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ABSTRACT

The afferent evoked electrical response in precentral and postcentral cerebral cortex was studied in anesthetized and unanesthetized monkeys. Responses to nerve volleys were obtained on both sides of the central sulcus, tactile evoked responses were limited to the contralateral postcentral gyrus. Precentral responses were evoked by stimulation of the fast cutaneous and slow muscle afferent fibers. The amplitude and distribution of precentral responses varied with the number of peripheral nerve fibers synchronously activated. The latency of precentral responses increased as a function of distance from the central sulcus. Precentral and postcentral potentials were altered in the same manner by topical application of Novocaine and strychnine and the spreading depression of Leão. The nature of the afferent precentral electrical response, the anesthetic conditions for its study and the possible functional role of this projection are discussed.

WITH THE DEVELOPMENT of modern electrophysiological techniques, it has been possible to produce a map of the region of the cerebral cortex from which a synchronized action potential can be evoked by activation of tactile receptors (1–3). These detailed maps obtained by mechanical displacement of a few hairs indicate that only a portion of the postcentral gyrus and the frontoparietal operculum in the primate is activated under these conditions. However, it has recently been recognized that electrical stimulation of dorsal roots (4) or of peripheral nerves (5–8) activate the precentral gyrus as well as the postcentral region. Furthermore, these studies reveal the existence of small potentials in the precentral and postcentral gyri evoked by ipsilateral nerve stimulation. The differences between the results obtained with tactile stimulation and those obtained with electrical stimulation of nerves are not easily explicable on the basis that different afferents were stimulated, since it was found that cutaneous (6, 7) and deep (5–8) afferent nerve volleys activate the same cortical regions. Since the physiological stimulus which will evoke precentral potentials is not yet known, it is clearly necessary to establish whether potentials evoked in this region are essentially different from the responses recorded in the postcentral gyrus and to define the conditions necessary for eliciting them. The present study constitutes an attempt to determine some of the similarities and differences between the precentral and postcentral cortices.

MATERIALS AND METHODS

Thirty-two immature monkeys (*Macaca mulatta*) were used. The animals were usually first used for mapping studies and consequently the cortex had been exposed for 8–20 hours. The animals weighed between 1.6–4 kg and were anesthetized with Nembutal or Dial administered intraperitoneally (30 mg/kg), but the anesthetic of choice proved to be Dial. Most stable records of the precentral afferent responses were obtained at an anesthetic level which virtually abolished spontaneous electrical activity, a condition which was usually achieved after many hours of exposure. Two

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experiments were performed on animals with chronically implanted plate electrodes placed on the Rolandic cortex. In these preparations the position of the recording points was determined by x-ray and postmortem examination. Electrical stimulation of nerves in the unanesthetized animals was accomplished by chronic implantation of a pair of wires wound around the proximal end of the cut sciatic nerve, which was encased in a polyethylene sheet sutured around the nerve.

In all acute experiments the calvarium and dura were opened extensively and in four experiments the spinal cord was also exposed. The tissues were kept moist with warm physiological saline. Exploration of the anterior bank of the central fissure was performed after subpial removal of the postcentral cortex with a fine suction tip. Cortical incisions were made with an iridectomy knife or a fine curved blade. For stimulation of small nerves the superficial peroneal and the lateral gastrocnemius were usually selected, but in most experiments the entire sciatic nerve was stimulated at two to three times threshold voltage strength.

Rectangular monophasic pulses 0.1–0.2 msec. in duration were applied to the cut central end of peripheral nerves and dorsal rootlets through a General Radio 578A isolation transformer. Tactile stimuli were applied with a small brush mounted on a solenoid. Recording electrodes were either stainless steel or silver wire. In all experiments in which 'monopolar' records were obtained the indifferent lead was shifted to various points around the scalp. Potentials were amplified with two differential amplifiers which were direct coupled except for a single RC network. These amplifiers (designed by Dr. L. I. Malis) have a common mode rejection ratio of 50,000 to 1 and a frequency response of 0.5–50,000 cycles. They were used either at full band width, or with limited band width to stabilize the baseline and reduce noise without obvious alteration of the response wave form. Potentials were displayed on a DuMont 304H oscilloscope, often used in conjunction with an electronic switch with which simultaneous recording of two channels without interaction was possible. Records were photographed on 35-mm film of the DuMont modification of the Land-Polaroid camera.

RESULTS

Properties of the Precentral Afferent Response. The electrical response recorded from the precentral region after a single pulse is applied to a somatic afferent nerve is in most respects identical with other cortical evoked potentials in having a short latency initially surface positive wave followed by a prolonged negativity. Ruch, Patton and Amassian (5) regarded these potentials as true precentral electrical phenomena because of their initial positivity without a preceding negative deflection and also because their latencies were comparable to those recorded from the postcentral gyrus. These potentials do not originate in the postcentral gyrus since they persist following acute (6, 7) and chronic (9) ablation of the postcentral gyri. Responses to ipsilateral nerve stimulation also remain after removal of the postcentral cortex and complete section of the corpus callosum (9). Under conditions of deep anesthesia it is possible to activate a considerable portion of the frontal agranular cortex with a peripheral nerve volley, the recorded potentials displaying a distinct caudorostral amplitude and latency gradient. Figure 1 shows the distribution of the response latencies in the precentral gyrus of a monkey in which the postcentral gyrus was removed in order to expose the depths of the central fissure. The latency becomes gradually longer as the electrode is advanced anteriorly across the precentral region. A vertical incision about 4 mm deep and about 3 cm long into the precentral agranular cortex 4–5 mm anterior to the central fissure (in 1 animal) did not eliminate the potentials recorded in the most rostral region, indicating that the response is probably not being conducted transcortically. The prolongation in onset latency of 8–9 msec. is comparable to the increase in latency as the electrode is moved away from the central fissure on the postcentral gyrus (1) and the magnitude of the latency changes renders it unlikely that these potentials are being electrotonically conducted. This consistent latency relationship was most easily demonstrated under conditions of prolonged deep Dial anesthesia (in 4 animals) and was not clearly present in the two unanesthetized animals studied. Although the latency distance function varied in each experiment, in all deeply anesthetized animals it was noted that anterior electrode placements had distinctly longer latencies. The distribution of latencies shows an interesting relationship to the density of Marchi degenerations seen in this region of cortex following interruption of its thalamic projection fibers (10). The latency of the responses prolongs as the density of degeneration decreases.

That the precentral afferent responses originate in the precentral cortex can be demonstrated by various manipulations affecting the state of this cortex.

Effects of Local Application of Novocaine. The application of a local anesthetic to the cerebral cortex has been employed by numer-

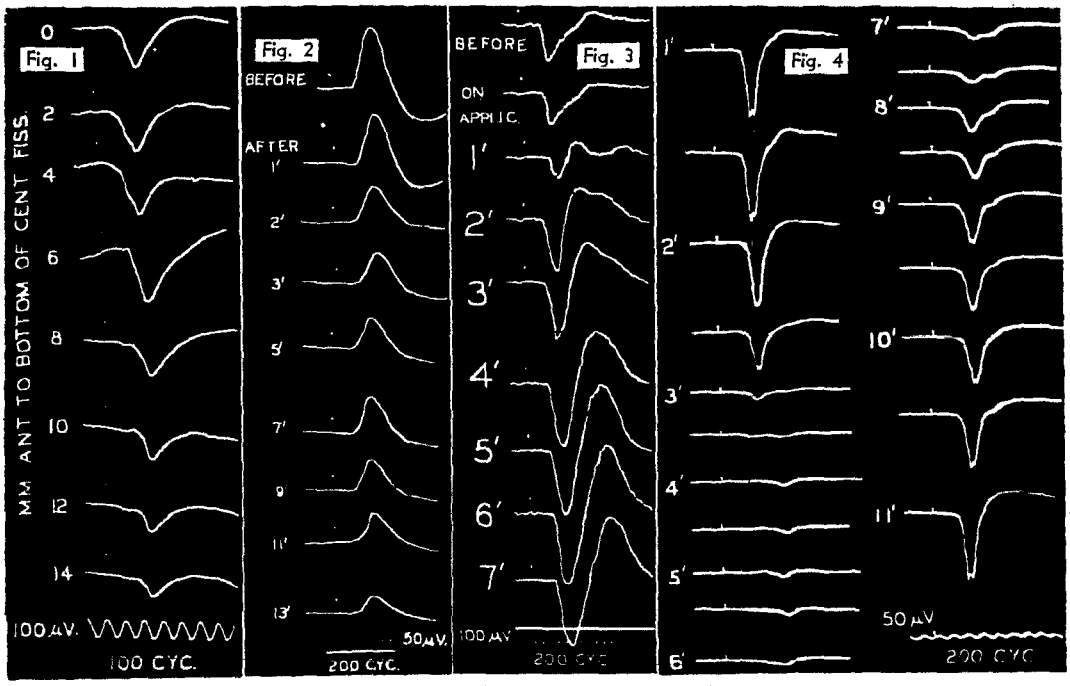

FIG. 1. Response of precentral cortex to stimulation of contralateral sciatic nerve, recorded in 2-mm steps moving forward along the precentral agranular cortex. The depths of the central fissure were exposed by subpial removal of the postcentral gyrus. Note prolongation in onset latency as the electrode was advanced along the precentral cortex. An upward deflection indicates surface negativity. Amplitude of the time line is set at a calibration voltage in all figures.

FIG. 2. Influence of topical 2% Novocaine application on the precentral response to contralateral sciatic nerve stimulation. An upward deflection indicates surface positivity. Note alteration of the positive as well as the negative component after several minutes. Additional drug application did not induce any further reduction in amplitude.

FIG. 3. Effect of topical strychnine application on the precentral response to contralateral sciatic nerve stimulation. An upward deflection indicates surface negativity. Note double deflection in the negative component in the 1 and 2-min. records and gradual enhancement of the positive wave in later records. (Dial anesthesia.)

FIG. 4. Precentral response to contralateral sciatic nerve stimulation following mechanical induction of the spreading depression of Leão approximately 5 mm anterior to the recording electrode. An upward deflection indicates surface negativity.

ous workers to demonstrate that the evoked response contains a local component which can be selectively affected when certain cortical neural elements are rendered inactive. The diffusion of the drug is assumed to be restricted to a relatively small region, since cortex a few millimeters away does not appear to be affected. Figure 2 shows the effect of one local Novocaine application (in 7 animals) on the precentral response evoked by a supraliminal electrical stimulus applied to the contralateral sciatic nerve. A 1 cm square piece of filter paper, pierced in the center for the passage of the electrode, was soaked in 2% Novocaine and placed on the precentral leg area about 1 cm anterior to the central sulcus. The local response to stimulation of the contralateral sciatic nerve was recorded at 30-second intervals after the application of the drug.

The surface-negative wave was always the more rapidly reduced, but before it disappeared the surface-positive wave had diminished somewhat. Although the surface-positive wave was less severely affected, it was reduced in every experiment. The last trace in figure 2 shows the maximum reduction obtained, since further application of Novocaine (in 2 experiments) did not cause any additional effect on amplitude or wave form. The same sequence of events is also seen when Novocaine is ap-

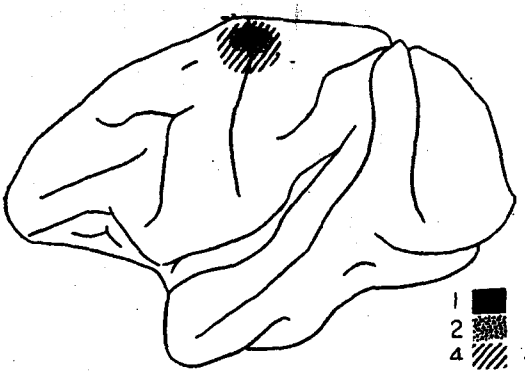

FIG. 5. Expansion of excitation in the Rolandic cortex with stimulation of 1, 2 and 4 lumbar rootlets, respectively. The extent of cortex from which a response of 75 μv or more is indicated. Deep Dial anesthesia.

plied to the postcentral gyrus. A similar result in other cortical evoked responses is well known in the cat. The reduction of the surface-positive wave of the cat auditory area which has recently been emphasized by Bremer (11) is strikingly similar to that seen in these experiments. The failure to prolong the onset latency of the responses, even with successive drug applications, is consistent with the idea that the effect of Novocaine is not largely due to any action on the afferent terminals.

Effect of local application of strychnine. The local application of convulsive drugs to the cortex has been used in many studies as a means of altering the excitability of cortical cells. In all of these studies, a rapid increase in amplitude and prolongation of the surface-negative component of the cortical response to an afferent volley has been seen. Figure 3 shows the time course of the effect of a 2% strychnine sulfate solution applied on a piece of filter paper to the precentral cortex on the response evoked by contralateral sciatic nerve excitation, in one of eight experiments. The surface negative component is always rapidly enhanced and is usually separable into two components during the initial period of enhancement as can barely be discerned in the records taken after 1 and 2 minutes. The initial positivity is also markedly enhanced, as Bremer (11) has shown in the auditory cortex of the cat. Applying strychnine to the exposed white matter after ablation of the overlying precentral cortex was without apparent effect in one animal. It is, therefore, likely that a portion of the surface-positive component of the precentral response is related to postsynaptic activation of the cortex, although a contribution from the presynaptic terminals is also probably present. The enhancement of the precentral afferent response by strychninization is localized to the site of drug application and does not alter the evoked response in the postcentral gyrus. In two animals, a similar enhancement of the smaller ipsilateral evoked response was also shown with strychnine application.

Effect of spreading depression. The spreading depression of Leão appears to be a suitable method for studying the local origin of cortical potentials because there is good reason to believe that it is conducted only in the cerebral cortex (12–14). Furthermore, the depression is reversible and transient and therefore, it can be produced many times in the same preparation. The phenomenon consists of a slow (approximately 3 mm/min.) wave spreading over the cortical surface from the point of stimulation.

Figure 4 shows the alteration of the precentral evoked response by a wave of spreading depression induced by mechanical deformation of the cortex approximately 5 mm anterior to the recording electrode on the precentral gyrus. After stroking the cortex with a small rod, the response evoked by contralateral sciatic nerve stimulation was observed and photographed at 30-second intervals until the wave regained its original form. Figure 4 shows the maximal depression of the afferent response in one of four such experiments. Other preparations showed varying degrees of attenuation of the surface-positive wave. Examination of the records shows that the depression wave reaches the point of recording within 2.5 minutes and that the negative component is completely abolished within 3 minutes. During the peak of the depression a small longer latency positive wave (records taken from 4–6 min.) appears at approximately the latency of the usual negative component and can be seen independently as a separate component when the early positive wave returns (records taken at 7–8 min.), and remains as a notch in the negative wave until it is obscured by the full negative wave. The negative wave is con-

sistently the last component to regain its previous amplitude. A similar sequence of events can be recorded in the postcentral gyrus.

Evidence that the spreading depression is limited to the cortex has been adduced by simultaneously recording the afferent response from cortex, white matter and subcortical structures (12, 14) and by observing the effect of cortical incisions on the alteration of spontaneous electrical activity produced by the wave of depression (13). In the experiments of Sloan and Jasper (13) it was found that although the depression of spontaneous activity is not abolished by a cortical incision between the point of wave induction and the point of recording, insulation of the incision did abolish the effect. In the present study this experiment has been repeated in two monkeys, using the alteration of the evoked response as a criterion of depression. Although a cortical incision does not block the wave of depression, insertion of cellophane in the incision obliterates the effect if the depression is induced beyond the cut but has no influence if the depression is induced on the opposite side of the recording point where neural contiguity remains. It is thus clear on the basis of experiments with cortical and subcortical recording (12, 14) and those with cortical incisions that the spreading depression of Leão is a cortical phenomenon.

Afferent Source of the Precentral Evoked Response. *Cutaneous and deep nerve stimulation.* Electrical stimulation of both cutaneous and muscle nerves evokes a response in the pre- and postcentral gyri of the monkey (5–7). In three experiments an attempt has been made to determine the fiber groups involved in this projection by monitoring the afferent volley at an intact lumbar (L_6–L_7) dorsal root while stimulating the superficial peroneal or sural nerves. With monopolar recording and no 'dead-end' lead, monophasic records are not obtained and it is, therefore, impossible adequately to identify more than the initial deflection. However, despite the limitation of the method, it is clear that the excitation of the low-threshold beta cutaneous fibers evoked a response in the pre-Rolandic cortex. Tactile stimulation or electrical excitation of the skin has always failed to excite the precentral region although responses to such stimuli are present in the dorsal roots and postcentral gyrus.

Stimulation of deep nerves (e.g., lateral gastrocnemius nerve) does not yield an evoked response in the pre- and post-Rolandic cortex when only the first deflection (*group I*) of the dorsal root record appears. Identification of the fiber group activating a cortical response would require appropriate monophasic recording, but it is clear that the appearance of the *group I* fiber deflection (which is largely derived from muscle spindle and tendon organs (15) is not accompanied by an evoked response in the Rolandic cortex of the anesthetized animal, a finding which is in agreement with earlier experiments on the cat (16). In several experiments an attempt was made to record a cortical response to rapid muscle stretch, in a leg devoid of skin, but no evoked responses were seen on either side of the central fissure. Strong stimulation of muscle nerves evokes a response in both precentral and postcentral gyri, but the physiological stimulus for these responses is unknown.

Since a specific receptor system, whose

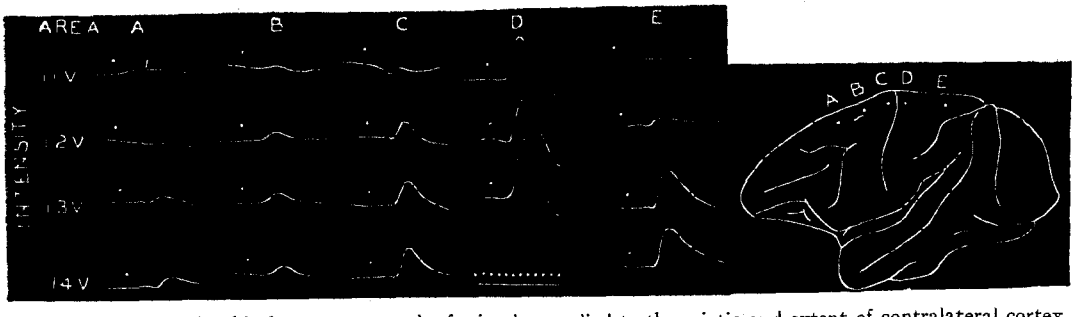

FIG. 6. The relationship between strength of stimulus applied to the sciatic and extent of contralateral cortex activated. An upward deflection indicates surface positivity. Postcentral cortex (point D) reaches maximum amplitude rapidly whereas more fibers must be stimulated to evoke responses in other regions. Time line: 200 cycles set at 50 μv amplitude.

stimulation will yield the precentral response is not yet known, it is possible that the more extensive maps obtained by electrical excitation of peripheral nerves are due to synchronous excitation of a large number of neural elements.

Effects of spatial summation. The application of an electrical pulse to the proximal end of a single dorsal root evokes a cortical action potential in a small area of cortex surrounding the central fissure (4). When an adjacent root of the same segment is activated in a similar manner, there is only a very small difference in the region and extent of cortex activated. The simultaneous excitation of both roots, however, substantially increase the size of the responsive cortical zone. The magnitude of the increase depending on the anesthetic level, temperature and blood pressure of the animal.

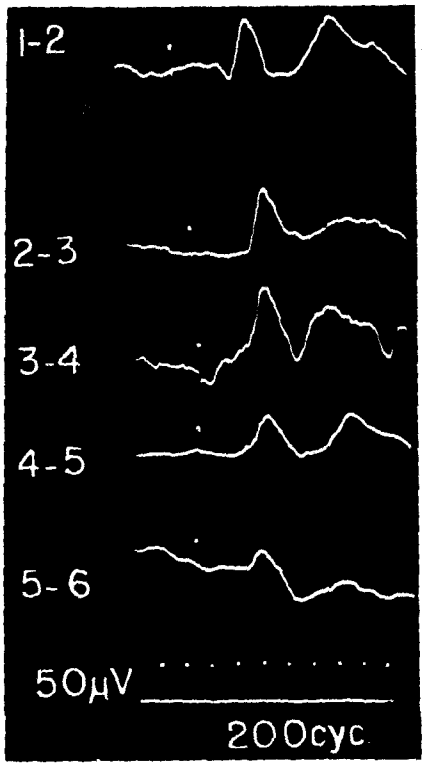

FIG. 7. Response of Rolandic cortex to stimulation of the contralateral sciatic nerve in an unanesthetized monkey with implanted electrodes. All records are 'bipolar' with interelectrode distances of 2 mm and surface positivity indicating by an upward deflection. *Points 1–3* are on the precentral gyrus with point 3 lying on the central fissure, all other points are on the postcentral gyrus.

Figure 5 diagrams a typical experiment out of four performed under deep Dial anesthesia which shows the expansion of the region of cortex from which an evoked response can be recorded after stimulation of 1, 2 and 4 root filaments at L_6. The region mapped consists of the area from which evoked potentials exceeding 75μv were recorded. The expansion of the active zone assumes an elliptical form with its longer axis in the anteroposterior direction.

Another appropriate means of varying the number of peripheral neural elements excited, is to apply electrical pulses of increasing strength to a large nerve such as the sciatic. If the increase in voltage does not exceed the threshold voltage by more than 1.5 times one can assume an increased number of fibers of the same group are excited. Figure 6 illustrates a series of records taken in sequence at several stimulus strengths applied to the sciatic nerve of a freshly exposed preparation under Dial anesthesia, with recording of the evoked response at five cortical points along the antero-posterior axis at each intensity level. The response occurs first near the central fissure, with lowest threshold and maximum amplitude recorded at the postcentral lead (*D*) adjacent to the central fissure. As the stimulus strength is increased the responsive cortical area expands in both anterior and posterior directions with an increase in latency when recording from points more distant from the focus (*D*). Both the spatial and temporal gradient can also be recorded in the dorsal ventral direction. The spread of cortical excitation was unaffected by transverse incisions which were made in the precentral and postcentral gyri in two of the five animals studied. The phenomenon is also demonstrable, although less extensive, with stimulation of either superficial or deep afferent nerves.

Effects of Anesthesia. In the course of these experiments, it was noted that the spatial summation effect described above as well as the extent of precentral activation varies with the level of anesthesia; preparations maintained under deep Dial anesthesia being most consistent. In order to determine whether the precentral response can be obtained only under such unphysiological circumstances, the problem was pursued with the use of unanesthetized animals.

In two animals, plate electrodes were im-

planted on the Rolandic cortex with three electrodes placed precentrally and three postcentrally in each animal. The inter-electrode distance was 2 mm, and a polyethylene plate containing the exposed recording surface was held in place on the pial surface by suturing the electrode to the under surface of the dura. In both animals, it was possible to observe the same spatial summation effects described above for sciatic nerve stimulation under Dial anesthesia, in the complete absence of any anesthetic effects. Although the evoked response in many sweeps is obscured by the enormous amount of spontaneous activity, it is possible to record afferent responses on both sides of the central fissure in the unanesthetized animal (fig. 7). Attempts to record tactile evoked responses in the precentral region of the unanesthetized monkey were unsuccessful.

DISCUSSION

It is generally agreed that the initial surface-positive deflection of the potential evoked in the cortex coincides with the arrival of an impulse in the afferent terminals of the cerebral cortex, since an essentially similar wave can be recorded from the exposed radiations (17). However, the 'afferent wave' recorded in the radiations is distinctly reduced in amplitude and duration, and Eccles (18) and Bremer (11) have observed that there is also much evidence to indicate that this wave is also partly attributable to the synaptic activity or discharge of cortical elements. It is clear from the present experiments that the surface-positive components can be depressed or enhanced by local application of an anesthetic or convulsant agent respectively. Whether the firing of cortical cells contributes greatly to the potential recorded from the surface is not yet clear. The integrated wave recorded at the surface is unreduced under conditions of deep anesthesia when individual units are more difficult to activate (19), but unit discharge can be recorded during the surface-positive phase of the evoked response (20).

The local origin of the response recorded in the precentral gyrus is clearly demonstrated by 'monopolar' recording against various reference leads and 'bipolar' recording, as well as by manipulation of the precentral wave form by the local action of Novocaine, strychnine and the spreading depression of Leão without simultaneous alteration of the postcentral response. The functional significance of an evoked potential which is not induced by physiological stimulation is difficult to determine. It is clear that the precentral region is maximally responsive to the synchronous excitation of a greater number of fibers than one can excite physiologically. It therefore must be recognized that the projection map obtained with sciatic nerve stimulation may not be reproduced with physiological stimulation of end organs. The increased amplitude and distribution of precentral responses in animals subjected to prolonged exposure and the peculiar anesthetic conditions induced by large doses of Dial or barbiturate supplemented with chloralose (7, 21) also requires cautious interpretation. However, since the precentral response is not dependent on conditions of deterioration and anesthesia, as shown in the unanesthetized animal, it is possible to interpret the enhancement of potentials in acute experiments as due primarily to a reduction in spontaneous electrical activity and the resulting tendency for synchronous activation in the cortex. It has long been recognized that postcentral responses to tactile stimulation also improve in the course of acute experiments in which barbiturate anesthesia is used (17). Despite all of these difficulties, it is also necessary to consider the possibility that excitation of large numbers of fibers activates the entire somatic afferent projection system, whereas discrete tactile stimuli may fail to do so. Experiments in which nerve volleys are used for mapping of the afferent projection system cannot be adduced as arguments against a precise tactile localization (22) since an increase in the number of fibers excited tends to obscure rather than to disprove the validity of somatotopic maps.

Although it is clear that the precentral region responds to somatic afferent stimulation, it is important to recognize the differences between the precentral and postcentral regions. The most obvious difference is that the precentral region is not activated by natural tactile stimulation in the anesthetized or unanesthetized animal. It is also interesting to note that the precentral response persists without latency alteration after chronic ablation of somatic receiving areas I and II (9), although histological examination of the thalamus reveals extensive cellular degeneration in the ventrobasal thalamic tactile region

as defined by Mountcastle and Henneman (23). It has been suggested that the precentral region may be activated by the controversial pyramidal tract afferents, but the cortical response to nerve volleys is not affected by acute transection of the medullary pyramids (9). It is also known that the precentral response is not altered by acute ablation of the cerebellum (6, 7). The most characteristic feature of the precentral response appears to be its relation to the magnitude of a synchronous peripheral nerve volley. The small evoked response in the pre- and postcentral gyri to an ipsilateral nerve volley (6–8, 21) also shares the feature of not being evoked by natural tactile stimulation. It is believed that the ipsilateral response is probably not due to a spinal cord reflex effect in the contralateral leg because such responses can be obtained in the curare immobilized animal (24). The interpretation of these findings must remain conservative unless a physiological stimulus which simulates the effect of a large synchronous nerve volley is found.

Whether the precentral afferent projection which is admittedly demonstrated under artificial conditions can be implicated in somatic sensation under physiological circumstances is uncertain. Lesions of the precentral gyrus in man (25) and the monkey (26) initially result in a defect in somesthesis which might be the result of interruption of the precentral afferent system. However, the transient nature of the defects and the possibility of unidentified damage to the postcentral gyrus render these observations as only suggestive. On the other hand, the finding that strychninization of the precentral cortex after postcentral resection produces symptoms of heightened somatic sensation in the monkey (27) suggests that the precentral cortex does play a role in somatic sensation.

The consistent anatomical proximity of 'sensory' and 'motor' fields in all mammals must be regarded as a feature of their functional unity. However, despite our knowledge of an extensive superimposition of afferent and efferent fibers in this region, there is also ample evidence of considerable differences between the precentral and postcentral regions in terms of the preponderance and source of afferent and efferent fibers as well as differences in structure and the effects of ablation. The pertinent question that remains is not whether functional localization exists, but rather how to interpret the lines of division in the continuous gradients of the cerebral cortex.

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