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Further investigations of the efferent linkage of the supplementary motor area (SMA) with the spinal cord in the monkey

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Summary. Intracortical microstimulation of the supplementary motor area (SMA) was studied in awake monkeys. With short trains of micropulses, contralateral muscle twitches mainly in shoulder and proximal arm muscles were elicited. There was an indication of a rostro-caudal representation of distal to proximal forelimb, trunk, and proximal to distal hindlimb muscles. However, an intermingling of efferent zones was much more prominent as compared to the precentral motor cortex (MI). All efferent zones to the spinal cord were clustered in the caudal half of the SMA, and we failed to detect face and ocular movements (except at one stimulation site) when microstimulating the rostral portions of the SMA. Single micropulses were also injected in efferent zones of the microexcitable cortex in order to investigate post-pulse facilitation of sustained EMG activity. For motor cortex (MI) stimulation, post-pulse facilitation was prominent and observed in 14 of 17 tested stimulation sites. The incidence of facilitation in comparable muscles obtained with SMA stimulation was only 20 out 54 tests. The onset latencies of EMG modulation obtained from the two areas were in the same range but the amount of modulation in the SMA was less conspicuous than in MI. These results indicate that the SMA has oligo- or possibly even monosynaptic connections with motoneurones, but that these connections are less dense than those from MI.

Key words: Supplementary motor area – Microstimulation – Somatotopy – Monkey

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Introduction

The supplementary motor area (SMA) of Penfield and Welch (1951) is located on the mesial wall of the hemisphere just in front of the motor-cortical hindlimb and tail representation. Recent anatomical and electrophysiological studies (Biber et al. 1978; Murray and Coulter 1981; Macpherson et al. 1982a, b) have shown that the SMA contains neurones projecting directly to the spinal cord. Electrical surface stimulation studies by Woolsey and collaborators (1952) revealed a rostro-caudal somatotopy in the SMA similar to the medio-lateral somatotopy found in the motor cortex. In their map, the SMA hindlimb representation undershoots the motor cortical hindlimb area. In a reinvestigation, it was shown that most of the motor effects obtained by surface stimulation of the SMA were mediated via the motor cortex suggesting a somatotopical projection from the SMA to the motor cortex (Wiesendanger et al. 1973). Macpherson et al. (1982a) obtained motor effects by intracortical microstimulation (ICMS) mainly in proximal musculature using currents of 30 μA or less. With conventional parameters of ICMS (12 stimuli at 330 Hz), as described by Asanuma and Rosén (1972), temporal and spatial summations are prominent (Jankowska et al. 1975) which complicate the interpretation of ICMS effects. In order to avoid this problem, we have investigated the modulation of ongoing EMG activity produced by single electrical micropulses applied intracortically. This technique was first introcuded by Cheney and Fetz (1978; cf. also Cheney and Fetz 1985 and Cheney et al. 1985) who showed that single pulse stimulation of the motor cortex results in post-stimulus modulation profiles of the EMG activity, comparable to those seen after spike-triggered averaging when action potentials of motor-cortical cells were used as a trigger (Fetz and Cheney 1978,

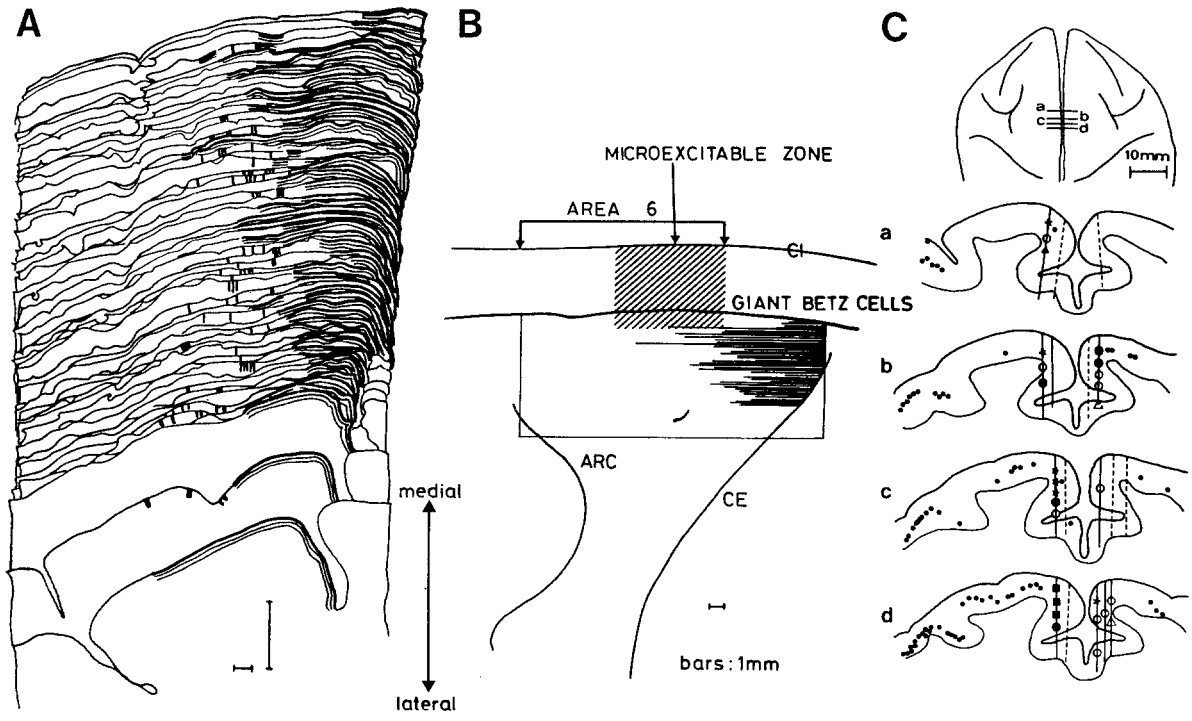

Fig. 1. **A** Histological reconstruction of border between medial area 4 with the large Betz cells (thick lines) and area 6. In the transition zone scattered Betz cells are also shown. Parasagittal sections. **B** Schematic representation (with unfolded mesial cortex) of approximate extent of the microexcitable zone from which forelimb effects were obtained by train ICMS (hatched area) in relation with the extent of mesial area 6. The sections with large Betz cells are marked with horizontal lines projected to the lateral surface. Monkey with multiple indwelling EMG electrodes. **C** Reconstruction (from another monkey) of stimulating tracks on 4 representative transverse sections (a–d), the position of which are also shown on the dorsal surface of the brain. The presence of Betz cells are marked with dots. Symbols: ● effects with train and single pulse ICMS in proximal forelimb muscles; ○ effects only with train ICMS; ▲ and △ same as above for distal forelimb muscles; ★ hindlimb ICMS effects; ■ trunk ICMS effects. ARC: arcuate sulcus, CE: central suclus

1980). The aim of the present study was to investigate the characteristics of the projections from the SMA to the spinal cord by the new technique of single stimulus-triggered averaging, thus avoiding the excessive intracortical spread of the stimulus inherent in the method of train stimulation. In particular, it was expected that the onset latencies of EMG modulation could provide a more accurate measure for the synaptic linkage of the SMA with spinal motoneurones than the latencies obtained with train stimulation. Since, in a previous study (Macpherson et al. 1982a, b), a rostro-caudal somatotopy was not evident, we also re-addressed the problem of somatotopical representation of the SMA.

Material and methods

Experiments were performed on 8 hemispheres of 5 Macaca fascicularis monkeys. The animals were first accustomed to sit in a primate chair. Under deep barbiturate anaesthesia, the SMA and MI (for another experiment, also the premotor cortex) were exposed, with the dura intact, and a large rectangular chamber was implanted. After a short period of recovery, daily microstimulation sessions were begun with the monkey's head fixed atraumatically by means of the semichronic head holder device of D. Kopf. The penetrations were performed systematically with each track 0.5 mm apart, following a grid system with a mark on the chamber as reference. Usually, the SMA of both sides and the precentral cortex on one side were investigated. The same monkeys served also for single unit recordings (to be reported separately). As a first step, trains of 12 constant cathodal pulses (0.2 ms, 330 Hz) were applied to elicit overt motor responses. The location of the twitches and the thresholds were examined by at least two observers. Moreover, in most cases, intramuscular electromyographic (EMG) recordings were photographed as superimposed traces from the oscilloscope. An arbitrary upper limit for positive intracortical microstimulation (ICMS) was set at 30 μA (cf. also Asanuma and Rosén 1972). However, higher currents were often used to search for ICMS sites. At all locations where positive ICMS effects could be obtained, we next used the technique of single pulse stimulation (2000 pulses, 8/s; Cheney and Fetz 1985). Each stimulus was taken as a trigger to average the ongoing EMG recorded from the muscle which had shown a response with train stimulation. The respective limb of the monkey was held in positions which resulted in a sustained EMG activity.

The EMG activity was recorded by pairs of fine needle electrodes (insect pins) which were insulated to about 2 mm from the tip. The amplified and filtered signal (100 Hz–5 kHz) was usually rectified and averaged at a bin width of 100 μs (5 ms prestimulus time and 30–45 ms poststimulus time). Stimulus intensity was first set at 30 μA and, if positive effets were seen,

more averages were obtained with successively lower intensities (in steps of 5 or 10 μA). At several occasions, when prolonged periods of stimulation had been used, it was ascertained that unchanging single unit activity could be recorded with the microelectrode at the stimulation sites suggesting that prolonged stimulation with single micropulses did not produce significant damage to the cortex. In one monkey, 7 pairs of wire electrodes (1 cm apart) were permanently implanted in the following muscles: wrist flexors, wrist extensors, brachialis, triceps, deltoid, trapezius, pectoralis major. The leads were multi-stranded stainless steel wires, insulated with teflon. The wires were tunneled under the skin to a socket cemented on the skull. During experimental sessions, connection to the amplifier was established via a multilead cable. With a selector switch, one of the EMG channels could be displayed on the oscilloscope together with the stimulating current. Five-times superimposed sweeps from the 7 channels could be recorded with a Grass camera in quick succession. This monkey served mainly for a closer analysis of the distribution of muscle activation as obtained by train stimulation from one particular efferent zone. The results were also used, together with the train stimulation results obtained from the other monkeys, to reinvestigate the problem of rostro-caudal somatotopy.

A typical session lasted for about 2 h. At the end of the experiments, some important regions of exploration were marked with Alcian blue. The animals were then deeply anesthetized and perfused with 10% formaldehyde. After postfixation of a block of the brain containing the motor cortex and the SMA, 50 μm coronal or parasagittal sections were cut for histological reconstruction of the stimulation tracks. In order to classify a track as passing through area 4 or area 6, it was necessary to rely on cytoarchitectonic criteria. Area 4 is characterized by the presence of giant Betz cells, whose density declines in the rostral direction. In the present experiments, the most difficult problem was to decide whether stimulation sites with hindlimb effects were in the SMA or in the hindlimb representation of the motor cortex. These two areas may even overlap in rostro-caudal direction (Woolsey et al. 1952; but see also Wise and Tanji 1982) which even more complicates the allocation to one or the other area in the medial cortex (cf. Results).

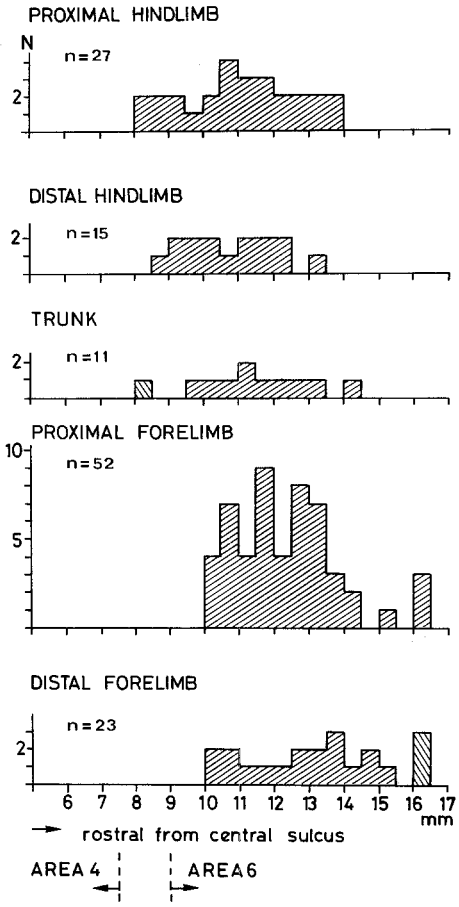

Fig. 2. Topographical representation of ICMS effects along the rostro-caudal axis of areas 4 and 6. The occurrences of positive motor effects with train stimuli below 30 μA are plotted as histograms for the various muscle groups. Note that inspite of a broad overlap of representation, there is a trend of a successively more rostral representation from the hindlimb muscles to the distal forelimb muscles. The ICMS effects were limited to the caudal part of medial area 6

Results

1. Histological reconstruction of positive ICMS sites in the SMA

In all animals it was possible to reconstruct the microelectrode tracks on the basis of an enlarged photograph of the brain surface with a number of surface and depth marks, the protocol of the grid of tracks, and the histological plots of the electrode tracks and marking tracks. As in previous experiments, we found that the microexcitable zone of the SMA was limited to the posterior portion of medial area 6. The posterior border of this zone was 7.5–10 mm rostrally from the central sulcus as it joins the midline, and it extended 6–8.5 mm in anterior direction (Figs. 1 and 2). Motor effects were rarely seen at levels more rostral to the arcuate spur. Some scattered Betz cells were seen occasionally in the microexcitable zone from where forelimb effects were obtained. Figure 1 shows a reconstruction of the Betz cell region in serial parasagittal sections (A) and the relative position of the microexcitable region from where forelimb effects were recorded in the animal with multiple EMG electrodes implanted (B). On the right of Fig. 1 is a schematic reconstruction of microstimulation effects on four representative coronal section from another monkey (C).

2. Somatotopical organization of the SMA as revealed by intracortical train stimulation

Figure 2 provides an overview of all positive sites of ICMS. In all animals, the medial cortex was systematically explored from the motor cortex far into the rostral-medial cortex where the agranular cortex joins the granular cortex of area 8. The general

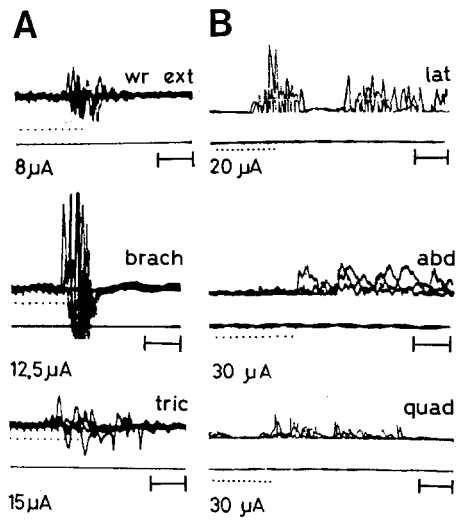

Fig. 3. A Examples of wide-spread motor effects obtained by train stimulation at one site in the SMA. The EMG was monitored from 7 different muscles by means of chronically implanted wire electrodes. Positive effects were seen at lowest intensity in wrist extensors (wr ext); at only slightly higher intensity of ICMS, motor units of the brachialis muscle (brach) and of the triceps muscle (tric) were also recruited. No responses were obtained with the other electrodes implanted in wrist flexors, trapezius, deltoid and pectoralis. Calibration 10 ms, 5 × superimposed traces. **B** Wide-spread effects obtained from one site of train stimulation in the SMA. At lowest intensity, prominent response in the latissimus dorsi muscle (lat); at higher intensity of ICMS, recruitment of rectus abdominis (abd) and of quadriceps (quad) muscle of the hindlimb. Rectified EMG, 3 × superimposed traces, calibration 10 ms

observation described previously (Macpherson et al. 1982a) was confirmed, namely that there is a considerable overlap of forelimb and hindlimb and of proximal and distal representation along the rostro-caudal axis of the medial cortex. Nevertheless, the histograms of Fig. 2 plotted on the rostro-caudal axis revealed a tendency of a rostro-caudal organization for the different muscle groups. Thus, exclusively forelimb responses were seen when stimulating the rostral-most portion of the microexcitable cortex. In spite of the rather pronounced intermingling of topographical zones, the sites for a particular motor response were sharply localized. We paid particular attention to face and ocular movements when exploring the rostral portion of the SMA. Saccades could be elicited at one single site only of the rostral SMA (not plotted in Fig. 2). As can be seen in the histograms of Fig. 2, the majority of motor responses were seen in forelimb muscles, mainly proximal ones, but distal forelimb muscles and hindlimb muscles were also represented. We failed to detect any ipsilateral limb movements. It is likely that the 27 efferent zones controlling proximal hindlimb muscles were really in the SMA since medial area 4 represents distal hindlimb muscles and tail muscles. We are not sure, however, that all of the distal hindlimb efferent zones were in the SMA since some of the tracks turned out to be in the border region between area 4 and area 6. Thus, the allocation of the distal effects to the SMA are somewhat arbitrary. The occurrence of responses in the trunk muscles was relatively low, but positive effects may well have escaped observation since the back of the animal was not visible and hardly accessible to palpation.

Figure 3A illustrates an example of recording EMG responses in the monkey with seven indwelling EMG electrodes. At that particular stimulation site in the SMA, a proximal forelimb muscle (triceps) was activated together with a distal muscle not acting on the same joint (wrist extensor), an observation which was already described previously (Macpherson et al.

Table 1. Comparison of motor response latencies in different muscle groups obtained from area 4 and from the SMA. For both areas, the latency values are listed for train stimulation on left columns and for single pulse stimulation on right columns (not tested in every case of train stimulation). The incidence of negative modulation responses are also listed

	Latencies of motor effects (in ms)			
	Area 4		Supplementary Motor Area	
Muscle groups	Train	Single pulse	Train	Single pulse
Shoulder	15–33 (n = 16)	5.5–7.0 (n = 3)	12–66 (n = 31)	6.25–12.0 (n = 9) 15 × negative
Upper arm	12–27 (n = 14)	5.0–7.5 (n =4)	12–36 (n = 11)	6 × negative
Forearm	8–32 (n = 17)	6.0–6.25 (n = 3)	18–48 (n = 12)	6.2–7.8 (n = 6)
and Hand		1 × negative		5 × negative
Trunk	18 (n = 1)	7.0 (n = 1)	18–35 (n = 5)	5 × negative
Tail	20–43 (n = 2)	—	—	—
Hip	15–33 (n = 2)	1 × negative	18–24 (n = 3)	10.0 (n = 1) 2 × negative
Ankle	12–33 (n = 2)	12–14.5 (n = 3)	15–24 (n = 5)	10–15 (n = 4)
and Toes		1 × negative		1 × negative

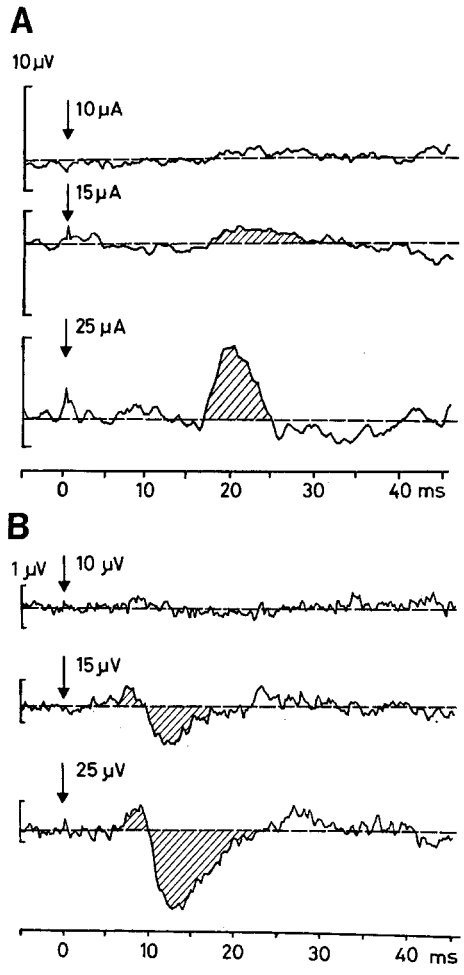

Fig. 4. **A** Post-stimulus facilitation profiles of the anterior tibial EMG obtained with single pulse stimulation of the hindlimb representation of area 4 while the monkey exerted a sustained contraction of the hindlimb muscle. Threshold of facilitation was at 15 μA and a sharp peak was building up at successively higher intensities of 20, 25 (illustrated) and 30 μA. The maximum 'peak-to-noise ratio' (providing an estimate of the strength of the modulation; cf. Cheney and Fetz 1985) was 5.1 at 30 μA. Averages of 2000 stimuli at 8/s. **B** EMG recording in a distal forelimb muscle (flexor digitorum communis) and single micropulse stimulation of the precentral hand area. A weak early facilitation (onset 6.5 ms) was followed by a prominent suppression which was maximal at 25 μA. The 'peak-to-noise ratio' was 1.7 for the facilitation and 5.3 for the suppression. Same procedure as in **A**

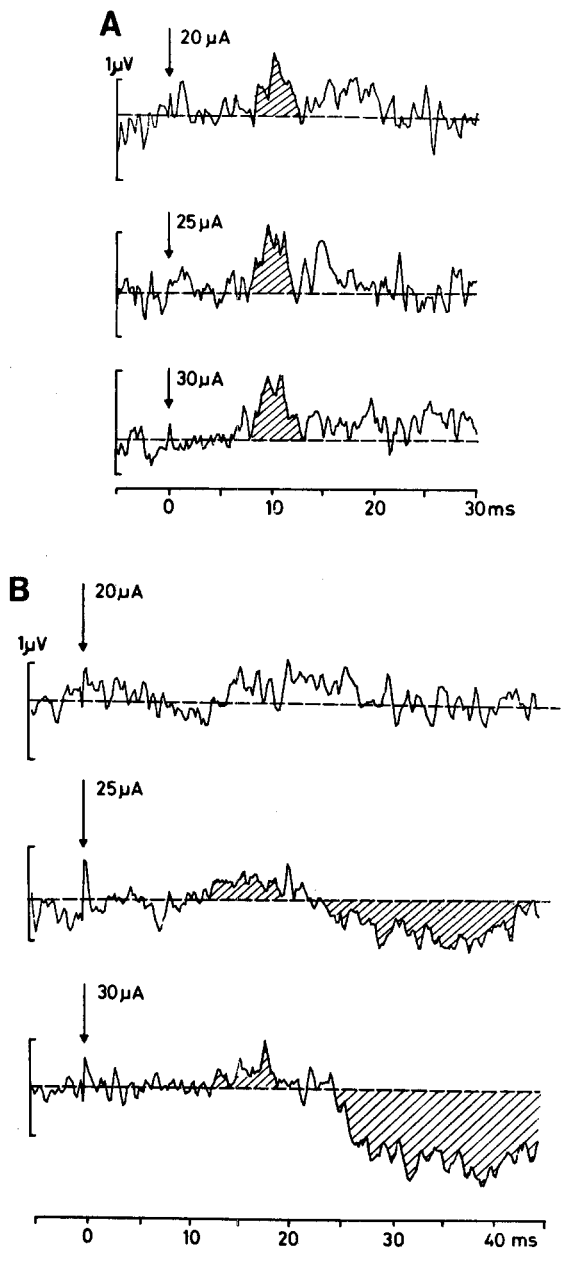

Fig. 5. **A** Single pulse ICMS in an efferent zone of the SMA and recording of sustained EMG activity in a distal forelimb muscle (flexor digitorum communis). A small, but consistent peak of facilitation appears in the noisy averaged trace. The 'peak-to-noise ratio' was about 2 at 30 μA. **B** Single pulse ICMS in the SMA and recording of sustained EMG activity in the anterior tibial muscle. A clear suppression with a 'peak-to-noise ratio' of about 2 appears at a long latency of 20 ms which was possibly preceded by a weak facilitation at 12 ms

1982a), but without EMG documentation. Figure 3B illustrates an efferent zone with the lowest threshold and largest response in the latissimus dorsi muscle. With only slightly higher intensity of stimulation, abdominal muscles and proximal hindlimb muscles were also recruited.

With train stimulation, the latencies of EMG responses were typically longer than 15 ms, even for proximal forelimb muscles (Table 1). This may partly be due to the slow conduction velocity of corticospinal neurones of the SMA (Macpherson et al. 1982b). However, the need of temporal summation in a multisynaptic chain of neurones may be a more important factor. This problem was further analyzed by means of single pulse stimulation as described below.

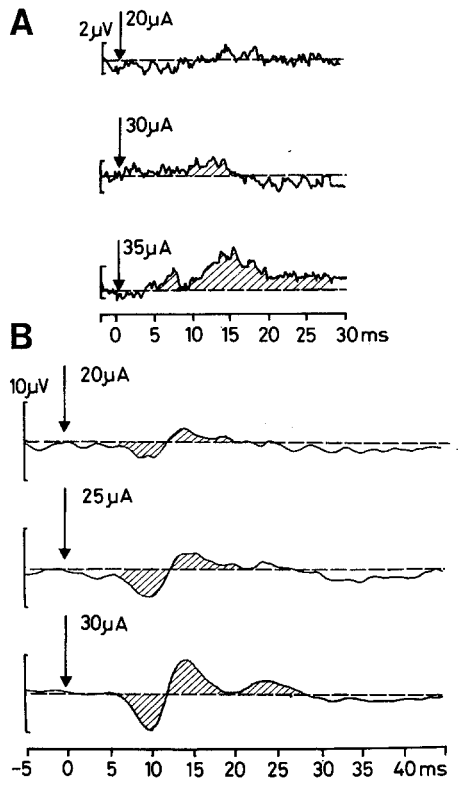

Fig. 6. **A** Example of a weak post-pulse facilitation in the deltoid muscle obtained by SMA stimulation. A consistent peak with a 'peak-to-noise ratio' of 2 was building up at an intensity slightly above 30 μA. **B** Example of an unusually prominent modulation obtained by SMA stimulation and recorded from the pectoralis muscle. The onset latency was 6.25 ms and the 'peak-to-noise ratio' 3.5. In this case, the average was from non-rectified EMG

3. The coupling of the SMA with spinal motoneurones as revealed by postpulse modulation of sustained EMG activity

Table 1 provides a summary of all experiments in which single pulse stimulation was used. In all of these stimulated sites, train stimulation had been shown to produce overt motor responses at threshold of 30 μA or less. In the motor cortex (area 4), single micropulses were applied at 17 sites and in 14 of them clear-cut modulations of the averaged tonic EMG activity was obtained. An example of a pronounced post-pulse facilitation of background EMG activity in the anterior tibial muscle is shown in Fig. 4A. The onset of this facilitation was sharp and occurred at 15 ms; the threshold was betwen 10 and 15 μA and the amount of facilitation progressively increased with higher intensities. Figure 4B is a postpulse facilitation-inhibition sequence which was obtained from the precentral hand area. The threshold response in the finger flexors was again between 10 and 15 μA, and the largest modulation occurred at 25 μA stimulus intensity.

By contrast, a much smaller proportion of ICMS sites in the SMA yielded also positive evidence for post-pulse modulation: out of 54 tested sites, only 20 were positive and the amount of modulation was less pronounced. Examples of a weak modulation obtained in the SMA are illustrated in Fig. 5. 'Noisy' facilitatory effects in a distal arm muscle and predominantly inhibitory effects in a distal hindlimb muscle are shown in Figs. 5A, B respectively. Occasionally, a rather prominent modulation was also seen with SMA stimulation. Figure 6 compares the modulation curves obtained in two shoulder muscles, showing weak effects in A and strong effects in B. The maximum strength of post-stimulus facilitation or suppression by single pulse of 30 μA applied to the SMA was estimated as 'mean percent modulation'

$$\left(\frac{\text{mean peak height} - \text{mean base line}}{\text{base line}} \times 100;\right.$$

Cheney and Fetz 1985).

The value for 6 measures were: + 5.8%, + 6.6%, + 10.5%, + 12.2%, +19.2% and −14.9%. In the remaining cases, the strength was estimated from the 'peak-to-noise ratio', measured in the graphic records of the averages (height of facilitation or suppression peak divided by the largest baseline fluctuation; Cheney and Fetz 1985). The average modulation strength thus established in 14 cases amounted to 2.3 (SD ± 0.9 range 1–4). In the motor cortex, the corresponding average value for the 'peak-to-noise ratio' was 5 (SD ± 3.9, range 1–14).

The small sample of positive effects does not permit statistical comparison of latencies obtained in the two motor fields. The latencies of modulation onset were, however, remarkably short in the SMA (6.25–12 ms for forelimb muscles and 10–15 ms for hindlimb muscles) and were in the same range as those seen in area 4.

Discussion

It is clear since the early stimulation studies on the cerebral cortex that the SMA of medial area 6 is part of the 'excitable' cortex. Often the evoked movements were complex and were elicited only at relatively high intensity of stimulation (Penfield and Welch 1951). This renders interpretation difficult as to the linkage of the SMA with the spinal motoneurones. Following unsuccessful attempts to obtain more discrete microstimulation effects from the SMA (Smith 1979; Wise and Tanji 1981), it was found that motor effects may be elicited by ICMS from the

posterior portion of the SMA (Macpherson et al. 1982a). This suggested that the SMA has a fairly direct access to the spinal cord, a notion which was also substantiated by the discovery of corticospinal neurones in the SMA (Biber et al. 1978; Murray and Coulter 1981; Macpherson et al. 1982a, b). The corticospinal neurones in the SMA are, however, relatively sparse and of small calibre (Murray and Coulter 1981; Macpherson et al. 1982a, b), and their exact site of termination in the spinal grey matter is presently unknown. On the other hand, the SMA has strong connections with precentral area 4 and may thus influence motoneurones via this cortico-cortical link. Finally, the SMA is also likely to influence the spinal cord via subcortical relays. Since intracortical train stimulation results in considerable temporal and spatial summation, the observed ICMS effects may well have involved a number of synapses along the above-mentioned indirect routes.

With the present investigation by means of single ICMS pulses, we sought to resolve the question of how tight the SMA is coupled with the spinal motoneurones, the rationale being that single pulses are less likely to activate corticospinal neurones transsynaptically. The reported positive evidence for a post-pulse modulation in hindlimb and forelimb muscles with single pulse ICMS of the SMA indeed suggests an oligosynaptic, possibly even monosynaptic linkage to motoneurones. However, post-pulse modulation was much less frequently observed in the SMA than in the precentral motor cortex. Furthermore, the amount of modulation was in most instances modest and 'noisy'. This is most likely explained by the sparse distribution of corticospinal cells in the SMA as compared to MI.

We suggest that the more striking motor effects seen with repetitive surface stimulation (Woolsey et al. 1952) or with longer trains and higher intensities of ICMS (Mitz 1985) are likely to be mediated by indirect pathways, including the link via the motor cortex. In this context, it is interesting to note that with long trains of ICMS and current intensities not limited to 30 μA, Mitz (1985) had no difficulty to evoke face and ocular movements. In addition, a more clear-cut somatotopy in the rostro-caudal direction was found than we did with shorter trains limited to 30 μA current intensity. This raises the question whether much of the rostro-caudal somatotopy revealed in electrical stimulation studies (Woolsey et al. 1952) and single unit studies of the behaving monkey (Brinkman and Porter 1979; Tanji and Kurata 1982) may be explained by the cortico-cortical connections of the SMA with the motor cortex which have been found to be arranged somatotopically in the rostro-caudal axis of the SMA (Muakassa and Strick 1979; Matsumura and Kubota 1979). This problem needs further clarification.

In recent years, the 'higher' role of the SMA in movement initiation has been advocated, mainly on the basis of cerebral blood flow and lesion studies on the human brain (reviewed in Wiesendanger 1986). In an anatomical sense, the SMA may indeed be regarded as being 'up-stream' to the motor cortex. However, the presence of corticospinal neurones in the SMA and the findings reported in this study also indicate that the SMA exerts a control on the motor apparatus *in parallel* with the motor cortex. Besides its alleged 'higher' role in movement initiation, the SMA is therefore susceptible to be involved also in a more direct control of movement execution.

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