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The Projection Field of the Stria Terminalis in the Rat Brain. An Experimental Study

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ABSTRACT The problem of the stria terminalis projection field has been examined by use of two versions of the cupric-silver technique as well as variations of the Fink-Heimer and Nauta-Gygax techniques applied to material fixed under different conditions using brains from very young rats surviving 30 hours to four days after production of lesions at different levels of the course of the stria terminalis and related structures. The findings are as follows:

(1) A *dorsal* subventricular portion of the stria terminalis divides into retrocommissural and supracommissural contingents which together account for degenerating terminals seen in the ipsilateral bed nuclei of the stria terminalis and of the anterior commissure, and in the medial preoptic-hypothalamic junction area. The supracommissural bundle also disseminates into the laterobasal septum, nucleus accumbens, olfactory tubercle, the posterior and medial divisions of the anterior olfactory nucleus, and the granular layer of the accessory olfactory bulb. Additional fibers end in the paucicellular capsule of the ventromedial hypothalamic nucleus, also in a small lateral parvocellular tuberal nuclear area, and throughout the premammillary nuclei. A small truly commissural division of the dorsal component was traced to the contralateral cortical amygdaloid nucleus and to small clusters of medial amygdaloid cells.

(2) A *ventral* juxtacapsular portion of the stria terminalis was traced to the ipsilateral strial bed nucleus, medial preoptic-hypothalamic junction area, the *entire* ventromedial hypothalamic nucleus, the lateral tuberal area and the premammillary nuclei. The lateralmost fibers of the dorsal strial component as well as those of the ventral component which lie lateral to the "commissural bundle" appear to terminate exclusively in the lateral portions of the bed nucleus of the stria.

(3) A "commissural bundle" or component, after crossing the midline in the anterior commissure, ends in the bed nucleus of the posterior limb of the latter, in the olfactory tubercle, prepiriform cortex, lateral amygdaloid nucleus and the strial bed nucleus. It is thus a decussation rather than a commissure. No contribution from stria terminalis to stria medullaris could be identified.

Behavioral and physiological experiments have indicated that the amygdala is involved in neural processes underlying complex behavioral activities such as food and water intake (Gloor, '60; Goddard, '64; Fonberg, '68; Morgane and Jacobs, '69), emotional expression, defense against danger, sexual reactions and social and learning behavior (Gloor, '60; Ursin and Kaada, '60; Goddard, '64; Hunsperger and Bucher, '67; Thompson, Schwartzbaum and Harlow, '69). It also seems to be concerned with the control of some extrapyramidal and electro-corticographic activities (Kaada, '51; Gloor, '60; Ursin and Kaada, '60; Koikegami, '64; Kreindler and Steriade, '64) as well as of autonomic and endocrine functions (Kaada, '51; Gloor, '60; Ursin and Kaada, '60; Goddard, '64; Koikegami, '64; Eleftheriou et al., '69; Lawton and Sawyer, '70).

The characteristics of most of these amygdalar effects suggest a widespread pattern of projection from this neuronal aggregate into brain structures — the hypothalamus most particularly — dealing with the above listed functions. In line with this, the stria terminalis, one of the

main efferent pathways of the amygdala, deserves particular attention, since its stimulation or severance has been shown to affect respiration (Kaada, '51), blood pressure (Koikegami, '64), cortically and reflexly induced movements (Kaada, '51; Koikegami, '64), affective defense reactions (Hunsperger and Bucher, '67), male sexual activity (Giantonio, Lund and Gerall, '70) and hormonal secretions (Critchlow and Bar-Sela, '69; Kawakami, Seto, Terasawa, Yoshida, Miyamoto, Sekiguchi and Hattori, '69; Velasco and Taleisnik, '69).

In view of these reports, it seems imperative to develop precise morphological data with respect to the spatial organization and field of distribution of the amygdalofugal components of the stria terminalis. However, until very recently, the numerous anatomical studies carried out with normal brain material have not provided reliable data in these contexts. Experimental approaches in which the Marchi, Glees and Nauta-type techniques for staining Wallerian degeneration were applied have not only given conflicting results but have also failed to disclose some very important aspects of the terminal distribution of this bundle. Consideration of the most pertinent contributions will be included in the discussion.

Introduction of the Fink-Heimer method for tracing degenerating axons and their terminals (Fink and Heimer, '67), and its further development for combined optic and electron microscopic observations (Heimer, '69) has overcome in some measure the limitations of the staining techniques just mentioned. As a result, Heimer and Nauta ('69) have been able to provide a more complete picture of the hypothalamic distribution of the stria terminalis and of its synaptic organization. More recently, Leonard and Scott ('71), using the same procedure, provided further support to the findings of these authors and in addition, showed that there is an intrinsic arrangement within the stria which correlates with the organization of the amygdala. However, in spite of these developments it has been possible to show still wider diencephalic and telencephalic terminal distributions for some fiber contingents of the stria by using the cupric-silver method developed in this laboratory (de Olmos, '69; de Olmos and Ingram, '71) for staining fibers and terminals undergoing experimental Wallerian degeneration. A description of the findings thus obtained will be the basis of this report.[1]

MATERIAL AND METHODS

The brains of 50 rats weighing between 47 to 140 gm were used. A variety of electrolytic and direct surgical lesions were placed unilaterally at different levels of the stria terminalis (fig. 1). Control lesions were made at various loci of the piriform cortex, Ammon's horn, fimbria fornicis and neocortex.

All operations were performed under ether anesthesia. Several surgical approaches were used but in most cases the lesions were placed stereotactically with a Kopf instrument using usually Bernardis' ('67) coordinates or sometimes, for different coronal planes, those of DeGroot ('59) and König and Klippel ('63). In several cases, the electrode was directed at an angle of 25–30° to a paramedian sagittal plane passing through the amygdaloid complex in order to avoid electrode track injury to the limbic cortex and hippocampus.

Electrolytic lesions were produced by passage of d.c. anodal current of 0.8 to 1.2 mA for 6 to 10 seconds. The electrodes were stainless steel wires less than 0.25 mm in diameter and insulated except for a few fractions of a millimeter at the tip.

In other experiments the stria terminalis was totally or partially interrupted mechanically after the dorsal hippocampus and overlying neocortex were removed by aspiration. In some instances the lesions were made through a ventral parapharyngeal approach using a small ophthalmic knife.

For control purposes, the hippocampus, overlying neocortex and the fimbria fornicis were aspirated with varying degrees of involvement of the cingulum fibers. Other controls were provided by making electrolytic lesions in the dorsal hippocampus

[1] The material presented here is largely based upon a thesis accepted in partial fulfillment of the requirements for the Ph.D. degree in The University of Iowa, see de Olmos, J. S. ('71).

and/or the fimbria fornicis as well as in the ventral hippocampal-subicular formation.

For lesions of the piriform cortex either by aspiration or electrolysis, the temporal approach of Powell, Cowan and Raisman ('65) was used.

Although in many instances the hemisphere contralateral to the lesions could be used as a control, normal brains originally stained for the demonstration of granular argyrophilic cells and their processes and in which no degeneration was present were also utilized for checking purposes.

After survival periods varying from 30 hours to four days, the animals were anesthetized and the brains in most cases perfused according to procedures previously outlined (de Olmos, '69). However, in later experiments it was found that the use of 4% paraformaldehyde in 0.067 M sodium cacodylate buffer (pH 7.2–7.4) containing 4% sucrose for the perfusion and subsequent postfixation steps, replaces to advantage the phosphate buffered paraformaldehyde solution previously recommended since the sections subsequently made have cleaner backgrounds, which facilitates the interpretation of degenerating areas.

Following removal and further fixation for two to six days, the brains were soaked for at least three days in a buffered gum-sucrose solution containing 0.88 M sucrose and 1% gum acacia. Frozen sections were then cut transversely, sagittally or horizontally at 20–30 μ depending upon the staining procedure to be used. The sections were collected serially in vials containing the perfusion solution and kept there for one to three days. Subsequently, most of them were stained with the cupric-silver method (de Olmos, '69) and the remainder, in an alternating way, by modifications of the Nauta-Gygax ('54) and Fink-Heimer ('67) techniques adapted to the present material. However, sections from later experiments were stained with a more recently developed modification of the cupric-silver method which has the advantage over the original version of giving sharper images of terminal degeneration and at the same time reducing greatly the staining of granular argyrophilic neuropil. This procedure has recently been described (de Olmos and Ingram, '71), and involves, besides careful control of the pH of the fixing fluid, use of acetone after impregnation with the cupric-silver mixture. While there is reduction of impregnation of normal argyrophilic neuropil, degenerating terminals are stained as discussed in the next paragraph.

The original cupric-silver method and its present modification belong to the category of silver impregnation procedures which stain the so-called "dust like" terminal degeneration. As such they cause particulate deposits of reduced silver which appear to mark nerve fiber terminals in early stages of the degeneration caused by axotomy. However, in contrast to other procedures, the modified cupric-silver method tends to show that at high magnifications the apparently solid spheroidal structures forming the "dust-like" degenerating terminals are actually ring-like in profile. This ring-like appearance plus the occasional presence of short tail-like lengths of axons attached to these structures makes them resemble the "boutons terminaux" of the neurofibrillar stains. This feature, together with others discussed elsewhere (de Olmos, '69) offers evidence that the cupric-silver methods actually stain terminal degeneration. A problem of interpretation may remain because of the

Abbreviations

aAC, anterior limb of the anterior commissure
AC, anterior commissure
Acc, nucleus accumbens septi
Acc. Olf. B., accessory olfactory bulb
Aon, anterior olfactory nucleus (m = aom; p = aop)
aod, anterior olfactory nucleus, pars dorsalis
aom, anterior olfactory nucleus, pars medialis
aop, anterior olfactory nucleus, pars posterior
a. prg. 25, area corticalis praegenualis 25
arc, arcuate nucleus
art, artifact
BL, basolateral amygdaloid nucleus
Bm, basomedial amygdaloid nucleus
bac, bed nucleus of the anterior commissure
bst, bed nucleus of the stria terminalis
CA, cornu Ammonis
Ce, central amygdaloid nucleus
CC, corpus callosum
Co, cortical amygdaloid nucleus
Coc, cortical amygdaloid nucleus, caudal portion
CST, commissural component of the stria terminalis
DG, dentate gyrus
dpm, dorsal premammillary nucleus
DST, dorsal strial component

Abbreviations

Ext. C, external capsule
f, convolutions of olfactory tubercle
Fi, fimbria fornicis
Fx, columna fornicis
GCC, genu corporis callosum
GP, globus pallidus
gr, internal granular layer of accessory olfactory bulb
Hb, habenula
hl, lateral hypothalamic area
hr, hypothalamic radiation of the supracommissural division of the dorsal strial component
ic, intercalate masses
IC, internal capsule
iCa, islands of Calleja
ipac, interstitial nucleus of the posterior limb of the anterior commissure
L, lateral amygdaloid nucleus
lpo, lateral preoptic area
LOT, lateral olfactory tract
LS, lateral septal nucleus
Lv, lateral ventricle
MB, mammillary body
MCH, medial cortico-hypothalamic tract
ME, median eminence
Me, medial amygdaloid nucleus
Me c, medial amygdaloid-nucleus, caudal portion
Me r, medial amygdaloid nucleus, rostral portion
mph, medial preoptic hypothalamic junction area
Ntol, nucleus of the lateral olfactory tract
OCh, optic chiasma
Olf. B., main olfactory bulb
OT, optic tract
ov, olfactory ventricular cleft
pAC, posterior limb of the anterior commissure
Pam, periamygdaloid cortex
PCH, precommissural hippocampus
Ped, pedunculus cerebri
pfh, perifornical hypothalamic area
pm, premammillary area
pr, parolfactory radiation of the supracommissural division of the dorsal strial component
Rtc, retrocommissural division of the dorsal strial component
S, subiculum
SM, stria medullaris thalami
Spc, supracommissural division of the dorsal strial component
ST, stria terminalis
Str, striatum
Th, thalamus
tl, nucleus tuberis lateralis (Diepen, 1962)
TuO, olfactory tubercle
TuOm, olfactory tubercle, medial
Tz, amygdalopiriform transitional zone
vm, ventromedial hypothalamic nucleus
vs, blood vessel
VST, ventral strial component
25, area praegenualis 25

Structures of the prepiriform areas and olfactory tubercle:

Ia, sublamina supratangentialis of the plexiform layer
Ib, sublamina tangentialis of the plexiform layer
II, superficial pyramidal-celled layer
III, deep polymorph-celled layer
Plx, external plexiform layer or lamina zonalis or molecular layer
Ply, polymorph layer
Pyr, pyramidal layer
51a, area praepiriformis 51a
51e, area praepiriformis 51e

Fig. 1 Diagrammatic representation on frontal sections of 18 of the most representative cases carrying lesions which damaged the stria terminalis totally or partially at different levels of its course, or involved adjacent brain areas which are used as controls. These lesions are represented at the level of their maximum diameter only and are differentiated by numbers and by the shading code shown at the lower portion of the figure which represents the patterns of degeneration induced by the various lesions.

Accordingly, the lesions numbered 1 and 2 (cases STH 1 and 2) involved the stria terminalis totally or partially, respectively, and the lesions numbered from 3 to 6 (cases STb 1, AF 7, 25 and 1) produced degeneration of the dorsal strial component. Those numbered from 7 to 13 involved either the whole of the ventral strial component or portions of it and an example of the first instance is given by lesion 11 (case AF 12). Lesion 9 (case AF 24) illustrates the type of case in which the medial sector of the ventral strial component underwent Wallerian degeneration, while lesions 8, 10, 12 and 13 (cases AF 17, 26 and 13, and AS 4) involved degeneration in the lateral sector of the ventral component. In lesions 10 and 13, the portions of periamygdaloid cortex and of the external capsule affected by the damage are separated from the remainder of the lesion by broken heavy lines since these structures do not appear to contribute to the degeneration pattern under consideration. The cases illustrated by numbers 7 and 14 (cases Stb 2 and HLF 1) are examples of lesions for controlling the validity of findings after coagulations in the anterior part of the corticomedial amygdala (lesions 9 and 11), since the latter type of lesion quite regularly encroaches upon the so-called ventral amygdalofugal pathways. Also, as is shown in figure 1d, lesion 9, the so-called "commissural" component of the stria terminalis is often involved by these intraamygdaloid lesions.

Lesions 15 in e, 16 in f, 17 in g and 18 in h (cases Str 1, FxF 4, AF 6 and CAF 2) illustrate the controls designed to establish the validity of the existence of a parolfactory radiation arising from the dorsal strial component. As discussed in the text such lesions do not produce a pattern of terminal degeneration like that found after degeneration of this parolfactory radiation and, more particularly, none of them evoke terminal degeneration in the internal granular layer of the accessory olfactory bulb.

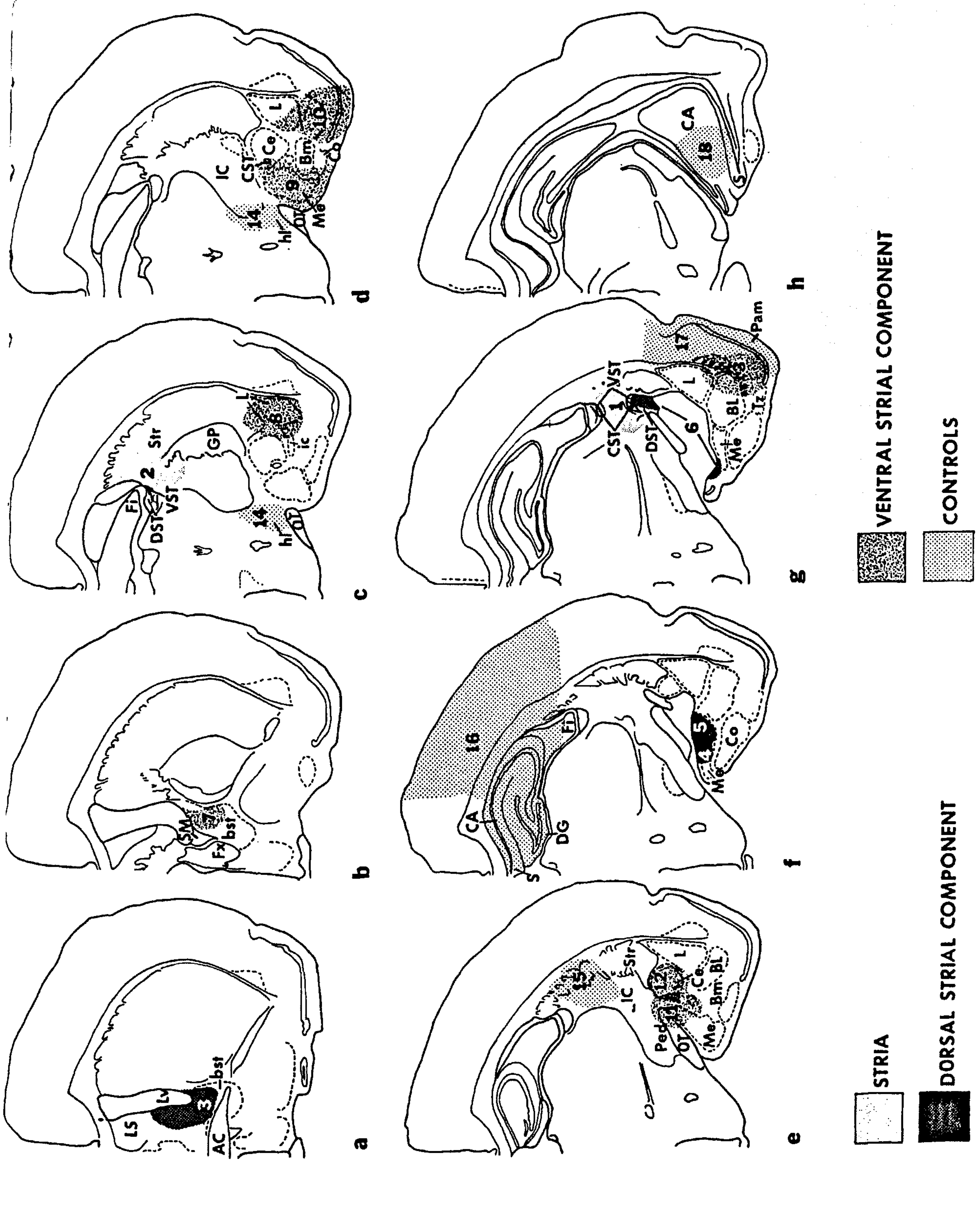
a
b
c
d
e
f
g
h
STRIA
DORSAL STRIAL COMPONENT
VENTRAL STRIAL COMPONENT
CONTROLS

Figure 1

variable occurrence of irregular, mainly extracellular, silver deposits in the olfactory bulbs — especially in the glomerular layers of normal and experimental brains. This type of pseudodegenerative picture can be distinguished from true degeneration because of its erratic character, lack of association with degenerating fibers and relationship to the sites of lesions.

RESULTS

The following account is based on the examination of sections obtained from brains bearing lesions at different levels of the course of the stria terminalis, which were produced either by electrolytic coagulation or by direct surgical transection and employing various approaches.

In many instances in which electrolytic lesions were made, the thin tracks caused by passage of the fine electrodes traversed various portions of neocortex, dorsal hippocampus, fimbria fornicis, internal capsule and lateral geniculate body, or passed through neocortex, upper portions of the external capsule and inferior portions of the striatum. Wallerian degeneration was induced in the fibers interrupted in this way, but such fibers were so few in number and so sparsely distributed that they will not be considered further. Besides, direct lesions limited to the structures ordinarily found in the electrode paths (fig. 1, e15, f16) showed in general little if any contribution from them to the patterns of fiber degeneration to be described. Such coincidental degeneration is in general readily distinguishable from that caused by lesions of the stria proper.

Since complete correlation between the various descriptions of the components of the stria terminalis given in the literature and the findings reported here is beset with difficulties, the following account will refer to these components according to their relative positions within the supracapsular portion of the looped course of the stria. Thus the stria may be considered as constituted of three parts: (1) a dorsal or subventricular component, (2) a ventral or juxtacapsular component and (3) a commissural component. It should be stated that the effects of total unilateral lesions of the stria (4 cases, see fig. 1g, 1) will not be described here, since such lesions usually involved extrastrial structures. The descriptions presented below, derived from many lesions of varying extent, encompass all results which might be expected from total lesions.

I. *The dorsal component.* Irrespective of the type of approach used, lesions of the dorsal subventricular half of the stria terminalis in 21 cases (examples, see fig. 1f, 4, 5, and g, 6) produced massive terminal degeneration in the bed nucleus of the stria (figs. 2, 8). This is particularly heavy in the anteromedial portion of the nucleus and less marked in the lateral. The oral part of the nucleus receives degenerating branches from a *supracommissural* division of the main dorsal bundle while the caudal portion is supplied by a smaller *retrocommissural* division of the latter (fig. 2).

Fibers from the supracommissural contingent also feed the rostral part of the bed nucleus of the anterior commissure while a small *commissural* division accounts for both fine and coarse degeneration in a dorsal stratum of the commissure and in the corresponding part of its bed nucleus. Some of the latter fibers swing dorsolaterally and appear to distribute degenerating terminals to a small central area of the contralateral bed nucleus of the stria. However, most of the crossed fibers continue caudally in the contralateral stria terminalis immediately dorsal to the latter's commissural component and reach the amygdaloid region. Once there they traverse a thin strip of medial amygdaloid cells to which they probably contribute some terminal degeneration and finally end in the caudal one-third of the contralateral cortical amygdaloid nucleus (fig. 3).

Anterior to the anterior commissure the supracommissural division divides into two main ipsilateral streams: *parolfactory* and *hypothalamic*.

The *parolfactory radiation* fans out toward the parolfactory region of the brain, supplying specific areas of septal, olfactory tubercle and retrobulbar formations. In the septal area terminal degeneration spreads out on the basal aspect of the lateral septal nucleus (figs. 2, 4) and continues into the medial portion of the nucleus accumbens septi (figs. 2, 4, 5).

From this general area of terminal de-

Fig. 2 Photomicrograph of a sagittal section through the bed nucleus of the stria terminalis (bst) to show the distribution of the supracommissural (Spc) and retrocommissural (Rtc) divisions of the dorsal strial component as indicated by the terminal degeneration in this nucleus. Modified cupric-silver method. × 27.

The terminal degeneration in the basal part of the lateral septal nucleus (LS) and in the nucleus accumbens septi (Acc) belongs to the parolfactory radiation of the supracommissural division. The arrows indicate the boundaries of the terminal degeneration fields.

generation, a diffuse stream of argyrophilic debris apparently representing axon-stem degeneration, passes rostrally along the sulcus limitans septi. Some of these degenerating elements incorporate into the ventral forceps of the corpus callosum and might account for terminal degeneration found in the deep layer V of the cortical area praegenualis 25 (fig. 6). The majority of these elements, however, pass forward at the ventromedial border of the olfactory ventricular cleft (figs. 6, thick arrow; 7) and along the olfactory peduncle where they form a loose stratum deep to the partes posterior and medialis of the so-called anterior olfactory nucleus. This offshoot of the parolfactory radiation accounts apparently for terminal degeneration found in the pars posterior of the anterior olfactory nucleus (fig. 4) and also in the deep polymorph-celled layer of the dorsal portion of its pars medialis (fig. 7). Other fibers of the parolfactory radiation branch off and scatter between the precommissural hippocampus and the pars medialis of the anterior olfactory nucleus and also, farther rostrally, between the dorsal and ventral portions of this latter gray mass (fig. 7, thick arrow). These appear to account for degenerating terminals found in the external plexiform layer of the dorsal portion of the pars medialis of the anterior olfactory nucleus (fig. 7). These degenerating terminals are found to be confined to the deep zone of the plexiform layer without any clear sign of termination in the cellular stratum immediately deep to it. Finally, a further stream of coarser degenerating elements reaches the accessory olfactory bulb and ends in its internal granular layer (fig. 8a; for control see fig. 8b).

The pattern of distribution of the parolfactory radiation in the medial formations of the olfactory peduncle appears to fit with the terminology of their control stratification [2] suggested by several authors (see Rose, '12; Krieg, '46; Vaz Ferreira, '51; White, '65). Accordingly, the deep main polymorph-celled layer III and the inner sublamina Ib of the external plexiform layer [3] which contains dendrites of deep-lying neurons, are receptors of this projection. On the other hand, the superficial pyramidal-celled layer II and outer plexiform sublamina Ia [4] of this region do not receive such connections.

The fibers supplying the olfactory tubercle descend from the field of degeneration in the nucleus accumbens and pass through the stream of normal fibers which separates the tubercle from the nucleus accumbens to reach the pars medialis of the former. Here, terminal degeneration is profusely disseminated among the cell bodies of the pyramidal cell layer with the heaviest concentration on the deep side (fig. 5).

The islands of Calleja, small clusters of

[2] Areas praepiriformis 51e and praepiriformis bulbaris 51g.
[3] The sublamina tangentialis Ib of area 51e.
[4] The sublamina supratangentialis Ia of area 51e.

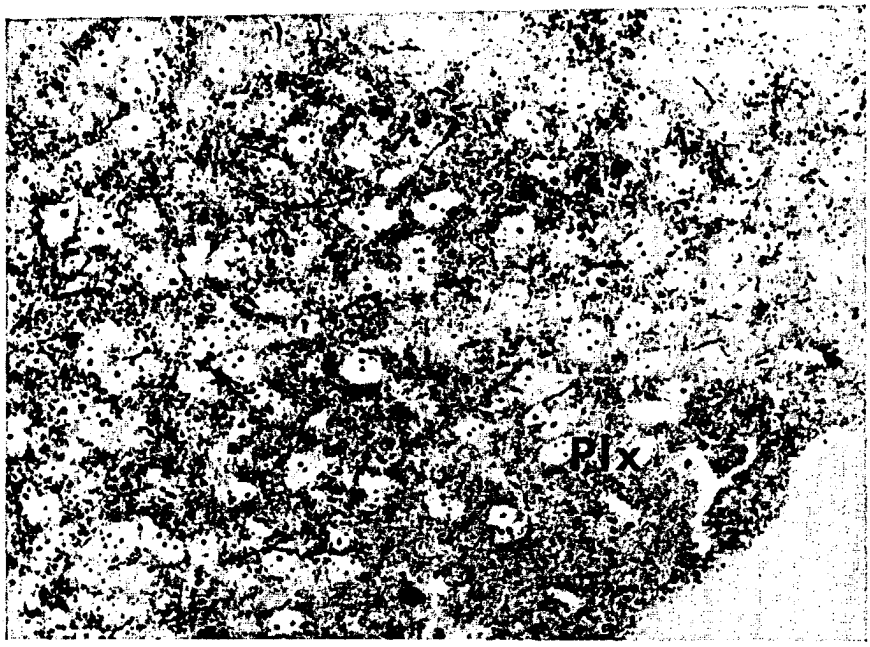

Fig. 3 Photomicrograph of a frontal section through the caudal pole of the amygdala to show terminal degeneration in the cortical amygdaloid nucleus contralateral to lesions damaging the dorsal strial component. Original cupric silver method. × 360.
Note that the degenerative changes are confined to the cellular portion of the nucleus, sparing its plexiform layer (Plx).

granule cells in the depths of the polymorph layer as well as superficially located groups, and the larger so-called medial island of Calleja, remain free of argyrophilic granules (fig. 5, iCa) except for some invasion of their peripheral portions. Conversely, an extended island of medium-sized cells paralleling very closely the medial surface of the "medial island of Calleja," shows profuse coarse terminal degeneration freely dispersed among its cell bodies.

The *hypothalamic radiation* of the supracommissural division curves around the rostral aspect of the anterior commissure and passes ventrocaudally dorsal to the medial suprachiasmatic region. Here, it contributes a heavy but diffuse terminal degeneration which fills a central and dorsal ovoid area which is included in the medial preoptic and anterior hypothalamic nuclei (to be called here the medial preoptic-hypothalamic junction area, mph in fig. 2) leaving the anterior and posterior poles of these nuclei free. This contribution from the supracommissural division overlaps that coming from the retrocommissural division which supplies only the laterocaudal sector of this strial projection field.

An important part of the supracommissural-hypothalamic system extends still farther and reaches the basal hypothalamic region. After supplying some terminals to the retrochiasmatic area, it disperses on the periphery of the ventromedial hypothalamic nucleus, accounting for the heavy fine and coarse terminal degeneration which encapsulates this cell group (vm in figs. 2, 9). A close examination reveals that this picture is not exclusively confined to the cell-poor capsular area of the ventromedial nucleus but also involves the more peripheral cell strata as well as granular argyrophilic neurons (de Olmos, '69) which border the dorsal aspect of this capsule, and also a small-celled tuberal region ventral to the fornix which was designated nucleus tuberis lateralis by Diepen ('62) perhaps because of its location relative to the primate nuclei of the same name (see also Christ., '69).

In addition, degenerating fragments pass farther caudally via the ventral portion of the ventromedial nuclear capsule and terminal degeneration fills the ventral premammillary nucleus. The ventro-lateral part of the dorsal premammillary nucleus also contains such degeneration. The arcuate, other periventricular nuclei and the

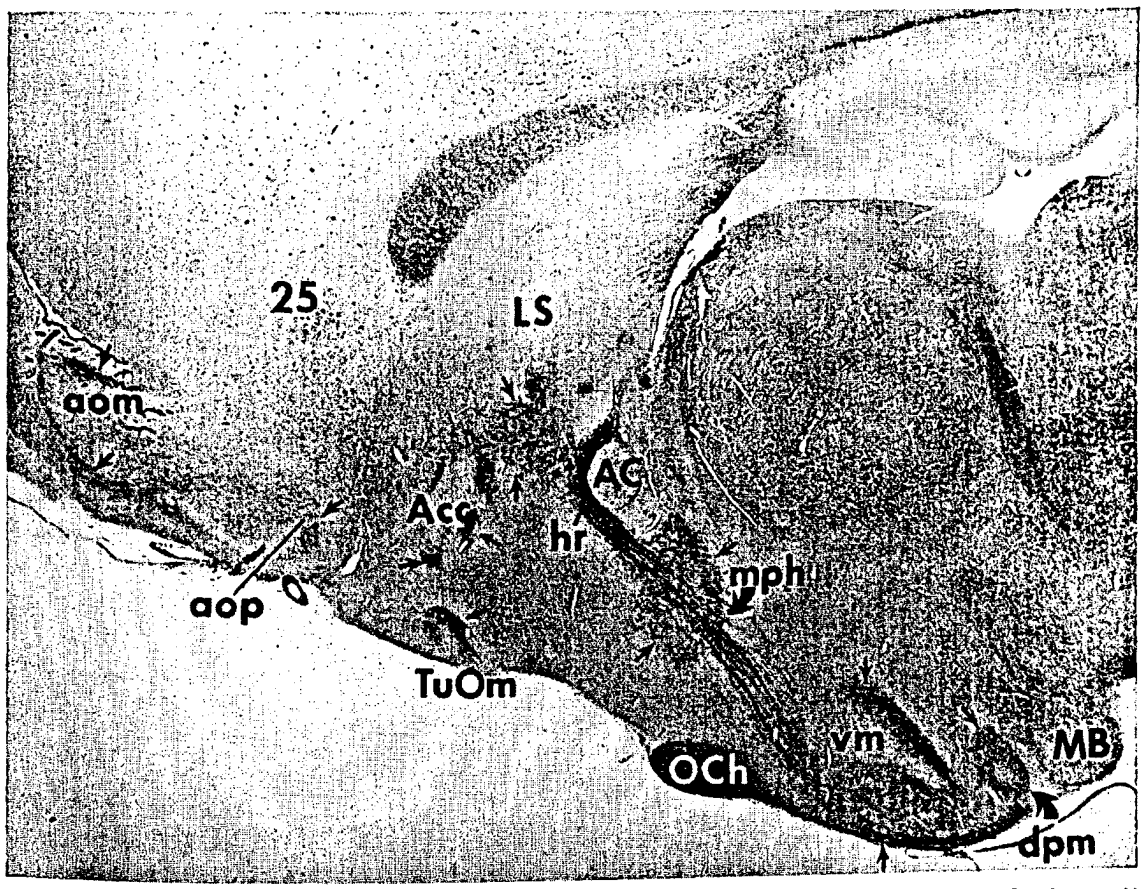

Fig. 4 Parasagittal section through the forebrain of a young rat to show the distribution pattern of terminal degeneration after lesions confined to the dorsal strial component. Modified cupric-silver method. × 16.

The terminal degeneration visible in the basal part of the lateral septal nucleus (LS), in the nucleus accumbens septi (Acc), in partes posterior (aop) and medialis (aom) of the anterior olfactory nucleus and pars medialis of the olfactory tubercle (TuOm) comes from the parolfactory radiation of the supracommissural division. The terminal degeneration in the medial preoptic-hypothalamic junction area (mph), in the capsule surrounding the ventromedial hypothalamic nucleus (vm) and in the dorsal premammillary nucleus (dpm) marks the distribution of its hypothalamic radiation (hr).

dorso-medial hypothalamic nucleus do not receive terminals from the supracommissural-hypothalamic system.

As noted before, the degenerating *retrocommissural* fascicle of the dorsal component distributes massive but diffuse terminal degeneration around the cells of the retrocommissural part of the bed nucleus of the stria (fig. 2). The remaining degenerating fibers of this fascicle gain the dorsolateral aspect of the medial preoptic-hypothalamic projection field (mph) where their terminal arborizations overlap to some extent those of the supracommissural-hypothalamic radiation. No contribution from this retrocommissural division to the supply of other hypothalamic cell aggregations has been identified.

Furthermore, partial lesions which involved fiber contingents running either in medial or lateral segments of the dorsal strial component disclose a medial-lateral organization within it. Accordingly, more medially placed lesions (fig. 1f, 4) which provoked sparse terminal degeneration in the nucleus accumbens septi, olfactory tubercle and the parvocellular "nucleus tuberis" region accounted for strong degeneration at the level of the granular layer of the accessory olfactory bulb and also in the medial hypothalamic nuclei. More laterally placed lesions (fig. 1e, 2) revealed a reverse pattern. Moreover, damage to fiber bundles in the lateral margin of the dorsal component provoked degeneration of a small bundle of fibers which passes

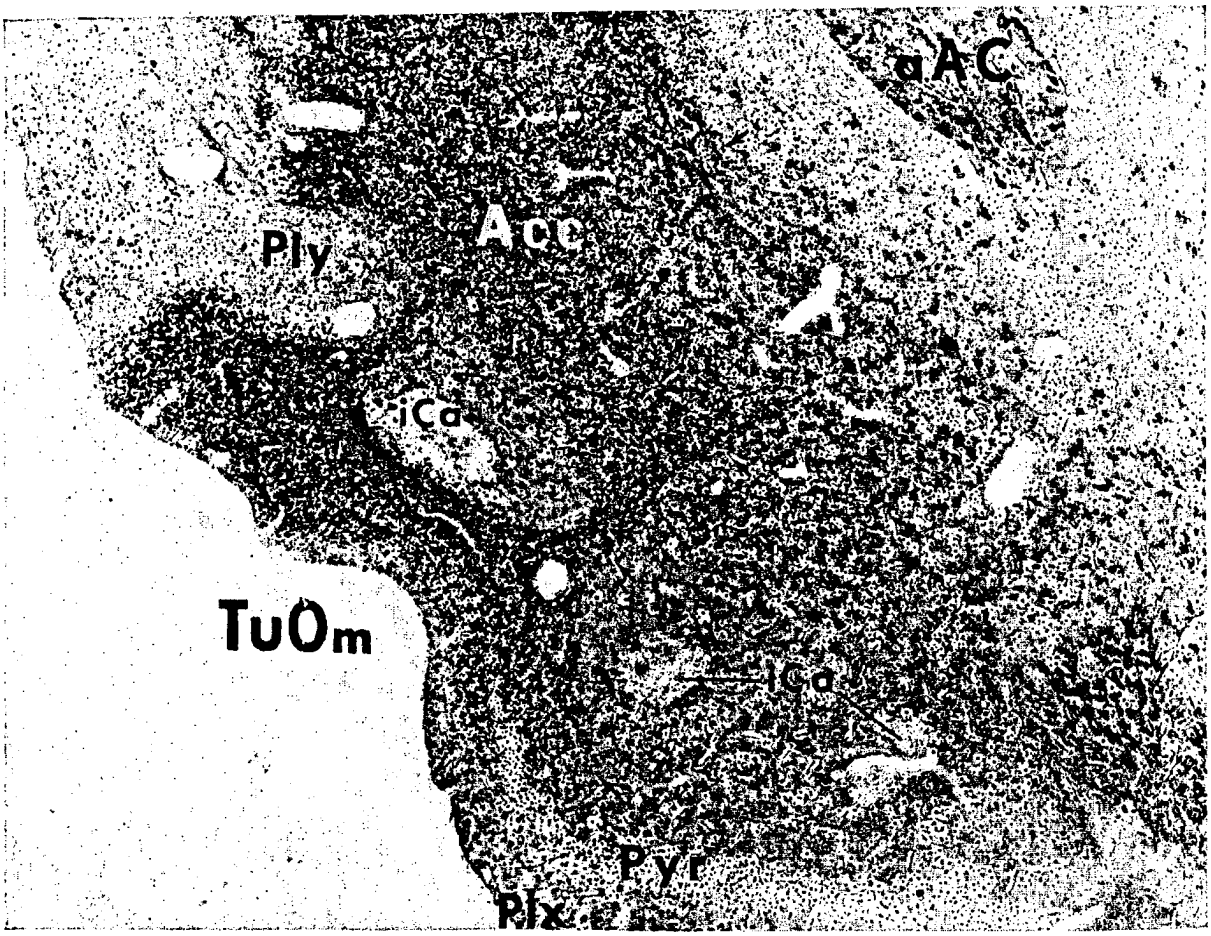

Fig. 5 Photomicrograph of a frontal section through the parolfactory region to show the distribution pattern of terminal degeneration after a lesion confined to the dorsal strial component. Modified cupric silver method. × 108.

The degenerated terminals are located in the pars medialis of the olfactory tubercle (TuOm) and in the nearby nu. accumbens septi (Acc). Note the foldings of the pyramidal cell layer (Pyr) and related degenerating terminals. The polymorph layer (Ply), the islands of Calleja (iCA) and plexiform layer (Plx) are free of such terminals.

immediately behind the anterior commissure near the medial edge of the internal capsule to end in the ventrolateral portion of the bed nucleus of the stria terminalis. Perhaps this bundle represents Johnston's ('23) infracommissural component (or bundle 3) of the stria terminalis.

2. *The ventral component.* Since the interruption of this component in most parts of its extraamygdaloid course necessarily causes coincidental damage to other components, more particularly the dorsal strial component, lesions were placed in 17 cases within the amygdala. The lesions involved in varying degree, together or singly, different amygdaloid nuclei and all or portions of the ventral strial component. Examples of these are given in figures 1c, 8; d, 9 and 10; e, 11 and 12; and g, 13. The ventral strial component was in addition damaged at the level of its course through the retrocommissural portion of the bed nucleus of the stria terminalis (figs. 1b, 7) as a way of checking the results obtained with the intraamygdaloid lesions described for figure 1.

From such an approach a degeneration picture develops which differs in some important features from those described by other workers.

Accordingly, the bulk of this fiber system is seen to radiate downward and caudally among the cells of the retrocommissural portion of the bed nucleus of the stria. It courses medially close to the dorsomedial border of the medial forebrain bundle without contributing to the latter and disseminates into the medial preoptic-hypothala-

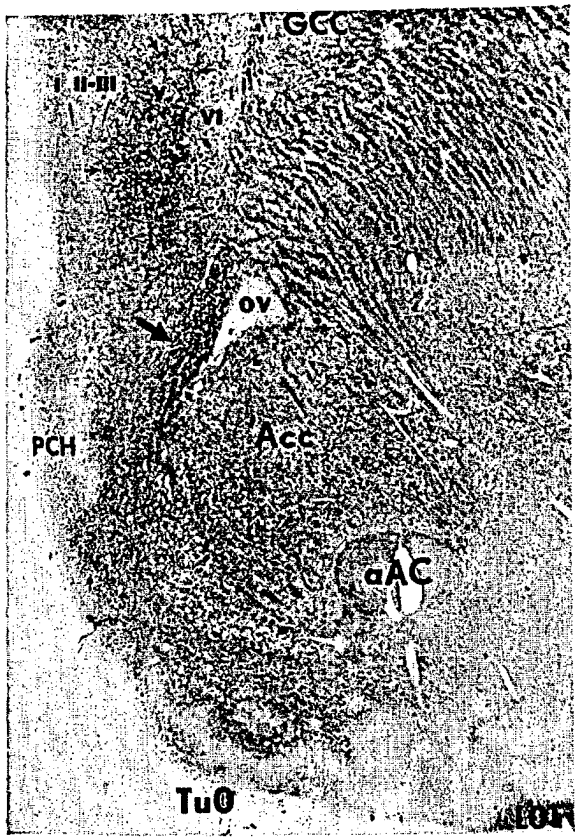

Fig. 6 Photomicrograph of a frontal section to show the distribution of degenerating terminals and fibers of the juxtaventricular offshoot (large arrow) of the parolfactory radiation. Modified cupric-silver method. × 17.

Note that the terminal degeneration in the area corticalis praegenualis 25 (25 in fig. 4) is confined to its layer V.

mic projection field (mph, figs. 11, 12). Along its course it distributes a heavy diffuse terminal degeneration to the retrocommissural bed nucleus of the stria except for a small rostromedial portion. This degenerative picture extends throughout the medial preoptic-hypothalamic field and is particularly heavy in its lateral and ventral aspects, from which a further stream of fiber fragments reaches the basal tuberal region. There the terminal degeneration covers uniformly the parvocellular tuberal gray beneath the fornix (Diepen's nucleus tuberis lateralis; see fig. 11), the dorsal part of the retrochiasmatic area, the dorsal and ventral premammillary nuclei, and also, in contrast with what happens after degeneration of the supracommissural-hypothalamic division, the *whole area* of the ventromedial hypothalamic nucleus (figs. 10, 12). Significantly, there is no degeneration in the lateral septal nucleus.

It has been said that degeneration appearing in the ventromedial hypothalamic nucleus may be due to coincidental damage of amygdalofugal pathways which pass by a ventral route to reach that nucleus (Szentágothai et al., '62; Ishikawa et al., '69). The relevance of this hypothesis is in doubt since the Golgi studies of Valverde ('65) and Millhouse ('69) do not offer evidence, at least in the rat brain, of a direct ventral amygdalo-hypothalamic pathway as reported for the cat by the above authors. Furthermore, in the present experiments lesions placed directly between the amygdala and the hypothalamus (figs. 1c, 1d, 14) failed to reproduce the pattern of terminal degeneration which characteristically follows damage to the ventral strial component or its nuclei of origin in the amygdala (cf., Heimer and Nauta, '69; Chi, '70; Eager et al., '71; Leonard and Scott, '71).

The ventral amygdalofugal system will be discussed in some detail in another publication. Evidence is at hand that degeneration traceable through the basal part of the medial forebrain bundle into the lateral hypothalamic area, through the inferior thalamic peduncle to the dorsomedial thalamic nucleus, and via the stria medullaris to the lateral habenular nucleus is due to involvement of the ventral amygdalofugal pathway and is not derived from lesions of the stria (cf., de Olmos, '72).

Terminal degeneration appearing in the bed nucleus of the anterior commissure on both sides of the midline and in several other contralateral gray areas must be ascribed to concurrent degeneration of the commissural component of the stria terminalis which is unavoidably involved in this type of lesion (figs. 1d, 9; 1e, 11, 12).

As was found in the case of the dorsal strial component, lesions (see figs. 1c, 8d, 9e, 12; 8, 13) causing degeneration limited to the lateral half of the ventral component do not cause terminal degeneration in the medial hypothalamic nuclei but are associated with such degeneration which is localized in the lateral part of the bed nucleus of the stria. Consequently, these lateral fiber contingents are shorter than the medial ones.

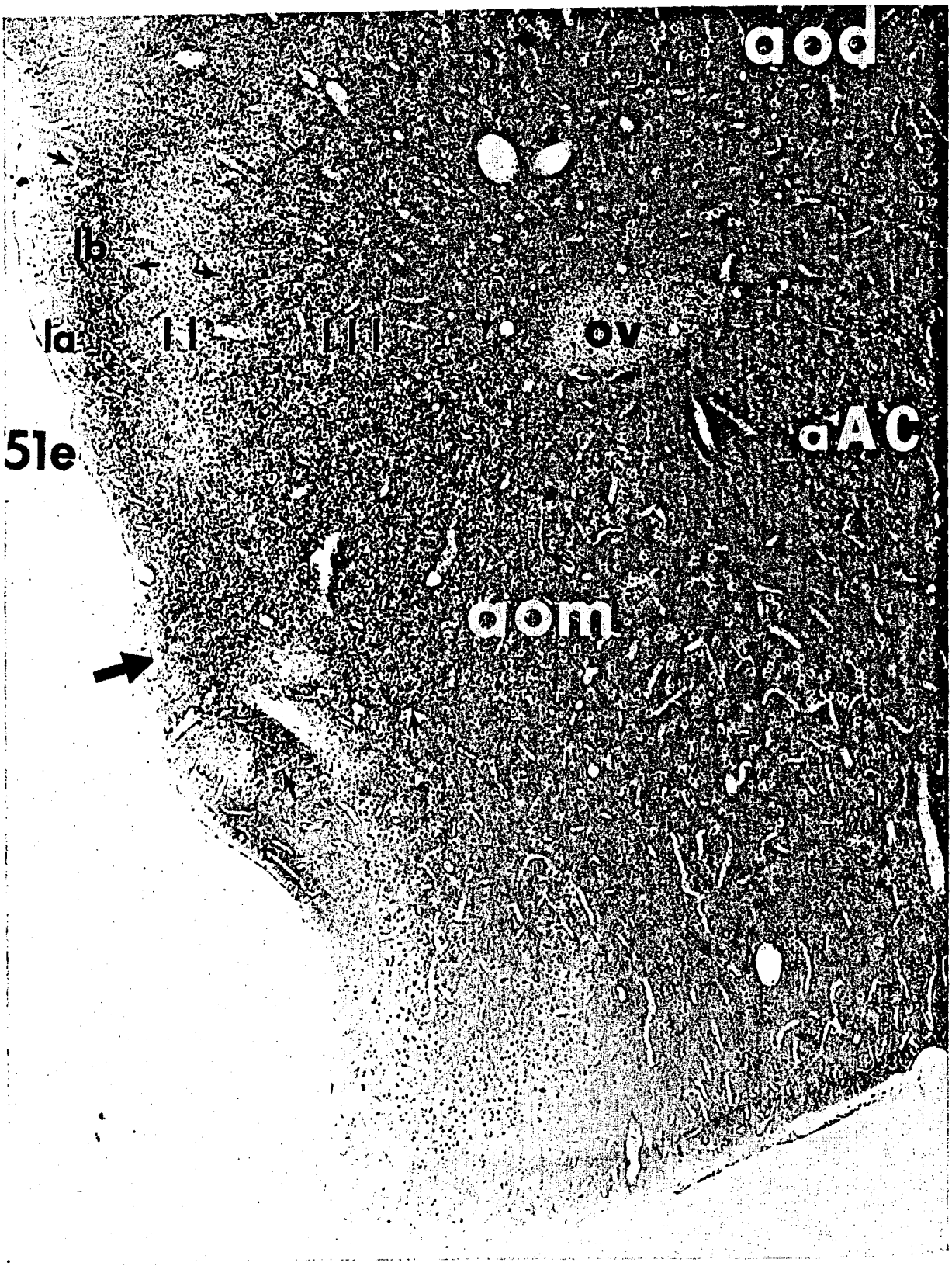

Fig. 7 Photomicrograph of a frontal section through the olfactory peduncle to show the distribution pattern of the terminal degeneration in this region after a lesion in the dorsal strial component. Modified cupric-silver method. × 120.

Degenerating terminals fill the deep polymorph-celled layer III and the inner sublamina Ib of the external plexiform layer of the dorsal portion of the anterior olfactory nucleus, pars medialis, or area praepiriformis 51e (51e). The big arrow marks the approximate location of the so-called medial olfactory tract as indicated in König and Klippel's atlas of the rat brain, the boundary between the dorsal and ventral portions of the nucleus and the location of the superficial offshoots of the parolfactory radiation.

3. *The commissural component.* In eight experiments (example in figs. 1d, 9; e11, 12), degenerating fibers which form this component or bundle 1 were found to differ from those of the dorsal and ventral components because of their coarser fragmentation and darker staining. Hence the round, compact fascicle which they form can be clearly followed as it passes forward between medial and lateral portions of the ventral component. It occupies a midventral position in the stria terminalis as the latter loops over the internal capsule. Anteriorly it swings ventrally and medially to enter the anterior commissure in which it decussates. This component apparently originates in the nucleus of the lateral olfactory tract.

Before and after decussation, the commissural component innervates the ipsi- and contralateral bed nuclei of the anterior commissure, respectively. Subsequent to crossing, a small contingent of fibers detaches from it, swings dorsolaterally through the contralateral bed nucleus of the stria, distributes some few terminals into a small central region of this latter gray mass and then passes backward in the stria terminalis of the side opposite the lesion to reach the contralateral amygdaloid region. It forms terminals with a few medial amygdaloid cells, and with neurons in Brodal's lateral amygdaloid nucleus. The latter also receives connections by way of the main contingent of the commissural stria.

This main division of the commissural stria having joined the posterior limb of the anterior commissure eventually reaches the external capsule, at which point it contributes to a fine diffuse terminal degeneration in the lateral amygdaloid nucleus. Along its course it also supplies degenerating terminals to the cellular masses which extend from the bed nucleus of the stria along the posterior limb of the anterior commissure (concerning these cell aggregations see Gurdjian, '25, and Brodal, '48, also see ipac in the figs.).

An additional and substantial projection of commissural strial fibers could also be traced to the contralateral olfactory tubercle and area praepiriformis 51a (fig. 13). An outstanding characteristic is the predominant distribution of degenerating terminals to the convulated folds (Beccari's, '10, "nidi di tipo primo") of the pyramidal cell layer of the olfactory tubercle and to the pyramidal cell layer in the medial half of the nonpeduncular portion of the area praepiriformis 51a. In the latter, the degenerating particles are both deeply and superficially placed (fig. 14). Finally, contrary to our expectation, no terminal degeneration nor axon stem degeneration could be seen to reach either the nucleus of the lateral olfactory tract nor the claustrum.

Control and miscellaneous observations. In spite of all precautions the possibility remains that structures in the paths of electrodes or involved in the surgical approaches could be related to at least some of the findings described above. Therefore, 13 control experiments were done in which lesions of varying extent were made. In these cases the survival periods were the same as those which are associated with optimal demonstration of terminal degeneration in brains in which the stria terminalis was involved.

Since a detailed description of the resulting patterns of terminal degeneration after lesions of the hippocampal formation and piriform cortex as well as of the contributions of the basolateral amygdaloid complex outside the stria terminalis is beyond the scope of the present report and since most of the findings agree in general with those published by other authors (see Guillery, '56; Nauta, '56; Lammers and Lohman, '57; Sanders-Woudstra, '61; Hall, '63; Cowan, Raisman and Powell, '65; Knook, '65; Powell, Cowan and Raisman, '65; Valverde, '65; Raisman, Cowan and Powell, '66; Raisman, '70; Heimer, '68 and cf. de Olmos, '71), brief comment only will be offered here.[5] Careful examination of these control specimens confirmed the general validity of the present account of the organizations of the stria terminalis. Especially strong support for this interpretation is derived from experiments with lesions localized to the precommissural or postcommissural portions of the bed nucleus of the stria terminalis, respectively (examples in fig. 1a, 3; 1b, 7). These

[5] Summary descriptions of the degeneration patterns associated with these lesions will be made available to interested persons.

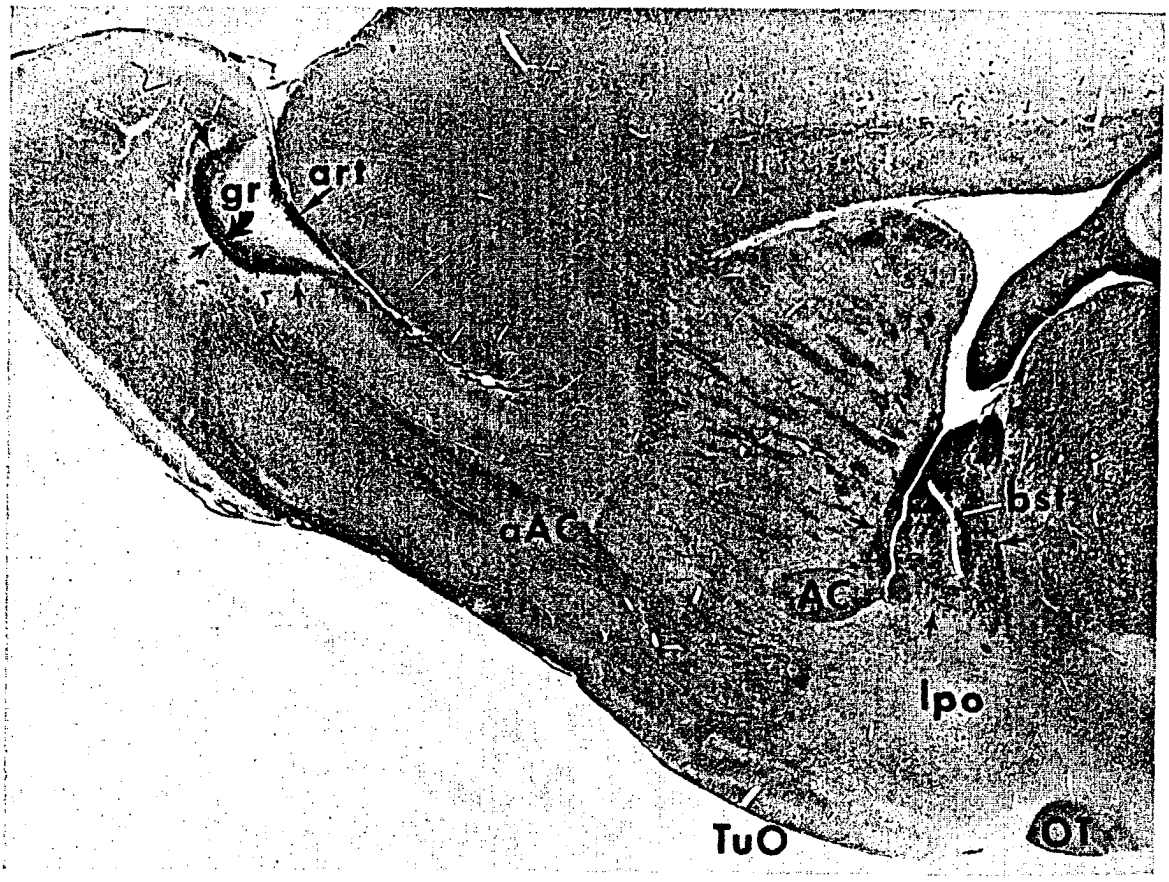

Figure 8a

Figs. 8a and 8b Parasagittal sections, stained with the modified cupric-silver method, which show in figure 8a the terminal degeneration in the internal granular layer (gr) of the accessory olfactory bulb after damage to the dorsal strial component as compared with the negative results in another experiment as illustrated in figure 8b after combined degeneration of the ventral amygdalofugal pathways, the lateral portion of the ventral strial component and the ventral hippocampus-fornix system. × 18.

It will be noted that in figure 8a degeneration of the dorsal strial component is also associated with degeneration in the bed nucleus of the stria terminalis (bst). In figure 8b degeneration of this area was due to involvement of the lateral portion of the ventral strial component. The lesion in this case affected the posterolateral amygdala and posterior piriform cortex, giving rise to degeneration in the ventral amygdalofugal paths as well as in the lateral part of the ventral strial component. There was also damage to the ventral hippocampal formation causing degeneration in the medial-corticohypothalamic tract (MCH). It seems evident that the degeneration in the nucleus accumbens septi (Acc), and the olfactory tubercle (TuO) was due to interruption of the ventral amygdalofugal pathway.

produced patterns of terminal degeneration which matched very closely those which followed interruption of the different components of the stria terminalis, except for the additional presence of terminal degeneration in the lateral habenular nucleus, perhaps due to involvement of stria medullaris fibers.

In addition, the distribution of terminal degeneration ensuing from lesions of the caudodorsal striatum was entirely confined to the overlying neocortex, the striatum itself, and the caudal subthalamic and thalamic regions (probably due to interruption of "en passage" cortico-thalamic, corticosubthalamic and thalamocortical fibers). Therefore, the pattern of terminal degeneration caused by coincidental electrode track damage to this portion of the striatum does not complicate that produced by interruption of the stria terminalis by the lesion proper.

DISCUSSION

From a survey of the literature, it seems that the gross topographical division of the

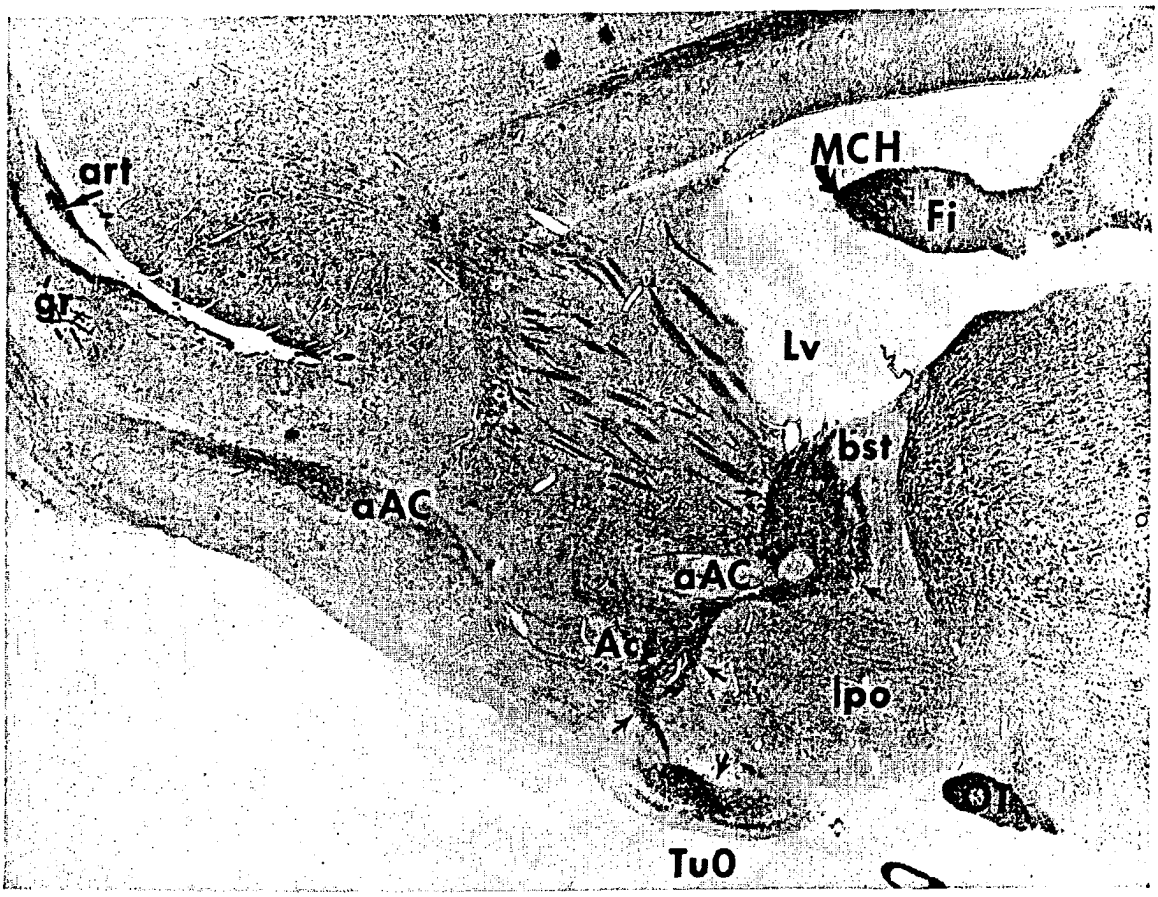

Figure 8b

stria terminalis into dorsal, ventral and commissural components proposed here is comparable, within certain limits, to Humphrey's ('36) and Young's ('36) designations of supracommissural, preoptic and commissural components. The infracommissural bundle (or bundle 3) of Johnston ('23) and Berkelbach van der Sprenkel ('26), if existent, is greatly reduced in the rat brain and forms part of the lateralmost portion of the dorsal stria component.

Although numerous reports describe a so-called stria medullaris component (or bundle 5) of the stria terminalis, both in normal material (Johnston, '23; Kappers, Huber and Crosby, '36; Fox, '40; Klingler and Gloor, '60; Miodonski, '66, and others) and in experiments with extensive lesions (Poirier, '52; Knook, '65, Leonard and Scott, '71), the present experiments do not permit a definitive conclusion as to the existence of an amygdalo-habenular projection via a stria terminalis-stria medullaris system [6] because our total transections of the stria included extrastrial tissue. However, when the dorsal and commissural components or the lateral part of the ventral one were completely interrupted singly or in combination, no signs of axon stem and terminal degeneration were seen in the stria medullaris thalami nor in the habenula. The same was true for the medial portion of the ventral component, except for its medialmost fibers which degenerated only after rostromedial amygdalar lesions. These coincidentally interrupted the ventral amygdalofugal pathways which are known to project to the lateral habenular nucleus via the inferior thalamic peduncle and the stria medullaris thalami (Sanders-Woudstra, '61; Cowan, Raisman and Powell, '65; Powell, Cowan and Raisman, '65). This prevents any definite conclusion concerning participation of the medialmost stria terminalis fibers in the innervation of the habenula. However, the morphological characteristics displayed by degenerating axons and their terminals in the stria med-

[6] The existence of an amygdalopetal component in the stria terminalis coming from the region of the stria medullaris thalami was suggested by Bürgi and Bucher in 1963.

ullaris and habenula show a thicker and larger fragmentation than is found in established stria terminalis components and projection fields. In addition, the same pattern of gross-fragmented degeneration is found in the inferior thalamic peduncle and stria medullaris after lesions in different portions of the piriform or prepiriform cortices and the substantia innominata but not in the stria terminalis (unpublished results). It therefore appears permissible to suggest that the stria terminalis does not contribute to the stria medullaris thalami. Such a suggestion finds support in the Golgi studies of Cajal ('11) and Valverde ('65) and experimental backing is supplied by the work of Adey and Meyer ('52), Ban and Omukai ('59) and Sanders-Woudstra ('61). Finally, Gloor ('55) was unable to record short latency evoked responses from the habenula after stimulation of the amygdala in the cat.

The Dorsal Strial Component

As previously described, this bundle divides into a major supracommissural contingent and minor retrocommissural and commissural ones when it reaches the level of the anterior commissure (fig. 15).

In this report the retrocommissural contingent of the dorsal strial component is described as separate from other postcommissural strial fascicles which arise from the ventral component, which has a separate origin in the amygdala (de Olmos, '72). Most authors in describing their results in this regard appear to have been guided by merely topographic criteria, disregarding or being unaware of the different origins of the two streams of stria fibers coursing behind the anterior commissure.

The demonstration of the existence in the rat brain of a commissural division of the dorsal component is an important finding provided by the procedures used. Although commissural connections to the medial and cortical amygdaloid nuclei were suggested by the normal studies of Smith ('30) and Jeserich ('45) they were thought to originate in Johnston's commissural component or bundle 1, known to have its source in the nucleus tractus olfactorius lateralis. In experiments described in another communication (de Olmos, '72) it has been possible to demonstrate that the commissural division of the dorsal component arises from the same region as the major bundle from which it branches, i.e., the caudal one third of the corticomedial amygdala. Thus, this fiber contingent forms a true commissural connection between the amygdalae, in contrast with the so-called "commissural" component of the stria terminalis.

It is now evident that the *supracommissural division* of the dorsal component is the largest amygdalofugal moiety of the stria terminalis in the rat brain. Shortly after passing through and contributing to the bed nuclei of the stria terminalis and of the anterior commissure it bifurcates into parolfactory and hypothalamic radiations. The latter system, which projects exclusively to the medial preoptic and hypothalamic nuclei as far as the caudal tuberal region, will be analyzed farther on.

The parolfactory radiation provides the most striking observation of this series. This is the experimental demonstration of the extraordinary proliferation of this radiation, which extends as far as the granular layer of the accessory olfactory bulb while contributing on the way to the innervation of the basal part of the lateral septal nucleus, the posteromedial aspect of the nucleus accumbens septi, the olfactory tubercle (pars medialis), the cortical area praegenualis 25, and the partes posterior and medialis of the anterior olfactory nucleus.

The existence of fiber connections between the stria terminalis and the precommissural septum has been proposed by several authors from the time of Déjerine ('01) to Klingler and Gloor ('60). The normal materials used were of limited value since they gave very little evidence of the conduction polarities of the fibers concerned. However, the Golgi studies of the mouse septal region by Cajal ('11) apparently disclosed branches from the anterior or precommissural "cord" of the stria which entered the basolateral septum. This finding was confirmed in similar Golgi studies by Valverde ('65) and also by experimental observations of this author and others (cf. Knook, '65; Ishikawa, Kawamura and Tanaka, '69). On the other hand, Nauta ('61), Hall ('63)

and Cowan, Raisman and Powell ('65) did not identify an amygdalo-septal component of the stria. However, it is possible that their lesions were not wide enough to provoke degeneration of all the fibers of the stria.

The data presented here indicating a projection from the stria to the medial portion of the nucleus accumbens septi are in good agreement with those reported by previous authors on the basis of experimental studies (Fox, '43; Gloor, '55; Cowan, Raisman and Powell, '65; Knook, '65; et al.). The terminal degeneration appearing in the posteromedial portion of the nucleus after damage to the ventral hippocampus (see fig. 1h, 18) was far less than that with strial degeneration. Also, the distribution patterns of terminal degeneration within this structure after lesions of the dorsal hippocampus (fig. 1f, 16) or the posterior piriform cortex (fig. 1, 8, 17) and/or the basolateral amygdaloid complex (fig. 1c, 8; d, 10; g, 13) differ substantially, one from the other, and from that due to degeneration of the dorsal strial component. Thus, dorsal hippocampal damage is associated with terminal degeneration in the rostral portion of the nucleus accumbens while lesions of the posterior piriform cortex cause degeneration in the caudal and ventrolateral aspects of the nucleus (de Olmos, '72 and unpublished observations).

The finding of terminal degeneration in the posterior and medial portions of the anterior olfactory nucleus (figs. 4, 7) appears to be new, since the only reference found for such relationship is that of Marburg ('48), which concerns an olfacto-amygdalar connection in normal human material. An interesting situation exists in this particular area. If the pars posterior of the olfactory nucleus is actually the deep cellular stratum of the area praepiriformis 51f (Rose, '12; Vaz Ferreira, '51; White, '65) it is evident that fibers from the dorsal strial component, the ventral piriform-amygdalofugal systems (de Olmos, '71; Lammers and Lohman, '57; Price and Powell, '70; et al.) and the precommissural fornix (unpublished observations) converge not only upon the deep cellular stratum of this paleocortical area but also on that of the areas praepiriformis 51g' and 51e which corresponds to the pars medialis of the anterior olfactory nucleus. In area 51e, additional overlapping occurs within the deep half of the external plexiform layer which involves terminals of the dorsal component of the stria and also of the ventral piriform-amygdalofugal systems, with little if any participation of the precommissural fornix.

No reference for afferent fiber connections from the stria terminalis to the area praegenualis 25 of the medial frontal cortex (fig. 6) could be found in the literature. However, the presence of such connections was confirmed in several studies of the terminal degeneration pattern which follows damage to the dorsal strial component (fig. 1f, 4, 5; 1g, 6). Furthermore, even if the medial frontal cortex is also the target for other converging pathways from the piriform cortex and perhaps from the basolateral amygdaloid complex via the ventral amygdalofugal system (de Olmos, '72) these regions were untouched by the lesions under consideration and therefore the dorsal strial origin of medial frontal afferents discussed above must be accepted.

Without doubt, the terminal degeneration traced to the granular layer of the accessory olfactory bulb provides one of the most interesting observations presented here. Because of its importance the genuineness of this projection was checked by making control lesions in structures lying in the paths of the approaches used for interrupting the stria terminalis. It can be safely said that neither the hippocampus, the cortical areas surrounding it, the posterior piriform cortex nor even the anteromedial amygdala and basolateral amygdala project to this particular area of the brain. The only region forming this connection is the posteromedial amygdala which does so via the dorsal component of the stria terminalis (fig. 8a). In this way, the projection which the accessory olfactory bulb sends to the posteromedial amygdala (Winans and Scalia, '70) is reciprocated.

The existence of a direct connection between the stria terminalis and the olfactory tubercle (figs. 2, 5) has been proposed only by authors working with normal human brains, and this connection was usually thought to be afferent to the amygdala

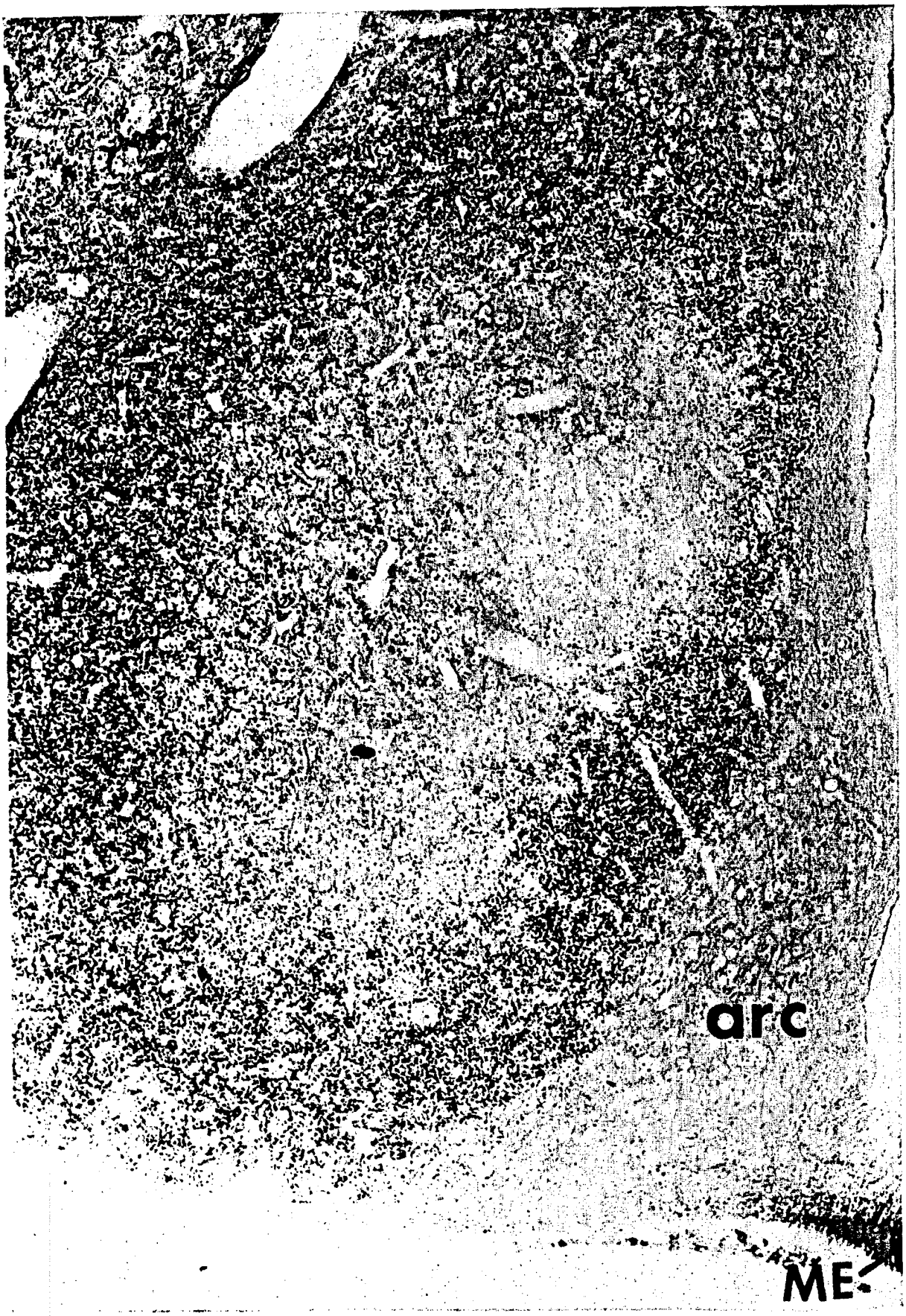

Fig. 9 The terminal degeneration encircling the central cellular core of the ventromedial hypothalamic nucleus as seen in a frontal section through the midtuberal level of the hypothalamus in a case with a lesion involving the dorsal strial component. Original cupric-silver method. × 180.

(Beccari, '10; Klingler and Gloor, '60; et al.). The present experiments show that the reverse is true with the dorsal strial projection limited to the pars medialis of the olfactory tubercle. This observation is supported by the finding of Powell, Clark and Mukawa ('68) that short latency responses can be recorded from the medial part of the tubercle after stimulation of the stria terminalis.

Here again there is overlapping with projections from the dorsal hippocampus. Little, if any, terminal degeneration was seen in the tubercle after lesions in the ventral hippocampal formation.

The olfactory tubercle also receives projections from the piriform cortex and the basolateral amygdaloid complex by way of the "ventral amygdalofugal pathway" (de Olmos, '72, but neither of these structures was encroached upon by lesions producing degeneration of the dorsal strial component in our experiments, although the posterior corticomedial amygdala was (fig. 1f, 4, 5).

The hypothalamic radiation. Except for the descriptive reports of Gurdjian ('27), Krieg ('32), Roussy and Mosinger ('35) and Kappers, Huber and Crosby ('36), authors working with normal materials were unable to trace fibers of the supracommissural contingent beyond the preoptic and anterior hypothalamic regions and this was in general true for experimental anatomical studies. However, Ban and Omukai ('59), Lundberg ('60, '62),

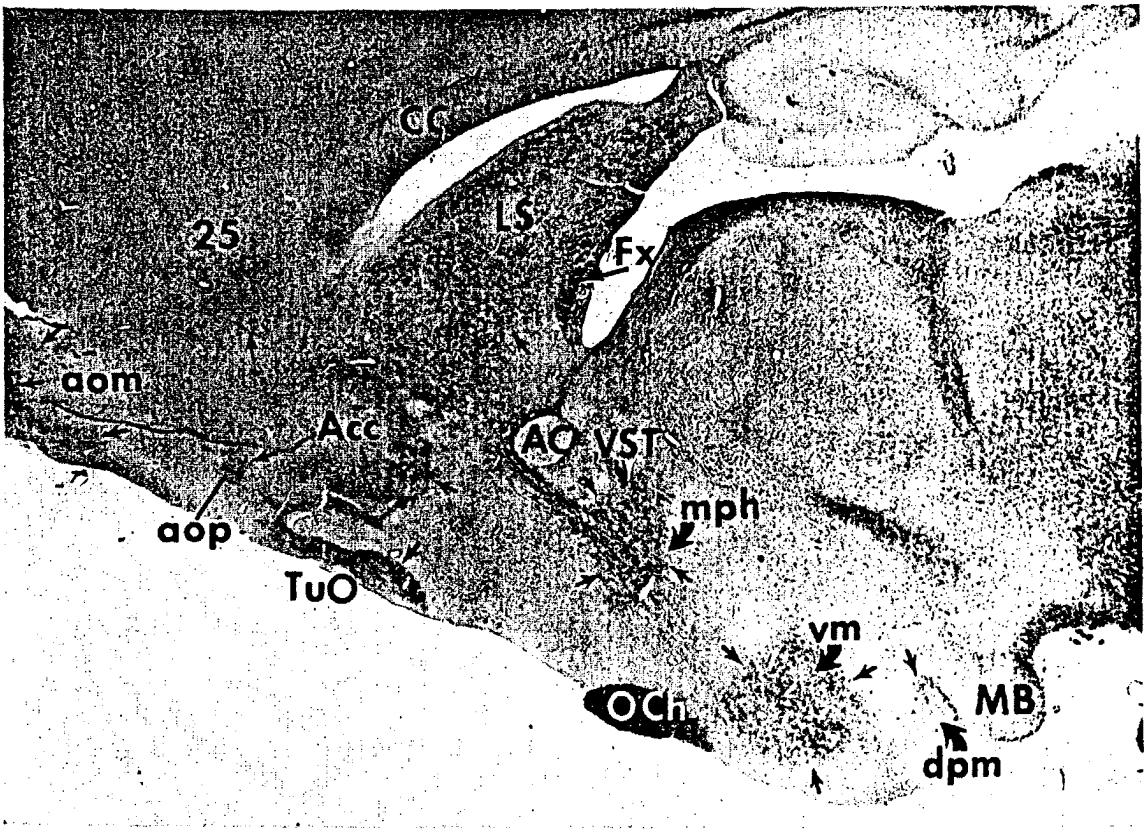

Fig. 10 A parasagittal section through the forebrain of a young rat to show the distribution pattern of terminal degeneration (small arrows) in different gray formations after lesions provoking degeneration of both dorsal and ventral components as well as the dorsal fimbria fornicis. Modified cupric-silver method. × 16.

Note in this case the filling by degenerating terminals of the entire extent of the ventromedial hypothalamic nucleus (vm) as compared with the capsule-like distribution of the terminal degeneration in the case illustrated by figure 4. Furthermore, the terminal degeneration indicated at the level of the nucleus accumbens septi (Acc), olfactory tubercle (TuO), partes posterior (aop) and medialis (aom) of the anterior olfactory nucleus and the area corticalis praegenualis 25 (25), is caused in most part by damage to the dorsal strial component, while the degenerating terminals in the dorsal part of the lateral septal nucleus (LS) belong to the precommissural fornix fibers degenerating because of a lesion of the fimbria.

Hall ('63), Knook ('65), and Ishikawa, Kawamura and Tanaka ('69) reported tracing such fibers to the rostral portion of the ventromedial hypothalamic nucleus. Electrophysiological evidence for such a connection was presented by Gloor ('55, '60), Gloor, Murphy and Dreifuss ('69), and Sutin ('63).

Knook was the first to present figures indicating the organization of this strial connection. He, however, interpreted a "cuff-like" spreading of the stria fibers around the ventromedial nucleus as indicating merely a by-pass of this nucleus.

Heimer and Nauta's 1969 report, based upon use of the Fink-Heimer method, offered more nearly complete information concerning the hypothalamic distribution of the stria, including the preoptic-hypothalamic junction area. Their findings at the optical microscope level with special reference to the ventromedial hypothalamic nucleus were backed by their own electron microscopic observations, by those of Raisman ('70), and supported by the electrophysiological studies of Murphy and Renaud ('68).

The use in the present experiments of the two cupric-silver methods, as well as variations of the Nauta-type stains has made it possible to trace the degenerating terminals of the dorsal strial hypothalamic radiation to the central and dorsal aspects of the junction area between the medial preoptic and anterior hypothalamic nuclei, the retrochiasmatic region, the paucicellular zone investing the ventromedial hypothalamic nucleus, Diepen's nucleus tuberis lateralis and the dorsal and ventral premammillary nuclei.

Comparing these results with those of Heimer and Nauta it is evident that except for differences in interpretation with respect to (a) the main channel source of terminals in the medial preoptic-hypothalamic junction area (mph), and (b) as to the inclusion of the dorsal premammillary nucleus within the strial projection field, there is close agreement.

With regard to discrepancy (a) Heimer and Nauta believed the terminals in the mph area to be derived from the postcommissural portion of the stria. However, in our experiments degeneration in this medial area was associated with lesions definitely limited to the supracommissural division.

Comparison of these results with those obtained after total lesions of the dorsal strial component suggest that the supracommissural division contributes to the rostromedial portion of the general area innervated by this component, while its retrocommissural division supplies the laterocaudal one. Termination of supracommissural fibers in the preoptic region has been described by Millhouse ('69) on the basis of Golgi studies on the rat brain.

The fact that Heimer and Nauta did not attempt to make discriminatory lesions of the several components of the stria may account for the difference in interpretation with regard to the medial preoptic hypothalamic terminals. This does not, however, explain why terminal degeneration was found in the dorsal premammillary nucleus in the present material but not mentioned by these workers. An explanation for the latter discrepancy might reside in the different conditions in the present experiment, i.e., the use of younger animals, usually shorter survival periods and fixation of the brain tissues with pH 7.2–7.4 buffered formaldehyde solutions.

Finally, with respect to the ventrolateral wing-like expansion of the tuberal terminal field into an area designated by Diepen ('62) as nucleus tuberis lateralis, it is interesting to note that this parvocellular area, besides being innervated by the supracommissural division of the dorsal strial component and by the ventral component also contains terminals derived from the postcommissural fornix. This latter connection is not shared by the ventral premammillary nucleus proper in which the lateral tuberal nucleus is included in the atlases of DeGroot and König and Klippel.

The ventral strial component

Accounts of the distribution of this bundle, commonly known as the preoptic or postcommissural component, offer vague and varied statements derived from studies of both normal and experimental material. This may be due in part to the thinness and feeble stainability of the fibers involved as well as to their fan-like spread into areas of termination which are difficult to define. Thus, although most authors

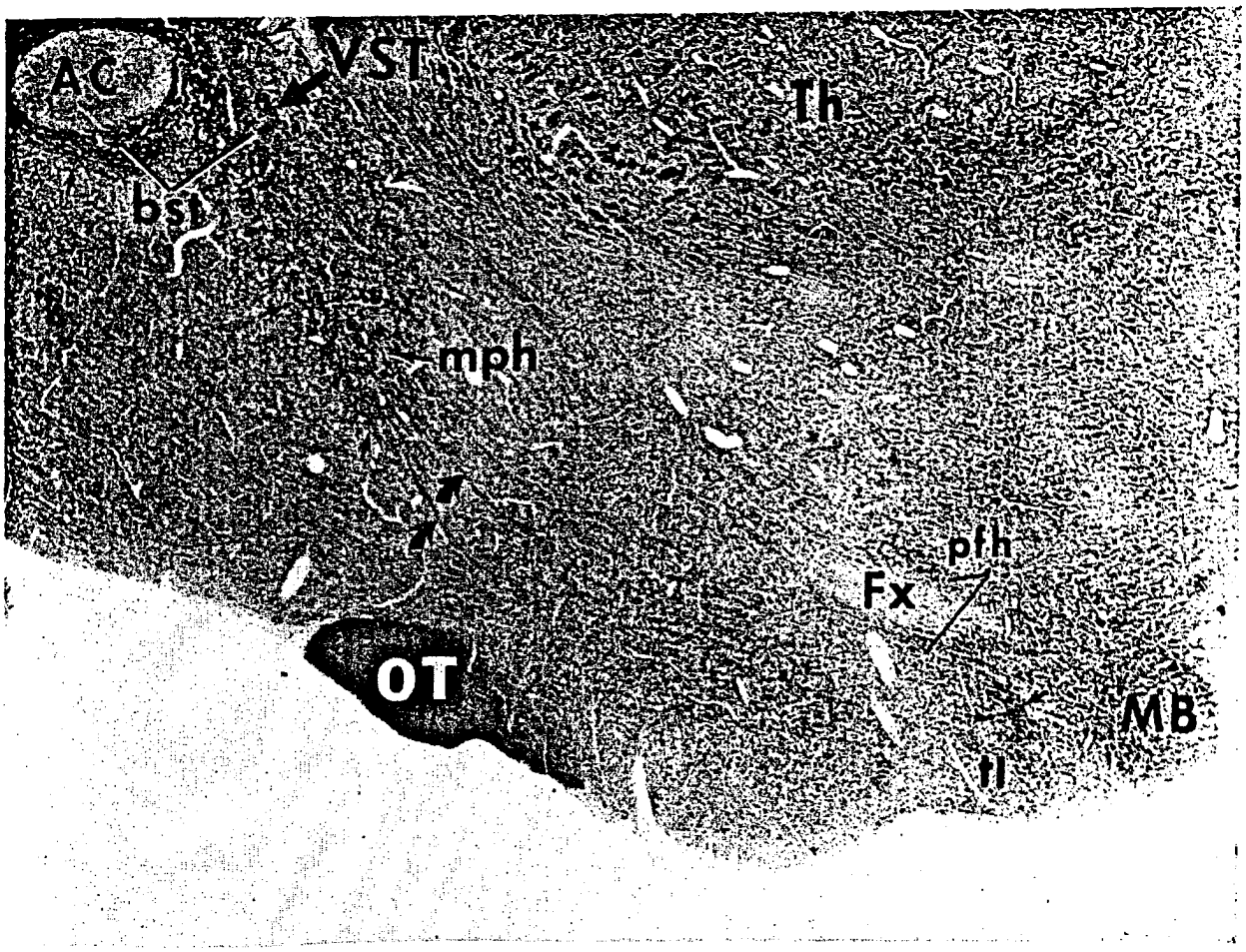

Fig. 11 Parasagittal section through the perifornical hypothalamic area (pfh) to show the arrival of the degenerating fiber contingents and distribution of the ventral strial component (VST). Modified cupric-silver method. × 37.

The thick small arrows point to fine degenerating fibers from this component leaving the area of terminal degeneration in the bed nucleus of the stria terminalis (bst) and medial preoptic hypothalamic junction area (mph) to reach eventually the parvocellular lateral tuberal area of Diepen's nucleus tuberis lateralis (tl) where degeneration is also present.

working with normal material agree in tracing this component to the preoptic and/or the hypothalamic regions, few indicate particular target nuclei. Such studies have sometimes implied that many of the fibers in question join the medial forebrain bundle in order to reach the hypothalamus, a suggestion supported by the Golgi studies of Cajal ('11), Valverde ('65) and Millhouse ('69).

In sharp contrast with the relative uniformity of descriptions derived from normal subjects, experimental work based upon Marchi or suppressive Nauta-Gygax stained material develops a quite confusing picture, with Knook's ('65), Morgan's ('68) and Ishikawa's ('69) reports offering the only really explicit statements about the ultimate destinations of the fibers of the ventral component. The first author traced them to the ipsilateral anterior hypothalamic nucleus and lateral hypothalamic area, the second one believed they ended in the anterior hypothalamic and lateral preoptic areas, and the third group described them as ending in the bed nucleus of the stria and in the lateral preoptic nucleus. Although Valverde ('65) found terminal degeneration in the lateral hypothalamic area and the subthalamic nucleus as well as in the preoptic and anterior hypothalamic regions in cats with lesions of this component, he was unable to establish if these terminals belonged to this bundle or to a more direct ventral amygdalo-hypothalamic fiber system in this

form. Adey and Meyer ('52), who used the Glees method in monkeys, also described terminals of this component in the preoptic and anterior hypothalamic regions, the dorsomedial, arcuate and ventromedial hypothalamic nuclei, bilaterally. These findings were supported by Adey, Rudolph, Hine and Harritt ('58) in spite of criticism based on the possible occurrence of Glees-positive pseudo-degenerative phenomena (Cowan and Powell, '56; see also comment by de Olmes, '69, Guillery, '70; Heimer, '70).

Most authors working with the suppressive Nauta-Gygax technique have been unable to discern bilateral projection of the stria terminalis to the hypothalamus (Nauta, '61; Cowan, Raisman and Powell, '65; et al.).

The present observations provided a clearer concept of the distribution of the ventral strial component perhaps because of the particular suitability of the methods used, particularly the current version of the cupric silver method. Thus, to recapitulate the observations already described (see fig. 15), this bundle distributes ipsilaterally to the bed nucleus of the stria terminalis, the medial preoptic-hypothalamic junction area, the retrochiasmatic region, the entire ventromedial hypothalamic nucleus, Diepen's nucleus tuberis lateralis, and both dorsal and ventral premammillary nuclei. No terminal degeneration of the ventral stria fibers was found in the dorsomedial hypothalamic nucleus, the arcuate infundibular nucleus and the lateral hypothalamic area nor were degenerating strial fibers detected within the diffuse composite called the medial forebrain bundle. A bilateral projection of the stria terminalis to the hypothalamus was not recognized. While the negative aspects of these results agree with those reported by Heimer and Nauta and Leonard and Scott, the present disclosure of terminal degeneration in the premammillary nuclei, Diepen's nucleus tuberis lateralis, and particularly in the central cellular core of the ventro-medial hypothalamic nucleus are added to the more limited distribution found by these authors in their Fink-Heimer preparations, which indicated terminations of the postcommissural fibers in the bed nucleus of the stria terminalis (Leonard and Scott) and in the "dorsal and medial hypothalamic regions" (Heimer and Nauta) only. Although their failure to detect a distribution to the premammillary nuclei and the nucleus tuberis lateralis may have been due in part to the type of lesion used, their lack of evidence for termination of strial fibers in the central core of the ventromedial nucleus must be due to other causes. Since all the staining procedures used in the present work show terminal degeneration in this portion of the nucleus (figs. 10, 12) it can be proposed that these causes include the lesser ages of the animals utilized, the briefer survival periods and even the rather alkaline pH of the fixing solutions. Any or all of these may have played some role in providing evidence for the more extensive terminal degeneration shown in the material presented here.

In support of findings of terminations of ventral strial fibers in the core of the ventromedial hypothalamic nucleus one might cite the Golgi studies of Cajal ('11). Careful examination of his description and illustrations of the stria terminalis and of the fiber supply to the ventromedial nucleus ("noyau principal," see Cajal, '11, pp. 474–479 and 721–724, and compare his figs. 312, 313, 314, 462), indicate that part at least of what he called "voie nerveuse issue du septum lucidum" contributing to the fibrillar capsule of the nucleus might correspond to the ventral strial component described in this paper. Terminal innervation of the core of the ventromedial nucleus was indicated by Cajal and related by him to a septal origin. However, lesions of the septal nuclei in our experiments have not provoked terminal degeneration in this nucleus. Direct amygdalar contribution of fibers to the core of this nucleus is attested by the work of Murphy and Renaud ('68). Their combined histological and electrophysiological studies of the neuronal population in the ventromedial nucleus of the cat suggest that while the bipolar inhibiting neurons receiving monosynaptic afferent impulses from the amygdala are most concentrated in the lateral margin of the nucleus, they are also located within the nucleus itself.

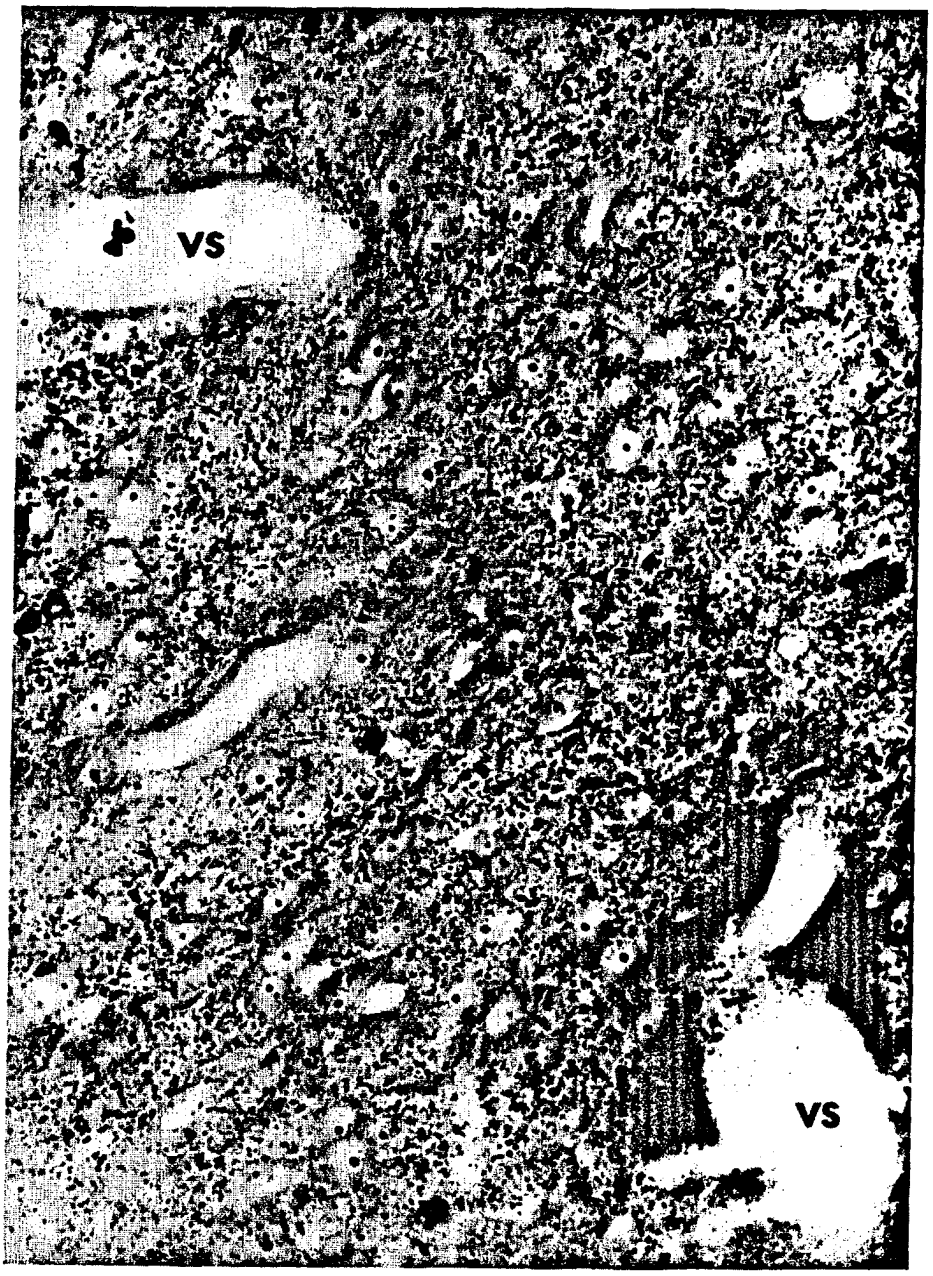

Fig. 12 A frontal section through the hypothalamus to show terminal degeneration throughout the right ventromedial hypothalamic nucleus 36 hours after production of a lesion involving the ipsilateral dorsal and ventral strial components in a very young animal. Original cupric-silver method. × 480.

The commissural strial component

According to many descriptive studies this bundle constitutes a commissural connection between the nuclei of the lateral olfactory tracts of the two hemispheres (Johnston, '23; Craigie, '25; Humphrey, '36; Young, '36; Fox, '40; Miodonski, '66). Other suggested connections involve projection from one of these to the contralateral piriform cortex (Cajal, '11), claustrum (Berkelbach van der Sprenkel, '26), central and lateral amygdaloid nuclei (Gurdjian, '28) and the medial and cortical amygdaloid nuclei (cf. Smith, '30; Jeserich, '45) as well as the median, medial and lateral preoptic nuclei and the bed nucleus of the anterior commissure (cf. Humphrey, '36; Valverde, '65; Millhouse, '69).

In contrast with these studies of normal

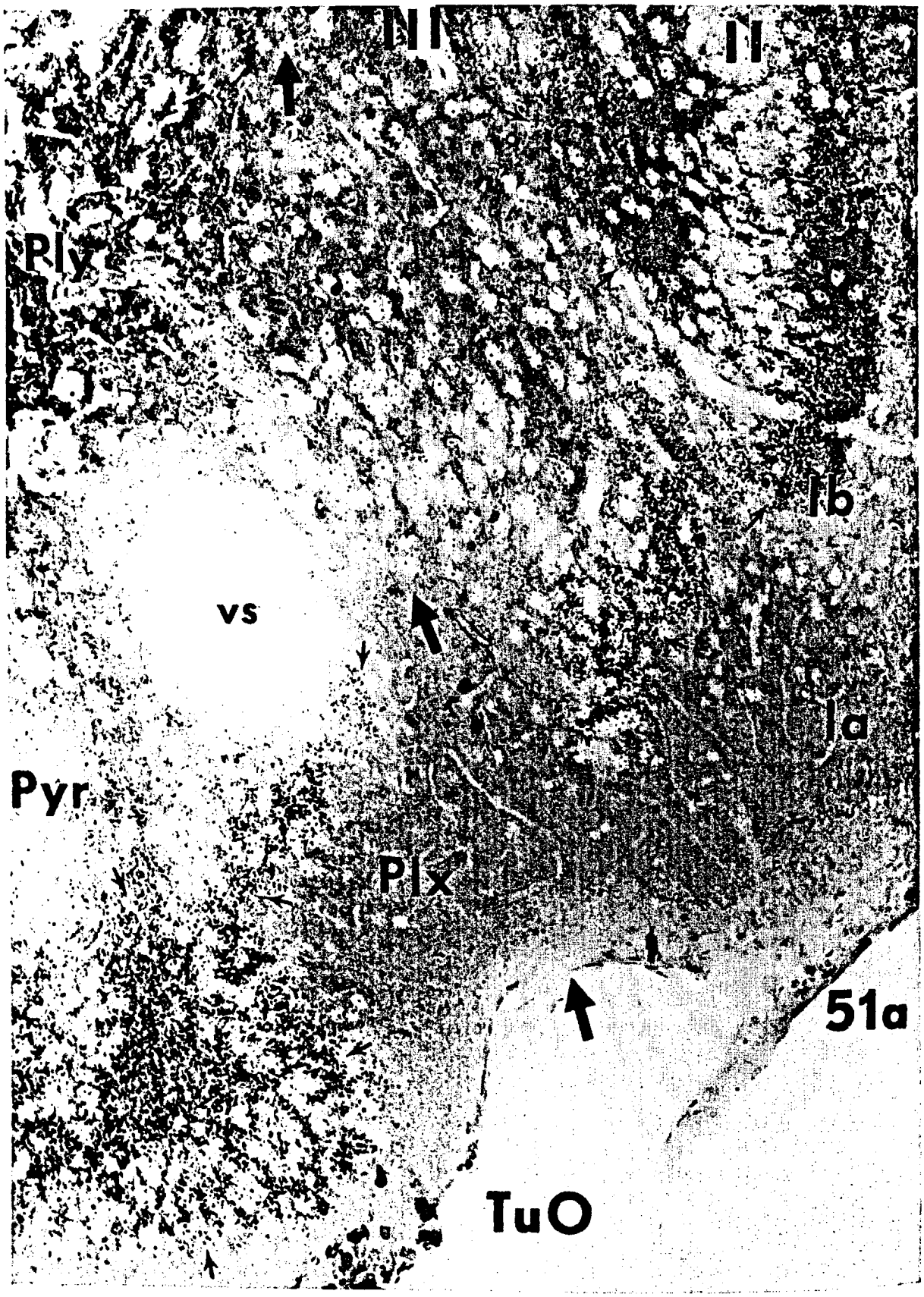

Fig. 13 A frontal section showing the distribution of terminal degeneration in the area praepiriformis 51a (51a) and in the lateral margin of the olfactory tubercle (TuO) after a lesion of the contralateral "commissural" component of the stria. Original cupric-silver method. × 280.

The degeneration encapsulates the superficial pyramidal-celled layer II of the area praepiriformis 51a and is also found in the convolution of the pyramidal cell layer (Pyr) of the tubercle. The thick arrows mark the boundary between these paleocortical formations.

brains, much experimental work based upon Marchi and suppressive Nauta-Gygax material has failed to show any site of termination of the commissural bundle (Fox, '43; Fox and Schmitz, '43; Ban and Omukai, '59; Nauta, '61; Cowan, Raisman and Powell, '65; et al.). However, findings based on strial lesions in the cat brain suggest, according to Lammers and Magnus ('55) and Lammers and Lohman ('57), that the fibers in this bundle end in the contralateral bed nucleus of the anterior commissure, or, according to Valverde ('65) in both homo- and contralateral bed nuclei of the anterior commissure and of the stria terminalis. Knook ('65) traced the degenerating commissural fibers into the vicinity of the nucleus of the lateral olfactory tract and of the claustrum, prepiriform cortex and lateral amygdaloid nucleus in rats, but was unable to establish terminal degeneration in these areas. Van Alphen ('69), using the Fink-Heimer method in the rabbit brain, found degenerating terminals in the contralateral bed nucleus of the stria, a finding in agreement with reports by Heimer and Nauta ('69) and Leonard and Scott ('71).

An evaluation of our results (see fig. 16) and those of others indicates that the so-called "commissural" component is more properly a *decussation*. Thus while we agree with most authors that the nucleus of the lateral olfactory tract is a site of origin (de Olmos, '72) of this fiber system we found no signs of terminal degeneration in this nucleus contralateral to lesions of the bundle or its source (cf. Van Alphen, '69). On the contrary, the commissural bundle contains not only projections directed to the homo- and contralateral bed nuclei of the anterior commissure and stria terminalis (bac and bst in fig. 16), cellular masses along the posterior limb of the so-called commissure (ipac in fig. 16), and the lateral amygdaloid nucleus (L in fig. 16), but also an important projection oriented to particular regions of the contralateral paleocortex, i.e., the olfactory tubercle and the nonpeduncular portion of the area praepiriformis 51a. Our material also suggests that these connections are established with the bodies and basilar dendrites of the pyramidal cells in the superficial cellular layers and with the basilar

Fig. 14 A higher magnification of the terminal degeneration in the area praepiriformis 51a (fig. 13) showing terminal degeneration in the sublamina tangentialis Ib of the external plexiform layer. Very dense terminal degeneration is also seen at the deep side of the superficial pyramidal-celled layer II while fewer silver granules are spread among the cell bodies of the neurons forming this layer. × 510.

portions of their apical dendrites (fig. 13, 14). This pattern of termination contrasts in localization with those amygdalofugal projections to homologous paleocortical fields ipsilateral to the lesion via the parolfactory radiation of the dorsal stria component.

Comparing these observations with those of others it appears again that such factors as species differences, age, survival period and staining methodologies might have contributed to some of the variations in findings.

Recapitulation

Recapitulation of the present findings as summarized in figures 15 and 16 show that the stria terminalis, at least in the rat brain, provides a major direct route by which the amygdala, and particularly its corticomedial portion can modulate some

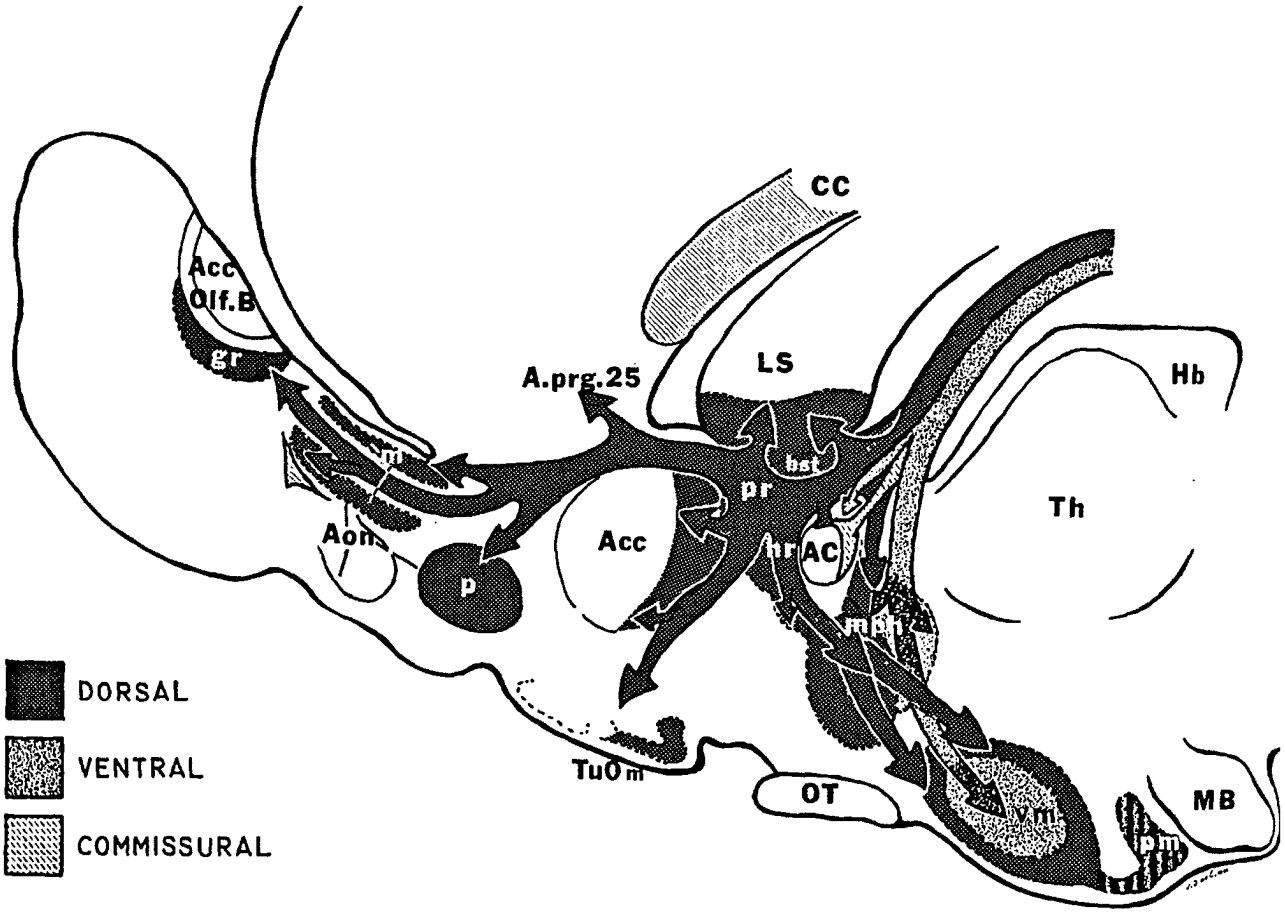

Fig. 15 Schematic representation of the distribution of the three components of the stria terminalis: dorsal, ventral and commissural. The diagram shows the stria of the right as seen from its medial side. The gray groups supplied from it are represented in broken lines and filled with the type of shading representing that particular component of the stria. The shading code is in the lower left corner of the figure and represents each of the three components illustrated. The dorsal strial component supplies the bed nucleus of the stria terminalis, the basal part of the lateral septal nucleus, the posteromedial part of the nucleus accumbens septi and olfactory tubercle, the internal granular layer of the accessory olfactory bulb, the medial preoptic hypothalamic junction area, the capsule encircling the cellular core of the ventromedial hypothalamic nucleus, and the premamillary area. Not represented in the figure are the retrochiasmatic area and Diepen's nucleus tuberis lateralis which are also recipients of fibers from this bundle. The ventral strial component distributes to the bed nucleus of the stria terminalis, the medial preoptic-hypothalamic junction area, the central core of the ventromedial hypothalamic nucleus, and the premamillary area. Again the retrochiasmatic area and the Diepen's nucleus tuberis lateralis are not represented although they also receive fibers from this component. Finally, the "commissural" component is seen to enter the anterior commissure as does also the commissural division of the dorsal strial component.

of the homeostatic and behavioral functions with which the hypothalamus is concerned. Furthermore, those amygdaloid neurons which send their axons into the stria could influence activities of the hypothalamus not only by forming synapses directly with neurons of its medial preoptic and tuberal regions via both the dorsal or ventral strial components, but also by relays in the olfactory and parolfactory centers where the dorsal component terminates, since these make connections with the lateral preoptic and hypothalamic areas via the medial forebrain bundle. By the same route they could influence the functional activities of the habenula and other thalamic nuclei.

In addition to the terminal spread of the stria this study has also demonstrated that fibers within the bundle are arranged in such a way that the longer axons occupy a more medial position than the shorter ones. A dorsoventral arrangement is also apparent especially with regard to the medial

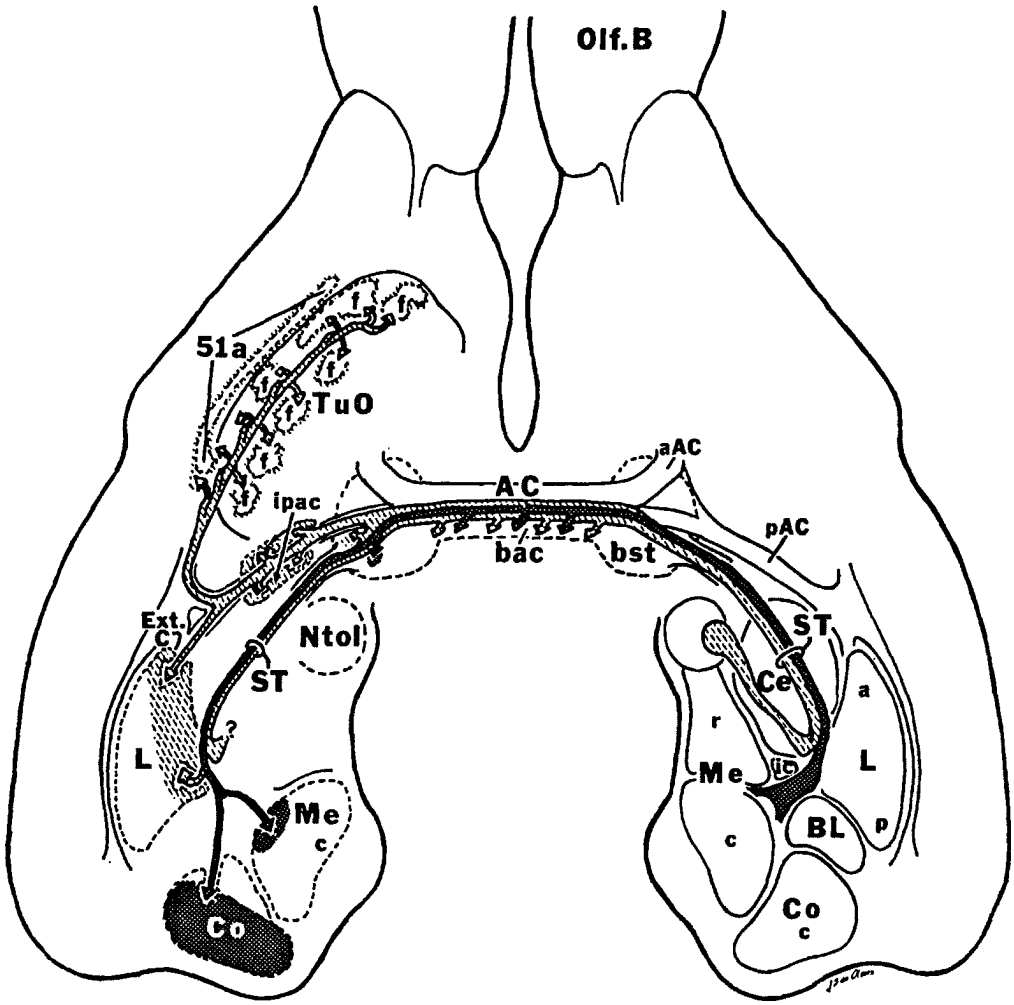

Fig. 16 Diagram, as viewed from above, illustrating the sources, courses and terminations of the commissural division of the dorsal component and of the so-called 'commissural" component. Both fiber systems are identified according to the shading code used in figure 15, but they can also be recognized by their pathways of departure from the right amygdala, i.e., the first bundle from the caudal portions of the cortico-medial nuclear complex, and in the second instance, from the nucleus of the lateral olfactory tract (Ntol). On the left hemisphere, the outlines of the gray formations where these two fiber systems terminate are represented in broken lines which contrast with the continuously lined outlines of the amygdaloid nuclei in the right hemisphere. Their respective relationships are further stressed by the shadings which match those of the fiber contingents supplying them. Thus, the commissural division of the dorsal component is shown to supply the bed nuclei of the anterior commissure and of the stria terminalis, and after joining the contralateral stria terminalis, restricted areas of the caudal portions of the medial and cortical amygdaloid nuclei. The "commissural" component, on the other hand, connects with the bed nuclei, and, after bifurcating and joining thereafter the contralateral stria terminalis and the posterior limb of the anterior commissure, terminates in restricted portions of the lateral amygdaloid nucleus, in cell masses surrounding the posterior limb of the anterior commissure, the area praepiriformis 51a and the convolutions (f) in the anterolateral portions of the olfactory tubercle.

long-projecting fibers. This supports the proposed descriptive division of the stria terminalis into dorsal and ventral components, with the "commissural" component lying between the medial and lateral portions of the latter. This concept is further supported by the fact that while fibers of the dorsal component reach the basal telencephalic centers and the medial hypothalamus, those of the medial portion of the ventral component reach the latter area only. Furthermore, even when there is some

overlap between the hypothalamic projections of these components, it has also been shown that the ventromedial hypothalamic nucleus forms a topical relationship with them; the dorsal strial fibers end largely in the cell-poor lamina encircling the nucleus and the ventral ones terminate in its cellular core.

Such topographical arrangement of fibers and terminals of the stria terminalis might have its basis in the type of contribution of each amygdaloid nucleus to the different strial components. Thus, experimental observations in our laboratory (de Olmos, '68, '70, '71) have indicated that while the caudal one third of the corticomedial amygdaloid complex is the source of many of the long axons in the dorsal component and that the rostral two thirds gives rise to those in the medial portion of the ventral one, most neurons of the basolateral complex contribute shorter projecting axons which pass via the lateral part of the stria to terminate mainly in the bed nucleus of the stria terminalis (cf. Ishikawa et al., '69; Leonard and Scott, '71). This topic is discussed in a subsequent communication (de Olmos, '72).

With regard to the so-called "commissural" component, it is interesting to note that the lateral amygdaloid nucleus, which constitutes the major amygdalar recipient of fibers in this bundle, makes only a small contribution of short projecting axons to the stria terminalis, an observation which will be elaborated in another place. However, this nucleus apparently possesses a strong efferent projection to the ipsilateral nucleus of the lateral olfactory tract (de Olmos, '72). This arrangement would provide for recircuiting of "commissural" impulses and/or their eventual transference to the telencephalic centers mentioned in the discussion of other components of the stria. Furthermore, the "commissural" bundle by means of its decussating connections with the olfactory tubercle and prepiriform cortex would also be situated so as to influence the activity of the lateral preoptic and hypothalamic areas as well as the thalamus and habenula of the contralateral side taking into account the same anatomical considerations as were given with regard to the homolateral projection of the stria terminalis to the olfactory tubercle.

Finally, the importance of the interconnections between the caudal corticomedial amygdalae of both hemispheres via the commissural division of the dorsal component is emphasized by the following observations. 1. The extent of the projection field of this particular portion of the amygdaloid complex. 2. The bed nucleus of the stria terminalis, one of the major recipients of amygdalofugal impulses via all components of the stria terminalis, appears to contribute a strong decussating projection to the above mentioned amygdaloid regions. 3. The caudal portion of the corticomedial amygdala receives a separate afferent supply from the accessory olfactory bulb (Winans and Scalia, '70), a connection which is reciprocated as has been shown in the present study.

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