ACTA BIOLOGIAE EXPERIMENTALIS

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AMYGDALOID COMPLEX OF THE DOG

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The amygdaloid complex occupies a space bordered anteriorly by the substantia innominata, medially by the cortex of the hippocampus and the inferior corner of the lateral ventricle, dorsally by the optic tract, putamen and internal capsule, laterally by the external capsule, and ventrally by the pyriform cortex. A number of nuclei, differing both in the density of fibre texture and in the size and stainability of cells, can be distinguished within the amygdaloid complex.

The amygdaloid complex was described by many authors. Völsch (1910) dealt with the amygdaloid complex of the ferret, lemur and macaque monkey, Jansen jun. with that of the whale, while Humphrey studied this system in the rat, which was described also by Gurdijan (1928), Craige (1925) and Brodal (1947). They applied different divisions of the amygdaloid complex, partly conformable to each other, partly varying.

The subject of the present work is the amygdaloid complex of the dog and its purpose has been to isolate individual nuclei from the amygdaloid complex of the dog and to make model of them which, it hoped, may be of some use in neurophysiological studies. Since the boundaries between the individual nuclei were frequently very indistinct (the nuclei merge with each other), the boundaries assumed were in many cases the conventional ones. The size, arrangement and stainability of the nuclear cells, as well as the course of fibres within the nuclei were regarded as the criteria of division.

MATERIAL AND METHOD

The model was made on the basis of the frontal sections of the dog brain, cut at 20 μ and stained by the Klüver-Barrera method. In addition, a series of frontal sections of the dog brain cut at 50 μ and stained by the Weigert method was used, and for control the horizontal and sagittal sections were stained in the same way. Every 12th or 13th preparation was drawn in a magnification of × 20. The drawings were projected on 5-mm.-thick wax plates, which were subsequently stuck together to form the model of the amygdaloid nuclei of the dog. Then the size of each nucleus was calculated as a percentage of the whole amygdaloid complex.

RESULTS

Basal Amygdaloid Nucleus (Figs. 1, 2, 3, 4 and 5)

This is a well-developed nucleus of the amygdaloid complex, situated in its central part. Laterally, it borders on the lateral amygdaloid nucleus, with which it merges without and manifest boundary. Ventrally, and somewhat medially, it neighbours upon the cortical amygdaloid nucleus, from which it is separated by a fairly wide band of rare fibres coming from a system which looks as if it was an extension of the external capsule. Medially, the basal amygdaloid nucleus is bounded by the medial amygdaloid nucleus, from which it is separated distinctly by the radiation of the stria terminalis.

The basal amygdaloid nucleus is irregular in shape, somewhat similar to a sector of a sphere and occupies about 21 per cent of the total volume of the amygdaloid complex. It is composed of large cells (about 17 μ in diameter), well-staining with the Nissl method and evenly arranged all over the nucleus. The whole nucleus is crossed by a great many fibres running in all directions. On a close examination it can be seen that the single fibres, running from the dorsomedial side to the ventrolateral, predominate. They come from the stria terminalis.

Lateral Amygdaloid Nucleus (Figs. 1, 2, 3, 4, 5 and 6)

The lateral amygdaloid nucleus, one of the large nuclei of the amygdaloid complex, lies most laterally. It constitutes about 37 per cent of the whole mass and is situated laterally to the basal amygdaloid nucleus. It borders dorsally on the putaminal nucleus, separated from it by apparent fibres, which seem to run from the internal to the external capsule. Laterally it neighbours upon the external capsule, which makes a well-defined boundary between it and the claustrum. Ventrally, the lateral amygdaloid nucleus merges with the cortical amygdaloid nucleus without a distinct boundary. Medially, in the anterior part, it adjoins the basal amygdaloid

nucleus, with which it fuses without a clear-cut boundary, while more posteriorly, where the basal amygdaloid nucleus vanishes, it reaches the lateral ventricle, being separated from it by a dense network of fibres.

The lateral amygdaloid nucleus is more or less semilunar in shape, and it is composed of fairly large cells (about 12 μ in diameter), well-staining with the Nissl method. They are irregularly disposed within the nucleus, forming numerous accumulations and rarefactions. A small number of fine fibrils, coming from the stria terminalis and running in all directions, pass through the lateral amygdaloid nucleus.

Cortical Amygdaloid Nucleus (Figs. 1, 2, 3 and 4)

This is irregular in shape and situated in the ventral and ventromedial part of the amygdaloid complex. Anteriorly the cortical amygdaloid nucleus comes into contact with the basal amygdaloid nucleus, separating it from the substantia innominata. Laterally it neighbours upon the lateral amygdaloid nucleus, into which it passes without any distinct boundary. On the border he cells of the cortical and lateral amygdaloid nuclei are so mixed that it is difficult to mark out the strict boundary between these nuclei. Dorsally the cortical amygdaloid nucleus passes into the medial, separated from it by the stria terminalis. The dorsomedial portion of the cortical amygdaloid nucleus forms the ventral wall of the inferior corner of the lateral ventricle. Medially, the cortical amygdaloid nucleus merges with the hippocampus, its boundary with the hippocampal cortex being very indistinct. It comes into contact with the optic tract in the dorsomedial portion, whereas anteriorly it borders on the nucleus of the lateral olfactory tract and dorsolaterally on the basal amygdaloid nucleus, from the latter of which it is separated by a band of rare fibres with few bundles. These fibres come partly from a system, which, being an extension of the external capsule, runs downwards and splits into two parts, a lateral and a medial, and partly from a system penetrating into the hippocampus. Within this band of fibres, the medium-sized cells of the cortical amygdaloid nucleus mix with the large cells of the basal amygdaloid nucleus.

The cortical amygdaloid nucleus occupies about 22 per cent of the volume of the amygdaloid complex. It is composed of medium-sized cells (about 9 μ) well-staining with the Nissl method.

Medial Amygdaloid Nucleus (Figs. 1 and 2)

This is an irregularly shaped nucleus occupying about 4 per cent of the amygdaloid complex mass and situated in its dorsomedial part. It is bordered dorsolaterally by the central amygdaloid nucleus and ventrally
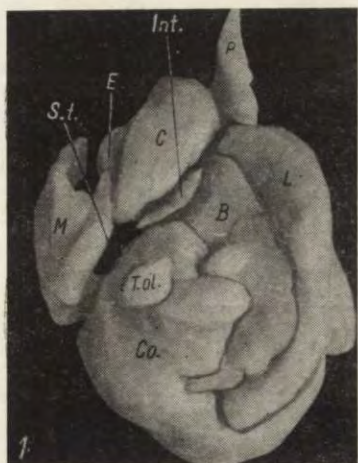
Fig. 1. Frontal view of the model of the left amygdaloid complex of the dog.
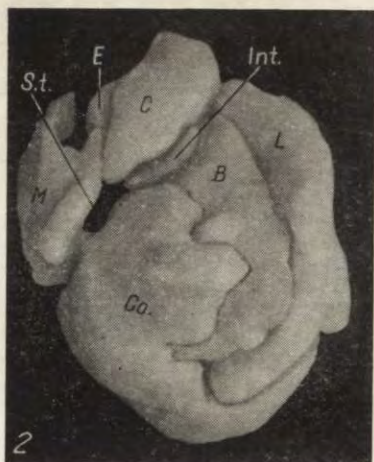
Fig. 2. As Fig. 1, after removal of the putaminal nucleus and the nucleus of the lateral olfactory tract.
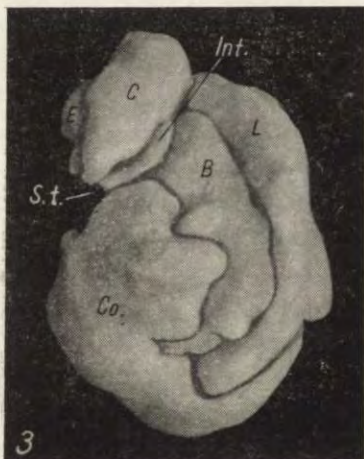
Fig. 3. Model of the left amygdaloid complex of the dog with the putaminal nucleus, nucleus of the lateral olfactory tract and medial nucleus removed.
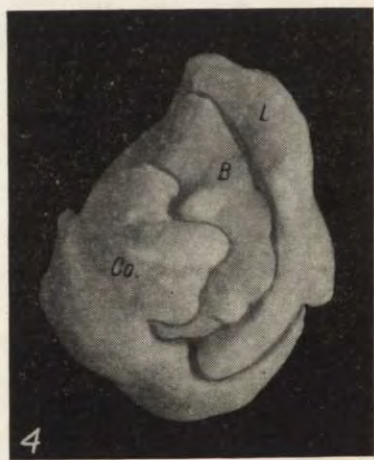
Fig. 4. Basal, lateral and cortical amygdaloid nuclei in the model of the amygdaloid complex of the dog.
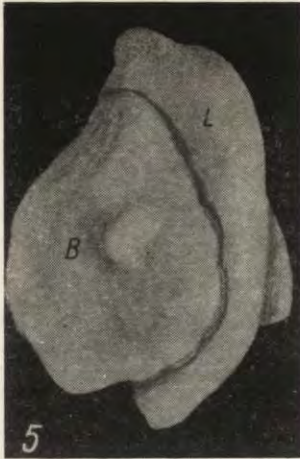
Fig. 5. Basal and lateral amygdaloid nuclei of the dog.

Fig. 6. Lateral amygdaloid nucleus of the dog.

ABBREVIATIONS FOR FIGURES

B	Central Amygdaloid Nucleus
C	Basal Amygdaloid Nucleus
Co.	Cortical Amygdaloid Nucleus
E	Nucleus E
Int.	Intercalate Nucleus
L	Lateral Amygdaloid Nucleus
M	Medial Amygdaloid Nucleus
P	Putaminal Nucleus
S.t.	Stria terminalis
T.ol.	Nucleus of the Lateral Olfactory Tract

by the cortical amygdaloid nucleus, merging with both without a distinct boundary. Dorsomedially it touches the optic tract and medially forms the wall of the lateral ventricle.

The cells of the medial amygdaloid nucleus are of medium size (5—6 μ). A small number of interwoven fibres can be seen within the nucleus.

Central Amygdaloid Nucleus (Figs. 1, 2 and 3)

This makes about 11 per cent of the volume of the amygdaloid complex. Irregular in shape, it is situated in the dorsomedial part of the complex. It passes dorsally into the putamen without any clear-cut

boundary. It is also difficult to trace its boundary with the medial amygdaloid nucleus, this being marked only in the posterior part by fine bundles of fibres, running ventromedially from the dorsolateral side. The central amygdaloid nucleus borders on nucleus E only along a short section in its dorsomedial part and is separated from it by fibres having the same course. These fibres rarefy somewhat inferiorly. The central amygdaloid nucleus is bounded ventrally by the basal amygdaloid nucleus, from which it is separated by faint and indistinct fibres coming from the stria terminalis. The intercalate nucleus holds a fairly large space between the central and the basal amygdaloid nucleus, evidently separated from them by fibres originating from the stria.

The central amygdaloid nucleus is composed of cells with a diameter of about $6\,\mu$, forming accumulations and rarefactions crossed by a small number of single fibres.

Intercalate Nucleus (Figs. 1, 2, 3)

The intercalate nucleus is one of the smallest nuclei of the amygdaloid complex, constituting barely 0.6 per cent of its volume. It is wedged in as a flat lamina between the central amygdaloid nucleus dorsally and the basal amygdaloid nucleus ventrally. Medially, it is in contact with the stria terminalis, which in sending out fibres to the depth of the amygdaloid complex, surrounds it on all sides with part of these fibres. The intercalate nucleus is composed of small cells, about $4\,\mu$ in diameter. Because of these small cells it is clearly distinguishable from the remainder of the nuclei. It shows hardly any fibres.

Putaminal Nucleus (Fig. 1)

Situated dorsolaterally, it is separated distinctly from the other nuclei by a ramification of the external capsule, and constitutes about 3 per cent of the amygdaloid complex. Triangular in shape in the frontal sections, it is composed of cells with a diameter of $9\,\mu$, fairly well-staining. Bundles of fibres pass through the whole mass of the nucleus, running ventrolaterally from the dorsomedial side and connecting the systems of the internal capsule with those of the external capsule. The putaminal nucleus is separated laterally from the claustrum by the external capsule. Ventrally, on the medial side it borders on the central amygdaloid nucleus and dorsomedially on the putamen. The ventral border runs between this and the lateral amygdaloid nucleus and is formed by a band of very fine fibres splitting from the external capsule and leading to the internal.

Nucleus of the Lateral Olfactory Tract
(Fig. 1)

This nucleus has the foremost position in the amygdaloid complex (Fig. 1). It turns out a group of medium-sized (9 µ), fairly well-stained cells amid the substantia innominata, somewhat dorsomedially to the lateral olfactory tract. Its boundaries are indistinct and it is cut across by a large number of fibres running in all directions. It occupies 0.5 per cent of the volume of the amygdaloid complex.

Nucleus E (Figs. 1, 2, 3)

This is a small, irregularly shaped nucleus situated medially to the central amygdaloid nucleus, between this nucleus and the upper portion of the stria terminalis. Nucleus E constitutes 0.7 per cent of the volume of the amygdaloid complex, and it is composed of large, rare, well-staining cells. Its ventral border is in contact with the medial amygdaloid nucleus, while the dorsal one reaches the internal capsule. Scanty, single fibres, running in all directions, pass through it.

Stria Terminalis

The stria terminalis is a large bundle of fibres entering the dorsocaudal part of the amygdaloid complex. Hence it runs anteriorly splitting into two bundles. One of them, called by V ö l s c h (1910) the sagittal bundle, runs in the dorsal portion of the amygdaloid complex without branching. Anteriorly it descends a little and mixes with the fibres of the substantia innominata. The other bundle of the stria terminalis runs ventrolaterally sending out numerous branches, which squeeze in between the particular nuclei of the amygdaloid complex. A part of the fibres extends ventrad, mixing with the fibres of the system that looks like an extension of the external capsule, and reaches the pyriform cortex. S y c h (1959) calls this a system accompanying the external capsule. Anteriorly the fibres of this system mix with those of the substantia innominata. Ventromedially a part of the second bundle of the stria terminalis also joins the system of fibres entering the hippocampus.

DISCUSSION

The foregoing division of the amygdaloid complex conforms to those applied by the other authors. Table I renders it possible to compare the results.

Table I

Maksymowicz (dog)	Jansen (fin whale)	Craigie (rat)	Gurdijan (rat)	Humphrey (bat)	Fox (cat)	Johnson (guinea pig)
Lateralis	Lateralis	Lateralis	Lateralis	Lateralis	Lateralis	Lateralis
Basalis	Basalis	Basalis	Basalis	Basalis	Basalis	Basalis
Corticalis	Corticalis	Corticalis	Corticalis	Corticalis	Corticalis	Corticalis
Medialis	Medialis	Medialis	Medialis	Medialis	Medialis	Medialis
Centralis	Centralis	Centralis	Centralis	Centralis	Centralis	Centralis
Intercalatus	Intercalatus	—	Intercalatus	Massa intercalata	Intercalatus	Intercalatus
Putaminalis	—	—	—	—	—	—
E	—	—	—	—	—	—
N. tr. olf. lat.	N. tr. olf. lat.	N. tr. olf. lat.	N. tr. olf. lat.	N. tr. olf. lat.	N. tr. olf. lat.	N. tr. olf. lat.

Unfortunately, a comparison with the studies on the human brain has failed. In humans, the amygdaloid complex along with the hippocampus is shifted and reversed, due owing to which the whole arrangement of the nuclei has changed entirely (S c h a l t e n b r a n d and B a i l e y 1959, H i l p e r t 1928). It seems also that some nuclei have undergone division. A comparison of the model of the amygdaloid complex of the dog with that of the man might help to identify these nuclei.

It is also difficult to trace particular nuclei in V ö l s c h's (1910), division (ferret, lemur), because he applies his own nomenclature and what is more, he divides the nuclei into subnuclei and combines them in various groups.

Comparing the nuclei of the amygdaloid complex of the dog with those in other animals it will be seen that in the dog, as in other mammals, the basal amygdaloid nucleus is composed of the largest cells (except for the rat, whose basal amygdaloid nucleus is quite a small-celled portion of the amygdaloid complex) (G u r d i j a n 1928, C r a i g i e 1925).

J a n s e n jun. (1953) distinguishes a large-celled lateral portion and a small-celled medial portion of the basal amygdaloid nucleus in the whale.

Similarly H u m p h r e y (1953) divides the basal amygdaloid nucleus of the bat into two parts, a magnocellular part situated laterally and a small-celled part lying medially (nucleus basalis accesorius).

In the basal amygdaloid nucleus of the macaque monkey L a u e r (1954) finds as many as six nuclei which, however, have no corresponding nuclei in the dog.

In the dog there is a slightly marked division of the basal amygdaloid nucleus into two parts, but this division is so indistinct that it seemed purposeless to divide this nucleus in the model of the amygdaloid complex. A detailed study of the myeloarchitecture of this region may lead to its division into parts.

The lateral amygdaloid nucleus, the second largest nucleus of the amygdaloid complex, has been divided by J o h n s o n (1959) (guinea-pig) into the pars anterior and the pars posterior. In the present study the lateral amygdaloid nucleus is treated as a whole, because there is no such division visible in the dog.

The division made by F o x (1940) in the cat does not vary from the remaining ones.

Nucleus E has been distinguished only by V ö l s c h (1910) and its position corresponds to the position of the same nucleus in the dog.

The anterior amygdaloid area has not been visualized in the model, though it is distinguished by many authors. It merges with the substantia

innominata so closely that it is impossible to mark out their boundary and it is difficult to recognize whether a given group of cells belongs to the anterior amygdaloid area or to the substantia innominata. Need for a myeloarchitectural study of this region again becomes apparent.

None of the autors mentions the putaminal nucleus. It is reckoned neither in the putamen nor in the amygdaloid complex in any of the studies, though in the drawings and photographs of this region in some papers it is quite visible. Situated in the bifurcation of the external capsule it may be reckoned both in the putamen and the amygdaloid complex. However, the character of the bundles of fibres passing through it as well as its closer connection with the amygdaloid complex than with the putamen speaks well for including it in the amygdaloid complex.

An interesting theory concerning the intercalate nucleus is offered by S a n i d e s (1957). He thinks that the intercalate nucleus is a piece broken off from the island of Calleja, and he finds more such broken-off pieces within the striatum. In fact, in the material of this study small-celled intercalations scattered occasionally among nuclei are to be seen within the amygdaloid complex of the dog. Nevertheless, only a minute embryological study of this region in the dog could throw light on the origin of the intercalate nucleus and of other small-celled intercalations throughout the amygdaloid complex and the striatum.

SUMMARY

The paper includes a description of the model of the amygdaloid nuclei of the dog based on the myelo- and cytoarchitectonics of this region. The nuclei distinguished are as follows: the basal amygdaloid nucleus, lateral amygdaloid nucleus, cortical amygdaloid nucleus, medial amygdaloid nucleus, central amygdaloid nucleus, intercalate nucleus, putaminal nucleus, nucleus of the lateral olfactory tract, and nucleus E, which all correspond to the nuclei described by other authors with the exception of the putaminal nucleus, which has not been described hitherto, and nucleus E which was recorded only by V ö l s c h.

REFERENCES

ADRIANOV O. S. and MERING T. A. 1959 — Atlas of the dog's brain. Moskva, (in Russian).
BRADLEY C. O. 1959 — Topographical anatomy of the dog. London.
BRODAL A. 1947 — The amygdaloid nucleus in the rat. J. com. Neur., 87, 1.
CRAIGIE E. H. 1925 — Finer anatomy of the central nervous system. Philadelphia, Pa.
FOX C. 1942 — The stria terminalis, longitudinal association bundle and praecommissural fornix fibers in the cat. J. com. Neur., 79, 277.

GURDIJAN E. S. 1928 — The corpus striatum of the rat. J. comp. Neur., *45*, 249.

HILPERT P. 1928 — Der Mandelkern des Menschen. J. Psych. und Neur., *36*, 44.

HUMPHREY T. 1936 — The telencephalon of the rat. J. comp. Neur., *65*, 603.

JANSEN J. jun. and JANSEN J. 1953 — A note on the amygdaloid complex in the fin whale (Balaenoptera physalus L.). Oslo.

JOHNSON T. N. 1957 — Studies on the brain of guinea pig J. comp. Neur., *107*, 353.

LAUER E. W. 1945 — The nuclear pattern and fiber connections of certain basal telencephalic centers in the macaque. J. comp. Neur., *82*, 215.

LAMMERS H. J. et MAGNUS O. 1955 — Étude expérimentale de la région du noyau amygdalien du chat. Extrait des Comptes Rendus de l'Association des Anatomistes, pp. 840—844.

SANIDES F. 1957 — Die Insulae terminales des Erwachsenengehirns des Menschen. J. f. Hirnforschung, *3* 243.

SCHALTENBRANDT G. and BAILEY P. 1959 — Introduction to stereotaxis with an atlas of the human brain. Stuttgart.

SYCH L. 1960 — The external capsule in the dog's brain (myeloarchitections and topography). Acta Biol. Exper. (Warsaw), *20*, 91.

VÖLSCH M. 1910 — Zur vergleichenden Anatomie des Mandelkerns und seiner Nachbargebilde. Archiv Mikroskop. Anatom. und Entwicklungsgeschichte. *76*, 373.

WINKLER C. and POTTER A. 1914 — An anatomical guide to experimental researches on the cat's brain. Amsterdam.

DEGENERATIONS OF THE MEDIAL GENICULATE BODY FOLLOWING ABLATIONS OF VARIOUS TEMPORAL REGIONS IN THE DOG

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The auditory area of the cortex has been a subject of interest for years (Vogt 1898), and the original studies dealt with the determination of its borders. The extent of the region was marked out by the physiological methods, namely, by an analysis of retrograde cell degenerations, the hearing range being checked after lesions of the temporal region and the results from the experiments confronted with the anatomical data. However, the results obtained in the studies carried out by various authors were not always identical (Yoshido 1924, Onishi 1931, Polyak 1932, Posthumus Meyjes 1934, Le Gros Clark 1936, D'Hollander and Stoffles 1937, Walker 1938).

Most authors agree that the auditory region is connected with the medial geniculate body. In the macaque monkey, the auditory area was described, as defined by the retrograde cell degeneration in the medial geniculate body, in the Sylvian fissure on the surface of the temporal lobe of the brain by Walker (1938) and Le Gros Clark (1935—1936). In the cat brain it was determined by many authors (Polyak 1927, Mettler 1932, Ades, Mettler and Culler 1939, Bremer and Down 1939, Wollard and Harpmann 1939, Waller 1940, Bremer 1943, Ades 1943, 1949, Rose 1949, 1955, Rose and Woolsey 1949, Stoll, Ajmone-Marsan and Jasper 1951, Hind 1953, Kiang 1955, Bremer, Bonnet and Te-

rzuolo 1955, Neff, Fisher, Diamond and Yela 1956, Neff 1957, 1958, Goldberg, Diamond and Neff 1957, Riss 1959) and the middle part of the medial ectosylvian gyrus (area A I), the superior part of the Sylvian gyrus and the posterior part of the anterior ectosylvian gyrus (area A II), as well as the posterior ectosylvian gyrus (area Ep) were reckoned in it.

The auditory region of the dog was worked out by few authors. In the available literature only T u n t u r i (1945—55) and M e r i n g (1952) dealt with it. M e r i n g, using the plucking method, marked out two bundles running from the medial geniculate body: a deep bundle leading from the magnocellular part and the other, superficial, from the principal part. According to the authoress, both these bundles reach the narrow cortical zone neighbouring on the suprasylvian fissure in the medial and posterior ectosylvian gyri. T u n t u r i, who based himself on the electrophysiological studies, arrived at different conclusions, his findings being analogous to those offered for the cat by the previous authors.

The present paper comprises the results of the histological studies on the brains of the dogs used previously for observations during experiments on conditioned reflexes made after ablations of the temporal area by C h o r ą ż y n a and S t ę p i e ń (1961). The dogs were allowed to live for 3 to 10 months after the operative lesion. Their brains were fixed by perfusion with a solution of formaldehyde, embedded in paraffin and sectioned at 20 μ. Every fifth section was stained by the Nissl and Klüver methods alternately.

Bilateral lesions of the temporal cortex were made in all the dogs. The extent of the lesions was various, nevertheless, the whole material may be divided into three groups. The first group includes the dogs with lesions of the anterior and posterior Sylvian gyri, the second those with lesions in the ectosylvian gyrus, while the dogs with injuries in the anterior and posterior Sylvian gyri as well as in the ectosylvian gyrus belong to the third group. In addition to these, one dog brain with asymmetrical lesions in both hemispheres is described. Within each of these groups there are individual differences in the extent and depth of ablations. The term "degeneration" is used whenever there is an isomorphous or anisomorphous gliosis in the region discussed or where cellular changes and degenerations of nerve fibres are present.

GROUP I

Discussion on observational material

The dogs with the anterior and posterior Sylvian gyri removed are characterized by having similar areas of lesions and similar resultant degenerations. The procedure consisted in the removal of all the cortex of the gyrus and, occasionally, of the white matter underlying the gyrus. The ectosylvian fissure arms generally constitute the outer limits of the lesion (dogs No. 2 left hemisphere, No. 6 right hemisphere). However,

the border of the lesion sometimes descends to the anterior rhinal fissure at the point where this fissure branches off the Sylvian fissure (dogs Nos. 3 and 6—8 left, No. 9 left and right). The outer boundary of the lesion does not always reach the ectosylvian fissure and in some lesions touches it along only short sections (dogs Nos. 2 and 9 right, Nos. 3 and 7 right and left), while in one dog (No. 8 right) a cortical zone is left undestroyed all along the ectosylvian fissure on the side adjacent to the lesion. In two cases the lesion border crosses the ectosylvian fissure (dog No. 3 left and right) passing on to the ectosylvian gyrus.

Just along the boundary the lesions are all superficial and involve only the outer cortical layers. All the cortex is ablated in the middle portion of the gyrus and, in some cases, the white matter under the gyrus (dogs Nos. 2 and 9 right and left, Nos. 3 and 8 left) or even under the Sylvian fissure (dogs Nos. 8 and 9 left) is destroyed, as well. The lesions made on both sides of the same brain show only slight differences.

In dog No. 2 (Fig. 1) the lesion of the left side is confined within the arms of the ectosylvian fissure. The juction of the arms forms the oralo-ventral boundary of the lesion. The cortex in the depth of the ectosylvian fissure is left undamaged and the lesion in the vicinity of this fissure is superficial. The injury within the area of the gyrus is deeper and includes the whole thickness of the cortex, even at the depth of the Sylvian fissure. Part of the subcortical white matter has been destroyed in the area of the anterior Sylvian gyrus on both sides. In the right hemisphere the cortex is maintained undamaged at the depth of the ectosylvian fissure and also in the form of a narrow strip running along this fissure on the anterior Sylvian side. The lesion involves the cortex of the posterior half of the Sylvian fissure, while more orally a broad zone of the cortex has been left undestroyed along this fissure. From the anterior and posterior Sylvian gyri two strips of the lesion encroach upon the anterior and posterior composite gyri. In addition to the cortex, a part of the white matter is destroyed in the anterior Sylvian gyrus in which the lession is narrower than in the posterior.

The lesions in both hemispheres of dog No. 3 (Fig. 1) cross the posterior ectosylvian fissure destroying the superficial cortical layers of the posterior ectosylvian gyrus. In addition, a narrow strip of cortex of the anterior ectosylvian gyrus, running along the ectosylvian fissure, has been damaged in the left hemisphere. On both sides the cortex at the depth of the ecto-sylvian fissure is not injured and neither is its narrow strip in the bend of this fissure. Moreover, in the right hemisphere a narrow cortical strip is left undercut along the anterior ectosylvian fissure. On both sides the cortex is destroyed in the depth of the Sylvian fissure, and the oroventral border of the lesion extends across the anterior and posterior composite

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Fig. 1. The extent of the cortical lesions in dogs and the corresponding degenerations of the nuclei of the medial geniculate body

A. Cortical area: a) lesion border, b) damage involving the outer cortical layers, c) damage involving the deep cortical layers, d) damaged white matter underlying the cortex; B. Degeneration of the medial geniculate body: a) complete degeneration, b) fairly severe partial degeneration with a large amount of glia and a small number of normal nerve-cells, c) partial degeneration with a large numbers of normal nerve-cells and a small amount of glia, d) no degeneration

gyri and reaches the point where the Sylvian fissure branches off the rhinal fissure. In this dog the deepest lesion is located in the anterior Sylvian gyrus in both hemispheres.

In dog No. 6 (Fig. 1) the lesions of both hemispheres border on the ectosylvian fissure, the cortex at the depth of this fissure being undamaged. On the left side the oroventral boundry extends down to the rhinal fissure, including the whole Sylvian fissure in the lesion. The cortex in the depth of the latter is destroyed superficially. The lesion is deeper in the anterior and posterior Sylvian gyri, where all the cortex is destroyed. On the right side the lesion does not involve the cortex in the depth of the ectosylvian fissure and the oraloventral margin of the lesion runs along the line connecting the opposite ends of this fissure.

The lesion on the right side of the brain of dog No. 7 (Fig. 1) could not be worked out in detail, for its cortex had been damaged while preparations were being made. It is, however, possible to reconstruct the lesion by describing its macroscopic appearance. In the left hemisphere the lesion covers a small area neighbouring on the whole Sylvian fissure. The cortex at the depth of the fissure is not completely destroyed, its deeper portions being left uninjured. The lesion reaches to the anterior rhinal fissure, but not to the posterior. Its boundary next runs across the anterior and posterior Sylvian gyri. On this side the lesion is comparatively shallow and involves the cortex over a small area of the convolution on either side of the Sylvian fissure. In the right hemisphere the lesion involves the anterior and posterior Sylvian gyri anteriorly. A macroscopic examination shows that it is just like the lesion of the other side and that it does not include the whole convolutions, only the areas lying close to the Sylvian fissure. On the right side the deepest portion of the lesion is in the anterior Sylvian gyrus.

The extent of the lesion in the left hemisphere of dog No. 8 (Fig. 1) is analogous to that of the ipsilateral lesion of dog No. 6. The only major difference is that in dog No. 8 the lesion is very deep in the region of the Sylvian fissure and penetrates into the white matter in the sulcus under the convolutions. In the dorsocaudal portion of the lesion, i.e. in the bend of the ectosylvian fissure the ablation is superficial and all the cortex has not been removed. The cortex is left in the depth of the ectosylvian fissure as well. In the right hemisphere of this dog the lesion is somewhat smaller, a cortical zone all along the ectosylvian fissure being undamaged. On the anterior Sylvian side the lesion extends into the anterior composite gyrus, while on the opposite side its boundary runs towards the arm of the ectosylvian fissure. The lesion of this hemisphere is shallower and, as a rule, includes the superficial layers of the cortex, only in the region of

the Sylvian fissure is all the cortex of the convolution together with the cortex of this fissure destroyed.

In dog No. 9 (Fig. 1) the lesion of the left hemisphere is similar to that in dog No. 8. It is limited by the ectosylvian fissure, and from both ends of the arms of this fissure the boundary runs down to the point where the Sylvian fissure branches off the rhinal fissure. The cortex at the depth of the ectosylvian fissure has not been destroyed and the portion of the lesion adjacent to the fissure is quite superficial. It is only in the region of the Sylvian fissure that the lesion depth exceeds the thickness of the cortex so that a portion of the white matter is included in the destruction. The lesion in the right hemisphere is a little smaller, for a cortical zone in the vicinity of the anterior and medial parts of the ectosylvian fissure is left undamaged. The lesion is very superficial: it involves the outer cortical layers and is somewhat deeper over a small area in the region of the anterior Sylvian gyrus, where it penetrates into the portion of the white matter underlying the convolution.

The lesion in dog No. 12 (Fig. 1) is the same on either side and extends on the anterior and posterior Sylvian gyri, being limited by the arms of the ectosylvian fissure. Its border runs from the tips of the arms to the junction of the Sylvian fissure with the rhinal. The lesion of the right hemisphere is superficial, confined only to the gyrus cortex, the cortex of the Sylvian and ectosylvian fissures being spared. In the left hemisphere a little more of the white matter has been removed in the area of the anterior Sylvian gyrus and the cortex of the Sylvian and ectosylvian fissures is partly damaged. The deepest portion of the lesion is situated in the anterior part of the anterior Sylvian gyrus, where the damage extends into the subjacent structures.

In dog No. 13 (Fig. 1) the lesions are analogous in both hemispheres. They involve the whole area confined within the arms of the ectosylvian fissure and extend to the rhinal fissure, including the portion of the anterior and posterior composite gyri neighbouring upon the Sylvian fissure. On both sides the lesion involves only the cortex of the convolutions and does not extend to the depth of the ectosylvian and Sylvian fissures.

The lesion in the right hemisphere of dog No. 1 (Fig. 3) is very deep and extends up to the lateral ventricle in its upper region. The anterior and posterior Sylvian gyri are removed up to the rhinal fissure. Also the cortex of the Sylvian and ectosylvian fissures as well as the white matter of the anterior and posterior Sylvian gyri is completely destroyed. In the left hemisphere the lesion is relatively shallow but involves a larger area of the gyri than on the opposite side. Here, the anterior and posterior Sylvian gyri, the medial and posterior ectosylvian gyri, the oral tip of the posterior composite gyrus, about the half of the anterior composite gyrus

(posterior portion) and the posterior section of the orbital gyrus have been removed. A cortical strip adjacent to the posterior suprasylvian fissure remains undamaged. The cortex in the depth of the rhinal, praesylvian as well as anterior and posterior ectosylvian fissures is also left uninjured. The deepest portion of the lesion lies in the region of the Sylvian fissure and extends somewhat posteriorly over the medial ectosylvian fissure.

Discussion on degenerations

In the discussion of the degenerations of the medial geniculate body destroyed as a result of the ablations described above I shall apply the division of this system into 7 separate structures, of which 6 are comprised within the area of the principal part, made up of small cells, and one in the magnocellular part (Sychowa 1962). Three of the nuclei of the principal part have connexions with the lower acoustic centres and lie in the posterior portion of the medial geniculate body. They are the superior, inferior, and ventral nuclei. Two nuclei (the lateral and dorsal nuclei) have associative connexions with the lateral geniculate body as well as with the adjacent nuclei of the medial geniculate body. One nucleus has a commissural connexion with the medial geniculate body on the other side. The auditory radiation originates from all these nuclei. The remaining large-celled portion has not undergone any changes in this division.

Lesions made in the area of the Sylvian gyrus bring about degenerations of two nuclei mainly (the superior and inferior nuclei), and, occasionally, of three (the ventral nucleus). The degenerative changes in the medial geniculate body are secondary ones. Within the degenerated nuclei a number of cells have atrophied, giving place to glial nuclei. The remaining cells assume an abnormal appearance, as if they were turgid, and their nuclei shift toward the periphery or fill the whole cell.

The intensity of the degeneration of the medial geniculate body in dog No. 2 (Figs. 1 and 2) seems to be proportional to the extent of the lesion. On the left side, where the lesion was more extensive, the degeneration is larger and involves the superior and inferior nuclei, partly the ventral nucleus and, to the lowest degree, the lateral nucleus. The superior nucleus has undergone complete degeneration, and only the glia is found within its area, no nerve-cells being present. The degeneration of the cells in the inferior nucleus is fairly severe, but besides a large amount of glia there occur numerous nerve-cells. A similar state of degeneration may be observed in the posterior portion of the ventral nucleus, whose anterior region is left intact. The lateral nucleus is partially degenerated only in the vicinity of the superior nucleus. On the right side the superior, inferior and lateral nuclei are degenerated. Only the posterior portion of
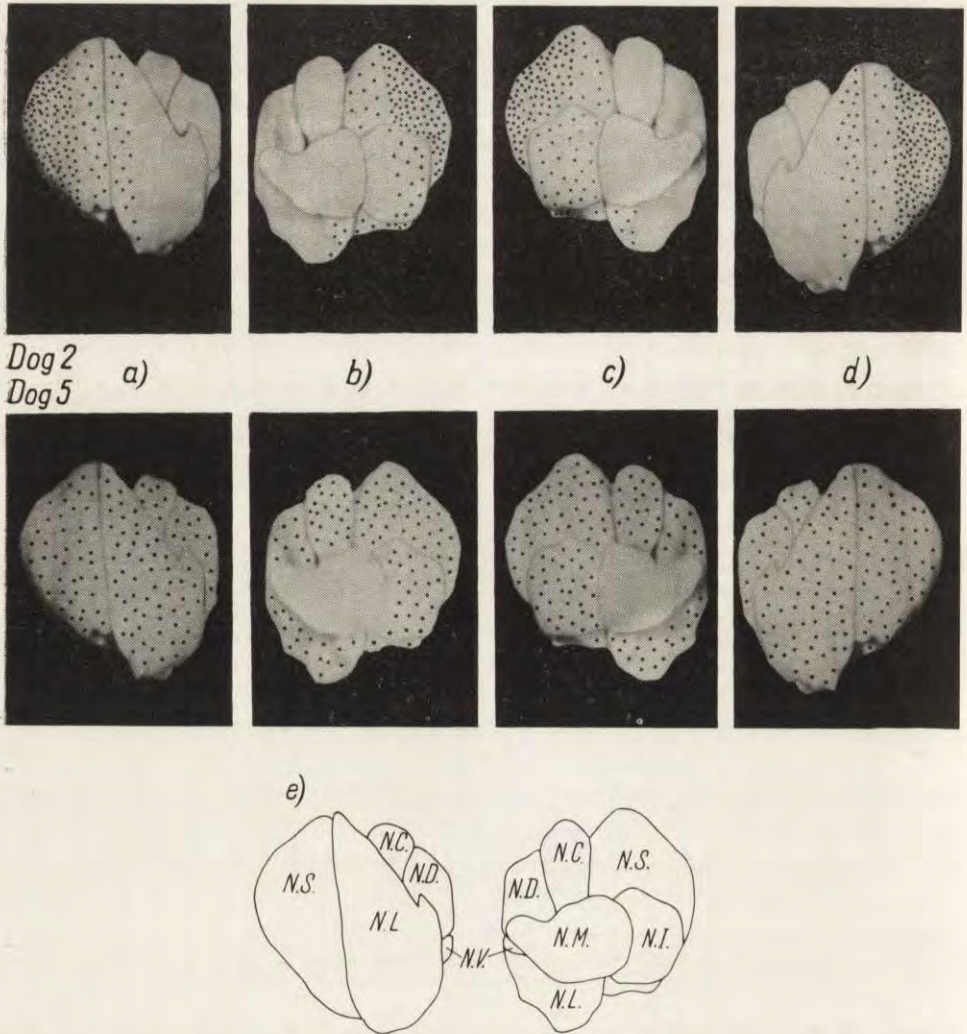
Fig. 2. The extent of the medial geniculate body following a cortical ablation as demonstrated by a wax model

a) right medial geniculate body, lateral view, b) right medial geniculate body, medial view, c) left medial geniculate body, medial view, d) left medial geniculate body, lateral view, e) nuclei of medial geniculate body: N.C., nucleus commissuralis, N.D., nucleus dorsalis, N.I., nucleus inferior, N. L., nucleus lateralis, N.M., nucleus magnocellularis, N.S., nucleus superior, N.V., nucleus ventralis

the superior nucleus is degenerated completely, its oral part having a great many normal nerve cells. The posterior region of the inferior nucleus shows a partial degeneration and so does the lateral nucleus in the part adjacent to the superior.

In dog No. 3 (Fig. 1) the degenerations of the medial geniculate body are analogous on both sides. Complete degeneration is found in the superior nucleus, where no nerve-cells are visible. The inferior and ventral nuclei have undergone partial degeneration and within their areas there are increased numbers of the glial cells and fairly numerous normal nerve--cells. The remaining nuclei of the medial geniculate body are unaltered.

In spite of the unequal cortical lesions in dog No. 6 (Fig. 1) the degenerations of the medial geniculate body are situated in the same nuclei and the difference consists only in the degree of degeneration. On the left side the superior nucleus has degenerated completely, while on the right side a small number of nerve-cells are left in it. The inferior nucleus of the right side shows a partial degeneration, but many normal cells are maintained in it. In the area of its counterpart on the opposite side there are no normal cells. In the small caudoventral portion of the lateral nucleus of either side slight degeneration of the nerve-cells is observed and the degenerated cells are replaced by the glia. In addition, slight degeneration of the commissural nucleus is noted on both sides in this dog; it is, however, a little more severe on the left side, where the lesion was more extensive. Nevertheless, a large number of normal nerve-cells are still present here.

Dog No. 7 (Fig. 1), with which I had much trouble in termining the cortical lesion exactly, shows small and rather uniform degenerations in the medial geniculate body on both sides. On the right side a small, caudoventral portion of the superior nucleus is completely degenerated. Somewhat orally to it there is an area of gradually diminishing degeneration. The anterior portion of this nucleus appears to be normal. Similarly, the superior nucleus of the left side is degenerated only in its dorsocaudal portion. This degeneration is, however, considerably milder. More normal cells are seen in the degenerated area on the left side than on the right.

The degenerations of the medial geniculate body of dog No. 8 (Fig. 1) are slightly larger on the left side and they include the superior and inferior nuclei. In the posterior portion of the superior nucleus the degeneration is complete. It diminishes anteriorly, where large numbers of normal cells are present. In the inferior nucleus the degeneration occurs in the posterior portion, accompanied by a large amount of glia. On the right side, where the cortex lesion is smaller, the degeneration of the medial geniculate body is present only in the posterior part of the superior nucleus and, similarly to what was found in the dogs discussed above, a more severe degeneration occurs at the caudal extremity of the nucleus. Followed anteriorly it fades away, and not farther than the medial region of the nucleus is the histological picture normal.

The degeneration of the nuclei of the medial geniculate body in dog No. 9 (Fig. 1) is just like that in the preceding dog. The cortical lesions in both these dogs are alike, though somewhat more extensive in the left hemispheres. On the left side the superior and inferior nuclei are degenerated more strongly, complete degeneration being present only in the posterior portion of the superior nucleus and diminishing anteriorly. In the oral part of this nucleus only a slight rarefaction of the normal nerve-cells occurs and the amount of the glia is normal. The inferior nucleus shows a lesser degeneration. Normal cells occur all over the area, though in the posterior region of the nucleus in a very small number. The number of normal cells increases anteriorly. In the right medial geniculate body the superior nucleus is only partly degenerated, in the manner resembling the degeneration of its fellow on the opposite side. The posterior portion of the medial geniculate body has degenerated most and yet the normal nerve-cells, though very few, remain in this area. More orally the degeneration becomes weaker, though there is more glia in the anterior part of the nucleus than in the normal picture. The remaining nuclei of the medial geniculate body of either side show no changes.

In dog No. 12 (Fig. 1) the degenerations of the medial geniculate body are more severe on the left side than on the right. On the left side the whole superior nucleus is markedly degenerated, although there are rather many normal nerve-cells within its area. At the same time the inferior nucleus has undergone a partial degeneration. Besides these, the ventral nucleus is partly degenerated in its posterior portion, though here the degeneration is smaller than in the two previous nuclei. There is slightly more glia in its posterior part. On the right side only the dorsal part of the superior nucleus is largely degenerated. The remaining area shows a partial degeneration, and so does the inferior nucleus, in which there is a large amount of glia.

The degenerations in dog No. 13 (Fig. 1) are small and they occur in the superior nucleus on both sides as partial ones. There is a fairly large amount of glia, with numerous normall nerve-cells occurring simultaneously. The inferior nucleus is partly degenerated, somewhat less than the previous nucleus. The remaining nuclei are free from degeneration.

In dog No. 1 (Fig. 3) part of the small cells are very strongly degenerated on both sides. On the left side the commissural nucleus shows no degeneration, while the other nuclei are degenerated completely (the dorsal and lateral nuclei) or partly nevertheless markedly (the superior, inferior, and ventral nuclei). The superior nucleus is completely degenerated in its posterior portion, and very few traces of cells are present in the anterior part. The inferior nucleus is characterized by a strong rarefaction of normal cells, a large amount of glia being found all over its area. The ventral

nucleus has degenerated only in part and a large quantity of glia and a number of normal nerve-cells can be observed in its area. On the right side all the nuclei of the principal part are degenerated, but a small number of cells occur in the inferior nucleus, which proves its partial degeneration. Similarly, some normal nerve-cells are present in the ventral nucleus, particularly in its anterior portion.

Besides the degenerations of the medial geniculate body in the dogs under study changes also occur in other structures.

A degenerated bundle leading from the cortical lesion area to the medial geniculate body is visible in all the brains examined. In the preparations stained by the Klüver method this bundle appears a very pale blue, or is completely decolourized, while in the Nissl preparations an isomorphous gliosis occurs profusely in the place of the bundle and marks out its course. The gliosis runs from the rostral convexity of the medial geniculate body. On the ventral side it passes between the optic tract and the anterior part of the lateral geniculate body, and sinks into the retrosplenial portion of the white matter. It then directs ventrolaterally on the surface of the ventral corner of the lateral ventricle, where it is difficult to trace in the broad inflammatory area underlying the lesion.

The white matter is strongly degenerated in the vicinity of the lesion, and inflammatory foci occur frequently in it, producing a slight rarefaction of the fibres in the semioval centre. The degenerations in the convolutions result from these inflammations and from the lesion. In each dog there are degenerated bundles (U-type bundles) running from the lesion to the neighbouring gyri: to the medial and posterior ectosylvian gyri and occasionally to the anterior ectosylvian, anterior and posterior suprasylvian, and entolateral gyri. Sometimes, if the cortical lesion approaches the anterior or posterior rhinal fissure, the degeneration of the U-type fibres is present, respectively, in the anterior or posterior composite gyrus. Also the medial region of the cingular gyrus is sometimes subject to a degeneration. The presence of glia and rarefaction of fibres are apparent in the cortex of this gyrus.

Rarefaction of fibres is also noted in the tapetum, in the inferior and, occasionally, superior parts of the lateral ventricles. It has been found that after deep lesions the lateral ventricles and sometimes also the third ventricle dilate greatly. The strong dilatation of the ventricles may cause the detachment of the corpus callosum from the fornix and hippocampus. The largest degenerations are present in the brain of dog No. 1, owing to an extensive lesion on one side and a deep one on the other. The degeneration affects the area surrounding the lesion through the internal capsule, from where it extends to various gyri and descends to the cerebral peduncles. In this dog the degeneration is more severe on the right side.

The state of the claustrum is dependent on the depth of the lesion. The shallow lesions are accompanied by the undamaged claustrum, but where the lesion is deep, involving the white matter, it destroys the posterior part of the claustrum. Sometimes, the claustrum is damaged in only one hemisphere. It is an interesting fact that the portion of the claustrum neighbouring on the part that has been removed does not show any signs of degeneration neither degeneration evident on the opposite side.

In some brains there is a partial degeneration in the thalamic nuclei, namely, in the posterior nucleus, the posterior part of the lateral nucleus and the lateral geniculate body. These nuclei show a slight degeneration of the normal nerve-cells and a larger amount of the glial cells than normal. The partial degenerations of the cerebral peduncles occur a little more rarely. They are situated on one side and presumably result from the retrograde degeneration of the semioval centre produced by the inflammatory foci underlying the lesion.

Conclusions

1. The cortical lesions of the anterior and posterior Sylvian gyri bring about the degeneration of the superior and inferior nuclei and sometimes a partial degeneration of the posterior portion of the ventral nucleus. The intensity of the degeneration of these nuclei depends upon the extent of the lesion. The degeneration of the inferior nucleus is generally less severe and, as a rule, a large number of normal cells is preserved in it along with an increase in the amount of glia.

2. A slight degeneration was found in the dog in which the lesion extended beyond the ectosylvian fissure. This observation, however, is inadequate, for there is a slight degeneration in the ventral nucleus on the left side of dog No. 2 without any crossing of the ectosylvian fissure by the lesion. So far, this fact can not be explained. The degenerations observed in other nuclei (lateral and commissural) are probably due to the fine connexions with the superior nucleus.

GROUP II
Discussion on observational material

This group consists of three brains in which the ectosylvian gyrus has been removed. In two dogs the ablations are very similar, for they are bounded by the suprasylvian and ectosylvian fissures. The outer margin of the lesion runs along the line connecting the tips of both fis-

sures. In the vicinity of the fissures the lesions are shallow and do not involve all the layers of the cortex. The deepest part of the lesion is situated in the middle of the medial ectosylvian gyrus and extends somewhat into the anterior and posterior ectosylvian gyri. The third dog brain of this group has the medial ectosylvian gyrus removed.

In dog No. 5 (Fig. 3) the anterior, medial and posterior gyri have been removed bilaterally and uniformly. The lesion lies between the ecto-

Fig. 3. The extent of the cortical lesions in dogs and the corresponding degenerations of the nuclei of the medial geniculate body

Further explanations as in Fig. 1

sylvian and suprasylvian fissures, the cortex at the depth of these fissures being uninjured. The deepest area of the lesion is confined to the middle zone in the posterior portion of the anterior ectosylvian gyrus and the medial ectosylvian gyrus.

The lesions in dog No. 10 (Fig. 3) are similar to those in the previous dog. They are bordered by the ectosylvian and suprasylvian fissures, in the depth of which the cortex is undamaged all along. In the left hemisphere and in the anterior and, partly, medial ectosylvian gyri of the right hemisphere the lesion is considerably deeper and involves a part of the white matter in the middle portion of the gyrus. The lesion shallows in the posterior part of the medial ectosylvian gyrus, but it is shallowest in the posterior ectosylvian gyrus including only the cortex of the con-volution. On the right of the ventral margin of the lesion the operative injury extends down over the posterior composite gyrus. In this region the lesion is fairly deep and involves part of the white matter underlying the gyrus. In the right hemisphere there is also an extensive degenerative plaque, which includes the white matter from the anterior ectosylvian gyrus through the internal capsule to the head of the caudate nucleus.

The lesions in dog No. 4 (Fig. 3) are markedly smaller and involve only the medial ectosylvian gyrus, thus being situated between the largest bend of the ectosylvian fissure and that of the suprasylvian. The depth of the lesion is the same on both sides. The cortex at the depth of both fissures remains intact and the deepest portion of the lesion occupies the middle zone of the destroyed gyrus. Besides, the dog had another, additional lesion made in the prefrontal region on both sides about two months before its being sacrificed. This is smaller in the right hemi-sphere and involves only the proreal gyrus and the cortex of the orbital gyrus adjacent to it. In the left hemisphere the lesion is larger and includes the proreal gyrus, dorsal portion of the orbital gyrus and a small anterior portion of the anterior composite gyrus. Medially the lesion on either side reaches the great fissure.

Discussion on degenerations

The removal of the whole ectosylvian gyrus is followed by the partial degeneration of all the nuclei of the principal part. This degeneration is characterized by a large number of glial cells and a small number of nerve-cells.

The degenerations of the medial geniculate bodies in dogs Nos. 5 and 10 are (Fig. 2, 3), as a rule, analogous and occur in all the nuclei of the principal part. The magnocellular part is unaltered. The difference between the degenerations in these dogs is that the posterodorsal part

of the superior nucleus has not degenerated on either side in dog No. 10. Anteriorly to this region spreads a degeneration which becomes broader and broader.

In dog No. 4 (Fig. 3) the degeneration is considerably narrower which corresponds to the smaller lesion. It involves only the anterior portion of the medial geniculate body on both sides. The ventral and dorsal nuclei are degenerated to a remarkably higher degree than the lateral and commissural nuclei. The degenerations of the former nuclei are severe, there being a large amount of glia and few normal nerve-cells in their area. On the other hand, in the lateral and commissural nuclei there is a large number of normal nerve-cells and a smaller quantity of glia.

Besides the changes in the medial geniculate body described above, degenerations also occur in other structures.

In both hemispheres of the dogs under study degenerated bundles are apparent in the white matter. The degenerated bundle of the temporal radiation runs from the injured region to the medial geniculate body, others pass to the medial suprasylvian gyrus, the ectolateral gyrus and the medial Sylvian gyrus. The dorsal portion of the medial region of the cingular gyrus is degenerated as in the case of the dogs in which the anterior and posterior Sylvian gyri have been removed. A gliosis and a reduction in the number of nerve fibres are observed in it.

A considerable decrease in the number of fibres is apparent in the tapetum in the dorsal part of the lateral ventricles. In addition, partial degenerations of the thalamic nuclei (lateral and posterior nuclei, lateral geniculate body) occur in the dogs discussed. Besides these, in dog. No. 4 there is a fairly strong, bilateral degeneration of the mediodorsal nucleus, which may be due to the frontal lesion.

Conclusions

1. The anterior region of the medial geniculate body degenerates after the removal of the medial ectosylvian gyrus, indicating the connexions of this part of the convolution with the anterior nuclei of the medial geniculate body (dorsal, lateral, ventral and commissural).

2. The partial degeneration of all the nuclei of the principal part suggests that the ectosylvian gyrus is largely connected with the medial geniculate body. However, the projection of the medial geniculate body is wider, since the degeneration following the ablation of this gyrus is not complete. It may be inferred from this fact that the area of the auditory region in the dog is larger than that of the lesion made in this series.

GROUP III
Discussion on observational material

This group consists of three dog brains in which broad temporal lesions, including the anterior and posterior Sylvian gyri as well as the ectosylvian gyrus, were made. The suprasylvian fissure and the anterior and posterior rhinal fissures constitute the outer boundary of all these lesions. However, the area of the composite gyrus lying between the ectosylvian gyrus and the rhinal fissure has not been removed in all of the dogs.

The lesions in both hemispheres of dog No. 14 (Fig. 3) have similar areas and the same depth. They are limited by the suprasylvian fissure. The area of the posterior composite gyrus neighbouring on the posterior ectosylvian gyrus remains undestroyed on both sides, but the part of the anterior composite gyrus contiguous to the anterior ectosylvian is involved by the lesion in the right hemisphere. The destruction of the portion of the anterior and posterior composite gyri adjacent to the anterior and posterior Sylvian gyri extends down to the rhinal fissure. The depth of the lesions is the same in both hemispheres. The whole area of the lesion is completely decorticated except for the bottom of the suprasylvian fissure, where the two-thirds of the cortical layer has been removed. The area of the deepest lesion with the subcortical white matter damaged lies in the middle bend of both convolutions. Moreover, in the anterior portion of the anterior Sylvian gyrus of the right hemisphere the lesion is deeper than elsewhere. The cortex at the depth of the ectosylvian and Sylvian fissures is destroyed almost completely.

In dog No. 15 (Fig. 3) the lesion of either side includes the anterior and posterior Sylvian gyri with the composite gyrus as far as the rhinal fissure. The whole ectosylvian gyrus has been removed. The portion of the anterior composite gyrus borderring on the anterior ectosylvian gyrus in the left hemisphere, and in the right hemisphere the portion of the posterior composite gyrus adjacent to the posterior ectosylvian region are removed. The lesions are deep in both hemispheres and reach to the bottom of the suprasylvian fissure. Thus, the destruction involves the white matter of the ectosylvian and Sylvian gyri. In addition, the lesion includes the cortex at the depth of the ectosylvian and Sylvian fissures.

The lesions of dog No. 16 (Fig. 3) are, as a rule, uniform. They involve the whole area embraced by the suprasylvian fissure and encroach on the anterior and posterior composite gyri in the vicinity of the Sylvian fissure, this extension being somewhat wider in the right hemisphere. The lesion is of the same depth in both hemispheres. The whole area is

decorticated, the destruction being deeper in the middle region of the medial ectosylvian gyrus, where the white matter underlying the cortex is infringed. The whole cortical layer has been removed in the ectosylvian and Sylvian fissures.

Discussion on degenerations

The wide temporal lesion results in the almost complete degeneration of the medial geniculate body. However, in some nuclei (the inferior and ventral) there is only a partial gliosis along with the destruction of a number of nerve-cells. The remaining cells of these nuclei are turgesced, each with a huge nucleus almost filling its whole area.

Throughout the medial geniculate body in the brain of dog No. 14 (Fig. 3) there is an intensive gliosis, some normal nerve-cells and a few turgid nerve-cells with huge nuclei being preserved. The superior and inferior nuclei are characterized by a larger number of glial nuclei than normal and few nerve-cells. The anterior nuclei, i.e. the dorsal, lateral and ventral, are completely degenerated and no traces of nerve-cells are visible in them. A very severe degeneration and turgid nerve-cells occur in the commissural nucleus. The area of the magnocellular part is unaltered.

The medial geniculate body of dog No. 15 (Figs. 3 and 4) shows a severe degeneration and an increased quantity of glia. A small number of normal nerve-cells and in addition to them a large number of glial nuclei are present in the area of the inferior and ventral nuclei.

The degeneration of the medial geniculate body in dog No. 16 (Fig. 3) is exactly like that in dog No. 14. The complete degeneration has affected the whole principal part except the ventral and inferior nuclei, in which there are traces of nerve-cells.

Besides the changes in the medial geniculate body the bundles of degenerated white matter leading to the entolateral, medial and posterior suprasylvian, coronal, posterior composite, and cingular (medioposterior part) gyri occur in the dogs of this group. Besides, a part of the tapetum situated in the dorsal region of the lateral ventricles has undergone a rarefaction of fibres. Partial degeneration is present in some of the thalamic nuclei: the pulvinar and posterior nuclei, the posterior parts of the lateral and ventral nuclei, the lateral geniculate body. A slight dilatation of the lateral ventricles also occurs in all these dogs, which may be connected with the extensive lesion. An inflammatory focus (supravital reaction), developed at the site of the subdural introduction of a foreign body (suture thread), is apparent on the ventral side of the lesion of the left hemisphere in dog No. 15.

Conclusions

1. The degenerations of the medial geniculate body produced by ablations of the temporal cortex in the dogs discussed above indicate that the auditory area is situated in the anterior and posterior Sylvain gyri and in the ectosylvian gyrus. It is only obscure why after such an ablation the complete degeneration does not occur in the inferior and ventral nuclei.

2. The lesion of the anterior and posterior composite gyri, neighbouring on the Sylvian and ectosylvian gyri, does not produce the degeneration of the medial geniculate body, which suggests that these gyri are not included in the auditory region.

DOG NO. 11

Discussion on observational material and degenerations

Now I shall discuss the lesions and degenerations of dog No. 11 (Fig. 3) in which the ablations were made asymmetrically.

On the right side the ablation includes the anterior, medial and posterior ectosylvian gyri. The lesion is bordered by the arms of the ectosylvian and suprasylvian fissures. It encroaches on the Sylvian gyrus from the side of the posterior ectosylvian gyrus and damages it superficially. In the anterior ectosylvian gyrus cortical strips are left undamaged in the vicinity of both fissures, the ecto- and suprasylvian. The lesion is shallower in the anterior ectosylvian gyrus than in the medial, where it involves the cortex at the bottom of the ectosylvian and suprasylvian fissures. In the left hemisphere the anterior and posterior Sylvian gyri are subject to superficial ablation. An undamaged cortical zone lies by the posterior ectosylvian fissure. The cortex bordering on the anterior ectosylvian fissure is destroyed superficially and left intact at the depth of the fissure. The cortex is also undamaged at the depth of the Sylvian fissure, and the destructions of the anterior and posterior composite gyri are superficial and affect the portions contiguous to the lesion.

The degeneration of the medial geniculate body is asymmetrical (Figs. 3 and 4). It is more extensive on the right side involving nearly all the nuclei of the principal part (similarly to the condition in dog No. 5 after the same cortical lesion). However, only a small degeneration occurs in the dorsal portion of the superior nucleus and it is less severe than in the remaining nuclei. The area of the other nuclei of the medial geniculate body is filled mainly with glia, though a few nerve-cells may

be found in it as well. On the left side the medial geniculate body is less degenerated than on the opposite side, but somewhat more than after the lesions confined to the anterior and posterior Sylvian gyri. The similarity of these degenerations consists in the marked destruction of the superior and inferior nuclei. Moreover, the marked destruction includes the commissural nucleus. It may be supposed that this is due to the degeneration of the right medial geniculate body. The degeneration of the

Dog 15
Dog 11   a)   b)   c)   d)
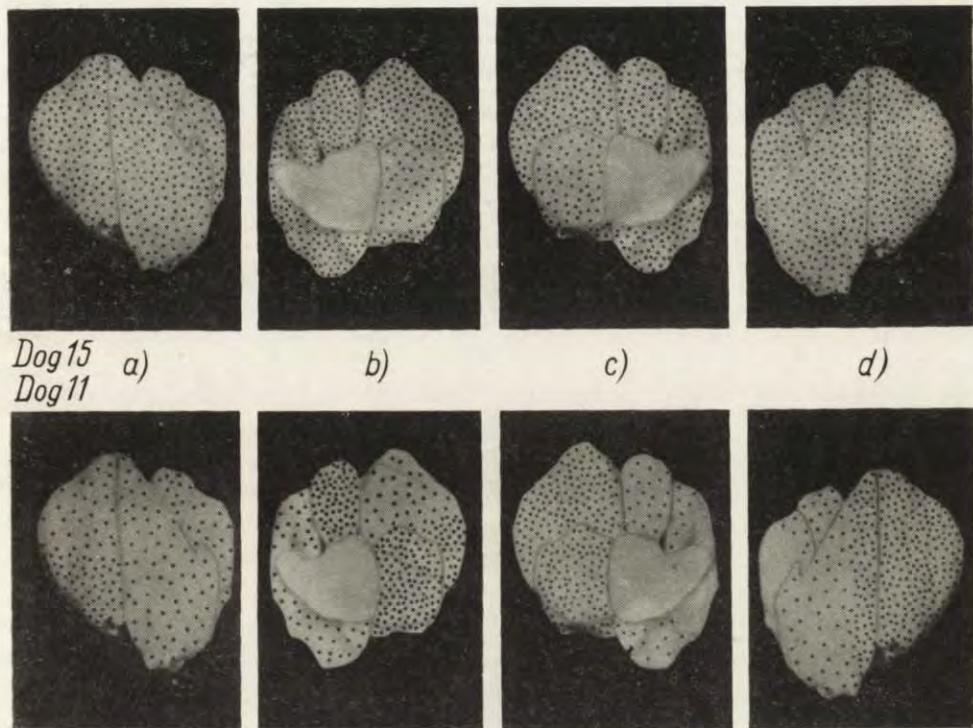
Fig. 4. The extent of the medial geniculate body following a cortical ablation as demonstrated by a wax model
Further explanations as in Fig. 2

commissural nuclei is, however, more severe in the posterior portion and diminishes anteriorly (cf. the description of the degenerations in dogs No. 2 and No. 6). A slight degeneration revealed by a slightly larger amount of glia than normal occurs in the dorsocaudal region of the ventral and dorsal nuclei. The associative connexions between the particular nuclei of the medial geniculate body are probably of some importance to the last degeneration.

The degenerated bundles of the temporal radiation, the area of which has been filled with glia, are also present. Fairly large degenerations

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spread under the lesions in both hemispheres and send out bundles of
degenerated white matter to the entolateral gyrus, the medial and
posterior suprasylvian gyri, as well as to the gyri adjacent to the lesion:
the posterior composite gyrus and the medial and anterior ectosylvian.
In addition, there is a slight degeneration of fibres in the cerebral
peduncles on both sides. The lateral ventricles are considerably dilated.

Conclusions

1. The commissural nucleus of the medial geniculate body connects
both the medial geniculate bodies, which is indicated by the fact that on
the complete degeneration of the nuclei of the principal part on one side
the degeneration is transmitted to the opposite side, where the change
would be poorer owing to the scanty cortical lesion.

2. The differences in the degree and range of the degenerations in the
nuclei of the medial geniculate body on both sides are caused by the
varying extents of the cortical lesions.

COMMENT

In this study degeneration of only the principal part of the medial
geniculate body has been observed after the lesions of the temporal
regions, while the magnocellular part remains unchanged. This fact has
already been stated by R o s e and W o o l s e y (1949). A partial de-
generation throughout the principal part of the medial geniculate body
follows the removal of all auditory areas (A I, A II and Ep) in the brain
of the cat. N e f f (1957) considers on the basis of his studies that it is
necessary to remove the cortex extending between areas A II and Ep as
far as the rhinal fissure to produce complete degeneration of the whole
principal part of the medial geniculate body. From this it results that the
map of the auditory cortex has been widened ventrally. Moreover, the
existence of a third auditory area (A III) in the brain of the dog has been
ascertained by the use of the physiological technic (T u n t u r i 1944,
1950). It is situated on the border of the anterior ectosylvian gyrus and
the anterior composite gyrus, rostrally to the remaining auditory areas
and isolated from them by a cortical area, which does not respond to the
acoustic stimuli.

The wide lesions made in the dogs of the third group of this series
include the temporal cortex, which comprises all the auditory areas so
far known. Degeneration of the medial geniculate body following these
ablations is almost complete with the exception of the inferior and ventral

nuclei, where the traces of normal nerve-cells have been preserved. This fact is as yet difficult to explain. The remaining nuclei of the principal part are completely degenerated.

Partial degeneration occurs in the medial geniculate body after the removal of the whole ectosylvian gyrus (Sychowa 1962). This seems to be comprehensible, as the rest of the auditory cortex has been left uninjured. In the case of dog No. 4, with the medial ectosylvian gyrus removed, the degeneration has been found only in the anterior nuclei of the medial geniculate body (the lateral, dorsal, ventral, and commissural nuclei), which conforms to the observations of N e f f (1957). Besides, my myeloarchitectural studies have revealed that the lateral and dorsal nuclei are connected with the lateral geniculate body by special tracts (S y c h o w a 1962). Using the electrophysiological method (C l a r e and B i s h o p 1954, B o r e n s t e i n, B r u n e r and B u s e r 1958) detected the optic area overlapping the auditory one in the medial ecto-sylvian gyrus. The juxtaposition of these two facts clearly explains the formation of the degeneration of the lateral and dorsal nuclei connected with the lateral geniculate body after the destruction of the medial ecto-sylvian gyrus.

The removal of the anterior and posterior Sylvian gyri produces the degeneration of the posterior nuclei of the medial geniculate body (the superior and inferior nuclei), although the ventral portions of those gyri are not included in any of the auditory areas. This fact has already been described (N e f f 1957, S y c h o w a 1961).

The complete degeneration of the medial geniculate body of dog No. 1 may be connected with the depth of the lesion and its wide extent, for the portion removed on the left side of the brain of this dog is that in which the fourth auditory region was found in the cat (D e s m e d t and M e c h e l s e 1959) and the third auditory region in the dog (T u n t u r i 1950).

The degenerated bundles running to the suprasylvian and entolateral gyri described in this paper indicate the associative connexions between the auditory cortex and the neighbouring convolutions, as has already been found by B o r e n s t e i n, B r u n e r and B u s e r (1958), who used the electrophysiological method. Similar degenerations of the bundles connecting the gyri were observed also by other investigators (N e f f e t a l. 1956).

The degeneration of some thalamic nuclei observed along with the degeneration of the medial geniculate body may be related to the con-nexions between the medial geniculate body and those nuclei. Similar observations have already been given by N e f f.

Attention should be given to the degree of degeneration of the individual nuclei of the medial geniculate body. In many cases the whole nucleus does not degenerate uniformly, and the degeneration in the posterior portion is more severe than that in the anterior portion (e.g. dogs Nos. 8 and 9, superior and inferior nuclei). This fact may be connected with the incomplete destruction of the cortical projection of the nucleus.

SUMMARY

The purpose of this paper is to present the degenerations following removal of the auditory cortex in 16 dogs. Bilateral lesions were made in all the brains. For histological studies the brains were fixed in a solution of formaldehyde, embedded in paraffin and sectioned at 20 μ. Every fifth section was stained by the Nissl and Klüver methods alternately. The extent of ablations was various. The whole of the material has been divided into three groups. The first group consists of the dogs with lesions in the anterior and posterior Sylvian gyri, the second with lesions in the ectosylvian gyrus, and the dogs with lesions in the anterior and posterior Sylvian gyri as well as in the ectosylvian gyrus are reckoned in the third group. In one brain the lesions are asymmetrical, the anterior and posterior Sylvian gyri being removed on one side and the ectosylvian gyrus on the other. Within each of these groups there are some slight differences in the extent and depth of lesions.

The histological analysis of this material shows that the cortical lesions of the anterior and posterior Sylvian gyri are followed by the degeneration of the superior and inferior nuclei and, occasionally, by a partial degeneration of the ventral nucleus of the medial geniculate body. The severity of these nuclear degenerations depends upon the extent of the lesion. The degenerations observed in the other nuclei of the medial geniculate body (the lateral and commissural nuclei) are probably due to the fine connexions with the degenerated nuclei.

The anterior part of the medial geniculate body degenerates after the removal of the medial ectosylvian gyrus which points to the connexions of this part of the gyrus with the anterior nuclei (dorsal, lateral, venteral and commissural). Myeloarchitectural studies show that in the anterior nuclei of the medial geniculate body there are connexions with the lateral geniculate body. On the other hand, physiological examination reveals two areas, an auditory and an optic, coinciding in the medial ectosylvian gyrus. For this reason that degeneration appears quite clear.

From the partial degeneration of all the nuclei of the principal part it follows that the whole ectosylvian gyrus is connected with the medial

geniculate body to a high degree, though the projection to the cortex is wider.

The large lesions of the temporal cortex produce very severe degenerations which indicate that the auditory region is situated in this area. It is only surprising that the ventral and inferior nuclei have not undergone complete degeneration. It should be also emphasized that the complete degeneration of these two nuclei does not occur in any brain of the series under study.

The encroachment of the lesion upon the anterior or posterior composite gyrus has no bearing on the degeneration of the medial geniculate body, which suggests that these gyri are not included in the auditory region. Moreover, it has been ascertained in the case of the dog with asymmetrical cortical lesions that the commissural nucleus of the medial geniculate body is the connective region by which the complete degeneration of one of the medial geniculate bodies is transmitted to the opposite side. Otherwise, the degeneration of the opposite side would be less marked owing to the smaller cortical lesion.

Besides the degeneration of the medial geniculate body there are also degenerative changes in the white matter. Here the degeneration has affected the bundle of the temporal radiation running from the damaged region to the medial geniculate body, as well as the bundles leading from the lesion to the entolateral, suprasylvian, ectosylvian and composite gyri. In addition, there are partial degenerations of such thalamic nuclei as the lateral geniculate body, the posterior nucleus, the posterior part of the lateral nucleus and the posterior part of the ventral thalamic nucleus.

REFERENCES

ADES H. W. 1943 — A secondary acoustic area in the cerebral cortex of the cat. J. Neurophysiol., 6, 59.

ADES H. W. 1949 — Functional relationships between the middle and posterior ectosylvian areas in the cat. Amer. J. Physiol., 159, 561.

ADES H. W., METTLER F. A. and CULLER E. A. 1939 — Effect of lesions in the medial geniculate bodies upon hearing in the cat. Amer. J. Physiol., 125, 15.

BORENSTEIN J., BRUNER J. and BUSER P. 1958 — Étude du système thalamo-cortical d'association visuelle et auditive chez le chat sous chloralose. Contrôre reticulaire des systèmes associatifs. J. Physiol, 50, 2.

BREMER F. 1943 — Etude oscillographique des responses sensorielles de l'aire acoustique corticale chez le chat. Arch. int. Physiol., 53, 53.

BREMER F. and DOWN R. S. 1939 — The acoustic area of the cerebral cortex in the cat. A combined oscillographic and cytoarchitectonic study. J. Neurophysiol., 2, 308.

BREMER F., BONNET V., and TERZUOLO C. 1955 — Étude electrophysiologique de des aires auditives corticales du chat. Arch. int. Physiol., 62, 390.

CHORĄŻYNA H. and STĘPIEŃ L. 1961 — Impairment of auditory recent memory produced by cortical lesions in dogs. Acta Biol. Exper., (Warsaw), 21, 177.

CLARE M. H. and BISHOP G. H. 1954 — Responses from association area secondary activated from optic cortex. J. Neurophysiol., 17, 271.

CLARK W. E. LE GROS 1935—36 — The thalamic connections of the temporal lobe of the brain in the monkey. J. Anat. (Lond.), 70, 447.

DESMEDT J. E. et MECHELSE K. 1959 — Mise end evidence d'une quatrième aire de projection acoustique dans l'écorce cerebrale du chat. J. Physiol., 51, 3.

DIAMOND I. T., CHOW K. L. and NEFF W. D. 1958 — Degeneration of caudal medial geniculate body following cortical lesion ventral to auditory area II in the cat. J. Comp. Neurol., 109, 349.

GOLDBERG J. M., DIAMOND I. T. and NEFF W. D. 1957 — Auditory discrimination after ablation of temporal and insular cortex in cat. Fed. Proc. Am. Soc. Exp. Biol., 16, 1.

HIND J. E. 1953 — An electrophysiological determination of tonotopic organization in auditory cortex of cat. J. Neurophysiol., 16, 475.

HOLLANDER D. and STOFFELS. 1937 — Nouveau procédé de localisation pour les recherches sur le cortex cerébrèl: le palliogramme. J. Belge. Neur. et Psych., 37.

KIANG N. 1955 — Organization of auditory cortex in the cat. Amer. J. Physiol., 183, 3.

MERING T. A. 1953 — On the topography of the auditory pathway in the dog (in Russian). Archiv. Anat. Gistol. i Embriol. 30, 61.

METTLER F. A. 1932 — Connections of the auditory cortex of the cat. J. Comp. Neur., 55, 139.

NEFF W. D. 1957 — Behavioral studies of auditory discrimination. Ann. Ot. Rhyn. Lar., 66, 506.

NEFF W. D. 1958 — Functions of the auditory cortex. Laryngoscope, 68, 413.

NEFF W. D., FISHER J. F., DIAMOND I. T. and YELA M. 1956 — Role of auditory cortex in a discrimination requiring localization of sound in space. Ibid., 19, 500.

OHNISHI G. 1931 — Über die zentrale Hörsbahn; besonders die Beziehung zwischen dem Temporallappen und dem Corpus geniculatum mediale. Okayama--Igakkai-Zasshi., 43, 1959.

POLYAK S. 1927 — An experimental study of the associational, callosal and projectional fibers of the cerebral cortex of the cat. J. Comp. Neurol., 44, 197.

POLYAK S. 1932 — The main afferent fiber systems of the cerebral cortex in Primates. Univ. California Publ. in Anat., 2.

POSTHUMUS MEYJES F. E. 1934 — Recherches expérimentales sur l'anatomie du système acoustique central. Encéphale, 29, 433.

RISS W. 1959 — Discrimination of sound direction. J. Neurophysiol. 22, 373.

ROSE J. E. 1949 — The cellular structure of the auditory region of the cat. J. Comp. Neurol. 91, 409.

ROSE J. E. 1955 — Cortical connections and functional organisation of the thalamic auditory system of the cat. In: Proceedings of the symposium on the biological and biochemical basis of behavior. August 29 — September 5, 1955. University of Wisconsin Press, Madison (in press).

ROSE J. E. and WOOLSEY C. N. 1949 — The relation of thalamic connections, cellular structure and evokable electrical activity in the auditory region of the cat. J. Comp. Neurol., *91*, 441.

STOLL I., AJMONE-MARSAN C. and JASPER H. H. 1951 — Electrophysiological studies of subcortical connections of anterior temporal region in cat. J. Neurophysiol. *14*, 305.

SYCHOWA B. 1961 — Degenerations after ablation of the anterior and posterior parts of the Sylvian gyrus in the dog. Bull. Acad. Polon. Sci., Ser. sci. biol., *9*, 183.

SYCHOWA B. 1962 — Degeneration after ablation of the Ectosylvian gyrus in dog. Bull. Acad. Polon. Sci., Ser. sci. biol., *10*, 17.

SYCHOWA B. 1962 — Medial geniculate body of the dog. J. Comp. Neurol., *118*.

TUNTURI A. R. 1944 — The audio frequency localization in the acoustic cortex of the dog. Amer. J. Physiol., *141*, 397.

TUNTURI A. R. 1946 — A study on the pathway from the medial geniculate body to the acoustic cortex in the dog. Amer. J. Physiol., *147*, 311.

TUNTURI A. R. 1950 — The physiological determination of the boundary of the acoustic area in the cerebral cortex in the dog. Amer. J. Physiol., *160*, 395.

TUNTURI A. R. 1955 — Effect of lesions of the auditory and adjacent cortex on conditioned reflexes. Amer. J. Physiol. *181*, 225.

VOGT O. 1898 — Sur la myélinisation de l'hémisphére cérèbral du chat C. R. Soc. Biol. (Paris), *10*, 54.

WALKER A. E. 1938 — The primate thalamus. The University of Chicago Press Chicago, Illinois.

WALLER W. H. 1940 — Thalamic degeneration induced by temporal lesions in the cat. J. Anat. (Lond.)., *74*, 528.

WOOLLARD, H. H. and HARPMAN A. 1939 — The cortical projection of the medial geniculate body. J. Neurol. Psychiat., *2*, 35.

YOSHIDO, J. 1924 — Über den Ursprung der kortikopetalen Hörbahn beim Kaninchen. Folia Anat. Japon., *2*, 289.

STIMULATION EFFECTS AND BIOELECTRICAL ACTIVITY OF THE CAUDATE NUCLEUS IN UNILATERALLY DECORTICATED RABBITS AND CATS

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M a g e n d i (1841), N o t h a n g e l (1873), and B a g i n s k y and L e h m a n n (1886) observed that destruction of the caudate nucleus gave rise to increased motor activity. Recent studies in monkeys in which the caudate lesions were more precise and the animal behaviour exactly registered by the actographic method also revealed a greater motility following operation (T u r n e r 1957). Other investigators, however, found no apparent behavioural changes following lesions of the caudate (S c h ü l l e r 1902 a) or lenticular nuclei (W i l s o n 1914).

Electric stimulation of the caudate nucleus in conscious animals evokes a number of phenomena. The most typical of them, described by F e r r i e r already in 1876, is contralateral turning of the head and trunk. Other effects are contralateral circling, chewing or licking movements and flexion of extremities (P a c h o n and D e l m a s - M a r s a l e t 1924, F o r m a n and W a r d 1957, R o z h a n s k i i and L a g u t i n a 1957, W h i t e and H i m w i c h 1957). In addition to these effects, which may be considered as excitatory in character, inhibitory phenomena have been reported. The latter appear as a decrease in spontaneous motor activity (A k e r t and A n d e r s s o n 1951), falling asleep (H e a t h and H o d e s 1952) or catatonic syndrome (H e s s jr, K o e l l e and A k e r t 1953). Catatonic syndrome was also obtained after intracaudate introduction of aluminum cream (S p i e g e l and S z e k e l y 1961).

It is likely that some of the motor effects are due to excitation of the internal capsule. This has been suggested in several earlier papers based

on the fact that the caudate nucleus stimulation effects appear to be elicitable only in animals with intact cerebral cortex and undamaged pyramidal pathways (M i n o r 1889, Z i e h e n 1889, 1890, S c h ü l l e r 1902 b). M c R i o c h and B r e n n e r (1938) consider the mammalian corpus striatum to have no autonomous function and to act in conjunction with other forebrain systems. On the other hand, stimulation of some points within the sensorimotor and premotor cortical areas produces phenomena very similar to those obtained from the caudate nucleus. This comprises both contralateral turning of the head (C l a r k and W a r d 1937, P e n f i e l d and R a s m u s s e n 1950, B r u c h e r 1955, W h i t e and H i m w i c h 1957) and chewing or licking movements (M a g o u n, R a n s o n and F i s h e r 1933, M c R i o c h 1934, G e r e b t z o f f 1941).

M e t t l e r e t a l. (1939 a, b, c) studied the effects of caudate nucleus stimulation upon cortically induced movements. Their results showed a noticeable inhibitory influence of the caudate nucleus on the evoked motor activity. M i l l e r (1936) found that strychnine injection into the caudate nucleus in cats produced essentially the same motor effects as electric stimulation. Similar effects followed intracaudate injection of cholinergic drugs (W h i t e and H i m w i c h 1957, S t e v e n s, K i m and M a c L e a n 1961).

Electrophysiological findings are contradictory. T e n C a t e, W a l- t e r and K o o p m a n (1940) found that electroencephalograms of striatal rabbits presented desynchronization patterns. On the other hand, K e n- n a r d (1943) observed, in EEG of decorticated monkeys, "bursts" of 8—12 cy/sec. waves arranged in spindles. M o r i s o n and B a s s e t t (1945) recorded similar spindles from the caudate nucleus of decorticated cats, but failed to obtain them in animals with an intact cerebral cortex.

In a previous paper (S a d o w s k i 1962) it has been shown that the caudate nucleus stimulation produces apparent changes in animal behaviour including the performance of conditioned reflexes. There is some difficulty in interpreting these results, however, because of the possibility of the excitatory process involving the adjacent ascending and descending fibres of the internal capsule. This not only could modify the functioning of the lower centres in the brain stem and spinal cord but produce a state of excitation in various cortical areas.

In this connection we thought it necessary to study how, if at all, the function of the caudate nucleus is dependent on the cerebral cortex, particularly on the sensorimotor area. As appears from the literature data, this problem is very difficult to solve. In our investigations we compared

the effects obtained by stimulation of the caudate nucleus in a decorticated and an intact hemisphere as well as bioelectric potentials recorded from both caudate nuclei in conscious animals.

METHODS

The experiments were made on 15 adult rabbits weighing over 2.5 kg., and on 4 cats weighing about 3 kg.

Hexobarbital was given intravenously or intraperitoneally to produce deep general anaesthesia, and the animals head put in a modified stereotaxic instrument described by R e i n o s o (1945). The head of a rabbit was immobilized in the attachment for a rabbit described by S a w y e r, E v e r e t t and G r e e n (1954). The skull bone was removed so as to expose the entire upper and part of the lateral surface of the left hemisphere. Holes about 1 mm in diameter were drilled at 2 mm intervals in the skull around the opening. The dura was cut and the left hemisphere was decorticated with a sharp bone spoon. An extensive, cortical ablation was made in the frontal and parietal areas with a slight encroachment on the temporal and occipital areas. Subsequently, electrodes were implanted pair-wise into the caudate nuclei, with a 1 to 1.5 mm. gap between the tips. The electrodes were made of platinum wire 0.2 mm in diameter and insulated with a glass capillary, except for 0.5 mm at the tip. One pair was implanted with a stereotaxic instrument into the right caudate nucleus. The implanted electrodes were immobilized with self polymerizing "Duracryl" resin poured over the skull surface. Another pair of electrodes was implanted in the left caudate nucleus (i.e., on the decorticated side), and the entire exposed brain surface was covered with "Duracryl", care being taken to fill all the small holes around the opening made by trephining. The extracranial ends of the electrode wires were connected to the plugs of a miniature radio valve, the wound was dusted with penicillin and streptomycin, and the skin sutured with catgut in such a way that the valve plugs would be at the back.

The coordinates of the caudate nucleus were taken from S a w y e r, E v e r e t t and G r e e n's stereotaxic atlas of the rabbit's diencephalon (1954) and J a s p e r and A j m o n e-M a r s a n's stereotaxic atlas of the diencephalon of the cat.

Antibiotics were administered postoperatively for the first week.

The above operation was performed in all the rabbits and one cat. The remaining three cats who served as controls had the cerebral cortex intact but electrodes were implanted in both caudate nuclei.

Experiments were begun two or three days after operation. The plugs on the animal's head were connected to an identical valve socket, and this by thin flexible leads to a stimulator to stimulate the caudate nuclei with rectangular impulses, or to an Alvar electroencephalograph to record the electrical activity.

The two caudate nuclei were stimulated alternately by bipolar method for 15 to 30 sec. at 1 min. intervals with electric current of 50 cy/sec. frequency and 0.5 msec. width of impulses. The amplitude was at the start 0.5 V and was raised 0.5 V a time until a distinct physiological effect was recorded. Usually, the amplitude did not exceed 10 V. The behaviour of the animals on stimulation of both the right and left caudate nucleus was noted. Attention was also paid to the amplitude of

the impulses in connection with the somatic effects attending stimulation. Several experiments were made on each animal at intervals of a few days for 3 to 8 weeks.

Electroencephalograms were recorded at varying intervals after operation, using bipolar derivations. The animals were in a cage, in a dark sound-proof room, but free to move. Cats were given luminal-sodium intramuscularly, dosage 50 mg./kg.

After termination of the experiments, the animals were sacrified, the heads perfused with 10 per cent formol and the brains removed and embedded in celloidin. Sections 50/μ thick were made in frontal plane, and every sixth stained with iron hematoxyllin after Weigert. All sections were stained in the immediate proximity of the electrodes. Cortical changes and localization of the electrode tips were checked.

Several sections from the anterior, middle and posterior parts of the caudate nuclei were stained with thionin after Nissl to verify neuronal changes resulting from the decortication.

RESULTS

Motor effects of caudate nucleus stimulation were investigated in all the rabbits and cats.

The usual effect was contralateral turning of the head, and, occasionally, of the trunk also which, sometimes, passed into contralateral circling or even running. This was noted in all the animals at an average threshold amplitude of 2 V for the intact hemisphere and 2.9 V for the decorticated one. In 7 rabbits and the hemidecorticated cat this effect was accompanied by an ipsilateral rolling movement of the head about the longitudinal axis, sometimes so pronounced that the head made a turn of 180°. This was evoked by an average threshold amplitude of 2.8 V for the intact hemisphere and 6.5 V for the decorticated hemisphere.

The next most frequent effect was the chewing movement. It was noted in 12 rabbits and in the hemidecorticated and control cats, at an average threshold amplitude of 1.6 V for the intact hemisphere and 4 V for the decorticated one. Less frequent effects were ipsilateral turning of the head, putting forward the ipsilateral forepaw, stamping, smelling and searching. All the effects described were evoked by stimulation of either caudate nucleus and were constant up to 8 weeks after operation (Fig. 1).

Electroencephalograms were recorded on 14 rabbits and the hemidecorticated cat from a few days to several weeks after operation.

In rabbits, the tracings recorded from both caudate nuclei were usually desynchronized, of a frequency 30 to 60 cy/sec., and not exceeding in amplitude 20 μV. Superimposed on this rhythm were „bursts" of waves
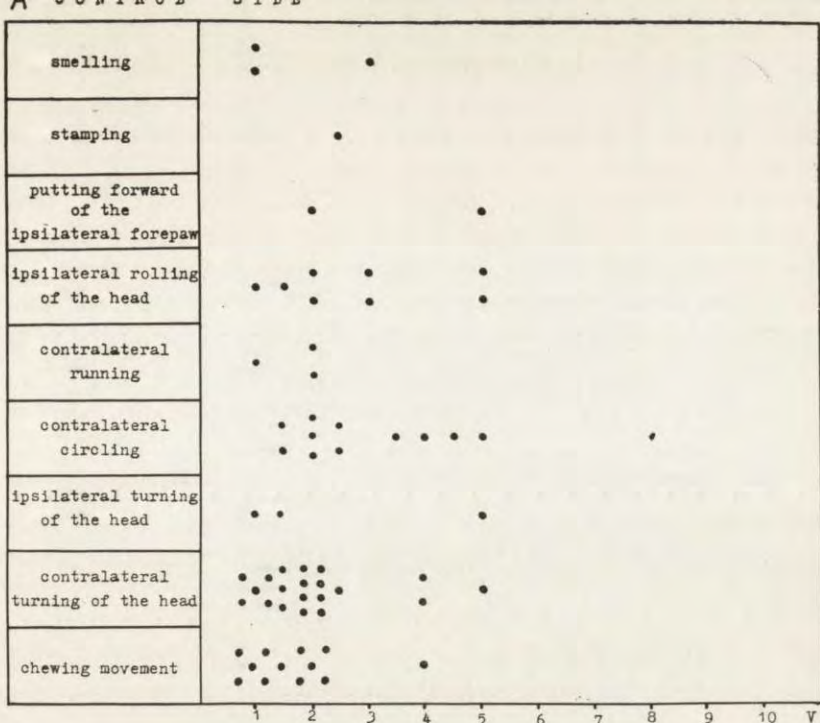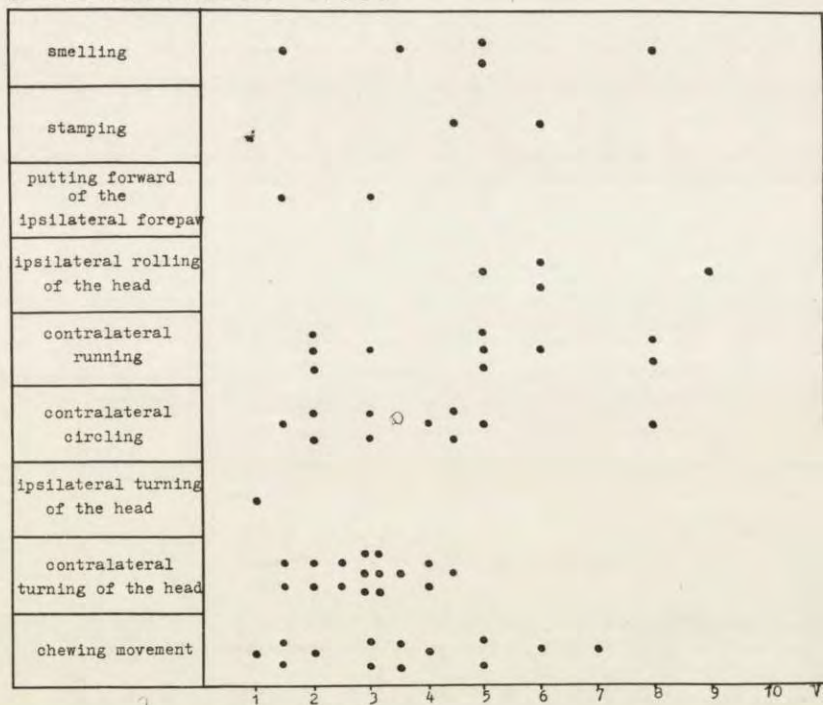
Fig. 1. Motor effects of caudate nucleus stimulation in the rabbits and cat within electrodes in the caudate nucleus. Each point corresponds to an effect observed in one experiment. The voltage scale indicates the lowest current amplitude to evoke the effect

8 to 15 cy/sec. in frequency and up to 150 μV in amplitude, arranged in characteristic spindles. They were recorded both from the intact and the decorticated side (Fig. 2).

In the cat, too, desynchronization was seen in the tracings from either caudate nucleus. Spindles similar to those recorded in rabbits appeared only after the administration of luminal. They were recorded from the caudate nuclei of both the intact and the decorticated hemisphere (Fig. 3).

Histological examination in rabbits demonstrated an extensive lesion of the left cortex which involved the grey matter and, to some degree,

Rabbit No 54

R. caudate nuc.

L. caudate nuc.

1 sec   100 μV
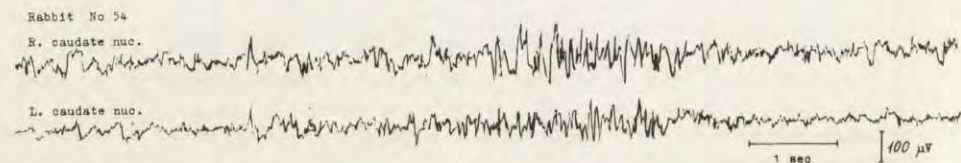
Fig. 2. Electroencephalogram of rabbit No. 54. Upper record, left caudate nucleus (decorticated side); lower record, right caudate nucleus. Bipolar lead. Experiment made 17 days after operation

the white matter. There were no signs of damage to the caudate nucleus. On the control side, both electrodes penetrated the caudate nucleus in 9 rabbits. In three animals one of the electrodes was in the internal capsule, and in two, both were in the capsule in close proximity to the caudate nucleus. On the decorticated side, both electrodes were in the caudate nucleus in all but one animal in which one of the electrodes was in the internal capsule. The region stimulated was either the anterior or the

Cat No 106

R. Caudate Nuc.

L. Caudate Nuc.

1 sec   100 μV

R. Caudate Nuc.

L. Caudate Nuc.

1 sec   25 μV

Fig. 3. Electroencephalogram of cat No. 106. Leads as in Fig. 2. Record made 72 days after operation, before and after luminal sodium 50 mg/kg, given intramuscularly

middle part of the nucleus (Fig. 4). In the hemidecorticated cat there was an extensive lesion of the left cortex and both pairs of electrodes were in the caudate nuclei.

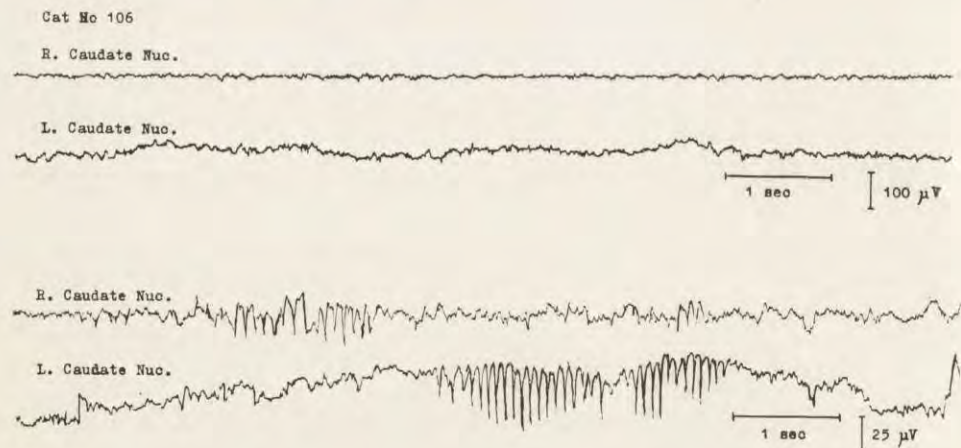In the rabbits and, to some degree in the hemidecorticated cat, the prosencephalic structures were slightly shifted towards the decorticated side which was responsible for the more medial localization of electrodes on this side, compared with the more lateral in the intact hemisphere.
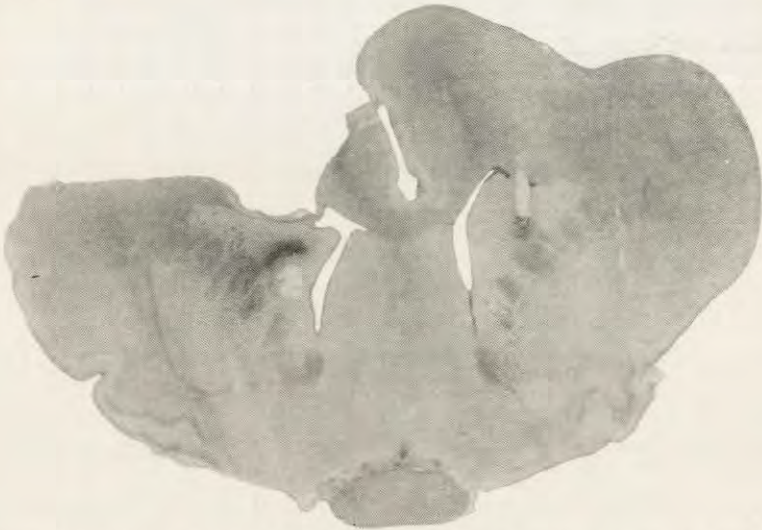
Fig. 4. Frontal section No. 50 of rabbit No. 45. Note electrode tips in both caudate nuclei and lesion of the cortex in the left hemisphere. Weigert's iron hematoxyllin staining

In thionin-stained sections some slight changes in nerve cells of the caudate nucleus were seen on the decorticated side. Their shape was altered, the nuclei deformed and flattened and the protoplasma showed a lesser affinity to the stain. The size and number of the neurones did not diminish, however. It is difficult to say if these changes were due to the decortication or if they depended on circulatory disturbances and/or inflammatory process.

DISCUSSION

Our findings confirm results of other investigators showing that the most typical effects of caudate nucleus stimulation are complex movements of the body. These movements consist in contralateral turning of the head passing, sometimes, to contralateral circling or running. Closely

related is the ipsilateral rolling of the head about the longitudinal axis which occurs in some animals.

It is known, however, that the caudate nucleus is not the only source of the mentioned phenomena. Contralateral turning of the head also appears upon stimulation of such structures as the nucleus amygdalae (S h e a l y and P e e l e 1957), nonspecific thalamic nuclei (H u n t e r and J a s p e r 1949), the zona incerta (H e s s 1954), the lateral hypothalamus (T r a c z y k 1962) as well as of the cerebral cortex.

M u s k e n s (1914, 1922) believes that these effects are produced by vestibular impulses running in the posterior longitudinal fasciculus to a group of nuclei in the region of the commissura posterior and finally reaching the globus pallidus. Owing to a partial crossing of this fasciculus at the commissura posterior level, the structures above this level are responsible for contralateral, and the structures below, for ipsilateral turning.

Recently, H a s s l e r (1956 a, b) developed a theory of two neuronal systems inducing contra- and ipsilateral adversive movements. The system of structures, stimulation of which evokes contralateral movements, involves the anterior nucleus of the thalamus, nucleus entopeduncularis, zona incerta, nucleus subthalamicus Luysi and areas 5, 6, 8 and 24 of the cerebral cortex. The caudate nucleus is included, too, in this system but the mechanism of its action is not clear.

On the basis of anatomical connections, the caudate nucleus can influence in three different ways. First, through the globus pallidus and ansa lenticularis the caudate nucleus is connected with diencephalic structures such as the thalamus, hypothalamus, zona incerta and nucleus subthalamicus Luysi (W i l s o n 1914, H u b e r and C r o s b y 1929, D r o o g l e e v e r, F o r t u y n and S t e f e n s 1951, 1952). Secondly, by the way of the nonspecific thalamic nuclei the impulses from the caudate nucleus reach the cerebral cortex (D u s s e r d e B a r e n n e and M c C u l l o c h 1938, 1939, 1941, P u r p u r a, H o u s e p i a n and G r u n d f e s t 1958). A third pathway involves the strionigral connections described earlier by L a n g l e y and G r ü n b a u m (1890) and now considered by V o n e i d a (1960) to be the only direct descending projection of the caudate nucleus.

The fact that essentially the same results can be obtained by stimulation of the caudate nucleus in the intact and the decorticated hemisphere permits us to assume that participation of the sensorimotor cortex is not necessary in producing contralateral adversive movements. This is consistent with the observations by L a u r s e n (1962) and may be correct only

in rabbits and cats. In higher species, as Essig et all. (1950) suggest for monkeys, the cortical influences are probably more pronounced.

The mechanism of contralateral turning of the head evoked by caudate nucleus stimulation must be related to impulses conveyed through the caudato-nigral fibres down to the brain stem, as well as to those transmitted to diencephalic structures. Probably the reticular formation of the brain stem plays an essential part in conveying the caudate impulses to the motor centres of the spinal cord (Hassler 1956 a, b, White and Himwich 1957).

Turning of the head may result from changes in neck muscle tonus produced by the caudate nucleus stimulation (Traczyk and Sadow-ski 1962). On the other hand, circling or running must have a more complex mechanism, probably of an extrapyramidal character, involving subcortical motor centres of the nonspecific formations of the brain (Ward 1958).

Chewing movements, although observed in cats by Miller (1936) and Rozhanskii and Lagutina (1957) on caudate nucleus stimulation, are rather related to the motor cortex and internal capsule (Magoun, Ranson and Fisher 1933, Gerebtzoff 1941). McRioch (1934), however, proposed also a subcortical mechanism localized in the brain stem. In our experiments, chewing movements were present both when the caudate nucleus was stimulated in the intact and the decorticated hemisphere. It may be assumed that they are at least partly dependent on the caudate nucleus.

Although there were no qualitative differences between the stimulation effects obtained from either caudate nucleus, the threshold amplitude on the decorticated side was usually higher. This fact is difficult to explain on the basis of our results. It may be due to the minor histologic changes found in the caudate neurones of the decorticated hemisphere. On the other hand, because of a shifting of the prosencephalic structures towards the decorticated side the points stimulated within the caudate nuclei were a little different in both hemispheres.

Regarding the results of electroencephalographic investigations, no differences were noted between the tracings recorded from the caudate nucleus in the intact and the decorticated hemisphere. This gives support to the view that synchronous electrical activity of the caudate nucleus in the form of regular spindles depends on neuronal circuits comprising subcortical structures, and the participation of the cortex is not necessary. Such results may contribute to a conclusion that the principal striatofugal connections end at subcortical level.

SUMMARY

1. The caudate nuclei were stimulated electrically in rabbits and one cat in which extensive cortical lesions of the entire sensorimotor and partly of other areas of the frontal, temporal, parietal and occipital lobes in the left hemisphere were made.

2. The stimulation effects consisted in a number of motor phenomena, the most typical being contralateral turning of the head passing to contralateral circling and running, as well as chewing movements. There were no qualitative differences between the intact and the damaged hemisphere.

3. In EEG records, taken from both caudate nuclei, spindles consisting of waves 8-15 cy/sec. in frequency were seen.

4. Considering that the motor effects of caudate nucleus stimulation are roughly alike, irrespective of whether or not a hemisphere is decorticated, the caudate nucleus may be considered to be relatively loosely connected with the cerebral cortex and to have its main system of connections directed towards lower lying structures of the diencephalon and mesencephalon. The similar EEG picture of tracings obtained from the two caudate nuclei gives further support to this point of view.

5. Our results suggest that motor effects from caudate nucleus stimulation in animals with intact cerebral cortex chiefly depend in the caudate nucleus and that excitation of the internal capsule is less important than might have been expected.

REFERENCES

AKERT K. and ANDERSSON B. 1951 — Experimenteller Beitrag zur Physiologie des Nucleus caudatus. Acta Physiol. Scand., 22, 281.

BAGINSKY A. and LEHMANN C. 1886 — Zur Funktion des Corpus striatum (Nucleus caudatus). Experimentelle Studie. Arch. Pat. Anat. Physiol. Klin. Med., 106, 258.

BRUCHER J. M. 1955 — Étude anatomophysiologique de la voie oculomotrice volontaire. Acta Neurol. Psych. Belg., 55, 633.

CLARK S. L. and WARD J. W. 1937 — Electrical stimulation of the cortex cerebri of cats: responses elicitable in chronic experiments through implanted electrodes. Arch. Neurol. Psychiat., 38, 927.

DROOGLEEVER-FORTUYN J. and STEFENS R. 1951 — On the anatomical relations of the intralaminar and midline cells of the thalamus. EEG Clin. Neurophysiol., 3, 393.

DROOGLEEVER-FORTUYN J. and STEFENS R. 1952 — Thalamus, striate body and cortex. EEG Clin. Neurophysiol., 4, 109.

DUSSER DE BARENNE J. G. and McCULLOCH W. S. 1938 — Sensorimotor cortex, nucleus caudatus and thalamus opticus. J. Neurophysiol., 1, 364.

DUSSER De BARENNE J. G. and McCULLOCH W. S. 1939 — Suppression of motor response upon stimulation of area 4 s of the cerebral cortex. Amer. J. Physiol., 126, 482.

DUSSER DE BARENNE J. G. and McCULLOCH W. S. 1941 — Suppression of motor response obtained from area 4 by stimulation of area 4 s. J. Neurophysiol., 4, 311.

ESSIG C. F., HAMPSON J. L., McCAULEY A. and HIMWICH H. E. 1950 — An experimental analysis of biochemically induced circling behavior. J. Neurophysiol., 13, 269.

FERRIER D. 1876 — The functions of the brain. London (cf. WILSON 1914).

FORMAN D. and WARD J. W. 1957 — Responses to electrical stimulation of caudate nucleus in cats in chronic experiments. J. Neurophysiol., 20, 230.

GEREBTZOFF M. A. 1941 — Contribution à la physiologie du corps strié. Arch. Int. Physiol., 51, 333.

HASSLER R. 1956a — Die zentralen Apparate der Wendebewegungen. I. Ipsiversive Wendungen durch Reizung einer direkten vestibulothalamischen Bahn im Hirnstamme der Katze. Arch. Psych. Nervenkr., 194, 456.

HASSLER R. 1956b — Die zentralen Apparate der Wendebewegungen. II. Die neuronalen Apparate der vestibulären Korrekturwendungen und der Adversivbewegungen. Arch. Psych. Nervenkr., 194, 481.

HEATH R. G. and HODES R. 1952 — Induction of sleep by stimulation of the caudate nucleus in Macaque rhesus and man. Trans. Amer. Neurol. Ass., 204.

HESS R. jr, KOELLA W. P. and AKERT K. 1953 — Cortical and subcortical recordings in natural and artificially induced sleep in cats. EEG Clin. Neurophysiol., 5, 75.

HESS W. R. 1954 — Das Zwischenhirn. Basel.

HUBER G. C. and CROSBY E. C. 1929 — Somatic and visceral connections of the diencephalon. Arch. Neurol. Psychiat., 22, 187.

HUNTER J. and JASPER H. H. 1949 — Effects of thalamic stimulation in unanaesthetised animals. EEG Clin. Neurophysiol., 1, 305.

JASPER H. H. and AJMONE-MARSAN C. 1954 — A stereotaxic atlas of the diencephalon of the cat. Ottawa.

KENNARD M. A. 1943 — Electroencephalograms of decorticate monkeys. J. Neurophysiol., 6, 233.

LANGLEY J. N. and GRÜNBAUM A. S. 1890 — On the degeneration resulting from removal of the cerebral cortex and corpora striata in the dog. J. Physiol., 11, 608.

LAURSEN A. M. 1962 — Movements evoked from the region of the caudate nucleus in cats. Acta Physiol. Scand. 54, 175

MAGENDI F. — 1841 (cf. WILSON 1914).

MAGOUN H. W., RANSON S. W. and FISHER C. 1933 — Corticifugal pathways for mastication, lapping and other motor functions in the cat. Arch. Neurol. Psychiat., 30, 292.

METTLER F. A., ADES H, CULLER E. A. and LIPMAN E. 1939 a — Effect of extrapyramidal stimulation upon cortically induced movements. Anat. Rec., 73: suppl. 2, 37.

METTLER F. A., ADES H., LIPMAN E. and CULLER E. A. 1939 b — The extra-pyramidal system. An experimental demonstration of function. Arch. Neurol. Psychiat., *41*, 984.

METTLER F. A., ADES H., LIPMAN E. and CULLER E. A. 1939 c — The physiology of the corpus striatum. Amer. J. Physiol., *126*, P 584.

MILLER F. R. 1936 — The excitability of the caudate nucleus. J. Physiol., *86*, 56P.

MINOR 1889 — (cf. WILSON 1914).

MORISON R. S. and BASSETT D. L. 1945 — Electrical activity of the thalamus and basal ganglia in decorticate cats. J. Neurophysiol., *8*, 309.

MUSKENS L. J. J. 1914 — An anatomico-physiological study of the posterior longitudinal bundle and its relation to forced movements. Brain, *36*, 352.

MUSKENS L. J. J. 1922 — The central connections of the vestibular nuclei with the corpus striatum and their significance for ocular movements and for locomotion. Brain, *45*, 452.

NOTHNAGEL H. 1873 — (cf. WILSON 1914).

PACHON V. and DELMAS-MARSALET M. 1924 — Effets produits par l'excitation électrique des noyaux caudés chez le chien eveillé. C. R. Soc. Biol. Paris, *91*, 558.

PENFIELD W. and RASMUSSEN T. 1950 — The cerebral cortex of man. New York.

PURPURA D. P., HOUSEPIAN E. M. and GRUNDFEST H. 1958 — Analysis of caudate — cortical connections in neuraxially intact and telencéphale isolé cats. Arch. Ital. Biol., *96*, 145.

REINOSO F. 1954 — Anatomisch-Cartesianischer Atlas zur Anwendung bei experimentellen Arbeiten am Katzengehirn durch Lokalisierungsmethoden von Horsley, Clarke und Hess. Anat. Anz., Ergänzungsheft, *100*, 151.

RIOCH McK. D. and BRENNER C. 1938 — Experiments on the corpus striatum and rhinencephalon. J. Comp. Neurol., *68*, 491.

RIOCH McK. J. 1934 — The neural mechanism of mastication. Amer. J. Physiol., *108*, 168.

ROZHANSKII N. A. and LAGUTINA N. I. 1957 — On the problem of significance of the striopallidal system nuclei. J. Physiol. USSR, *43*, 622. (in Russian).

SADOWSKI B. 1962 — Investigations on the function of the caudate nucleus (in Polish). Rozpr. Wydz. Nauk Med., 7, part 1, 63.

SAWYER C. H., EVERETT J. W. and GREEN J. D. 1954 — The rabbit diencephalon in stereotaxic coordinates. J. Comp. Neurol., *101*, 801.

SCHÜLLER A. 1902 a — Experimente am Nucleus caudatus des Hundes. Jahrbücher für Psychiatrie u. Neurologie, *22*, 90.

SCHÜLLER A. 1902 b — Reizversuche am Nucleus caudatus des Hundes. Pflüg. Arch. Ges. Physiol., *91*, 477.

SHEALY C. N. and PEELE T. L. 1957 — Studies on amygdaloid nuclei of cat. J. Neurophysiol, *20*, 125.

SPIEGEL E. A. and SZEKELY E. G. 1961 — Prolonged stimulation of the head of the caudate nucleus. Arch. Neurol., *4*, 55.

STEVENS J. R., KIM C. and MacLEAN P. D. 1961 — Stimulation of caudate nucleus. Behavioral effects of chemical and electrical excitation. Arch. Neurol., *4*, 47.

TEN CATE J., WALTER W. G. and KOOPMAN L. J. 1940 — Electro-encephalography in rabbits after removal of the neopallium. Arch. Néerland. Physiol., *24*, 578.

TRACZYK W. 1962 — Feeding and flight reactions evoked in rabbits by hypothalamic stimulation. Acta Physiol. Polon., *13*, 239. (in Polish).

TRACZYK W. and SADOWSKI B. 1962 — Alterations of the muscle tonus due to stimulation of the caudate nucleus in rabbits. Acta Physiol. Pol. *13*, 609.

TURNER E. 1957 — The significance of hyperkinesis in lesions of the anterior striatum in rhesus macaccus. J. Neurol. Neurosurg. Phychiat., *20*, 216.

VONEIDA T. J. 1960 — An experimental study of the course and destination of fibers arising in the head of the caudate nucleus in the cat and monkey. J. Comp. Neurol., *115*, 75.

WARD A. A. 1958 — Efferent functions of the reticular formation. In: Henry Ford Hospital International Symposium Reticular Formation of the Brain. Boston — Toronto, 263.

WHITE R. P. and HIMWICH H. E. 1957 — Circus movements and excitation of striatal and mesodiencephalic centers in rabbits. J. Neurophysiol., *20*, 81.

WILSON S. A. K. 1914 — An experimental research into the anatomy and physiology of the corpus striatum. Brain, *36*, 427.

ZIEHEN T. 1889 — Zur Physiologie der infracorticalen Ganglien und über ihre. Beziehungen zum epileptischen Anfall. Arch. Psychiat. Nervenkr., *20*, 584.

ZIEHEN T. 1890 — Zur Physiologie der infracorticalen Ganglien und über ihre Beziehungen zum epileptischen Anfall. Arch. Psychiat. Nervenkr., *21*, 863.

FOOD INTAKE AND TYPE II CONDITIONING IN LATERAL HYPOTHALAMIC RABBITS SURVIVED UNDER FORCED HYDRATION

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Stimulation and ablation studies have shown that the lateral hypothalamus controls the food intake in animals (Hetherington 1941, Hetherington and Ranson 1942, Anand and Brobeck 1951 a, b, Delgado and Anand 1953, Anand, Dua and Shoenberg 1955). It has also been found that this hypothalamic structure appears to be implicated in both feeding and motivational behavior patterns (Miller 1955, 1960, Morrison, Barnett and Mayer 1958, Morgane 1961 a,b). Thus rats upon electrical stimulation of the lateral hypothalamus, whose cells are perplexed with the fibres of the medial forebrain bundle (Nauta 1960, 1961, Morgane 1961 a,b), display an increased drive for food as well as an elevation of the effort to satisfy the drive (Miller 1955, 1960, Morgane 1961 a, b).

More recently, Williams and Teitelbaum (1959) found that in spite of the fact that rats with lesions of the lateral hypothalamus refuse the ordinary food and water, and starve to death if these are the only substances offerred, they may at the same time overeat a liquid diet containing milk, eggs, sugar and vitamins, and eventually become obese like the dynamic hyperphagics after ventromedial lesions. Feldman, Larsson, Dimick and Lepkovsky (1957) as well as Morrison, Barnett and Mayer (1958) have also demonstrated that the eventual recovery of eating and drinking behavior in animals with lateral hypothalamic lesions may be possible by the application of the forced drinking technique.

Recently, Wyrwicka (1957) showed that lateral hypothalamic rabbits, who had survived due to forced feeding with carrot, were temporarily

affected on their type II conditioned reflex performance. The purpose of the present experiment was to investigate in detail the behavior of lateral hypothalamic rabbits, who survived due to subcutaneous hydration during the aphagic period, in a situation in which food was presented in response to a correct instrumental (type II) reflex activity acquired prior to operation.

MATERIAL AND METHODS

Experiments were carried out on 14 naive, male and female, rabbits, about one year old at the beginning of the investigation. They were numbered from 19 up to 32. Conditioned reflex type II activity (the instrumental type conditioning) was trained in a 100 × 50 × 50 cm. cage within which two food trays were mounted on opposite walls. A small board was attached to the food tray situated on the left side of the cage, while above the right food tray a bakelite ring was fixed. Both the board and the ring were connected with a recording apparatus.

E x p e r i m e n t a l   g r o u p. In 9 rabbits (Nos. 19, 20, 21, 22, 23, 24, 25, 26 and 27) two kinds of conditioned reflexes type II were trained. One of them consisted of the animal placing its fore limb on the board attached to the left food tray. This response was established by passive placing the animal's limb on the board in accordance with our standard procedure (B a l i ń s k a 1963) used primarily by K o n o r s k i and M i l l e r in the dog (1933). Each active response was followed by food reinforcement.

The second conditioned reflex was established with the M a l i n o w s k i technique (1952). Briefly, the animals were trained in pulling the bakelite ring with their teeth. In the initial training, small pieces of carrot were fasten to the ring or carrot's juice was put on the ring in order to focus the animal's attention on it. Within a few days the animals learned to grasp and pull the ring voluntarily, and each of these responses was followed by food presentation. The animals were divided into 2 groups: (A) in Nos. 19, 20 and 21, the pulling-the-ring response was followed by the presentation of carrot, whereas the fore limb response was reinforced by oats; in Nos. 22 and 23, the pulling-the-ring habit was followed by cats, and the fore limb response was combined with the carrot reinforcement; (B) in Nos. 24, 25 and 26, the pulling-the-ring response was, too, paired with carrot, but the fore limb performance was reinforced by the presentation of cooked, purée-type potatoes. In rabbit No. 27, potatoes were presented after the pulling-the-ring response, while carrot followed the fore limb response (Table I).

Table I

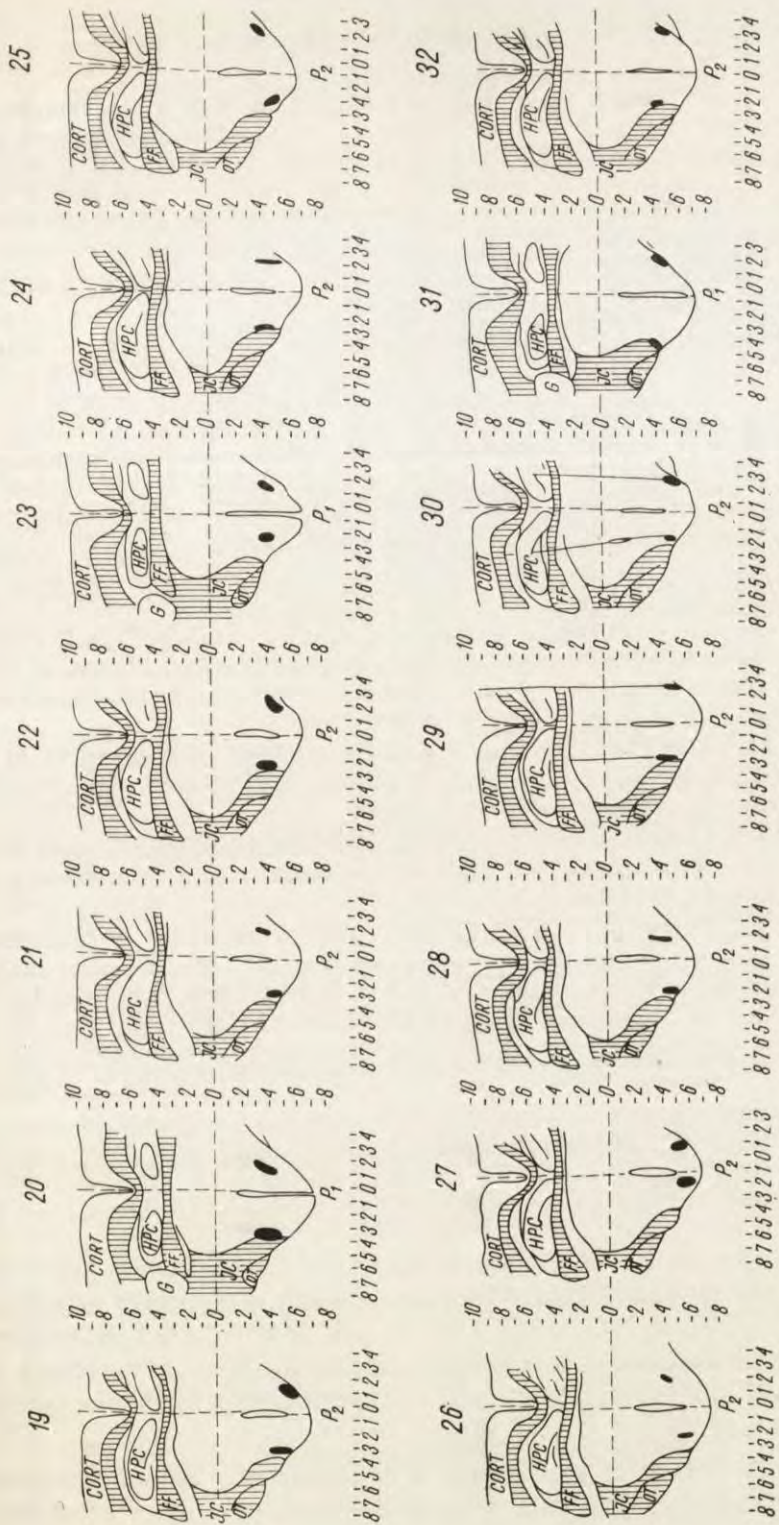| Food	Subgroup A		Subgroup B	
	Pulling-the-ring response	Fore-limb response	Pulling-the-ring response	Fore-limb response
Carrot	Nos. 19,20,21	Nos. 22,23	Nos. 24,25,26	No. 27
Oats	Nos. 22,23	Nos. 19,20,21		
Potatoes			No. 27	Nos. 24,25,26

Fig. 1. Representative reconstructions of lateral hypothalamic lesions described in the text

Cort — celebral cortex; FF — fimbria of fornix; G — mammillotegmental tract; HPC — hippocampus; IC — internal capsule; OT — optic tract

During testing sessions the animals ate ad libitum, that is, as long as they performed the acquired responses. Out of the testing cage the animals were maintained on a standard diet which consisted of potatoes in rabbits which were fed with carrot and oats in the experimental situation, or, it contained oats if carrot and potatoes were offerred under testing conditions. In addition, all rabbits received hay in their living cage.

When conditioned reflexes were well established, all animals were subjected to bilateral lesions of the lateral hypothalamus.

O p e r a t i o n. Lesions were placed stereotaxically in the Horsley-Clarke instrument with the head holder for rabbits described by S a w y e r, E v e r e t t and G r e e n (1954). Animals were anaesthetized with Nembutal (37 to 42 mg. per 1 kgm. of body weight) injected intravenously, and all operating manipulations were made under aseptic conditions. In each instance, lesions were placed bilaterally at points 1 mm. posterior to the bregma, and 2 mm. lateral to the midline. The tips of the electrodes were inserted 14.5 mm. below the dura and lesions were produced by passing a direct current of 3 mA for 15 secs.

F o r c e d   s u b c u t a n e o u s   h y d r a t i o n. In an attempt to keep the lateral hypothalamic rabbits alive forced hydration was administered by subcutaneous injection of 100 ccm. of 40 per cent glucose with isotonic salt solution. In isolated instances, if the aphagic animals lost weight rapidly, 10 ccm. of 20 per cent glucose was injected intravenously in addition. Forced inhydration was discontinued when voluntary acceptance of food recovered.

C o n t r o l   a n i m a l s. In a control group of 5 rabbits (Nos. 28, 29, 30, 31 and 32) the lateral hypothalamus was also destroyed but the animals were not injected with glucose and Ringer solution postoperatively.

W e i g h t   m e a s u r e m e n t s of all animals were taken once a week before operation. In the initial postoperative period, the weight was measured every day, and, later on, every third day.

H i s t o l o g y. Three to four months after operation the animals were sacrificed, their brains removed and fixed in formalin, and prepared for histological analysis. Serial frontal sections were thereafter cut and stained with hematoxiline and cosine or thionine in order to verify the location of the lesions (Fig. 1).

RESULTS

Effects of forced hydration on food intake in rabbits with hypothalamic lesions

All lateral hypothalamic animals used in this experiment refused to eat and drink postoperatively. They also showed a decreased mobility as well as a diminished responding towards any external cues. Reactions to nociceptive stimuli were reduced or absent. In their living cages the lateral hypothalamic animals crouched or sat immobile for hours, ignoring the ordinary food.

After a few days of subcutaneous hydration the animals markedly improved. They began to walk around the cage, paid more attention to

some of the external cues and they also often gnashed their teeth. The latter response was noted in all lateral hypothalamic rabbits and it persisted for about three weeks postoperatively. In the first days after operation, some of the aphagic animals approached food, sniffed it, and eventually, attempted to take it. Spontaneous eating recovered 3 to 7 days following operation. Each animal began to eat hay at first. This was followed by voluntary acceptance of potatoes, small pieces of carrot, and, eventually, grains of oats. However, in spite of recovery of the eating behavior, the lateral hypothalamic animals initially ate less comparing to the preoperative level and showed great reluctance to take oats. Most of the animals ate 10 gms. potatoes and 10 to 20 gms. carrot per day. Only some of the operated rabbits took food of the same amount and quality as prior to the hypothalamic lesions. The recovered rabbits Nos. 19, 20, and 21 increased their food intake, and became hyperphagic towards the end of the observation period (Table II).

In the immediate postoperative period, that is, when complete aphagia occurred, the lateral hypothalamic animals lost their weight very rapidly

Table II

Average food intake (in grms) before and after lateral hypothalamic lesions in the experimental situation in which two kinds of food were offered at the same time

Rabbit No.	Carrot				Oats				Potatoes			
	Days pre-op.				Days pre-op.				Days pre-op.			
	1—5	5—15	15—25	25—35	1—5	5—15	15—25	25—35	1—5	5—15	15—25	25—35
19	160	170	150	180	8	10	14	17	—	—	—	—
20	170	160	180	200	9	20	21	27	—	—	—	—
21	130	110	60	90	10	13	15	18	—	—	—	—
23	20	80	80	120	28	23	20	20	—	—	—	—
24	50	90	40	50	—	—	—	—	70	60	80	50
25	170	160	90	120	—	—	—	—	70	80	70	50
26	200	170	190	110	—	—	—	—	80	60	70	30
27	230	190	200	80	—	—	—	—	120	80	60	30
	Days post-op.				Days post-op.				Days post-op.			
19	150	220	230	260	0	0	8	31	—	—	—	—
20	40	230	220	170	0	0	16	34	—	—	—	—
21	40	180	180	290	0	26	30	61	—	—	—	—
23	0	60	80	130	0	2	22	46	—	—	—	—
24	10	90	120	190	—	—	—	—	0	7	30	67
25	0	40	90	100	—	—	—	—	30	90	80	70
26	50	150	130	140	—	—	—	—	0	25	30	60
27	40	140	140	180	—	—	—	—	0	27	40	40

(Table III). Due to the subcutaneous inhydration, which resulted in voluntary food intake, the animals began to gain weight, although they periodically lost some. It is to be stressed that even in the late postoperative period, when hyperphagia used to occur, the weight did not exceed the preoperative level.

Table III

Alterations of body weight before and after lateral hypothalamic lesions

Rabbit No.	Pre-op.				day of operation	aphagic period	Post-op.				
	weeks						days after recovery of spontaneous eating				
	I	II	III	IV			1−5	5−15	15−25	25−35	60
19	+2	+3	+7	+5	0	−300	−290	−150	−70	−50	+6
20	+1	+3	+4	+6	0	−270	−261	−203	−80	−120	+7
21	+3	+1	+2	6	0	−380	−369	−356	−303	−230	+8
23	+6	+1	0	0	0	−650	−647	−600	−309	−226	−3
24	+9	+3	+2	+7	0	−500	−493	−490	−400	−300	+4
25	+4	+5	+7	+3	0	−320	−300	−250	−300	−150	−2
26	+5	+10	+9	+8	0	−260	−249	−203	−100	−50	−5
27	0	0	0	+1	0	−230	−225	−200	−150	−48	−5
28	+4	+6	+3	+7	0	−1300	died 15 days after operation				
29	+6	+3	+2	0	0	−1260	died 16 days after operation				
30	+3	0	0	0	0	−1000	died 17 days after operation				
31	+5	+1	+4	0	0	−800	−795	−790	−778		
32	+5	−2	+6	+1	0	−960	−953	−955	−941		

Numbers indicate variations from the body weight in grms, measured on the day of operation and reduced to zero (0).

Three animals of the control group (Nos. 28, 29 and 30) which had not been subjected to forced subcutaneous hydration, starved to death within 15 to 17 days following hypothalamic lesion. Two remaining controls recovered their voluntary eating behavior in 14 to 16 days after operation, although the recovery was never complete and the animals showed a striking preference for carrot.

Effect of lateral hypothalamic lesions on conditioned reflex type II activity

Testing the animals was resumed after the recovery of voluntary eating behavior in the living cage. Initially, under experimental circumstances the animals showed abnormalities similar to those noticed out of the testing situation: they were apathetic and immobile. However, due to the procedure of glucose and isotonic salt solution injection, they changed considerably. Despite the absence of conditioned reflex type II responding they began to walk and sniff as well as to show a variety of basic food responses, such as approaching the food trays, inserting mouth in the

cup and attempting to turn it over in order to get food from the next cup. All these reactions in the alimentary situation were obstinately perseverated despite the remarkable tameness and placidity of the lateral hypothalamic rabbits.

The complete loss of the preoperatively acquired instrumental reflex activity with marked expressions of primary food activities were seen during the first two postoperative weeks. Subsequently, the animals began to work for food, although the conditioned reactions often were awkward. In most instances, the animals recovered at first only one conditioned reflex, and it was that previously paired with the presentation of carrot, no matter whether it was the response of the right fore limb (rabbits Nos. 23 and 27) or that consisting in pulling the ring (rabbits Nos. 19, 20, 21, 24 and 26). Either of these two responses recovered gradually. In the initial period of recovery of the pulling-the-ring performance the animal approached the ring, rubbed itself against it or tried to grasp it with its teeth with no signs of pulling it. Very soon, however, at times even within one testing session, the reflex was restored entirely. In the case of the fore limb response a mincing step habit had developed at first which, thereafter, was followed by climbing-the-food-tray performance. A few days later, the conditioned response had acquired a further refinement, and eventually, after a number of reinforcements its recovery became complete. However, in the later postoperative period there were isolated experimental sessions in which the instrumental response did not occur.

A few days after the recovery of the conditioned reflex activity combined with the presentation of carrot the lateral hypothalamic animals began to exhibit also some activity towards the other types of food. After a number of responses at the food tray where carrot was offered, the animal passed over to the food tray situated on the opposite side of the cage, and showed a full picture of basic food behavior patterns there. This persisted for a few days, and, eventually, the animal recovered also its second conditioned reflex activity (Fig. 2).
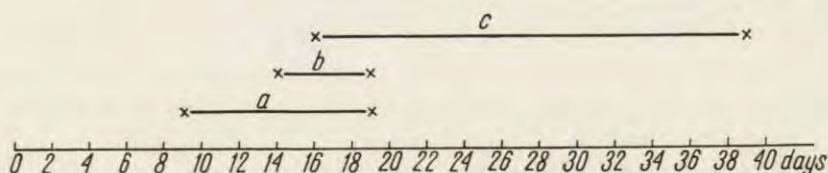

Fig. 2. Recovery of food conditioned reflex type II activity in individual rabbits after lateral hypothalamic lesion
Horizontal lines indicate time extent of recovery of conditioning:
a — basic food-oriented reactions in the experimental situation, b — recovery of the first CR, c — recovery of the second CR

DISCUSSION

It was found that due to the procedure of forced hydration by subcutaneous injection of glucose and isotonic salt solution the lateral hypothalamic rabbits showed only a temporary aphagia which was followed by a decreased food intake. With time the animals began to accept more food, and eventually they recovered their preoperative level of eating, and some of them became even hyperphagic. The evidence that rabbits after lateral hypothalamic lesions survived due to subcutaneous hydration confirms the earlier observation of F e l d m a n, L a r s-s o n, D i m i c k and L e p k o v s k y (1957), and M o r r i s o n and M a y e r (1957) indicating a recovery of aphagic animals under conditions of forced drinking technique. Three out of five non-inhydrated rabbits in the present experiment died following the removal of the lateral hypothalamus, whereas the two remaining animals showed a severe and relatively long aphagia. It is to be emphasized that during the recovery period hay was the first meal which was voluntarily taken in by the surviving lateral hypothalamic rabbits. In the subsequent period, the operated animals successively began to eat potatoes, carrot and oats, although some of them showed great reluctance to eat oats.

The findings also indicate that the conditioned reflex type II activity was lost immediately after operation. Despite a progressively increasing food-directed activity which consisted of a variety of primordial eating behavior patterns the lateral hypothalamic animals were unable to perform the preoperatively acquired instrumental food response. The temporary failure of instrumental performance in lateral hypothalamic rabbits might be related to a partial damage to the motivational system localized within the portion of the medial forebrain bundle passing through the lateral nucleus. However, within several testing sessions the animals, like some of the lateral rabbits in W y r w i c k a's (1957) experiments recovered one of the two conditioned reflexes trained prior to operation, and it usually was that associated with the presentation of carrot. Only then was the conditioned reflex recovered which was followed by potatoes or oats. This may suggest that rabbits with lateral hypothalamic lesion show a preference for the kind of food containing more water. W i l l i a m s and T e i t e l b a u m (1959), and T e i t e l-b a u m and S t e l l a r (1959) demonstrated that animals with lesions of the lateral hypothalamus refused to drink water but they showed a marked preference for milk, or other kinds of liquid foods containing milk, sugar and eggs. It is likely that acceptance of liquid diet maintains the water balance in aphagic and adipsic animals and prevents them from dehydration.

SUMMARY

1. Experiments were carried out on 9 rabbits in which two kinds of conditioned reflexes type II were trained preoperatively. One of these instrumental activities was paired with the presentation of carrot, whereas another one was paired with potatoes or oats.

2. Following lesions of the lateral hypothalamus the animals were induced to eat and drink by subcutaneous injection of 100 ccm. of 40 per cent glucose with isotonic salt solution.

Control animals, who had not been hydrated, died or developed aphagia over a relatively long period of time.

3. Despite adequate motor activity the conditioned reflex type II performance was temporarily los after removal of the lateral hypothalamus.

4. The conditioned reflex which recovered first after lesion of the lateral hypothalamus was that associated with carrot presentation.

5. The findings suggest that rabbits with lateral hypothalamic lesions show a preference for food containing more water.

REFERENCES

ANAND B. K. and BROBECK J. R. 1951 a — Hypothalamic control of food intake in rats and cats. Yale J. Biol. Med., *24*, 123.

ANAND B. K. and BROBECK J. R. 1951 b — Localization of "feeding centre" in the hypothalamus of the rat. Proc. Soc. exp. Biol. (N. Y.)., 77, 323.

ANAND B. K., DUA S. and SHOENBERG K. 1955 — Hypothalamic control of food intake in cats and monkeys. J. Physiol., *127*, 143.

BALIŃSKA H. 1963 — Food preference and conditioned reflex type II activity in dynamic hyperphagic rabbits. Acta Biol. Exper. (Warsaw), *23*, 1.

DELGADO J. M. R. and ANAND B. K. 1953 — Increase of food intake induced by electrical stimulation of lateral hypothalamus. Amer. J. Physiol., *127*, 162.

FELDMAN S. E., LARSSON S., DIMICK M. K. and LEPKOVSKY S. 1957 — Aphagia in chicken. Amer. J. Physiol., *191*, 259.

HETHERINGTON A. W. 1941 — The relation of various hypothalamic lesions to adiposity and other phenomena in the rat. Amer. J. Physiol., *133*, 326.

HETHERINGTON A. W. and RANSON S. W. 1942 — The spontaneous activity and food intake of rats with hypothalamic lesions. Amer. J. Physiol. *136*, 609.

KONORSKI J. and MILLER S. 1933 Foundations of the physiological theory of acquired movements (Polish text with a French summary). Książnica Atlas, Warszawa.

MALINOWSKI O. W. 1952 — Method of motor food conditioned reflexes in rabbits (in Russian). Fiziol. Zhurn. SSSR., *38*, 637.

MILLER N. E. 1955 — Shortcomings of food consumption as a measure of hunger: Results from other behavioral technique. Ann. N. Y. Academy of Science, *63*, 141.

MILLER N. E. 1960 — Some motivational effects of brain stimulations and drugs. Presented at Symposium on Animal Bahaviour and Neurophysiological Correlates. Federation Meeting, Chicago 1960.

MORGANE P. J. 1961 a — Medial forebrain bundle and hypothalamic feeding centers. Fed. Proc., *19*, 292.

MORGANE P. J. 1961 b — Medial forebrain bundle and feeding center of the hypothalamus. J. Com. Neurol., *117*, 1.

MORRISON S. D., BARNETT R. J. and MEYER J. 1958 — Localisation of lesions in the lateral hypothalamus of rats with induced adipsia adn aphagia. Amer. J. Physiol., *193*, 230.

MORRISON S. D. and MAYER R. J. 1957 — Adipsia and aphagia in rats after lateral subthalamic lesions. Amer. J. Physiol., *191*, 248.

NAUTA W. J. H. 1960 — Anatomical relationships between the amygdaloid complex, the dorsomedial thalamic nucleus, and the orbitofrontal cortex in the monkey. Annual Meeting and VII-th International Congress of Anatomists. Amer. Record., *136*, 251.

NAUTA W. J. H. 1961 — Fibre degeneration following lesions of the amygdaloid complex in the monkey. J. Anat., *95*, 515.

SAWYER C. G., EVERETT J. W. and GREEN J. D. 1954 — The rabbit diencephalon in stereotaxis coordinates. J. Comp. Neurol., *101*, 801.

WILLIAMS D. R. and TEITELBAUM P. 1959 — Some observations on the starvation resulting from lateral hypothalamic lesions. J. comp. physiol. Psychol., *59*, 458.

WYRWICKA W. 1957 — Preliminary results on the effects of lesions of the lateral hypothalamus on food conditioned reflex type II (in Polish). Acta Physiol. Polon., *8*, 575.

CONDITIONED REFLEXES ESTABLISHED IN UNILATERALLY DECORTICATED RABBITS TO CAUDATE NUCLEUS STIMULATION

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Unilateral lesions within the caudate nucleus produce some minor changes in the conditioned reflex activity, such as an increase in the number of trials needed to establish a conditioned reflex (K l o s o v s k i i and V o l z h i n a 1956 a,b) or a decrease in its regularity (S a d o w s k i 1959). Bilateral injury of the caudate nucleus, on the other hand, leads to noticeable impairment on conditioning performance. This has been established in dogs by K l o s o v s k i i and V o l z h i n a (1956 a, b), R o m a n o v s k a y a (1957, 1958) and S o ł t y s i k (cf. K o n o r s k i 1961), in rabbits by R o m a n o v s k a y a (1957, 1958), in cats by T h o m p s o n (1959) and in monkeys by R o s v o l d, M i s h k i n and S z w a r c b a r t (1958).

Similarly, electrical or chemical stimulation of the caudate nucleus affects the conditioning process. Inhibition of different motor conditioned reflexes was observed by the majority of authors. This was noted by C h e r k e s (1955, 1957, 1958) studying the influence of electric stimulation of the caudate nucleus on the defence reflexes in dogs. R o s v o l d and D e l g a d o (1956) observed a deterioration of the alternation test in monkeys. B u c h w a l d et a l. (1960, 1961 a) found impairment of visual discrimination in cats, while S t e v e n s, K i m and M a c L e a n (1961) obtained inhibition of the avoidance response both by electric stimulation and by introducing cholinergic compounds into the caudate nucleus. In addition to the inhibitory effect, the excitatory influence of caudate nucleus stimulation upon conditioned behaviour has been shown by T h o m p s o n (1958) using a reversal test in cats. S a d o w s k i (1962), in dogs and rabbits, also noted enhancement of the defence conditioned reflexes.

According to B u c h w a l d et a l. (1961 b) caudate nucleus stimulation can either inhibit or facilitate instrumental conditioned responses in cats, depending on the frequency of the current. Low frequency is inhibitory, whereas high frequency produces an excitatory effect.

Two questions arise from the above presented data. First, in considering the effect of caudate nucleus stimulation upon the conditioned reflexes it is necessary to take into account the possibility of concomitant excitation of the internal capsule. Secondly, the finding that the caudate nucleus exerts an important influence on the conditioned reflexes does not explain the mechanism of its action. It would be interesting to know if this influence results from the action of the caudate nucleus on the cerebral cortex or is due to subcortical connections of this structure. Therefore, investigations were undertaken on unilaterally decorticated rabbits in which a defence conditioned reflex was established to caudate nucleus stimulation, used as a conditioned stimulus. Electric stimulation of the brain used as a conditioned and/or unconditioned stimulus has been employed by a number of authors (L o u c k s 1935, K o n o r s k i and L u b i ń s k a 1939, D o t y, R u t l e d g e and L a r s e n 1956, G i u r g e a and R a i c i u l e s c u 1959, S e g u n d o, R o i g and S o m m e r - S m i t h 1959, T a r n e c k i 1962). This technique may contribute to elucidate the role of different nervous structures in the formation of the temporary link. Caudate nucleus stimulation was first used as a conditioned stimulus in cats by L a g u t i n a and R o z h a n-s k i i (1949) and, later, by N i e l s o n, D o t y and R u t l e d g e (1957) to investigate alimentary reflexes. S a d o w s k i (1960) applied it to study defense conditioning in rabbits.

METHODS

Experiments were performed on 11 adult male rabbits weighing over 2.5 kg. Operations were performed using the technique described elsewhere (S a d o w-s k i and T r a c z y k 1963). Briefly, the cerebral cortex of the left hemisphere was removed and electrodes implanted pair-wise into both caudate nuclei, using a stereotaxic instrument.

Establishing of conditioned reflexes was begun 2 to 4 weeks after operation. The method of V o l o k h o v and O b r a z t s o v a (1953) was applied in which the unconditioned reflex was shaking of the ears and head on electrical stimulation of the pinna. The nociceptive stimulus, electric current, was applied with a pair of electrodes clamped to the right pinna. The shaking movement was registered on a kymograph, using a mercury switch placed on the rabbit's head.

As the conditioned stimulus electric rectangular impulses of 50 cy/sec. frequency and 0.5 msec. duration were applied to the caudate nucleus, the amplitude being so chosen as to evoke no apparent behavioural effects (S a d o w s k i 1960). In each experiment the conditioned and unconditioned stimuli were paired 10 to 12 times, the former being applied alternately to the right and left caudate nucleus.

If a conditioned reflex upon caudate nucleus stimulation failed to develop, control experiments were carried out to see whether it could be established to a buzzer in the given animal.

After termination of the experiments a histological examination of the brain was made in the way described elsewhere (S a d o w s k i and T r a c z y k 1963).

RESULTS

Conditioned reflexes were investigated from 14 to 45 days after operation. First, the motor effect of the caudate nucleus stimulation was checked, and the amplitude of the current to be used as conditioned stimulus chosen. Out of the rabbits no conditioned reflex could be formed in six either to caudate nucleus stimulation or to the buzzer. These animals were, however, in poor health: did not eat, lost weight, etc. In 5 rabbits conditioning was established to stimulation of the caudate nucleus both in the decorticated and in the intact hemisphere. The reflex, however, was very irregular. While it attained a high level of regularity in single experiments, ranging from 80 to 100 per cent, the mean values were low and did not exceed 50 per cent of positive responses in relation to the number of trials (Fig. 1).

It should be emphasized that the shaking of ears and head is never by itself the effect of caudate nucleus stimulation and was obtained only through the process of conditioning.

Anatomical and histological analysis revealed an extensive cortical lesion comprising the entire left sensorimotor cortex and also large parts of the temporal and occipital areas. In all the rabbits the electrodes were localized in the head of the caudate nucleus, except for rabbit No. 43 in which the electrodes in the intact hemisphere entered the internal capsule.

DISCUSSION

The problem of conditioning in mammals after cortical ablation has been discussed in many papers. Although Z e l i o n y (1913) failed to establish conditioned reflexes in decorticated dogs, later investigations demonstrated that conditioning in such animals is possible. Defense conditioned reflexes in dogs after bilateral removal of the cerebral cortex were established by P o l t y r e v and Z e l i o n y (1929, 1930) and B r o m i l e y (1948 a), though in the opinion of C u l l e r and M e t t l e r (1934) only simple forms of conditioning may develop.

Motor conditioned reflexes, bath defense and alimentary, were quite easily formed in unilaterally decorticated dogs (P o l t y r e v and A l e k s e e v 1936), and light could be used as a conditioned stimulus in dogs after lesion of the optic cortex (T e n C a t e 1938, W i n g and S m i t h 1942, W i n g 1946). Motor conditioned reflexes in cats could
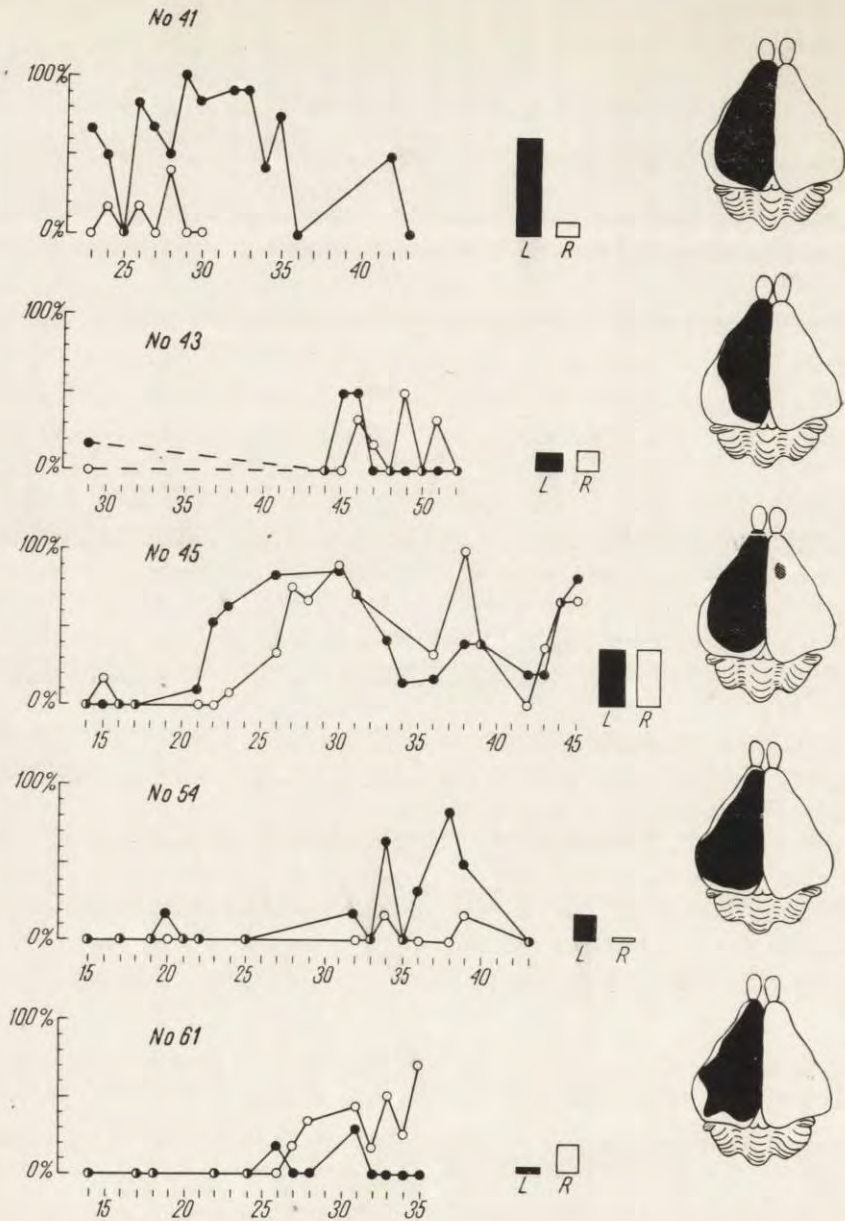
Fig. 1. The conditioned defense reflex in five rabbits. Horizontal axis shows the number of days after operation. Vertical axis shows the percentage of positive conditioned responses in relation to the number of trials in one experiment. Solid circles represent conditioned responses to electrical stimulation of the left caudate nucleus (in the decorticated hemisphere). Open circles represent responses to the right caudate nucleus stimulation. Black columns represent the average percentage of conditioned responses in the given animal to left caudate nucleus stimulation, the white ones the average percentage of conditioned responses to right caudate nucleus stimulation. Black spot on the brain scheme represents the ablated cortical region.

be obtained both after unilateral (B r o m i l e y 1948 b) and bilateral (T e n
C a t e 1934) decortication. T e n C a t e and v a n H e r k (1933) de-
monstrated that in rabbits after extensive bilateral cortical lesion it was
possible to establish alimentary motor conditioned responses whose me-
chanism resembled approach learning. This indicates that the lower the
species is phylogenetically, the less significant is cortical ablation for its
behaviour. G a s t a u t (1958) recognizes three levels of structures in-
volved in the formation of a temporary link. The lowest is the reticular
formation of the brain stem. The next comprises the thalamus, the striate
body and the limbic system. The highest level is represented by associat-
ive areas of the cerebral cortex. According to this point of view, the
formation of a simple temporary link occurs at the subcortical level, where-
as the cerebal cortex is necessary for the more complicated components
of the conditioned reflex.

In our rabbits with unilaterally ablated cerebral cortex, caudate
nucleus stimulation used as a conditioned stimulus evoked typical con-
ditioned responses. These responses were essentially the same, irrepective
of whether the caudate nucleus in the intact or decorticated hemisphere
was stimulated. This may indicate that the influence of the caudate nucleus
upon the conditioned reflex activity possesses an extracortical mechanism.
Two points, however, need clarifying. First, why only in half the rabbits
could the defense conditioned reflex be established, and, secondly, why
were the reflexes induced by the caudate nucleus stimulation so irregular.
As was said, the animals not responding to caudate nucleus stimulation
also did not respond to the buzzer. From the above mentioned literature
data it seems improbable that this unresponsiveness could be related to the
cortical lesion. It may be due, however, to the poor state of health of the
animals. The low regularity of the conditioned reflex in the remain-
ing rabbits possibly depends on the non-physiological character of
the conditioned stimulus which was, in our experiments, stimulation of the
caudate nucleus. Even in the work performed on rabbits with intact cere-
bral cortex (S a d o w s k i 1960) reflexes obtained in the same way were
found to be irregular, rarely attaining 80 per cent of performance. It
could be thought, too, that some inhibitory action develops to caudate
nucleus stimulation which reverses the final conditioning effect. Never-
theless, it is evident that the stimulation of the caudate nucleus in hemi-
decorticated rabbits does evoke conditioned responses and that the pre-
sence of an intact cerebral cortex is not essential. On the basis of this
finding it is necessary to look for descendent pathways of the caudate
nucleus which could intermediate the transmission of the excitatory pro-
cess to the structures responsible for the formation of the conditioned
reflexes. It is known that such pathways exist between the caudate nu-

cleus and the diffuse activating system. One of them connects this structure with the thalamus (W i l s o n 1914, H u b e r and C r o s b y 1929, D r o g l e e v e r - F o r t u y n and S t e f e n s 1951, 1952). The other comprises strionigral fibres recently studied by V o n e i d a (1960) through which the transmission of impulses to the mesencephalic reticular formation is possible. In this way, the formation of a temporary link could be established at the nonspecific systems level.

It is likely that the effect of caudate nucleus stimulation upon the conditioned reflex activity in animals with intact cerebral cortex depends also on the connections of this structure with the reticular system.

SUMMARY

1. In rabbits, in which the cerebral cortex had been unilaterally removed, it was possible to establish defense motor conditioned reflexes by associating the caudate nucleus stimulation with a nociceptive stimulus.

2. Although the reflex so obtained was irregular, there were no apparent differences between the intact and the decorticated side.

3. It is suggested that the influence exerted by the caudate nucleus upon the conditioned reflex activity involves extracortical mechanisms, probably through its connections with the diffuse activating system.

REFERENCES

BROMILEY R. B. 1948 a — Conditioned responses in a dog after removal of neocortex. J. comp. physiol. Psychol., 41, 102.

BROMILEY R. G. 1948 b — The development of conditioned responses in cats after unilateral decortication. J. comp. physiol. Psychol., 41, 155.

BUCHWALD N. A., WYERS E. J., CARLIN J. and FAIRLEY R. 1960 — Effects of stimulation of the caudate nucleus on visual discrimination in cats. Anat. Rec., 136, 172.

BUCHWALD N. A., WYERS E. J., CARLIN J. and FAIRLEY R. 1961 a — Effects of caudate stimulation on visual discrimination. Exper. Neurol., 3, 556.

BUCHWALD N. A., WYERS E. J., LAUPRECHT C. W. and HEUSER G. 1961 b — The "caudate-spindle". IV. A behavioral index of caudate-induced inhibition. EEG Clin. Neurophysiol., 13, 531.

CHERKES V. A. 1955 — Changes of conditioned reflexes in dogs depending on the site of stimulation of subcortical structures. J. Higher Nerv. Activity, 5, 415 (in Russian).

CHERKES V. O. 1957 — On the physiology of subcortico-cortical relationships. Physiol. J. (Kiev), 3, 25 (in Ukrainian).

CHERKES V. A. 1958 — The caudate nucleus stimulation and motor conditioned reflexes. XVIII Symposium on the Problems of the Higher Nervous Activity, Leningrad 1, 141 (in Russian).

CULLER E. and METTLER F. A. 1934 — Conditioned behavior in decorticated dog. J. comp. Psychol., *18*, 291.

DOTY R. W., RUTLEDGE L. T. and LARSEN R. M. 1956 — Conditioned reflexes established to electrical stimulation of cat cerebral cortex. J. Neurophysiol., *19*, 401.

DROOGLEEVER-FORTUYN J. and STEFENS R. 1951 — On the anatomical relations of the intralaminar and midline cells of the thalamus. EEG Clin. Neurophysiol., *3*, 393.

DROOGLEEVER-FORTUYN J. and STEFENS R. 1952 — Thalamus, striate body and cortex. EEG Clin. Neurophysiol., *4*, 109.

GASTAUT H. 1958 — Some aspects of the neurophysiological basis of conditioned reflexes and behaviour. Ciba Foundation Symposium on the Neurological Basis of Behaviour, London pp. 255—276.

GIURGEA C. and RAICIULESCU N. 1959 — Étude électroencéphalographique du réflexe conditionnel à l'excitation électrique corticale directe. First International Congress of Neurological Sciences, London, *3*, 156.

HUBER G. C. and CROSBY E. C. 1929 — Somatic and visceral connections of the diencephalon. Arch. Neurol. Psychiat., *22*, 187.

KLOSOVSKII B. N and VOLZHINA N. S. 1956 a — On the functional rôle of the corpus striatum. Problems of Neurosurgery, *20*, 8 (in Russian).

KLOSOVSKII B. N. and VOLZHINA N. S. 1956 b — The method of ablation of the corpus striatum. J. Physiol. USSR, *42*, 817 (in Russian).

KONORSKI J. and LUBIŃSKA L. 1939 — Sur un procédé nouveau d'élaboration des réflexes conditionnels du II type et sur les changements d'excitabilité du centre cortical moteur au cours de l'apprentssage. Acta Biol. Exper., *13*, 143.

LAGUTINA N. I. and ROZHANSKII N. A. 1949 — On the distribution of subcortical alimentary centres. J. Physiol. USSR, *35*, 587 (in Russian).

LOUCKS R. B. 1935 — The experimental delimination of neural structures essential for learning: the attempt to condition striped muscle response with faradization of the sigmoid gyri, J. Psychol., *1*, 5.

NIELSON H. C., DOTY R. W. and RUTLEDGE L. T. jr — 1957. Subcortical stimulation as motivation and as signal for conditioned responses. Feder. Proc., *16*, 93.

POLTYREV S. S. and ALEKSEEV V. A. 1936 — Über die Möglichkeit der Bildung bedingter Reflexe bei Hunden mit exstirpierter Hirnrinde von der der Hemisphäre gegenüberliegenden Körperoberfläche aus. Z. Biol., *97*, 297.

POLTYREV S. S. and ZELIONY G. P. 1929 — Der Hund ohne Grosshirn. Amer. J. Physiol., *90*, 475.

POLTYREV S. and ZELIONY G. 1930 — Grosshirnrinde und Assoziationsfunktion. Z. Biol., *90*, 157.

ROMANOVSKAYA E. A. 1957 — Some effects of lesions within the corpora striata of rabbits and dogs. Bull. Exp. Biol. Med., *44*, 43 (in Russian).

ROMANOVSKAYA E. A. 1958 — The effect of lesion and ablation of corpora striata on the conditioned reflex activity of rabbits and dogs. XVIII Symposium on the Problems of the Higher Nervous Activity, Leningrad, *1*, 124.

ROSVOLD H. E. and DELGADO J. M. R. 1956 — The effect on delayed-alternation test performance of stimulating or destroying electrically structures within the frontal lobes of the monkey's brain. J. comp. physiol. Psychol., *49*, 365.

ROSVOLD H. E., MISHKIN M. and SZWARCBART M. K. 1958 — Effects of sub-
cortical lesions in monkeys on visual discrimination and single alternatior.
performance. J. comp. physiol. Psychol., 51, 437.

SADOWSKI B. 1959 — Defensive conditioned reflexes in rabbits after unilateral
extirpation of caudate nucleus. Acta Physiol. Polon., 10, 1.

SADOWSKI B. 1960 — Electric stimulation of the caudate nucleus as a stimulus
for conditional defence reflex in rabbits. Bull Acad. Pol. Sci. Série Sci
Biol., 8, 369.

SADOWSKI B. 1962 — Investigations on the function of the caudate nucleus (in
Polish). Rozpr. Wydz. Nauk Med., 7, 63.

SADOWSKI B. and TRACZYK W. 1963 — Stimulation effects and bioelectrical
activity of the caudate nucleus in unilaterally decorticated rabbits and
cats. Acta Biol. Exper. (Warsaw), 23, 101.

SEGUNDO J. P., ROIG J. A. and SOMMER-SMITH J. A. 1959 — Conditioning of
reticular formation stimulation effects. EEG Clin. Neurophysiol., 11, 471.

SOŁTYSIK S. 1961 — In: Brain Mechanisms and Learning. Chapter by J. KO-
NORSKI, The physiological approach to the problem of recent memory,
Oxford, pp. 115—132.

STEVENS J. R., KIM C. and McLEAN P. D. 1961 — Stimulation of caudate nucleus.
Behavioral effects of chemical and electrical excitation. Arch. Neurol., 4, 47.

TARNECKI R. 1962 — The formation of instrumental conditioned reflexes by di-
rect stimulation of sensori-motor cortex in cats. Acta Biol. Exper. (War-
saw), 22, 114.

TEN CATE J. 1934 — Können die bedingten Reaktionen sich ausserhald der Gros-
shirnrinde bilden? Arch. Néerl. Physiol., 19, 469.

TEN CATE J. 1938 — Bedingte Reflexe bei Hunden nach beiderseitiger Exstirpation
der Regio occipitalis der Grosshirnrinde. Arch. Néerl. Physiol., 23, 219.

TEN CATE J. and van HERK A. W. H. 1933 — Beobachtungen an Kaninchen nach
Exstirpationen im Neopallium. Arch. Néerl. Physiol., 18, 337.

THOMPSON R. 1958 — The effect of intracranial stimulation on memory in cats.
J. comp. physiol. Psychol., 51, 421.

THOMPSON R. L. 1959 — Effects of lesions in the caudate nuclei and dorsofrontal
cortex on conditioned avoidance behavior in cats. J. comp. physiol. Psy-
chol., 52, 650.

VOLOKHOV A. A. and OBRAZTSOVA G. A. 1953 — A method of investigation of
conditioned reflexes in animals in ontogenesis. Bull. Exp. Biol. Med., 36, 68
(in Russian).

VONEIDA T. J. 1960 — An experimental study of the couse and destination of
fibers arising in the head of the caudate nucleus in the cat and monkey.
J. Comp. Neurol., 115, 75.

WILSON S. A. K. 1914 — An experimental research into the anatomy and physio-
logy of the corpus striatum. Brain, 36, 427.

WING K. G. 1946 — The rôle of the optic cortex of the dog in the retention of
learned responses to light: conditioning with light and shock. Amer. J. Psy-
chol., 59, 583.

WING K. G. and SMITH K. U. 1942 — The rôle of the optic cortex in the determi-
nation of the functional properties of conditioned reactions to light.
J. Exper. Psychol., 31, 478.

ZELIONY G. P. 1913 — Observation sur des chiens auxquels on a enlevé les
hémisphères cérébraux. C. R. Soc. Biol. Paris, 74, 707.

PROPERTIES OF MULTIPLE CONDITIONED REFLEX TYPE II ACTIVITY

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According to the general experimental practice the technique of instrumental conditioned reflexes (CRs) is such that a conditioned stimulus (CS) elicits the trained motor response which is immediately or almost immediately reinforced by food, or by avoidance of a nociceptive stimulus. Only in those experiments, in which the salivary outflow is recorded in parallel with the motor response, is it useful to apply a constant interval between the CS and US amounting up to 15 seconds or more. In such instances, the animal usually performs the trained movement several times, but irrespective of the number of motor performances, the food reinforcement is presented only once per trial.

In this connection, it is interesting to study the behaviour of the animal in a situation in which instead of a single movement being necessary for obtaining food, the animal would be required to perform such a movement n times in succession. It is well known from Skinner's experiments (F e r s t e r and S k i n n e r 1957) that rats may be trained to perform several dozen lever pressings for one pellet of food.

It was felt that this problem should be reinvestigated on dogs by applying somewhat different methods. In contrast with the Skinner's procedure the multiple conditioned reflex was trained not only in response to the experimental situation, but, also to the presentation of a CS. Next, in an attempt to analyze the mechanism of this reflex behaviour, both the motor performance and the salivary outflow during the multiple conditioned reflex activity were simultaneously recorded.

MATERIAL AND METHODS

Experiments were carried out on 6 mongrel male dogs aged from 3 to 7 years at the beginning of the investigation. During testing the animals were placed in harness in a Pavlovian frame mounted within a relatively soundproof conditioned--reflex chamber. In front of the animal a food tray containing 10 small cups was situated. The food cups were successively delivered by the observer from outside the experimental room. The animal faced only one cup each time as the remaining 9 cups were hidden under the top lid of the food tray. Before every testing session one piece of bread weighing approximately 3 gms. was placed into each cup.

In the preliminary training (experimental series No. 1) a single performance of the CR type II response was established according to Konorski and Miller technique (1933, 1936).

In Group I (3 animals) the animal's right fore limb was passively placed on the food tray, whereupon food was presented. After a few days the animals began to react by active placing their limb on the food tray and received food reinforcement. Immediately after ingestion of food they again produced the trained response which was followed by the second presentation of food. They then performed the next response, received food, etc.

During testing the Group I animals ate ad libitum, and they signalled the end of each experimental session by their refusing to take more food, and turning away. Out of the experimental situation the animals were given meals of a definite size which consisted of cereal, broth, meat and vegetables.

Once the single performance of the CR was well established, the animals were induced to repeat the acquired response. Thus, in the experimental series No. 2, they were required to double the CR performance in order to get the same food reinforcement which had previously been given for a single response; in series No. 3, the response had to be tripled, etc. Briefly, in every consecutive experimental series, which comprised 8 to 14 testing sessions, one, or from series No. 10 up, more than one, additional performances of the CR per trial were trained. In consequence, a multiple conditioned reflex type II (MCR) habit was trained.

The animals in Group II (3 animals) were treated like those in Group I, except that the CR was trained to the presentation of a conditioned stimulus (CS). After establishment of a single fore limb response to the sound of a bell in the initial training (experimental series No. 1), the performances of the CR were progressively increased in consecutive testing series until the MCR type II response was produced. Each trial, which consisted of a single performance of CR in the experimental series No. 1, or its multiplicity in further series, was followed by food reinforcement of the same size. The initial experimental series differred from one another by one additional CR performance. From series No. 6 onward, 2, 3, 4 or 5 additional performances of the CR were executed in successive series. In all instances, however, the series number denoted the CR performances, e.g., the experimental series No. 13 indicated that the CR response was produced 13 times in each trial, No. 25 indicated 25 responses. The initial series consisted of 10 testing sessions. Series Nos. 4 and 5 composed 8 sessions, and every subsequent experimental series, 6 sessions. In each series, 9 trials daily were used.

The course of every testing session was recorded. The animal's right fore limb was connected to a flexible rubber tape which allowed him to activate a pneumatic device as the limb got flexed. In order to show the instrumental response graphically, a kymograph was used which moved horizontally at a speed of 5 cm. per sec.

Also, all details of the animal's behaviour were carefully observed throughout the entire experiment, and changes in them were noted.

The meal offered during testing of Group II animals was prepared by mixing 8 gms. ground biscuits, 25 cc. broth and 5 gms. cooked horse meat. Out of the experimental situation the animals were maintained on a diet consisted of cereal, broth meat and vegetables.

The Group II animals had a shortened right parotid gland fistula made according to the surgical procedure of S o ł t y s i k and Z b r o ż y n a (1957) which allows one to avoid artefacts in salivary records obtained by the application of the classical G l i ń s k i operation (P a v l o v 1894—1895). The salivary outflow was registered on the kymograph by the use of K o z a k's recording technique (1950) by which the volume and the rate of salivation may be defined.

RESULTS

Group I animals

Training of a single CR type II performance was uneventful. All animals acquired this type of responding within a few days, and they performed it regularly after each ingestion of the food. Since food was presented until the dogs worked for it, the testing session terminated when the CR activity discontinued, and the animal turned away from the food tray. After that the animals were released, and they readily left the experimental room. If food was then offered to them in the pre- -experimental room, they refused to take it.

As the results obtained on the animals of Group I were very similar, a description of dog No. 1 will be given in detail, and only some peculiar aspects of the two remaining dogs' behaviour will be added.

T r a i n i n g   o f   MCR   i n   d o g   No. 1. In the experimental series No. 1, the dog made 60 single conditioned limb responses in average and received 60 small pieces of bread per day.

In the experimental series No. 2, the dog was required to perform a double CR response, and he did it quite easily if the single performance was not reinforced. Also, in the experimental series No. 3 the dog immediately learned to repeat the response three times for a piece of bread.

Towards the end of series No. 4 the animal began to show some irritation, and in isolated trials, he stopped working after performing the response twice or three times. He often did not take off his leg from the food tray, and occasionally, 15, 20 or even 45 seconds elapsed before he produced the final one or two performances to complete the trial.

No disturbances were noticed in further testing series up to No. 20. The dog was relatively quiet, and worked well. First appreciable irregularities occurred during the establishment of 20 CR performances per one trial. Since the animal refused both to work and to eat in the con-

ditioned-reflex chamber, testing was discontinued. However, after a 10-
-day break full recovery occurred which persisted up to the experimental
series No. 40.

It is to be stressed that with the increase of CR performances per trial,
the dog reduced the number of trials per day, and signalled the termin-
ation of testing sessions after a smaller number of reinforcements than
at the beginning of the investigation, that is to say, he ate less in the
experimental room than primarily. For example, in the experimental
series No. 20, food was presented 18 times daily on an average, what is
only one third of that given during the series No. 1. The dog also continued
to refuse eating immediately after testing, if he was offered food close
to the conditioned-reflex chamber. Nevertheless, his food intake in the
animal building was quite all right.

In further experimental series, in which the testing sessions were pro-
gressively extended, and, sometimes, persisted for nearly one hour, the
conditioning and feeding patterns changed considerably. The rate of
responding and ingestion increased, and the dog appeared to gobble each
piece of bread, or produced the next series of responses before having
swallowed the food which had been given previously. He also showed
a burst-type of responding in some of the trials, that is, the very rapid
CR performances were interrupted by short intervals of inactivity. For
example, after performance of responses with increased frequency the
animal stopped working, looked around or stared into the space, and then
continued responding at a rapid rate until the presentation of food. At
times, a few bursts of fast activity were separated by relatively long
periods of no-responding.

Such was the animal's behaviour up to the testing series No. 90 in
which the number of trials dropped down to 2 or 3 per day, and the dog
began to accept food after the end of the experimental session in the
vicinity of the testing room.

In the experimental series No. 100, a further decrease in the number
of daily trials occurred. In most instances, there was in fact only one
trial within a session, that is, after being placed in the frame the dog
began to work at a very rapid rate, and after completion of the required
number of CR performances and ingestion of food, he turned away and
refused to responding. If he was given a "gratis" piece of bread, he ate it,
but this did not induce him to restore his type of working. However, he
improved after a 2-day pause.

On the other hand, a 24-hour food deprivation did not promote the
dog to increase the number of trials.

To sum up, one may say that, up to the testing series No. 25, the increase of the number of CR performances per trial was associated with a decrement of the number of reinforcements (what is tantamount to a decrease of the number of trials), although the total number of CR responses within a session tended to grow. But, as may be seen in Table I, beyond this point both the trial number and the conditioning effort showed a sharp decrease (Table I).

Table I

Relationship between the size of MCR performance per trial and the total MCR performance per testing session in some of experimental series in dog No. 1

Number of CR performances per trial (=No. of testing series)	Average number of trials (reinforcements) per testing session	Total number of CR performances per testing session (mean values)
1	60	60
10	45	455
15	33	515
25	35	925
40	9	280
70	4	250
100	2	200

Extinction and restoration of MCR in dog No. 1

After completing the experimental series No. 100, attempts were made to test the resistance of MCR to extinction. This was carried out by the acute extinction procedure which implied no food presentation for several days in succession under the testing circumstances.

Table II

Extinction of MCR. Dog No. 1

Extinction session No.	1	2	3	4	5	6	7	8	9	10	11	12	13	14
Total number of CR responses	250	560	260	320	260	200	420	220	170	280	220	0	0	0

Table II shows that despite the absence of reinforcement the MCR did occur in a variable size during 11 extinction sessions. From the 12-th day onward, the animal refused working. However, within a few further sessions the MCR performance was restored by simply offering the bread to the animal, a procedure which had previously been utilized by W y r-

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w i c k a (1952) for restoring a single CR performance. Under our circumstances isolated responses or short-lasting bursts of MCR activity recovered first, and they thereafter developed into series of 40 to 60 rhythmic performances per trial.

T r a i n i n g o f M C R i n d o g s N o s. 2 a n d 3. The course of MCR training in these two dogs was principally the same as in dog No. 1, except that dog No. 2 stopped working for food in the experimental series No. 40, and dog No. 3 did it in series No. 30. Table III illustrates the relationship between the number of responses per trial and the total performance of MCR per testing session in some of the experimental series. No extinction of MCR was made in these two dogs.

Table III

Relations between the size of MCR performance per trial and the total MCR performance per testing session in some of experimental series in dogs Nos. 2 and 3

Number of CR performances per trial (No. of testing series)	Average number of trials (reinforcements) per testing session	Total number of CR performances per testing session (mean values)
Dog No. 2		
1	68	68
35	13	448
40	8	328
Dog No. 3		
1	39	39
4	53	212
30	4	120

G r o u p II a n i m a l s

T h e M C R p e r f o r m a n c e. The acquisition of a single CR type II performance was essentially identical with that described in the former animal group, except that the response occurred to the sound of a bell. The animals worked perfectly, and readily ate the proffered food.

The situation became complicated, however, during the establishment of the double CR response (experimental series No. 2). Initially, when the single performance was not followed by food reward, it was duplicated after a delay. However, in the next trials the animals often refused doubling the CR, and instead, they insisted on receiving the food reward by scratching at the food tray and trying to turn over the cups. Eventually, they produced the second response after 5 to 20 seconds.

In dog No. 4, disturbances in the double responding pattern occurred again on the second testing day of series No. 2. Following three correct trials the animal stopped working, and ignored the CS. To avoid extinction of the instrumental activity the last few trials were reinforced despite the absence of responding. On the next day, the animal performed well on all the trials, and from then on, he easily produced the double CR to each presentation of CS. However, in two subsequent experimental series disturbances of this type occurred again in this dog.

In two other dogs, the training of MCR was uneventful throughout several initial testing series: the animals produced the required number of responses at a maximal rate, and thereafter ingested the food (Fig. 1 and 1a).

An interesting finding was the fact that with an increase in the size of the MCR the rate of responding was still very rapid, but the original performance within each trial got progressively delayed. In other words, in contrast with the initial training, the CR did not occur immediately to the presentation of CS, but was elicited after a latency of 5 to 10 seconds.

In further experimental series, namely from No. 16 onwards, the animals began to display a variety of changes in their behaviour. Thus, they resisted to enter the testing room, and after their placement in the frame they tended to escape or get rid of the harness. They also scratched and gnawed the food tray, or screamed and howled, and tore off the saliva collection capsule attached to their cheek. As to the MCR, it was clearly seen that a depression of the instrumental activity began (Fig. 2). Although the animals kept the MCR performance approximately normal on most of the trials, they, occasionally, refused working at all, or reduced the frequency of their responding pattern. With time, the decrement of MCR became even more pronounced by the animals failing to respond on an increasing number of trials, or by not completing the performance. Since they were not offerred food if the required number of responses failed to be elicited, they became even more irritated, and finally they discontinued to respond at all. It is to be emphasized, however, that the depression phase of the MCR habit was very slow. Thus, although the animals remarkably extended the latency of their conditioning performance (see Fig. 2), they often showed some tendency to revert to the normal pattern of responding. To illustrate this Table IV with an excerpt of a typical protocol of series No. 25 in dog No. 4 is given, but the above holds also true for the two remaining dogs.
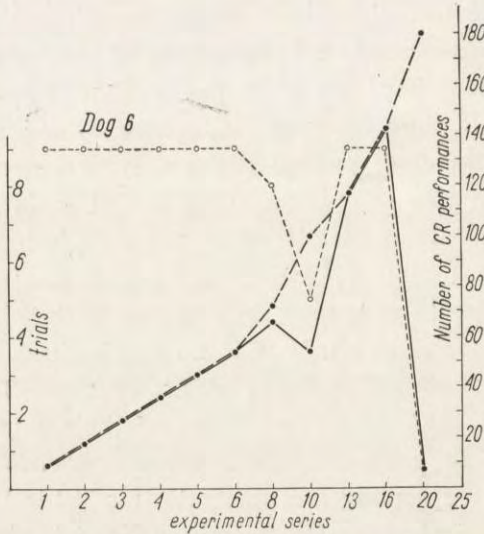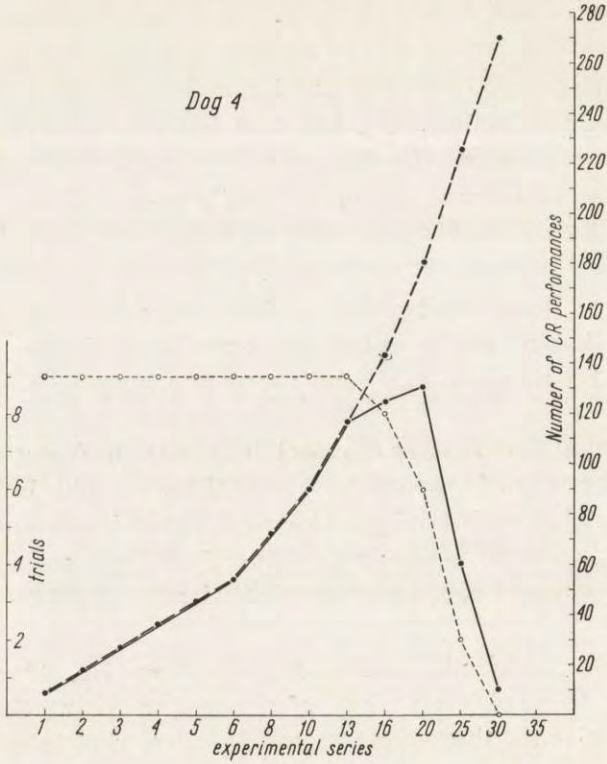
Fig. 1. Variations of MCR in succesive experimental series
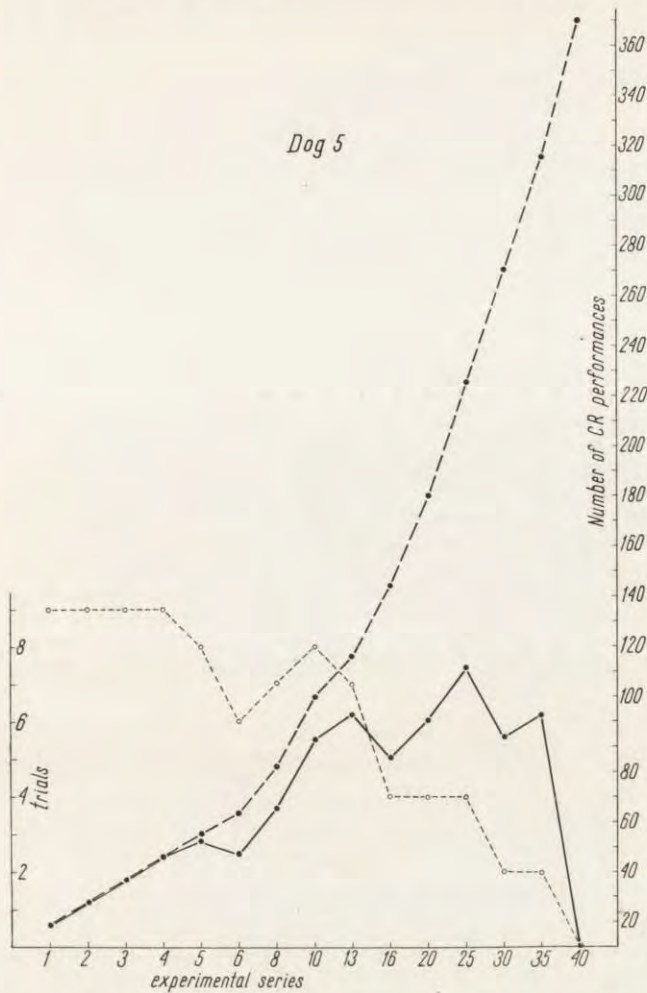Full description in Fig. 1a

Fig. 1a. Variations of MCR in successive experimental series

Abscissa: experimental series (the experimental series number denotes the number of CR performances per trial). Ordinate: on the left, number of trials per day; on the light, average total MCR performance per day. Light dashed line, changes in the number of trials per day as a function of the number of CR performances per trial. Note that the number of trials per day drops with increasing number of CR performances per trial. Heavy dashed line, hypothetical (calculated) MCR performance (the required number of responses to be elicited). Solid line, actual MCR performance. Note that the total MCR performance declines with increasing number of CR performances per trial

The animals' irritation, which attained its maximum before the abolition of MCR, seemed to interfere with the instrumental activity, since it systematically occurred when the acquired response pattern was delayed or absent. Thus, in most of the trials during the late MCR depression period the dogs at first showed a tripping step in response to the sound of the bell, and only after a while did they produce an abortive flexion
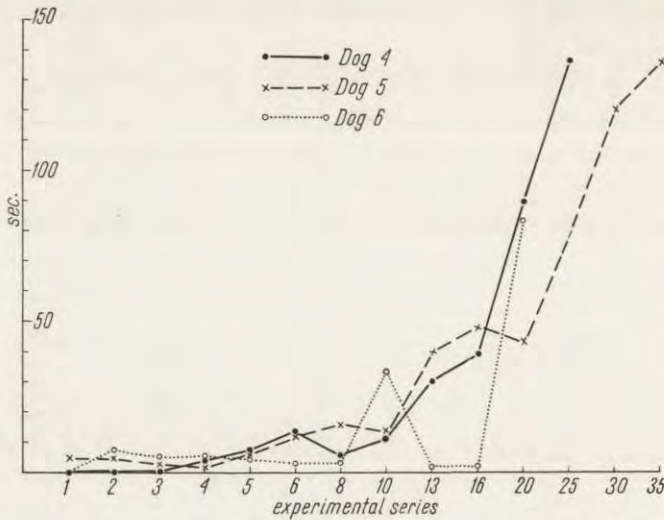
Fig. 2. Average latency of MCR

Abscissa: experimental series (= number of CR performances per trial), ordinate: time in secs. Each point represents average of 10 days. Note that the latent period of MCR performance increases with increasing number of responses per trial

which was immediately cut down, and again followed by tripping at the food tray. This type of behaviour prevailed, although, in isolated instances, the animals did produce the MCR and ingested food.

Table IV

Experimental series No. 25. Dog. No. 4

Trials	Time from the beginning of the testing session	Latent period of MCR in sec.	Duration of the CS in sec.	Number of CR performances	Reinforcement
1	15 sec.	no response	180	0	no reinforcement
2	4 min. 30 sec.	no response	180	0	no reinforcement
3	11 min. 30 sec.	105	285	4	no reinforcement
4	15 min.	no response	180	0	no reinforcement
5	21 min.	15	240	25	food reward
6	28 min.	175	305	25	food reward
7	36 min.	no response	180	0	no reinforcement
8	41 min.	no response	180	0	no reinforcement
9	47 min.	80	230	25	food reward

As mentioned, above, the MCR showed a striking tendency to occur after a long latency (see Fig. 2), but once it occurred, it practically always was elicited in its full size, although its course was marked by two distinctly separated phases: the very slow responding pattern at the

beginning was followed by an increasingly rapid performance towards the end of the trial (Fig. 3).

When the MCR performance was abolished, and it did not recover within three consecutive testing days, a two week interval was applied, but no recovery was noted after that period either. Conversely, the animals showed a profound apathy under the testing conditions, and appeared to be very upset by the presentation of the CS. While during
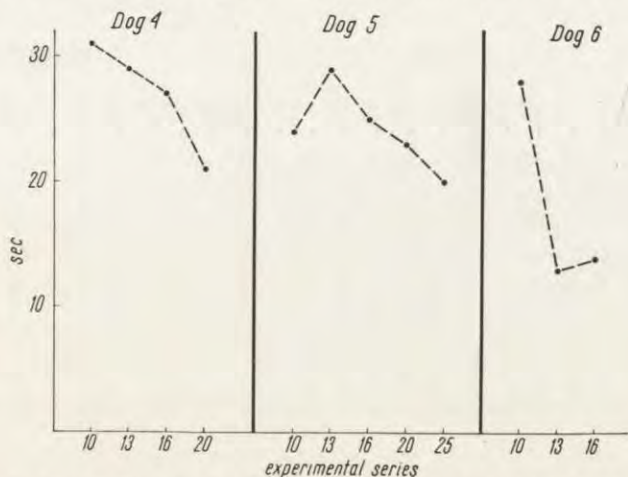

Fig. 3. The duration of the final 10 components of MCR
Abscissa: experimental series (= number of CR performances per trial). Ordinate: time in secs. Note that the rate of MCR performance increases towards the end of the trial with increasing number of CR performances per trial

the intertrial intervals they stood nearly motionless and maintained an upright position, in response to the CS they leaned their head on the food tray, kept their eyes closed, and moaned. They also continued to refuse responding after a 24-hour starvation. Only in one case did the MCR occur, and this was when the food reward was offered „gratis" to the animals. However, the recovery was transient: the MCR was produced a few times, and thereafter it declined again. From then on, it sustained a permanent suppression.

As seen in Fig. 1 the MCR performance was discontinued at different times in the animals of Group II.

T h e   s a l i v a r y   o u t f l o w   d u r i n g   t h e   MCR   p e r f o r m-
a n c e. Analysis of kymograms indicates that a steady level of performance on MCR coincides with a relatively constant equilibrium of salivary outflow whereas an abrupt depression of the MCR is associated with an immediate drop of the saliva production.

On the other hand, if the animal performs well on all the trials, the
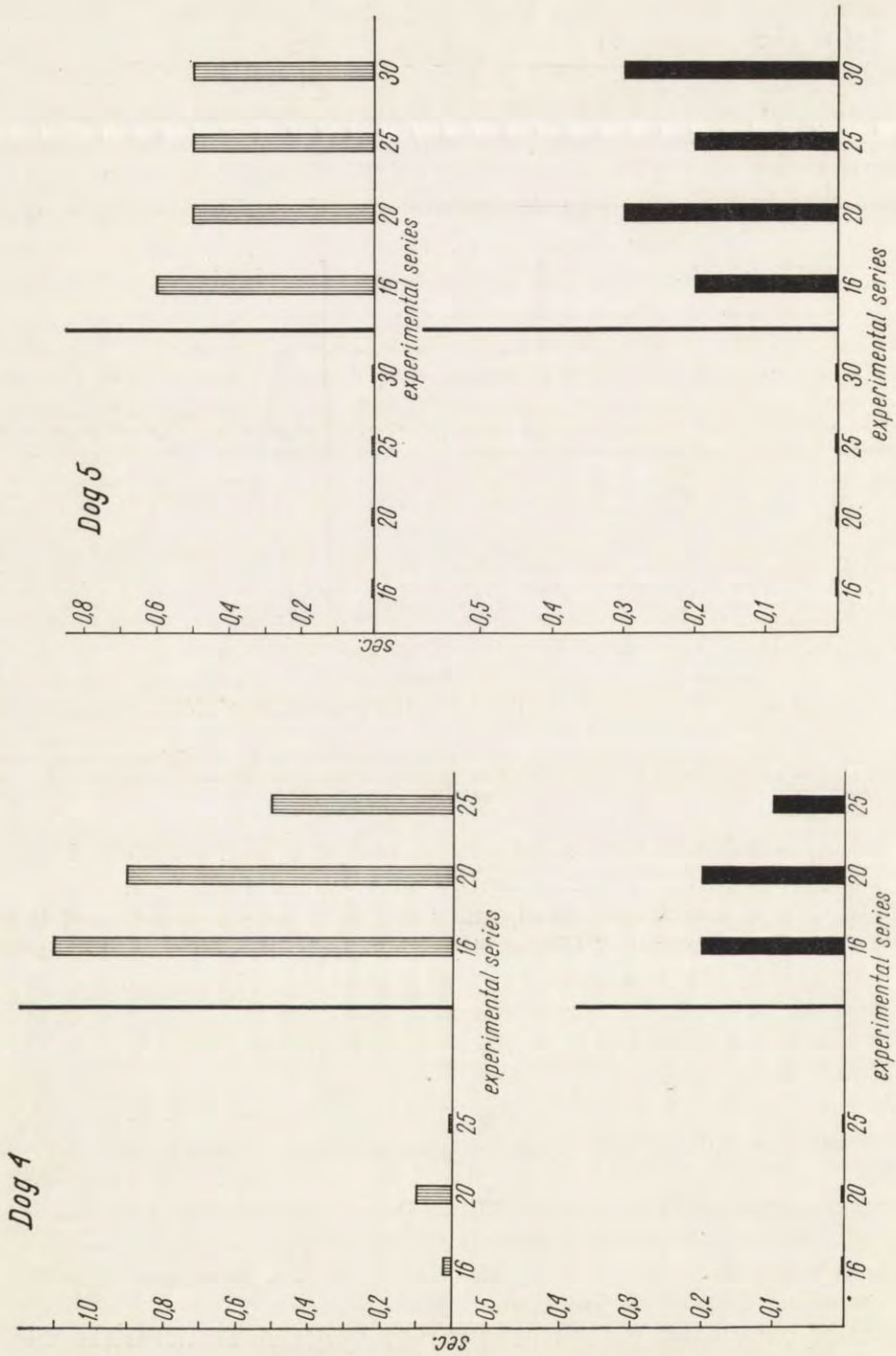
Fig. 4. Salivary outflow during the latent and active periods of MCR. See text
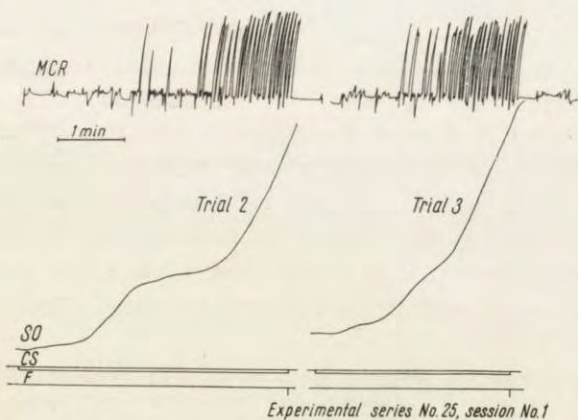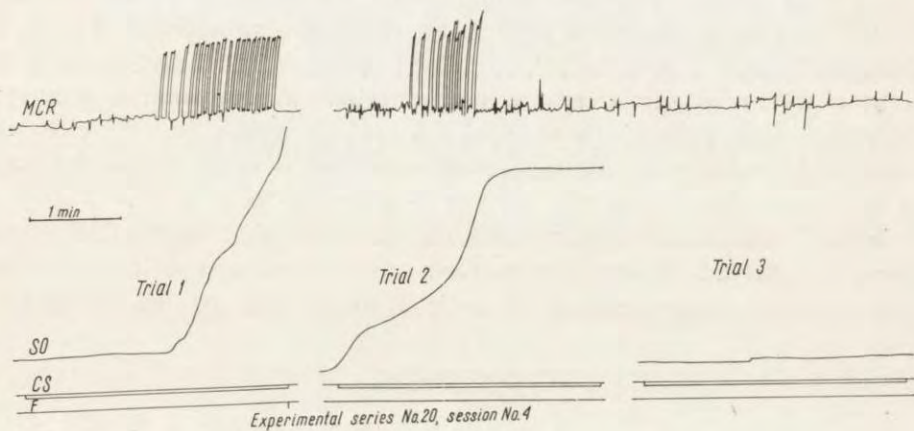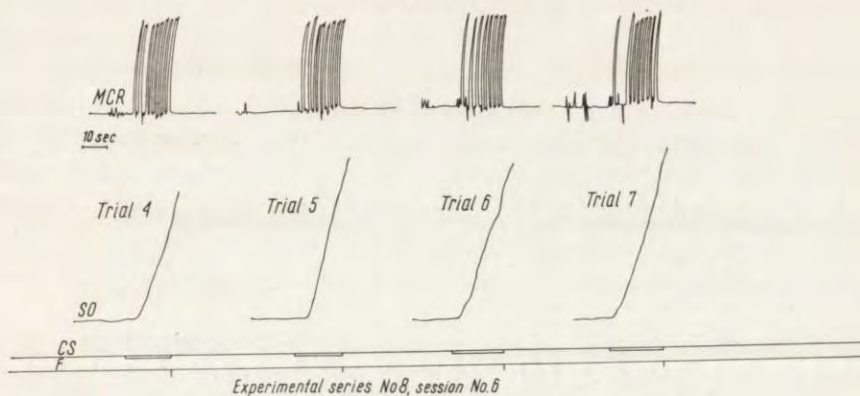
Dog 4

Fig. 5. Voluminograms of salivary outflow correlated with MCR performance in dog No. 4

MCR, tracing of the multiple conditioned reflex; SO, tracing of salivary outflow; CS. conditioned stimulus; F, food presentation

saliva remains at a balanced level throughout the entire experimental
session, and even by the very end of the testing day no appreciable
fluctuations are seen.

Rocords of experiments clearly indicate that the MCR, which has been
associated with food reinforcement, is intimately correlated with the
salivary reflex, and that regardless of the fact of whether the motor
performance immediately follows the presentation of CS or occurs after
some latency, it is paralleled with salivation. In other words, the saliva
is never produced independently of MCR, as simply a product determined
by CS.

Fig. 4 shows the mean values for salivary flow obtained during the
latent and active periods of MCR. In the upper left part of the figure the
quantity of salivary flow per second during the phase of motor inactivity
is plotted, and in its upper right part, the saliva production during the
MCR performance may be seen. In the lower left and right parts of Fig.
4, the MCR during the latent and active periods of performance are shown
respectively. In general, these results point out again to a very close cor-
relation between the autonomic salivary reflex and the complicated
pattern of voluntary activity represented by the MCR habit.

Since a cumulative record does not ascertain this correlation as pre-
cisely as individual comparisons, tracings representing continuous record-
ings from a kymogram taken directly from testing session are given in
Fig. 5.

DISCUSSION*

The results obtained in this paper show that the dogs are able to
perform a multiple conditioned motor response (MCR) ranging up to
about 100 trained movements in a single trial. This effect was achieved
by training consisting in a gradual increase of the instrumental responses
required for each reinforcement.

The establishment of the MCR occurred not quite smoothly. First,
disorders of the animal's behaviour were seen at the very beginning of
training when two or three instrumental responses, instead of a single
one, were required for food reinforcement. The dogs became restless and
sometimes refused to take food presented to them. Only after a few days
did the animals become quiet again and easily perform a double, triple or
even multiple conditioned response.

The explanation of this fact does not present difficulty. When only
a single movement was required to receive food, after its performance
the animal immediately displayed a classical conditioned response con-
sisting in turning to the bowl and copious salivation. When the food was

* Discussion was written jointly by J. K o n o r s k i and the author.

not presented, the animal performed the movement again and this time reinforcement followed. In effect, after several repetitions of such trials a conflicting situation arose connected with "irregular" reinforcement of the performed movement. However, when this training continued, the conditioned role of the first movement was changed in a way that instead of eliciting a classical conditioned response, it become a signal for producing the next movement, and thus the animal's behaviour again become regular.

The successive stages of the training of MCR were much facilitated by the fact that the animal had already learnt that the performance of a movement might be a signal of performing the next movement, and that such repetition eventually led to food reinforcement. Thus, the series of mevements required for one reinforcement could be made longer and longer till another crisis, more deep and decisive, crept gradually in.

The first characteristic change in the animal's responses which occurred with the increase of the number of required movements consisted in gradual prolongation of the latency of the performance of the first movement. During this latent period the animal was restless, whined and barked till he "decided" to perform the movement. After that he immediately quietened down and started to perform matter-of-factly the trained movement with increasing frequency. This was accompanied by copious salivation. When the number of required movements was increased beyond a certain limit, the animal refused to work altogether and displayed general restlessness throughout the operation of the CS. This total inactivity could not be removed either by food deprivation, or by making food more attractive. The only way of making the animal active again was to return to shorter series of required movements.

It should be noted in parenthesis that all this behaviour cannot be easily explained by any psychological or rationalistic considerations. Of course, fatigue is here totally out of the question, since the animal is able to perform the trained movements hundreds of times if they are interspersed by food reinforcement, and his difficulty lies exclusively in the initiation of the series of movements and not in its termination. And so, if the animal is hungry and the food reinforcement is attractive his most rational behaviour should be to start the series of movements at once and in this way get food as quickly as possible within the given condition. Thus the prolongation of the latent period of the response is clearly "antihedonistic".

However, if we attempt to explain these facts from the positions of the physiology of higher nervous activity, we may find that they are not only evident but also easy to be foreseen.

The prolongation of the series of movements required for reinforce-

ment necessarily leads to the prolongation of the CS-US interval. This
in turn leads to the formation of inhibition of delay in the first period
of the operation of the CS. This inhibition is clearly documented by
absence of the salivary conditioned reflex, and, of course, exerts its
influence upon the instrumental response, too. However, during the
operation of the CS the excitatory CR gradually takes the upper hand, the
trained movements begin to be performed, and salivation starts. The
nearer the moment of reinforcement, the more frequent the instrumental
responses become and the more rapid is the flow of saliva.

It is easy to observe that the prolongation of the latent period of
movements causes a vicious circle, since it delays the initiation of the
movements and, in consequence, the moment of reinforcement is still
more delayed. Because of this, we often noticed that with the greater
numbers of required movements the latent periods gradually increased
and eventually the animal refused to working altogether, which means
that inhibition totally overruled the excitatory processes.

Since, in our experiments both salivary and motor conditioned respon-
ses were recorded, it was possible to examine closely their mutual relations
in the MCR test.

Generally, the problem of interrelations between the salivary and
motor response in the type II CRs is far from being simple. According to
the original concepts of K o n o r s k i and M i l l e r (1933, 1936, see also
K o n o r s k i 1962), in the type II conditioning training only the compound
composed of the exteroceptive stimulus and proprioceptive stimulus gener-
ated by the movement, is the type I signal of food while the extero-
ceptive stimulus acting alone, being never reinforced, is differentiated.
In consequence, the prediction of the theory was that salivation to the
external stimulus alone should be nil or negligible, and would start only
after the performance of the trained movement. However, experimental
practice does not confirm this prediction, since in many instances sali-
vation starts, and is even very copious, right from the onset of the exte-
roceptive stimulus, while the movement performed after a longer latency
does not add anything to its rate. The impression is that in those cases
in which the type II CR was not "pure" but contaminated by type I CR,
as happened, for instance, when the type II CR was transformed from the
type I CR, salivation occurred independently of, and prior to, the motor
response (cf. K o n o r s k i and W y r w i c k a 1950, S o ł t y s i k,
unpublished experiments). On the other hand, very rarely was the oppo-
site true, namely the performed movement was not followed by saliva-
tion; we have, however, some evidence to believe that this happened either
in neurotic states, or after some cortical lesions.

Since in the well established MCR the first movements in the given

trial were never reinforced by food, it was expected that they would not be followed by salivation which would appear only later when the moment of reinforcement was imminent. Therefore, it was thought that the MCR should be a good physiological model of the case of discrepancy between the motor and salivary response, the latter appearing later than the former one.

However, what really happened was quite opposite. It has been found that the salivary response was amazingly parallel to the motor one, so much so that each movement was accompanied by a definite and nearly stable "quantum" of saliva. During the latent period salivation was practically nil, and it started immediately after the performance of the first movement. The increase of salivation ran pari passu with the increase in the rate of movements, and when, sometimes, the motor performance was interrupted (which happened with long series of movements), salivation was more or less abruptly discontinued.

Although this fact is in close agreement with the original K o n o r- s k i and M i l l e r idea, its explanation does not seem to be simple. After all, most of the movements performed in MCR are n o t reinforced by food, and it is quite probable that the animal did learn that at least the first few movements will not be followed by food. In addition, what the animal learnt, too, in his early training with MCR was that the performance of a movement is not necessarily a signal of food, but may be also a signal of the next movement. So, the question is open as to why the animal starts to salivate exactly at the moment of the performance of the first movement.

It seems that the answer to this question is again closely connected with the above discussed fact of prolonged latencies of the MCR. We have emphasized that when a series of movements required for reinforcement is long enough, the animal simply develops a delayed CR, the first phase of the operation of the CS being inhibitory. In consequence, when the dog already starts the instrumental response, he does so after the inhibitory period is over, and he now expects food after not very many, but rather a few movements. In other words, the animal does not learn to develop a classical CR (i. e. expectation of food) after a given (approximately) number of movements, but rather after a given lapse of time. This result is in a striking agreement with an earlier finding of Ł a w i c- k a confirmed recently by S z w e j k o w s k a (unpublished experiments) that in dogs go-no go alternation is also based exclusively on the principle of time, namely the animal learns to react positively not to every second (or even every third of forth) presentation of the CS, but simply to the CS presented after a definite lapse of time from the last reinforcement.

To sum up, we see that when only a few movements are required for obtaining food, the animal starts his motor performance immediately, since then the CS-US interval is relatively short. On the other hand, when the number of movements required is increased so that the food reinforcement is delayed, the change of animal's behaviour consists only in inhibition of delay causing the prolongation of the latency of instrumental response.

SUMMARY

1. It is possible to train the multiple conditioned reflex (MCR) in the dog by multipling single performance of CR type II.

2. The growth of MCR performance is followed by a depression phase.

3. The limit of the growth of MCR performance is individually specific.

4. MCR habit is very resistant while submitted to the acute extinction procedure.

5. The MCR performance is closely correlated with the salivary outlow.

REFERENCES

FERSTER C. B.. and SKINNER B. F. 1957 — Schedules of reinforcement. Appleton--Century-Crofts. Inc. New York. Ed.: R. M. Elliot, pp. 741.

KONORSKI J. and MILLER S. 1933 — Foundations of the physiological theory of acquired movements. (Polish text with a French summary) Książnica Atlas, Warsaw.

KONORSKI J. and MILLER S. 1936 — Conditioned reflexes of the motor analyzer. (in Russian). Trudy Fiziol. Lab. Pavlova, 6, 1.

KONORSKI J. and WYRWICKA W. 1950 — Researches into conditioned reflexes of the second type. I. Transformation of conditioned reflexes of the first type into conditioned reflexes of the second type. Acta Biol. Exper. (Warsaw), 15, 193.

KONORSKI J. 1962 — Changing concepts concerning physiological mechanisms of animal motor behaviour. Brain, 85, 277.

KOZAK W. 1950 — Method of graphic registration of salivary secretion. Acta Biol. Exper. (Warsaw), 15, 185.

PAVLOV I. P. 1894—1895 — On the Dr. Glinski's experiment on the function of the salivary glands. Trudy Obcestva russkich vracej v S-Petersburge p. 340 (in Russian).

SOŁTYSIK S. and ZBROŻYNA A. 1957 — The chronic fistula of shortened Stensen's duct in dog. Acta Biol. Exper. (Warsaw), 17 399.

WYRWICKA W. 1952 — Studies on motor conditioned reflexes. Acta Biol. Exper. (Warsaw), 16, 131.

Book Review

SENSORY COMMUNICATION. *Contributions to the Symposium on Principles of Sensory Communication.* July 19 — August 1, 1959, Endicott House, M.I.T. Book, Ed.: Walter A. ROSENBLITH. Published jointly by the M.I.T. Press, Massachusetts Institute of Technology and John Wiley & Sons, Inc. New York-London, 1961, Pp. XIV + 834.

If we make a survey of major developments in contemporary science in the course of the last few decades, there is no doubt that an important place among them is occupied by a domain which may be briefly called "Sensory Communication". Perhaps the origin of this development starts with A d r i a n's discovery of the "code" by which the sensory input is transmitted to the central nervous system. It has been found that stimulation of a receptor is transformed into „trains of brief pulses separated from each other by a refractory phase", in other words "a series of "clics" is in fact all that our brains ever receive — at any rate from nerves" as R u s h t o n concisely put it in his illuminating article in the reviewed volume. In this way a new form of "communication" has been discovered, concerning specifically the living organisms endowed with the nervous system, and what now we are concerned with is to understand better and better this form of communication and to see how the animals make use of it. Due to the developments of new techniques, especially utilizing microelectrodes, our knowledge in this field is growing now very rapidly, and it seems that at present we have some insight into the general principles of this peculiar "language" by means of which the animals decipher what is going on in their surroundings.

The present volume fulfills an important role of supplying much information concerning this fascinating domain. A number of outstanding authors working in this field have presented their recent results and their ideas in respect to the rules which this communication system obeys. In consequence, we have now an important and nearly exhaustive source of information which will be very useful not only for the research workers concerned with these problems but also for psychologists and naturalists.

The volume begins with a few papers dealing with sensory communication on the so called psychophysical level, the line of research which started more than hundred years ago. It is amazing how many new and interesting "molar properties" of sensory communication can be still discovered by utilizing these relatively simple methods of investigation, to quote the study by C h e r r y on binaural fusion, or in suppression of pain by sound dealt with in L i c k l i d e r's paper.

However, the chief bulk of papers is devoted to the analysis of trains of impulses generated by the stimulated receptors and transmitted to the higher parts of neural axis. Here we have several contributions on stimulation of gustatory receptors, and transmission of their messages to the brain (B e i d l e r, Z o t t e r-m a n, P f a f f m a n n and others), on the somatic afferent system (M o u n t-c a s t l e, L a n d g r e n, B u s e r and I m b e r t, W a l l, R o s n e r), on auditory system (D a v i s, W o o l s e y, N e f f, R o e d e r and T r e a t, K a t s u k i) and vision (R a t l i f f, G o l d s m i t h, A r d e n and S ö d e r b e r g, J u n g, L e t-

t v i n and others). There are also several articles dealing with some theoretical aspects of sensory communication and the volume ends with the general comments by the editor, Dr. W. A. R o s e n b l i t h.

The reviewer is, of course, tempted to draw some conclusions which emerge from the large body of evidence concerning the coding of sensory input as represented in this volume. If one asks the question how much faithfully the nervous system of animals (man included) "reflects" the external world, in other words, how exact is the description of the environmental events in terms of nervous processes, how literal is the translation from the physico-chemical language of external stimulation into the Morse-like coding performed by the nervous system, we see that the exactitude of this translation, or description, is very far from being perfect. This is not only because of the natural limitations of the system which is, for instance, not able to receive ultraviolet waves of light or ultrasonic waves of sound, or to follow flicker beyond certain frequency. This is, of course, an inherent imperfection of the system, the imperfection which is perhaps pertaining to all recording systems, both already invented, and to be invented in future.

But not this imperfection of the system have we in mind. What is true about the nervous system is its, so to speak, "d e l i b e r a t e" inaccuracy which is clearly seen in all studies concerning the nervous coding beginning from the very first A d r i a n's papers. The picture of the external world transmitted by afferents to the brain is quite specifically distorted both in space and in time, being thus quite different from photography or tape recording. One may say that the picture is not impartial which shows, by the way, that partiality is inherent for all living organisms.

These distortions concern a number of features. First, there is exaggeration and exacerbation of contours which makes the separation of the object from the background more pronounced than it is in reality. By the way, the same trick is generally used in theatre (make-up of actors) and in many paintings. The same trick is abundantly used in vision by *Limulus poliphemus* and other lower animals.

Another trick permanently used in the nervous system is its "loosing interest" in the unchanging continuous stimulation. On the other hand, each change both in visual field, in auditory sequences and in tactile stimulation is immediately grasped and largely amplified. In consequence, the animals see the objects only when they move, and since we are often interested in immovable objects, too, we manage to perceive them by moving our eyes.

Of course, the biological reasons of such arrangements are quite clear. The chief aim of sensory communication is not "objectivity", but u t i l i t y for survival. Therefore, the organism ruthlessly rejects all those forms of information which in the long run are not needed for this aim and much exaggerates in paying attention to those which are useful.

Last remark for very absent-minded reader: if so, why is it that both photography and tape recording imitate the "true" picture of the external world, although there is no make-up in them, and they are faithfully objective. The answer, of course, is that it is because when seeing a photo or listening to the tape record we distort the information transmitted by them exactly in the same way as we do in relation to the "real" external world.

To return to the reviewed volume it seems that it would be even more useful and readable if the papers were divided into several parts, dealing each with clearly separate problem.

<div align="right">*J. Konorski*</div>

Cena zł 20.—

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