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THE EFFERENT CONNECTIONS OF THE BASAL AND LATERAL NUCLEI OF THE AMYGDALA IN THE CAT

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TABLE OF CONTENTS

CHAPTER I

INTRODUCTION

Review of the Literature

The earliest investigations of the amygdaloid complex dealt necessarily with its morphology and phylogenetic development. Among the first to note its existence were C. L. Herrick (1891, 1893; occipito-basal lobe) and Kölliker (1896; nucleus amygdalae). However, Völsch's report (1906, 1910) on the amygdala in mammals, was the first really comprehensive study, and in spite of the fact that the terminology he established is applied only occasionally today, his description of the nuclear subdivisions and their fibre connections formed the basis on which other workers were very soon to build.

Crosby (1917) recognized that the nucleus of the lateral olfactory tract and the ventromedial nucleus and lateral part of the dorso-lateral area of the basal nuclei of the lateral wall of the hemisphere in the brain of alligator were comparable with the amygdala in higher forms. C. J. Herrick (1921) described the amygdala in the anura and urodela. In 1923, Johnston presented his theory concerning its phylogenetic development.

Drawing on the work of other authors, but using principally his own great volume of material, Johnston compared the amygdalae of reptiles, marsupials and mammals. On the basis of his observations he divided its nuclear subdivisions into a more primitive group consisting of the central, medial, cortical and nucleus of the lateral olfactory tract, and a younger group consisting of the basal and lateral nuclei. He stated that the central and medial nuclei were continuous with the phylogenetically old bed nucleus of the stria terminalis and thus constituted a part of the primitive olfactory area. The basal and the lateral nuclei, on the other hand, developed as a migration and proliferation of cells from the medial surface of the amygdaloid fissure; that is, from the phylogenetically younger pyriform lobe. Johnston observed that only the basal nucleus could be seen in the turtle, the lateral nucleus first making its appearance in marsupials. He considered the cortical nucleus, although it arose from the same area, a relatively primitive structure that had simply been pushed "inward and medialward" as the basal and lateral nuclei developed. Humphrey (1936) introduced the term corticomedial complex for the older nuclei and basolateral complex for the younger.

In his morphological study on the cat brain, Fox (1940) found it more convenient to divide the nuclei into three groups: a lateral, consisting of the basal and lateral, a corticomedial consisting of the cortical, central and medial, and an anterior consisting of the intercalated mass, the nucleus of the lateral olfactory tract and the anterior amygdaloid area.

Crosby and Humphrey (1941) however considered it advisable to divide the amygdala into four groups: 1) the basolateral complex, in which they included the intercalated mass; 2) the corticomedial complex, in which they included the nucleus of the lateral olfactory tract; 3) the anterior amygdaloid area; and 4) the cortico-amygdaloid transition area. It is this terminology that is generally employed today.

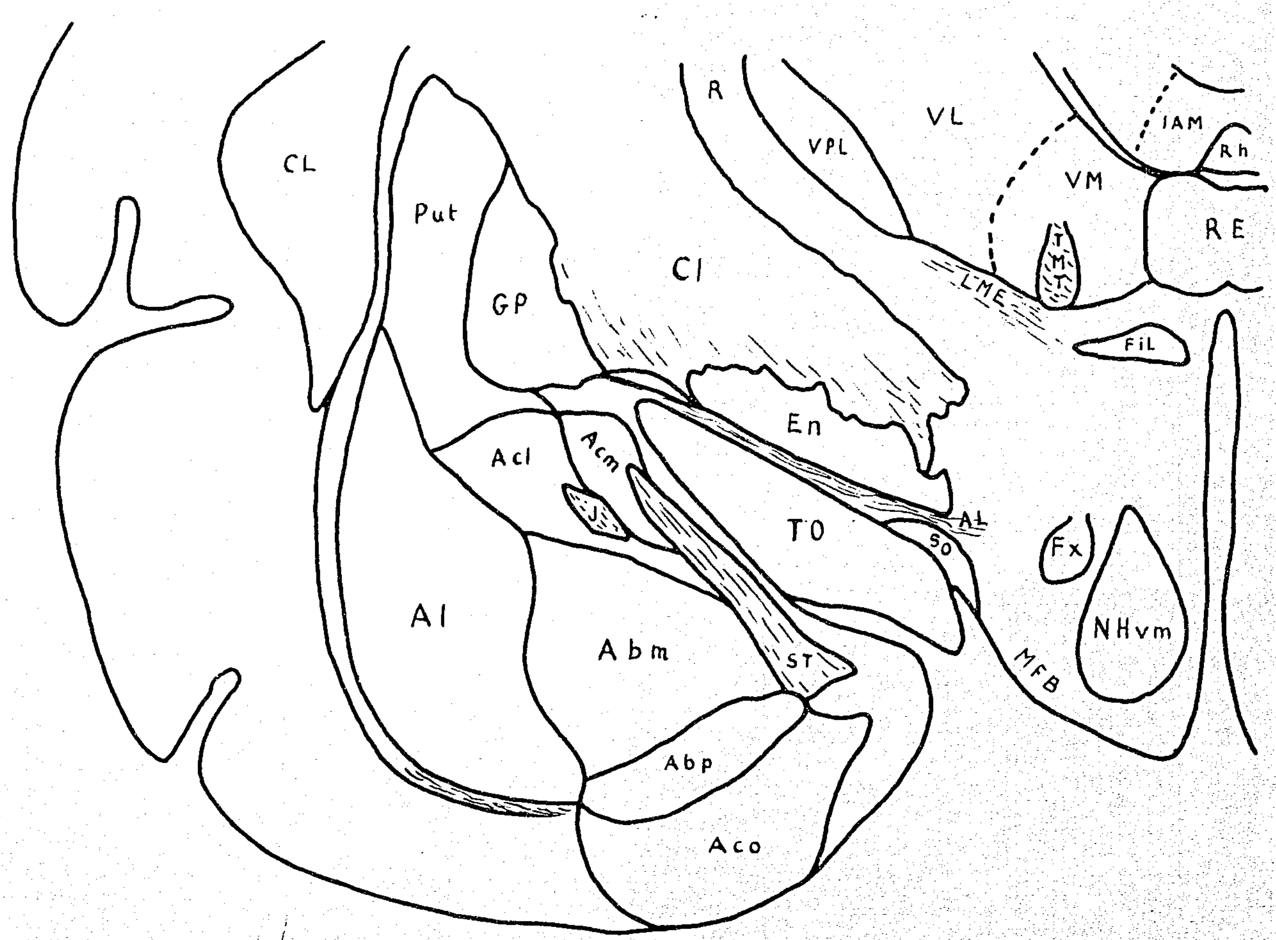
Following Johnston's work, data concerning the nuclear configuration of the amygdaloid complex accumulated steadily. Berkelbach van der Sprenkel (1926) and Loo (1931) confirmed Johnston's description of the opossum brain. Young (1936), Humphrey (1936) and Lauer (1945) did the same in rabbit, bat, and monkey. Gurdjian (1928) gave a preliminary report on the amygdala in the rat, which was followed by Brodal's more complete study in 1947. Investigations were also carried out on the shrew (Crosby and Humphrey, 1944), ant-eater (G. Smith, 1930), panda (Lauer, 1949), man (Crosby and Humphrey, 1941), cat (Fox, 1940), and porpoise (Breathnach and Goldby, 1954).

It has been observed that the nuclear pattern of the amygdala is essentially the same in all the marsupials and mammals that have been studied. Two types of variation, however, have been noted. (Crosby and Humphrey, 1944). First, there are differences in the size and cellular differentiation of the individual nuclei, and, second, there are variations in their position. The latter is not a change in the position of one nucleus relative to another, but is an inward rotation of the entire amygdala, caused by the increased development of the temporal lobe in higher forms.

Since the cat was the animal used in the series of experiments to be presented below, a brief summary of Fox's description of the amygdala in this animal will be given.

The amygdaloid complex is located in the medial part of the pyriform lobe and extends from the nucleus of the diagonal band of Broca to the hippocampus. The position of the central, cortical and medial nuclei is illustrated in Fig.1. The anterior amygdaloid area is a "transitional region" of diffusely arranged cells that is continuous anteriorly with the tuberculum olfactorum and posteriorly with the basal, central and medial nuclei. The nucleus of the lateral olfactory tract is found in the antero-medial part of this area. Several tiny groups of small neurons are interposed between the amygdaloid nuclei. These are the intercalated masses.

Fig.1. The amygdala of the normal cat.
(Modified after Jasper & Ajmone-Marsan: for abbreviations see chapt. II)



The lateral nucleus which is composed of moderately large cells, is the largest and longest in the amygdaloid complex. In its central region it is bounded laterally and ventrally by the external capsule, medially by the basal nucleus, and superiorly by the putamen and central nucleus. Anteriorly and, with the exception of its dorsal surface, posteriorly, it is surrounded by the external capsule. Posteriorly the dorsal surface forms part of the ventricular floor. The basal nucleus can be divided into a large-celled lateral and a smaller-celled medial part. In higher forms an accessory basal nucleus is also described (Lauer, 1945; Crosby and Humphrey, 1941). Laterally it is bordered by the lateral nucleus, ventrally by the cortical nucleus, medially by the medial nucleus, and superiorly by the central nucleus.

It should be noted here that the stria terminalis has been recognized for many years as one of the main pathways of the amygdaloid complex. Johnston (1923) observed that in the opossum it has five components: 1) supracommissural, 2) infracommissural, 3) hypothalamic, 4) commissural and 5) stria medullaris.

The studies of normal material, from a variety of species by the authors mentioned above, have suggested three main efferent pathways for the basolateral complex; the stria terminalis, the posterior (temporal) limb of the anterior commissure, and the longitudinal association bundle of Johnston. While there is general agreement that the stria terminalis is the main pathway of the corticomedial complex and that the basal nucleus contributes fibres to the supra- and infracommissural bundles there is a division of opinion concerning contributions from the lateral nucleus. Johnston (1923) stated that the lateral nucleus of the opossum did not contribute to the stria. Fox (1940) expressed the same opinion concerning the cat. Young (1936) and O. Smith (1930) were uncertain about this in the rabbit and ant-eater. On the other hand Humphrey (1936) and Lauer (1945) observed a few fibres from this nucleus joining the stria terminalis in the bat and the monkey.

There has been general agreement that the lateral nucleus sends fibres to the temporal limb of the anterior commissure. However, not all the fibres are described as being commissural in nature. Some end in the nucleus of the anterior commissure and the preoptic region.

Very few authors mention the longitudinal association bundle of Johnston, (Johnston referred to it as the amygdalo-pyriform association bundle) and although it is called an association bundle, it should be pointed out that Johnston recognized that it was composed of both an association bundle and efferent pathway. The former interconnected the amygdaloid nuclei and the cortex of the pyriform lobe. The latter passed inferior to the internal capsule at the level of the anterior commissure and was lost in the region of the diagonal band of Broca and the medial forebrain bundle. Johnston considered the longitudinal association bundle the main pathway of the basal and lateral nuclei.

Although most authors do not refer to the longitudinal association bundle, and Lauer (1945) only notes its existence, Fox (1940) gives a fairly detailed description of it in the cat. According to the author, it contains fibres from the pyriform cortex and lateral and basal nuclei. It is a distinct bundle lying dorsal to the border between these two nuclei, separating them in this region from the central nucleus. It begins to form at the level where the stria terminalis leaves the amygdala, and it continues anteriorly to the anterior limit of the basal nucleus. Here it bends dorsomedially and as it passes through the medial nucleus it divides into a large posterior bundle and a smaller anterior bundle. The former passes beneath the internal capsule and can be followed to the preoptic area. The latter continues anteriorly and ventrally through the anterior amygdaloid area, then curves medially, passes beneath the anterior commissure, and joins the medial forebrain bundle. Fox suggests that the longitudinal association bundle is, at least in part, comparable with Crosby's (1917) and Loo's (1931) ventral olfactory projection tract (alligator and opossum), and Gurdjian's (1927) lateral cortico-hypothalamic tract (rat).

Of special importance is Fox's reference to "a surprising number of diffuse fibres which cannot be allocated to either the stria terminalis or the longitudinal association bundle" (p.56). He states that some of these, from the lateral, basal and central nucleus, form a distinct bundle ("c" bundle) that can be followed medially to the region of the entopeduncular nucleus.

Following his study of normal material, Fox (1943) became the first to apply experimental anatomical procedures to the amygdala. First, he determined by Marchi studies that the stria terminalis was a completely efferent pathway for the amygdaloid region. Thus he confirmed the work of Gurdjian (1928), who reported that in Golgi-Cox stained material axons of amygdaloid neurons could be seen entering the stria terminalis. However, Fox was unable to confirm his own earlier observations (1940) regarding the pathways of the lateral nucleus. Lesions involving only this nucleus failed to produce degeneration in either the stria terminalis or the longitudinal association bundle while those involving both the lateral and basal nuclei produced degeneration in both. He also modified his description of the longitudinal association bundle. Instead of a posterior and an anterior component, he found a smaller lateral component that disappeared at the anterior limit of the basal nucleus, and a larger medial component that curved medially, passed ventral to the internal capsule and continued dorsally to reach the bed nucleus of the stria terminalis. He also carried out experiments (1943) with the method of retrograde degeneration but since they were not successful there is no advantage in reporting and commenting on them in this introduction.

Fox's Marchi studies of the anterior commissure in the cat (Fox and Schmitz, 1943) and monkey (Fox, Fisher and Desalva, 1948) did not demonstrate any interamygdaloid fibres in the posterior limb. Brodal (1948) however, employing retrograde degeneration methods after severing the anterior commissure of the rat, observed that while the lateral and large-celled part of the basal nucleus do not send fibres into the anterior commissure, the small-celled part of the basal nucleus does send a few.

The results obtained by Omukai (1958) in his Marchi study of the rabbit agree in part with Fox's Marchi study and in part with his description of normal material. The former found that the basal nucleus contributes fibres to the supracommissural and preoptic (hypothalamic) components of the stria terminalis, and that the lateral nucleus contributes none (as Fox, 1943). He also observed that the longitudinal association bundle contained fibres from both the lateral and the basal nucleus (as Fox, 1940). Moreover Omukai described fibres from both nuclei reaching the anterior commissure by way of the external capsule.

In their investigation of the temporal lobe of the monkey, using the Glees stain, Adey and Meyer (1952) discuss the efferent pathways of the amygdala. Although their lesions were not restricted to this complex only, they observed that the supracommissural and preoptic components of the stria terminalis were more closely associated with the basolateral complex than with the corticomedial, and added that while the former component could be traced only to the region of the anterior commissure, terminal degeneration of the latter could be identified in the ventromedial and periventricular hypothalamic nuclei. Degeneration found in the same region of the contralateral hypothalamus was thought to reach this site via the anterior commissure.

A further communication by Adey et al (1958) on one monkey brain stained by both the Glees and Nauta-Gygax techniques confirmed the results of Adey and Meyer (1952). In addition, the sections obtained with the Nauta-Gygax method demonstrated degeneration in the anterior commissure that could be followed to the contralateral limit of the anterior commissure. Some of these fibres entered the base of the septum bilaterally. Degeneration was also noted in the fornix bilaterally and in the thalamic nuclei. The latter finding adds support to Fox's (1949) description of degenerated fibres from the monkey amygdala entering the ventral thalamic peduncle and coursing to the nucleus medialis dorsalis of the thalamus.

Recently, Nauta has applied his staining technique to the investigation of the monkey amygdala, (1958, 1960). He also describes fibres coursing into the ventral thalamic peduncle. Some of these terminate in the paramedian thalamic nuclei, but most of them continue caudally in the internal medullary lamina to end in the medial, magnocellular part of the dorsomedial thalamic nucleus. Still other fibres maintain a ventral position and spread medially into the substantia innominata, the lateral preoptic region, the nucleus of the diagonal band of Broca, and anteriorly into the olfactory tubercle. Some enter the basal septal area. It should be pointed out here to avoid any confusion, that in all the studies mentioned above, in which amygdaloid lesions have been performed, the lesion has not been confined to the basolateral complex, except in the study of Fox (1943).

The significance of morphological and experimental anatomical studies, as discussed above, lies in the clarification of the pathways that may be involved in the development of certain somatic, autonomic or emotional effects, not in the determination of function. The stimulus that prompted anatomists and physiologists to begin an investigation into the function of the amygdala was the report of Klüver and Bucy (1937, 1939) on monkeys which had undergone bilateral temporal lobectomy. In these animals they observed a syndrome consisting of "psychic blindness", loss of anger or fear reactions, marked oral tendencies, a tendency to attend and react to all visual stimuli, and an increase in sexual behaviour. As this syndrome did not occur after removal of the first or second and third temporal convolutions only, it was thought that it might have developed because of removal of the amygdala. Weiskrantz (1956) observed the syndrome in monkeys with bilateral amygdalectomies. Rosvold, Mirsky and Pribram (1954) found a decreased aggressiveness in monkeys which shared a cage with others. If they were isolated, they showed an increased aggressiveness. Jameson, Settlage and Bogumill (1957), while they were able to reproduce the "Klüver-Bucy" syndrome, correlated its development with the onset of secondary radiation necrosis beyond the amygdaloid complex. They used Cobalt 60 to make their lesions.

The initial experiments in the cat resulted in a rage reaction rather than increased docility (Spiegel et al, 1940; Bard and Mountcastle, 1948). However, Spiegel et al (1940) were unable to produce the rage reaction by bilateral section of the stria terminalis.

Schreiner and Kling duplicated the "Klüver-Bucy" syndrome in the cat in 1953. Psycho-physiological studies of these animals (Brady et al, 1954) ascribed the decrease in anxiety and fear to a decrease in the learning of an avoidance response. Anand and Brobeck (1952) and Martin et al (1958) did not observe a rage reaction in rats or cats, or dogs and cats respectively. Kling and Hutt (1958) after producing the syndrome in a series of cats, destroyed the ventromedial nucleus of the hypothalamus in these animals bilaterally. Immediately they became wild and vicious.

Green, Clemente, and de Groot (1957) noted rage reactions only in those cats in which the lesions extended beyond the amygdala into the hippocampus. When one considers these last two investigations together, the possibility that the hippocampus sends inhibitory impulses to the ventromedial nucleus is not at all remote.

In assessing these recent experiments, one feels satisfied that the amygdala is concerned with emotional expression, but it must be pointed out that the lesions in every case have extended to a greater or lesser degree into surrounding structures. King (1958) did not observe emotional alterations in rats which had bilateral lesions confined to the basolateral complex.

The amygdala may also influence endocrine function. Koikegami et al (1955, 1958) noted that bilateral amygdalectomy caused retardation of growth in pups and infant rats. Martin et al (1958) and Endröczi et al (1959) found that both ablation and stimulation of the amygdala in dogs and cats caused a rise in the adrenocorticoid hormone level of the adrenal venous blood.

The reports that stimulation of the amygdala in the waking animal produces fear and rage seems to be in agreement with the communications on the tameness and docility of amygdalectomized animals. A number of physiologists feel that this result is produced mainly by stimulation of the phylogenetically older nuclei (MacLean and Delgado, 1953; Magnus and Lammers, 1956; Fernandez de Molina and Hunsperger, 1959).

Kaada et al (1954) observed this behaviour during stimulation of the newer basolateral complex but Fernandez de Molina and Hunsperger (1959) could not produce affective behaviour by stimulating the longitudinal association bundle of Johnston.

It is interesting to note that Chapman et al (1954) and Heath (1955) observed fear, anxiety and rage on stimulating the amygdala in humans, (5 epileptics and 1 schizophrenic). This lends support to the conclusion of Crosby and Humphrey (1944) that the constancy of pattern in the amygdala of mammalian forms "permits the application of results obtained through experimental studies to the understanding and solution of clinical cases" (p.296).

Electrical stimulation of the amygdala in both unanaesthetized and anaesthetized animals has produced a variety of somatic and autonomic effects. Movement of the face and jaw and chewing have been attributed in some studies to the stimulation of the basolateral complex (Baldwin et al, 1954, 1956; Wood et al, 1958) while other authors attribute it to stimulation of the phylogenetic older nuclei and the basal nucleus (Kaada et al, 1954; Magnus and Lammers, 1956).

Koikegami and his co-workers have studied the autonomic effects for a number of years. They have found alteration in body temperature (1952a), blood pressure (1953, 1957), amplitude and rate of respiration (1952b), gastro-intestinal motility (1952a, 1953), Uterine (1954) and bladder contractions and in urine flow (1957). They have also noted ovulation (1954). Shealy and Peele (1957) have confirmed the effect on gastric motility and ovulation, Andy et al (1959), the effect on blood pressure.

While localization of these autonomic effects in a particular nucleus does not appear acceptable (Shealy and Peele, 1957), the work of Koikegami et al indicates that they are elicited most frequently by stimulation of the central, medial, cortical and small-celled part of the basal nucleus.

Problem Formulation

The review of the literature indicates that a thorough investigation of the connections of the amygdala, particularly in view of the recent functional data, is necessary. It is especially important to determine which structures are directly concerned in the emotional and autonomic reactions initiated in this complex; that is, to determine the site of termination of the efferent pathways.

In the experimental anatomical studies that have been carried out to date, little attempt has been made to localize lesions within the individual nuclei of the amygdala. Thus it is not known which nuclei share a common pathway and site of termination. Until this has been determined, there can be no anatomical basis to explain any overlap or isolation of effect observed in physiological studies.

In the review of the literature the conflicting results obtained by various authors employing experimental anatomical techniques have been noted. These may be explained by species variations and by differences in the size and position of the lesions. Probably the most important explanation, however, is the difference in the staining techniques that were employed.

The Marchi technique employed by Fox (1943) and Umukai (1958) stains degenerated myelin selectively (Mettler, 1932). Thus it is useful only for the identification of myelinated fibres of passage. It does not stain unmyelinated fibres or terminal arborizations. The Glees method does stain the latter and is especially useful in identifying sites of termination (Glees and Nauta, 1955; Evans and Hamlyn, 1956). However, it is not selective. Thus if the terminals are sparse and scattered, it is difficult to identify them among the normal fibres. The Nauta technique combines the advantages of the Marchi and the Glees methods. It is relatively selective for degeneration and stains unmyelinated fibres and preterminals.

It is felt that an investigation of the individual nuclei of the basolateral complex is required. At present, the Nauta-Gygax technique is the method of choice for such a study.

CHAPTER II

MATERIAL AND METHODS

Forty-three adult cats ranging in weight from 5.0 to 10.0 lbs. were used for this series of experiments. In 27, an attempt was made to place a lesion in the lateral nucleus, in 16 in the basal nucleus. All lesions were made in the right amygdala. In each case, the following surgical procedure was performed under antiseptic conditions.

The cat was anaesthetized with an intraperitoneal injection of 0.8 cc of a 5% nembutal solution per kilo body weight. The head was secured in the stereotaxic apparatus (Lab-tronics) and the shaved scalp painted with iodine. By incising and retracting the skin and galea aponeurotica, the sagittal suture was exposed. Next, the right temporalis muscle was reflected from its origin in the temporal fossa. The electrode was then moved to within a few millimetres of the parietal bone and the required site of entry marked. An opening approximately 4.0 mm in diameter was made with a dental drill. After trimming the margin with bone forceps the dura was opened. A monopolar electrode was then inserted to the desired position and coagulation was carried out with a Blendtome electro-surgical unit. Following the removal of the electrode the operative site was rinsed with warm physiological saline and the bony defect filled with Gel-foam. The galea aponeurotica was closed with interrupted black silk sutures after the temporalis muscle had been pressed gently against the temporal fossa. The skin was closed with metal clips and the incision then painted with iodine and covered with a thin coating of collodion.

The post-operative course was, in most cases, uneventful. Occasionally, on the first day the cat had to be fed milk by dropper. Slight transient oedema of the conjunctiva was occasionally encountered.

The electrodes were made by the author. Silver or platinum wire 0.01 inches in diameter was used. Neither was found to have an advantage over the other with regard to the size of the lesion produced, and although the silver was easier to handle, the platinum was more durable. The wire was insulated with approximately 10 cm of straight capillary glass tubing. A tip of 1.0 mm to 2.0 mm was left exposed, the length of exposure being governed by the site chosen for the lesion. The glass was fused to the metal at this point. The outside diameter of the electrodes did not exceed 0.35 mm.

The atlas of Jasper and Ajmone-Marsan (1954) was used to determine the desired coordinates. In addition, sagittal and horizontal atlases were made from it by plotting the points at which the outlines of the various nuclei crossed the reference lines. These were found helpful not only in interpreting the slides but also in visualizing a proposed course for an electrode.

Three methods of inserting the electrode were employed: 1) vertical (7 cats); 2) at an angle of 60° to 75° with the horizontal of the frontal plane (20 cats); 3) at an angle of 55° with the horizontal of an oblique plane (16 cats).

The limitations of the apparatus made a special procedure necessary to determine the settings for the electrode holder when using method 2. This procedure is illustrated with an example picture in Fig.2.

Suppose the electrode is to be inserted on a 60° angle so that its tip is at a point 10 mm from the midline and 4 mm above the horizontal zero line of the apparatus (i.e.: 6 mm below the horizontal zero line of the atlas) in the frontal 10.0 plane.

1. Adjust the electrode holder in frontal plane zero so that its tip is at point A (Fig.2).
2. Move the electrode holder 10 mm laterally to point B.
3. With reference to Triangle DBC retract the electrode the distance of 4 cosecant 60° to point C.
4. Move the holder horizontally a distance of 4 cotangent 60° to point D.
5. Move the electrode holder anteriorly to frontal plane 10.0.
6. Record the readings on all parts of the electrode holder and begin the experiment.

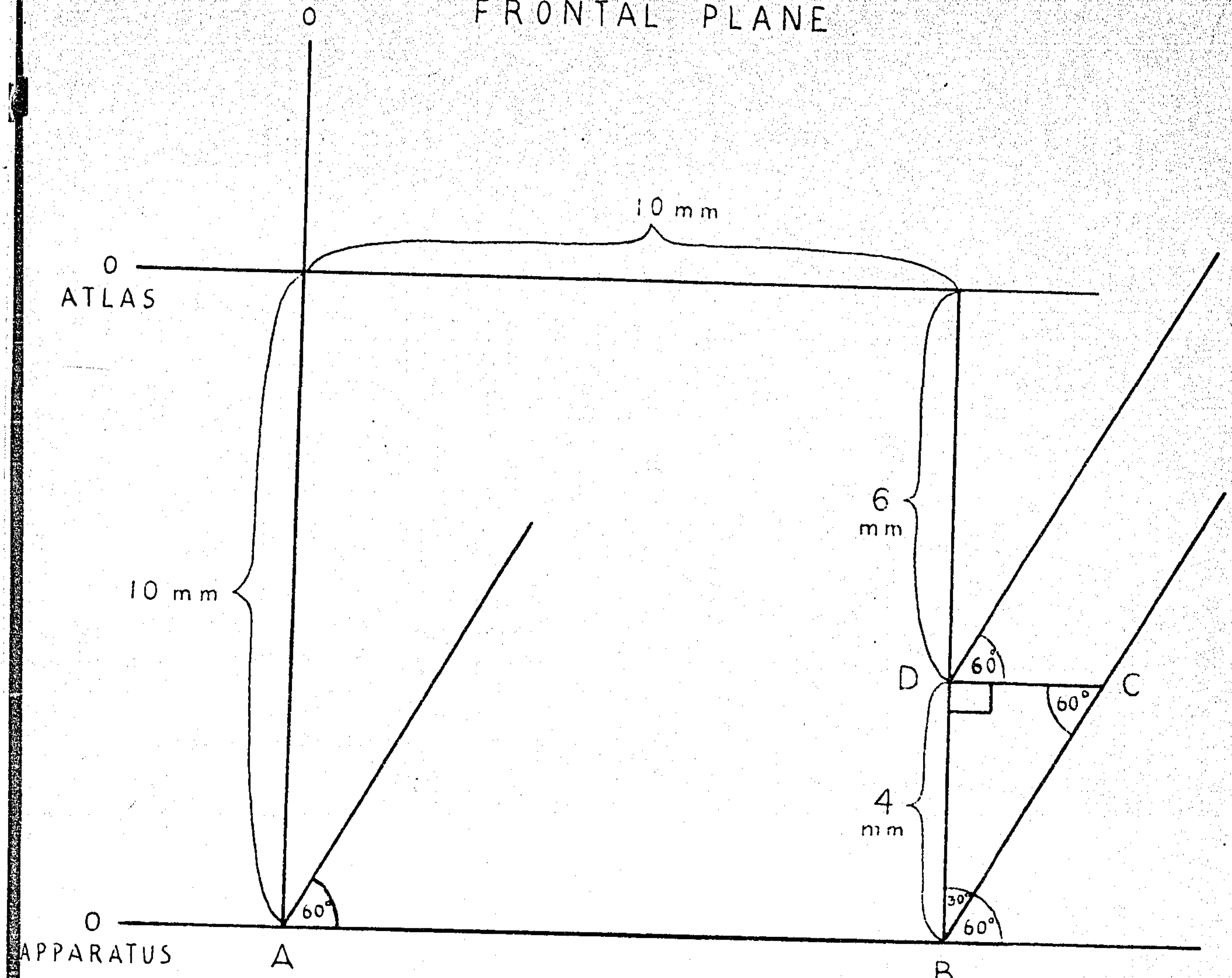

O
FRONTAL PLANE
10 mm
O
ATLAS
10 mm
6
mm
D
60°
C
60°
4
mm
30°
60°
60°
O
APPARATUS
A
B

Fig. 2

To determine the settings for the electrode in method 3, the following procedure was carried out.

Suppose the electrode is to be inserted on a 55° angle to the horizontal plane and on a 45° angle to the frontal plane so that its tip is at frontal 10.0, 10 mm lateral to the midline and 4 mm above the horizontal zero line of the apparatus.

1. Adjust the electrode holder to the required angles (Fig.3b and 3c).
2. Centre the tip of the electrode at point O of Fig.3a.
3. Move the holder 10 mm laterally by moving Bar 2 $\frac{10}{2}$ units to the right and Bar 3 $\frac{10}{2}$ units to the right. The electrode is now a point B. Fig.2a.
4. Move the electrode holder anteriorly 10 mm along Bar 1. The electrode is now at point C.
5. With reference to Triangle BCD, retract the electrode the distance of 4 cosecant 55° to point D.
6. Move the electrode holder horizontally a distance of 4 cotangent 55° to point E.

Record the readings on all parts of the electrode holder and begin the experiment.

In an attempt to produce a somewhat larger lesion, two electrodes were inserted 1.5 mm apart in 4 brains. It was

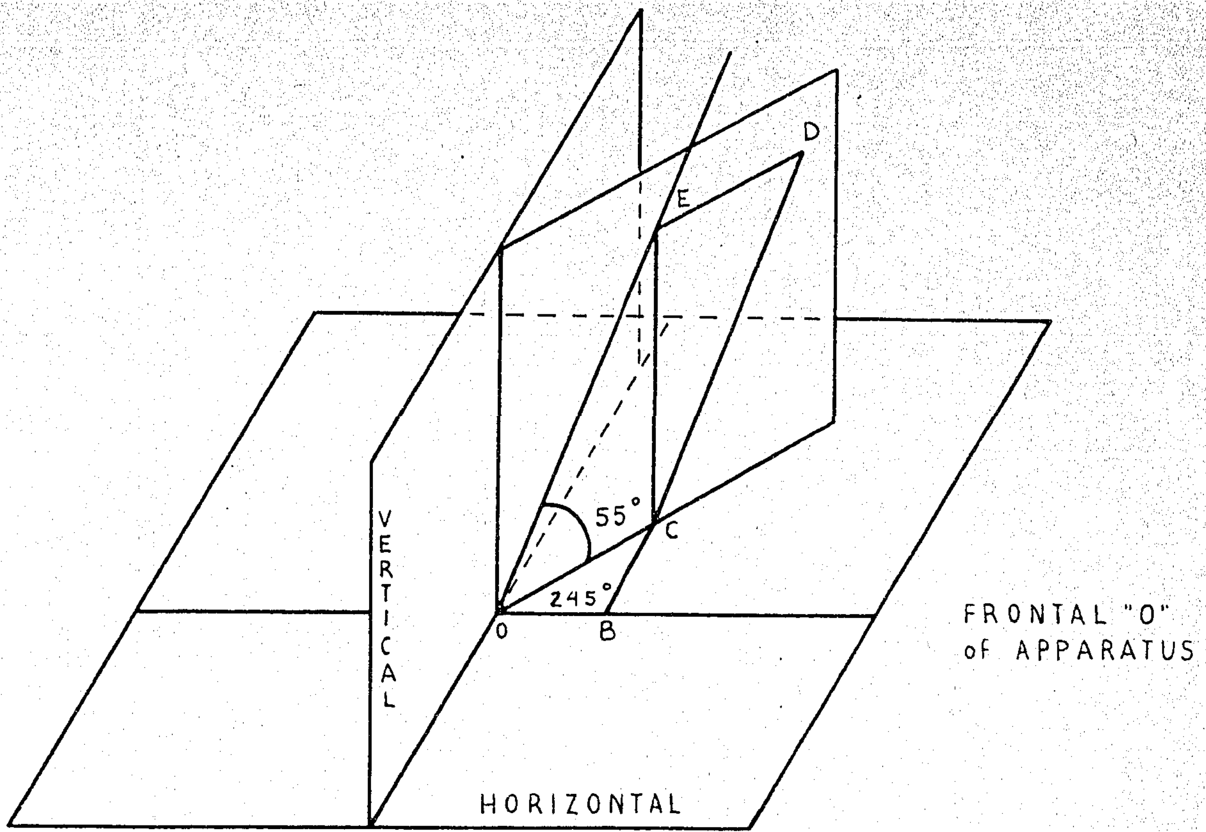


Fig. 3 A

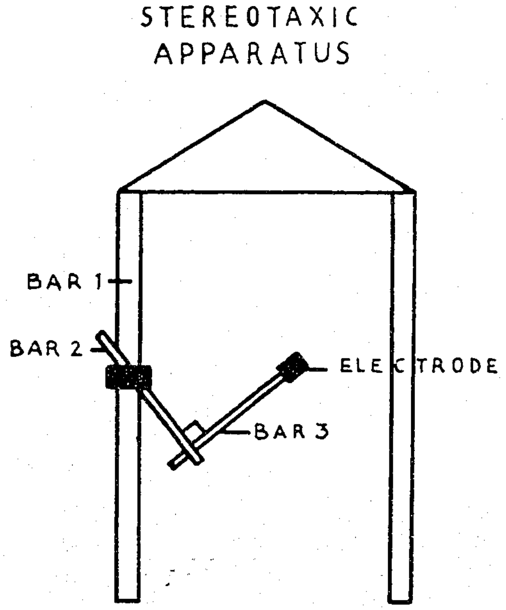


Fig. 3 B

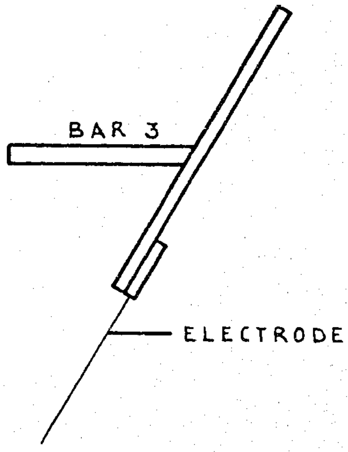


Fig. 3 C

found however that the lesions were a bit too large and the method was abandoned. It should also be mentioned that when this series of experiments was started, discrete lesions in the most anterior, the intermediate and most posterior regions of the lateral and basal nuclei appeared attractive. However, the difficulties involved in such a program were soon appreciated and thereafter the central area of each nucleus was the desired target.

In most cases (see table 1 on next page), the animal was sacrificed on the tenth post-operative day. After anaesthetizing the cat and clipping the fur from the anterior chest wall, the skin was incised and reflected. A window was made quickly in the left side of the thoracic cage to expose the heart. The pericardium was then incised and the right ventricle secured with arterial forceps. After severing the inferior vena cava approximately 400 cc of physiological saline was introduced into the left ventricle through a 19 guage needle. This was followed by 300-400 cc of 10% formalin. The solutions were pushed in manually with a 50 cc syringe. The brain was removed immediately and stored in ten times its own volume of 10% formalin.

After at least eight weeks in formalin, the brain was sectioned with the aid of a freezing microtome. In the first twenty-seven brains the sections were made 40 u in thickness. In the remaining sixteen they were cut at 30 u.

Sections from the region of the amygdala were

SUMMARY OF PROCEDURE FOR EACH EXPERIMENT

TABLE I.

CAT	ELECTRODE			CO-ORDINATES			METHOD	SUBSEQUENT COURSE.			
Nō	Nō	WIRE	TIP. PROJECT	FR.	LAT	INF. TO "0" LINE	OF ELECT. INSERTION.	SACRIF. POST-OP.	PLANE OF SECTION	LESION	STAIN.
1	1	SILVER	1.0. mm.	12.0	11.0.	5.0	I	8 DAYS	FR	S	S
2	1	PLATIGNUM	2.0. "	12.0	11.0	5.0	I	8 "	SAG	S	S
3	1	"	2.0 "	11.0	11.0	6.5	I	8 "	FR	S	U
4	1	SILVER	1.5 "	11.0	11.0	6.5	I	8 "	FR	S	U
5	1	"	1.5 "	12.0.	9.5	6.0	I	8 "	FR	U	
6	2	"	1.5 " 1.5 "	12.0 10.5	11.0 11.0	6.0 6.0	II - 60°	8 "	FR	S	S
7	2	"	1.5 " 1.5 "	12.0 10.5	11.0 11.0	6.0 6.0	II - 60°	8 "	FR	S	S
8	2	"	1.5 " 1.5 "	12.0 10.5	10.5 10.0	6.0 7.0	II - 60°	8 "	FR	S	S
9	2	"	1.5 " 1.5 "	12.0 10.5.	10.5 10.0	6.0 7.0.	II - 60°	8 "		FOR A.R.+Y. TECH.	
10		EXPERIMENTAL	PROCEDURE	UNSATISFACTORY							
11		"	"	"							
12	1	PLATINUM	1.2 mm.	12.0	10.5	6.5	II - 75°	9 "	FR	U	
13	1	"	1.2 "	12.0	10.5	6.5	II - 75°	10 "	FR	U	
14	1	"	1.2 "	12.0	11.0	6.5	II - 65°	10 "	FR	U	
15	1	"	1.2 "	11.0	11.0	7.0	II - 70°	10 "	FR	U	
16	1	"	1.2 "	11.0	10.5	7.0	II - 65°	10 "	FR	U	
17		EXPERIMENTAL	PROCEDURE	UNSATISFACTORY							
18	1	PLATINUM	1.2 mm.	10.5	11.0	6.5	II 60	10 "	FR	U	
19	1	"	1.2 "	10.0	10.0	7.0	II 65	10 "	FR	U	
20	1	"	1.2 "	11.0	10.5	6.5	II 70	10 "	SAG	S	S
21		"	1.2 "	10.5	10.0	7.0	II 65		FR	S	S
22		"	1.2 "	10.5	10.0	7.0	II 65			FOR A.R.+Y. TECH.	

S. - SATISFACTORY

U. - UNSATISFACTORY.

SUMMARY OF PROCEDURE FOR EACH EXPERIMENT

TABLE I. (CONT'D).

CAT	ELECTRODE			CO-ORDINATES			METHOD	SUBSEQUENT COURSE			
Nō	Nō	WIRE	TIP. PROJECT	FR.	LAT.	INF. TO "0" LINE	OF ELECT. INSERTION	SACRIF. POST-OP.	PLANE OF SECTION	LESION	STAIN
23	1	PLATINUM.	1.2 mm.	11.5	10.0	7.0	II - 65°	10 DAYS	SAG	S	S
24	1	"	1.2 "	11.5	10.0	7.0	II - 65°	10 "		FOR A.R.+Y. TECH.	
25	1	"	1.2 "	11.0	10.0	7.0	II - 65°	10 "	SAG	S	S
26	1	"	1.2 "	11.0	10.0	7.0	II - 65°	10 "		FOR A.R.+Y. TECH.	
27	1	"	1.2 "	12.0	10.0	7.0	II - 65°	10 "	SAG		
28	1	"	1.2 "	12.0	10.0	7.0	II - 65°	10 "		FOR A.R.+Y. TECH.	
29	1	"	1.2 "	10.5	8.0	8.0	III - 55° - 45°	10 "	FR	S	S
30	1	"	1.2 "	10.5	8.0	8.0	III - 55° - 45°	10 "	FR	S	U
31	1	"	1.5 "	10.0	9.0	7.0	III - 55° - 45°	10 "	FR	U	
32	1	SILVER	2.0 "	10.5	8.0	8.0	III - 55° - 45°	8 "	FR	S	S
33	1	"	2.0 "	10.5	8.0	8.0	III - 55° - 45°	8 "		U	
34	1	"	2.0 "	10.5	8.0	8.0	III - 55° - 45°	8 "	FR	S	S
35	1	"	2.0 "	10.5	8.0.	8.0	III - 55° - 45°	8 "	FR	U	
36	1	PLATINUM	2.5 "	10.5	8.0	8.0	III - 55° - 45°	10 "	FR	S	S
37	1	"	2.5 "	10.5	8.0	8.0	III - 55° - 45°	10 "	FR	S	S
38	1	"	2.5 "	10.5	8.0	6.0	III - 55° - 45°	9 "	SAG	U	
39	1	"	2.5 "	10.5	8.0	8.0	III - 55° - 45°	DIED 4 DAYS POST-OP.			
40	1	SILVER	2.3 "	12.0	6.5	5.5	III - 55° - 45°	10 DAYS.	SAG	S	S
41	1	"	2.3 "	12.0	6.5	5.5	III - 55° - 45°	10 "		U	
42	1	PLATINUM	2.5 "	12.0	8.0	7.0	I	10 "	FR	S	U
43	1	"	2.5 "	12.0	8.0	7.0	I	10 "	FR	S	S

S. - SATISFACTORY.
U. - UNSATISFACTORY.

stained with cresyl echt violet so that the extent of the lesion could be determined. This method was also employed as required for identification of areas showing preterminals.

Serial sections of each brain were stained by the Nauta-Gygax technique (1954) with the modifications of Chambers et al (1956) and Nauta (1956). Following consultation with Dr. Nauta in May, 1959, a few minor changes in the method were instituted. Although it is still not known what product of degeneration is impregnated with silver by this technique, the basic principles of the method have been described (Nauta and Ryan, 1952).

First the sections are treated with phosphomolybdic acid and potassium permanganate. This prevents the normal fibres from forming insoluble "silver nuclei" when the tissue is transferred to a silver nitrate solution. The degenerated fibres still have this ability however, and submicroscopic particles of metallic silver ("silver nuclei") collect in them. On placing the sections in Laidlaw's ammoniacal silver solution, nacent silver is deposited on the "silver nuclei". Thus, each fragment of a degenerated fibre becomes impregnated and is seen microscopically as a row of black dots. As the method is not completely selective, a few normal fibres, impregnated throughout their length, are also seen.

It will be noted in table 1 that a number of brains were set aside for staining of terminal degeneration with the Armstrong, Richardson and Young technique. Modification of the technique was indicated and this was attempted over a period of about four months. Although some progress was made, it was decided that the modification was really a project in itself and thus it was set aside until a future date.

The diagrams illustrating the lesions and pathways were made by first outlining an enlarged image of the slide. (Hansa photographic enlarger). The nuclear groups were outlined free hand while examining the section microscopically. The abbreviations used are the same as those in the Atlas of Jasper and Ajmone-Marsan. They are as follows:

Aa - Area amygdaloidea anterior.
Ab - N. amygdaloideus basalis.
Abm - N. amygdaloideus basalis (pars magno-cellularis).
Abp - N. amygdaloideus basalis (pars parvi-cellularis).
Ac - N. amygdaloideus centralis
Acl - N. amygdaloideus centralis (pars lateralis).
Acm - N. amygdaloideus centralis (pars medialis).
Aco - N. amygdaloideus corticalis.
AD - N. anterior dorsalis.
Al - N. amygdaloideus lateralis.
AM - N. amygdaloideus medialis.
Av - N. anterior ventralis.
Cd - N. caudatus.
Ch - Chiasma opticum.
CL - N. centralis lateralis.

En - N. entopeduncularis.
Fcd - Fundus caudati.
Fx - Fornix.
GL - Corpus geniculatum laterale.
GLV - Corpus geniculatum laterale (pars ventralis).
GM - Corpus geniculatum mediale.
GP - Globus pallidus.
Hb - Habenula.
HbL - N. habenularis lateralis.
HbM - N. habenularis medialis.
Hipp. - Hippocampus.
Hp - Hypothalamus posterior.
J - Longitudinal association bundle of Johnston.
LD - N. lateralis dorsalis.
LP - N. lateralis posterior.
MD - Medialis dorsalis.
Ml - N. mamillaris lateralis.
Mm - Corpus mamillare.
NCM - N. centralis medialis.
HHVM - N. hypothalami ventromedialis.
PC - N. paracentralis.
Ped - Pedunculus cerebralis.
Pul - Pulvinar.
Put - Putamen.
PVA - N. periventricularis anterior.
PVH - N. periventricularis hypothalami.
R - N. reticularis.

RE - N. reuniens.
Rh - N. Romboidens.
S - Stria medullaris.
Sch - N. suprachiasmaticus.
SM - N. submedius.
SO - N. supraopticus.
ST - Stria terminalis.
STh - N. subthalamicus.
TMT - Tractus mamillo-thalamicus.
TO - Tractus opticus.
VA - N. ventralis anterior.
VPL - N. ventralis postero-lateralis.

Other abbreviations used:

C.E.V.- Cresyl echt violet cell stain
N - Nauta-Gygax stain
Fr. - Frontal
SAG - Sagittal

CHAPTER III

OBSERVATIONS

Both the lesion and the selective staining of degenerated fibres were satisfactory in sixteen cats. A description of each of these brains is presented below.

Cat No. 1

The lesion is confined to the medial half of the lateral nucleus (Fig.4). It is fusiform in shape with a maximum medio-lateral measurement of 0.8 mm and supero-inferior measurement of 2.0 mm. Antero-posteriorly it extends from Fr. 11.5 to Fr. 10.0. The electrode tract is small, but can be identified descending vertically through the cortex, the internal capsule, the external medullary lamina of the lentiform nucleus and the nucleus centralis pars lateralis of the amygdala.

The degeneration surrounding the lesion is scattered and sparse. Laterally and inferiorly a few fibres are seen coursing towards the external capsule but these are soon lost as they mingle with degenerated fibres from the electrode tract where it passed through the internal capsule. Medially, a few degenerated fibres enter the basal nucleus where their preterminals can be identified. A greater number enter the lateral aspect of the longitudinal association bundle of Johnston and continue forward to the level of the optic chiasma. Here, they begin to fan out medially as they continue their anterior course. At the level of, and just anterior to the anterior commissure, the preterminals of these fibres are seen in the medial third of the anterior amygdaloid area and the lateral preoptic region (Fig.5). A few fibres dorso-medial to the lesion course medially and approach the ansa lenticularis. Here they are lost as they join degenerated fibres from the electrode tract where it passes through the lentiform nucleus. A few degenerated fibres can be seen immediately posterior to the lesion.

CAT #1
C.E.V. #48
FIG.4.

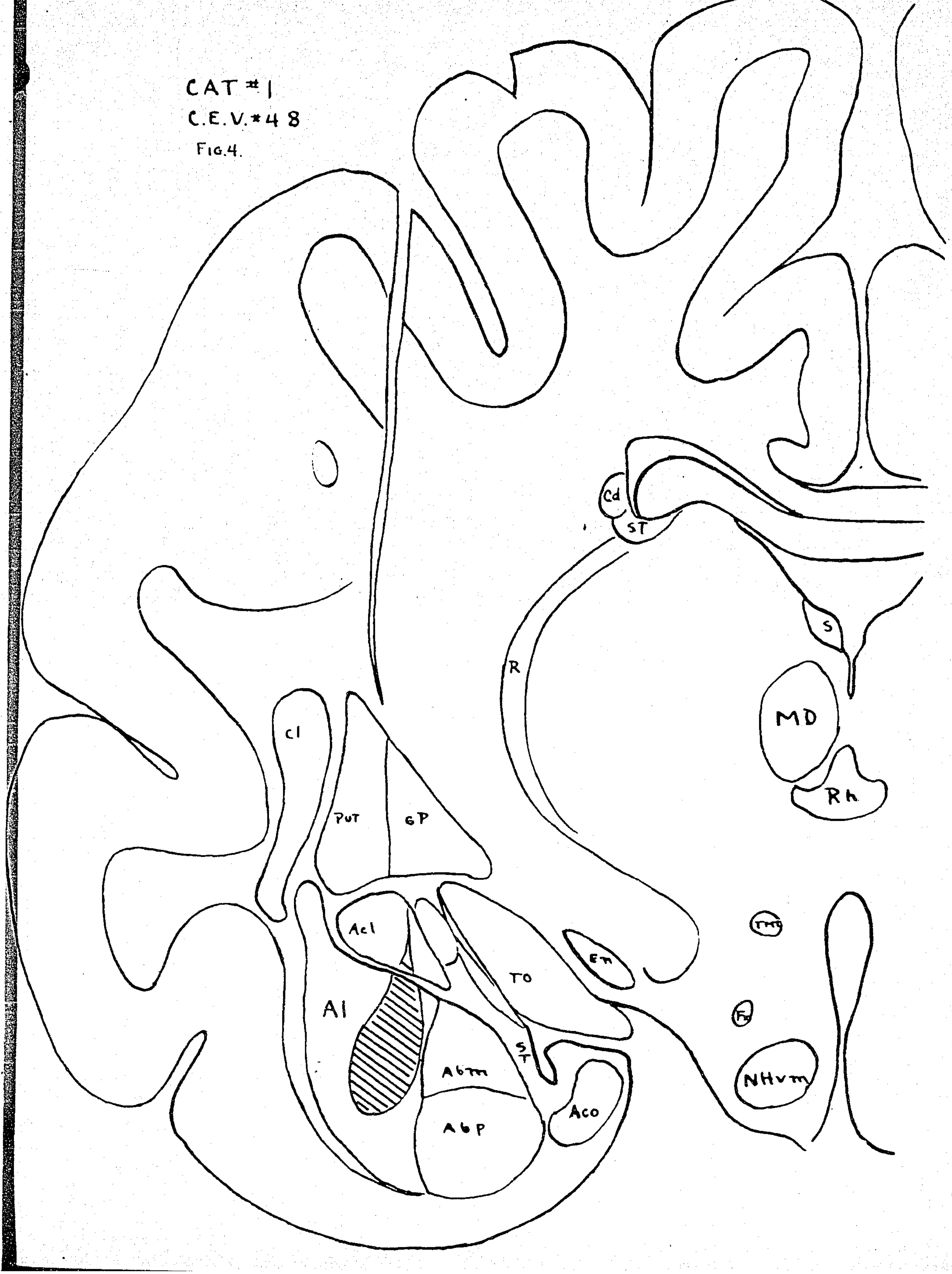


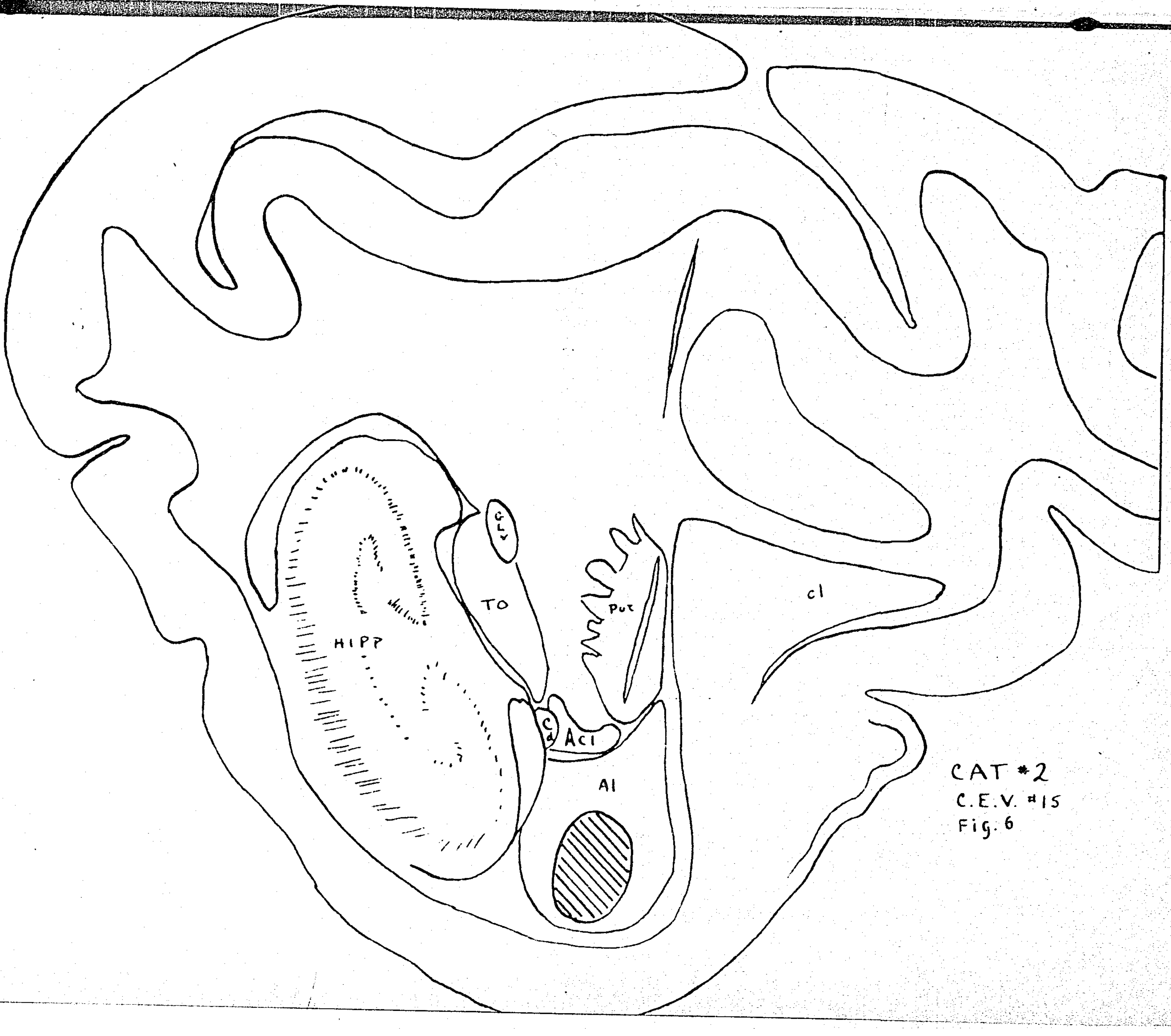
Fig.5: Cat No. 1. Section No. 36. Frontal Plane, Nauta-Gygax Stain.

Degenerated fibres and preterminals in the medial part of the anterior amygdaloid area. X700.

Cat No. 2

The lesion is fusiform in shape and is almost completely confined to the lateral third of the lateral nucleus (Fig.6). However, it does encroach slightly upon the external capsule laterally and inferiorly. Medio-laterally, it extends from sagittal plane 11.0 to 12.0. Its maximum antero-posterior measurement is 2.0 mm while its supero-inferior is 2.5 mm. The electrode tract is small but can be identified passing vertically through the cortex, the internal capsule and the putamen.

The staining of this brain was successful only in the more lateral sections, and thus only findings in this region are reported. Rather sparse, scattered degeneration surrounds the lesion in the lateral nucleus. There are many degenerated fibres in the external capsule due to the encroachment of the lesion on this structure. Some of these fibres are seen coursing between the external capsule and the lateral nucleus (Fig.7). Although their direction cannot be determined with certainty, the presence of preterminals in the lateral nucleus suggests that at least some are amygdaloid afferents. It is important to note that degeneration was not found in the longitudinal association bundle. An occasional fibre can be seen passing just inferior to the ansa lenticularis but these could not be traced medially due to the poor quality of the staining in that area.



CAT * 2
C.E.V. #15
Fig. 6

Fig.7: Cat No. 2. Section No. 12. Sagittal Plane, Nauta-Gygax Stain.

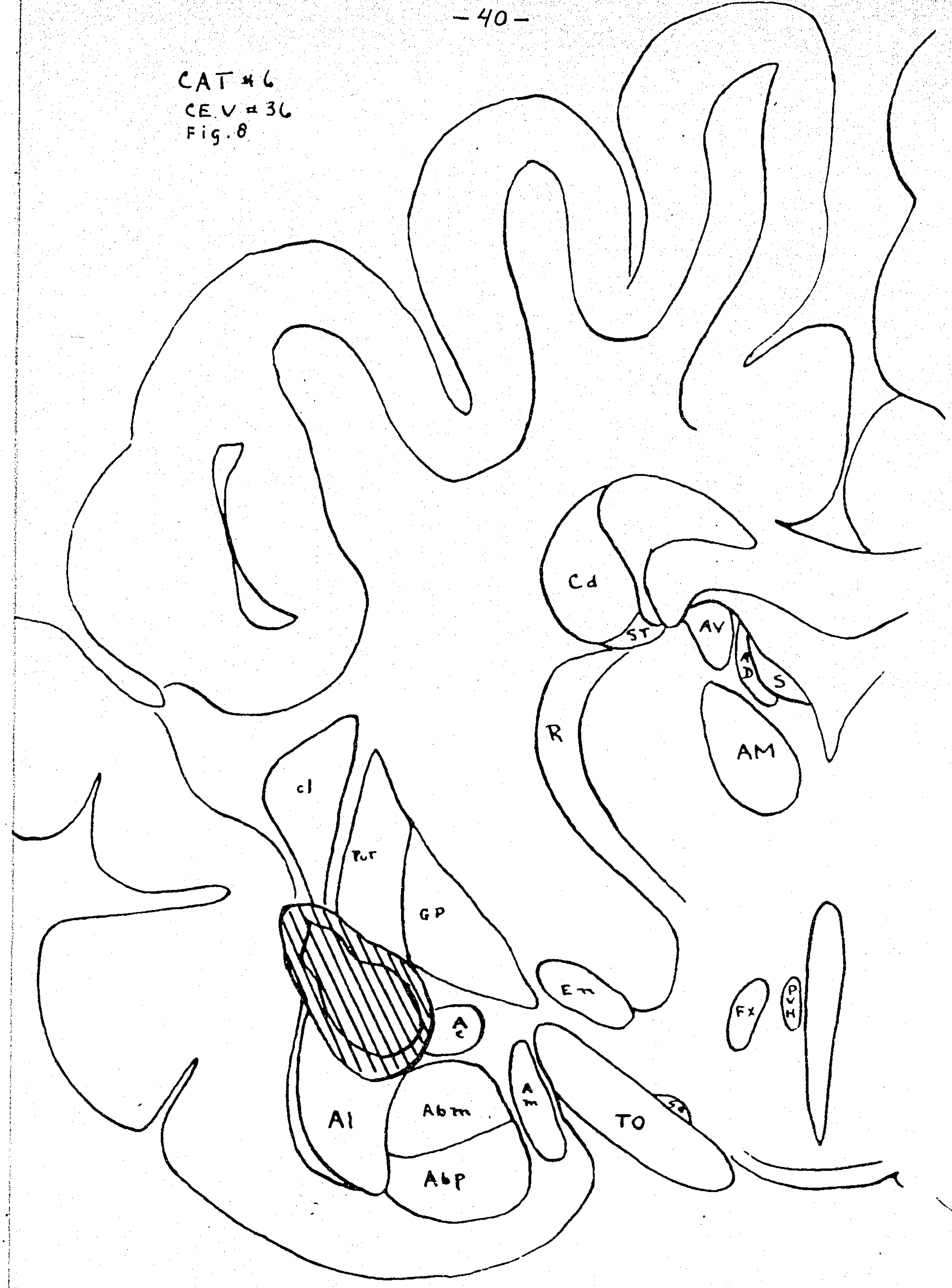
Degenerated fibres coursing between external capsule and lateral nucleus just superior to the lesion. X700.

Cat No. 6

In this brain there is a large irregular lesion which has its long axis directed obliquely in the frontal plane (Fig.8). It extends from the anterior to the posterior limit of lateral nucleus (Fr. 13.5 to Fr. 9.0) occupying mainly its superior one-half. In frontal section, its greatest medio-lateral diameter is 2.0 mm., its supero-inferior 2.5 mm. Anteriorly, it encroaches slightly upon the anterior amygdaloid area. In the mid-region of the nucleus it encroaches dorso-medially on the lower margin of the putamen, the lateral two-thirds of the nucleus centralis pars lateralis and the superior part of the basal nucleus. Only the posterior third of the lesion is completely confined to the lateral nucleus. Both electrode tracts are very fine. They can be seen passing obliquely downward through the cortex and the external capsule just ventral to the claustrum.

Degenerated fibres can be seen surrounding the lesion, but they are more concentrated on its dorsal and medial aspects. Laterally and inferiorly, fibres course between the external capsule and the lateral nucleus. However, it is impossible to determine whether they are afferents or efferents due to the degeneration caused by the electrode tract. The more inferior fibres on the medial aspect of the lesion end as preterminals scattered through the lateral half of the basal nucleus. More superiorly, fibres enter the longitudinal association bundle of Johnston. They form a compact bundle that can be followed anteriorly to the level of the optic chiasma. Here, they become more diffuse and begin to turn medially. Their preterminals can be observed in the medial half of the anterior amygdaloid area and the lateral half of the preoptic region at the level of, and just anterior to the anterior commissure. Between Fr. 11 and Fr. 12.5 some fibres leaving the supero-medial aspect of the lesion course medially just inferior to the ansa lenticularis and join the medial forebrain bundle. They then continue posteriorly in this bundle, giving off preterminals in the lateral hypothalamic region as far caudal as the mammillary bodies (Fig 9). In the posterior half of the lesion, some fibres of passage from the sublenticular fasciculus have been destroyed as they course through the upper third of the lateral nucleus.

CAT #6
CE.V #36
Fig. 8



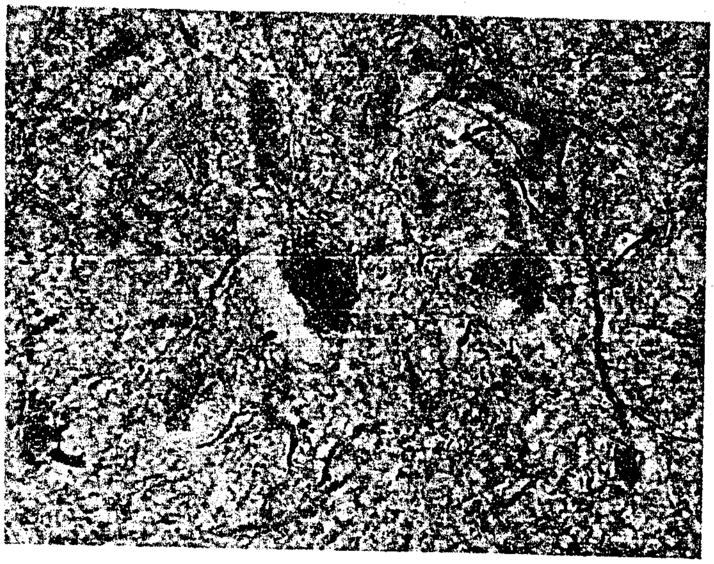


Fig.9: Cat No. 6. Section No. 38. Frontal Plane, Nauta-Gygax Stain.

Degenerated preterminals in the lateral hypothalamus at the level of the nucleus ventralis medialis. X700.

Cat No. 7

The lesion is irregular in shape with its long axis placed obliquely in the frontal plane. It occupies the medial half of the posterior two-thirds of the lateral nucleus (Fig.10) extending from Fr. 12.0 posteriorly to Fr. 9.5. Medio-laterally its maximum measurement is 1.5 mm; supero-inferiorly its maximum measurement is 2.0 mm. The more rostral portion of the lesion encroaches upon the inferior one-third of nucleus centralis pars lateralis and the superior border of the magnocellular part of the basal nucleus. Posteriorly, it involves the most caudal tip of nucleus centralis pars lateralis and the superior margin of the basal nucleus. Both electrode tracts descend obliquely through the cortex and the external capsule and just touch the inferior tip of the putamen. The posterior tract also courses through the lower tip of the claustrum.

Throughout the entire extent of the lesion scattered intra-amygdaloid association fibres can be identified. Those from the lateral aspect of the lesion end within the lateral nucleus itself. Those from the infero-medial aspect end in the basal nucleus. Among the latter are scattered a few large droplet preterminals. These may be the preterminals of afferents reaching the basal nucleus via the external capsule. A few fibres from the lateral aspect of the lesion continue into the external capsule. Here they are lost in the degeneration caused by the electrode tract. In the anterior region of the lesion two main efferent pathways can be identified. The smaller is composed of rather diffusely scattered fibres that course first upwards and medially from the superior two-thirds of the lesion and then medially and slightly inferiorly, passing just beneath the ansa lenticularis. They enter the medial forebrain bundle and a few of the fibres can be traced as far posterior as the mammillary bodies. Their preterminals are confined to the lateral hypothalamus. The second pathway is made up of fibres coursing antero-medially to enter the longitudinal association bundle of Johnston. Their preterminals are found throughout the medial part of the anterior amygdaloid area and in the most lateral part of the preoptic region (Fig.11). In one slide only a few fibres can be followed to the anterior commissure. Their course through the commissure cannot be identified.

CAT #7
C.E.V. #13
Fig. 10

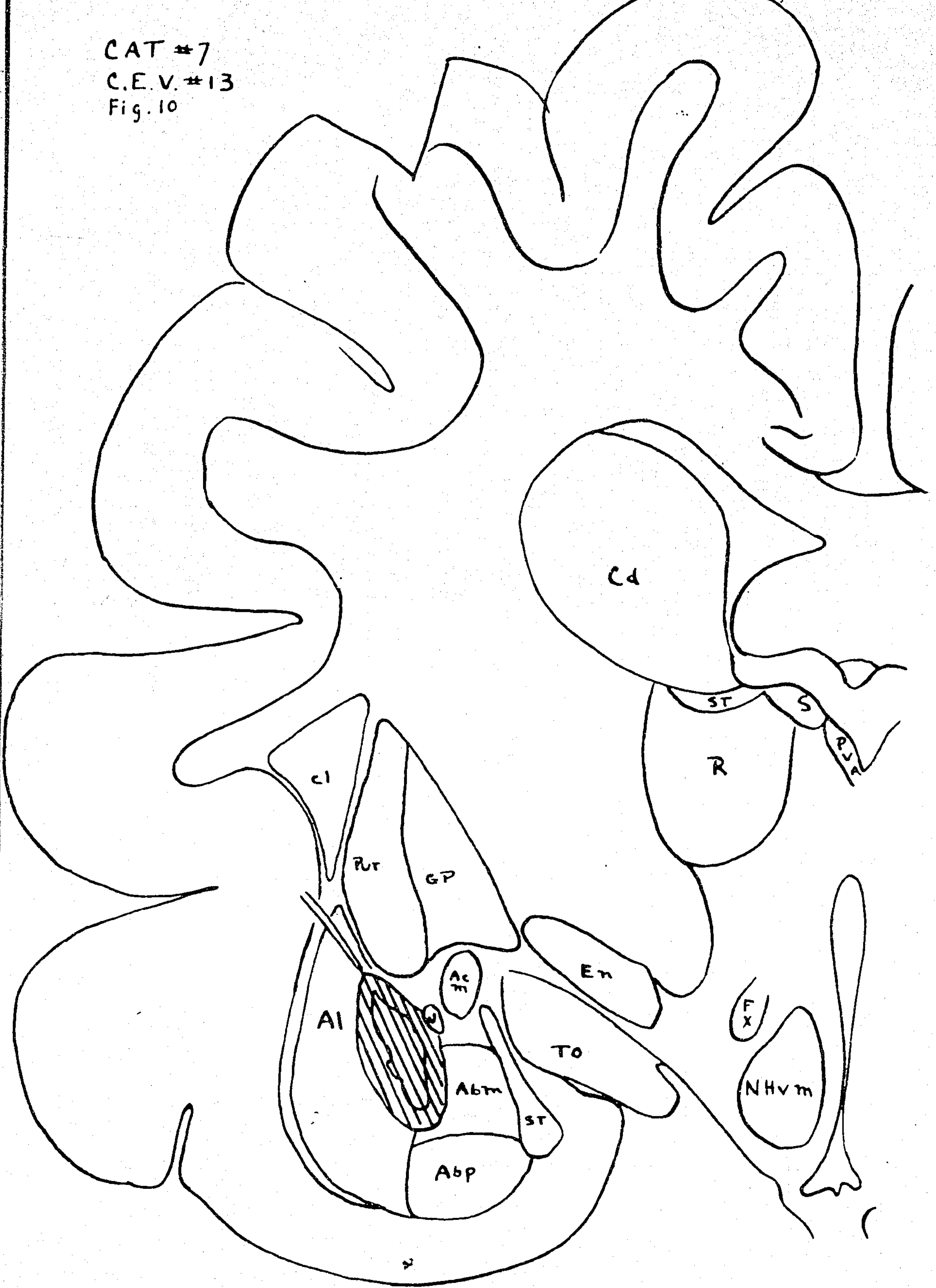


Fig.11: Cat No. 7. Section No. 5. Frontal Plane. Nauta-Gygax Stain.

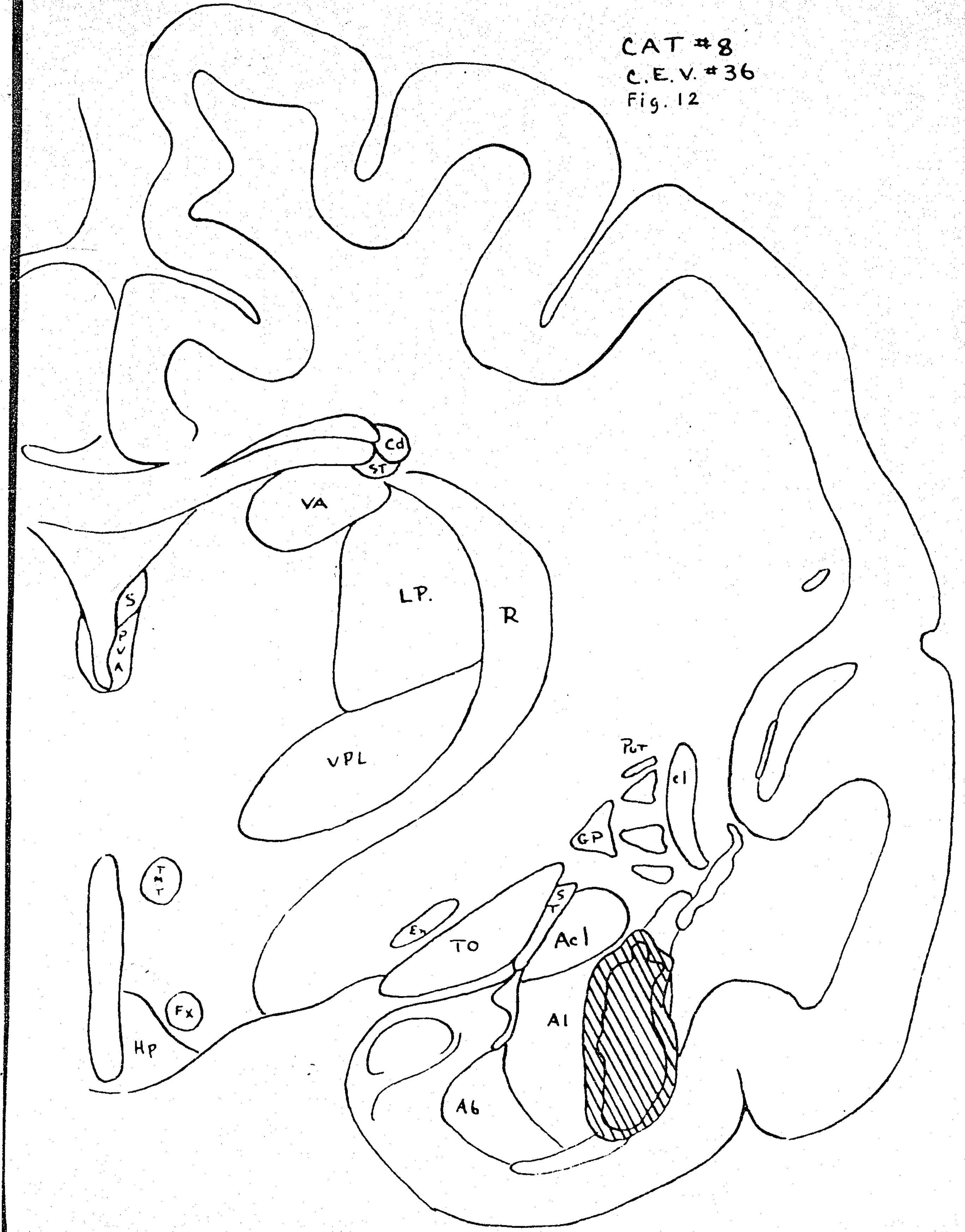
Arrows point to degenerated preterminals in the lateral preoptic region. X700.

Cat No. 8

The lesion is large and roughly ellipsoid in shape (Fig.12). It extends through the posterior two-thirds of the lateral half of the lateral nucleus from Frontal 11.5 to 9.0, the more anterior part involving its upper half, the more posterior involving all but its most medial margin. Its maximum supero-inferior dimension is 2.5 mm, its medio-lateral 2.0 mm. Anteriorly it encroaches upon the infero-lateral border of nucleus centralis pars lateralis, while more posteriorly it extends slightly into the external capsule laterally and the periamygdaloid cortex inferiorly. There is also a minimal encroachment upon the lateral border of the basal nucleus. Both electrode tracts descend obliquely through the cortex and the external capsule. The anterior also passes through the inferior tip of the claustrum.

In the most posterior part of the lateral nucleus a few short scattered degenerated fibres are seen in the immediate vicinity of the lesion. Their preterminals cannot be identified. In the central part of the lesion degenerated fibres and their preterminals are sparsely scattered in the inferior part of the lateral nucleus (Fig.13). Fibres course slightly upwards and medially from the more superior part of the lesion and pass just beneath the ansa lenticularis. They enter the medial forebrain bundle and a few can be followed as far posterior as the mammillary bodies. Their scattered preterminals are confined to the lateral hypothalamus. In the anterior one-third of the lesion degeneration can be observed coursing antero-medially to enter the longitudinal association bundle. It can be followed in this bundle to the anterior amygdaloid area. Here, however, the staining of the sections is not too satisfactory so that even on scanning carefully under high power only an occasional preterminal can be found in the medial part of the anterior amygdaloid area and the lateral preoptic region.

CAT #8
C.E.V. #36
Fig. 12



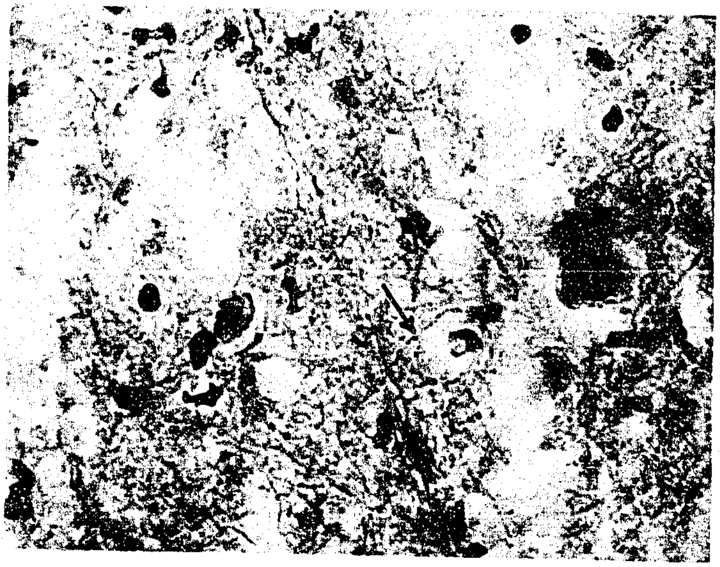


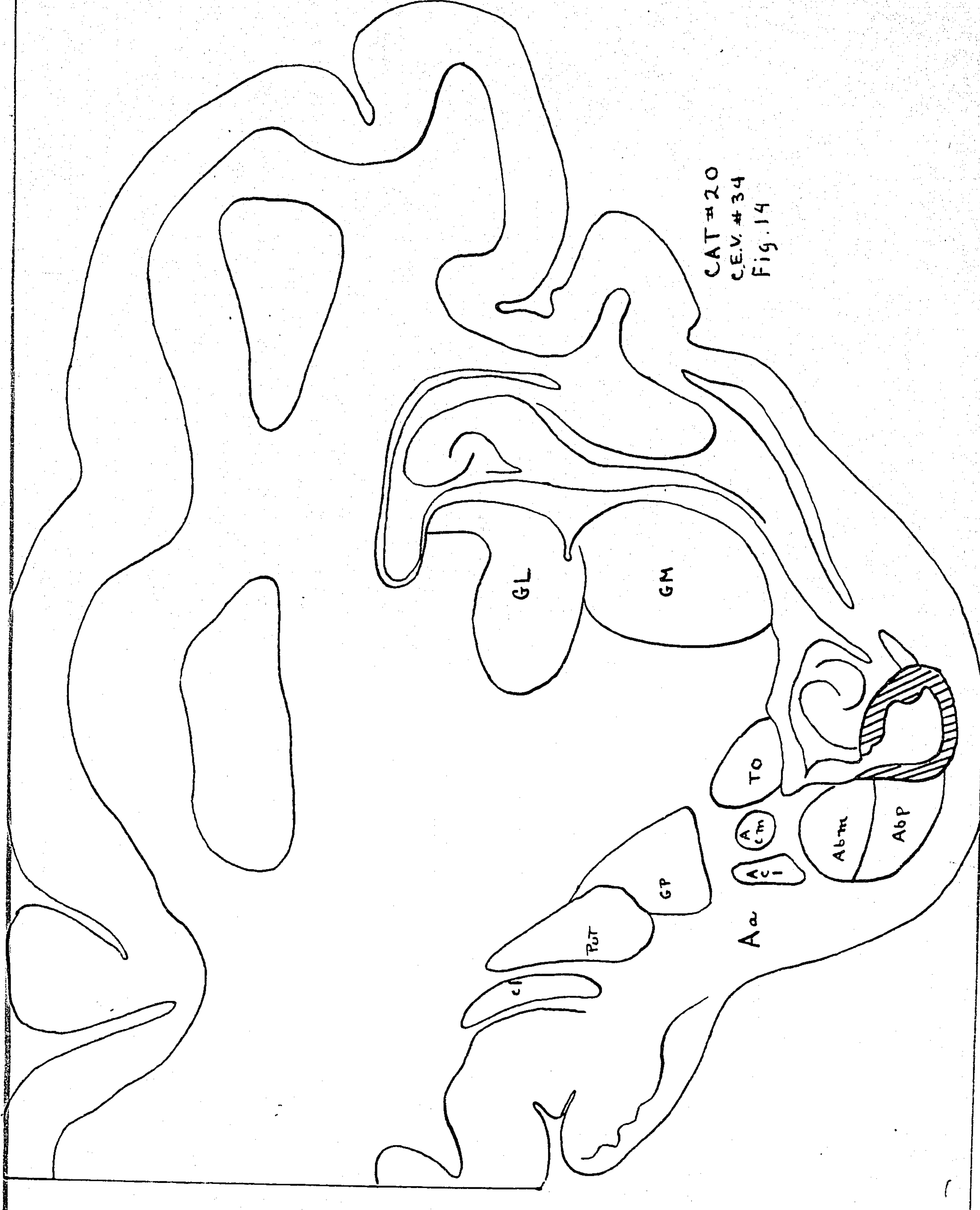
Fig.13: Cat No. 8. Section No. 34. Frontal Plane. Nauta-Gygax Stain.

Degenerated fibres just inferior to the lesion. One is indicated by the arrow. X700.

Cat No. 20

The lesion in brain number 20 involves surrounding structures more than the amygdala itself (Fig.14). It is fusiform in shape and extends medio-laterally from sagittal 9.0 to 11.0. Its maximum supero-inferior measurement is 2.5 mm, its antero-posterior 2.0 mm. It involves both the magnocellular and parvicellular divisions of the basal nucleus. Continuing inferiorly and posteriorly it invades one-half the depth of the cortical nucleus as well as the anterior tip of the hippocampus. The electrode tract passes through the cortex and the external capsule.

In sections lateral to the lesion, that is, in the region of lateral nucleus, degeneration is seen only in the external capsule and the fornix. There are no preterminals in the lateral nucleus. More medially, in the lateral part of the basal nucleus, a great number of degenerated fibres are observed streaming superiorly and anteriorly to join the longitudinal association bundle of Johnston (Fig.15). This degeneration is so heavy that it must be concluded that it arises not only from the basal nucleus, but also from the cortical nucleus. As the longitudinal association bundle is followed medially, the degenerated fibres can be seen fanning out a little as they reach the anterior amygdala. Their preterminals are observed in the lateral two-thirds of the preoptic region. Other fibres course upwards and medially from the lesion to join the stria terminalis. They can be observed along the length of the U-shaped course of the stria terminalis just medial to the tail of the caudate nucleus. They are lost as they approach the anterior commissure. Degeneration in the fornix is due to the encroachment of the lesion upon the hippocampus.



CAT #20
c.E.V. #34
Fig. 14

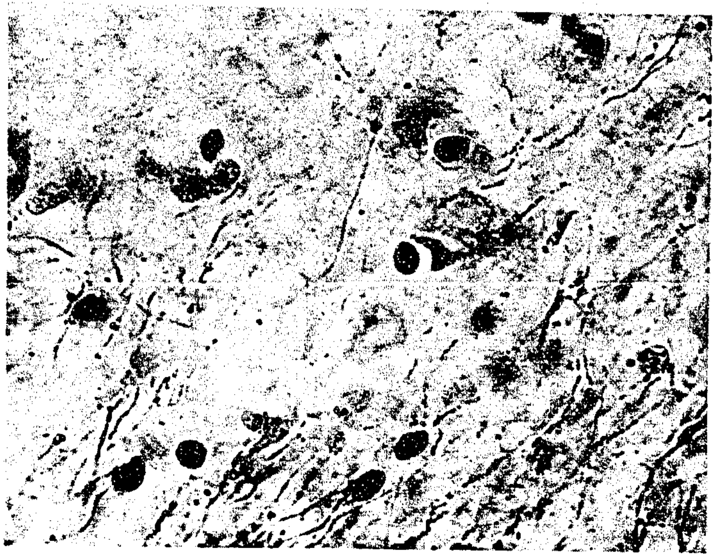


Fig.15: Cat No. 20. Section No. 33. Sagittal Plane, Nauta-Gygax Stain.

Degenerated fibres coursing superiorly and anteriorly in the longitudinal association bundle. X700.

Cat No. 21

The lesion in this brain is small and fusiform in shape. It occupies the lateral one-third of the inferior half of the lateral nucleus (Fig.16) and extends antero-posteriorly from frontal 10.5 to 9.5. Its maximum medio-lateral dimension is 1.0 mm, its supero-inferior 2.0 mm. Except for slight encroachment of its inferior tip on periamygdaloid cortex, the lesion is confined to the lateral nucleus. The thin electrode tract passes obliquely downwards through the cortex, the inferior tip of the claustrum, and the external capsule.

Scattered degeneration is seen in the lateral and basal nuclei in the region of the lesion. A few of their preterminals can be identified. An occasional preterminal is also present in the nucleus centralis pars lateralis. Degenerated fibres can also be traced from the lesion where it has invaded cortex into the lateral and basal nuclei. Their scattered preterminals which are thicker than those of intra-amygdaloid fibres can be found scattered throughout both nuclei. From the lateral aspect of the lesion a few fibres course superiorly and laterally to join the external capsule. Here they are lost among degenerated fibres from the electrode tract. From the more superior part of the medial aspect, a few fibres are directed upwards and medially towards the inferior surface of the ansa lenticularis. They can be followed to the medial forebrain bundle and their preterminals are seen in the lateral hypothalamus. A slightly denser concentration of degenerated fibres just anterior to the lesion courses medially and slightly superiorly and anteriorly into the lateral aspect of the longitudinal association bundle of Johnston (Fig.17). A few slightly larger degenerated fibres that may be cortical in origin course upwards between the lateral and basal nuclei to enter the medial aspect of the bundle. All the degenerated fibres in the longitudinal association bundle continue forwards to the level of the optic chiasma. Here they begin to fan out as they continue anteriorly. Their preterminals are found in the medial half of the anterior amygdaloid area and the lateral preoptic region.

CAT #21.
C.E.V. #35

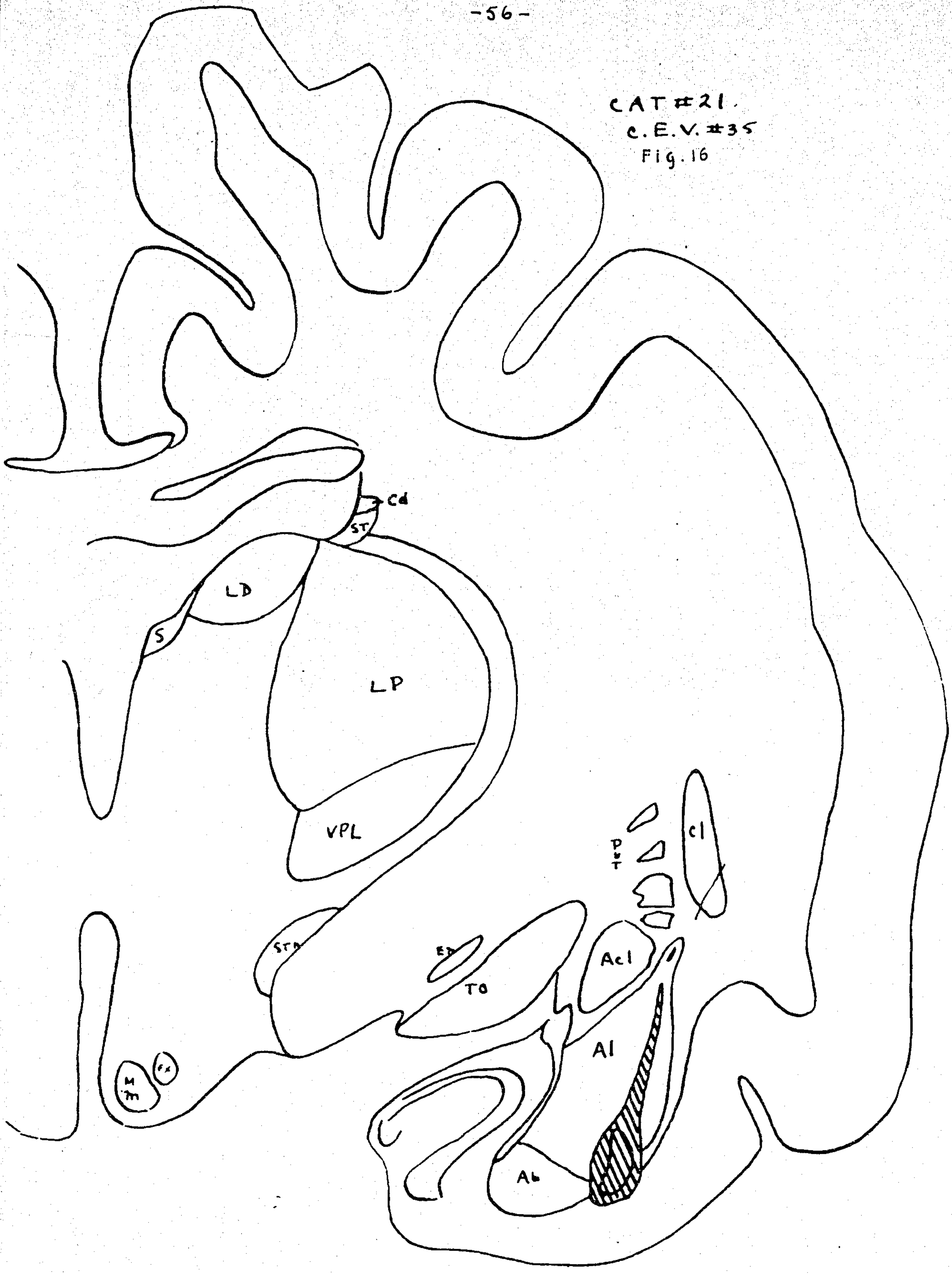


Fig. 16

Fig.17: Cat No. 21. Section No. 23. Frontal Plane, Nauta-Gygax Stain.

Degeneration coursing superiorly medially and anteriorly to enter the longitudinal association bundle. X700.

Cat No. 23

The lesion is small, ellipsoid in shape, and almost completely confined to the inferior one-third of the lateral nucleus (Fig.18). It extends medio-laterally from sagittal plane 10 to 11. Its maximum supero-inferior dimension is 1.5 mm, its antero-posterior 1.0 mm. There is very slight encroachment upon the external capsule inferiorly.

Although this brain was not stained well it has not been excluded from the series because of some rather interesting normal fibres that can be observed. These fibres course upwards almost at a right angle to the external capsule from the cortex just inferior to the most posterior and medial part of the lateral nucleus and join the longitudinal association bundle of Johnston. Only a few degenerated fibres have been stained. These course antero-medially to enter the longitudinal association bundle but cannot be followed along its course.

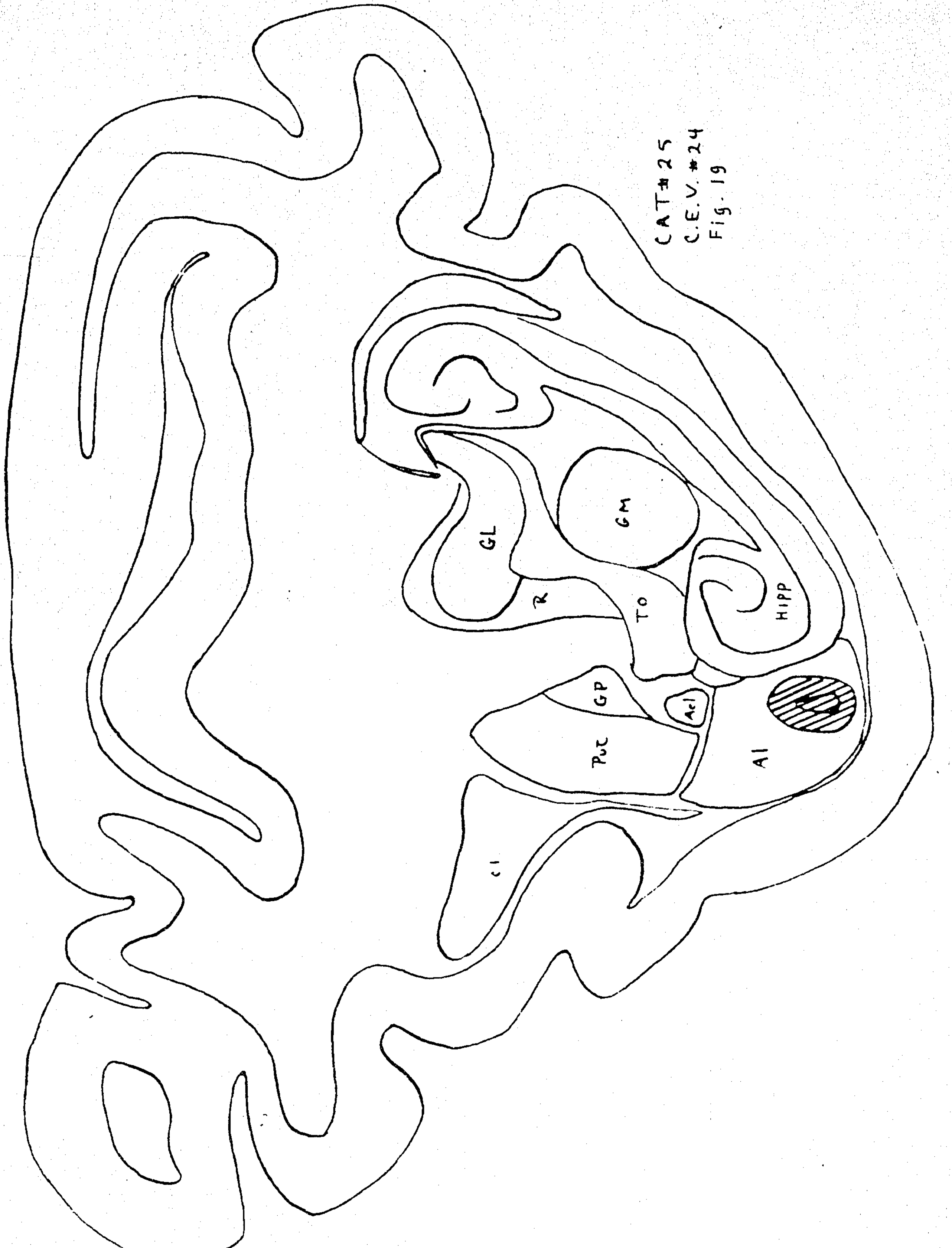
CAT #23
C.E.V. #23
Fig. 18

Cat No. 25

The lesion is small, fusiform in shape and located in the posterior two-thirds of the lateral half of the lateral nucleus (Fig.19). It extends medio-laterally from sagittal 9.5 to 11. Its maximum antero-posterior measurement is 1.5 mm, its supero-inferior 2.0 mm. Medially, it just touches the lateral border of the basal nucleus. There is no other encroachment on surrounding structures. The electrode tract passes through the cortex and the external capsule.

Degenerated fibres coursing inferiorly from the lesion fan out anteriorly and posteriorly as they join the external capsule. However, once they enter the external capsule they can no longer be identified. They do not appear to continue medially or laterally nor can they be found in the cortex, even though the staining is quite good. A few fibres end as preterminals within the lateral nucleus and a very occasional one ends within the inferior part of the basal nucleus. Degenerated fibres course superiorly and medially from the lesion to enter the lateral aspect of the longitudinal association bundle (Fig.20). They can be followed anteriorly in this bundle to the medial part of the anterior amygdaloid region and lateral preoptic region where their preterminals can be identified.

Degeneration could not be followed to the medial forebrain bundle. Rather large degenerated fibres and preterminals are seen in the superior part of the lateral nucleus. They appear to be entering the nucleus from the external capsule.



CAT #25
C.E.V. #24
Fig. 19

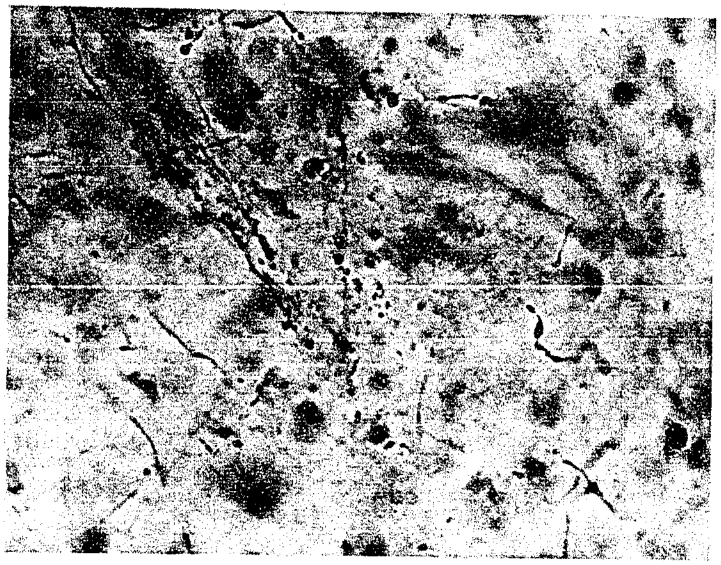


Fig.20: Cat No. 25. Section No. 25. Sagittal Plane, Nauta-Gygax Stain.

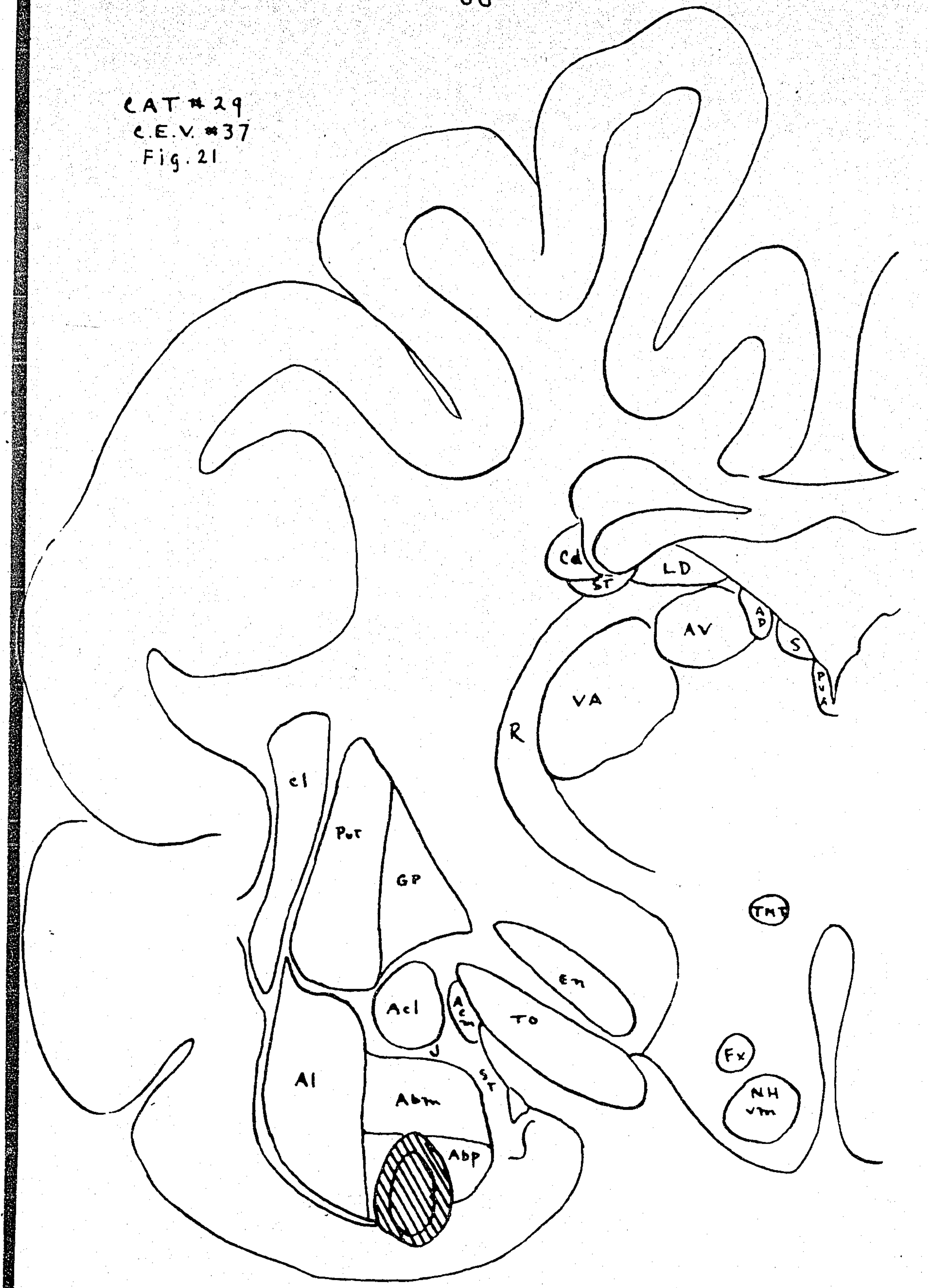
Degeneration leaving the anterior aspect of the lesion and coursing superiorly and anteriorly to join the longitudinal association bundle. X700.

Cat No. 29

The lesion is almost spheroid in shape and occupies the inferior half of the lateral two-thirds of the basal nucleus. It extends antero-posteriorly from Fr. plane 12.0 to 10.0. The maximum medio-lateral dimension is 2.0 mm., the supero-inferior 2.5 mm. The posterior half of the lesion encroaches inferiorly upon the cortical nucleus (Fig.21) and at its most posterior limit extends through its entire depth. The electrode tract passes obliquely downwards, medially and inferiorly through the cortex, the external capsule, and the anterior part of the lateral nucleus.

In the region of the lesion, short degenerated fibres and their preterminals are seen scattered through the basal, lateral and cortical nucleus. From the superior and medial aspect (Fig.22), and to a lesser extent from the lateral border of the lesion, a great number of degenerated fibres course superiorly. Many form small compact bundles. As they approach the upper margin of the nucleus they branch into two main pathways. The great majority course laterally to join the medial aspect of the longitudinal association bundle of Johnston. A few turn medially to enter the stria terminalis. The former fibres continue anteriorly and slightly medially in the longitudinal association bundle to the level of the optic chiasma. Here they become less compact as they bend upwards and medially to form a distinct bundle that courses just inferior to the internal capsule. Fibres leave the medial aspect of this bundle as it continues anteriorly to the level of the anterior commissure. Many preterminals are found in the intermediate part of the preoptic region, while only a few are found in its most lateral part. A few fibres are seen entering the anterior commissure but they cannot be followed with certainty across the midline. The smaller band of fibres that joined the stria terminalis can be followed to the level of the optic chiasma. Their preterminals cannot be located.

CAT #29
C.E.V. #37
Fig. 21



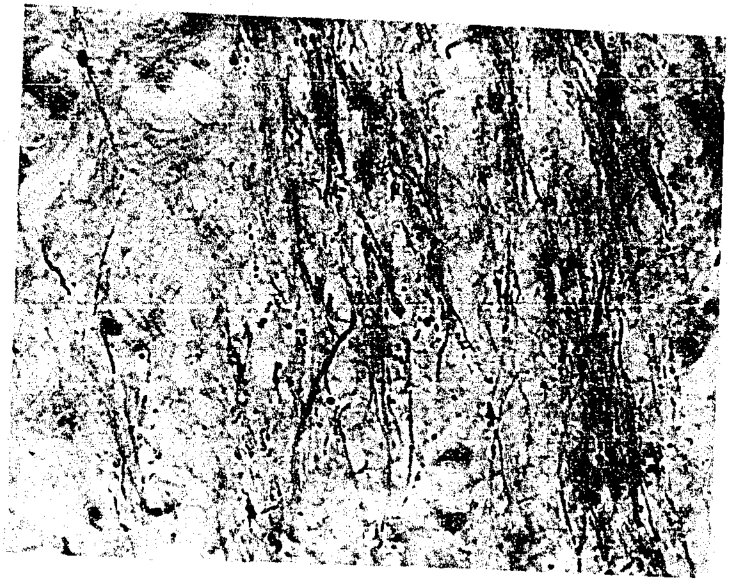


Fig.22: Cat No. 29. Section No. 33. Frontal Plane, Nauta-Gygax Stain.

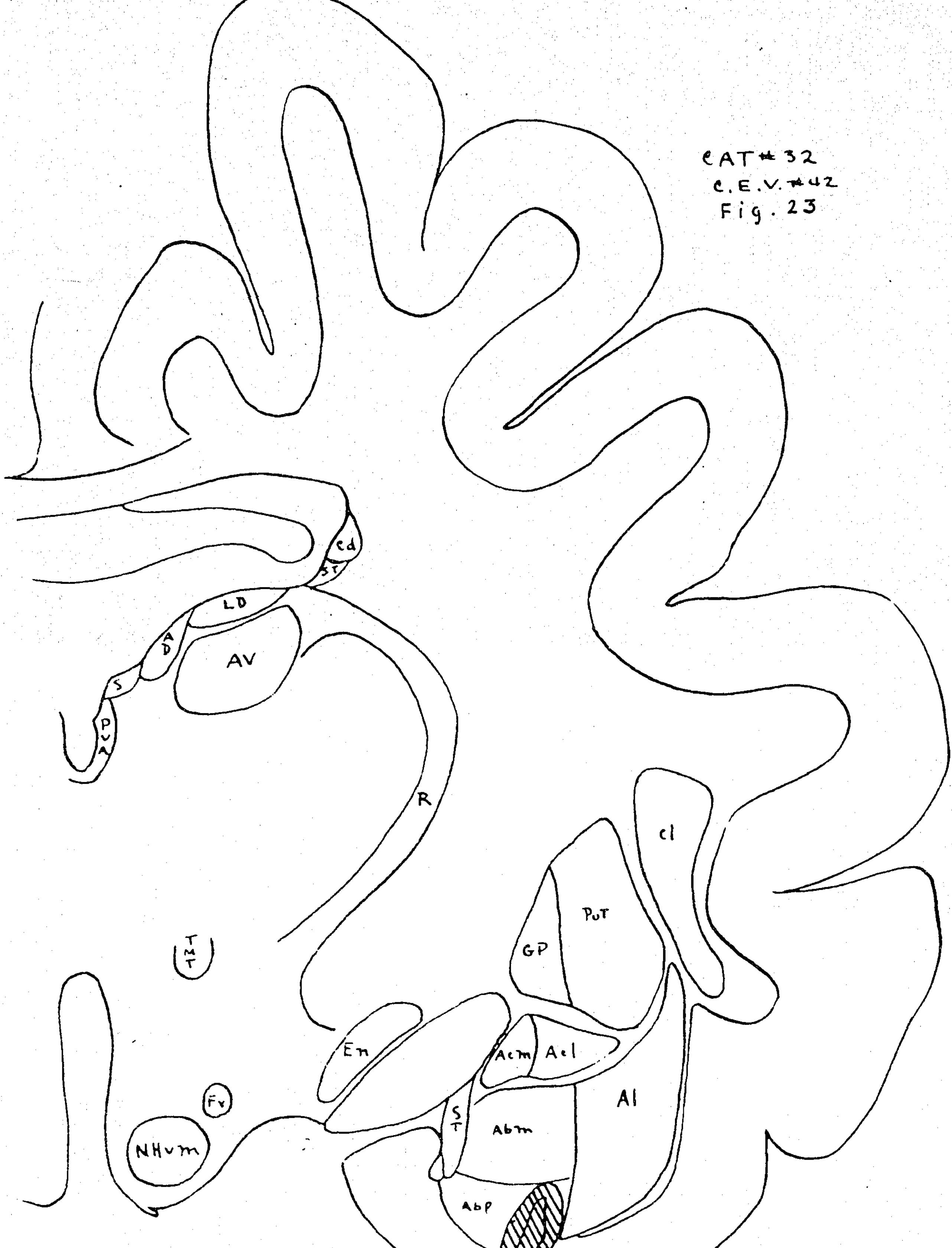
Degeneration coursing superiorly from the upper margin of the lesion. X700.

Cat No. 32

The lesion in this brain is small and ellipsoid in shape. It occupies the inferior lateral quadrant of the basal nucleus. Antero-posteriorly it extends from Fr. 11.0 to 10.0. Its greatest medio-lateral dimension is 1.5 mm., its supero-inferior 2.0 mm. The posterior half of the lesion encroaches upon the cortical nucleus (Fig.23). At its most posterior limit it reaches the inferior surface of the hemisphere. The electrode descends obliquely through the cortex, the external capsule and the lateral nucleus.

A great number of degenerated fibres course superiorly and anteriorly from the lesion. They curve slightly medially (Fig.24), then laterally and join the medial aspect of the longitudinal association bundle. They continue anteriorly in this bundle to the level of the optic chiasma. Between the level of the optic chiasma and the anterior commissure these fibres form a less compact bundle that bends medially and courses just beneath the base of the internal capsule. Unfortunately, the sections at the level of the anterior commissure are rather pale, and only a few proterminals of the bundle can be identified. They are scattered throughout the intermediate third of the preoptic region. A small number of degenerated fibres continue medially and superiorly from the lesion to join the stria terminalis. They can be followed in this bundle of fibres to the level of the most posterior part of the septum.

Here they are lost because of the poor quality of the stain. In the most posterior part of the lesion degenerated fibres from the cortical nucleus are also seen joining the stria terminalis. A few preterminals are scattered throughout the basal nucleus in the region of the lesion. They are probably intra-nuclear fibres.



CAT # 32
C.E.V. # 42
Fig. 23

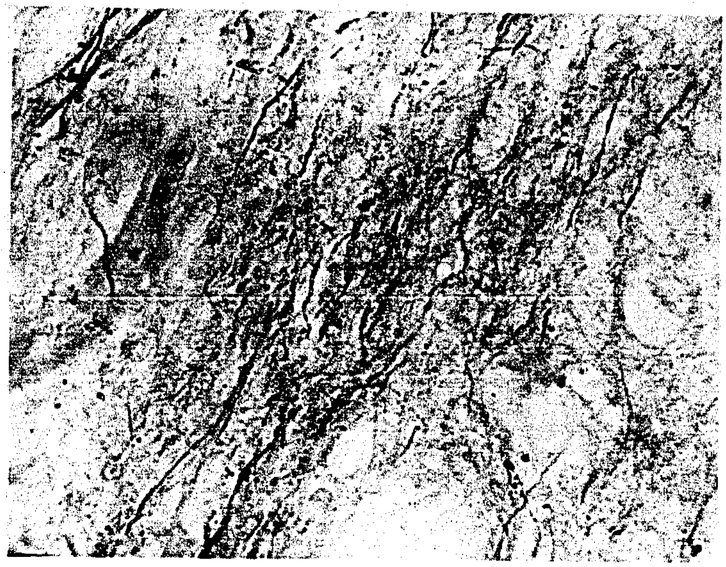


Fig.24: Cat No. 32. Section No. 31. Frontal Plane, Nauta-Gygax Stain.

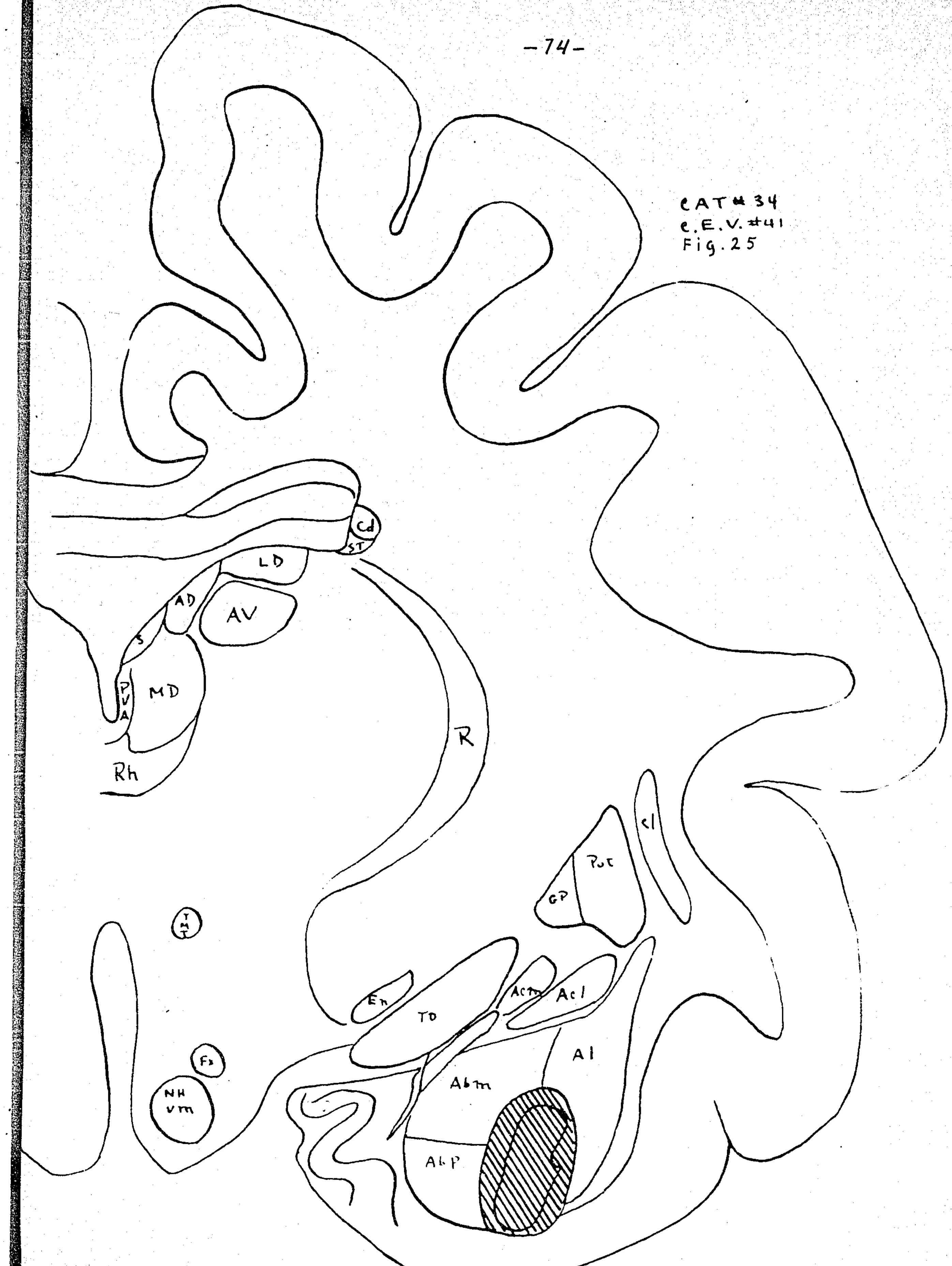
Degenerated fibres coursing upwards and medially just anterior to the lesion to enter the longitudinal association bundle. X700.

Cat No. 34

The lesion is fusiform in shape and is located principally in the lateral nucleus (Fig.25). It extends from Frontal 12.5 to Frontal 10.0. Its maximum medio-lateral dimension is 2.0 mm., its supero-inferior 3.5 mm. Anteriorly, the lesion is confined to the medial and inferior two-thirds of the lateral nucleus. Its posterior half however, encroaches increasingly upon the basal nucleus until, most posteriorly, it involves its lateral half. In this region the lesion also encroaches slightly upon the cortical nucleus. The electrode tract passes obliquely through the cortex, the external capsule, and the lateral nucleus.

The degenerated fibres course upwards and anteriorly from the lesion and enter the central part of the longitudinal association bundle. Most of these fibres come from the superior, anterior and medial aspects of the lesion. Very few arise from its lateral aspect. They continue anteriorly in this bundle to the level of the optic chiasma. Here they fan out medially (Fig.26) into the lateral half of the preoptic region where their preterminals can be identified.

A few fibres from the medial aspect of the lesion in the area involving the basal nucleus course upwards and medially to join the stria terminalis. These fibres can be followed along the course of the stria terminalis to its most anterior limit. Here a few continue in its supracommissural component. Their preterminals can not be identified. In the region of the lesion a few short fibres and their degenerated preterminals are found in the basal nucleus. Degenerated cortico-amygdaloid or amygdalo-cortical fibres are not present. Degeneration cannot be followed into the medial forebrain bundle.



CAT# 34
C.E.V. #41
Fig. 25

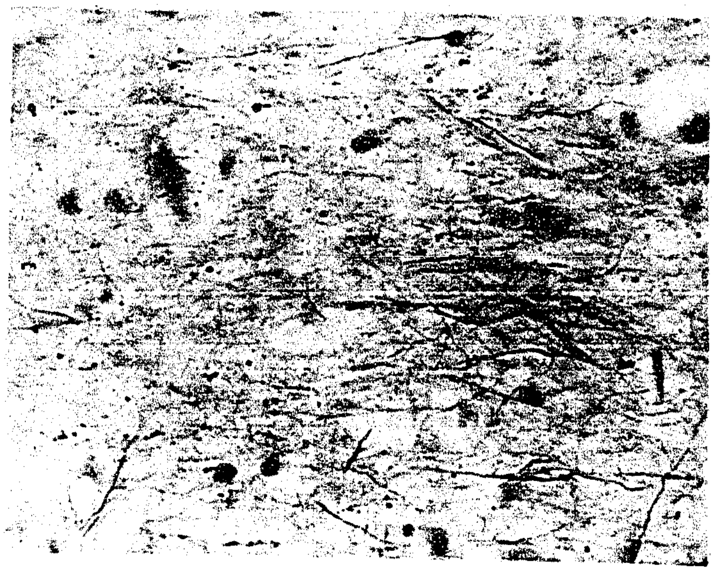


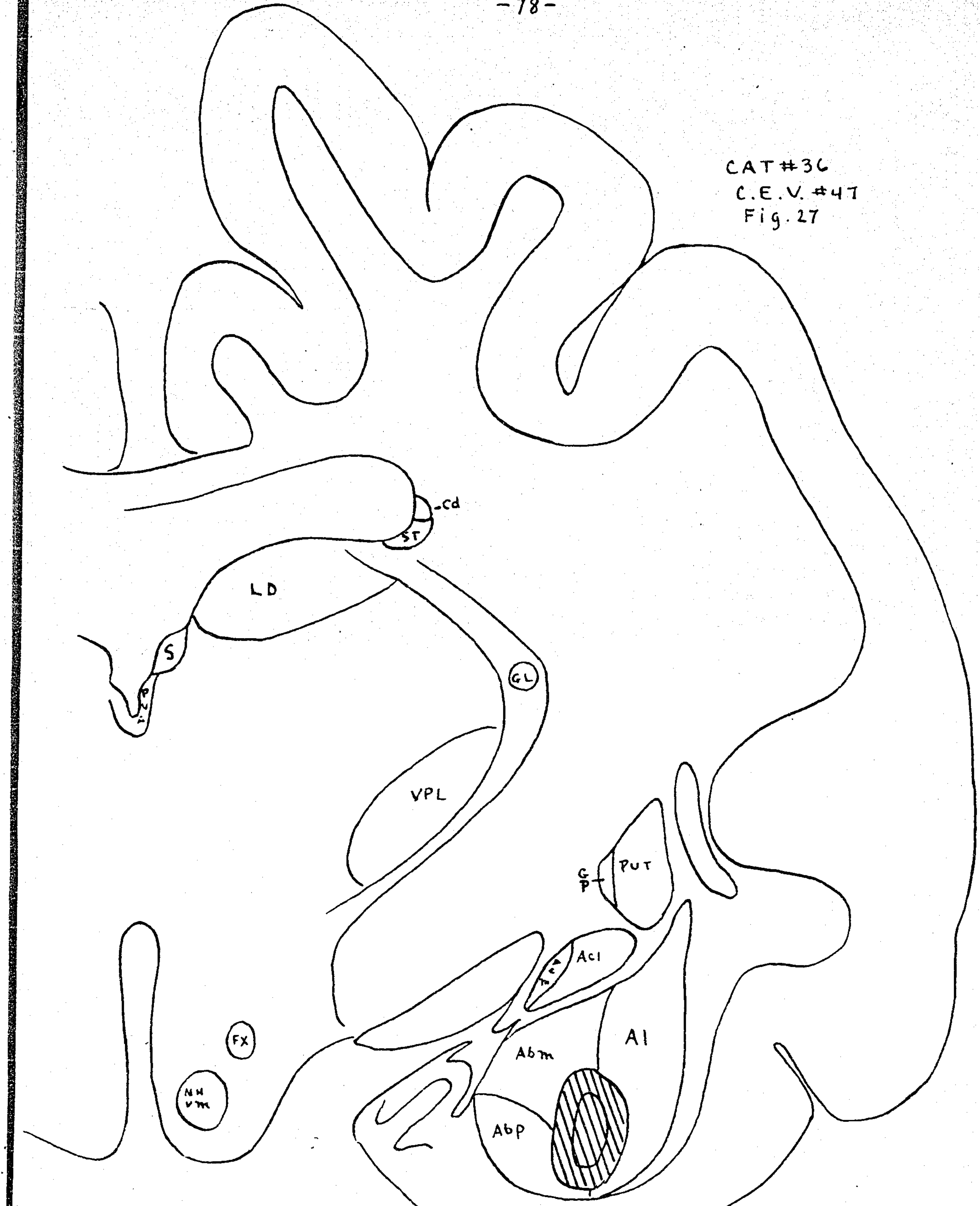
Fig.26: Cat No. 34. Section No. 19. Frontal Plane, Nauta-Gygax Stain.

Degenerated fibres coursing medially in the lateral part of the preoptic region. X700.

Cat No. 36

The lesion is fusiform in shape and occupies the lower part of the medial one-third of the lateral nucleus and the lateral one-third of the basal (Fig.27). Most anteriorly it is confined to the lateral nucleus, while most posteriorly it involves only the basal nucleus. The lesion extends antero-posteriorly from Frontal 12.5 to 9.5. Medio-laterally its maximum measurement is 1.5 mm., supero-inferiorly 2.5 mm. There is very slight encroachment into the cortical nucleus inferiorly. The electrode tract passes through the cortex, the external capsule and the lateral nucleus.

Many degenerated fibres course upwards and medially from the lesion. They are especially dense on its medial and superior aspect. After coursing medially for a short distance they continue upwards anteriorly and laterally to join the central part of the longitudinal association bundle. A dense concentration of degenerated fibres is found in the longitudinal association bundle as far anterior as the optic chiasma. Here the bundle takes a sharp bend medially and slightly anteriorly just beneath the base of the internal capsule. The fibres then scatter diffusely through the lateral two-thirds of the preoptic region (Fig.28). Their preterminals are found in this region and also in the lateral part of the bed nucleus of the anterior commissure. A few degenerated fibres course medially and superiorly from the lesion and enter the stria terminalis. They can be followed throughout the course of this bundle and their preterminals can be identified in its bed nucleus. An occasional fibre can be followed to just beneath the ansa lenticularis and a few can be identified in the medial forebrain bundle. The preterminals of short intra-amygdaloid fibres can be identified in the basal and lateral nuclei in the region of the lesion.



CAT #36
C.E.V. #47
Fig. 27

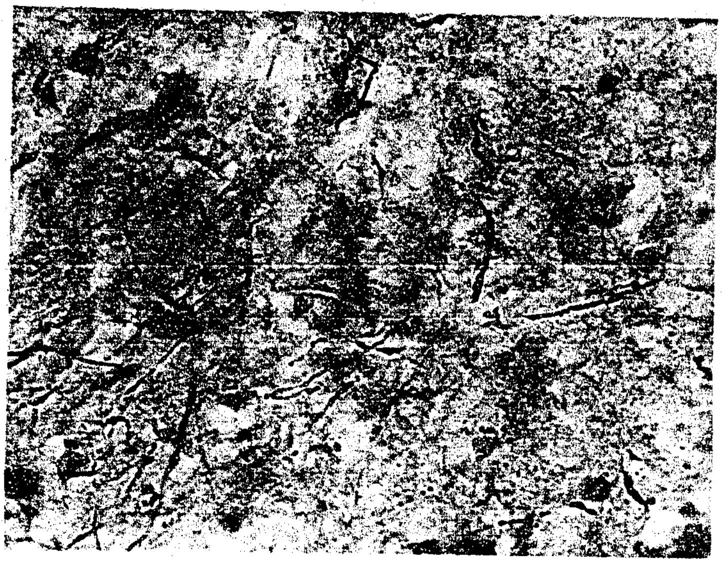


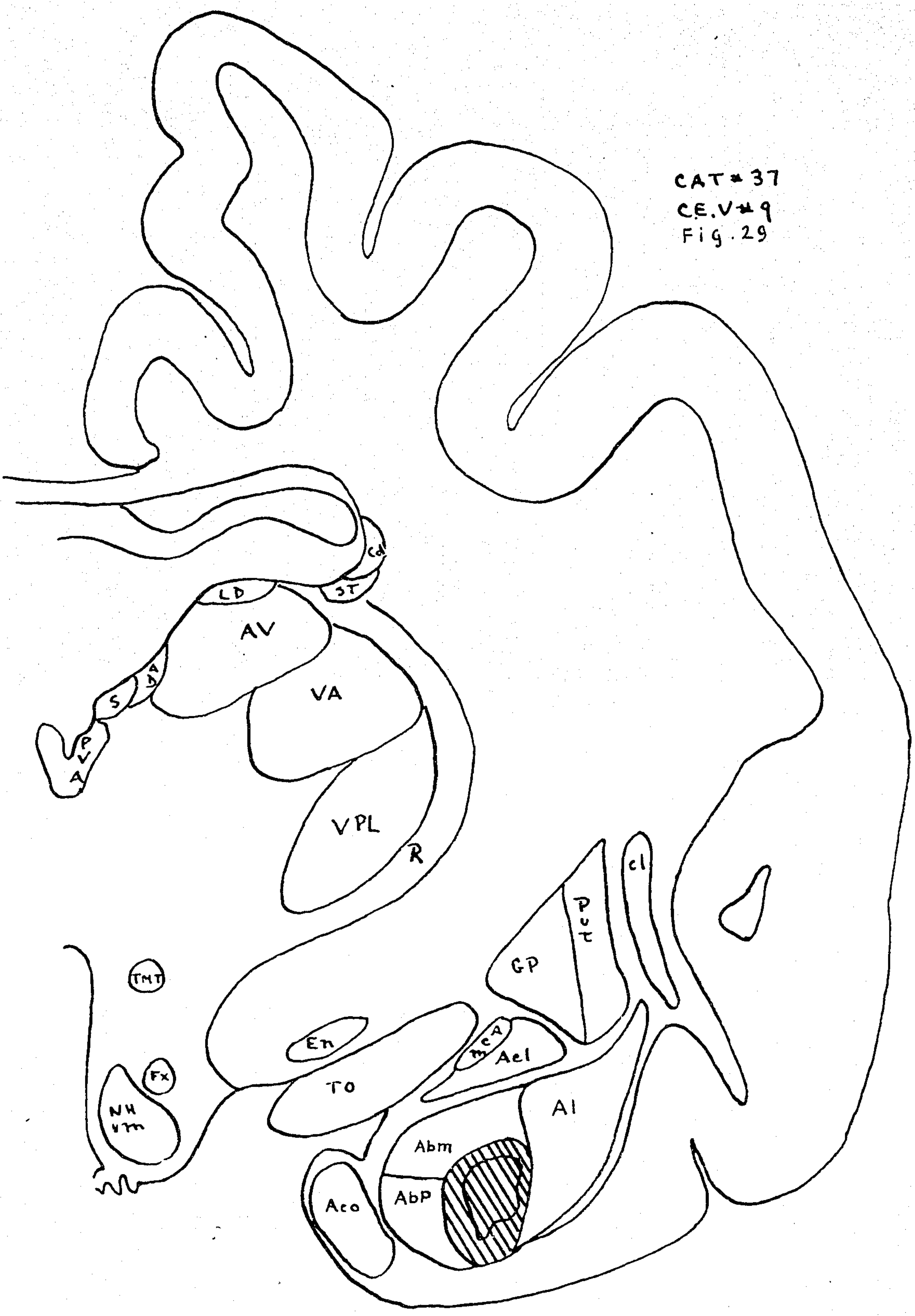
Fig.28: Cat No. 36. Section No. 22. Frontal Plane, Nauta-Gygax Stain.

Degenerated fibres of passage and preterminals in the intermediate part of the preoptic region. X700.

Cat No. 37

The lesion is ellipsoid in shape and occupies the lateral half of the inferior two-thirds of the basal nucleus (Fig.29). It extends antero-posteriorly from Frontal 11.5 to 9.5. Medio-laterally its maximum dimension is 2.0 mm., supero-inferiorly 3.0 mm. The lesion is almost completely confined to the basal nucleus. There is very slight encroachment into the cortical nucleus inferiorly. The electrode tract courses through the cortex, the external capsule, and the lateral nucleus.

Heavy bundles of degenerated fibres course upwards from the superior and medial margin of the lesion. The more medial fibres bend slightly medially, then laterally as they course anteriorly towards the longitudinal association bundle. At the level of the optic chiasma the fibres turn medially and scatter in the preoptic region and bed nucleus of the anterior commissure (Fig.30). Their preterminals are found in the medial two-thirds of these nuclei. A few fibres continue into the anterior commissure but they cannot be followed across the midline. Degeneration from the lesion also enters the stria terminalis. A few of those fibres can be followed to the bed nucleus of the stria and a few more can be seen entering the supracommissural bundle. Their preterminals can be identified in the bed nucleus of the stria and the precommissural part of the bed nucleus of the anterior commissure respectively. The occasional preterminal of an intra-amygdaloid fibre can be identified in the basal and lateral nucleus in the region of the lesion.



CAT # 37
CE.V # 9
Fig. 29

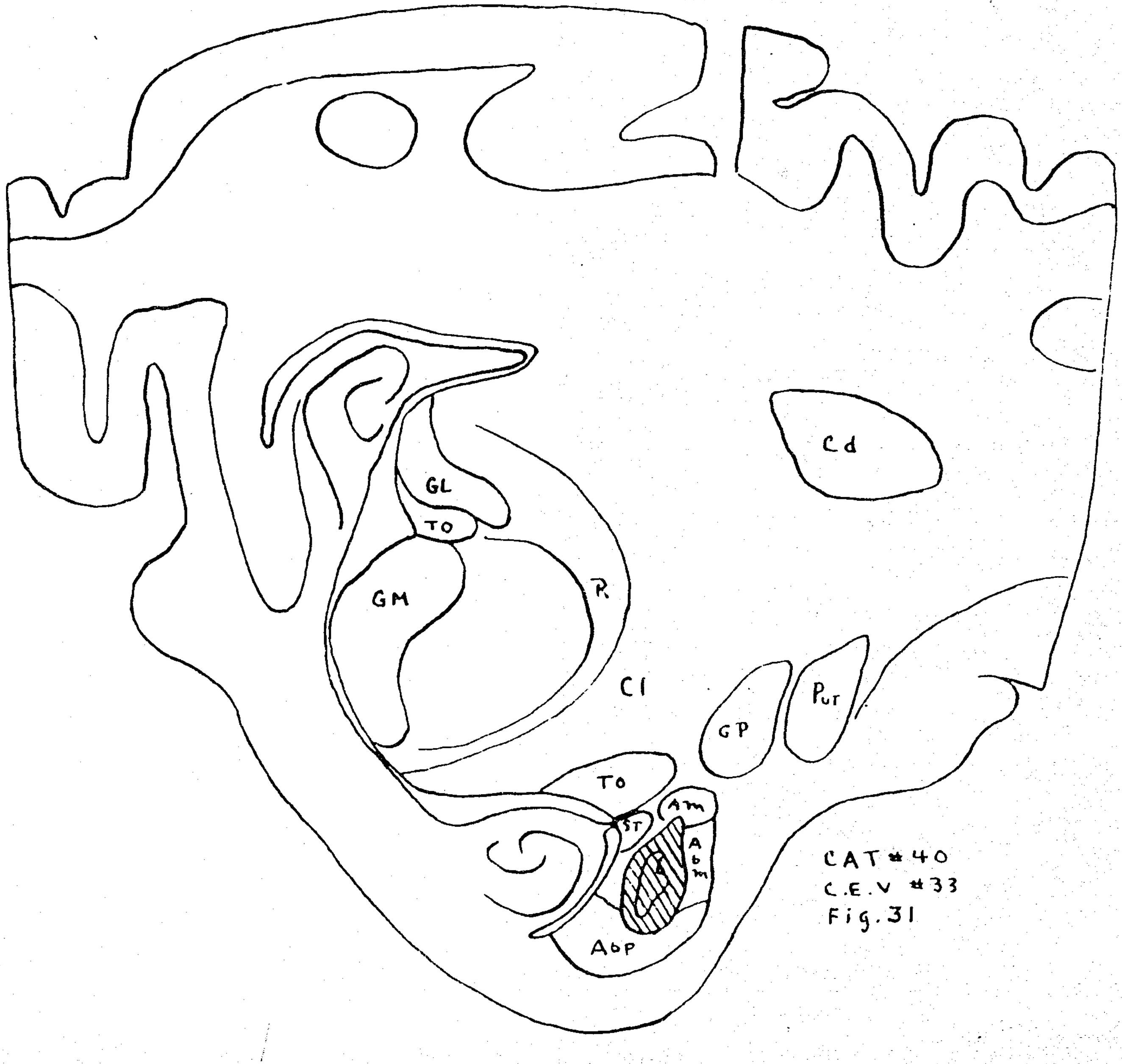
Fig.30: Cat No. 37. Section No. 10. Frontal Plane, Nauta-Gygax Stain.

Degenerated fibres and preterminals in the bed nucleus of the anterior commissure. X700.

Cat No. 40

The lesion is fusiform in shape and is almost completely confined to the central part of the basal nucleus (Fig.31). It extends from Sagittal 9.0 to 7.5. Antero-posteriorly its maximum dimension is 1.5 mm., supero-inferiorly 2.5 mm. Superiorly, it encroaches very slightly upon nucleus centralis pars lateralis. The electrode tract passes through the cortex, the external capsule and the superior part of the lateral nucleus.

Many degenerated fibres are seen in the region of the lesion. A great number course upwards slightly medially and anteriorly to enter the longitudinal association bundle. These can be traced as a dense band to just anterior to the optic chiasma. Here they fan out, coursing anteriorly and medially. Their preterminals are found scattered through the medial half of the preoptic region and the nucleus of the anterior commissure. Many fibres join the stria terminalis. They can be followed along the course of the stria to its point of division (Fig.32). Here they enter the supracommissural component. Unfortunately they become lost at this point in an area of granular artefact. Short intra-amygdaloid fibres and their preterminals are found in the basal and lateral nuclei.



CAT #40
C.E.V #33
Fig. 31

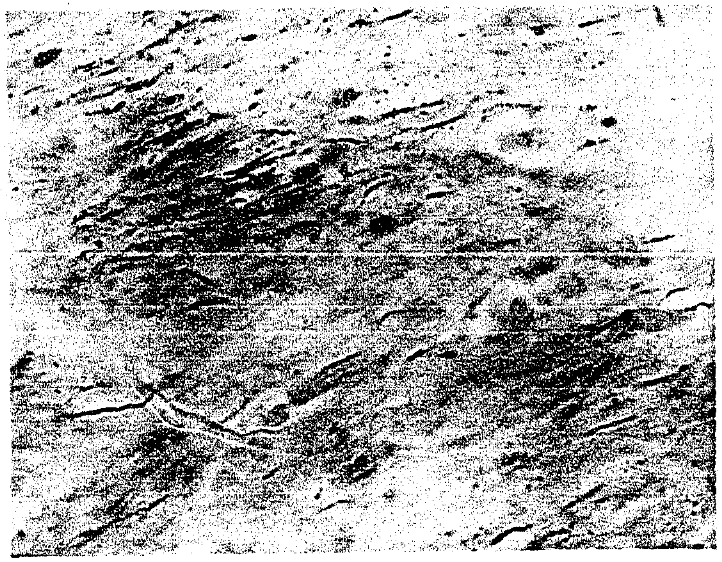


Fig.32: Cat No. 40. Section No. 29. Sagittal Plane, Nauta-Gygax Stain.

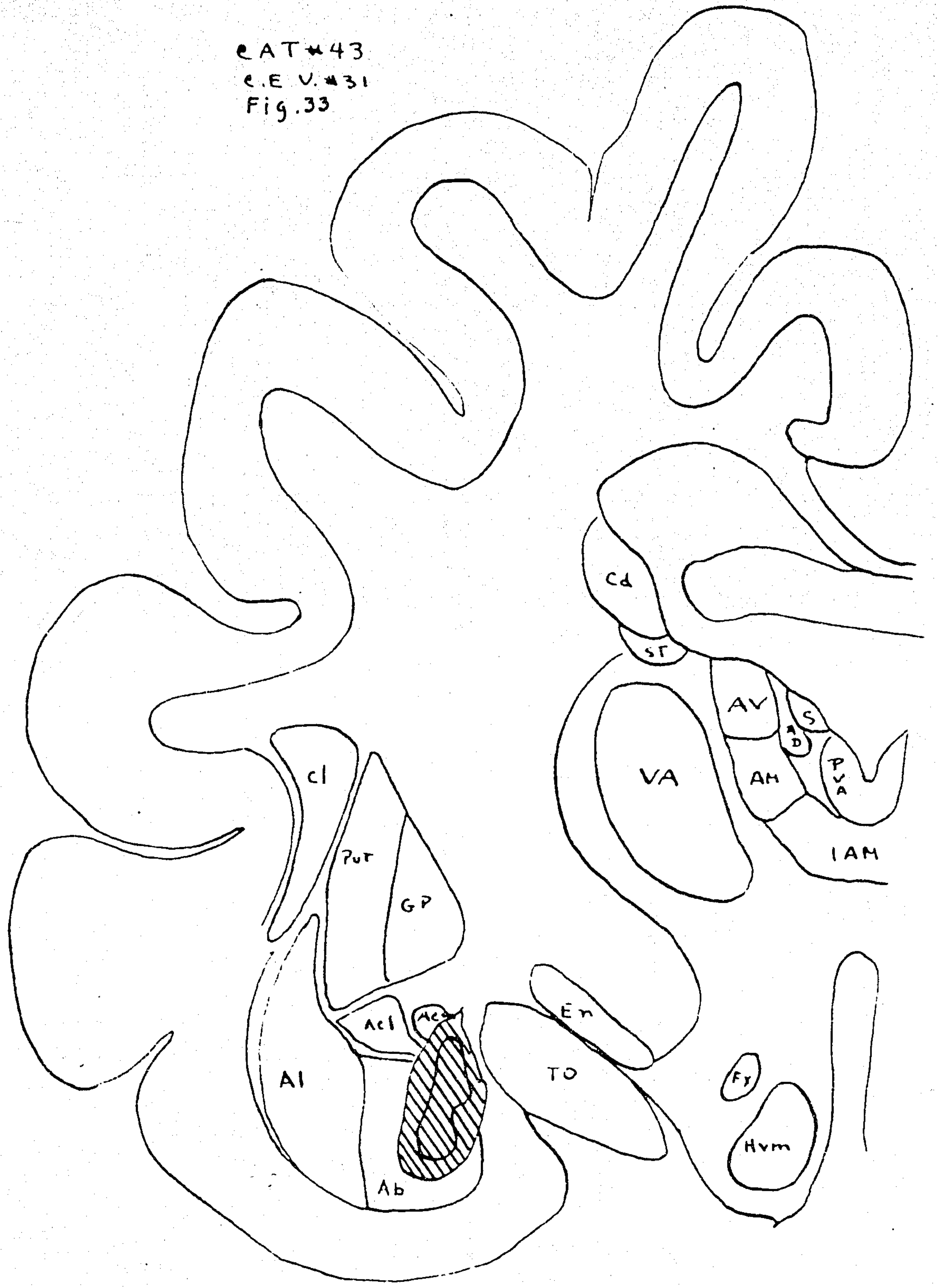
Degeneration in the superior part of the course of the stria terminalis. X700.

Cat No. 43

The lesion in this brain is ellipsoid in shape and occupies the medial half of the basal nucleus and the superior third of the medial nucleus (Fig.33). It encroaches slightly upon nucleus centralis pars medialis. Antero-posteriorly it extends from Frontal 12.0 to 11.0. Its maximum medio-lateral dimension is 1.0 mm., its supero-inferior 2.5 mm. The electrode tract passes vertically through the cortex, the internal capsule, the lateral tip of the optic tract and the stria terminalis.

Dense bundles of degenerated fibres leave the superior, medial and anterior aspect of the lesion. About equal numbers join the longitudinal association bundle and the stria terminalis. Those going to the former join it on its medial aspect and continue with the bundle to the level of the optic chiasma. Here they take a sharp medial bend and continue anteriorly and medially into the region of the nucleus of the anterior commissure and the medial part of the preoptic region. Although a few continue into the anterior commissure, most of them have their preterminals in the nuclei mentioned above. The large bundle that joins the stria terminalis can be followed to the anterior division of the stria. Here some end in the bed nucleus of the stria terminalis but most of them continue anterior to the anterior commissure in the supracommissural bundle. Their preterminals are scattered in the antero-inferior part of the nucleus of the anterior commissure, the medial preoptic region and into the most anterior part of the nucleus ventralis medialis of the hypothalamus as well as the anterior nucleus of the hypothalamus (Fig.34). In the region of the lesion, preterminals are seen in both the basal and cortical nuclei.

CAT #43
C.E.V. #31
Fig. 33



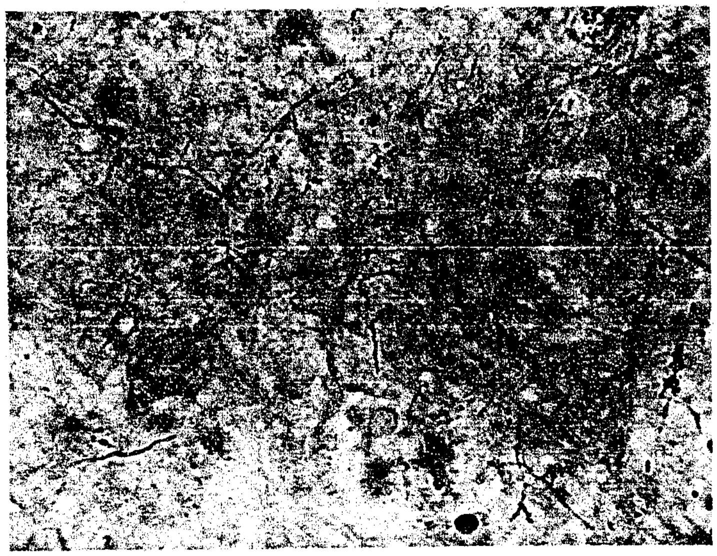


Fig.34: Cat No. 43. Section No. 26. Frontal Plane, Nauta-Gygax Stain.

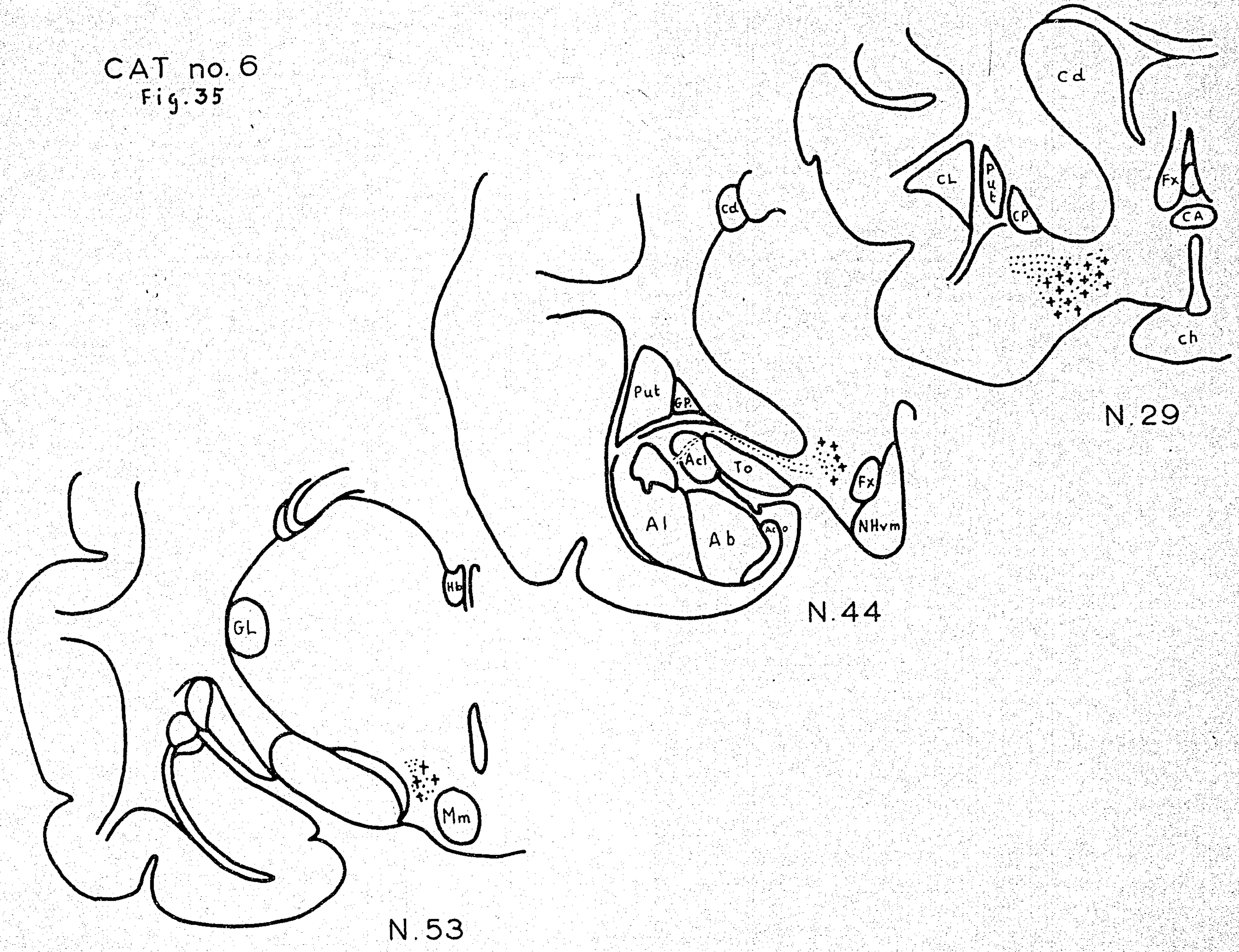
Degenerated preterminals in the anterior nucleus of hypothalamus. X700.

SUMMARY OF OBSERVATIONS

The lateral nucleus has two efferent pathways. These are illustrated in Fig.35. The dots represent degenerated fibres of passage, the plus signs represent preterminals. The smaller of the two pathways is seen in frontal sections as a thin flat band of fibres coursing upwards and medially through the nucleus centralis to a position just inferior to the ansa lenticularis. From here, it continues in an infero-medial direction between the ansa lenticularis and the optic tract to join the medial forebrain bundle. The preterminals of these fibres can be identified throughout the lateral hypothalamus.

The second and larger efferent pathways of the lateral nucleus is the longitudinal association bundle. The only brain in which degeneration to this bundle could not be traced was No. 2. However, in this brain the lesion was small and placed very laterally. Fibres course upwards and medially in the lateral nucleus to enter the longitudinal association bundle on its lateral aspect. They continue in an anterior and slightly medial direction to the level of the optic chiasma. They then curve medially and become more diffusely distributed as they continue antero-medially to the level of the anterior commissure. Their preterminals are located in the medial half of the anterior amygdaloid area and the lateral preoptic region.

CAT no. 6
Fig. 35

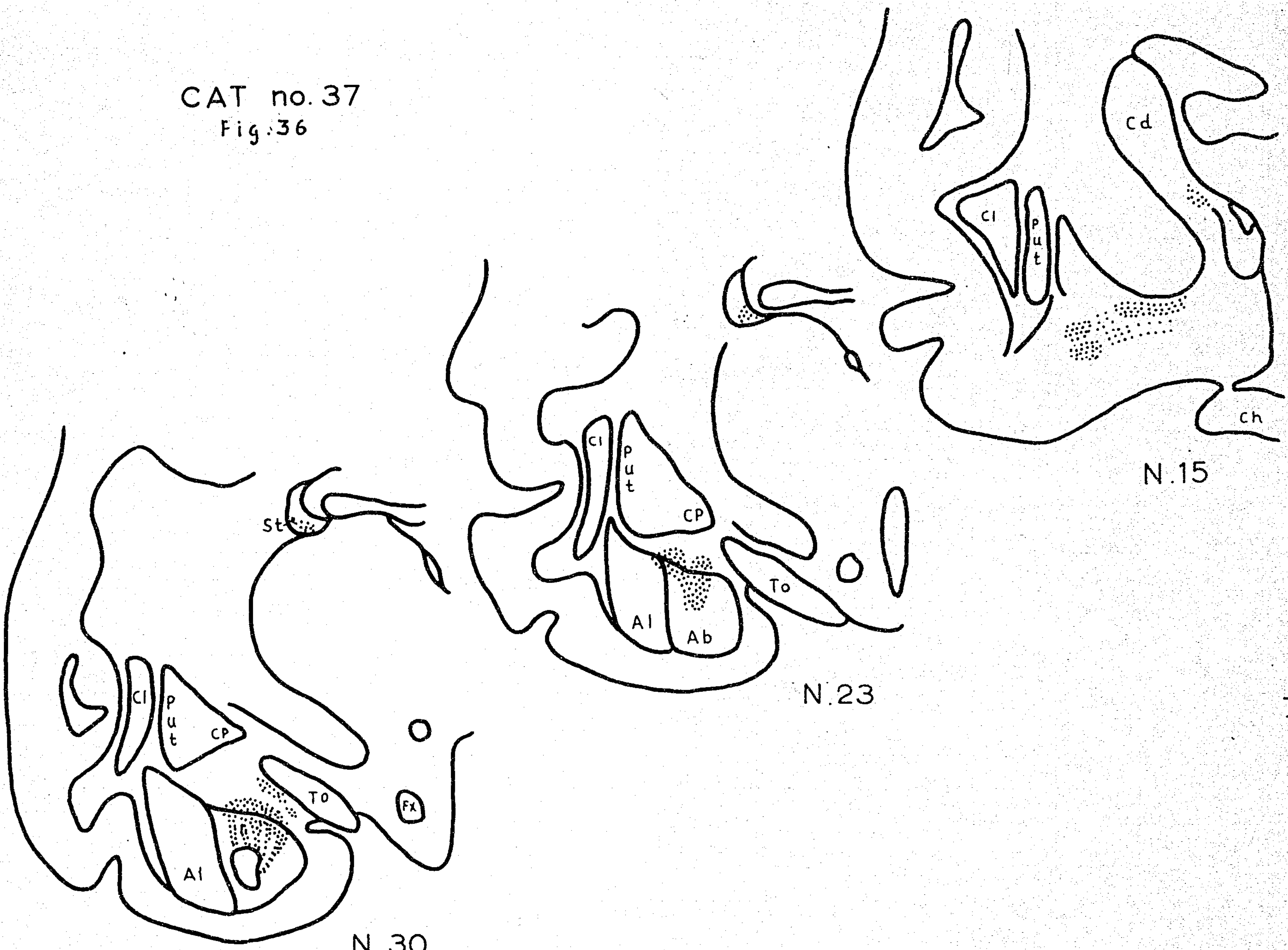


Although degeneration could not be traced into the temporal limb of the anterior commissure, there was some indication (Cat Nos. 1,6,7,21,25) that the lateral nucleus sends fibres into the external capsule. The latter observation requires further confirmation as there is the possibility that these fibres are afferents to the lateral nucleus which were interrupted by the electrode tract.

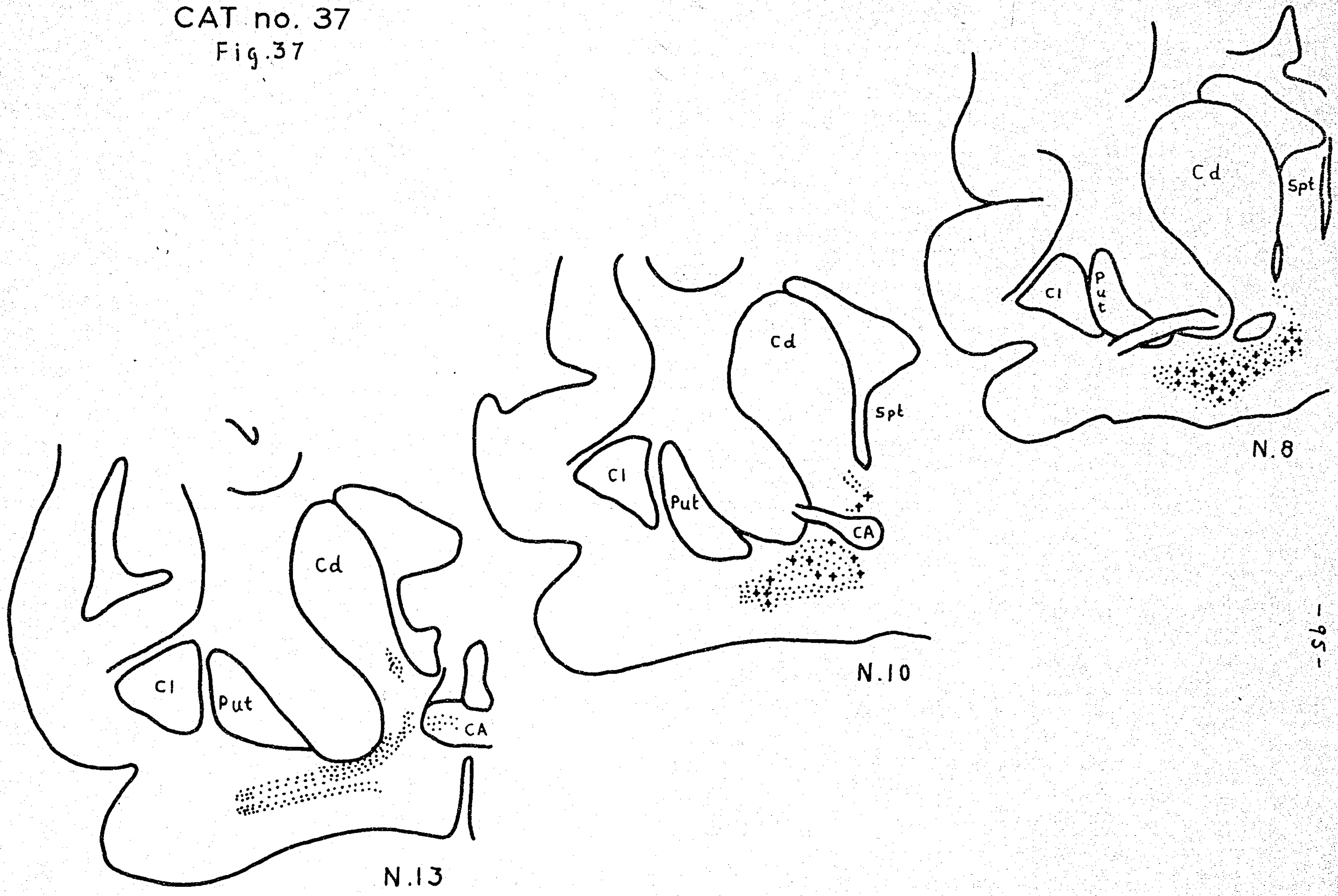
The basal nucleus also has two efferent pathways. (Fig.36 & Fig.37). Small compact bundles of fibres course together in an upward and slightly medial direction. As they approach the superior margin of the nucleus, the more lateral fascicles turn laterally into the longitudinal association bundle, the more medial join the stria terminalis. The latter can be seen coursing in the stria to its point of division just posterior to the level of the anterior commissure. Some fibres terminate here in the bed nucleus of the stria terminalis, while others continue in the supracommissural bundle and terminate in the bed nucleus of the anterior commissure. However, in Cat No. 43, where the lesion involved the most medial part of the basal nucleus and the superior part of the medial nucleus, the degenerated fibres continue further caudally in the supracommissural bundle and terminate in the anterior nucleus and the anterior part of the ventromedial nucleus of the hypothalamus.

The fibres that join the longitudinal association

CAT no. 37
Fig. 36



CAT no. 37
Fig.37



bundle course anteriorly in this fasciculus to the level of the optic chiasma. Here they show a partial division into a lateral and a medial component (Fig.37 N.15). The more medial, which arises chiefly in the medial half of the basal nucleus, curves antero-medially beneath the internal capsule and the majority of its fibres terminate in the medial two-thirds of the preoptic region and the bed nucleus of the anterior commissure. A few continue into the anterior commissure. The more lateral component courses more gradually in a medial direction and terminates in the lateral two-thirds of the preoptic region, the bed nucleus of the anterior commissure, and the substantia innominata (Fig.37). Fibres from the lateral half of the basal nucleus terminate in the lateral and intermediate part of the preoptic region while those from the medial half terminate in the medial preoptic region.

The basal and the lateral nucleus are interconnected by short association fibres. There is some evidence that both the pyriform cortex and the cortical nucleus contribute fibres to the longitudinal association bundle (Cat Nos.23 & 32). Finally, it should be noted that lesions in the basal nucleus cause heavier degeneration than those in the lateral.

CHAPTER IV

DISCUSSION

The observations presented above support, in general, the description of earlier authors concerning the efferent pathways of the basolateral complex of the amygdala. In addition, the size and position of the lesions and the application of the Nauta staining technique has allowed not only a more precise description of the contributions of the basal and lateral nuclei to these pathways but also the identification of their sites of termination.

In agreement with the studies of many other investigators (Johnston, 1923; Humphrey, 1936; Fox, 1940, etc.) the basal nucleus has been found to be one of the nuclei that contributes to the supracommissural component of the stria terminalis. Omukai (1958) reported that in the rabbit this nucleus also contributes to the preoptic component, but in this investigation of the cat, no such contribution has been found. The discrepancy between observations in the rabbit on the one hand and in the cat on the other, can be explained in part by the fact that there is a species difference in the divisions of the stria terminalis. The preoptic component in the rabbit is very large (Young, 1936), but in the cat it is very small (Fox, 1940; Lammers and Magnus, 1955).

Omukai (1958) stated that the supracommissural component could be followed to the bed nucleus of the stria terminalis, the bed nucleus of the anterior commissure, the septal area, and the rostral part of the ventromedial nucleus of the hypothalamus. The present study demonstrates that the contribution of the central and lateral part of the basal nucleus terminates only in the bed nucleus of the stria terminalis and the bed nucleus of the anterior commissure. The fibres from the medial nucleus and possibly those from the most medial part of the basal nucleus terminate in the rostral part of the ventromedial nucleus. This constitutes an anatomical basis for Gloor's report (1955) that in the cat, short latencies are recorded in the ventromedial nucleus only on stimulation of the corticomedial division of the amygdala.

In agreement with the studies of Johnston (1923) in the opossum, Fox (1940) in the cat and Omukai (1958) in the rabbit, it was observed that the lateral nucleus does not have efferent fibres in the stria terminalis. This again supports the work of Gloor, who recorded only long latencies in the stria terminalis on stimulation of the lateral nucleus.

It was mentioned in the introduction that a number of workers have observed fibres from the basal and lateral nuclei entering the temporal limb of the anterior commissure. The present investigation provides no additional information concerning this pathway, as the electrode tract in every case passed through the external capsule.

The longitudinal association bundle is, as Johnston stated in 1923, a main efferent pathway for both the basal and lateral nuclei. Omukai has demonstrated that this is also the case in the rabbit. While the description of the course of this tract is in agreement with that of Fox (1943), it has been found that the lateral component does not remain in the lateral part of the anterior amygdaloid area, but curves medially to terminate in the medial part of the anterior amygdaloid area and the lateral preoptic region. It may also be added that the lateral nucleus distributes to the lateral preoptic region, the lateral part of the basal nucleus to the lateral and intermediate part of the preoptic region and the medial part of the basal nucleus to the medial preoptic region.

Gloor (1955) has reported that on stimulation of the basolateral complex short latencies are recorded in the medial and lateral preoptic regions. On the basis of this study, it would seem probable that these impulses reach the preoptic region by way of the longitudinal association bundle.

The present investigation has also revealed that the lateral nucleus sends fibres directly medially to the medial forebrain bundle, and that these fibres terminate throughout the lateral hypothalamus. At least some of these fibres probably correspond to Fox's "C" bundle (1940). However, there is no evidence to support his statement that these fibres also arise from the basal nucleus. It is interesting to note that similar medially directed fibres have been described in the monkey, (Fox, 1948; Adey et al, 1958; Nauta and Valenstein, 1958). However, the majority of these fibres continue into the thalamus. Nauta and Valenstein (1958) observed a few fibres that remained in a ventral position, but these terminated anterior to the anterior hypothalamus. The difference between the monkey brain and the cat brain in this instance can be accounted for on the basis of the difference in the lesions, and on the basis of species variation.

One important consideration remains to be discussed: are the anatomical observations presented of any assistance in explaining, on a structural basis, the effects of stimulation and ablation of the amygdala? It was noted in the review of the literature that the amygdala is concerned with a wide variety of somatic and autonomic effects that can, for the most part, be related to emotional expression. A number of investigators have tried by stimulation of the individual nuclei, to determine whether these effects (e.g. movement of the face and jaws, gastrointestinal motility) have a specific localization within the amygdala. Although some of these workers (Wood et al, Kaada et al, Koikegami et al) have described such localization either within individual nuclei or within the phylogenetically older and younger nuclear groups, their results are contradictory. Thus on the basis of the physiological data available localization of effect within the amygdala would appear unlikely. This receives some support from the observation that the basal and lateral nuclei are interconnected by short association fibres.

In addition, the sites of termination of the efferent pathways of these nuclei do not suggest a localization of effect. Although the lateral nucleus does send fibres directly into the lateral hypothalamus, they are diffusely scattered throughout its antero-posterior extent. The majority of the projections of both the basal and lateral nuclei end in the preoptic region, an area that is connected with all levels of the hypothalamus through short multisynaptic pathways (Gloor, 1955). This could explain how both parasympathetic and sympathetic effects can be elicited on stimulation of individual nuclei. This type of structural organization would also facilitate the production of complex emotional reactions.

However, it must be remembered that neither the afferent connections of the amygdaloid complex, nor the efferent connections of the corticomedial group have been completely defined and that the contribution of the lateral nucleus to the temporal limb of the anterior commissure requires further investigation.

Only when all these anatomical details are available might it be possible to correlate the effects of stimulation and ablation of the amygdaloid complex with the structural organization.

CHAPTER V

SUMMARY

1. Small circumscribed lesions were placed in either the basal or lateral nucleus of the amygdala in 43 cats.
2. The animals were sacrificed ten days post-operatively and the brains fixed in formalin for at least two months.
3. Serial sections were made with the aid of the freezing microtome. These were stained by the method of Nauta and Gygax. The cresyl echt violet cell stain was used to determine the extent of the lesion.
4. Both the lesion and the impregnation of the degenerated fibres were satisfactory in 16 brains.
5. It was observed that the lateral nucleus has two efferent pathways: the longitudinal association bundle and a more diffuse pathway that extends directly medially to join the medial forebrain bundle. The preterminals of the former are found in the medial part of the anterior amygdaloid area and the lateral preoptic region. Those of the latter are scattered diffusely through the lateral hypothalamus.

6. The basal nucleus also has two efferent pathways: the stria terminalis and the longitudinal association bundle. The preterminals of the former are found in the bed nucleus of the stria terminalis and the bed nucleus of the anterior commissure. Those of the latter are distributed throughout the intermediate and medial part of the preoptic region.
7. The lateral and basal nuclei are interconnected by short association fibres.
8. These observations are in general agreement with the anatomical studies of earlier investigators. In addition they are compatible with the conclusion of Gloor (1955) and Shealy and Peele (1957) that localization of function within the individual nuclei of the amygdala is unlikely.

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