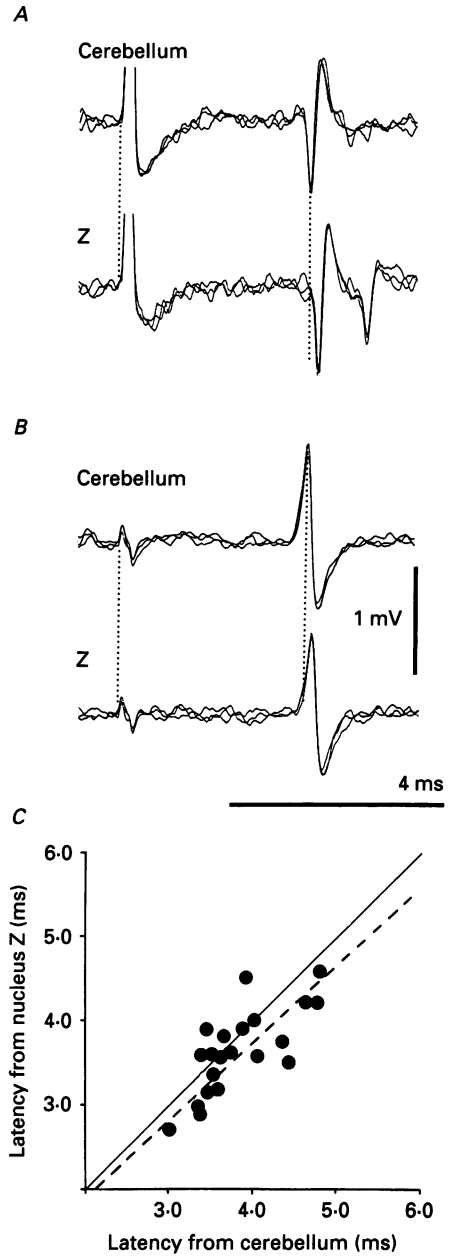

Fig. 3. Comparison of latencies of antidromic activation from the cerebellum and from the region of nucleus Z. A and B show extracellular recordings from two different dorsal horn spinocerebellar tract neurones in which the spikes evoked from the region of nucleus Z had latencies longer than those evoked from the cerebellum, both being measured at threshold. *C* is a plot of latency from nucleus Z against latency from the cerebellum. The diagonal is the line of quality, the dashed line shows the expected additional delay in the responses evoked from the cerebellum assuming a conduction distance of 200 mm from recording site to nucleus Z and 215 mm from recording site to the cerebellar stimulating site.

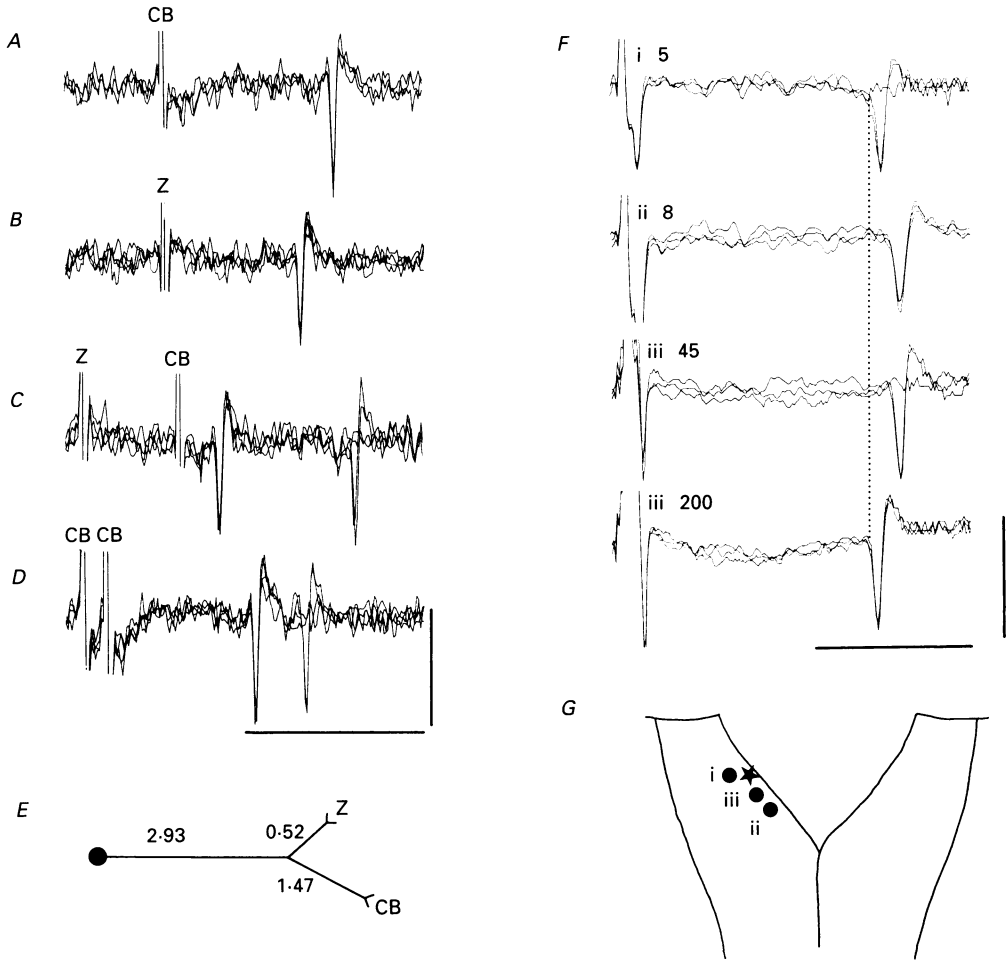

Fig. 4. *A–D* show interaction of antidromic spikes evoked from the cerebellum and from the medulla in the region of nucleus Z. Antidromic activation of a neurone from the cerebellum and the medulla are shown in *A* and *B*, respectively. *C* shows the critical interval following a stimulus to the medulla (Z) at which a cerebellar stimulus (CB) evoked a spike, in this case in two out of four trials. The stimulus separation is 2·6 ms. At 2·5 ms the cerebellar stimulus was ineffective. *D* shows the refractory period at the cerebellar stimulation site (0·6 ms). All stimuli are at 1·5 times the threshold for activation. *E* shows the estimated conduction times, in ms, in the axonal segments of the neurone in *A–D*, derived as described in the text. *F*, antidromic activation of a spinocerebellar tract neurone from three different sites, marked i–iii, on the surface of the medulla. At each point antidromic activation at threshold is shown (stimulus intensities in μA). At site iii stimulation at a stronger intensity is also illustrated. The stimulus sites on the dorsal medulla are shown in *G*, the star marks a reference point 3·0 mm rostral and lateral to the obex. Voltage calibrations are 0·5 mV. Time calibrations are 5 ms in *A–D*, 2 ms in *F*.

(the difference between the antidromic latencies from the two sites) plus the refractory period at the second stimulation site. In all four neurones the critical intervals were greater than these values, implying the presence of an additional

conduction delay in a collateral branch. An example of this test for one neurone is illustrated, in Fig. 4*A–D*. In this case antidromic latencies from the cerebellum (*A*, 4·40 ms) and the medulla (*B*, 3·45 ms) differ by 0·95 ms. The critical interval following a stimulus to the medulla at which a cerebellar stimulus begins to elicit a spike (in about half of the stimulus deliveries) is 2·6 ms and the refractory period at the cerebellar stimulating site is 0·6 ms (Fig. 4*C* and *D* respectively). The critical interval (2·6 ms) is much greater than the difference in antidromic conduction times from the two sites (0·95 ms) plus the cerebellar refractory period (0·6 ms), implying an additional conduction time in a collateral branch. With measures of critical interval, refractory periods at the stimulating sites and antidromic latencies from the two sites it is possible to estimate the conduction time of the different segments of the axon (see Shinoda *et al.* 1976). The conduction delay from the branch point to the terminal in the cerebellum is given by:

$$X = 1/2(I_{zc} + L_c - L_z - RP_c),$$

where X is the conduction time from cerebellum to the branch point, I_{zc} is the critical interval for collision of a spike evoked from the cerebellum by a preceding stimulus to the medulla, L_c is the antidromic latency from the cerebellum, L_z is the antidromic latency from the medulla, and RP_c is the refractory period at the cerebellum.

For the derivation of the equation see Shinoda *et al.* (1976).

Using this equation the values of conduction time in the collateral branches to the region of nucleus Z in the four neurons tested were 0·15, 0·40, 0·41 and 0·52 ms. The conduction times calculated for the different segments of the axon illustrated in Fig. 4*A–D* are shown in Fig. 4*F*.

The refractory periods of these four neurones could have been overestimated, as those of the two rejected neurones might have been. The stimuli were close to threshold and might have been affected by for example changes in current shunting by cerebrospinal fluid. In an attempt to obtain accurate values the refractory periods were measured immediately following the measurement of critical intervals. The possibility that the refractory period at one of the stimulation sites may have been overestimated because it was less than the refractory period at the cell soma, as is the case for some neurones (Swadlow, 1977), was not tested. Without an exact measure of the refractory periods at the stimulation sites, the conduction times between the branch point and the stimulation sites and therefore the location of the branch point cannot be determined with accuracy. However, if the refractory periods at the stimulation sites were overestimated then this would strengthen the conclusion that these neurones have collateral projections to the medulla.

Additional observations provide further evidence for collateral branching in the medulla; the latency of antidromic activation often shortened dramatically as stimulus intensity increased (Fig. 4*F*, site iii). In addition the antidromic latency at threshold was often substantially different with antidromic activation from different sites on the medulla, as in Fig. 4*F* i, ii and iii. These observations imply that the stimuli were activating the neurone at different locations in a region where the axon was branching. Some neurones which had shorter latencies of antidromic activation from the medulla than from the cerebellum and which were not tested for collision may also have had collateral projections to the medulla, in particular four of them were activated with thresholds of 25 μA or less.

Lowest threshold points on the dorsal medulla

Wherever stability permitted, the threshold for antidromic activation of dorsal horn spinocerebellar tract neurones from different locations on the surface of the medulla were determined. Maps of this type (Fig. 5) were constructed for six different

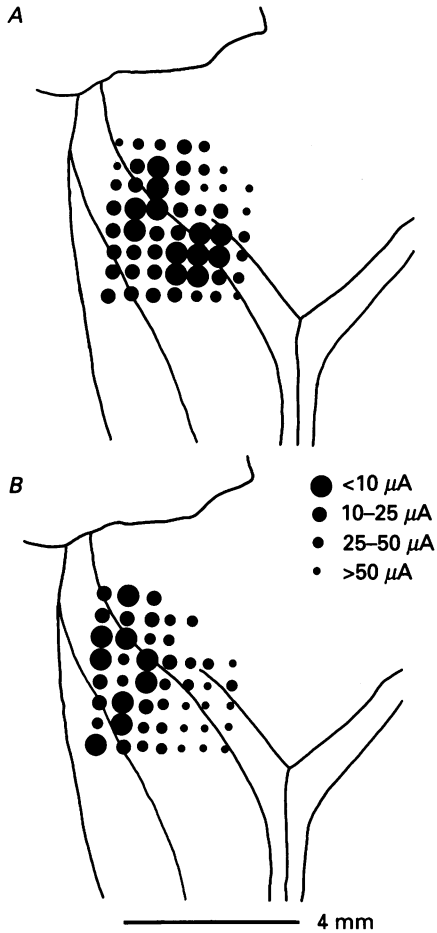

Fig. 5. Antidromic thresholds from different sites on the surface of the medulla for two dorsal horn spinocerebellar tract neurones. *A* and *B* are maps showing antidromic thresholds from different sites on the dorsal surface for two dorsal horn spinocerebellar tract neurones. Filled circles indicate different thresholds for antidromic activation, the larger the circle the lower the threshold, as indicated.

neurones and more limited mapping (6–9 points) was made for two others. The two neurones illustrated in Fig. 5 were both recorded in the same experiment. Very low thresholds were sometimes seen at several locations overlying the rostral gracile and cuneate nuclei, as well as overlying nucleus Z. Two neurones, one of which is illustrated, Fig. 4*A*, had low threshold points over the gracile and cuneate nuclei 1·5–2·5 mm lateral and 1·0–2·0 mm rostral to the obex. Three neurones had low

threshold points caudal to nucleus Z, but further laterally, 3·0–4·0 mm lateral and 0·5–2·0 mm rostral to the obex. One of these neurones is illustrated in Fig. 4*B*. The exposure of the dorsal medulla did not allow thresholds to be determined at points more than 4·5 mm lateral or 4·5 mm rostral to the obex. Antidromic maps of this type were not made for Clarke's column cells in these experiments.

DISCUSSION

Activation of dorsal horn spinocerebellar tract neurones from the medulla

Our data indicate that a high proportion of dorsal horn spinocerebellar tract neurones have projections to the medulla. Seven neurones were activated at the same or longer latency from the medulla than from the cerebellum and must have been activated via collaterals; if the current from the medullary stimuli had spread to the axons in the DSCT then the latencies would always have been shorter than those from the cerebellar anterior lobe. On the basis of the results of collision tests, four additional neurones could be shown to have collateral projections to the medulla. Thus we have strong evidence that eleven of the neurones sampled had collateral projections to the medulla. Other neurones may also have had similar projections. in particular those with low thresholds for activation from the medulla (four had thresholds less than 25 μA, while other neurones recorded in the same experiments had much higher thresholds or were not activated). For other neurones it is difficult to exclude the possibility that the medullary stimuli may have spread to activate DSCT axons directly.

The frequency of projections to the medulla among dorsal horn spinocerebellar tract neurones (at least 11/25 neurones; 44%) is high in comparison to estimates for Clarke's column neurones. In the rat, double-labelling studies (Low, Mantle St John & Tracey, 1986) indicate that only 3% of Clarke's column cells send projections to both the cerebellum and the region of nucleus Z. This was despite the fact that the dorsal spinocerebellar tract provided the great majority of afferent projections to nucleus Z, as also indicated in electrophysiological studies in the cat (see Johansson & Silfvenius, 1977*a*; McIntyre *et al.* 1985). Our impression, gained from recordings made from Clarke's column cells during fixation of the stimulating electrodes in these experiments, is that the incidence of double projections for Clarke's column cells in the cat is likely to be higher, but this has not been studied systematically.

It is difficult to equate the conduction times obtained from the collision tests with distances and thus to estimate the locations of the branch points; in the absence of any terminal slowing of conduction velocity the conduction times would imply branches between 10 and 27 mm caudal to the medulla, but these values are likely to be erroneous.

Targets of dorsal horn spinocerebellar tract neurones in the medulla

Dorsal horn spinocerebellar tract neurones have convergent input from group II afferents from many different muscles (Edgley & Jankowska, 1988), but afferents in certain nerves (quadriceps, sartorius, the deep peroneal) excite the large majority of neurones while others are much less effective. This pattern of input has been taken to imply that dorsal horn spinocerebellar tract neurones signal specific

movements of the limb, in particular full extension (Edgley & Jankowska, 1988). In contrast, Clarke's column cells are powerfully excited by group I afferents of a single, or small group of muscles and the representations of different muscles appear to be equally prominent. Neurones in nucleus Z appear to conform to the pattern seen in Clarke's column (Landgren & Silfvenius, 1971; Johansson & Silfvenius, 1977*a*). Some neurones in nucleus Z which were activated by group I afferents were also activated by group II afferents. As the convergence was almost always from group I and II afferents from the same muscle, it is likely that these actions were mediated by convergence of group I and II afferents on Clarke's column rather than dorsal horn spinocerebellar tract neurones (Eccles *et al.* 1961). Other nucleus Z neurones were activated by cutaneous or joint afferents (Johansson & Silfvenius, 1977*b*), both of which also excite dorsal horn spinocerebellar tract neurones, the former very powerfully (Edgley & Jankowska, 1988). Group II afferent inputs to this class of nucleus Z neurones were not described. Neurones in the region of nucleus Z with group II inputs from the muscles which excite dorsal horn spinocerebellar tract neurones and with an axonal projection to the thalamus have not been reported. Not all of the neurones in the region of nucleus Z project on to the thalamus and Johansson & Silfvenius (1977*b*) have suggested that non-relay cells act as interneurones modulating the activity of neurones which do project on to the thalamus. Low-threshold points caudal to nucleus Z over the rostral parts of the gracile and cuneate nuclei were found in some neurones, as has been described for some Clarke's column neurones (Johansson & Silfvenius, 1977*c*). It is not possible to determine from the current data whether these structures represent targets for dorsal horn spinocerebellar tract neurones or regions through which collaterals pass. The course taken by collaterals from the DSCT to the medulla is unknown in the cat but in the rat the fibres run quite close to the surface of the medulla (Low *et al.* 1986). Some of the low-threshold points on the dorsal medulla could thus represent activation of branches running to the region of nucleus Z. Extensive antidromic mapping would be required to determine which points on an antidromic map represent activation of fibres of passage and which represent activation of terminals.

It is clear that group I muscle receptors provide signals important for the appreciation of limb position and movement (e.g. Matthews, 1982; Burgess, Wei, Clark & Simon, 1982). Since the discharges of secondary muscle spindle afferents are closely related to muscle length (see Matthews, 1981), they seem to be strong candidates to contribute to position sensation. Like group I afferents, group II muscle afferents from the hindlimb do not ascend directly to the brain via the dorsal columns (see Fern *et al.* 1988). There is evidence that ascending axons which signal hindlimb position can be found in the dorsolateral funiculus and that the receptors which provide the positional signal are slowly adapting afferents from muscles (Wei, Simon, Randic & Burgess, 1984). The location of these axons corresponds closely to the location of the axons of dorsal horn spinocerebellar tract neurones. Other ascending axons occupying this location (e.g. spinocervical tract and Clarke's column cell axons) do not have inputs appropriate to a role in signalling limb position. Dorsal horn spinocerebellar tract neurones provide a pathway for group II information to ascend and there is good evidence that spindle secondary afferents contribute to the excitation of the neurones (Edgley & Jankowska, 1988; Harrison, Jami &

Jankowska, 1988). The further connections of nucleus Z (Landgren & Silfvenius, 1971), together with these observations suggest that dorsal horn spinocerebellar tract neurones are components in a pathway which conveys information from hindlimb group II afferents to the cerebral cortex.

We wish to thank the Wellcome Trust for support.

REFERENCES

ASIF, M. & EDGLEY, S. A. (1990). Evidence for a collateral projection from dorsal horn spinocerebellar tract neurones to the region of nucleus Z in the cat. *Journal of Physiology* **430**, 111*P*.

BURGESS, P. R., WEI, J. Y., CLARK, F. J. & SIMON, J. (1982). Signalling of kinaesthetic information by peripheral sensory receptors. *Annual Reviews of Neuroscience* **5**, 171–187.

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

EDGLEY, S. A. & GALLIMORE, C. M. (1988). Morphology and projections of dorsal horn spinocerebellar tract neurones in the cat. *Journal of Physiology* **397**, 99–111.

EDGLEY, S. A. & JANKOWSKA, E. (1988). Information processed by dorsal horn spinocerebellar tract neurones. *Journal of Physiology* **397**, 81–97.

FERN, P., HARRISON, P. J. & RIDDELL, J. (1988). The dorsal column projection of muscle afferent fibres from the cat hindlimb. *Journal of Physiology* **401**, 97–113.

GRANT, G. (1962). Spinal course and somatotopically localised termination of the spinocerebellar tracts. *Acta Physiologica Scandinavica*, suppl. 193.

GRANT, G. & XU, Q. (1988). Routes of entry into the cerebellum of spinocerebellar axons from the lower part of the spinal cord. An experimental anatomical study in the cat. *Experimental Brain Research* **72**, 543–561.

HARRISON, P. J., JAMI, L. & JANKOWSKA, E. (1988). Further evidence for synaptic actions of muscle spindle secondaries in the middle lumbar segments of the cat spinal cord. *Journal of Physiology* **402**, 671–686.

JOHANSSON, H. & SILFVENIUS, H. (1977*a*). Axon–collateral activation by dorsal spinocerebellar tract fibres of group I relay cells of nucleus Z in the cat medulla oblongata. *Journal of Physiology* **265**, 341–369.

JOHANSSON, H. & SILFVENIUS, H. (1977*b*). Input from large ipsilateral proprio- and exteroceptive hind limb afferents to nucleus Z of the cat medulla oblongata. *Journal of Physiology* **265**, 371–393.

JOHANSSON, H. & SILFVENIUS, H. (1977*c*). Connexions from large, ipsilateral hindlimb afferents to the rostral main cuneate nucleus and to the nucleus X region in the cat. *Journal of Physiology* **265**, 395–428.

LANDGREN, S. & SILFVENIUS, H. (1971). Nucleus Z, the medullary relay in the projection path to the cerebral cortex of group I muscle afferents from the cat's hind limb. *Journal of Physiology* **218**, 551–571.

LLOYD, D. P. C. & MCINTYRE, A. (1950). Dorsal column conduction of group I muscle afferent impulses and their relay through Clarke's column. *Journal of Neurophysiology* **13**, 39–54.

LOW, J. S. T., MANTLE ST JOHN, L. A. & TRACEY, D. J. (1986). Nucleus Z in the rat; spinal afferents from collaterals of dorsal spinocerebellar tract neurones. *Journal of Comparative Neurology* **243**, 510–526.

MCINTYRE, A. K., PROSKE, U. & RAWSON, J. A. (1985). Pathway to the cerebral cortex for impulses from tendon organs in the cat's hind limb. *Journal of Physiology* **369**, 115–126.

MATTHEWS, P. B. C. (1981). Muscle spindles: their messages and their fusimotor supply. In *American Handbook of Physiology*, vol. 2, part 2, chap. 6, ed. BROOKS, V. B., pp. 189–228. American Physiological Society, Bethesda, MD, USA.

MATTHEWS, P. B. C. (1982). Where does Sherrington's 'muscular sense' originate? Muscles, joints, corollary discharges? *Annual Review of Neuroscience* **5**, 189–218.

SHINODA, Y., ARNOLD, A. P. & ASANUMA, H. (1976). Spinal branching of corticospinal axons in the cat. *Experimental Brain Research* **26**, 215–234.

SWADLOW, H. A. (1982). Antidromic activation: measuring the refractory period at the site of axonal stimulation. *Experimental Neurology* **75**, 514–519.

WEI, J. Y., SIMON, J., RANDIC, M. & BURGESS, P. R. (1984). Ascending axons that signal the positions of the hindlimbs under static conditions: location and receptor input. *Experimental Brain Research* **54**, 7–22.