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The Cingular Vocalization Pathway in the Squirrel Monkey

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Summary. In 39 squirrel monkeys (Saimiri sciureus), the effects of various brain lesions on vocalizations elicited from the precallosal cingulate gyrus were tested. It was found that lesions abolishing the "cingular vocalization" completely can be traced from the stimulation site continuously down to the laryngeal motoneurons in the nucleus ambiguus. The pathway thus determined (Fig. 4) travels from the precallosal cingulate gyrus through the frontal white matter and enters the internal capsule from a dorsolateral position. The pathway then follows this structure in a medio-caudal direction down to the caudal diencephalon. Here, the effective lesions leave the corticospinal tract and ascend dorsally into the periaqueductal grey. The pathway follows this structure to its end where it sweeps lateral through the parabrachial area and then descends through the lateral pons and ventrolateral medulla to the nucleus ambiguus.

In nine of the animals, in addition, the effects of bilateral anterior cingular lesions on vocalizations elicited in other brain areas were tested. It was found that the only vocalization-eliciting area which becomes ineffective after destruction of the anterior cingulate gyrus is the postero-medial orbital cortex.

Key words: Cingulate gyrus – Vocalization – Squirrel monkey

There are several observations which indicate that the anterior cingulate gyrus is involved in phonatory processes. Electrical stimulation of this area yields vocalization in the rhesus monkey (Smith, 1945; Kaada, 1951; Showers and Crosby, 1958; Hughes and Mazurowski, 1962; Robinson, 1967) and squirrel monkey (Dua and MacLean, 1964; Jürgens and Ploog, 1970). Lesions within the anterior cingulate cortex severely affect conditioned vocalization in the rhesus monkey (Sutton et al., 1974) and cause a more or less prevalent inertia to speak in man (Barris and Schuman, 1953; Botez and Barbeau, 1971; Buge et al., 1975; Rubens, 1975). Single unit recording in the rhesus monkey reveals

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that there are a number of cells within the anterior cingulate gyrus which consistently change their activity 200–800 ms before vocalization (Sutton et al., 1978). Anatomical studies show that the cingular vocalization area is connected directly with the majority of other vocalization-eliciting brain areas (Müller-Preuss and Jürgens, 1976; Jürgens and Müller-Preuss, 1977) and that its most dorsal part receives a direct input from the cortical vocal fold representation within the motor face cortex (Jürgens, 1976a). An investigation of the motivational effects accompanying gyrus cinguli-elicited vocalizations reveals that these vocalizations cannot be explained as secondary reactions due to stimulus-induced motivational changes (Jürgens, 1976b).

In two recent studies, we have investigated the anatomical projections of the cingular vocalization area (Müller-Preuss and Jürgens, 1976; Jürgens and Müller-Preuss, 1977). These studies did not decide which of the many pathways starting in the anterior cingulate gyrus is or are responsible for the vocal response evoked from this area. The present study is an attempt to answer this question.

Methods

Thirty-nine squirrel monkeys (Saimiri sciureus) were provided with a stereotaxic platform cemented to the skull (for details of the technique see Jürgens and Ploog, 1970). The platform contained a number of electrode guides through which holes were drilled in the underlying bone, followed by the introduction of electrodes into the brain. Stimulation and lesioning electrodes consisted of a 0.47-mm stainless steel tube with a 0.15-mm Teflon-insulated steel wire protruding 2 mm out of the tube. The tip of the wire was bared for 1 mm.

The awake animals were first explored by electrical brain stimulation for vocalization-eliciting loci in the precallosal cingulate gyrus. For this purpose, electrodes were lowered in 1-mm steps through this area until a point was found whose stimulation yielded vocalization. The electrical stimuli used for testing were biphasic rectangular pulses with a repetition rate of either 10, 30, or 70 Hz, a pulse width of 1 or 3 ms and an amplitude of 0.1–0.4 mA. If the elicited vocalization proved to be reproducible, lesioning electrodes were implanted into brain areas anatomically connected with the cingular vocalization area, and lesions were made. The lesions were high-frequency coagulations with an approximate diameter of 2 mm. If the lesion had no effect on the vocalization evoked from the anterior cingulate cortex, it was enlarged in most cases. The enlargement was carried out by either moving the electrode 1.5 mm up or down and repeating the coagulation or, if a rostro-caudal or medio-lateral enlargement was desired, by implanting an additional lesioning electrode immediately adjacent to the first one (1.4 mm tip-to-tip distance). The total lesions, therefore, were different in size and consisted of between one and 17 coagulations.

At later stages of the study, when parts of the "cingular vocalization pathway" had been clarified, stimulation electrodes were implanted not only in the anterior cingulate gyrus but also along the pathway so far determined. In this way several lesions could be tested at different brain levels within the same animal. In the beginning, lesions were made bilaterally; in these cases the first lesion was always made ipsilateral to the stimulation electrode. After it had been found that unilateral lesions alone may block the vocalization, testing was carried out in each brain half separately.

In a number of additional experiments, stimulation electrodes were implanted along the "cingular vocalization pathway" and other vocalization areas directly connected with the cingulate gyrus to test the effects of cingular lesions on these vocalizations.

All vocalizations were recorded on tape. Most of them were analyzed spectrographically with a Kay Sonagraph. The type of call elicited was quite uniform being either "peeping" or "keckering" or

both; a detailed description of the acoustic characteristics and functional significance of these calls is given by Winter et al. (1966).

All experiments were concluded by perfusing the animals with warm physiological saline followed by 4% formaldehyde. The brains were then removed and freeze-cut. The sections were stained alternately with Cresyl violet and with Luxol fast blue – Nuclear fast red.

Results

Altogether the effects of 112 lesions ipsilateral to the stimulation site were tested. The lesions were distributed throughout the projection system of the cingular vocalization area and further caudally down to the level of the nucleus ambiguus. According to the effects, the lesions could be classified into two groups: (1) lesions abolishing completely the gyrus cinguli-elicited vocalizations (or their caudal equivalents). (2) lesions with either no effect or partial effect (partial effects were either increases in threshold or changes in quality of vocalization).

Figures 1–3 represent the results according to this classification in a semi-schematic form. The lesions are not shown in their full rostro-caudal extent but each lesion is represented on only one AP plane. This plane corresponds within ± 0.75 mm to the frontal section with the greatest extent of the lesion. In those cases where a lesion abolished vocalization after the second or a later coagulation, not only the effective coagulation but the whole lesion is shown in the diagrams. The effective lesions (right-hand side of diagrams) at the level of the genu of the corpus callosum are found near the dorsolateral edge of the head of the caudate nucleus. Somewhat more caudally, they invade the internal capsule. Within this structure there is a shift from lateral to medial the more caudally the lesion is situated. At the transition between telencephalon and diencephalon the responsible fibers seem to have reached the most ventro-medial part of the internal capsule. The effective lesions can be then followed in the same relative position down into the cerebral peduncle. At the diencephalic-mesencephalic junction, the effective lesions suddenly leave the cortico-spinal bundle and ascend dorsally. Here they are found in the medial substantia nigra, ventral tegmental area of Tsai, and medial part of Forel's field H. With the appearance of the posterior commissure, the effective lesions cluster around the aqueduct. They remain in a periaqueductal position throughout the mesencephalon. At the mesencephalic-pontine junction, there is again a shift of the effective lesions which now take a lateral position. This lateral position is maintained down into the medulla. The effective lesions in the upper pons are situated in the lateral parts of the parabrachial nuclei and the trigeminal motor nucleus; in the lower pons, they lie immediately dorsal to the superior olive; in the medulla, they overlap with the nuclei facialis and ambiguus.

Lesions which result in an increase in threshold all lie in the vicinity of lesions with complete effects. Lesions consisting of several coagulations occasionally showed an increase in threshold one coagulation before a complete effect was obtained. Changes in quality of vocalization also occurred after lesions quite distant from the "cingular vocalization pathway", as for instance in

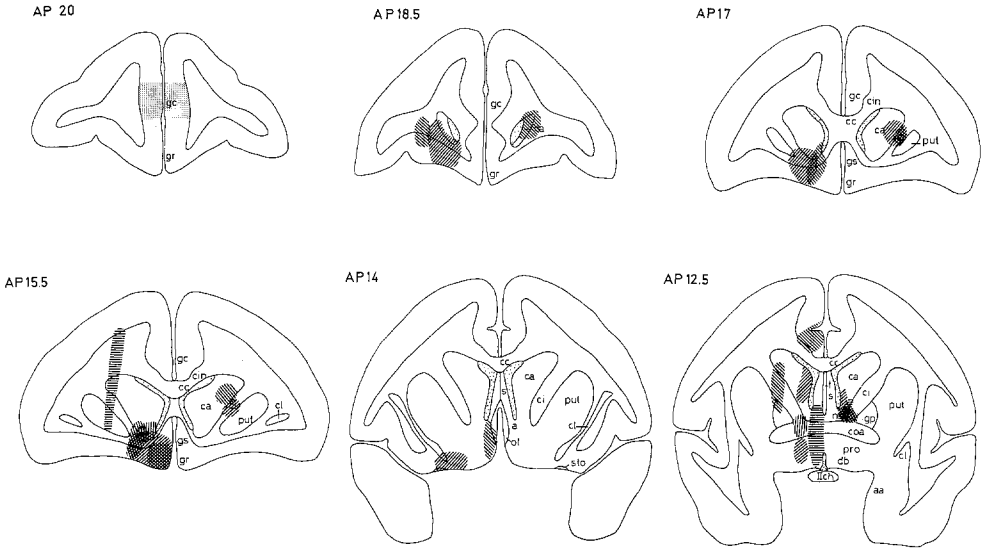

Fig. 1. Frontal sections of the squirrel monkey's brain indicating the cingular vocalization area (dotted area at AP 20) and lesion sites (hatched areas). Lesions which were effective in abolishing ipsilateral gyrus cinguli-vocalizations are shown on the right-hand side of the diagrams, ineffective lesions on the left-hand side

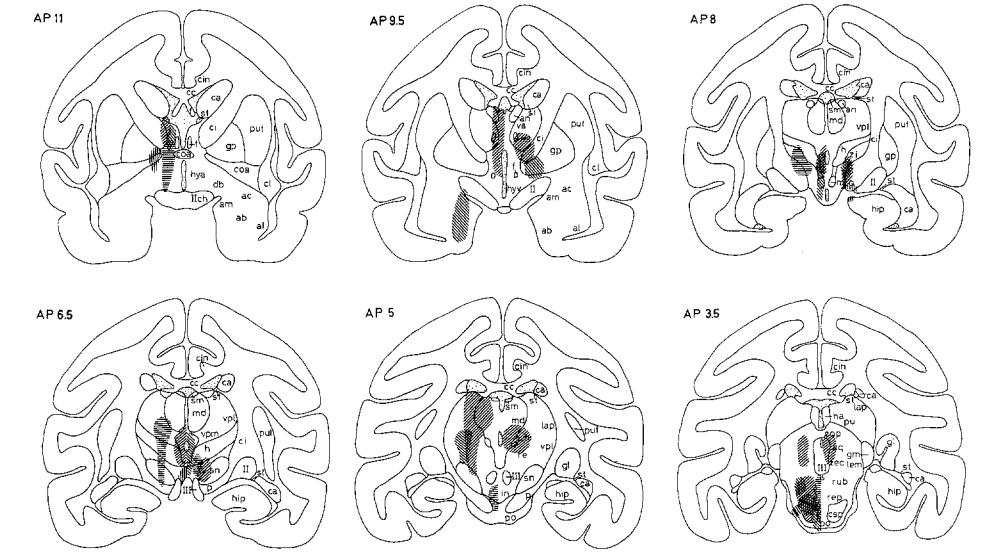

Fig. 2. For explanation see Fig. 1

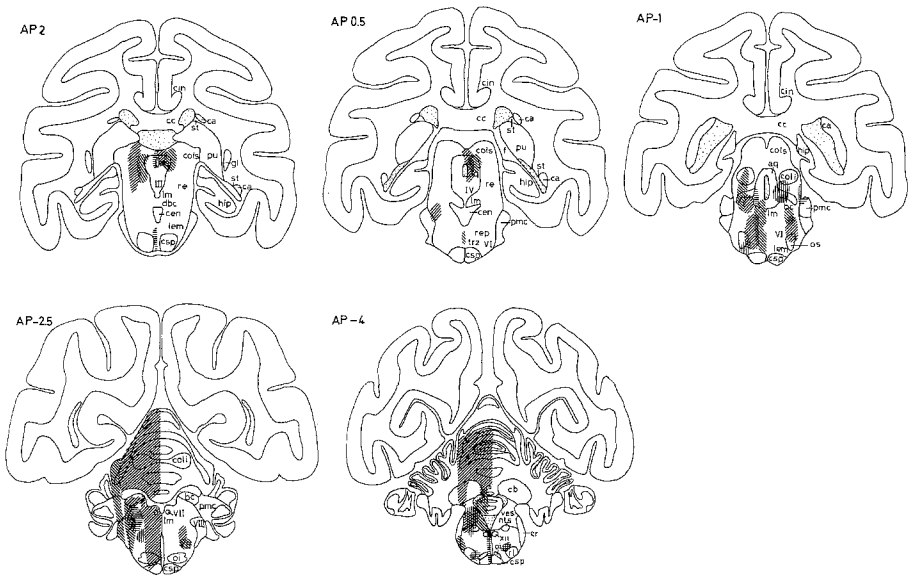

Fig. 3. For explanation see Fig. 1

the nucleus solitarius, ascending brachium conjunctivum and medullary medial lemniscus. These lesion effects will be the subject of a later publication (Jürgens and Pratt, in prep.).

Cingular Lesion Effects

In three animals, the effects of bilateral destruction of the cingular vocalization area were tested on vocalizations elicited along the "cingular vocalization pathway". The stimulation electrodes were placed in the periaqueductal grey, in the caudal periventricular grey of the diencephalon and at the ventromedial edge of the internal capsule just behind the anterior commissure. In all three cases, the vocalizations were not affected by the cingular lesions.

In another six animals, vocalization-eliciting electrodes were implanted in brain areas outside the "cingular vocalization pathway" but known to receive direct projections from the cingular vocalization area. The electrodes were located in the postero-medial orbital cortex (gyrus rectus), nucleus striae terminalis, substantia innominata, central amygdaloid nucleus, dorsomedial hypothalamus, midline thalamus, ventrolateral, and dorsolateral midbrain tegmentum. Bilateral lesions within the cingular vocalization area left all these vocalizations undisturbed except those from the gyrus rectus. Vocalizations from the gyrus rectus were abolished not only after anterior cingular lesions, but also after interruption of the "cingular vocalization pathway" dorsal to the gyrus rectus and outside the projection system of the latter. A lesion immediately caudal to the gyrus rectus, destroying the diagonal band of Broca, olfactory

tubercle, medial forebrain bundle, ventromedial caudato-putamen and ventromedial capsula interna was without effect on the vocalization elicited from the gyrus rectus.

In two of the nine animals with bilateral anterior cingular lesions, the vocal reaction to an external stimulus was tested. For this purpose a dummy leopard was presented to the animals immediately before and after cingular lesioning. In both animals, the vocal sequence appropriate to this situation (alarm peeping, yapping, and, during approach of the leopard, cawing and shrieking, were the same before and after the lesions.

Discussion

The results show that there is a discrete ipsilateral pathway connecting the cingular vocalization area with the laryngeal motoneurons in the nucleus ambiguus. Lesions anywhere within its course block the transmission of the vocalization-inducing neural activity of the anterior cingulate gyrus. The study does not answer the question of how many synapses are involved in this pathway. In our anatomical studies (Müller-Preuss and Jürgens, 1976; Jürgens and Müller-Preuss, 1977), no direct connections between gyrus cinguli and nucl. ambiguus were found. There is, however, a direct connection between nucleus ambiguus and two of the most caudal terminal fields of the cingular projection system, namely the periaqueductal grey and laterally adjacent parabrachial nuclei (Jürgens and Pratt, in press). This means that the "cingular vocalization pathway" contains at least one synapse either in the periaqueductal grey or adjacent parabrachial area.

The course of the "cingular vocalization pathway" according to the lesion experiments follows exactly the course of the cingular efferents found in the anatomical studies. The only difference is that the former represents just one component of the latter. Whereas the anatomically determined projections show manifold branching, wide spreading and looping, the functionally determined pathway is linear and circumscribed. Figure 4 summarizes the course of the latter. On leaving the cingulate gyrus, the effective fibers traverse the frontal white matter and enter the internal capsule from a dorsolateral position at about the level of the genu of the corpus callosum. Within the capsula interna, the fibers descend in a mediocaudal direction down to the caudal diencephalon. In our anatomical studies we found that a great number of cingular efferents leave the internal capsule in the most rostral part of the diencephalon. These fibers partly join the periventricular fiber system, partly follow the inferior thalamic peduncle into the midline thalamus and amygdala. The amygdalar and periventricular fiber contingent, in addition, is reinforced by cingular efferents taking their course through the external instead of the internal capsule. All these projections seem to be without significance for the gyrus cinguli-elicited vocalizations. According to the anatomical studies, cingular efferents leave the cortico-spinal pathway (internal capsule, cerebral peduncle, pyramidal tract) throughout the diencephalon, mesencephalon, and pons in order to join the periventricular/periaqueductal fiber system. The present study, however, shows

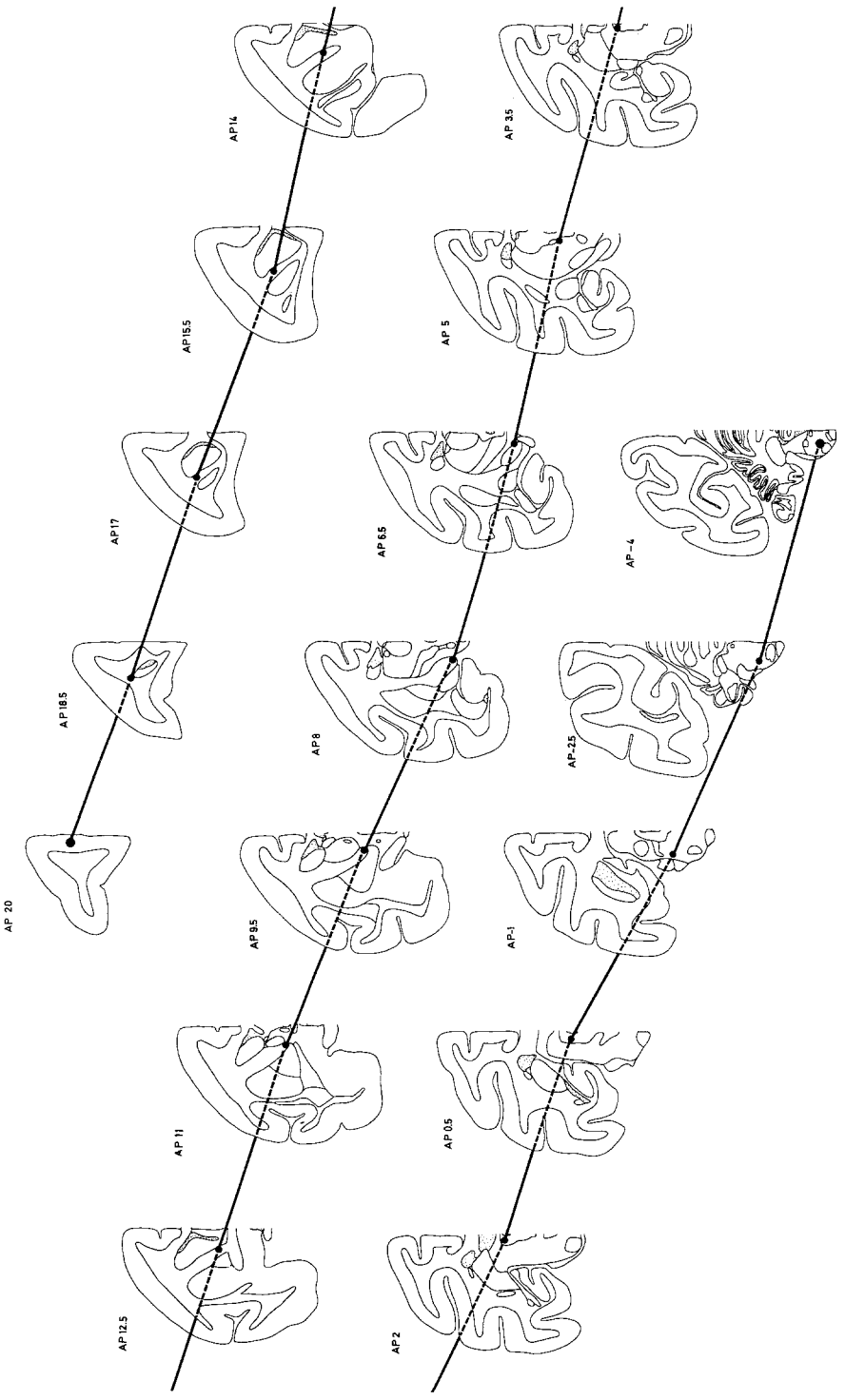

Fig. 4. Summary of the cingular vocalization pathway as determined by the lesion experiments

that the fibers responsible for vocalization leave the cortico-spinal pathway only at the caudal diencephalic level.

On leaving the cerebral peduncle, the effective fibers ascend dorsally to the periaqueductal grey and follow its course down to the mesencephalic-pontine junction (isthmus). Within the rostral half of the periaqueductal grey, the effective fibers occupy a dorsolateral position, in its caudal part they shift slightly ventralward just before they leave this structure with a sharp sweep lateralward into the parabrachial area. The caudal periaqueductal grey and laterally adjacent parabrachial nuclei are the most caudal structures of the "cingular vocalization pathway" which receive direct projections from the anterior cingulate gyrus. In recent anatomical studies (Jürgens and Pratt, in press), we found that the main descending projections from the periaqueductal grey take a lateral course through the parabrachial nuclei and then descend through the lateral pons ventrocaudalward. On reaching the dorsal border of the superior olive, the main fiber contingent turns in a purely caudalward direction until it reaches the nucl. ambiguus. As can be seen from Fig. 4, this projection system is identical with the "cingular vocalization pathway" as determined by the lesion experiments.

In considering the course of the "cingular vocalization pathway" as a whole, one wonders why there is the detour through the periaqueductal grey instead of the more direct route which would follow the corticospinal pathway down into the medulla and then pass dorsolateralward to the nucleus ambiguus. One possible explanation would be that the periaqueductal grey/parabrachial area represents a crucial relay station in the "cingular vocalization pathway". This suggestion is supported by our observation that this area represents the caudalmost terminal field of the cingular projection system. The role played by the periaqueductal-parabrachial area in call production, however, seems to be even more important. De Molina and Hunsperger (1962) observed that hypothalamus- and amygdala-evoked growling in cats is also abolished by periaqueductal lesions. The same authors and a number of others (Kelly et al., 1966; Skultety, 1958; Adametz and O'Leary, 1960; Randall, 1964) also found that periaqueductal lesions interfere with spontaneous vocalization in cats; that is, such lesions often cause transient and sometimes even permanent mutism. In our own stimulation experiments (Jürgens and Ploog, 1970), we found that the shortest latencies for electrically elicited vocalizations are found in the periaqueductal grey. Furthermore, in contrast to most other vocalization-eliciting brain areas, this structure shows almost no habituation of the vocal reaction to the electrical stimulation. That is, the animal vocalizes throughout the duration of the stimulation.

All these findings indicate that the periaqueductal-parabrachrial area plays a more general role in vocalization production.

In our anatomical studies (Müller-Preuss and Jürgens, 1976; Jürgens and Müller-Preuss, 1977), it was found that almost all vocalization-eliciting areas within the brain receive direct afferents from the cingular vocalization area. As this area is, at the same time, the most rostral of all vocalization-eliciting brain structures, it was assumed that the anterior cingulate gyrus might represent a higher order vocalization area. This suggestion was corroborated by our

self-stimulation results (Jürgens, 1976b) which indicate that the gyrus cinguli-evoked vocalizations seem to be independent of the accompanying stimulation-induced motivational changes. The present study, nevertheless, shows that the significance of the anterior cingulate gyrus for call production is quite limited. Bilateral destruction of this area neither abolishes subcortically evoked vocalizations, nor changes their quality, nor affects the "spontaneous" vocal reaction to external alarm stimuli. The control exerted by the anterior cingulate gyrus on the subcortical vocalization areas is therefore facilitatory or inhibitory rather than one of coordination; that is, the vocalization-specific neural patterns can be produced subcortically without the participation of the anterior cingulate cortex.

The only vocalization area found in the present study which seems to be totally dependent upon the anterior cingulate gyrus is the posteromedial orbital cortex (gyrus rectus). Vocalization loci within the latter become ineffective after destruction of the cingular vocalization area. Theoretically, this dependency could be explained in two ways. Either, the gyrus rectus needs a specific input from the anterior cingulate gyrus which cannot be simulated by the electrical stimulation; or, the anterior cingulate gyrus is a necessary relay station on the way from the gyrus rectus to the laryngeal motoneurons. The latter interpretation is favoured by our findings that large lesions immediately caudal to the gyrus rectus (thus blocking its direct caudal output) do not affect gyrus rectus vocalizations, while lesions interrupting cingular efferents which by-pass the gyrus rectus by a distance of several mm are effective. This interpretation is further supported by the observation that gyrus rectus-vocalizations have much longer average latencies than gyrus cinguli-vocalizations (8.5 s against 1.5 s).

With regard to the function of the cingular vocalization area in call production in general, the present study has yielded more negative than positive results. That is, the results show in which cases the anterior cingulate gyrus is dispensable, but not (except for the gyrus rectus) where it is needed. For a positive characterization, we have to go back to the findings mentioned in the introduction, namely, that anterior cingular lesions interfere with conditioned vocalization in the rhesus monkey (Sutton et al., 1974) and reduce speed fluency in man (Barris and Schuman, 1953; Botez and Barbeau, 1971; Buge et al., 1975; Rubens, 1975). From these observations and the fact that cingular lesions do not interfere with call structure, it may be concluded that the anterior cingulate gyrus plays a major role in the voluntary initiation of vocal utterances.

A still open question is the relationship between anterior cingulate gyrus and supplementary motor area in man. In contrast to the monkey, electrical stimulation of the anterior cingulate gyrus in man does not yield vocalization (Talairach et al., 1973). There is an area, however, somewhat dorsocaudal to the anterior cingulate gyrus, the so-called supplementary speech area, which when electrically stimulated does yield vocalization in man (Brickner, 1940; Penfield and Welch, 1951). This area lies just above the cingulate sulcus and about 4 cm in front of the central sulcus. Although in one sub-species of the squirrel monkey, vocalization can be elicited also from above the cingulate sulcus, the effective sites lie at about the same level as the genu of the corpus callosum and thus are clearly localized rostral to the supplementary speech area in man

(Jürgens and Ploog, 1970). Lesions within this latter area in man produce the same kind of effects as anterior cingular lesions in monkey and man, namely difficulties in the voluntary initiation of vocal utterances (Botez and Barbeau, 1971). These difficulties manifest themselves as a reduction of verbal utterances, difficulty in finding words, speech hesitation, decreased voice volume, monotony – but intact articulation. It is possible, therefore, that the anterior cingulate gyrus and supplementary motor area do not represent functionally separate brain structures but instead form a functionally coherent area. On this view, the supplementary motor area would not have gained its phonatory function in parallel with and independently of the anterior cingulate gyrus but would have taken over the latter's function progressively during phylogeny. This limbic-neocortical shift of the "cerebral representation of vocalization" probably reflects the shift from an innately preprogrammed vocal repertoire to one consisting predominantly of learned articulatory patterns. The supplementary motor area is a structure which anatomically seems to be especially suited to this taking-over of vocal function as it lies in the immediate vicinity of the phylogenetically old vocalization area but, in contrast to the latter, receives a massive input from classic motor pathways, like the cerebello-thalamo-cortical system (Hassler, 1964) or projections from the primary motor face cortex (Jürgens, 1976a). This close relationship with the motor system, however, seems to be a prerequisite for mastering such complex motor tasks as human speech – even if the task consists "only" of the initiation of this behavior.

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Abbreviations

a = nucl. accumbens
aa = area anterior amygdalae
ab = nucl. basalis amygdalae
ac = nucl. centralis amygdalae
al = nucl. lateralis amygdalae
am = nucl. medialis amygdalae
an = nucl. anterior thalami
aq = griseum centralis
bc = brachium conjunctivum
ca = caudatum
cb = cerebellum
cc = corpus callosum
cen = nucl. centralis superior Bechterew
ci = capsula interna
cin = cingulum
cl = claustrum
coa = commissura anterior
coli = colliculus inferior
cols = colliculus superior
cop = commissura posterior
cr = corpus restiforme
csp = tractus corticospinalis
db = fasciculus diagonalis Brocae
dbc = decussatio brachii conjunctivi
f = fornix
gc = gyrus cinguli
gl = geniculatum laterale
gm = geniculatum mediale
gp = globus pallidus
gr = gyrus rectus
gs = gyrus subcallosus
h = area tegmentalis (Forel)
ha = habenula
hi = tractus habenulo-interpeduncularis
hip = hippocampus
hya = hypothalamus anterior
hyv = hypothalamus ventromedialis
in = nucl. interpeduncularis
lap = nucl. lateralis posterior thalami
lem = lemniscus medialis
lm = fasciculus longitudinalis medialis
m = nucl. mammillaris

md = nucl. medialis dorsalis thalami
mt = tractus mammillothalamicus
nst = nucl. striae terminalis
nts = nucl. solitarius
oi = oliva inferior
ol = fasciculus olfactorius (Zuckerkandl)
os = oliva superior
p = pedunculus cerebri
pmc = brachium pontis
po = griseum pontis
pro = area praeoptica
pu = nucl. pulvinaris
put = putamen
re = formatio reticularis mesencephali
rep = nucl. reticularis tegmenti pontis
rl = nucl. reticularis lateralis
rub = nucl. ruber
s = septum
sm = stria medullaris
sn = substantia nigra
st = stria terminalis
sto = stria olfactoria lateralis
tec = tractus tegmentalis centralis
trz = corpus trapezoideum
va = nucl. ventralis anterior thalami
ves = nucl. vestibularis
vpl = nucl. ventralis posterior lateralis th.
vpm = nucl. ventralis posterior medialis th.
zi = zona incerta
II = tractus opticus
IIch = chiasma n. opticorum
III = nucl. n. oculomotorii and n. oculomotorius
IV = nucl. n. trochlearis
VI = n. abducens
VII = nucl. n. facialis and n. facialis
VIII = n. acusticus
XII = nucl. n. hypoglossi

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