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ELECTROMYOGRAPHIC RESPONSES EVOKED IN MUSCLES OF THE FORELIMB BY INTRACORTICAL STIMULATION IN THE CAT

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SUMMARY

1. Chronically implanted microwires were used to deliver brief trains of electrical stimuli (11 cathodal pulses at 330 Hz and intensity 5–35 μA) to sixty-two locations in the grey matter of the pericruciate cortex in cats.

2. Electromyographic (e.m.g.) responses in the contralateral forelimb were recorded from a total of ten muscles (four to eight in each animal) acting about the shoulder, elbow and wrist and on the digits. The animals were relaxed with little background e.m.g. in the muscles and as a result only excitatory effects could be described.

3. Five muscles which are flexors in the locomotor context were excited from more electrodes, distributed more widely across the motor cortex, than another five muscles which are extensors during locomotion; this difference in 'accessibility' was present both at 35 μA stimulus intensity and at 15 μA.

4. At a stimulus intensity of 15 μA, effective cortical electrodes tended to cluster either in the most lateral part of the anterior sigmoid gyrus (rostromedial focus) or in the coronal gyrus just caudal to a line prolonged beyond the lateral end of the cruciate sulcus (caudolateral focus). This is consistent with the existence of a double motor representation within the forelimb motor cortex (Pappas & Strick, 1981).

5. The two foci were similar in that both gave rise to more flexor than extensor responses and to fewer responses in digit or wrist muscles than in muscles acting about more proximal joints (elbow and shoulder).

6. At stimulus intensity 35 μA the latency of the earliest e.m.g. responses ranged from 11 to 14 ms in different muscles.

7. For some muscles and electrodes the amplitude of the e.m.g. responses was substantially altered by a quite small postural change.

8. After pyramidectomy the cortical thresholds and the e.m.g. latencies were both greatly increased.

INTRODUCTION

A previous paper (Armstrong & Drew, 1984*a*) described movements evoked in cats by brief trains of stimuli delivered to the motor cortex via chronically implanted micro-electrodes (which were also used to record the activity of single cortical

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neurones during locomotion; see Armstrong & Drew, 1984*b*). Movements were produced from many loci within the pericruciate cortex and maps were made to delimit the areas of cortex yielding different movements of the contralateral forelimb (see also Nieoullon & Rispal-Padel, 1976).

Whilst useful information was provided regarding the localization and the excitability of the cortical mechanisms whose excitation gave rise to the movements, it was frequently observed that thresholds for movement were higher than those for producing electromyographic (e.m.g.) responses in the forelimb muscles. Moreover, *digit and wrist movements were detected less often than e.m.g. responses in forearm* muscles which contribute to control of the digits and wrist.

In view of these findings, and because Pappas & Strick (1981) have found in anaesthetized cats that contractions of digit muscles are produced from two distinct low-threshold areas in the forelimb motor cortex, we investigated e.m.g. responses evoked in a range of forelimb muscles by intracortical microstimulation. Muscles acting at the shoulder, elbow and wrist and on the digits were examined and the spatial distributions of the cortical loci from which they were excited at stimulus intensities between 5 and 35 μA were determined.

The findings demonstrate that motoneurones supplying all the muscles studied can be discharged by intracortical stimulation and they also indicate that the double representation of muscles shown in the cat motor cortex by Pappas & Strick (1981) is present in the awake animal. However, this appears not to be confined to digit muscles, but extends also to muscles acting around the wrist, elbow and shoulder. The effects of pyramidectomy on the e.m.g. responses are described.

METHODS

Nine cats were used, drawn from among those used to obtain recordings of the discharges of single motor cortical neurones at rest and during locomotion (Armstrong & Drew, 1984*b*, *c*). Electrical stimuli were applied to the right motor cortex via a total of sixty-two platinum–iridium microwire electrodes, all of which were previously shown to evoke detectable movements of the contralateral forelimb when trains of 11 pulses (0·2 ms width at a frequency of 330 Hz and intensity 35 μA) were used. For details of the electrodes and the procedures used to implant them at an initial aseptic operation using full general anaesthesia see Armstrong & Drew (1984*b*). The motor cortex was examined histologically (see Armstrong & Drew, 1984*b*) and most electrode tracks were found to terminate in the cortical grey matter rather than the underlying white matter. Because twenty to twenty-five electrodes were implanted in each animal it was not usually possible to establish which electrode made which track. However, such a reconstruction was achieved in one animal and it showed that although single- and multi-unit activity was not recordable via every electrode tip in the grey matter, all tips which did record such activity were in the deeper layers of the grey matter. In addition, all tips in the white matter failed to record such activity. Most of the present electrodes recorded neuronal activity and we therefore believe that most, if not all, had their tips in the deeper layers of the grey matter.

Electromyography

In five cats e.m.g. recordings were obtained from only four muscles in the contralateral (left) forelimb but in each of the four remaining animals eight or nine muscles were recorded. Altogether ten muscles were studied acting at the shoulder, elbow and wrist and on the digits. They included brachialis, biceps brachii, cleidobrachialis, extensor carpi ulnaris, extensor digitorum communis, latissimus dorsi, triceps brachii lateral head and long head, flexor carpi ulnaris and palmaris longus. The e.m.g. electrodes were chronically implanted as described by Armstrong & Drew (1984*b*). E.m.g.

responses to cortical stimulation were amplified and tape recorded (Racal Thermionic Store 7D; over-all bandpass 100 Hz–1·3 kHz).

For analysis the taped e.m.g. responses were low-pass filtered at 400 Hz and digitized at 1 kHz. The data were then full-wave rectified and twenty–thirty successive responses were averaged using a suitably programmed PDP 11/34 computer. Averaging was initiated 50 ms prior to stimulus delivery and continued for a further 100 ms. The system included cursor-setting facilities which permitted measurement of response latencies. Response sizes are not of particular concern in this study but it should be noted that the peak amplitude of the smallest responses was *ca.* 0·1 mV and amounted to *ca.* 10% of the peak instantaneous amplitude of the (much longer) bursts of e.m.g. recorded from the same muscle during steady walking at the relatively low speed of 0·5 m/s. In general, such small responses were not accompanied by visible movement. The largest responses encountered were about 5 times the peak amplitude of the locomotor bursts of e.m.g. and were accompanied by a brisk flick movement.

Intracortical stimulation

As in a preceding paper (Armstrong & Drew, 1984*a*), the cortical stimuli were trains of 11 pulses at 330 Hz. Cathodal pulses of duration 0·2 ms were employed throughout, a diffuse anode being provided by a stainless-steel electrode implanted under the scalp. Stimulus trains were presented at rates of 0·5 Hz or less. Stimulus intensities ranged between 5 and 35 μA.

The point of entry of all sixty-two micro-electrodes into the pericruciate cortex was charted on a photograph of the brain surface. Because the electrodes were inserted to a depth of less than 2 mm and most entered the cortex normal to the pial surface, the entry points provide a fair approximation of the distribution of the electrode tips across the cortex. The electrodes were in the coronal gyrus (definition of Livingston & Phillips, 1957) and the adjoining (lateral) part of the anterior and posterior sigmoid gyri. Entry points from individual animals were pooled onto a single cortical diagram according to their mediolateral and rostrocaudal distances from the lateral tip of the cruciate sulcus (see Armstrong & Drew, 1984*a*).

In three animals the right medullary pyramid was transected (see Armstrong & Drew, 1984*a*) and the e.m.g. responses to cortical stimulation were re-investigated one week later.

Post mortem

At the end of the experiment each animal was killed by anaesthetic overdose and the positions of the e.m.g. electrodes were confirmed by dissection. The method of Swank & Davenport (1935) was used to verify the completeness of the pyramidal transections (see Armstrong & Drew, 1984*a*).

RESULTS

Nature of the electromyographic responses to intracortical microstimulation

Intracortical microstimulation (trains of 11 pulses at frequency 330 Hz) was first applied at intensity 35 μA to a total of sixty-two locations in nine cats whilst electromyographic responses were recorded from muscles of the contralateral forelimb (see Methods). Because 35 μA stimuli applied via each of the electrodes were previously shown to elicit forelimb movements (see Armstrong & Drew, 1984*a*), it is not surprising that e.m.g. responses were almost always produced in one or more of the muscles studied. The responses were invariably brief (usually <50 ms), in agreement with the observation that the movements had a brief flick-like character. The usual finding was that the responses fluctuated in amplitude (and sometimes also in latency) from trial to trial. Variation was present even when the animal was immobile but its extent was least when postural changes were minimized. For this reason the animals were routinely held by the loose skin over the shoulders and back of the neck and supported under the belly with both forelimbs and hind limbs out of contact with any supporting surface. Care was taken to ensure that the animals

were comfortable. They purred frequently, made few spontaneous movements and evidence of relaxation was given by the low levels of background e.m.g.

The rectified e.m.g. signals were computer-averaged (see Methods) and examples of the resultant displays are shown for eight different muscles in Fig. 1. Fig. 1 *A–F*, respectively, illustrate the effect of stimulation at six different cortical locations. The

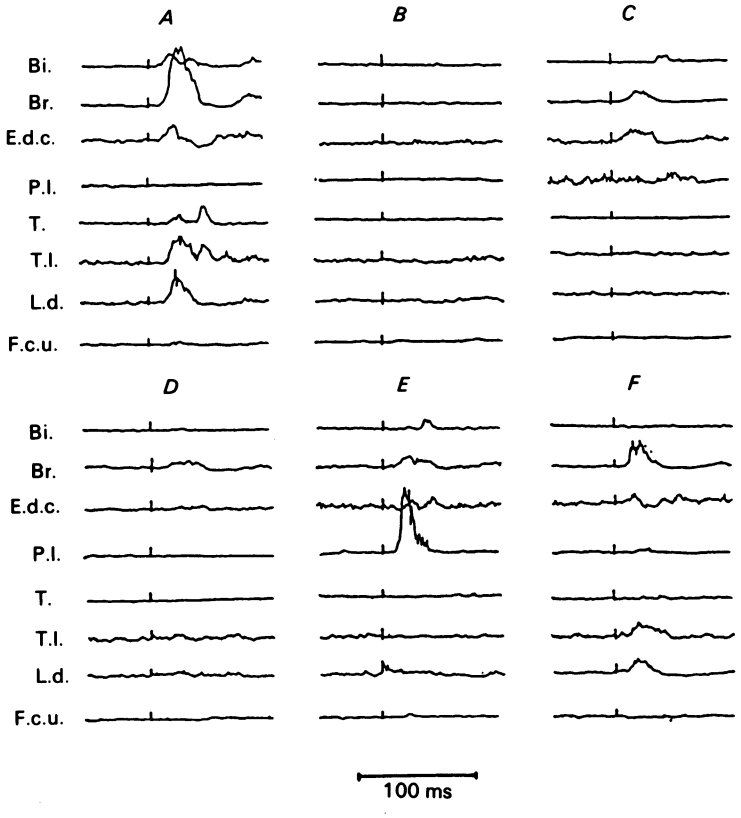

Fig. 1. E.m.g. responses evoked in eight muscles of the contralateral forelimb by intracortical stimulation. *A–F* each represent responses evoked from a different locus in the same animal. All traces are full-wave rectified and are the averages of twenty successive responses evoked at a repetition rate of 0·5 Hz. Vertical ticks indicate onset of the stimulus train which consisted of 11 pulses at 350 Hz and intensity 35 μA. Time calibration below *E* applies to all records. Muscle abbreviations: Bi., biceps brachii; Br., brachialis; E.d.c., extensor digitorum communis; P.l., palmaris longus; T., lateral head of triceps brachii; T.l., long head of triceps brachii; L.d., latissimus dorsi; F.c.u., flexor carpi ulnaris.

largest responses shown (brachialis in Fig. 1 *A* and palmaris longus in Fig. 1 *E*) had peak amplitudes approximately twice the peak instantaneous e.m.g. developed in the same muscle during the bursts of activity which occurred in locomotion at a slow walking pace (0·5 m/s). Occasionally tonic activity was present in one or more of the muscles and on some of these occasions it was evident that cortical stimulation was capable of producing decreases as well as increases in muscle activity. Examples of

such 'inhibitory' effects can be seen in the traces for extensor digitorum communis in Fig. 1*A* and *F*. Such effects were not deliberately sought or studied in detail.

Fig. 1 exemplifies the fact that most electrodes produced excitation of several muscles (see especially Fig. 1*A*) whilst the actions of others were confined to one muscle (see Fig. 1*D*, brachialis). A few electrodes were ineffective (see Fig. 1*B*), though since they evoked visible movement it can be presumed that actions were exerted on muscles not displayed.

Out of thirty-five electrodes (in three cats) for which eight particular muscles were studied (brachialis, biceps brachii, cleidobrachialis, triceps brachii lateral head, triceps brachii long head, latissimus dorsi, extensor digitorum communis and palmaris longus) there were no electrodes from which all these muscles were influenced and five electrodes which influenced none of them. The number of electrodes influencing one, two, three, four, five, six and seven muscles was three, five, six, seven, two, one and six, respectively. The most usual finding, therefore, was that from two to four muscles were influenced but it is noteworthy that six electrodes influenced as many as seven muscles. The locations of these electrodes will be specified below.

Cortical topography for the e.m.g. responses to 35 μA stimulation

Fig. 2*A–J* each indicate for a particular muscle the distribution within the pericruciate cortex of the electrode locations from which e.m.g. activity was elicited by stimulation at intensity 35 μA. Filled circles represent effective electrodes while the open circles represent loci from which no response was evoked in that muscle (although a limb movement was evoked and e.m.g. was usually evoked in at least one other muscle). Note that each map displays the pooled results from all nine animals.

Two features of the maps are worthy of emphasis. First, it is clear that for each muscle the effective points were rather widespread. This scatter may result partly from the procedure of pooling results from different animals (see Pappas & Strick, 1981; Armstrong & Drew, 1984*a*) but in any one animal the locations which produced activity in a single muscle were frequently widely separated and were sometimes on opposite boundaries of the distributions in Fig. 2.

Secondly, although some locations produced excitation of several muscles (see above), it is clear that some muscles were excited from a larger number of electrodes than others (cf. Fig. 2*A*, brachialis, forty-one out of sixty-two locations and Fig. 2*G*, lateral head of triceps brachii, eighteen out of sixty-two locations). The rank order of 'accessibility' for the different muscles is shown in Fig. 3*A*. It is noteworthy that the five muscles most frequently excited were the three which flex the elbow (brachialis, cleidobrachialis and biceps brachii) plus the two which dorsiflex the wrist (extensor carpi ulnaris) and digits (extensor digitorum communis). These five muscles are all physiological flexors in the context of locomotion, i.e. they are most active during the swing phase of the forelimb step cycle (Drew, 1981). The five remaining muscles were excited less often. They include two which extend the elbow (lateral and long heads of triceps brachii), latissimus dorsi, which acts about the shoulder and two muscles which ventroflex the wrist (flexor carpi ulnaris) and digits (palmaris longus). All five are physiological extensors during locomotion, i.e. active during the stance phase of the step cycle (Drew, 1981).

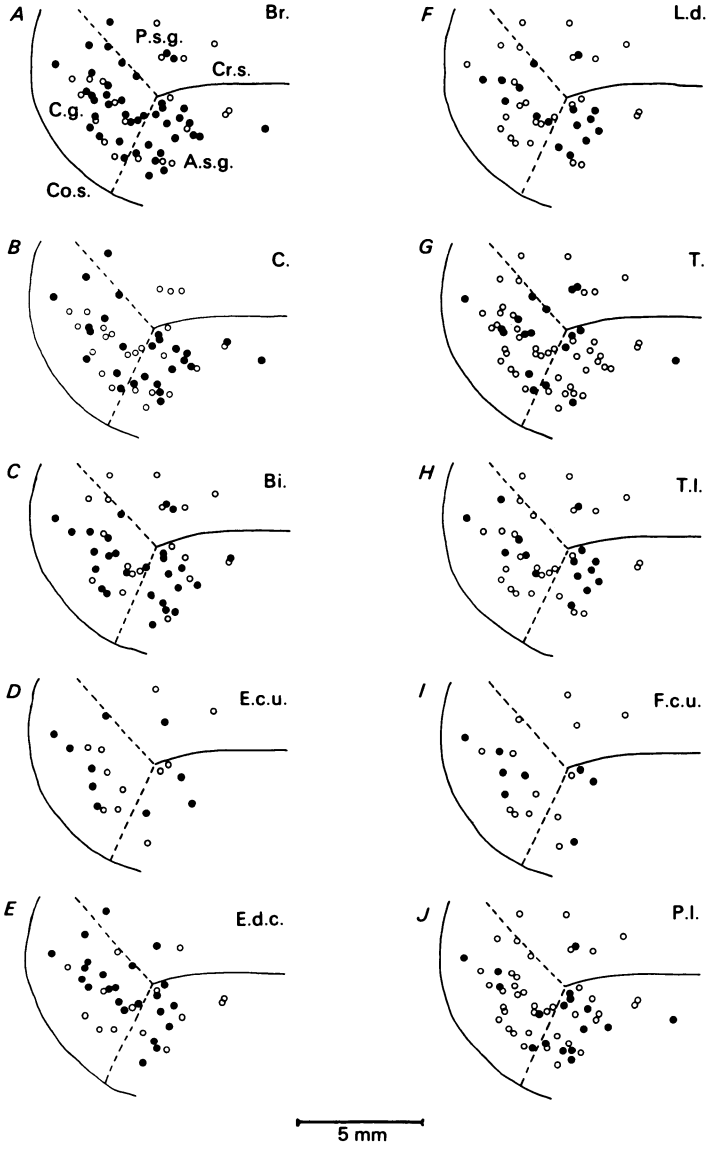

Fig. 2. Maps showing distribution within motor cortex of electrodes from which responses were evoked in different muscles of the contralateral forelimb by trains of 35 μA stimuli. In each case ● indicates an effective electrode, ○ an ineffective electrode. Note that *A–E* are for muscles which are locomotor flexors while *F–J* are for locomotor extensors. Muscle abbreviations as in Fig. 1 and C., cleidobrachialis; E.c.u., Extensor carpi ulnaris. Other abbreviations in *A* apply to all maps: Co. s., coronal sulcus; Cr. s., cruciate sulcus; A.s.g., anterior sigmoid gyrus; P.s.g., posterior sigmoid gyrus; C.g., coronal gyrus. Dashed lines show (arbitrary) lines of demarcation between the different gyri (cf. Livingston & Phillips, 1957; Armstrong & Drew, 1984*b*).

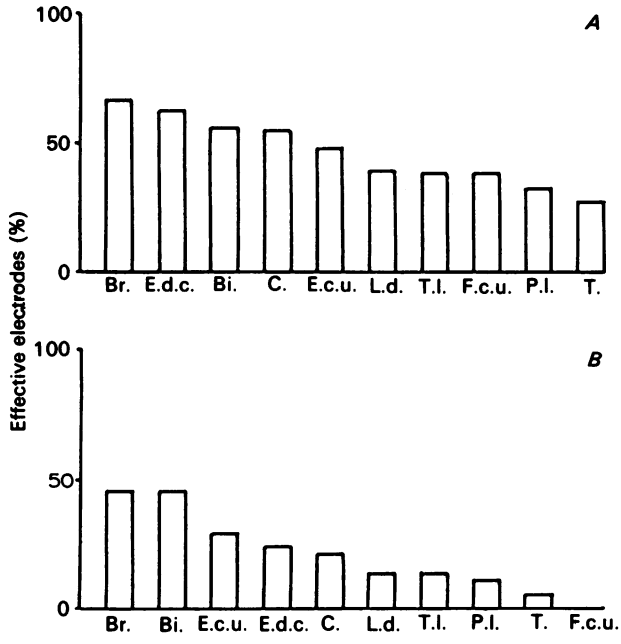

Fig. 3. 'Accessibility' of different muscles of the contralateral forelimb to stimulation in the motor cortex. Each column shows the number of cortical electrodes from which responses were evoked in a particular muscle, expressed as a percentage of the number of electrodes tested. In *A* stimulus intensity was 35 μA; in *B* intensity was 15 μA. Muscle abbreviations as in Figs. 1 and 2.

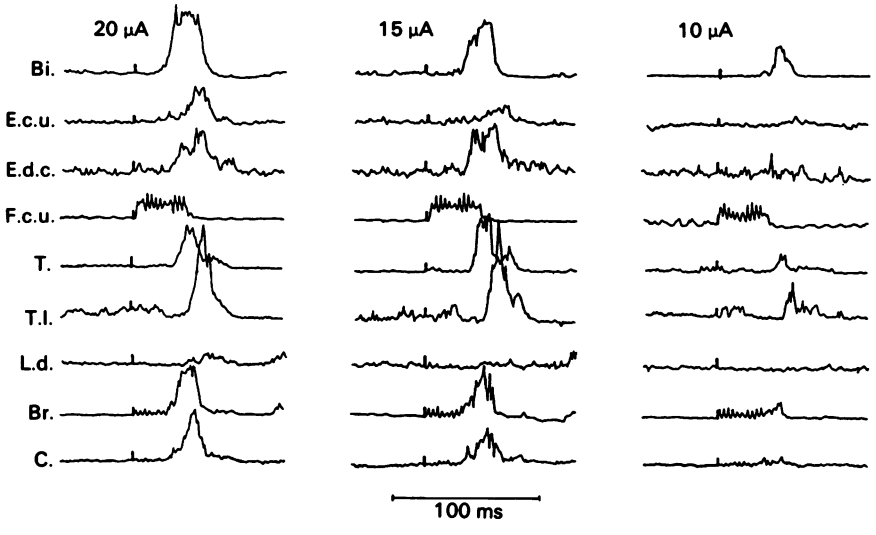

Fig. 4. Effect of reducing stimulus intensity on the rectified averaged e.m.g. responses evoked from one cortical electrode in nine different muscles. Stimulus intensity shown at head of each column of traces. Muscle abbreviations as in Figs. 1 and 2. Note that 'sawtooth' beginning just after stimulus marker in traces for F.c.u. and Br. is an artifact created by the stimulus train. Same time scale throughout.

Responses to near-threshold stimulation

It is of course possible that the location and extent of the electrode distributions from which the muscles could be activated at 35 μA are not entirely a reflexion of the intrinsic functional organization of the motor projections issuing from the cortex. They may also result, at least in part, from physical spread of current within the cortex and/or from indirect (i.e. synaptic) excitation of cortical efferents distant from the electrode tip following direct electrical excitation of cortical afferent fibres or of interneurones within the cortex. These possibilities prompted a further investigation in which for thirty-three of the electrodes (in four cats) stimulus intensity was progressively reduced, usually in 5 μA steps. In general, responses declined progressively in amplitude until at some threshold current they were barely detectable. In some cases latency remained unchanged but quite frequently the reduction in amplitude was accompanied by a progressive increase in latency (see later).

The effects of reducing stimulus intensity are illustrated for one electrode in Fig. 4 which shows the responses of nine different muscles at three different intensities (20, 15 and 10 μA). In some traces (flexor carpi ulnaris and brachialis) a 'sawtooth' series of deflexions just after stimulus onset is an artifact due to the stimulus train. In one muscle (flexor carpi ulnaris) no responses were evoked at any intensity but all the remaining muscles responded at 35 μA (not shown) and also at 20 μA. However, at 10 μA a response was no longer evoked in latissimus dorsi and the responses in three further muscles (extensor digitorum communis, brachialis and cleidobrachialis) were very small. A slight reduction in current abolished these three responses and reduction to 5 μA (not shown) also abolished the responses in biceps brachii, extensor carpi ulnaris and lateral head of triceps, leaving only a (long-latency) response in long head of triceps.

For the majority of electrodes (twenty-two out of thirty-three; 67 %) the threshold for the most excitable muscle or muscles lay between 5 and 15 μA and in a few cases (three out of thirty-three; 9 %) the threshold was less than 5 μA (see for example long head of triceps in Fig. 4); for the remaining eight electrodes (24 %) threshold lay between 15 and 35 μA.

The topographical aspects of the findings made with near-threshold stimulation are displayed in Fig. 5*A–I* in which the filled circles represent those electrodes which were effective on the different muscles at intensities of 15 μA or less, while open circles are electrodes from which no response was evoked at 15 μA. Flexor carpi ulnaris is omitted because no responses could be evoked from any locus unless the stimulus exceeded 15 μA.

These maps reveal that most responses were elicited from electrodes located in two fairly restricted areas within the forelimb motor cortex. One of these 'low-threshold' areas was the most lateral part of the anterior sigmoid gyrus: for each muscle except lateral head of triceps there were at least two electrodes here which evoked responses. A second low-threshold area lay further caudolaterally in the middle of the coronal gyrus. For each muscle there was at least one electrode here which evoked a response and in some cases the area included several effective electrodes (most notably for biceps brachii; see Fig. 5*C*). The cortex sandwiched between the two low-threshold areas (i.e. the rostromedial part of the coronal gyrus) yielded few responses, and only

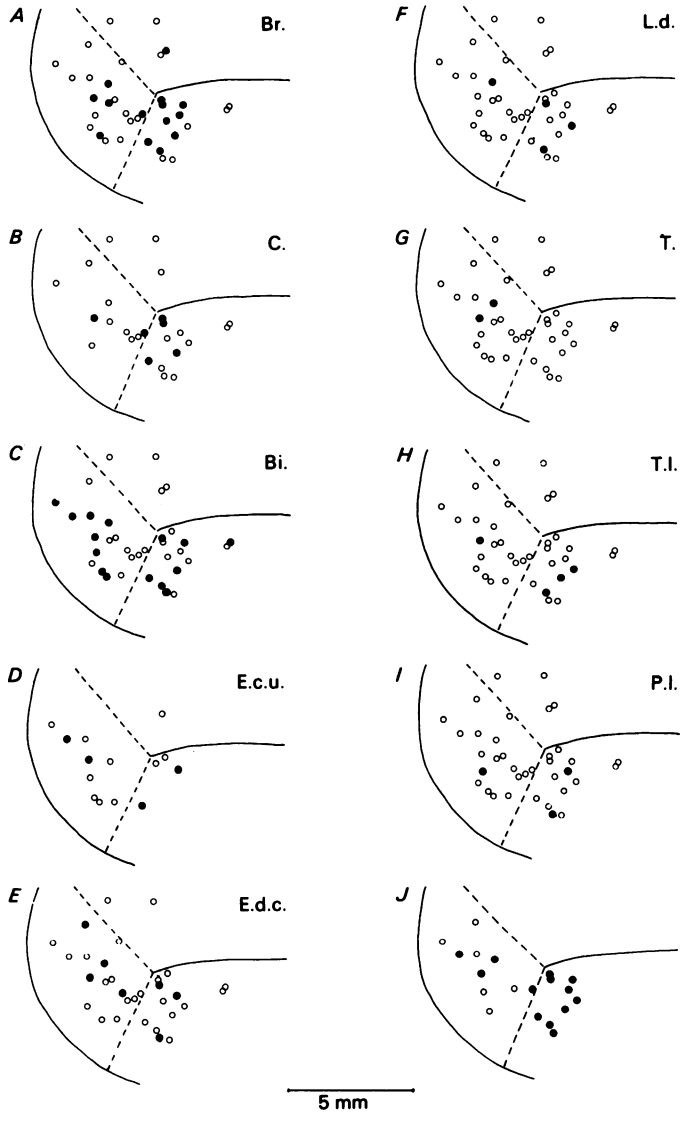

Fig. 5. Maps showing the distribution in the motor cortex of electrodes from which e.m.g. responses were evoked by 15 μA stimuli. *A–I* are maps for individual muscles (cf. Fig. 2) and effective and ineffective electrodes are represented by ● and ○ respectively. *J* maps the distribution of those electrodes which evoked responses in more than one muscle. ○, electrodes evoking responses in two muscles. ●, electrodes effective on three or more muscles. See text.

one response (in brachialis, see Fig. 5*A*) was evoked from the lateral part of the posterior sigmoid gyrus.

In Fig. 5*A–I* some individual effective loci are represented on more than one map because even at 15 μA there were fifteen electrodes which evoked responses in more than one muscle. These are indicated in Fig. 5*J* where open circles represent

electrodes which influenced two muscles and filled circles represent those which influenced three or more muscles. It is clear that there are two well marked clusters of particularly effective loci, one in the lateral part of the anterior sigmoid gyrus and the other in the middle of the coronal gyrus. It is of interest that five of the six electrodes mentioned earlier as exciting seven muscles when 35 μA stimuli were used (see above) are represented in Fig. 5*J*. The sixth of these (which influenced only one muscle at 15 μA) was in the coronal gyrus just lateral to the most lateral electrode in Fig. 5*J*.

In connexion with Fig. 5*J* it may also be noted that of the eighteen e.m.g. responses evoked from the five electrodes making up the caudolateral focus five (28 %) were in locomotor extensor muscles. Of the thirty-one responses evoked from the ten electrodes making up the rostromedial focus seven (23 %) were in the extensor muscles. It appears, therefore, that the average number of muscles influenced per electrode was similar in the two foci and that the ratio of extensor to flexor responses was roughly the same. As regards the 'accessibility' of distal (i.e. forearm) *versus* more proximal muscles, five (28 %) of the responses evoked from the caudolateral focus were in distal muscles; for the rostromedial area seven responses (23 %) were in distal muscles.

With 15 μA stimuli, as at 35 μA, the number of electrodes from which responses could be evoked varied considerably between different muscles. This variation is evident in Fig. 5*A–I* but is better displayed in Fig. 3*B* in which the number of electrodes from which 15 μA stimuli caused activity in each muscle is expressed as a proportion of all the electrodes for which that muscle was studied. Comparison with the similar histogram for 35 μA stimulation (Fig. 3*A*) shows that the rank order for 'accessibility' amongst the different muscles is somewhat changed, but the five muscles which are flexors during locomotion remain clearly more 'accessible' than the five extensors.

Comparison of Fig. 3*A* and *B* shows also that the reduction in stimulus intensity from 35 to 15 μA always reduced the percentage of electrodes from which responses were evoked in any one muscle. However, the reductions were larger for the locomotor extensor muscles than for the locomotor flexors. The least affected extensor was latissimus dorsi (reduction from 39 % to 13·5 %) whereas the flexor most affected was cleidobrachialis (reduction from 55 % to 21 %). The largest reduction occurred for flexor carpi ulnaris (reduction from 39 % to 0 %).

Latency of evoked responses

The latency to onset of the e.m.g. responses evoked using 35 μA stimuli was determined from the rectified averaged traces and the values obtained are shown in the normalized histograms of Fig. 6. Beside each histogram is shown the number of electrode locations involved. For each muscle the range of latencies was quite wide and in some muscles there were a few responses which began after more than 50 ms delay. However, most latencies were less than 30 ms and in all ten muscles the shortest latencies fell within the 10–15 ms bin. In fact they were all within the narrow range of 11–14 ms. The actual values were 11 ms for brachialis and latissimus dorsi, 12 ms for extensor digitorum communis, 13 ms for cleidobrachialis and the two heads

of triceps brachii and 14 ms for biceps brachii, extensor carpi ulnaris, flexor carpi ulnaris and palmaris longus.

All latencies were measured from the first pulse in the stimulus train and although the influence of number of stimuli was not systematically studied it was frequently observed that a minimum of three pulses was required to elicit an e.m.g. response.

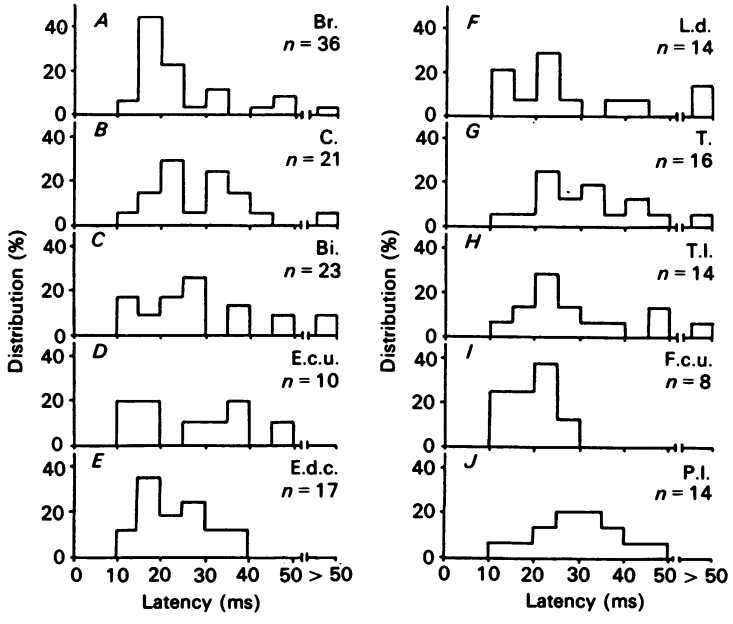

Fig. 6. Frequency histograms for the latencies to onset of e.m.g. responses evoked in individual muscles by cortical stimulation at 35 μA. Each histogram column represents the number of responses in a particular latency range expressed as a percentage of the total number of responses for which latency was measured. Number of responses is shown to right in each histogram. Muscle abbreviations as in Figs. 1 and 2. Note that left-hand column includes locomotor flexors, and the right-hand column locomotor extensors.

This suggests that if the latency values were to be used to estimate conduction time in the pathway mediating the responses then 6 ms should be deducted. Furthermore, it should be noted that the latencies include a conduction time in the motor fibres of the muscle nerve plus a neuromuscular delay.

As regards the topography of response latencies, those electrodes for which latency was not intensity-dependent were invariably within the low-threshold areas shown in Fig. 5. However, these areas included some electrodes for which latency *was* intensity-dependent (i.e. increased as intensity was decreased) and electrodes yielding the shortest latency responses at 35 μA were not always confined to the low-threshold areas. There were no systematic differences between the latencies of responses evoked in individual muscles from the rostromedial and the caudolateral low threshold areas.

Effects of posture on the e.m.g. responses

Because successive responses often fluctuated in amplitude an attempt was made to determine whether posture was likely to be an important influence on response magnitude. Responses observed when the animal was maintained in the position routinely used were compared with those when it was held in the same position but

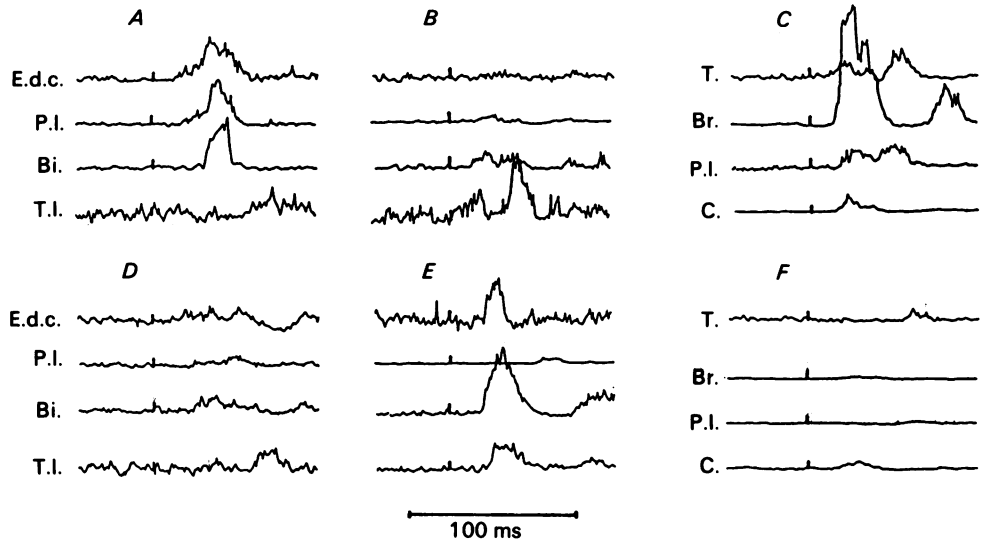

Fig. 7. Effect of postural differences and pyramidectomy on the e.m.g. responses to cortical stimulation. *A* shows rectified averaged responses evoked in four muscles from one cortical electrode by a 35 μA train of stimuli delivered when the animal was in the standard (supported) posture (see text). *D* shows responses evoked in the same muscles by similar trains delivered while all four limbs were load-bearing. *B* and *E* are similar records for stimulation at another cortical locus. *C* and *F* show responses evoked in four muscles from one cortical electrode respectively before and 1 week after pyramidectomy (see Methods). Stimulus train intensity was 20 μA in *C* and 50 μA in *F*. Muscle abbreviations as in Figs. 1 and 2. Time scale below *E* applies throughout.

with the forelimbs and hind limbs in contact with a supporting surface and bearing some of the body weight. For some electrodes the responses in some muscles were unchanged (not illustrated) but more often there were changes of the kind illustrated in Fig. 7*A*, *B*, *D* and *E*. Fig. 7*A* and *B* illustrate responses evoked in four muscles from two different electrodes when the animal was in the standard posture. For comparison Fig. 7*D* and *E* show the responses evoked when the limbs were load-bearing. For the electrode of Fig. 7*A* and *D* the responses in extensor digitorum communis, palmaris longus and biceps brachii were dramatically reduced during load-bearing, while for the electrode of Fig. 7*B* and *E* the response in long head of triceps was reduced but that in biceps brachii was markedly increased; in extensor digitorum communis a substantial response appeared which was previously absent. These findings indicate that many of the responses are heavily dependent on static posture.

Effect of pyramidectomy

In order to investigate the role of the direct corticospinal projection in production of the responses the effects of cortical stimulation were determined in three animals both before and one week after section of the ipsilateral medullary pyramid. In one animal a sham operation produced no change in the latency or threshold of the responses.

Typical results are shown in Fig. 7*C* and *F* in which Fig. 7*C* shows responses evoked in four muscles by trains of 20 μA stimuli delivered pre-operatively to one cortical electrode. Fig. 7*F* shows traces obtained from the same muscles when stronger stimuli (50 μA) were applied via the same electrode after pyramidectomy. For most electrodes and for most muscles 35 μA stimulation after operation was completely ineffective. At intensities around 50 μA responses were occasionally evoked (see Fig. 7*F*, triceps and cleidobrachialis) but they were invariably small and the latency usually exceeded 30 ms.

DISCUSSION

Distribution and thresholds of cortically evoked e.m.g. responses

Our results demonstrate that in the resting animal intracortical microstimulation can discharge α-motoneurones supplying each of the forelimb muscles studied, which included muscles acting at the shoulder, elbow and wrist and on the digits. All muscles were 'accessible' to 35 μA stimuli and all but one (flexor carpi ulnaris) gave responses when stimulus intensity was lowered to 15 μA (or less at some cortical loci). The experiments do not prove conclusively that the responses were mediated either solely or in part via the direct corticospinal projection but the effects of pyramidectomy provide strong evidence for participation of this pathway; it is unlikely that the abolition of all short-latency, low-threshold responses can be attributed to any generalized depression of spinal mechanisms because at the time of testing the animals walked with ease and their general behaviour was virtually indistinguishable from that of unlesioned animals.

The wealth of e.m.g. responses evokable is not surprising because Sakata & Miyamoto (1968) have previously detected a wide variety of flick movements accompanied by e.m.g. responses (see also Armstrong & Drew, 1984*a*). Moreover, in cats sedated with small doses of barbiturate, Asanuma, Stoney & Abzug (1968) were able to evoke e.m.g. responses in eight forelimb muscles, including six of those studied here plus extensor carpi radialis and flexor digitorum profundus. In our experiments responses were found in three additional muscles, namely cleidobrachialis, latissimus dorsi and brachialis muscle.

In the studies by Sakata & Miyamoto (1968) and by Asanuma *et al.* (1968) threshold currents were slightly lower on average than in our experiments, so that in the case of Asanuma *et al.* (1968) all effects studied were produced by currents less than 10 μA. The difference is not, however, unexpected because in those studies the cortical electrodes were tracked systematically through the grey matter in search of low-threshold loci. Moreover, the electrodes were slightly smaller (see Armstrong & Drew, 1984*c*) which should result in a slightly higher current density near the electrode tip.

In the present study there were in fact fifteen cortical electrodes for which eight

or ten muscles were studied and for which threshold was 10 μA or less in at least one muscle. For eight of these electrodes the threshold was distinctly lower for one muscle than for the remaining muscles but in seven cases the weakest effective stimulus evoked responses in two (or more) muscles. In one of these cases three muscles and in another case four muscles displayed the same threshold. By comparison Asanuma *et al.* (1968) found that among 156 loci for which the threshold was less than 10 μA there were 94 for which responses were confined to a single muscle and 62 for which responses were evoked in two or (rarely) three muscles. Inevitably, the extent to which such divergence is observable will be influenced by the number of muscles studied which, for technical reasons, cannot include all those in the limb. It will also be influenced by the excitability of the α-motoneurones and the spinal interneurones interposed between the corticospinal terminals and the motoneurones. For this reason Asanuma *et al.* (1968) manipulated the forelimb to generate peripheral inputs to the spinal motor mechanisms and this indeed resulted in significant lowering of thresholds.

Manipulation was not attempted in our experiments but the absence of sedation may have produced a compensatory increase in the opportunity to observe divergence. In any event, it is clear from both studies that threshold responses to intracortical stimulation quite frequently involve more than one muscle and this accords well with recent demonstrations that individual corticospinal axons frequently possess terminal arborizations sufficiently extensive to permit an influence on several motoneurone pools (Shinoda, Arnold & Asanuma, 1976; Shinoda & Yamaguchi, 1978; Futami, Shinoda & Yokota, 1979).

In the present experiments thresholds were similar for muscles which are flexors or extensors in the locomotor context but flexors were influenced from substantially more cortical electrodes than extensors and this was so whether stimulus intensity was 35 μA (Fig. 2) or 15 μA (Fig. 5). This pattern of differential 'accessibility' is in good correspondence with the fact that hypoflexion of the contralateral limbs is prominent among the behavioural deficits produced in cats by motor cortical lesions (e.g. Adkins, Cegnar & Rafuse, 1971) or by pyramidectomy (e.g. Liddell & Phillips, 1944). The paucity of extensor responses also fits well with observations made when the effects of corticospinal volleys on motoneurone pools were investigated by monosynaptic reflex testing in pyramidal cats (Preston, Shende & Uemura, 1967). Flexor pools uniformly received initial facilitation but the excitability of extensor pools were usually reduced; when extensor facilitations did occur they were usually routinely preceded (and followed) by depression. Inhibitory effects were not of course observable in our experiments but, when microstimulation was applied in walking animals, ongoing locomotor e.m.g. activity was frequently reduced or even abolished at short latency (Armstrong & Drew, 1985). Some of the cortical loci yielding such effects gave no extensor responses in the resting animal.

Perhaps surprisingly, in view of the results of Preston *et al.* (1967), the latencies of those excitatory responses which did occur in extensor muscles were no longer than in flexors. It is theoretically possible that the earliest extensor responses were mediated via fast pathways descending outside the pyramidal tract but this seems unlikely because both flexor and extensor responses were abolished by pyramidectomy. More probably, the balance of excitability between excitatory and inhibitory groups

of spinal interneurones acting on the extensor motoneurones was systematically different between our animals and the anaesthetized pyramidal preparations of Preston *et al.* (1967). Certainly our finding that the responses often showed marked posture dependence does emphasize the importance of the excitability of spinal mechanisms in determining the distribution of responses among the muscles. Changes in motor cortical responsiveness to microstimulation might also play a role here but are unlikely to provide a complete explanation (cf. experiments in man by Berardelli, Cowan, Day, Dick & Rothwell, 1985).

Responses were as frequent among distal muscles (i.e. acting at the wrist and on the digits) as they were among more proximal muscles, and this contrasts markedly with our own findings when *movements* rather than e.m.g.s were studied (Armstrong & Drew, 1984*a*). The relative scarcity of wrist and digit movements is probably not explicable on a basis that flexors and extensors were co-contracted (so as to fix joints rather than move them) because such co-contractions occurred no more frequently among distal than among proximal muscles. Small movements of the digits may sometimes have been overlooked, but the most likely explanation is that many contractions of distal muscles were too weak to produce overt movement.

Response latencies

In the cat, unlike primates, at least one spinal interneurone is interposed between the corticospinal terminals and the α-motoneurones. Some of the interneurones receiving monosynaptic corticospinal input are segmental (Asanuma, Stoney & Thompson, 1971) while others are propriospinal neurones which lie in segments C3–C4 and have axons descending in the ventrolateral part of the lateral funiculus (Illert, Lundberg & Tanaka, 1977; Illert, Lundberg, Padel & Tanaka, 1978).

The responses evoked in each muscle from different cortical loci ranged widely in latency (see Fig. 6) but minimum latencies varied little between muscles. The earliest responses (in brachialis and latissimus dorsi) had a latency of 11 ms and the other muscles yielded values of 12, 13 or 14 ms. These values are slightly less than the approximate 20 ms quoted by Asanuma *et al.* (1968) and by Sakata & Miyamoto (1968) but the *majority* of our latencies were close to this value.

Allowing *ca.* 1·5 ms for conduction of the fastest corticospinal impulses to the cervical cord and a peripheral delay of *ca.* 3 ms (for conduction in the motoneurone axons plus a neuromuscular delay), an interval of 6·5–9·5 ms remains for intraspinal events leading to the earliest e.m.g. responses. However, when the number of stimulus pulses was varied, 3 was the minimum required. The third pulse occurred 6 ms after the first so the intraspinal delay available in respect of that pulse would be approximately 0·5 ms for brachialis and latissimus dorsi and 1·5–3·5 ms for the other muscles. These estimates are very approximate but if the earliest responses were mediated via the corticospinal tract (as appears probable from the results of pyramidectomy) then some are likely to have been mediated via the disynaptic pathway mentioned above. The requirement for 3 stimuli agrees well with the finding by Illert, Lundberg & Tanaka (1976) that 3 pyramidal volleys are required to discharge the C3–C4 propriospinal neurones. That many responses had latencies compatible with a more complex pathway is not surprising because the short latency effects of pyramidal volleys on α-motoneurones are much weaker than longer-latency

(presumably polysynaptic) effects (Agnew, Preston & Whitlock, 1963; Preston *et al.* 1967; see also Phillips & Porter, 1977).

Cortical topography for the e.m.g. responses

For each muscle, contractions were elicited at 35 μA from loci quite scattered across the forelimb motor cortex. This emphasizes the functional complexity of the motor representation, as also does the fact that sites effective on one muscle were intermingled with ineffective sites (which nevertheless mostly yielded responses in one or more other muscles). The complexity does not end here because the e.m.g. investigation was confined to sites which produced forelimb movements and these were intermingled with a substantial number of others which evoked no movements. When stimulus intensity was reduced to 15 μA (presumably reducing stimulus spread) a tendency was revealed for responses to be most readily evoked either from a rostromedial 'focus' in the lateralmost part of the anterior sigmoid gyrus or from a more caudolateral 'focus' in the middle of the coronal gyrus. This finding is in agreement with those of Pappas & Strick (1981) who found in ketamine-anaesthetized cats that these two locales constitute separate low-threshold regions for evoking contractions of digit muscles. Pappas & Strick (1981) could not usually find a double representation for more proximal muscles (except occasionally for wrist or elbow muscles) but, perhaps because of the absence of anaesthesia, we found that double representation was evident for proximal as well as distal muscles.

In both locales there were some electrodes which evoked responses in several muscles (see for example Fig. 5 *J*) but equally not all muscles were influenced from any one electrode. This suggests that, although the low-threshold representations for the different muscles certainly overlap, they are not precisely co-extensive. Unfortunately our experiments permit no more exact conclusion because a relatively small number of fixed electrodes was used and it was necessary to pool results from several animals. Pappas & Strick (1981) have shown that the surface landmarks in the cat pericruciate region are sufficiently variable that pooling is likely to blur maps made to reveal functional subdivisions within the cortex. However, any blurring should have acted approximately similarly on our maps for different muscles. Moreover, although we used pooling to demonstrate the two low-threshold areas their existence was confirmed in individual animals because effects on some muscles were obtained from within both areas but not from loci interposed between them.

The data of Pappas & Strick (1981) show (see their Fig. 2 *C* and *D*) that when double representations were present both for the digits and for another joint they did not overlap. It seems likely, therefore, that further investigation in the absence of anaesthesia will reveal a pattern of double representation in which the areas related to different joints overlap at their edges but not at their centres.

Pappas & Strick (1981) found essential similarity between their rostromedial and caudolateral low-threshold zones in that both flexors and extensors of the digits were represented in each zone, as also were the extrinsic and intrinsic muscles acting on the digits. This similarity is paralleled in our own findings. Thus, each of our low-threshold regions influenced both proximal and distal muscles and the proportional representation of forearm and more proximal muscles was roughly similar. Also, each area had 'access' to muscles, both flexor and extensor in the locomotor context, and in each area the flexors were collectively about four times as accessible to 15 μA

stimuli as extensors. In addition, response latencies were not significantly different between the two areas.

Collectively, our findings and those of Pappas & Strick (1981) strongly suggest that a genuinely double representation of excitatory efferent function exists within the motor cortex in the cat, and it is interesting to note that a double motor representation of hand and wrist has also been reported for the monkey (Strick & Preston, 1978*a*). However, well known problems of interpretation arise when electrical stimuli are applied to the motor cortex (see for example discussions in Asanuma, Arnold & Zarzecki, 1976; Phillips & Porter, 1977) and it is therefore of great interest that other evidence exists to suggest a rostrocaudal duality of function.

Ghez & Yumiya (1984) have provided anatomical evidence for differences in afferent projections to the rostral and caudal areas and Vicario, Martin & Ghez (1983) have found that whereas cells with 'simple', well localized, temporally stable peripheral receptive fields are scattered throughout the motor cortex, cells exhibiting temporal lability, directional specificity and other 'complex' receptive field properties are preferentially located rostral to the cruciate sulcus. This was also the preferred location for 'lead' cells which discharge in advance of volitional movements of the elbow and wrist. 'Lag' cells discharging only after movement onset were present throughout the motor cortex and usually had simple receptive fields.

In the monkey, also, there are rostrocaudal differences between the receptive field properties of motor cortical neurones: caudally, cutaneous receptive fields predominate heavily over deep fields while the reverse is true rostrally (Strick & Preston, 1978*b*; Tanji & Wise, 1981).

What the precise significance of a dual organization may be for movement control has yet to be determined, but it has been reported that ablations of the anterior and posterior sigmoid gyri produce different patterns of motor deficit in the cat (Adkins *et al.* 1971). Vicario *et al.* (1983) have suggested that the rostral area may be involved in movement initiation while the caudal part utilizes specific somatosensory inputs to achieve regulation of ongoing movements. A functional differentiation of this nature is certainly compatible with our finding that the two low-threshold areas have similar patterns of 'access' to the forelimb musculature.

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