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Two types of rhythmical jaw movements evoked by stimulation of the rat cortex

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Abstract: Two different patterns of rhythmical jaw movements (RJM) resembling masticatory movements were evoked by stimulation of the cerebral cortex in the rat. The first type of RJM was evoked from the primary jaw motor area, and it always began with a sustained jaw-opening and consisted of high frequency (5-7 Hz) simple opening-closing movements of the jaw. The anterior digastric electromyographic (EMG) activity showed discharges apparently time-locked to each stimulus pulse. The second type of RJM was evoked from the ventral part of the insular cortex. In contrast to the first type of RJM, the second type started with a jaw closing movement in many cases. This type of RJM had large and complex lateral and sagittal movements and the frequency of RJM was low (3-4 Hz). The digastric EMG activity did not show the stimulus-locked component. The pattern of these RJM did not depend on the intensity or frequency of stimulation, but on the site stimulated. These two cortical RJM areas appeared to work independently from each other, because ablation of one area did not affect the pattern of the RJM evoked from the other cortical area.

Introduction

Repetitive electrical stimulation of a certain area of the cerebral cortex can evoke rhythmical jaw movements (RJM) resembling masticatory movements in many species of mammals (cf. Luschei and Goldberg[1]). Stimulation of the anterolateral part of the rat cortex also evokes RJM[2,3]. However, there is no report that clearly delineates the cortical masticatory area of the rat and describes the patterns of the RJM evoked from it.

Bremer[4] has described that three different types of RJM are evoked by stimulation of three different areas within the cortical masticatory area in the awake rabbit. Those patterns are, an incisal gnawing evoked from the anterior part, a vertical mastication evoked from the middle part and a rumination evoked from the posterior part of the masticatory area. Lund et al.[5] have evoked two different types of RJM by stimulation of the cortical masticatory area in the urethane anesthetized rabbit, although they have not been able to evoke Bremer's "gnawing". They have described that stimulation of the anterior part of the masticatory area evokes simple opening-closing movements of the jaw, whereas the posterolateral part evokes RJM which have a contralateral deviation of the jaw and resemble natural mastication. It has also been indicated in the cat that there are two spatially separated cortical areas for jaw and orofacial movements[6].

The aim of the present study is to delineate the cortical RJM area of the rat and to ascertain whether different patterns of RJM are evoked from spatially separated areas in the

rat cortex.

Part of the results were presented elsewhere in an abstract form[7,8].

Materials and methods

Experiments were performed using 34 male Wistar rats (body weight : 250-300 g) that were obtained from a local breeder (Seiwa experimental animals, Ltd.). Animals were maintained on a 12 h light-dark cycle, were given free access to food and water. Guiding principles for the care and use of animals in the field of physiological sciences, as outlined by the Physiological Society of Japan, were followed carefully.

The animals were anesthetized with urethane (1.0 g/kg, i.p.). Supplementary doses (0.2 g/kg, i.p.) were given to maintain the rat at a level that suppressed retraction of the hindlimb following a firm pinch of the foot. The masseter and anterior digastric muscles on both sides were exposed and bipolar electromyographic (EMG) wire-hook electrodes (copper strings with an enamel coating, diameter : 0.13 mm, tip exposure : 2 mm, interelectrode distance : 1.5 mm) were inserted into the muscle bellies. A small infrared light emitting diode (LED, diameter : 3.1 mm, weight : 0.08 g) was attached to the mandibular symphysis with acrylic resin. Total weight of the LED and resin block ranged from 0.15 to 0.20 g and we considered that the weight did not disturb the jaw movement of the rat. The animal was fixed on a stereotaxic apparatus (SN 2, Narishige) with ear bars and an incisal bar. A small screw was implanted into the nasal bone. A metal rod was attached to the stereotaxic apparatus and this rod and the screw on the nasal bone were connected with acrylic resin. Then the incisal bar was removed out of the rat's mouth so that the jaw could move freely. The left superficial temporalis muscle was removed. The frontal, parietal and squamosal bones were removed and the anterolateral part of the cortex was exposed by cutting the dura mater carefully. The surface of the exposed cortex was covered with a mixture of liquid-paraffin and Vaseline. The dura mater was punctured at the foramen magnum to allow the cerebrospinal fluid to flow out and to prevent a swelling of the brain. The body temperature was maintained at 37 °C with a heating pad throughout the experiments.

A stimulator (SEN-7103, Nihon Kohden) and a constant current isolator (SS-201 J, Nihon Kohden) were used to stimulate the cortex. All stimuli were applied to the left side of the cortex. Three cathodal square-wave pulses of 0.1 ms duration and 500 Hz frequency were applied to elicit short-latency EMG activities in the anterior digastric muscles. Train pulse stimulation composed of 1 ms duration square-wave pulses of 15-100 Hz (mostly 30-50 Hz) frequency were applied for 5-60 s to evoke RJM. In three rats, a broad area of the cortex was stimulated with a monopolar electrode of a glass-coated tungsten (tip exposure : 50-80 μm). Rows of penetrations were made in the frontal and parasagittal plane. The stimulating electrode was inserted into the cortex at a right angle to the cortical surface and advanced through the cortex in 200 μm steps. The effect of stimulation was examined at each depth and precise maps for a short-latency digastric response or the RJM were made. An indifferent electrode was placed on the exposed neck muscle. In other rats, a bipolar concentric stainless steel electrode (diameter of inner electrode : 0.15 mm, diameter of outer electrode : 0.5 mm, interpolar distance : 1.5-2.0 mm) was used because the stimulus artifacts recorded by the EMG electrodes were smaller with this type of electrode than with the monopolar electrode. In the bipolar stimulation, the tip of the electrode was placed at the most effective depth which was in the range of 2.0-2.5 mm for the short-latency digastric response and RJM in the primary motor area or 1.5-2.0 mm for the RJM in the insular cortex.

Jaw movements were monitored by recording the movements of the LED in the vertical, horizontal and sagittal axes with a photodiode transducer system. The EMG activities of the masticatory muscles were recorded with wire-hook electrodes. Each recording was amplified and led to a cathode ray oscilloscope (VC-10, Nihon Kohden) and stored in a data recorder (FE-39 A, SONY). The records were replayed so that the movements and EMG activities could be drawn on paper

and analyzed with a personal computer system (PC-9801 F 2, NEC).

To clarify the relationship between two cortical RJM areas, the effects of stimulating one area were examined, before and after an ablation of another area, and vice versa (four rats in each case). Cortical ablation was accomplished by aspiration. Furthermore, the effect of stimulating the white matter immediately beneath the ablated cortex was examined in each case. To avoid confusion in analyzing the effect of stimulating the cortex or the passing fibers which run in the subcortical area, the cortical stimulation experiment was performed two weeks after a complete ablation of the primary motor area was done on both sides in four rats. In the preliminary experiment, it was confirmed with Nauta and Gygax method[9] that the cortical descending fibers were degenerated completely 2 weeks after cortical ablation. In case of chronic cortical ablation, after bilateral craniectomy under pentobarbital anesthesia (Nembutal, Abbott, 30 mg/kg, i. p.), an impression of the opening of the cranial bone was taken with an elastomeric polysiloxane impression material (Coltex fine, Coltène Inc.). A resin core was made on the platter model prepared from the impression. After ablation of the primary motor areas on both sides, hemostasis was maintained with a piece of gelform and saline. Then the resin core was attached to the opening of the cranial bone with wax. The skin wound was sutured. Antibiotics (penicillin-G, Banyu, 50000 units/rat, i. m.) was administered. The rats did not take food by themselves for two days after surgery. However, they could eat food paste and drink water when an operator put them into their mouth. In the following days, the rats ate ordinary pellets. Reduction of body weight during the first postoperative two days ranged from 20 to 30 g.

After the experiments, the rats were anesthetized deeply with urethane (1.2 g/kg) and decapitated. The head was stored in 10 % formalin over night. The brain was removed and stored in 10 % formalin for an additional 3 to 7 days. The lateral view of the brain was photographed. A trace of the brain was drawn from the photograph, and stimulated points were plotted on the trace. Frontal sections of the brain were cut at a thickness of 50 μm on a freezing microtome. The sections were stained with cresyl violet and stimulated sites were verified.

Student's t-test was used to investigate difference between means of latencies of cortically evoked digastric responses. $p<0.05$ was considered statistically significant.

Results

Cortical areas which induced jaw movements

Figure 1 illustrates the cortical jaw motor areas. Figures 1 A and B show the areas which had low threshold intensity for evoking a short-latency EMG activity in the left (ipsilateral) and right (contralateral) anterior digastric muscles, respectively. The ipsilateral anterior digastric area was narrower and located more dorsally than the contralateral one. We could not evoke a short-latency activity of the masseter muscle under the present experimental conditions. There were two separate areas from which the RJM could be evoked by low intensity repetitive stimulation (Fig. 1 C). In this paper, we will call the area located in the anterior part of the cortex, A-area (anterior RJM area) and the other, P-area (posterior RJM area). The center of the A-area was located at 1.8-2.2 mm posterior to the frontal pole and 5.0-5.2 mm lateral to the midline and the diameter of the area was 1.5-2.0 mm. The center of the P-area was located at 0.3-0.5 mm posterior to the middle cerebral artery and 0.3-0.5 mm dorsal to the rhinal sulcus and the diameter of the area was 1.5-2.0 mm. The A-area overlapped with the area for the short-latency response of the anterior digastric muscle. These cortically induced RJMs could be evoked even when the white matter was stimulated after the covering cortex was ablated.

Short-latency digastric responses evoked by stimulation of the cortex

Short train stimulation of the A-area evoked short-latency EMG activity in the anterior digastric muscles on both sides (Fig. 2). The threshold of EMG activity of the contralateral digastric muscle at the most effective point ranged from 10 to 25 μA and

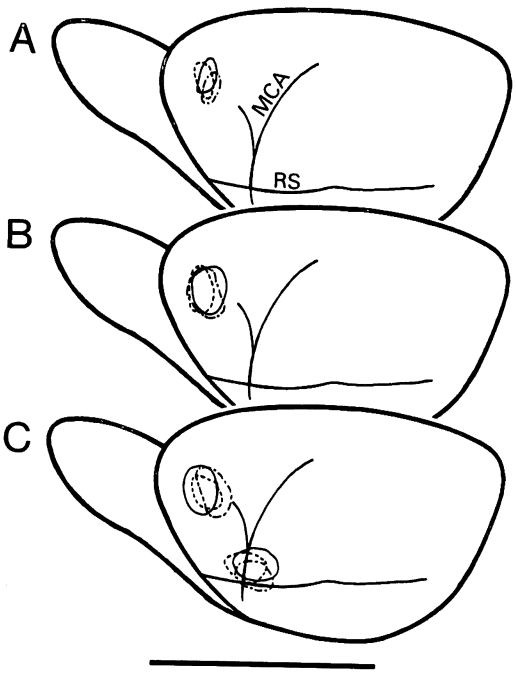

Fig. 1 Cortical jaw motor areas. MCA : middle cerebral artery, RS : rhinal sulcus. Data from the 3 rats (drawn with different line style) are superpositioned with a reference of a cross point of MCA and RS. A and B : limits of the areas from which ipsilateral (A) and contralateral (B) anterior digastric short-latency responses could be elicited with currents of less than 30 μA are drawn on outlines of the left cortex. Sites 2.5 mm in depth were stimulated with a monopolar electrode with 3 shocks (0.1 ms, 500 Hz). C : outlines of areas from which the rhythmical jaw movements could be obtained with 50 Hz train of shocks of 1 ms duration and less than 50 μA through the same electrode. Scale bar : 10 mm.

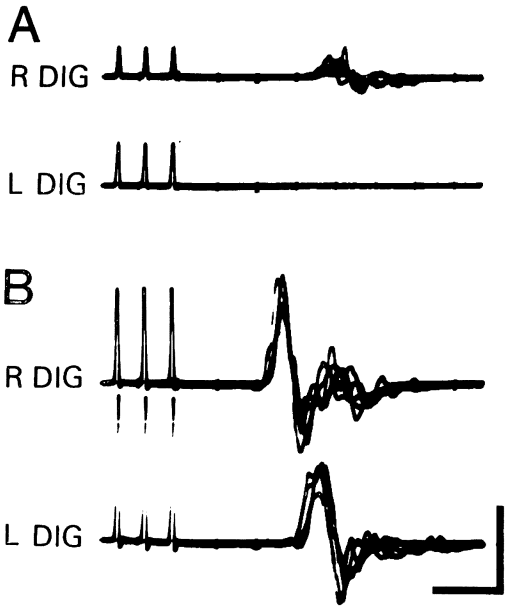

Fig. 2 Recordings from right (R DIG) and left (L DIG) anterior digastric muscles during stimulation of the left A-area. Five responses are superimposed. Three pulses were delivered at 500 Hz. Stimulus intensity is 20 μA (1.1 times threshold for R DIG) in A and 84 μA (3.3 times thresh old for L DIG, 4.2 times threshold for R DIG) in B. Calibration : 5 ms, 2 mV.

that of the ipsilateral one from 20 to 30 μA. The shortest latency from the first stimulus pulse at supramaximum stimulation (3.3 times threshold for each muscle) was 10.82$\pm$0.37 ms (n=10, mean$\pm$SD) for the right anterior digastric EMG and 12.11$\pm$0.69 ms (n=10) for the left one. Thus, there is a significant difference between the latencies of the digastric EMG on both sides ($p<0.001$). Much higher stimulus current (usually more

Fig. 3 Rhythmical jaw movements evoked by stimulation of A-area. A : jaw movements in three planes. From top, vertical (VERT, opening down), horizontal (HORZ, right down) and sagittal (A-P, posterior down) jaw movements are displayed. Dotted lines indicate resting levels. Onset and end of stimulation are indicated by downward and upward arrows, respectively. Data in figures "a" and "b" in B-D are taken from periods of "a" and "b" in A. B : frontal and sagittal views of the movements. R : right of rat, L : left, C : closing, O : opening. Vertical zero level is the resting level. Arrows indicate direction of movements. Several cycles are superimposed. C : time between successive data point is 0.5 ms. Jaw movements in three planes (VERT, HORZ, A-P) are displayed. EMGs of right (R) and left (L) anterior digastric (DIG) and masseter (MA) muscles and stimulus train (STIM) are also displayed. D : peristimulus histograms of rectified and averaged EMG activity triggered by each stimulus pulse (n=40). Arrows indicate stimulus timing. Bipolar stimulation was employed in this and following figures. Threshold for this point at 50 Hz, 1 ms=85 μA.

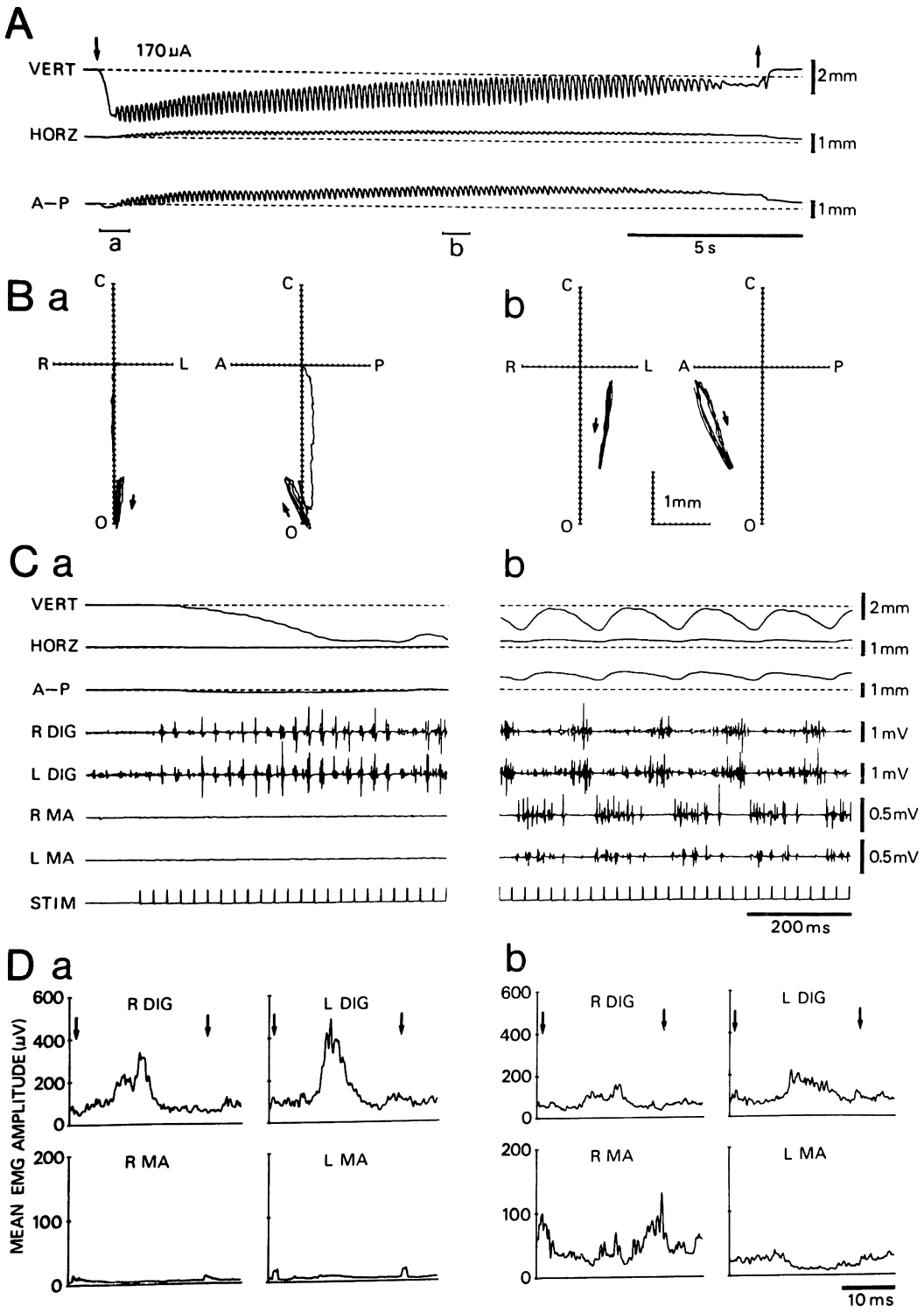
A
170μA
VERT
HORZ
A–P
2mm
1mm
1mm
a
b
5s
B a
C
R
L
A
P
O
b
1mm
C a
b
VERT
HORZ
A–P
R DIG
L DIG
R MA
L MA
STIM
2mm
1mm
1mm
1mV
1mV
0.5mV
0.5mV
200ms
D a
b
MEAN EMG AMPLITUDE (μV)
600
400
200
0
R DIG
L DIG
200
100
R MA
L MA
10ms

Fig. 3

than several hundred μA) was required to evoke digastric activity when stimulating the P-area, while stimulation of a deep part of the cortex (2.5-3.0 mm) was necessary to evoke it with low stimulus intensity. The characteristics of the evoked digastric activity were similar to those evoked from the A-area.

The rhythmical jaw movements evoked by stimulation of the A-area

Repetitive stimulation of the A-area induced RJM (A-RJM). Threshold of the RJM at the most effective point ranged from 15 to 50 μA with the monopolar stimulation and 30 to 100 μA with the bipolar stimulation. When a stimulus intensity was less than the threshold of the RJM, only a simple sustained jaw opening was induced.

Figure 3 shows an example of the A-RJM. The simple pattern of RJM with small amplitude and high frequency (5-7 Hz) occurred after a jaw-opening (Fig. 3 A, B). The amplitude of the RJM became large upon continued stimulation. Although the jaw stayed at the level below the resting position during the RJM in many cases, it closed to the resting level in the closing phases of the cycle in a few cases. The separation between the upper and lower incisor teeth at the resting level ranged from 2.5 to 3.5 mm when it was measured between trials and this value was constant for each animal. Lateral and sagittal movements were very small and simple. The EMG activity of the anterior digastric muscles were time-locked to each stimulus pulse during the early jaw-opening period (Fig. 3 Ca). The averaged anterior digastric EMG activity showed a sharp peak (Fig. 3Da, top). The latency of the onset of the effect was 5.56 ± 0.66 ms ($n=5$) for the right digastric muscle and 7.44 ± 0.88 ms ($n=5$) for the left one. The peak latency was 10.51 ± 1.48 ms ($n=5$) for the right and 10.44 ± 0.52 ms ($n=5$) for the left. Thus, there was a significant difference between the onset latencies ($p<0.01$) but not between the peak latencies ($p>0.05$). The EMG activity was absent in the masseter muscle in the early stage of the A-RJM (Fig. 3Ca, Da, bottom). Stimulus-locked component of the EMG activities in the anterior digastric muscle became weak in the late stage when the pattern of the RJM fully developed and the burst activity occurred simultaneously with the opening phases of the RJM (Fig. 3Cb, Db, top). Reduction in amplitude of stimulus-locked activity was prominent in the right digastric EMG during the closing phases of the cycle (Fig. 3Cb). The masseter EMG showed the bursts in the closing phases (Fig. 3Cb). The averaged left masseter EMG indicated a weak stimulus-locked inhibition, whereas the right masseter seemed to show a mixed effect of weak stimulus-locked activation and inhibition (Fig. 3Db, bottom).

The rhythmical jaw movements evoked by stimulation of the P-area

Repetitive stimulation of the P-area induced another type of RJM (P-RJM). Threshold of the RJM was higher than that for A-RJM. The range of the threshold at the most effective point was from 30 to 50 μA with monopolar stimulation and 50 to 200 μA with bipolar stimulation.

Figure 4 shows an example of the P-RJM. The frequency of the P-RJM was about 3.5 Hz which was lower than that of the A-RJM. Lateral and sagittal movements were larger and more complex than those of the A-RJM (Fig. 4 A, Bb). The P-RJM did not have the early jaw-opening, and a small closing movement initiated it in many cases. The jaw closed over the resting level in the closing phases. The digastric EMG activities did not show the stimulus-locked component even in the early stage, but did show the bursts in the opening phases of the RJM (Fig. 4 C, D). The EMG activity of the masseter muscle was weak and did not occur simultaneously with the closing phase (Fig. 4 Cb). When the stimulus intensity was raised or a deep part of the cortex was stimulated, the stimulus-locked digastric EMG and consequent jaw-opening appeared as in the A-RJM.

The pattern of the RJM evoked from any point within the A- or P-area was always similar to that shown in Fig. 3 or Fig. 4, respectively, and it was not affected by varying the frequency or intensity of stimulation.

The effects of ablation of the cortical RJM areas on the pattern of the RJM

Because the A-area and P-area have a reciprocal projection[10], acute ablation of the

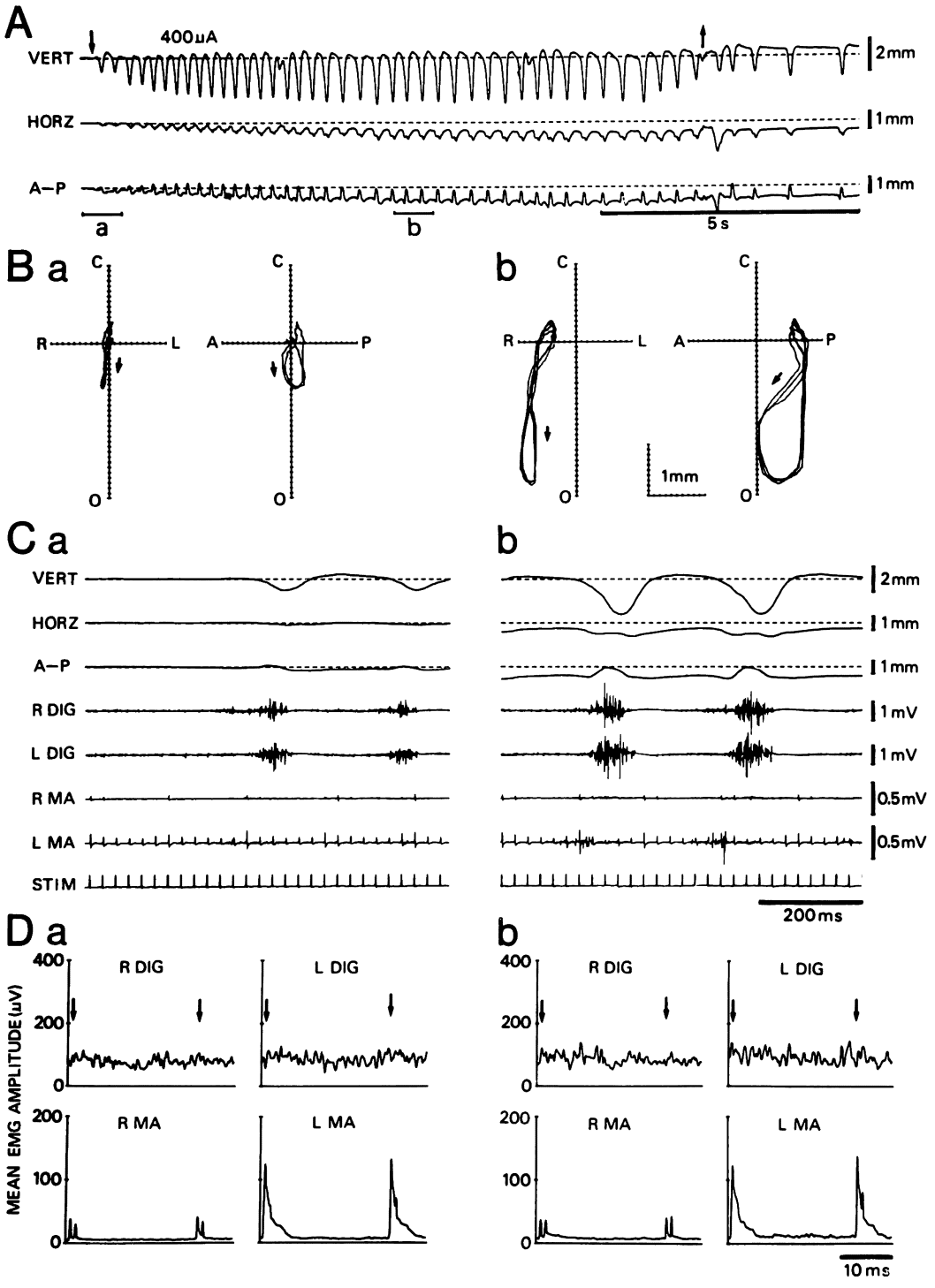

Fig. 4 Rhythmical jaw movements evoked by stimulation of P-area. As in Fig. 3. Threshold, 200 μA.

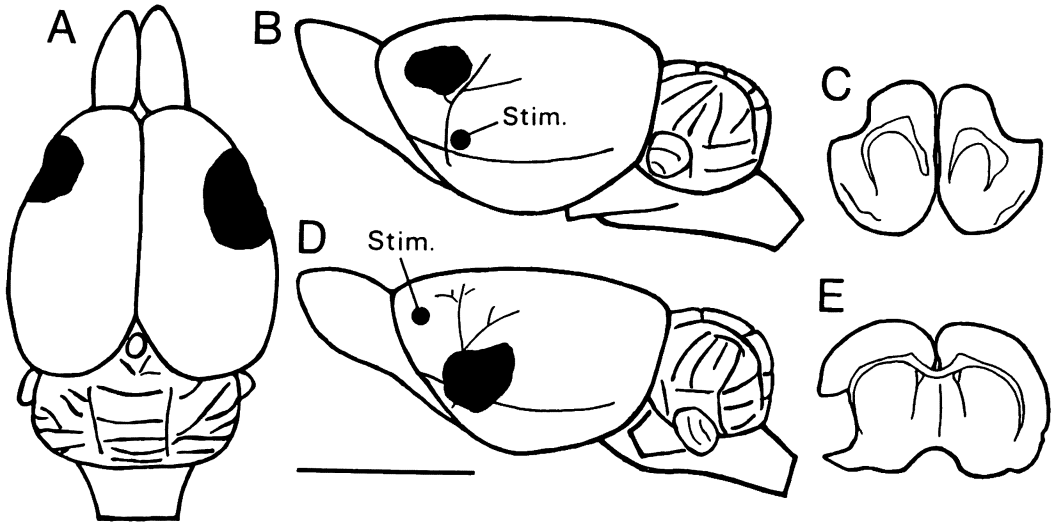

Fig. 5 Semischematic drawings of ablated areas in cortex. A-C : chronically ablated A-areas are indicated by black areas in the dorsal (A) and lateral (B) view of brain. C : trace from histological section of case shown in A and B. D and E : acutely ablated areas (D) and trace from the histological section (E) of P-area. Stimulated points are indicated by dots. Scale bar : 10 mm.

left side P-area was performed to examine the role of the projection on the A-RJM (Fig. 5 D, E). When the same site as before the P-area ablation was stimulated, the pattern of the A-RJM remained essentially the same as before ablation (not shown in figure). Namely, a jaw-opening followed by small and simple RJM was observed. The pattern of the RJM was simple even in the late stage. The digastric EMG activity was stimulus-locked. The masseter EMG activity became weak but occurred in the closing phase. When ablation of the P-area extended to the subcortical area, the pattern of the A-RJM was apparently affected and when it reached the caudate-putamen complex, the A-RJM disappeared.

Acute ablation of the A-area was performed to examine the possibility that the P-RJM was produced via the A-area. The ablation essentially did not affect the pattern of the P-RJM, although the threshold increased (not shown in figure). Chronic ablation of the A-area was performed to examine the possibility that the P-RJM was produced by direct stimulation of the fibers descending from the A-area (Fig. 5 A-C). The P-area was stimulated two weeks after ablation, because the fibers descending from the A-area are considered to have been degenerated completely by then. The pattern of the P-RJM was essentially the same as the case of stimulating the intact cortex (compare Fig. 4 with 6). Namely, a closing movement initiated RJM. Lateral and sagittal movements of the RJM were large and complex and the jaw closed over the resting level in the closing phases (Fig. 6 A, C). The EMG activities of the anterior digastric muscle showed the burst discharges in the opening phases of the RJM (Fig. 6 B, C). The masseter EMG activity was weak and did not appear in the closing phases of the cycle.

Discussion

In the present study, we found two spatially separated cortical areas responsible for different types of RJM in the rat.

The present study suggests that the A-area corresponds to the primary jaw motor area[11-13] because of its location and ability to induce short-latency EMG activities of the anterior digastric muscles. The P-area was

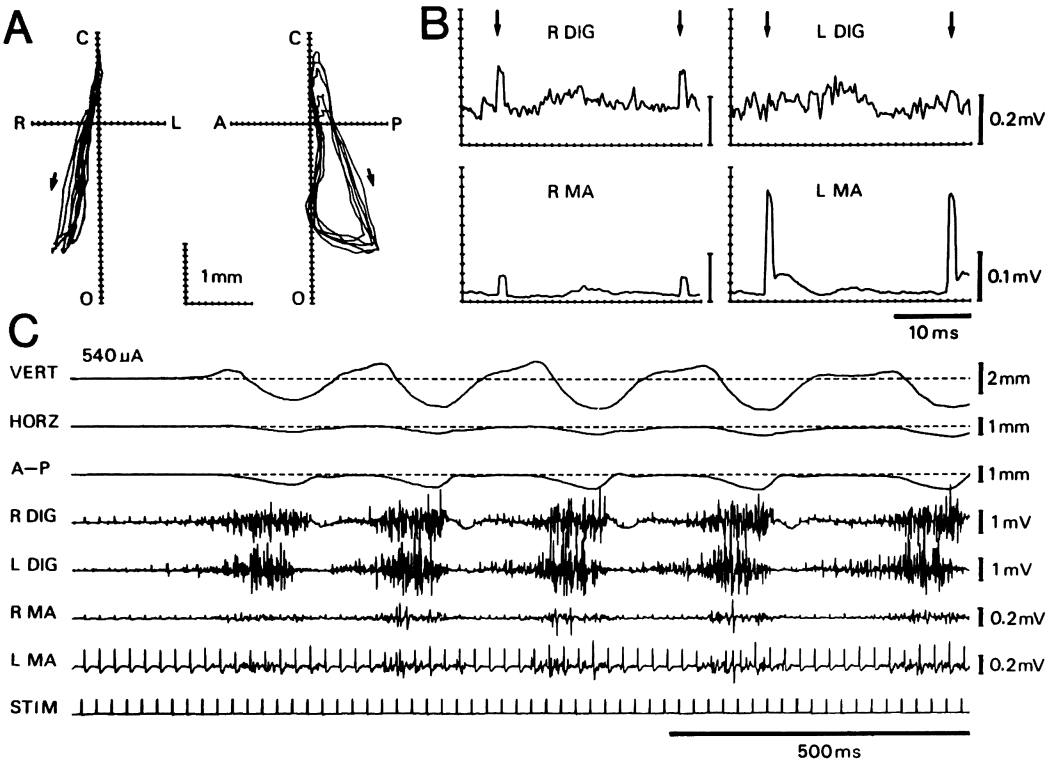

Fig. 6 Rhythmical jaw movements evoked by stimulation of P-area after chronic ablation of A-area. As in Fig. 3, except for amplification of MAs. Threshold, 270 μA.

located in the ventral part of the insular cortex. From its cytoarchitecture, the insular cortex of the rat is divided into a dorsal granular area, an intermediate dysgranular region, and a ventral agranular strip[14]. The P-area appears to be located in the ventral agranular strip.

The cortex controls the jaw motoneurons through at least two pathways. One is the short-latency pathway which inhibits the jaw-closing motoneurons and excites the jaw-opening motoneurons (reviewed in 1) ; see also refs. 15, 16)) and the other is a polysynaptic pathway through the central pattern generator (CPG) which has been considered to produce RJM without rhythmical inputs from higher centers or the periphery[17-19]. In the present study, it appears that the A-area has both pathways, because short train stimulation of the A-area induced short-latency digastric responses and repetitive stimulation of this area produced the RJM with an opening movement. Conversely, we had to stimulate a deep site or use a strong stimulus to elicit the digastric activity from the P-area. This effect may be due to direct stimulation of the fibers descending from the A-area, because those fibers pass through the caudate-putamen complex beneath the P-area[10]. Therefore, it seems that the P-area has no short-latency pathway to the trigeminal motoneurons and repetitive stimulation of the P-area activates only the CPG and then evokes the RJM without stimulus-locked digastric activity.

Nozaki *et al.*[20] have suggested that the paragigantocellular and the gigantocellular nuclei in the medial bulbar reticular formation are essential in forming the rhythm of the RJM in the guinea pig. On the other hand, Marini and Sotgiu[21] have located neurons which show a phasic modulation with the rhythm of cortically induced RJM in the lateral reticular nucleus in the rabbit. Moriyama[22] has shown that some neurons in the lateral reticular formation are also modulated with the rhythm of the RJM evoked from

the A- or P-area in the rat. He has also indicated that the neurons which receive dominant input from the A-area are located somewhat medially in the recticular formation and neurons with a similar input from the P-area are located laterally. We got the same results as those of Moriyama[22] with a horseradish peroxidase tracing method in the rat[10]. These neurons in the lateral reticular formation may possibly contribute to form a complex pattern of the RJM. In the present study, stimulation of the white matter beneath the ablated cortex could produce the same pattern of RJM as before ablation. From these facts, it may be suggested that the difference in the patterns of the RJM is due to the difference in projection patterns from these two cortical areas to the brain stem and is not due to the difference in intracortical mechanism.

In the guinea pig, Goldberg *et al.*[23] have suggested that when the CPG is activated by repetitive stimulation of the cortex, it pro duces RJM by modulating the short-latency cortical excitatory and inhibitory pathways. They postulate no direct effect of the CPG on the motoneurons. However, Lund *et al.*[5] have shown that stimulation of the postero-lateral part of the rabbit cortical masticatory area produces the RJM with no or slight evidence of activation of the short-latency pathways. They have suggested that the CPG is acting directly on the motoneurons and is not simply acting as a gate for the short-latency responses. In the present study, although stimulus-locked activities of the digastric EMG resulting from the activation of the short-latency pathway were observed in the A-RJM, no evidence of the activation of the short-latency pathway could be found in the P-RJM. Therefore, as far as P-RJM is concerned, as suggested by Lund *et al.*[5], we agree that the CPG controls motoneurons directly and activation of the short-latency pathways is not essential for generation of RJM. However, there remains the possibility that it is essential for generation of the A-RJM.

Iwata *et al.*[6] found two separate jaw and orofacial motor areas in the cat cortex. One area is the anterior part of the coronal and lateral sigmoid gyri (C-area) and the other is the anterior part of the orbital gyrus (O-area). They have described that short train stimulation of the C-area evokes a simple movement like a jaw-opening and that of the O-area evokes two or more movements of the jaw and orofacial region, and RJM can be evoked only by repetitive stimulation of the O-area. Furthermore, Itoga *et al.*[24] have described that most of the O-area-induced movements disappear after ablation of the C-area, but the C-area-induced movements are not affected by ablation of the O-area. Suzuki *et al.*[25] have found that these two areas have, not only an independent projection to the brain stem, but also a reciprocal projection with intense projection from the O-area to the C-area. From these lines of evidence, they have postulated that the C-area is the primary motor area and the O-area is the premotor area for the jaw and orofacial region. Although they have not evoked RJM from the C-area, it seems to correspond to the A-area in the present study. However, the O-area does not correspond to the P-area because, under the present stimulatory conditions, we could not evoke the complex short-latency responses which they have evoked from the O-area, and ablation of the A-area did not show an apparent effect on the P-RJM. Therefore, at least in the rat, it would seem that two separate cortical areas, which evoke different types of RJM, independently control the CPG despite the reciprocal projection between two cortical areas.

In the present study, although the burst activities of the masseter EMG occurred simultaneously with the closing phases in the A-RJM, weak burst activities of the masseter EMG in the P-RJM did not occur in the closing phases. In the preliminary experiments, we recorded EMG activity from the superficial temporalis and medial pterygoid muscles. Both muscles did not show the activity occurred simultaneously with the closing phase in the P-RJM. We did not test the other deep situated closing muscles to avoid too much damage. It is possible that these muscles may contribute to the closing movements over the resting level during the P-RJM. However, further investigation will

be necessary to clarify which muscle contributes to the closing movement in the P-RJM.

Although the pattern of the P-RJM resembles natural mastication, it is hard to say if this area plays a main role in natural mastication since there are many regions which can produce RJM by repetitive stimulation in the brain. For example, stimulation of the lateral amygdaloid nucleus of the rabbit[26] and cat[27] elicits the RJM with closing-dominant movements. Furthermore, various forebrain and midbrain structures can also evoke RJM[28,29]. In natural mastication, these areas may function in a coordinated manner, give the proper inputs to the CPG and produce an optimal pattern of RJM.

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抄録：ラットの大脳皮質の二つの領域の連続電気刺激により，二つの異なる型のリズミカルな顎運動（RJM）が誘発された。第1型は第1次顎運動野から誘発され，常に開口運動が先行し，高頻度（5～7 Hz）の単純な顎の開閉運動からなるものであった。前顎二腹筋の筋電図活動は，個々の刺激に対し一定の潜時で応じる刺激結合型のものであった。第2型は島皮質腹側部から誘発された。第1型と対照的に，この型の RJM は多くの例で閉口運動から開始した。大きく複雑な側方および前後運動を持ち，RJM の頻度は低かった（3～4 Hz)。前顎二腹筋筋電図は刺激結合型成分を持たなかった。これらの RJM の型は刺激部位により決定され，刺激強度や頻度の変化には影響されなかった。これらの皮質 RJM 領域の片方を破壊しても，他方の刺激により誘発される RJM の型には影響がなかったので，これらの領域は互いに独立して機能していると考えられる。

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