J. Physiol. (1983), **337**, *pp.* 257–285
With 8 text-figures
Printed in Great Britain

ORIGIN AND SAGITTAL TERMINATION AREAS OF CEREBRO-CEREBELLAR CLIMBING FIBRE PATHS IN THE CAT

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(*Received 2 July 1982*)

SUMMARY

1. Climbing fibre responses were recorded in the cerebellar anterior lobe on stimulation of the cerebral cortex. A zonal pattern was demonstrated in the cortical projection, which was related to the cerebellar sagittal zones, as identified from peripheral climbing fibre input. In all zones, except c2, a co-variation of the responses evoked on peripheral nerve stimulation and on stimulation of the corresponding part of the sensorimotor cortex was found.

2. There was a bilateral projection to the a, b, c2 and d1 zones which also, to a varying extent, receive a bilateral peripheral input. The x, c1 and c3 zones, receiving an ipsilateral peripheral input, were activated exclusively from the contralateral cortex.

3. Stimulation of the posterior sigmoid gyrus (p.s.g.) evoked responses in all the zones. These responses had, in all zones except d1, lower thresholds and shorter latencies than the responses from other cortical areas.

4. Two separate p.s.g areas were shown to project to the pars intermedia zones (c1, c2, c3 and d1), the lateral area to the caudal parts and the medial area to the rostral parts of the zones. In contrast, the b zone received a projection from only one p.s.g. area, centred between, but overlapping, the two areas projecting to the pars intermedia zones.

5. Stimulation of the anterior sigmoid gyrus evoked short-latency responses in the d1 zone and long-latency responses in all other zones.

6. Stimulation of the first and second somatosensory areas (SI and SII) was generally less effective in evoking climbing fibre responses than was stimulation of the p.s.g. The only exception was the c2 zone, in which responses were evoked from the SII with nearly as low thresholds and short latencies as on p.s.g. stimulation.

7. From the parietal cortex, long-latency responses were regularly evoked in the d1 zone and less frequently in the a, b and c2 zones.

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INTRODUCTION

It is now generally accepted that the inferior olive is the major, if not exclusive, source of climbing fibres to the cerebellum (Szentágothai & Rajkovits, 1959; Eccles, Ito & Szentágothai, 1967; Armstrong, 1974; Desclin, 1974; Batini, Corvisier, Destombes, Gioanni & Everett, 1976). The functional role of the olivo-cerebellar system is, however, still unknown despite many anatomical and physiological studies (for references see Eccles *et al.* 1967; Armstrong, 1974; Brodal & Kawamura, 1980; Ito, 1980; Oscarsson, 1980). It has been proposed that the climbing fibres convey signals about errors in motor performances (Barmack & Hess, 1980; Barmack & Simpson, 1980; Ito, 1980). These signals would induce plastic changes in the cerebellar cortex and result in improved performance (Marr, 1969; Albus, 1971; Ito, 1980). It has been suggested that the errors are detected by the inferior olive acting as a comparator (Miller & Oscarsson, 1970; Oscarsson, 1980); the inferior olive receives information from descending pathways which mediate motor commands and from ascending pathways which monitor the on-going activity in the lower motor centres and the resulting movement. From a comparison of these converging inputs, the inferior olive might detect deviations from the intended movement and inform the cerebellum about these deviations.

The ascending paths, the spino-olivo-cerebellar paths (s.o.c.p.s), have been thoroughly investigated with respect to their functional properties, as well as to their origin and course in the spinal cord and their termination areas in narrow sagittal strips in the cortex of the cerebellar anterior lobe (for references see Ekerot, Larson & Oscarsson, 1979; Armstrong & Schild, 1980; Oscarsson, 1980).

The climbing fibre input to the cerebellum from the cerebral cortex has also been described in several reports (Provini, Redman & Strata, 1968; Miller, Nezlina & Oscarsson, 1969*a*; Allen, Azzena & Ohno, 1974*a*, *b*; Miles & Wiesendanger, 1975*a*, *b*; Sasaki, Oka, Matsuda, Shimono & Mizuno, 1975; Oka, Yasuda, Jinnai & Yoneda, 1976; Oka, Jinnai, & Yamamoto, 1979; Rowe, 1977*a*), as have the responses in the inferior olive to stimulation of the cerebral cortex (Armstrong & Harvey, 1966; Crill & Kennedy, 1967; Crill, 1970). These studies showed a topographically organized convergence of peripheral and cortical climbing fibre inputs to the cerebellum, which is consistent with the comparator hypothesis. However, in these studies, the climbing fibre responses were not localized in relation to the sagittal zones which presumably constitute the functional units of the cerebellar cortex, each controlling a particular motor mechanism (Oscarsson, 1980). Nor were the cortical areas giving rise to the climbing fire responses adequately delineated.

Therefore, the present study was conducted in order to investigate the cortico-olivo-cerebellar projection to each of the climbing fibre zones in the anterior lobe. Particular emphasis was laid on the projection from the pericruciate cortex to the forelimb parts of the zones. A preliminary report has been given (Andersson & Nyquist, 1980).

METHODS

The experiments were performed on thirty adult cats (2·0–3·5 kg), twenty-one under pentobarbitone (iNitial dose 40 mg/kg intraperitoneally) and nine under chloralose anaesthesia (initial dose 80 mg/kg intravenously). All animals were given supplementary doses (2–5 mg/kg) of pentobarbitone as required. The animals were paralysed with gallamine triethiodide and artificially ventilated. The arterial blood pressure, end-expiratory CO_2 concentration and rectal temperature were continuously monitored and kept within physiological limits. The anaesthesia was maintained at such a level that the size of the pupils was always minimal.

The sciatic and ulnar nerves were dissected bilaterally and mounted for stimulation. In some experiments, the superficial and deep radial nerves were also stimulated. The infraorbital branches of the trigeminal nerves (henceforward referred to as trigeminal nerves) were stimulated bilaterally with needle electrodes inserted through the gingiva (Andersson & Eriksson, 1981). All nerves were stimulated at 20 times nerve threshold.

Craniotomies were performed to expose the cerebellar anterior lobe on the left side and the cerebral pericruciate cortex bilaterally. In many cases, the cerebral exposure was extended to include other cortical areas such as the parietal cortex and the first and second somatosensory areas. Photographs were taken of the cerebellar and cerebral surfaces and the exposed parts were then covered with warm mineral oil to prevent drying. Climbing fibre responses were recorded, with silver ball electrodes, as positive field potentials at the surface. In some experiments, the activity of single Purkinje cells was recorded with glass micropipettes filled with a 3 M-KCl solution and having a resistance of 3–6 MΩ.

The cerebral cortex was stimulated either at the surface with mono-polar silver ball electrodes or at a depth of 2 mm with a needle electrode, insulated except for the tip and having a resistance of 10–20 kΩ. The cortex was stimulated cathodally with 1–5 square pulses of 200 μs duration using a constant current stimulator. The train frequency was 300–400 Hz and the stimuli were applied 1–2 times per s. The maximal stimulus intensities employed were 3 mA for surface stimulation and, with the exception of the experiment illustrated in Fig. 1, 1·5 mA for intracortical stimulation. Prior to cortical stimulation, the responses on peripheral nerve stimulation were recorded at the stimulation site on the surface of the cerebral cortex.

RESULTS

(*1*) *Functional properties of cortico-cerebellar pathways*

The cortico-olivo-cerebellar projection was studied by recording climbing fibre responses evoked in the cerebellum on stimulation of the cerebral cortex. Fig. 1*A* shows records of responses recorded, under pentobarbitone anaesthesia, from the cerebellar surface on stimulation of the contralateral cerebral cortex with different stimulus intensities and number of shocks. The cortex was stimulated cathodally with a mono-polar needle electrode at a depth of 2 mm. The evoked responses consisted of two components: a short-latency (3 ms), slowly rising positivity due to synaptic activation of granule cells by a mossy fibre input and a second, steeply rising positivity at 13–16 ms due to a climbing fibre activation of Purkinje cells (Eccles *et al.* 1967; Eccles, Provini, Strata & Táboříkova 1968; Armstrong & Harvey, 1968; Provini *et al.* 1968; Allen, Azzena & Ohno, 1972; Allen *et al.* 1974*a*).

In most preparations under barbiturate anaesthesia, the mossy fibre responses were considerably smaller than illustrated here. Under chloralose anaesthesia, they were large and almost concealed the climbing fibre responses. Since the aim of the present study was to investigate the latter, small amounts of pentobarbitone (2–5 mg/kg at intervals of 1–2 h) were given in these cases in order to depress the mossy fibre responses. Under such circumstances, it has been proposed that the amplitude of the climbing fibre responses is increased (Gordon, Rubia & Strata, 1973; Allen, Azzena

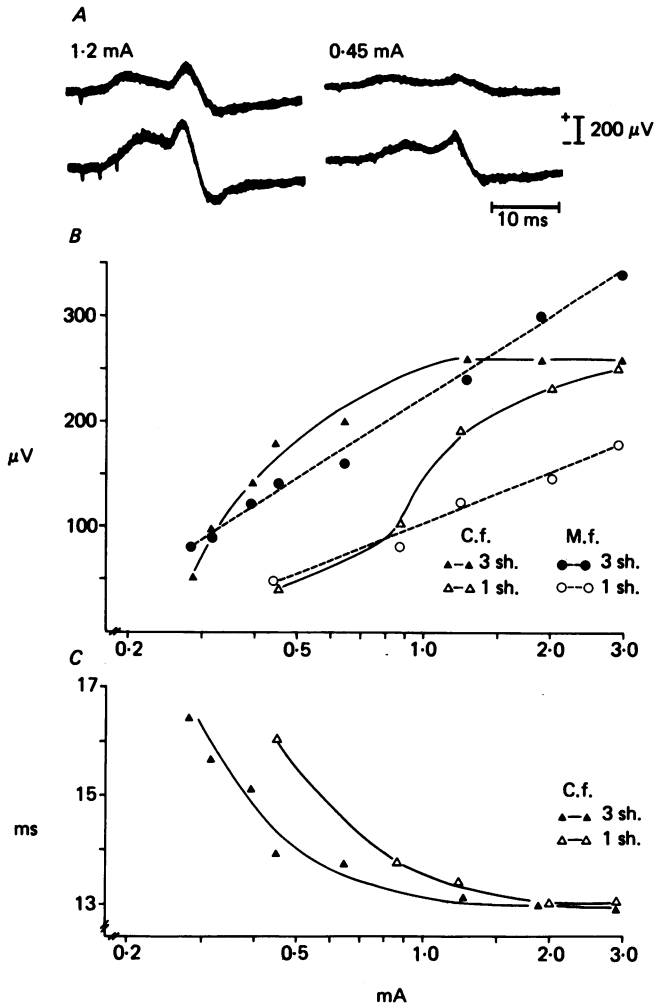

Fig. 1. Characteristics of mossy and climbing fibre responses to cerebral intracortical stimulation. *A*, responses recorded at the cerebellar surface to stimulation of the contralateral posterior sigmoid gyrus with an intracortical needle electrode. A single shock (sh.) and a train of three shocks were used with varying intensities. Positivity upwards, as in all Figures. *B*, relation between stimulus strength and the amplitudes of climbing fibre (c.f.) and mossy fibre (m.f.) responses. *C*, relation between stimulus strength and climbing fibre response latencies. Pentobarbitone anaesthesia.

& Ohno, 1979). Additionally, pentobarbitone stabilized the climbing fibre respons which fluctuate markedly under chloralose anaesthesia (Oscarsson & Sjölund, 197

With a high stimulus strength (e.g. 1·2 mA), the climbing fibre response stimulation of the cortex with one or three shocks was essentially the same (Fig. 1*A* In some cases, as in the example illustrated in Fig. 1, the climbing fibre respons evoked with two shocks were similar to those evoked with three shocks. In other cas stimulation with three shocks evoked climbing fibre responses with slightly low thresholds and larger amplitudes than those evoked with two shocks. An increase the number of shocks above three did not reduce the threshold further nor incres the amplitudes. Therefore, stimulation with three shocks was regularly employe

When using lower stimulus intensities (e.g. 0·45 mA; Fig. 1*A*), there was a clear difference between responses evoked with one and with three shocks. This is further illustrated in Fig. 1*B* and *C*. In *B*, the mossy fibre and climbing fibre response amplitudes are plotted against the stimulation strengths. The climbing fibre responses evoked with a single shock were much smaller than those evoked with three shocks at low stimulation strengths. At higher stimulus intensities, the amplitudes did not grow further when the stimulus strength or the number of shocks was increased, implying a maximal activation of the cortico-olivo-cerebellar pathway. In contrast, the amplitudes of the mossy fibre responses (dashed lines) were increased both by increasing the stimulus strength and the number of stimuli at all strengths tested.

The latencies of the cortically evoked climbing fibre responses depended, to a large extent, on the stimulus intensities used (Fig. 1*C*). When the stimulus intensity was increased from threshold, there was a gradual decrease of the latencies, by several milliseconds, to a plateau level, in this case 13 ms. Thereafter, further increase of the stimulation strength resulted in no, or only a very slight, decrease of the response latency. At low strengths, the response latency to a single shock was longer than that to double or triple shock stimulation.

In this study, the response latencies used for analysis (cf. Fig. 3) were measured from the first shock at stimulation strengths where a moderate increase in strength did not reduce the latency further, i.e. when the synaptic delays in the cortico-olivo-cerebellar pathways had become minimal. Usually, this plateau on the latency curve was reached between 0·5 and 1·0 mA with intracortical stimulation and between 1·0 and 2·5 mA with surface stimulation, if the stimulus electrode was optimally located.

(*2*) *Identification of cortico-olivo-cerebellar projections*

The mapping of the climbing fibre input to the cerebellar anterior lobe from the cerebral cortex was achieved using two different strategies: (a), ten to twenty stationary stimulus electrodes were placed at fixed sites on the cerebral surface, which were identified by the surface configuration (sulci and gyri) and the peripheral input (e.g. maximal responses in the first somatosensory area). A recording electrode was moved in small steps (0·3–1 mm) along the surface of a cerebellar folium. Peripheral nerves were stimulated in order to identify the cerebellar sagittal zones (Oscarsson, 1973; Oscarsson & Sjölund, 1977; Ekerot & Larson, 1979*a*), and then the responses elicited from different cortical stimulation sites were studied. The amplitudes of the responses from peripheral nerves and cortical stimulation sites were plotted along the folium (see Fig. 2*A*), and the cortical input to each zone was specified. (b), Stationary recording electrodes were placed on the cerebellar surface. The recording sites were chosen so that each electrode recorded maximal responses evoked from the periphery in each of the studied zones. Either a silver ball electrode for surface stimulation or a needle electrode for intracortical stimulation was moved in small steps along the cerebral cortex. The responses evoked from peripheral nerves were recorded at each cortical stimulation site prior to stimulation of that site. Based on the results from this procedure, a map of the cerebral cortical area(s) projecting to a certain zone was constructed (see Fig. 5).

The first strategy permitted an analysis of the zonal organization in the projection from any stimulation site. However, it was not possible to determine the borders of

the cerebral cortical areas projecting to the cerebellum, nor to locate the minimum threshold sites. When a response was evoked from a certain site only with a rather high stimulus intensity, it was impossible to know whether the response was due to stimulus spread to an adjacent cortical area or to activation of a cortico-olivo-cerebellar pathway originating at the site, but requiring much spatial summation.

With the second strategy, it was possible to delineate the cerebral projecting areas as well as to identify the sites from where responses with minimal thresholds and latencies and maximal amplitudes were evoked. On the other hand, responses could be recorded from only a few sites in each cerebellar zone.

In most experiments, the recording session was begun with the first strategy and, when it had been determined in which zones the most favourable recordings could be made in that particular experiment, the second strategy was employed.

Fig. 2 shows an experiment in which the caudal folium of lobule Vc was mapped with a surface recording electrode. Fig. 2*B* is a map of the left cerebellar anterior lobe. The points and stars along the folium indicate the recording sites and the borders between the sagittal zones are indicated with dotted lines. Fig. 2*C* shows the cortical stimulation sites and the responses evoked at these sites on stimulation of the ulnar (upper traces) and sciatic (lower traces) nerves contralateral to the cortical sites.

In Fig. 2*A*, the amplitudes of the climbing fibre responses recorded from lobule Vc are plotted and the latencies indicated. The cerebral cortex was stimulated at a strength of 1·5 mA with surface electrodes. To the right are records from the sites marked with stars. Responses in successively more lateral zones, identified from their peripheral inputs, will be described.

The *a zone* in the medial vermis receives a long-latency hind-limb input which is often bilateral (Oscarsson & Sjölund, 1977). In the experiment illustrated in Fig. 2, no, or only small, responses were evoked in the lateral part of the zone on cerebral cortical stimulation. More medially, responses were evoked from nearly all stimulation sites. In fact, when the stimulation strength was 2 mA, responses were evoked from all ten stimulation sites.

The *x zone*, lateral to a, receives information from the ipsilateral forelimb and the

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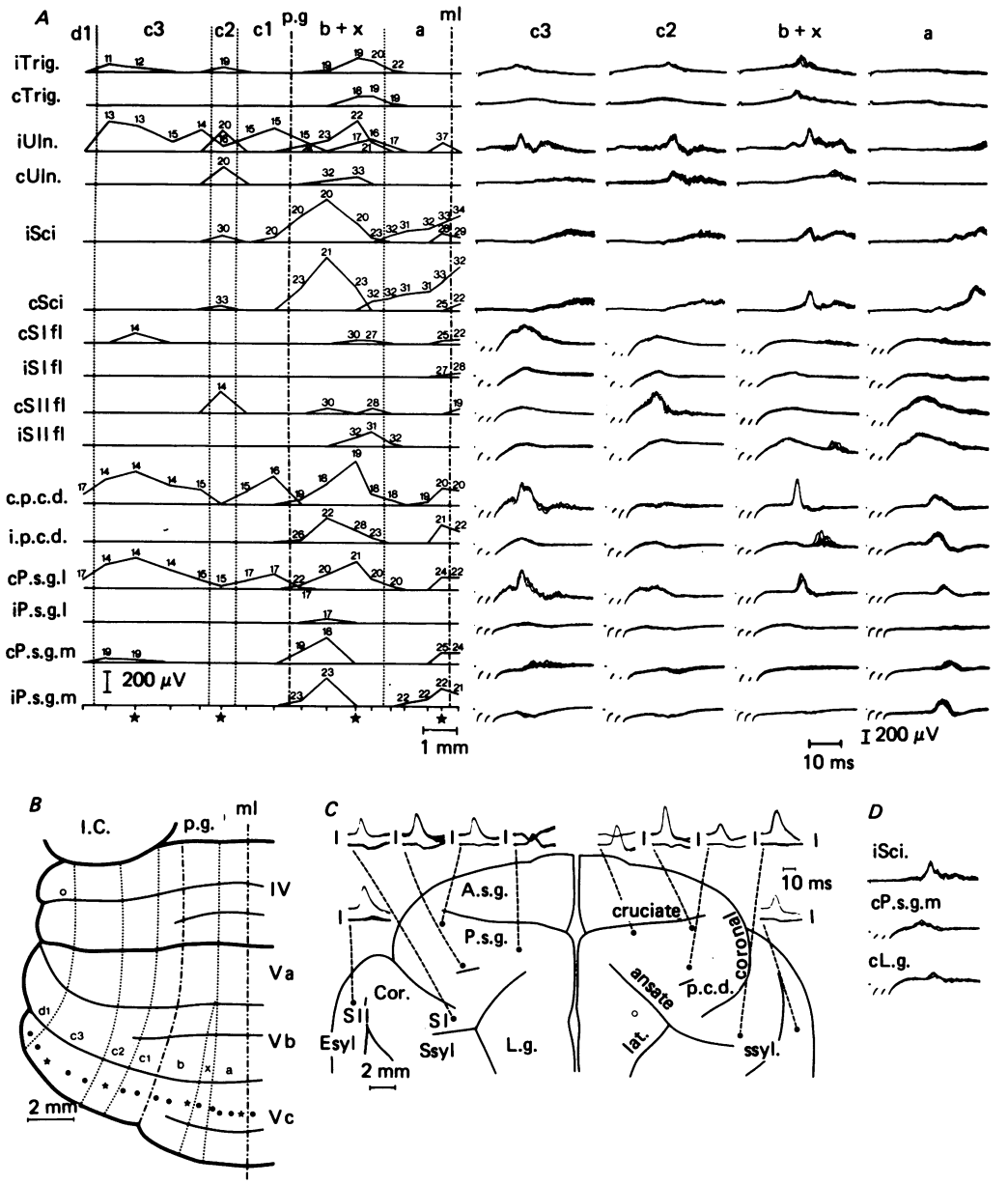
Fig. 2. Distribution of climbing fibre responses elicited in one folium on stimulation of peripheral nerves and cerebral cortex. *A*, distribution of climbing fibre responses along one folium in the cerebellum. Response latencies indicated. To the right, records of responses in four of the zones, recorded at the sites indicated with stars to the left and in *B*. *B*, left cerebellar anterior lobe, lobules IV–Vc (nomenclature of Larsell, 1953). Dots and stars indicate recording sites. *C*, cerebral cortex and stimulation sites (viewed from dorsal direction). At each site, records of responses evoked on contralateral ulnar nerve (upper traces) and sciatic nerve (lower traces) stimulation are shown. Vertical bars 200 μV. Open circle indicates stimulation site in the contralateral lateral gyrus. Names of gyri indicated to the left and names of sulci to the right, according to Hassler & Muhs-Clement (1964). *D*, records of responses evoked in the d1 zone in lobule IV (indicated with a ring in B). Abbreviations: i, ipsilateral; c, contralateral; Trig., trigeminal; Uln., ulnar; Sci., sciatic nerves; SIfl and SIIfl, forelimb areas of first and second somatosensory areas; p.c.d. post-cruciate dimple; P.s.g., posterior sigmoid gyrus; l lateral; m medial; A.s.g., anterior sigmoid gyrus; Cor, coronal gyrus; Esyl, ectosylvian gyrus; Ssyl, suprasylvian gyrus; L.g. lateral gyrus; lat, lateral sulcus; ssyl, syprasylvian sulcus; p.g., paravermal groove; ml, midline; i.c., inferior colliculus. Cerebellar zones according to Oscarsson (1980). Chloralose anaesthesia.



short-latency response (16–17 ms) on ipsilateral ulnar nerve stimulation in the medial part of the b+x division in Fig. 2*A* can be ascribed to the activation of Purkinje cells in the x zone (Ekerot & Larson, 1979*a*; Andersson & Eriksson, 1981). When the x zone was as narrow as in Fig. 2*A*, it was impossible to separate it spatially from the medial part of the b zone when the responses were recorded only as surface potentials (Andersson & Eriksson, 1981).

The *b zone* in the lateral vermis receives a bilateral input from the periphery. Caudal body segments project to the lateral part, and more rostral segments project to successively more medial parts (micro-zones) of the zone (Oscarsson & Sjölund, 1977; Andersson & Oscarsson, 1978; Andersson & Eriksson, 1981). In the experiment illustrated in Fig. 2, responses were evoked in the lateral part of the b zone from the medial posterior sigmoid gyrus (p.s.g.) electrodes, bilaterally. Overlapping these responses but shifted slightly medially, responses were recorded on stimulation of the forelimb parts of both posterior sygmoid gyri (contralateral and ipsilateral p.s.g.l. and post-cruciate dimple (p.c.d.)). Due to the difficulty in separating the responses in the medial b zone and the x zone, it was not possible to ascribe the responses from the SI and SII specifically to one of these zones. However, when the stimulation strength was increased to 2·5 mA, more laterally located responses of considerably larger amplitudes, with latencies of 22–25 ms, were evoked bilaterally from the SI and SII electrodes. This suggests that the responses were evoked in the b zone.

The *c1 zone* in the medial pars intermedia receives, in lobule V c, ipsilateral forelimb input (Ekerot & Larson, 1979*a*). In the illustrated experiment (Fig. 2*A*), responses were evoked on cortical stimulation only from the forelimb part of the contralateral p.s.g.

The *c2 zone* receives a long-latency input from all four limbs without a distinct somatotopy (Larson, Miller & Oscarsson, 1969*b*; Ekerot & Larson, 1979*a*). In addition, in the present investigation, responses were recorded on ipsilateral and, usually also, contralateral trigeminal nerve stimulation (latencies 15–22 and 18–28 ms, respectively). In the illustrated experiment (Fig. 2*A*), the forelimb site in the contralateral second somatosensory area (cS II fl) was the only cortical site from which a climbing fibre response could be evoked in the c2 zone.

The *c3 zone* receives, in lobule V, a short-latency climbing fibre input mainly from the ipsilateral forelimb (Ekerot & Larson, 1979*a*, *b*). In the present investigation, a short-latency response (9–12 ms) in a lateral strip in the c3 zone (in lobule V c) was also frequently observed on stimulation of the ipsilateral trigeminal nerve. In the experiment of Fig. 2, short-latency responses (14 ms) were evoked mainly from the forelimb parts of the contralateral first somatosensory area (cS I fl) and posterior sigmoid gyrus (cp.s.g.l and cp.c.d.).

The *d1 zone* receives ipsilateral forelimb input in lobule V and ipsilateral hind-limb input in lobule IV and, to some extent, in lobule V (Larson, Miller & Oscarsson, 1969*a*; Ekerot & Larson, 1979*a*). In the present study, responses to contralateral limb nerve stimulation were also occasionally evoked (latencies 21–24 ms). In the experiment illustrated in Fig. 2, the d1 zone was very narrow in lobule V c and no, or only very small, responses were recorded there. Recordings were also made from a folium in lobule IV in this experiment. Here the d1 zone was wider, and at the site indicated with an open circle in Fig. 2*B*, a large response from the ipsilateral sciatic nerve was evoked. In 2*D*, records are shown of responses evoked at this site. Of the cortical stimulation sites in *C*, only the medial one on the contralateral p.s.g. (cp.s.g.m) was effective in evoking a response (latency 15 ms). In addition, stimulation of the rostral part of the lateral gyrus (cl.g.), indicated with a circle in 2*C*, evoked a response at a latency of 19 ms. Stimulation of this site evoked no responses in lobule V c.

The results presented in Fig. 2 demonstrate that climbing fibre responses can be

evoked from several cerebral-cortical areas. The histograms in Fig. 3 show the response latencies to stimulation of the different areas in the contralateral and ipsilateral cortex, displayed above and below the abscissa, respectively. The values were obtained by selecting, in each experiment, the shortest response latency in each zone. The characteristics of the projections from each cortical area will be briefly described.

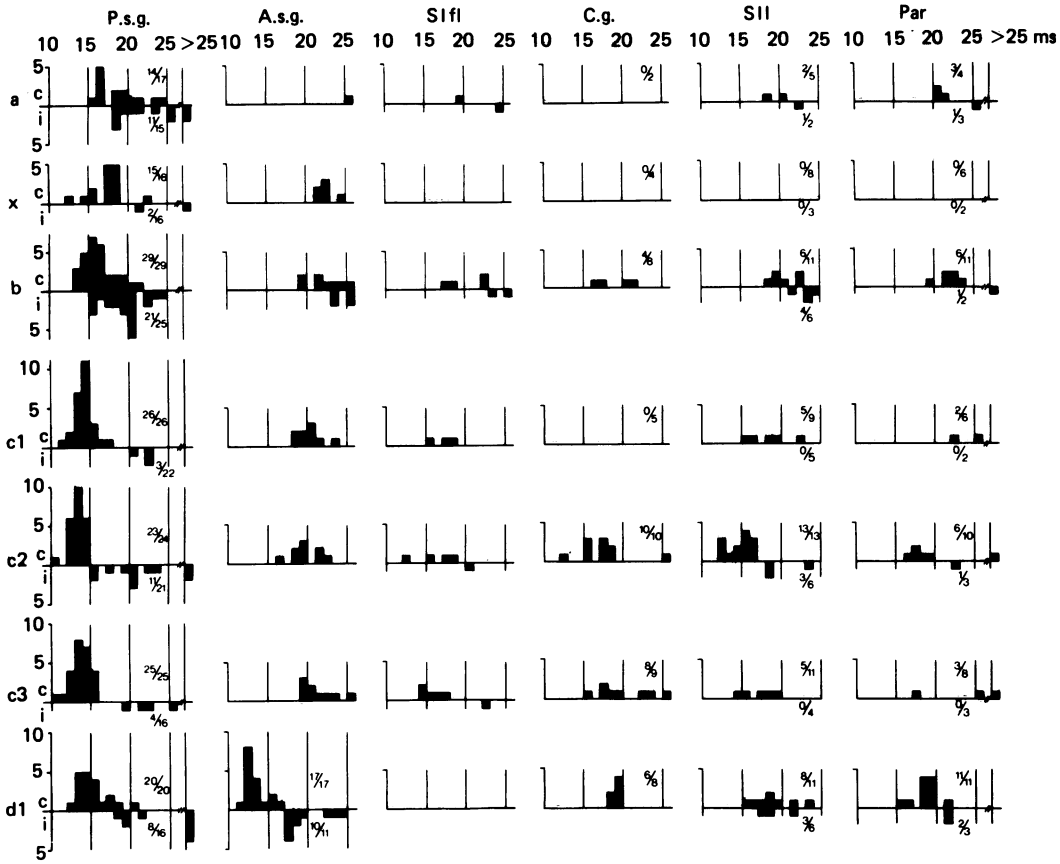


Fig. 3. Latencies of climbing fibre responses to stimulation of cerebral cortex. Contralateral (c) response latencies above and ipsilateral (i) below the abscissa. In each experiment, the shortest latency was selected for each zone. Numbers indicate in how many experiments responses were evoked in a certain zone out of the number of experiments tested for that particular projection. Abbreviations: P.s.g., posterior sigmoid gyrus; A.s.g., anterior sigmoid gyrus; SI,fl, forelimb area of first somatosensory area; C.g., coronal gyrus; SII, second somatosensory area; Par, parietal cortex.

Stimulation of the p.s.g. evoked responses in all zones in nearly all experiments. In all zones, except d1, these responses had lower thresholds, larger amplitudes and shorter latencies than the responses evoked from other cortical areas.

There was a topographical organization in the projection from the p.s.g. Stimulation of the lateral part evoked responses in the x, medial part of the b and caudal parts of the pars intermedia zones. In each zone except c2, these responses showed the same distribution pattern as that found on stimulation of large forelimb nerves (cf. Ekerot

& Larson, 1979*a*). Correspondingly, the medial part of the p.s.g. projected to the a, the lateral part of the b and rostral parts of the pars intermedia zones, which are the main hind-limb receiving areas in the cerebellar anterior lobe (see below, section 3).

Stimulation of the ipsilateral p.s.g. was almost as effective as stimulation of the contralateral p.s.g. in evoking responses in the a and b zones. Responses were less frequently seen in the c2 and d1 zones. In the zones receiving only ipsilateral peripheral input, small responses were observed only occasionally on stimulation of the ipsilateral p.s.g.

From a large area in the anterior sigmoid gyrus (a.s.g.), short-latency responses (12–13 ms) were evoked exclusively in the d1 zone (Fig. 3). These responses had as low, or lower, thresholds and as large amplitudes as those evoked from the p.s.g. Responses were also evoked in the d1 zone on ipsilateral a.s.g. stimulation. These had approximately 5 ms longer latencies and 2–3 times higher thresholds than the contralaterally evoked responses.

In the other zones, responses were evoked only from the lateral part of the a.s.g. These responses had considerably longer latencies than those evoked from the p.s.g. (Fig. 3). At least in the b zone (the only zone tested), these long-latency responses were evoked also from the ipsilateral a.s.g.

The first somatosensory area (SI) was stimulated with a fixed stimulus electrode where maximal surface positive responses were evoked on forelimb nerve stimulation (Fig. 2). The climbing fibre responses evoked from SI occurred exclusively in the forelimb parts of the zones. The responses had higher thresholds and longer latencies than those evoked from the p.s.g. (Fig. 3). In three experiments, intracortical stimulation was employed in the parts of SI with maximal responses on stimulation of the ulnar and superficial radial nerves. Climbing fibre responses were only evoked with stimulus strengths which were considerably higher than those required in the p.s.g.

When fixed stimulus electrodes were used, it was not always possible to determine if the responses from the SI electrode were due to activation of the SI only, or to stimulus spread into the p.s.g. area, particularly in those cases where the distance between the SI and p.s.g. area was short and the threshold for evoking a response from the SI was considerably higher than that from the p.s.g. area. Therefore, only the latencies of those responses that could be clearly ascribed to activation of the SI, i.e. if they had only slightly higher thresholds than the responses from the p.s.g., are included in Fig. 3 and only positive findings are considered.

On stimulation of the coronal gyrus at the site of maximal responses evoked on trigeminal nerve stimulation, climbing fibre responses were evoked in the b, c2, c3 and d1 zones. The latencies were usually 15–20 ms (Fig. 3). The responses were evoked in those parts of the zones receiving a trigeminal input. The occasional absence of responses in the b and c3 zones, indicated in the latency histograms in Fig. 3, reflects the inclusion of experiments in which recordings were made only from 'non-trigeminal' parts of the zones.

The d1 zone has not previously been reported to receive a trigeminal input. In the present study, climbing fibre responses were evoked in the d1 zone on stimulation of the ipsilateral trigeminal nerve in five (of twenty-five tested) cats (latencies 17–24 ms).

The second somatosensory area (SII), in the anterior ectosylvian gyrus (see Jones & Powell, 1973) was stimulated in thirteen cats. Climbing fibre responses were regularly evoked in the c2 zone and less frequently in the other zones (Figs. 2 and

3). In five experiments, the SII was systematically mapped with an intracortical stimulus electrode. The minimal thresholds for evoking responses in the c2 zone were nearly as low as those for evoking responses from the p.s.g. To evoke responses in the other zones from the SII, considerably higher strengths were required.

A topographical organization was found such that the anterior, forelimb part of the SII projected to the medial part of the b zone and the caudal parts of the pars intermedia zones, while the caudal, hind-limb area projected to the lateral part of the b zone and the rostral parts of the pars intermedia zones. There was, however, a considerable overlap in the projecting areas. The minimum threshold sites were usually found in that part of the SII which received convergent fore- and hind-limb inputs.

In six cats, the ipsilateral SII was also stimulated. In the two pentobarbitone anaesthetized cats, no climbing fibre responses were evoked. In the four cats under chloralose anaesthesia, climbing fibre responses were observed in the b, c2 and d1 zones (Fig. 3).

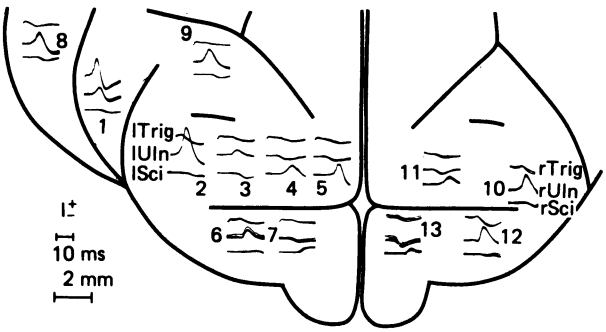
On stimulation of the parietal cortex (anterior parts of the lateral and suprasylvian gyri), responses were evoked in the d1 zone in all cases tested (Figs. 2 and 3). In the other zones, responses were less frequently observed. The effective area extended caudally from the ansate sulcus for a distance of approximately 3 mm. The thresholds for evoking responses in the d1 zone were usually as low as those for evoking responses from the p.s.g. and a.s.g. To evoke responses in the other zones, higher stimulus strengths were usually required (with the exception of the c2 zone in chloralose anaesthetized cats).

There was a topographical organization in the projection from the parietal cortex to the d1 zone such that stimulation of the suprasylvian gyrus tended to evoke larger responses in the caudal (forelimb) part of the d1 zone and stimulation of the lateral gyrus evoked larger responses in the rostral (hind limb) part of the zone.

The results presented above demonstrate a projection from several cortical areas. Stimulation of different cortical areas often evoked responses at the same cerebellar recording site. In order to test if there was a convergence of the paths from several cortical areas also at a unitary level, recordings from single Purkinje cells were obtained (cf. Rowe, 1977*a*). Fig. 4 shows intracellular records of climbing fibre excitatory post-synaptic potentials from three Purkinje cells encountered in successively more lateral micro-zones (Andersson & Oscarsson, 1978; Andersson & Eriksson, 1981) in the b zone in one experiment. The map of the cerebral cortex (viewed from a rostral direction) shows the positions of the stimulus electrodes and the responses evoked at each of these sites on stimulation of the trigeminal (upper trace), ulnar (middle trace) and sciatic (lower trace) nerves contralateral to the respective sites.

The three cells were identified as trigeminal (A), forelimb (B) and hind-limb (C) cells from their dominant peripheral inputs. The cells were activated on bilateral nerve stimulation, but only records of the ipsilateral nerve responses are shown. The trigeminal cell (A) was activated with the lowest threshold (0·6 mA) and shortest latency (18 ms) from the contralateral coronal gyrus (position 1). The cell was also activated from the lateral parts of the p.s.g. on both sides but with higher thresholds and longer latencies (note that the cell was also activated from the limb nerves but with long latencies).

The forelimb cell (B) was activated from most of the sites. The shortest latency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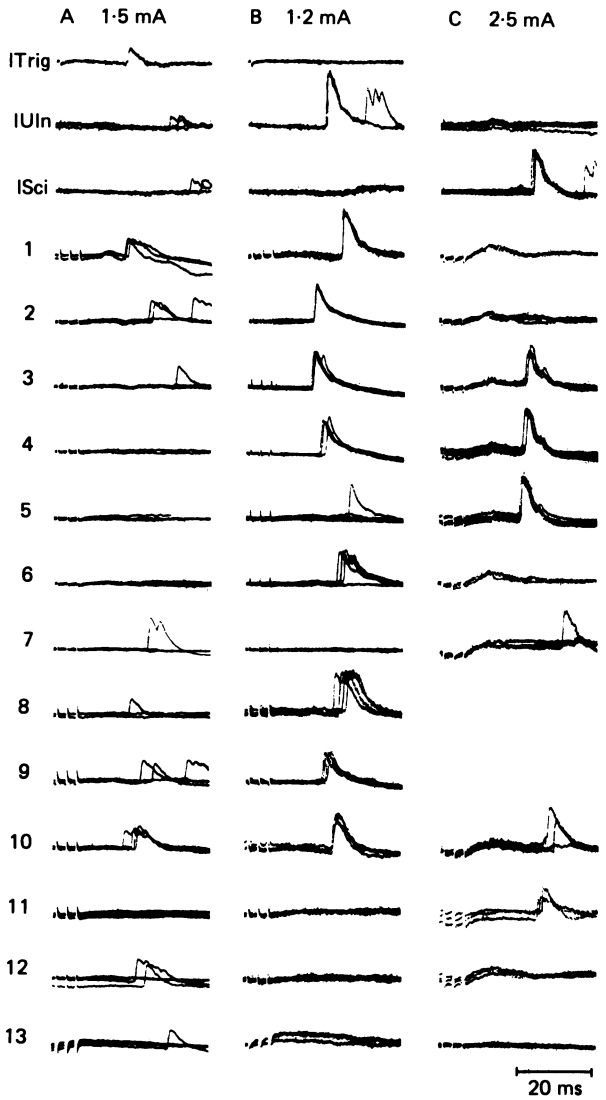
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13
20 ms

(16 ms) and lowest threshold (0·5 mA) was obtained from position 3. The threshold for evoking a response from the SI (site 9) was 0·7 mA and from SII (site 8), 1·2 mA. The response from the ipsilateral p.s.g. (site 10) had a threshold of 1·0 mA.

The hind-limb cell (C) was activated from the medial parts of the p.s.g. on both sides.

Thus, because each Purkinje cell is presumably innervated by a single olivo-cerebellar axon, it can be concluded that the cells received convergent inputs from several cortical areas, the convergence occurring at or before the olivary level. In addition, there was a topographical organization in the projection to these cells with matching peripheral and cortical inputs. In two experiments, recordings were made from forty-five Purkinje cells in the b, c1, c2 and d1 zones. With few exceptions, they all received convergent inputs from more than one cortical area. The cortical input also corresponded to the peripheral input.

(3) Functional organization of climbing fibre projection from the pericruciate cortex

The pericruciate cortex was the most effective cortical area in evoking climbing fibre responses. Therefore, the projection to the cerebellar anterior lobe from the pericruciate cortex was investigated more systematically.

The projection from the pericruciate cortex to the c1 zone will be described in detail. In most respects, the description of this projection to the caudal (forelimb) part of the c1 zone also applies to the projection from the pericruciate cortex to the x zone and to the caudal parts of the c2, c3 and d1 zones. Likewise, the projection to the rostral (hind limb) part of the c1 zone is similar to the projection to the rostral parts of the c2, c3 and d1 zones.

Fig. 5*A* is a map of the right pericruciate cortex (viewed from a rostral direction) in which records of responses evoked in the left caudal c1 zone are shown at those sites from where the responses were evoked (upper traces). The cerebral cortex was stimulated with an intracortical electrode (stimulation strength 1·0 mA). Before the electrode was inserted into the cortex, the responses evoked on left ulnar nerve stimulation were recorded from the surface (lower records). The dots and stars indicate the stimulation sites and, additionally, a latency of 15 ms. The traces shown in the upper right corner are the responses recorded in the left caudal (forelimb) c1 zone on stimulation of some ipsilateral peripheral nerves before (upper) and after (lower) cerebral mapping. There was a small decrease of the response amplitudes but no change of latencies during the experiment.

Responses could be evoked in the c1 zone from an area in the p.s.g. between the post-cruciate dimple and the lateral tip of the cruciate sulcus and, further rostrally, from the a.s.g. From the central sites of the area in the p.s.g., large, short-latency

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Fig. 4. Climbing fibre activation of single Purkinje cells (A, B and C) in the left b zone. Numbers in the cerebral map indicate positions of surface stimulus electrodes. Inlaid traces are responses at the stimulation sites evoked on stimulation of trigeminal (Trig.), ulnar (Uln.) and sciatic (Sci.) nerves contralateral to the respective site. Cerebral stimulation strength indicated above the column of intracellular records from each cell. Voltage calibration: 200 μV for surface recordings, 2 mV for cells A and C, 1 mV for cell B. (Differences in amplitudes of cell A responses due to deterioration of the cell. Cell C lost before the series of recordings was finished). Chloralose anaesthesia.

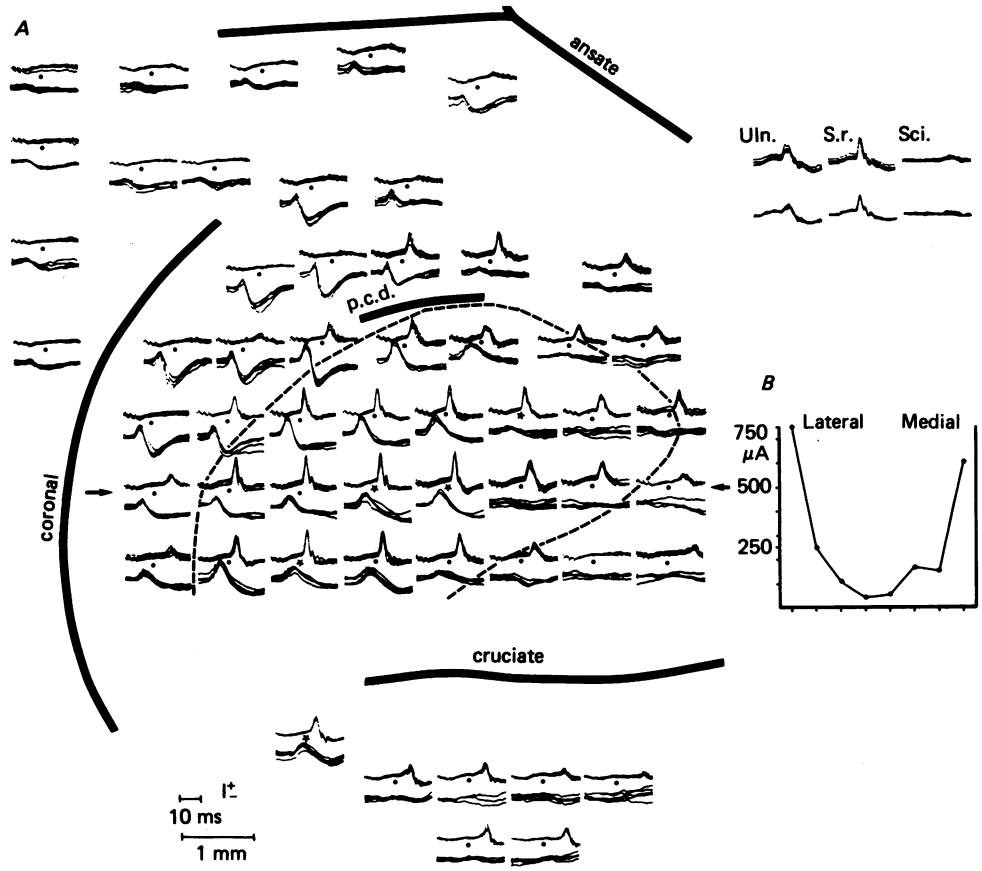

Fig. 5. Climbing fibre projection to the caudal c1 zone from the pericruciate cortex. *A*, map of the right pericruciate cortex. Dots and stars indicate stimulus sites and latency of 15 ms. Upper traces: responses in the left c1 zone to intracortical stimulation at a depth of 2 mm (1 mA, 3 shocks). Lower traces: surface responses evoked at the site on stimulation of the left ulnar nerve. Interrupted line encloses area from which c1 responses could be evoked with stimulus strengths below 0·5 mA. Stars indicate sites with thresholds less than 100 μA. Upper right: responses in the c1 zone to stimulation of left ulnar (l Uln.), superficial radial (l S.r.) and sciatic (l Sci.) nerves before (upper traces) and after (lower traces) mapping of the cerebral cortex. Vertical bar: 200 μV for cerebellar responses and 100 μV for responses in cerebral cortex. *B*, stimulus thresholds for evoking climbing fibre responses in the c1 zone from the row of stimulus sites indicated with two arrows in *A*. Pentobarbitone anaesthesia.

(15 ms) responses were evoked. When stimulating away from this central area, gradually smaller responses with longer latencies were evoked.

Fig. 5*B* shows the thresholds for evoking responses from the sites along the row indicated with arrows in *A*. Centrally, there was an area with thresholds at or below 250 μA. When moving out from this central area, the thresholds increased steeply to values well above 500 μA. In other experiments, similar observations were made. Therefore, a threshold of 500 μA, or less, was used as the criterion of a cortico-olivo-cerebellar projection from a stimulation site.

The interrupted line in Fig. 5*A* encircles the area from which responses were evoked

with a stimulus strength of 500 μA or less. The responses from the a.s.g. sites all had thresholds below 500 μA. The stars indicate the sites where the thresholds were less than 100 μA (in fact, all had thresholds of about 50 μA). They were distributed in a crescent-shaped strip stretching from midway between the dimple and the lateral end of the cruciate sulcus to an area rostrolateral to the lateral end of the sulcus.

The response latencies in the c1 zones were 15–17 ms from the whole area within the interrupted line. The responses evoked from the a.s.g. had longer latencies (21–23 ms), with the exception of the response from the most lateral point which had a latency of 17 ms. This suggests that this site belonged to the same functional area as the one in the p.s.g. which henceforth will be referred to as the short-latency area. The long latencies of the responses from the other a.s.g. sites indicate that these responses were due to activation of a slower conducting cortico-olivo-cerebellar pathway (see below).

It was a general finding that the short-latency projecting area was shifted medially relative to the areas receiving input from the ulnar and superficial radial nerves (cf. Fig. 5*A*). Usually, the lateral half of the projecting area overlapped the medial half of the area receiving input from the two forelimb nerves.

In some cats, the short-latency projecting area extended to behind the dimple. In four experiments, the deep radial nerve was stimulated in order to identify the area receiving group I a input (Oscarsson & Rosén, 1963, 1966). In all these cats the group I area lay 1–1·5 mm caudal to the cortical site with the lowest threshold for evoking climbing fibre responses in the forelimb zones. The lowest thresholds for evoking a response from group I receiving sites were 2–3 times higher than the minimum thresholds for evoking a response from the short-latency area. Climbing fibre responses evoked from group I sites were observed in all zones but a, which was not investigated.

Fig. 6*A* shows the minimum threshold sites for evoking short-latency responses in the caudal (forelimb, dots) part of the c1 zone in those experiments in which the pericruciate cortex was systematically mapped. The lowest threshold for evoking short-latency climbing fibre responses in the caudal c1 zone were found within two areas. The caudal area lay midway between the lateral tip of the cruciate sulcus and the post-cruciate dimple and the rostral area at the lateral tip of the cruciate sulcus (within 2 mm in a lateral or rostral direction). In some cats, only one of these two areas could be distinguished. In other cats, both areas were observed, either as two separate areas or (as in Fig. 5*A*) as a strip of cortex extending from the caudal to the rostral area (indicated with dotted line in Fig. 6*A*).

The minimum threshold sites did not always coincide with the minimum latency and maximum amplitude sites. The optimal sites of these different parameters were often separated by one or a few millimetres. No systematic pattern in these differences was found.

The rostral (hind limb) part of the c1 zone received a short-latency projection from an area in the medial p.s.g. (Fig. 6*A*, crosses). The medial area projecting to the rostral c1 never overlapped the area projecting to the caudal c1. The lateral border of the medial area lay 4–7 mm lateral to the mid line. In most experiments, the area extended rostrally to the cruciate sulcus and caudally to the level of the dimple. In each experiment, the minimum threshold site lay 4–5 mm medial to the corresponding site for the caudal c1 zone.

The minimum threshold sites for the rostral c1 zone sometimes coincided with the area receiving maximal sciatic input, but more often they were found slightly lateral to this area.

The response latencies in the rostral c1 zone on stimulation of the medial p.s.g. were usually 1–3 ms longer than those in the caudal c1 zone on stimulation of the lateral short-latency area. The difficulty in exposing the most medial part of the p.s.g. and thus finding, and stimulating, the optimal hind-limb site could account for part of this difference. However, in those five experiments in which the minimum latency sites for both the caudal and rostral c1 were identified when mapping the p.s.g., the shortest response latencies in the rostral c1 zone on medial p.s.g. stimulation were 0·5–4 ms (mean 1·5 ms) longer than the shortest response latencies in the caudal c1 zone on stimulation of the lateral short-latency area.

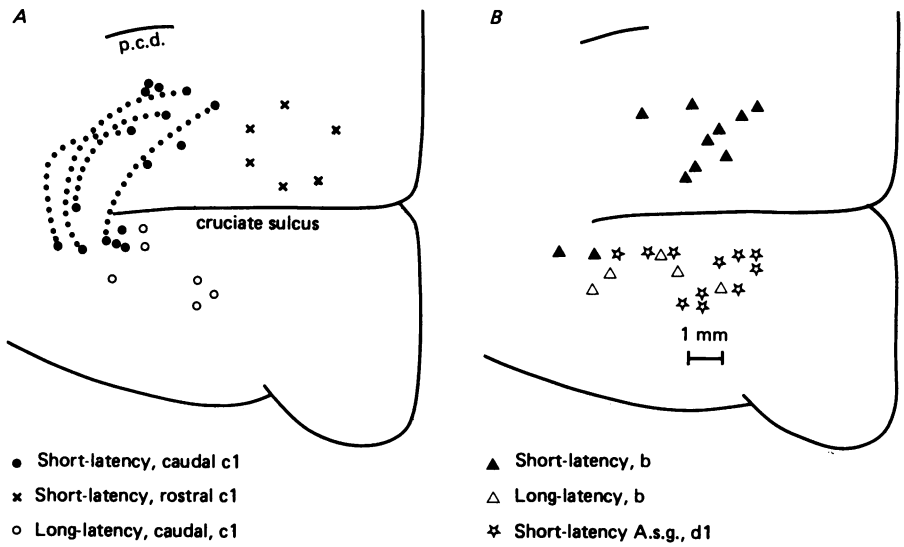

Fig. 6. Minimum threshold sites for evoking climbing fibre responses in the c1 (*A*) and band d1 (*B*) zones. Filled circles and crosses in *A* indicate the minimum threshold sites in the short-latency area for caudal and rostral parts of the c1 zone, respectively; filled triangles in *B* for the b zone. Dotted lines connecting two filled circles indicate strip of cortex with equally low thresholds. Unfilled circles (*A*) and triangles (*B*) indicate the minimum threshold sites in the long-latency a.s.g. area for the caudal c1 and the b zone, respectively. Stars in *B* indicate the minimum threshold sites in the A.s.g. short-latency area for the d1 zone. p.c.d.: post-cruciate dimple.

In seven experiments, long-latency responses (17–27 ms) were evoked in the rostral parts of the c1 and c3 zones on stimulation of the lateral short-latency area in the contralateral p.s.g. The response latencies were 4–13 ms (mean 6·5 ms) longer than the response latencies in the caudal parts of the zones evoked from the same area. In four cats, long-latency responses were evoked in the caudal parts of the c1 and c3 zones on medial p.s.g. stimulation. The response latencies were 18–20 ms, i.e. 3–6 ms (mean 4·5 ms) longer than the response latencies in the rostral parts of the zones. These long-latency responses always had higher thresholds than the short-latency responses from the same area and were not studied further.

The results illustrated in Figs. 5 and 6*A* apply to the x, c1, c2 and c3 zones and also to the d1 zone (with the exception of the projection from the a.s.g., see below). In the experiment illustrated in Fig. 5, for example, recordings were also made in the x zone and the caudal parts of the c2 and d1 zones. These zones received a

projection from the same cortical area as that projecting to the caudal c1 zone. The minimum threshold sites corresponded exactly for the c1 and c2 zones, while for the x and d1 zones, only the caudal-most site, indicated with a star, had a distinctly lower threshold than all the other sites.

In some experiments, small differences were observed between the areas projecting to the x and pars intermedia zones with respect to the borders and the minimum threshold sites. There were, however, no systematic differences, except that the x and d1 zones received a projection from a slightly smaller area. This might be correlated with the fact that the responses in these two zones often had higher thresholds than in the other zones.

Contrary to the vague somatotopy in the peripheral projection to the c2 zone (Larson *et al.* 1969*b*; Ekerot & Larson, 1979*a*), the projection from the pericruciate cortex showed a distinct topographical organization. Thus, the medial short-latency area projected to the c2 zone in lobule Va and further rostrally. The lateral short-latency area projected to the entire lobule V and further caudally (at least to the rostral folium in lobule VI). Between these two projecting areas was a strip of cortex, 1–2 mm wide, from which responses could usually not be evoked with moderate stimulus strengths (0·5 mA, intracortical stimulation) in the c2 zone.

In contrast to the zones in the pars intermedia which received a projection from two short-latency areas, the *b zone* received a projection from only one area. In Fig. 6*B*, the minimum threshold sites for evoking short-latency responses in the b zone are shown (▲). Most of the optimal sites were found in an area in the p.s.g. located between the minimum threshold sites for the caudal and rostral parts of the c1 zone (cf. Fig. 6*A*). There was a topographical organization such that the minimum threshold sites for evoking short-latency climbing fibre responses in the medial (forelimb) part of the b zone lay laterally in the p.s.g. and in the a.s.g. close to the lateral tip of the cruciate sulcus, whereas the minimum threshold sites for evoking responses in more lateral parts of the b zone lay successively more medially in the p.s.g. (but not in the a.s.g.). This is further illustrated in Fig. 4; the optimal site in the p.s.g. for activating the forelimb cell (B) was site 3 and for the hind-limb cell (C) site 5.

The other Purkinje cells encountered in the b zone in this experiment showed the same pattern. The optimal cortical stimulation sites were position 1 for trigeminal cells, 2 and 3 for forelimb cells, 3 for cells with a dominant forelimb and weak hind-limb input, 3 and 4 for cells with equal forelimb and hind-limb inputs, and 4 and 5 for hind-limb cells. For comparison, the cells recorded in the caudal parts of the c1 and c2 zones in this experiment were all activated with the shortest latency from position 2, i.e. slightly lateral to the optimal site for the b zone forelimb cells.

The responses from peripheral nerves in the a zone are usually suppressed by barbiturate anaesthesia (Oscarsson & Sjölund, 1977) and the cortical projection to this zone was studied in only a few experiments. In those two experiments in which the area in the p.s.g. projecting to the a zone was mapped with an intracortical stimulus electrode, the projecting area was shifted slightly laterally to the area projecting to the hind-limb part of the c1 zone and overlapped, almost completely, the area projecting to the lateral part of the b zone. Stimulation of the sciatic nerve evoked no responses in the p.s.g. area projecting to the a zone. Sciatic nerve responses were found more medially.

In experiments with stationary stimulus electrodes, responses in the a zone were often evoked

also from the lateral p.s.g. site and the electrode just rostral to the dimple (see Fig. 2). In these experiments, long-latency responses from forelimb nerves were often seen in the a zone.

The areas in the ipsilateral p.s.g. projecting to the a, b, c2 and d1 zones were homologous to the contralateral areas. The thresholds for evoking ipsilateral responses were usually 1·5–4 times higher than those for contralateral responses.

Responses evoked from the a.s.g. are illustrated in Fig. 5. Long-latency responses (20-22 ms) were evoked in the caudal c1 zone from all but the most lateral stimulation sites in the a.s.g. The response latencies were distinctly longer (average difference 5 ms) than those from the short-latency area. The thresholds for evoking these long-latency responses were rather low, 180–420 μA. Thus, it is unlikely that they were due to stimulus spread into the short-latency area. In fact, the minimum threshold site for evoking a long-latency response was the medial of the two rostral sites, i.e., the site being most distant from the short-latency area.

Such long-latency responses were observed in the forelimb parts of all zones, except the d1 zone (see below), on stimulation of the lateral part of the a.s.g. The a zone was not investigated.

These observations suggest the existence of a cortico-olivo-cerebellar pathway originating in the a.s.g. which has a slower conduction velocity and/or a greater number of intercalated neurones than the pathway from the short-latency area.

Fig. 6 (*A* and *B*) shows the minimum threshold sites for evoking long-latency responses in the forelimb parts of the c1 (○) and b (△) zones, taken from those experiments in which the sites were identified on intracortical stimulation of the a.s.g. In some cases, there were small differences between the minimum threshold site locations for the different zones, but no systematic difference was found.

The minimum thresholds for evoking the long-latency responses from the a.s.g. were usually 1·5–4 times higher than those for evoking short-latency responses. The area from which long-latency responses were evoked varied between the experiments from only one or two stimulation sites to an area covering several square millimetres.

It was a general finding that short-latency climbing fibre responses could be evoked in the cerebellum on stimulation of sites in the lateral a.s.g. where ulnar nerve stimulation evoked responses at latencies of 7–9 ms (see Fig. 5*A*). At the sites from which long-latency climbing fibre responses were elicited, no, or only very small, surface positive potentials were evoked from the limb nerves in cats under pentobarbitone anaesthesia. Under chloralose anaesthesia, rather large responses with latencies of 20–25 ms were evoked from all four limbs in the a.s.g. long-latency area.

The long-latency a.s.g. area could not be identified in all experiments. When successively more medial sites in the a.s.g. were stimulated, a gradual increase in the climbing fibre response latencies, without a distinct low-threshold area, was observed in some cases. In other experiments, double responses were evoked from certain sites indicating that the pathways from both the short-latency and long-latency areas were activated simultaneously. In those cases, it was difficult to measure the latencies and amplitudes of the second response component.

In Fig. 3, the shortest latencies of the responses from the a.s.g. long-latency area are selected from those experiments where long-latency responses could clearly be identified. Because of the above-mentioned difficulties, only positive results are considered in Fig. 3.

Unlike the other zones, the *d1 zone* received a short-latency climbing fibre input (12–13 ms) from the a.s.g. (Fig. 3). These responses usually had lower thresholds and shorter latencies than those evoked from the p.s.g. Fig. 7 shows the responses in the left d1 zone in lobule IV from an experiment in which the right a.s.g. was mapped

with intracortical stimulation (stimulation strength 0·5 mA). Responses, with latencies of 13–15 ms, were evoked from a rather large area in the a.s.g. When the stimulation strength was increased to 1·0 mA, the response latency from most of the sites decreased to 12 ms. The minimum threshold for evoking a response was 150 μA; the minimum threshold sites are indicated with stars. The response from the optimal site in the right (r) p.s.g. was considerably smaller than from the a.s.g. in this experiment.

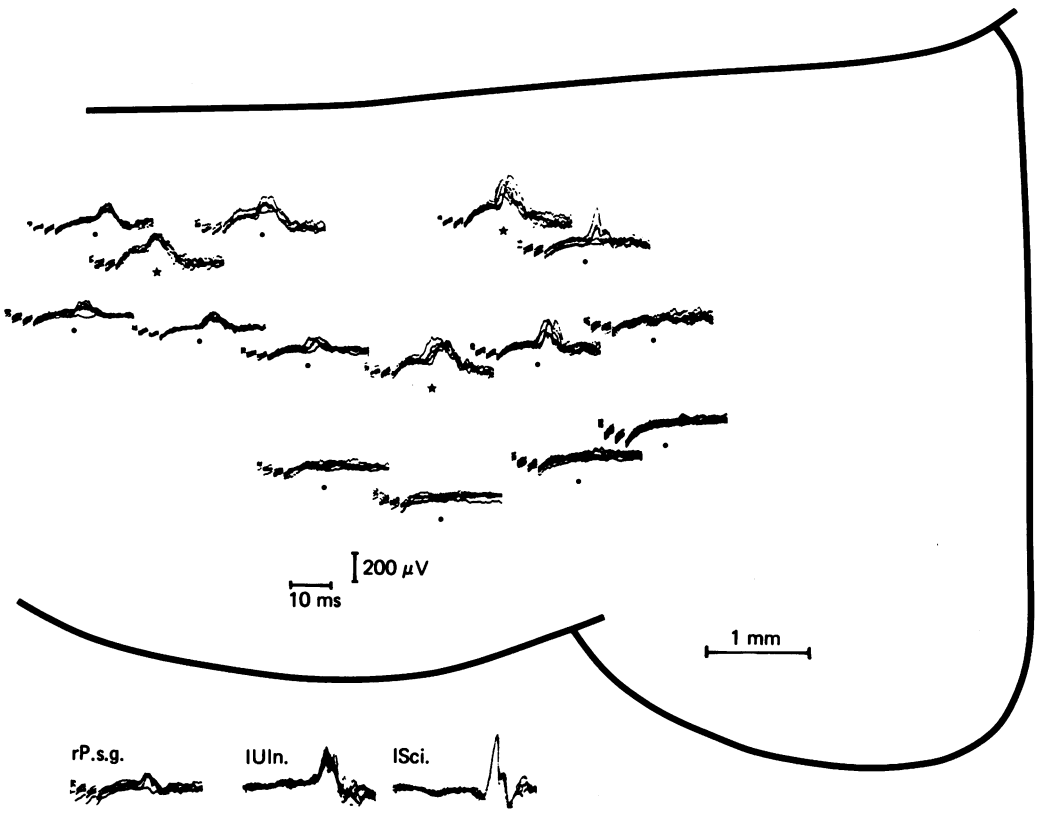

Fig. 7. Climbing fibre projection from the right anterior sigmoid gyrus to the left d1 zone in lobule IV. Inlaid records are responses in the d1 zone on intracortical stimulation at a depth of 2 mm at the sites indicated with dots and stars, which also indicate latency of 15 ms. Stimulation strength 0·5 mA. Lower row of records: responses in the d1 zone on stimulation of the optimal site in the right posterior sigmoid gyrus (r.P.s.g.), left ulnar (Uln.) and sciatic (Sci.) nerves. Pentobarbitone anaesthesia.

In other experiments, the response amplitudes on p.s.g. stimulation were the same as, or even larger than, those on a.s.g. stimulation.

The area from which short-latency responses could be evoked in the d1 zone often extended to at least 6 mm medially from the lateral tip of the cruciate sulcus. The a.s.g. was seldom stimulated further medially. The locations of minimum threshold sites for the d1 zone, as identified in experiments with intracortical stimulation, are shown in Fig. 6*B* (stars). There was a large inter-experimental variation in the spatial relation between the a.s.g. area projecting to the d1 zone and the short-latency and long-latency areas projecting to the other zones. The area from which long-latency

responses were evoked in the other zones usually overlapped the lateral half of the a.s.g. area projecting to the d1 zone.

Responses were also evoked in the d1 zone on stimulation of the ipsilateral a.s.g. The stimulus strength required was usually 2–3 times higher than that required for evoking responses from the contralateral a.s.g. The ipsilateral response latencies were 17–19 ms, i.e. 5 ms longer than the contralateral ones (Fig. 3).

DISCUSSION

The results of the present study demonstrate a zonal pattern in the climbing fibre projection to the cerebellar anterior lobe from the cerebral cortex. This zonal pattern is related to the sagittal zones which can be identified from the peripheral climbing fibre input.

Fig. 8 is a summary diagram showing the cortical areas projecting to each zone in lobule V. Dense hatching indicates areas from where responses were evoked in more than 80 % of the experiments. Sparse hatching indicates areas from where responses were evoked in at least 40 % of the experiments. In addition to these projections, responses were occasionally evoked in some of the zones from areas not hatched in Fig. 8 (cf. Fig. 3). These responses were recorded mainly under chloralose anaesthesia and often had higher thresholds and lower amplitudes than the responses which have been considered for Fig. 8.

The use of dense hatching in the long-latency area in the a.s.g. and sparse hatching in the SI is somewhat tentative. Although it was impossible, in some cases, to identify the minimum threshold sites for evoking long-latency responses from the a.s.g. and to obtain a plateau on the latency curves of these responses, long-latency responses were evoked from the lateral a.s.g. in almost all experiments. Therefore, it seems justified to indicate this area with dense hatching. Stimulation of the SI less frequently evoked climbing fibre responses at moderate stimulus strengths. Hence, this area is indicated with sparse hatching.

In addition, the medial p.s.g. area which projects to the c2 and d1 zones in the rostral part of lobule V is, for the sake of clarity, not shown.

The predominant projection originates in the pericruciate cortex. From sensory and parietal cortical areas considerably weaker projections were generally observed. The zones receive characteristically different inputs. The most conspicuous features are: the a and b zones receive almost equally strong p.s.g. inputs from both sides; the x, c1 and c3 zones, which have similar, ipsilateral peripheral inputs, receive contralateral cortical inputs only, mainly from the p.s.g. Two separate areas project to these zones. A lateral area extends rostrally into the lateral part of the a.s.g. and projects to the forelimb parts, and a medial area projects to the hind-limb parts of the cerebellar zones.

The p.s.g. projection to the c2 zone resembles that to the x, c1 and c3 zones but is bilateral. There are also projections from the second somatosensory area (SII), the coronal gyrus and the parietal cortex.

The d1 zone contrasts with the other zones by receiving a strong projection from the parietal cortex and a short-latency projection from a large area in the a.s.g.

A comparison with the cytoarchitectonic map of Hassler & Muhs-Clement (1964) reveals that the major part of the short-latency (p.s.g.) area belongs to area 4γ. A projection from the group I receiving area 3a (Oscarsson & Rosén, 1963, 1966;

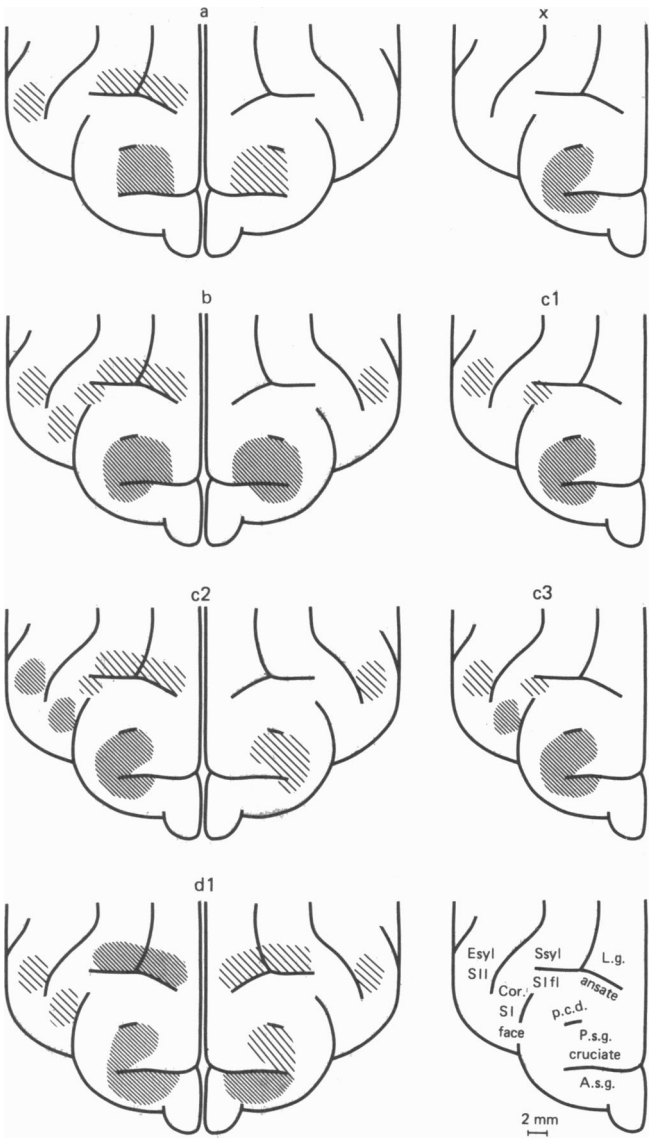

Fig. 8. Summary diagram of cerebral cortical areas from which climbing fibre responses could be evoked in the respective zones in more than 80% of the experiments (dense hatching) or in at least 40% of the experiments (sparse hatching). Abbreviations as in Fig. 2.

Landgren & Silfvenius, 1969) cannot be excluded. The p.s.g. area projecting to the hind-limb zones (Fig. 6*A*) also seems to correspond to the dorsal hind-limb group I locus of Landgren & Silfvenius (1969).

Long-latency responses were evoked in several zones on stimulation of an area in the lateral a.s.g. (Figs. 5 and 6), which seems to lie within area 4γ. In the d1 zone, short-latency responses were elicited from an area which extended further medially and, hence, might include part of area 6aβ. However, since histological analysis was

not made, the relationship between the projecting areas and the cytoarchitecture remains uncertain.

The tendency for two minimum threshold areas to occur within the short-latency area projecting to the forelimb zones might be related to the finding that two spatially separate representations exist for the distal forelimb muscles in area 4γ (Pappas & Strick, 1981). Although located slightly more laterally, these two 'digit zones' had approximately the same position as the two minimum threshold areas in the present study, i.e. the caudal area, midway between the lateral tip of the cruciate sulcus and the post-cruciate dimple, and the rostral area, close to the tip of the cruciate sulcus (Fig. 6*A*). Pappas & Strick (1981) suggest that the double representation may reflect the existence of two motor control systems dealing with different components of motor behaviour. If so, it might be important that both these motor control systems project also to the cerebellum (see below), as suggested by the present findings.

Interestingly, the minimum threshold sites in the p.s.g. for the b zone (Fig. 6*B*) seem to be located in the area which sends corticospinal fibres both ipsilaterally and contralaterally (Armand & Kuypers, 1980) and fibres with collaterals to both the cervical and lumbar enlargements (Hayes & Rustioni, 1981). The efferent projection from this area thus resembles the afferent projection to the b zone, bilateral and with some overlap of the forelimb and hind-limb projections.

In general, the present results have confirmed those of previous studies on the cortico-olivo-cerebellar projection (Provini *et al.* 1968; Miller *et al.* 1969*a*; Allen *et al.* 1974*a*, *b*; Miles & Wiesendanger, 1975*a*, *b*; Sasaki *et al.* 1975; Oka *et al.* 1976, 1979; Rowe 1977*a*). Summarizing these reports, there is a somatotopically organized climbing fibre input to nearly all Purkinje cells in the anterior lobe from the p.s.g. A substantial proportion of the cells receive also an input from the SI and SII. Most Purkinje cells receive converging climbing fibre inputs from peripheral nerves and somatotopically corresponding parts of the sensorimotor cortex. There is also a projection from the a.s.g. which does not seem to be somatotopically organized. Long-latency responses can be evoked on stimulation of the parietal cortex. The responses from the SI, SII and parietal cortex are mediated through private cortico-olivary paths, since ablation of the pericruciate cortex did not abolish these responses (Sasaki *et al.* 1975; Rowe 1977*a*).

The results of the present and previous studies suggest the existence of a multitude of cortico-olivo-cerebellar pathways, originating in different areas of the cerebral cortex and terminating in separate cerebellar zones (see above, Fig. 8). The difference in thresholds for evoking responses, as well as in response latencies, between some of the zones on stimulation of a single cortical site indicates that cortico-olivo-cerebellar pathways originating in one cortical area, but terminating in different zones, can have different synaptic organizations.

In the experiment illustrated in Fig. 2, for example, stimulation of the p.s.g. evoked responses in all zones except the c2 zone. However, in other experiments, a projection from the p.s.g. to the c2 zone was observed (cf. Fig. 3). Its absence in that particular experiment might be explained by a low excitability of the olivary neurones projecting to the c2 zone. This is, however, rather unlikely since responses were evoked in the c2 zone on stimulation of peripheral nerves and the contralateral SII. It seems more likely that the pathway from the p.s.g. to the c2 zone has a different synaptic organization from the pathways to the other zones and that, in the illustrated experiment, the transmission through this pathway was suppressed.

The pathways mediating the cortically induced climbing fibre responses are largely unknown. Anatomical investigations have indicated a direct, bilateral cortico-olivary path (Walberg, 1956; Sousa-Pinto, 1969; Sousa-Pinto & Brodal, 1969; Bishop, McCrea & Kitai, 1976; Brown, Chan-Palay & Palay, 1977; Berkley & Worden, 1978). These results have, however, been questioned (Saint-Cyr & Courville, 1980). Thus, the climbing fibre responses observed in the present, as well as in previous studies, might be mediated through indirect cortico-olivary paths. The relay nuclei, with one exception, have not been identified, but the inferior olive receives a descending input from several structures. These projections have recently been reviewed in detail (Brodal & Kawamura, 1980; Cintas, Rutherford & Gwyn, 1980; Martin, Culberson, Laxon, Linauts, Panneton & Tschismadia, 1980; Saint-Cyr & Courville, 1980).

Physiological studies have demonstrated that climbing fibre responses evoked in both the vermis and pars intermedia on sensorimotor cortex stimulation are mediated by slowly conducting fibres in the pyramidal tract (Cervetto, Marchesi & Strata, 1969; Kitai, Oshima, Provini & Tsukahara, 1969). Despite the rather slow conduction velocity, the response latency on pyramidal tract stimulation seems to be sufficiently long to include in addition a pre-olivary synaptic relay. Interestingly, the conduction velocity of pyramidal tract fibres mediating responses to the forelimb part of the cerebellum was faster (mean 10·0 m/s) than the velocity of the fibres mediating a response to the hind-limb part (mean 8·4 m/s) (Kitai *et al.* 1969). This is in agreement with the findings in the present study, where responses from the medial p.s.g. in the hind-limb zones usually had slightly longer latencies (mean difference 1·5 ms) than those from the lateral p.s.g. in the forelimb zones.

The only well-established relay in a cortico-olivary path is the parvocellular part of the red nucleus (Oka & Jinnai, 1978*a*, *b*; Oka *et al.* 1979). This has been shown to mediate parietal-evoked climbing fibre responses to the d1 zone (cf. Miller *et al.* 1969*b*; Jeneskog, 1974*a*, *b*, 1981). Whether the parietal-evoked responses in the other zones also are mediated through the parvocellular red nucleus is uncertain.

A general finding of both the present and previous studies is that stimulation of one cortical site evoked a response not only in a large part of one zone but in several zones. A response at one cerebellar recording site, or in a single Purkinje cell, could also be evoked from many stimulation sites within one cortical area, as well as from several separate areas. This divergence and convergence must be considered when discussing the functional role of the cortico-olivo-cerebellar projection.

The inferior olive has been suggested to function as a comparator of descending and ascending information (Miller & Oscarsson, 1970; Oscarsson, 1980). Each sagittal cerebellar-cortical zone and its associated cerebellar or vestibular nucleus probably constitute a functional unit concerned with controlling a particular motor mechanism (Oscarsson, 1980). Each zone would then receive information which is relevant to this motor function.

The striking parallelism in the distribution pattern of the climbing fibre responses evoked in the cerebellar zones (with the exception of c2) on stimulation of peripheral nerves and the corresponding areas in the sensorimotor cortex reveals that each part of the inferior olive receives convergent inputs from certain segments of the spinal cord and the corresponding part of the motor cortex. This is consistent with, and is, indeed, a prerequisite for, the comparator hypothesis.

If stimulation of a particular cortical site evokes responses in several zones, it would imply that this stimulation also affects each of the motor mechanisms controlled by the activated zones.

Indeed, it has been shown that, in addition to exciting motoneurones in a topographically organized manner (Livingston & Phillips, 1957; Asanuma & Sakata, 1967; Asanuma, Stoney & Abzug, 1968; Nieoullon & Rispal-Padel, 1976; Pappas & Strick, 1981), stimulation of the sensorimotor cortex produces many other effects. For example, stimulation of the area which activates distal forelimb muscles via the pyramidal tract, also activates the proximal forelimb muscles through extrapyramidal pathways (Asanuma, Babb, Mori & Waters, 1981). This effect is probably part of a postural adjustment (Gahéry & Massion, 1981). Furthermore, stimulation of the sensorimotor cortex facilitates and/or depresses the transmission through different spinal reflex arcs (Lundberg & Voorhoeve, 1962; Hongo & Jankowska, 1967), through jaw reflex arcs (Olsson & Landgren, 1980), through ascending spinal tracts (Magni & Oscarsson, 1961; Lundberg, Norrsell & Voorhoeve, 1963; Hongo, Okada & Sato, 1967; Hongo & Okada, 1967) and through the dorsal funiculus nuclei (Jabbur & Towe, 1961; Andersen, Eccles, Oshima & Schmidt, 1964; Andersen, Eccles, Schmidt & Yokota, 1964*c*; Gordon & Jukes, 1964; Levitt, Carreras, Liu & Chambers, 1964). In addition, depolarization of presynaptic fibre terminals in the spinal cord (Carpenter, Lundberg & Norrsell, 1963; Andersen, Eccles & Sears, 1962, 1964; Abdelmouméne, Besson & Aléonard, 1970) and in the dorsal funiculus nuclei (Anderson, Eccles, Schmidt & Yokota, 1964*a*, *b*) is also produced by stimulation of this cortical area.

Thus, a large number of interneurone pools, participating in different mechanisms, are activated from the sensorimotor cortex. If the cerebellar zones participate in the control of these mechanisms, each zone should receive information about the activation of its particular interneurone pool. Hence, the divergence to several zones from those cortical sites which activate functionally separate interneurone pools would be expected.

Similarly, the convergence from several stimulation sites to one cerebellar recording site, or to a single Purkinje cell, could be compared to the convergent effects from more than one cortical site, or area, onto particular interneurone pools. Many of the effects on sensorimotor cortex stimulation mentioned above were elicited from a large area rather than from only one site or from several separate areas. Some of the effects were also bilateral.

Thus, more than one cortical area can be concerned with the regulation of a particular function and, consistent with the comparator hypothesis, can also converge onto a certain cerebellar zone.

The cortically induced inhibition of ascending spinal tracts, mentioned above, also applies to the spino-olivo-cerebellar pathways (Leicht, Rowe & Schmidt, 1972, 1973; Fennel & Rowe, 1973; Rowe, 1977*b*). It was often shown in those studies that both climbing fibre excitation of a Purkinje cell and inhibition of its response to a peripheral stimulus were produced from the same cerebral cortical stimulation site. Usually, the inhibition was achieved with a lower stimulus strength than the excitation. At least part of the inhibition occurred at a preolivary level. In some, but far from all, Purkinje cells, the cortically induced inhibition was somatotopically organized.

Thus, the cerebral cortex can inhibit the peripheral climbing fibre input to the cerebellum. This might be a mechanism by which the cerebral cortex cancels peripheral input which is expected in connection with a movement. Unexpected peripheral input, however, brought about by an error in the executed movement, might be transmitted to the cerebellum. In such a case, the comparator function would be exerted through a comparison of the cortical inhibitory and ascending excitatory inputs to the inferior olive.

This work was supported by grants from the Medical Faculty, University of Lund, and to Dr O. Oscarsson from the Swedish Medical Research Council (Project No. 1013). The authors wish to thank Dr O. Oscarsson for helpful discussion and valuable criticism throughout this work.

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