THE THALAMIC PROJECTION UPON THE TELENCEPHALON IN *LACERTA VIRIDIS*

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INTRODUCTION

The basic principles of the organization of the mammalian thalamus have been defined largely as the result of experimental comparative studies in different species of mammals. Thus the afferent and efferent connexions of most of the nuclei of the dorsal thalamus have been established with both anatomical and physiological techniques. It has been shown that all of these nuclei project upon the telencephalon, the majority being related to the neocortex, and the remainder to either the rhinencephalic cortex (Rose & Woolsey, 1943) or to the striatum — directly (Stefens & Droogleever Fortuyn, 1953; Powell & Cowan, 1956*a*) or by way of collaterals (Nashold, Hanbery & Olszewski, 1955).

In contrast to this knowledge of the mammalian thalamus little is known of the connexions of this region in other classes of vertebrates, and what is known has been derived mainly from studies of normal material. The question of the projection of the thalamic nuclei in the bird and reptile is of particular interest because of the radically different structure of the telencephalon in these animals, and especially in view of the relatively greater development of the striatum. In the reptile the striatum can be readily differentiated into a dorsal hypopallial portion, and a ventral palaeostriatum composed of a medial part connected mainly with the hypothalamus and a lateral part connected with the thalamus. Goldby & Gamble (1957) point out, however, that these connexions need experimental confirmation, and that their functional significance is speculative. It has been thought that an extension of experimental investigations similar to those which have been done in the mammal might provide significant data for the interpretation of the phylogenetic development of the dorsal thalamus.

In the present study attention has been directed to the problem of the projection of the thalamus upon the telencephalon in the reptilian brain. The technique of retrograde cell degeneration has been used as it has proved one of the most effective methods for similar studies in the mammal; it may be mentioned that the limitations of the technique are fully realized. The earlier work of Goldby and his associates (Goldby, 1937; Armstrong, 1950; Gamble 1952, 1956; Armstrong, Gamble & Goldby, 1953) on the connexions of the cerebral hemisphere, particularly with the olfactory and visual systems, has encouraged us to employ experimental techniques in the reptile. *Lacerta viridis* has been used in this investigation because it is readily obtainable, and convenient to handle for experimental purposes, and because a detailed description of the cerebral hemisphere has been given by Goldby (1934).

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MATERIAL AND METHODS

Forty-three green lizards (*Lacerta viridis*) were used in these experiments. The operations were performed under ether anaesthesia, and the cerebral hemispheres were exposed by removing parts of the overlying frontal and parietal bones. Lesions of varying size were made in the cerebral hemispheres, either by suction or by a fine needle, with the aid of a binocular dissecting microscope. Many of the lesions were bilateral, so that in all fifty-eight hemispheres were available for study. The opening in the skull was closed with thrombin foam; no signs of infection were found in the brains. The animals were kept at approximately 28° C. for periods ranging from 28 days to 82 days. They were killed by an overdose of ether and the brains fixed in 70 % alcohol and 2 % acetic acid. After embedding in paraffin wax most of the brains were cut in the coronal plane, but some were cut horizontally. The sections were 10μ in thickness; a 1 in 2 series was stained with thionine, and an additional series of many brains was stained by Bodian's protargol method.

RESULTS

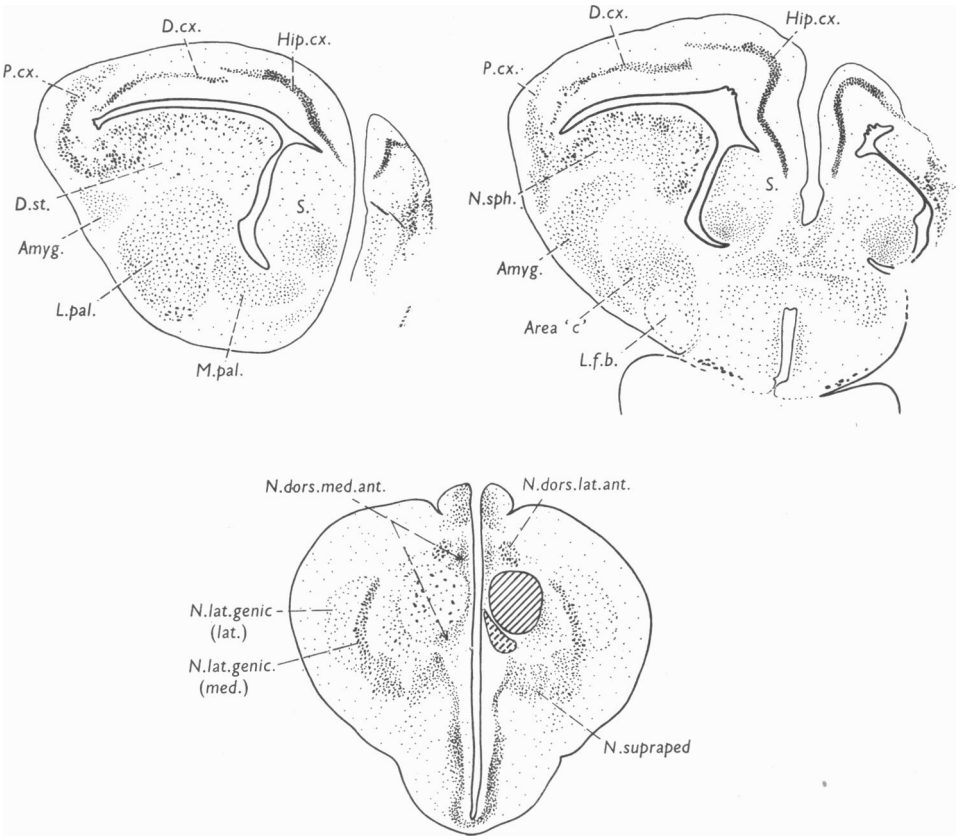
In general the terminology suggested by Goldby & Gamble (1957) in their recent review of the literature of the reptilian cerebral hemisphere has been used in the present study. The thalamic nuclei which will be considered are easily delimited and can be readily identified in the photographs; a well illustrated account of these nuclei in *Lacerta* has been given by Armstrong (1950).

The difficulty in producing localized lesions in the small brain of *Lacerta* has resulted in only a few lesions being restricted to individual structures in the hemisphere. To facilitate the description of the material the lesions have been divided into three groups, and only representative examples of each will be described in detail. In the first group the telencephalon has been almost completely removed; in the second group there has been extensive destruction of the cortex and sub-cortical structures without involvement of the palaeostriatum, and in the last group are the smaller lesions by which the thalamic projection has been more accurately determined.

A striking finding in these experiments in *Lacerta* is that in contrast to mammals most of the thalamic nuclei have survived after removal of the telencephalon. Furthermore, the nature of the retrograde degeneration in the thalamus differs in certain respects from that found in mammals; cell loss is not accompanied by any appreciable glial reaction, and consequently partial degeneration is more difficult to evaluate. The cellular degeneration appears to have reached its maximum by 28 days, and there is no further appreciable change in the appearance of the affected nuclei after 82 days.

In Exp. OL 71 (survival 44 days) which is representative of the first group, the lesion is strictly unilateral, and almost the entire telencephalon has been removed (Text-fig. 1). The anterior third of the right hemisphere has been completely destroyed, and with the exception of a small portion of the medial hippocampal area all the cortex of this hemisphere has been removed, together with the entire dorsal striatum, lateral part of the palaeostriatum, nucleus sphaericus and the central amygdaloid mass. The most rostral part of the remaining tissue consists of

the small-celled area of the hippocampal cortex, and more caudally the posterior half of the septum and the ventromedial corner of the hemisphere, including the nucleus of the diagonal band and the medial palaeostriatum, are preserved. The medial palaeostriatum appears to be shrunken, presumably because of the loss of fibres passing through this region, and the cells are swollen. The medial and lateral forebrain bundles have undergone extensive direct involvement. The medial and lateral preoptic areas are intact, but there is marked atrophy of the forebrain bundles at this level.



Text-fig. 1. Drawings made by means of a projection apparatus of transverse Nissl-stained sections to show the extent of the lesion and the distribution of the thalamic degeneration in Expt. OL71. On the operated side only the hippocampal cortex, posterior part of the septum and the ventromedial corner of the hemisphere are preserved.

In the thalamus severe retrograde cell degeneration is found in the nucleus rotundus where virtually no normal cells remain. Marked cell loss has occurred, and it is difficult to distinguish the residual shrunken neurons from glial elements. The entire nucleus is involved, but the cell loss is more pronounced in the caudal half. In this brain (as in all others regardless of survival period) retrograde cell degeneration is not accompanied by glial proliferation (Pl. 2. fig. 4; Pl. 3, fig. 5). The only other thalamic nucleus in which degeneration is found is that portion of the nucleus

dorsomedialis anterior lying medial and ventral to the nucleus rotundus. In this nucleus there is little, if any, cell loss, but there is distinct shrinkage of all the cells, and the nucleus as a whole is smaller than on the normal side. As no other nuclei show degeneration in this or in any other experiment, the nucleus rotundus and the adjacent part of the nucleus dorsomedialis anterior appear to constitute the total thalamic projection upon the telencephalon as determined by this technique.

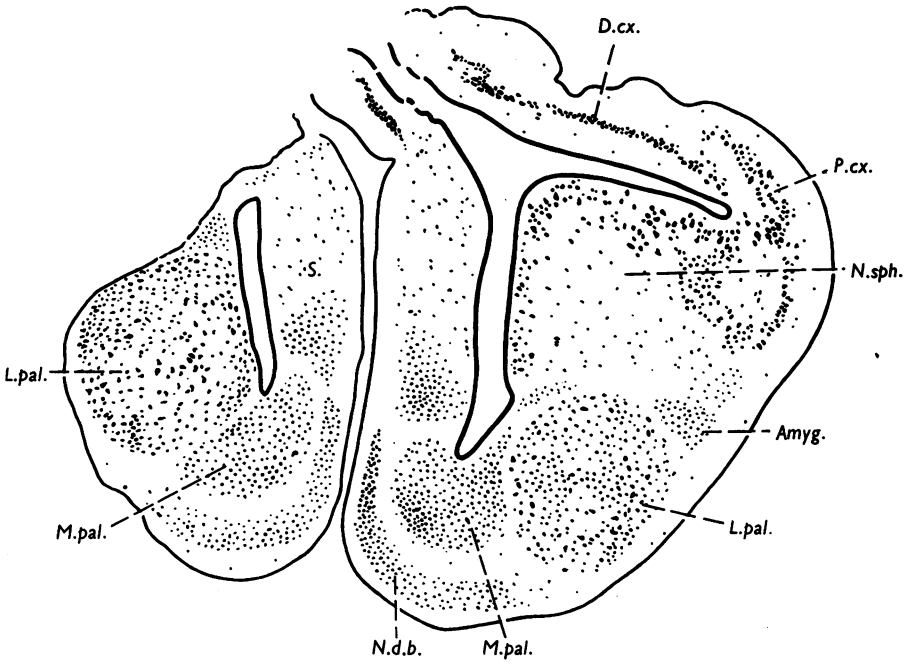
Another experiment (OL 65) is essentially similar to OL 71 except that there is less extensive involvement of the medial palaeostriatum and a little more damage to the septum; in addition there is slight involvement of the dorsolateral margin of the lateral preoptic area. In the thalamus the changes are similar to those described in the previous experiment: the nucleus rotundus shows severe cell loss throughout its extent, and no normal cells remain; in the adjacent part of the nucleus dorsomedialis anterior all the cells are shrunken (Pl. 3, fig. 5).

In an additional series of sections of this brain stained with Bodian's protargol method the thalamic components of the lateral forebrain bundle have been studied. On the normal side three separate groups of fibres can be seen passing from the dorsal aspect of the lateral forebrain bundle to enter the thalamus. In the rostral part of the diencephalon an anterior component is given off which first passes backwards and upwards and then forwards to reach the anterior parts of the dorsal nuclei. More posteriorly an intermediate component passes upwards and medially to the nucleus rotundus and the nucleus dorsomedialis anterior, and at a slightly more posterior level a medial component passes medial to these nuclei. This description agrees closely with that given for these fibre bundles in the alligator brain by Huber & Crosby (1926), but it has not been possible to distinguish in *Lacerta* the internal component described by these authors in the alligator. It should be noted that immediately posterior to the level at which the intermediate component is given off to the nucleus rotundus the dorsal peduncle of the lateral forebrain bundle undergoes a marked decrease in size, but the ventral peduncle can be clearly traced backwards through the diencephalon to the mid-brain.

On the operated side of this brain both the medial and lateral forebrain bundles show severe fibre loss, but this is more marked in the lateral bundle, the lateral two-thirds of which are almost totally devoid of fibres at the level of the preoptic areas (Pl. 3. fig. 6). Some fibres remain in the medial one-third of the lateral forebrain bundle, and these fibres can be seen to join (or leave) the bundle as it passes through the lateral preoptic area. More posteriorly in the diencephalon clear spaces sharply outline the degenerated dorsal and ventral peduncles. The anterior and intermediate thalamic components of the dorsal peduncle are severely degenerated, and only a small proportion of the normal fibres remain (Pl. 4, figs. 7, 8). The medial component, however, appears to be unchanged. The dense fibre plexus of the nucleus rotundus is not appreciably reduced, and in the posterior part of the nucleus the afferent fibres from the tecto-thalamic tract can be seen passing in to form the main contribution to this plexus.

The results of this experiment indicate that the axons of the cells of the nucleus rotundus and the adjacent part of the nucleus dorsomedialis anterior pass into the intermediate component of the lateral forebrain bundle to reach the telencephalon, but they do not preclude the possibility that there are also descending fibres in this

component. The profound degree of degeneration of the anterior thalamic subdivision, taken together with the absence of any retrograde cell degeneration in the anterior parts of the nuclei dorsomedialis anterior and dorsolateralis anterior, strongly suggests that this subdivision is made up largely of fibres which arise in the telencephalon and pass to terminate in these nuclei.



Text-fig. 2. Drawing traced from a section to show the extent of the lesion in Expt. OL 73. The palaeostriatum is completely preserved as the ventral limit of the lesion coincides with the dorsolateral margin of the lateral palaeostriatum.

The second group of experiments indicates that those nuclei which project upon the telencephalon do not project upon the cortex, dorsal striatum and nucleus sphaericus. The best example is experiment OL 73 (survival 80 days) in which no thalamic degeneration is found after extensive involvement of these telencephalic structures (Text-fig. 2). The lesion in the left hemisphere begins near the anterior pole of the hemisphere where it involves the dorsolateral surface, and it rapidly increases in extent so that all three cortical areas, the entire dorsal striatum and the nucleus sphaericus are destroyed. The extent of the lesion remains essentially the same up to the caudal end of the hemisphere where a small portion of hippocampal cortex is preserved. Throughout its antero-posterior extent the ventral limit of the lesion coincides with remarkable precision to the dorsolateral border of the lateral palaeostriatum (Pl. 1, fig. 1). Medially the lesion reaches the junction of the hippocampal cortex and the septum. The septum is not directly involved, but is slightly shrunken and shows compacting and apparent increase of cells, presumably the result of atrophy of fibres of passage. The preoptic areas are also intact. In addition, there has been slight damage to the dorsomedial margin of the right hemisphere.

In several other experiments similar but smaller lesions in the cerebral hemisphere have not resulted in any thalamic degeneration.

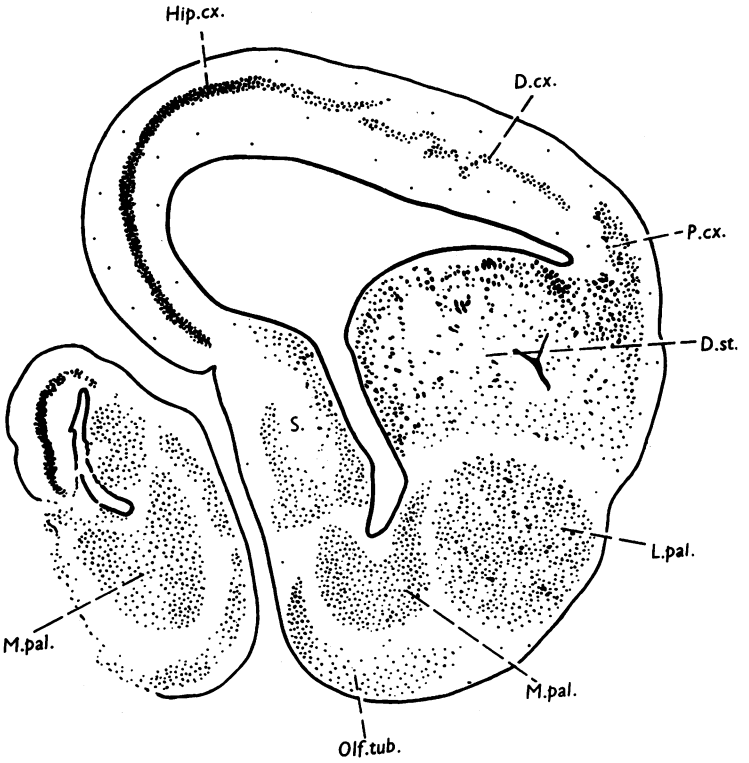
The Bodian-stained sections of this experiment show that a distinct loss of fibres has occurred in the lateral half of the lateral forebrain bundle at the level of the preoptic areas. The medial and intermediate thalamic components show no appreciable change, but there has been a distinct loss of fibres from the anterior component. The severity of this degeneration is not as marked as in the previous experiment, but it would appear that about half of the normal complement of fibres has degenerated. From a correlation of the extent of the lesion with the fibre loss in this and the previous experiment it is apparent that the intermediate thalamic component of the lateral forebrain bundle is intimately related to either the palaeostriatum or the amygdala because the preservation of this component in Exp. 73 is undoubtedly due to the lack of involvement of these more ventrally situated telencephalic structures. Moreover, it may also be concluded that if this component of the lateral forebrain bundle contains fibres descending from the telencephalon to the thalamus these fibres must arise in these two structures. The finding that no appreciable number of fibres of the intermediate component has degenerated after such an extensive lesion suggests that the thalamic projection upon the telencephalon is localized rather than diffuse. As the severity of the degeneration of the anterior component in this experiment is not as marked as in OL 65 it is clear that this bundle receives fibres partly from the palaeostriatum and amygdala on the one hand and partly (at least one-half) from the dorsal striatum, nucleus sphaericus and cortex on the other hand.

In seven other experiments with smaller lesions involving these three more dorsally placed structures both thionine and Bodian series have been prepared. In all of these brains the medial and intermediate components are found to be unaffected, but the anterior component shows varying degrees of fibre loss. The severity of this loss is proportional to the extent of the damage to the dorsal striatum and nucleus sphaericus, and minimal degeneration is seen in those brains in which the striatal involvement is slight. In three further experiments the lesion is virtually restricted to the cortex, and in these brains Bodian-stained sections show no appreciable change in the lateral forebrain bundle or in any of its thalamic components. These results indicate that in *Lacerta* the number of fibres which enter or leave the dorsal cortex from the lateral forebrain bundle is very small, and this agrees with Goldby's findings (1937).

If the thalamic projection is localized rather than diffuse, it would follow from the results of the preceding experiments that the termination of the thalamic axons must be the palaeostriatum or the central amygdaloid mass. The first experiment of the third group (OL 66, survival 28 days) not only excludes the medial part of the palaeostriatum but also shows that degeneration in the thalamus occurs after involvement of the lateral palaeostriatum and the central amygdaloid mass. The central amygdaloid mass is that group of cells found dorsal and posterior to the lateral palaeostriatum and described in detail by Goldby (1934); it is morphologically distinct from the nucleus sphaericus with which it is often grouped as the reptilian homologue of the mammalian amygdala.

The lesion in OL 66 has destroyed the frontal pole of the hemisphere and has

severed the olfactory peduncle (Text-fig. 3). The ventromedial corner of the hemisphere reappears at the level of the rostral end of the medial palaeostriatum and slightly more caudally the septum and hippocampal cortex are preserved. Back to the level of the preoptic areas, the structures which are intact include virtually the entire medial palaeostriatum, the nucleus of the diagonal band, septum and the small-celled portion of the hippocampal cortex. The septum shows the same features of cellular compacting as were described in OL 73. The lesion extends posteriorly to completely destroy the posterior pole of the hemisphere, but the preoptic areas and

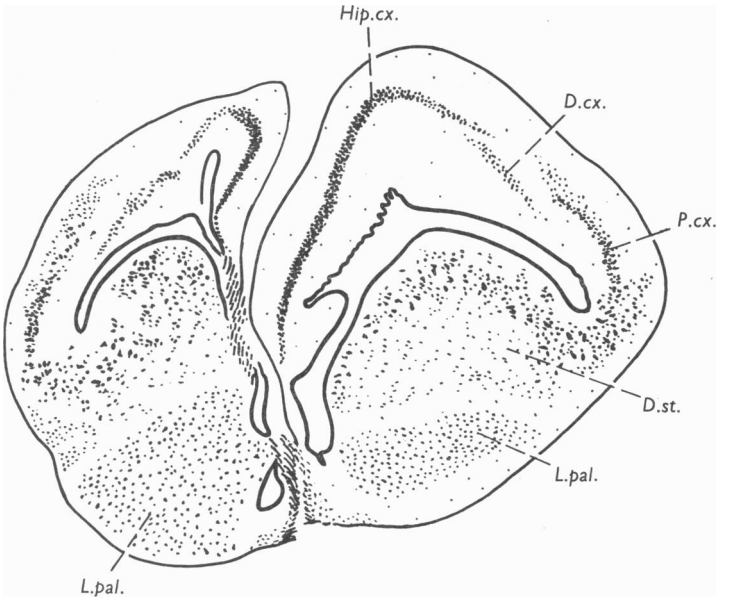


Text-fig. 3. Drawing traced from a section of the cerebral hemispheres of Expt. OL 66. The medial part of the palaeostriatum has not been damaged on the operated side, and the other structures which are preserved include the hippocampal cortex, the septum and the nucleus of the diagonal band.

the forebrain bundles are not directly involved. The thalamic degeneration is restricted to severe cell loss in the nucleus rotundus and marked shrinkage of the cells of the adjacent portion of the nucleus dorsomedialis anterior (Pl. 2. fig. 4). The only structures involved by the lesion in this experiment which are not destroyed in OL 73 (in which no degeneration is found) are the lateral palaeostriatum and central amygdaloid mass.

The next experiment of the third group to be described strongly suggests that the thalamic degeneration results from destruction of the lateral palaeostriatum. In animal OL 67 (survival 28 days) there is a bilateral lesion involving different portions

of the palaeostriatum on each side but with unilateral retrograde degeneration in the thalamus. This lesion has been produced by a needle inserted between the two hemispheres resulting in asymmetrical destruction of the ventromedial regions of both sides (Text-fig. 4). In the right hemisphere the damage begins close to the frontal pole medial to the ventricle, and the medial hippocampal cortex and olfactory tubercle are destroyed. The medial margin of the dorsal striatum is slightly involved only near its rostral extremity, and further caudally the entire extent of the ventricular margin of the striatum is intact. The damage is restricted to the medial and lateral palaeostriatum ventral to the inferior angle of the ventricle, the dorsolateral part of the lateral palaeostriatum being preserved. The damage remains restricted to the palaeostriatum, and posteriorly the lesion ends abruptly



Text-fig. 4. Drawing traced from a section to show the asymmetrical damage to the cerebral hemispheres in Expt. OL 67. On the left side the lateral palaeostriatum is uninvolved, but on the right side only the dorsal part of this structure is preserved.

at the level of the appearance of the nucleus of the diagonal band. It should be emphasized that the central amygdaloid mass, together with the cells capping the lateral forebrain bundle, are uninvolved. The lateral forebrain bundle shows slight atrophy. The retrograde cell degeneration in nucleus rotundus is less severe than in the experiments previously described but is quite definite. There is little cell loss, but all the cells are considerably shrunken. Similar changes have occurred in the adjacent portion of the nucleus dorsomedialis anterior. On the left side only the medial palaeostriatum has been destroyed, below and medial to the lateral ventricle over the same antero-posterior extent as the damage on the right side. There is no involvement of the lateral palaeostriatum. No retrograde cell degeneration is seen in the thalamus on this side.

The absence of degeneration on the left side after such a lesion again excludes the

medial palaeostriatum as a projection area of the degenerated thalamic nuclei. Furthermore, the lack of involvement of the amygdaloid complex by the lesion on the right side of this experiment indicates that this structure can be included with those regions which have already been eliminated as the thalamic projection area in previous experiments. It is possible that fibres do pass from the thalamus to the amygdaloid complex through the adjacent lateral palaeostriatum, and that the degeneration in this experiment could be interpreted as being the result of the interruption of these fibres. This possibility appears unlikely, however, unless the fibres first pass anteriorly through the lateral palaeostriatum and then curve back to the amygdaloid complex. The findings in this experiment, taken together with those previously described, show that the only common factor in all lesions resulting in thalamic degeneration is involvement of the lateral palaeostriatum.

The final experiments of this group indicate that small lesions of the lateral palaeostriatum result in more restricted thalamic degeneration. In animal OL 52 (survival 28 days) only the posterior part of the lateral palaeostriatum has been involved, and partial degeneration is found in nucleus rotundus. In the anterior half of the hemisphere the hippocampal and general cortex have been destroyed, and the lateral ventricle opened. Although it has not been directly involved, the dorsal striatum is severely atrophied. Immediately rostral to the nucleus of the diagonal band the needle used to make the lesion has passed through the inferior angle of the ventricle into the dorsal part of the medial palaeostriatum. The damage to the medial palaeostriatum does not increase posteriorly, but the caudal half of the lateral palaeostriatum and the overlying dorsal striatum at this level have been completely destroyed. The central amygdaloid mass is extensively involved, but the cells overlying the lateral forebrain bundle (Goldby's area *C*) and the nucleus sphaericus are preserved. The damage to the cortex progressively diminishes in the caudal half of the hemisphere. The anterior half of the nucleus rotundus shows no appreciable change, but in the posterior half there is severe cell shrinkage and partial cell loss. The nucleus dorsomedialis anterior adjacent to the degenerated portion of nucleus rotundus shows no distinct changes, but in the anterior half (adjacent to the undegenerated part of nucleus rotundus) the cells appear shrunken. In another experiment, OL 89 (survival 82 days) a small lesion has destroyed the posterior part of the lateral palaeostriatum together with the immediately adjacent part of the central amygdaloid mass and the dorsal striatum. This brain has been cut in the horizontal plane so that the cell shrinkage, which has occurred in the nucleus rotundus and the adjoining nucleus dorsomedialis anterior as a result of this circumscribed area of damage, is clearly seen (Pl. 2, fig. 3).

In three other brains (OL 80, 87 and 93), in which there was less extensive involvement of the posterior part of the palaeostriatum, partial retrograde cell degeneration has occurred in the nucleus rotundus, the cells of the posterior half of the nucleus being considerably shrunken. In two of these experiments an additional series of Bodian-stained sections has been available. These series show that the anterior thalamic component of the lateral forebrain bundle has undergone severe degeneration, and that although the rostral part of the intermediate component is unaltered its caudal part is partially degenerated.

DISCUSSION

From the results of the present experiments it would appear that in *Lacerta viridis* the total thalamic projection upon the telencephalon, as determined by the method of retrograde cell degeneration, consists of the nucleus rotundus and the adjacent part of the nucleus dorsomedialis anterior. The cells of these two nuclei could either project diffusely to make contact with several structures through terminal branching of their axons or by way of collaterals. From a correlation of the site and extent of the lesion on the one hand with the thalamic degeneration on the other it may be concluded that the projection is restricted to the lateral part of the palaeostriatum. Thus damage of the lateral part of the palaeostriatum is always found to result in cellular degeneration in these two nuclei, but no changes are found after extensive lesions involving most of the other structures of the cerebral hemisphere. The palaeostriatum is generally subdivided into a medial 'olfacto' part connected mainly with the hypothalamus and a lateral 'somatic' part connected with the thalamus. The present experimental results show that this morphological subdivision is justified in so far as the two parts have differential connexions from the thalamus.

In those experiments in which the involvement of the lateral palaeostriatum is circumscribed partial degeneration is found in the thalamic nuclei. The distribution of the degeneration varies with the position of the lesion, and the areas affected in the two nuclei vary independently of each other. Although this evidence is derived from few experiments, and partial degeneration is difficult to evaluate because of the absence of accompanying gliosis, it does suggest that there is a topical organization in the thalamic projection in the antero-posterior direction at least, and also that the two nuclei project independently.

There is a distinct qualitative difference in the reaction of the cells in the nucleus rotundus and in the nucleus dorsomedialis anterior after removal of the telencephalon. The degeneration in the nucleus rotundus is the more severe and appreciable cell loss occurs, whereas only cell shrinkage is apparent in nucleus dorsomedialis anterior. Such differences in cellular reaction almost certainly reflect distinct differences of axonal distribution, and the persistence of the cells in nucleus dorsomedialis anterior suggests that they give off collateral branches to other parts of the diencephalon or brain stem.

Although the nucleus dorsomedialis anterior in *Lacerta viridis* cannot be subdivided on the basis of normal morphology it can be clearly differentiated into an anterior and posterior portion on the basis of differences of the reaction of the cells in these portions to removal of the telencephalon. The main anterior part of the nucleus shows no appreciable changes, but in the posterior extension of the nucleus lying medial to the nucleus rotundus the cells show definite shrinkage. The results of these experiments indicate, therefore, that these two parts of the nucleus have different efferent connexions and are also probably distinct functionally. In the corresponding position in the thalamus of the alligator Huber & Crosby (1926) have differentiated two nuclei on the basis of their morphological differences, the nucleus dorsomedialis anterior, and more posteriorly, adjacent to the nucleus rotundus, the nucleus medialis which in turn could be subdivided into an anterior and a posterior part. These authors conclude that few, if any, of the fibres of the lateral forebrain

bundle are related to the nucleus dorsomedialis anterior, but that the nucleus medialis anterior is connected with the striatum through the intermediate thalamic component of this bundle and the nucleus medialis posterior through the medial component. It would appear that the nucleus dorsomedialis anterior of *Lacerta viridis* is homologous with the more differentiated nucleus medialis anterior of the alligator on the basis of its position and connexions.

The simplest interpretation of the absence of cellular degeneration in other thalamic nuclei is that these nuclei do not project upon the telencephalon. In general it may be stated that most of the comparable evidence from experimental work on the thalamic projection in the mammal would support such an interpretation. An important corollary of this interpretation would be the absence of a thalamic projection to telencephalic structures other than the palaeostriatum, and in particular the general cortex. Because of the limitations of the method of retrograde cell degeneration, and especially in view of the limited experience of this technique in the reptile, this lack of cellular change cannot be considered to exclude completely the possibility of an additional projection to the telencephalon from these nuclei. The axons of the cells of these nuclei could terminate in the telencephalon, but a collateral may be given off to the diencephalon or to the brain stem which may be sufficient to maintain the integrity of the cell. It would be of interest to determine which of these possibilities is correct by tracing the course and termination of the degenerating fibres following lesions in the thalamus.

The findings in the Bodian-stained sections must also be interpreted with caution as this technique only demonstrates fibre loss of considerable degree. The present observations are concerned only with the lateral part of the basal forebrain bundle and its dorsal peduncle and thalamic components; no reference will be made to any changes in other tracts or commissures. The results of these experiments indicate that the fibres in the three thalamic components have different origins. The medial thalamic component shows no appreciable change after virtually complete removal of the telencephalon, which suggests that its constituent fibres arise and terminate in the diencephalon or brain stem. The intermediate thalamic component only degenerates if the palaeostriatum is involved, and the close correlation between the fibre degeneration in this component and the cellular degeneration in the nucleus rotundus and nucleus dorsomedialis anterior—whether total or partial—makes it highly probable that this component is composed largely of the efferent fibres from these nuclei. The anterior thalamic component is related to both the palaeostriatum and the dorsal striatum, as it shows almost complete atrophy after destruction of both these structures and a loss of approximately one half of its fibres after involvement of the dorsal striatum only; few, if any, of the fibres of this component are related to the cortex. The absence of any cellular change in the closely related anterior group of thalamic nuclei strongly suggests that the fibres of this subdivision are descending from the striatum to the thalamus. In this discussion of the constitution of the lateral forebrain bundle the palaeostriatum has not been subdivided into medial and lateral parts as there is insufficient evidence to justify this distinction. If the results on the fibre loss are considered in the light of the retrograde cell degeneration findings, however, it is clear that the intermediate thalamic component, at least, is related to the lateral palaeostriatum.

In general, these conclusions agree with those derived from the study of normal material (Kappers, Huber & Crosby, 1936) because it has long been known that the lateral forebrain bundle forms the main pathway between the striatum and the thalamus, but it has not been possible to be certain of the direction of conduction of the fibres. They are also in general accord with the experimental work of Goldby (1937) using the Marchi technique. Goldby concludes that few fibres from the cortex enter the lateral forebrain bundle, that most of the fibres in this bundle are efferent from the thalamus and that the majority of them end in the striatum. The two differences between his conclusions and those presented above are not serious. Firstly, the apparent absence of degeneration in the anterior thalamic component in the Marchi material is probably to be explained in part by the fact that Goldby is referring mainly to that part of the dorsal peduncle related to the nucleus rotundus (where fibre degeneration would not be expected), and partly because, as he emphasizes, the Marchi technique only demonstrates degenerated myelinated fibres. Secondly, although Goldby concludes that most of the efferent fibres from the thalamus end in the striatum he considers that they are particularly related to the hypopallial part; he states, however, that the degree of this degeneration is relatively slight, and secondary to a direct lesion of the lateral forebrain bundle and palaeostriatum so that the origin of these ascending fibres is not certain, and they could have arisen from the palaeostriatum itself.

The most significant result of the present series of experiments is the finding that the thalamic projection upon the telencephalon in *Lacerta* is restricted to the lateral part of the palaeostriatum. This result is particularly surprising in view of the descriptions from normal material of thalamic efferent connexions with other parts of the striatum (Kappers, Huber & Crosby, 1936), and it immediately raises the problem of the origin of the afferent connexions to other structures such as the dorsal striatum, the nucleus sphaericus and the dorsal cortex. The finding of a restricted thalamic projection in the lizard is strikingly similar to that obtained with the same technique in the avian brain where it has been shown that most of the thalamic nuclei (including the nucleus rotundus and the nucleus ovoidalis), which degenerate after removal of the telencephalon, are related to the palaeostriatum augmentatum (Powell & Cowan, 1957). On the basis of their morphology and of their connexions one might therefore be tempted to suggest that the nucleus rotundus and the adjacent part of the nucleus dorsomedialis anterior of the lizard are homologous with the nucleus rotundus and nucleus ovoidalis of the bird, and that the lateral part of the palaeostriatum of the reptilian brain is the homologue of the avian palaeostriatum augmentatum. The question as to which structures are homologous in the brains of *modern* reptiles and birds is much more complex than this, however, and it is probably sufficient to say that in using as a criterion the fibre connexions between two structures it is not necessarily to be inferred that the thalamus or the palaeostriatum of the reptile is homologous in its nuclear components with the corresponding regions in the bird. Experimental evidence of the afferent connexions of the thalamic nuclei in both groups of animals and of the other afferent and efferent connexions of the palaeostriatum would help to clarify this problem. The conclusion that may be drawn is that in both the reptilian and avian brain the telencephalic projection of the thalamus is predominantly to the palaeostriatum. This similarity

of the thalamic projection is perhaps not unexpected in view of the closely related evolutionary history of the two groups and the generally accepted view of the close resemblance between the brains, the avian brain differing mainly in the degree of development of the striatum. As Goldby & Gamble (1957) have recently pointed out, even more difficulty would be encountered if the attempt were made to draw homologies between the reptilian and mammalian brain where the thalamus is known to be intimately related to the neocortex. On the one hand, there is no evidence to indicate whether the palaeostriatum of the reptile is homologous with the globus pallidus or the caudate nucleus and putamen of the mammal, and on the other hand the reptilian thalamus may be the homologue not of the mammalian dorsal thalamus but of one of the other main subdivisions of the diencephalon such as the subthalamus. That the thalamus of these two classes is probably organized in a fundamentally different manner is suggested by the differences which have been found in the central connexions of the optic nerve. The dorsal nucleus of the lateral geniculate body of the mammal receives optic nerve fibres, and its cells either project upon the neocortex or are interneurons; there is no unequivocal evidence to indicate that the ventral nucleus of the lateral geniculate body receives optic nerve fibres. The lateral geniculate nucleus of *Lacerta* has also been shown to be the site of termination of fibres of retinal origin (Armstrong, 1950) but observations on normal material have shown that its predominant efferent connexions are with the tectum of the midbrain and not with the telencephalon, and these observations are in agreement with the findings of the present investigations in which no retrograde cell degeneration has been found in the nucleus after removal of the telencephalon. One may speculate as to whether the predominant striatal projection of the thalamus which has been found in the reptile and bird may not form the basic pattern, upon which the close thalamo-cortical relationship of the mammalian dorsal thalamus has been superimposed as a specialized evolutionary development.

SUMMARY

1. The projection of the thalamus upon the telencephalon in *Lacerta viridis* has been investigated using the method of retrograde cell degeneration. A study of Bodian-stained sections of the same experimental material has provided evidence of the origin of certain components of the lateral forebrain bundle.

2. After virtually complete removal of the telencephalon retrograde cell degeneration in the thalamus is restricted to the nucleus rotundus and the adjacent part of the nucleus dorsomedialis anterior.

3. Analysis of the retrograde cell degeneration in the thalamus following lesions of varying size has shown that these nuclei project to the lateral palaeostriatum.

4. The intermediate thalamic component of the lateral forebrain bundle consists largely of the efferent fibres from the nucleus rotundus and the adjacent part of the nucleus dorsomedialis anterior to the lateral palaeostriatum. The anterior component is composed of fibres which arise from the dorsal striatum, nucleus sphaericus and palaeostriatum. There are few, if any, fibres in the lateral forebrain bundle which are related to the dorsal cortex.

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ABBREVIATIONS

A.	Anterior thalamic component of lateral forebrain bundle.
Amyg.	Central amygdaloid mass.
Area 'c'	Area *C* of the amygdala described by Goldby (1934)
D.cx.	Dorsal cortex.
D.st.	Dorsal striatum.
Hip.cx.	Hippocampal cortex.
I.	Intermediate thalamic component of lateral forebrain bundle.
L.	Lateral forebrain bundle.
L.f.b.	Lateral forebrain bundle
L.pal.	Lateral palaeostriatum.
M.	Medial forebrain bundle.
M.pal.	Medial palaeostriatum.
N.d.b.	Nucleus of the diagonal band.
N.dors.lat.ant.	Nucleus dorsolateralis anterior.
N.dors.med.ant.	Nucleus dorsomedialis anterior.
N.lat. genic. (lat.)	Lateral part of lateral geniculate nucleus.
N.lat. genic. (med.)	Medial part of lateral geniculate nucleus.
N.sph.	Nucleus sphaericus.
N.supraped.	Suprapeduncular nucleus.
Olf.tub.	Olfactory tubercle.
P.cx.	Pyriform cortex.
R.	Nucleus rotundus.
S.	Septum.

EXPLANATION OF PLATES

Plate 1

Fig. 1. Photomicrograph showing the extent of the lesion in Expt. OL 73; note the complete preservation of the lateral palaeostriatum. Stained with thionine. × 36.

Fig. 2. Photomicrograph to show retrograde cell degeneration in the nucleus rotundus and the adjacent part of the nucleus dorsomedialis anterior in Expt. OL 56. Stained with thionine. × 40.

Plate 2

Fig. 3. Photomicrograph of a horizontal section to show severe cell shrinkage in the nuclei rotundus and dorsomedialis anterior in Expt. OL 89. Stained with thionine. × 69.

Fig. 4. Photomicrograph showing severe cell loss in nucleus rotundus and cell shrinkage in nucleus dorsomedialis anterior in Expt. OL 66. Stained with thionine. × 87.

Plate 3

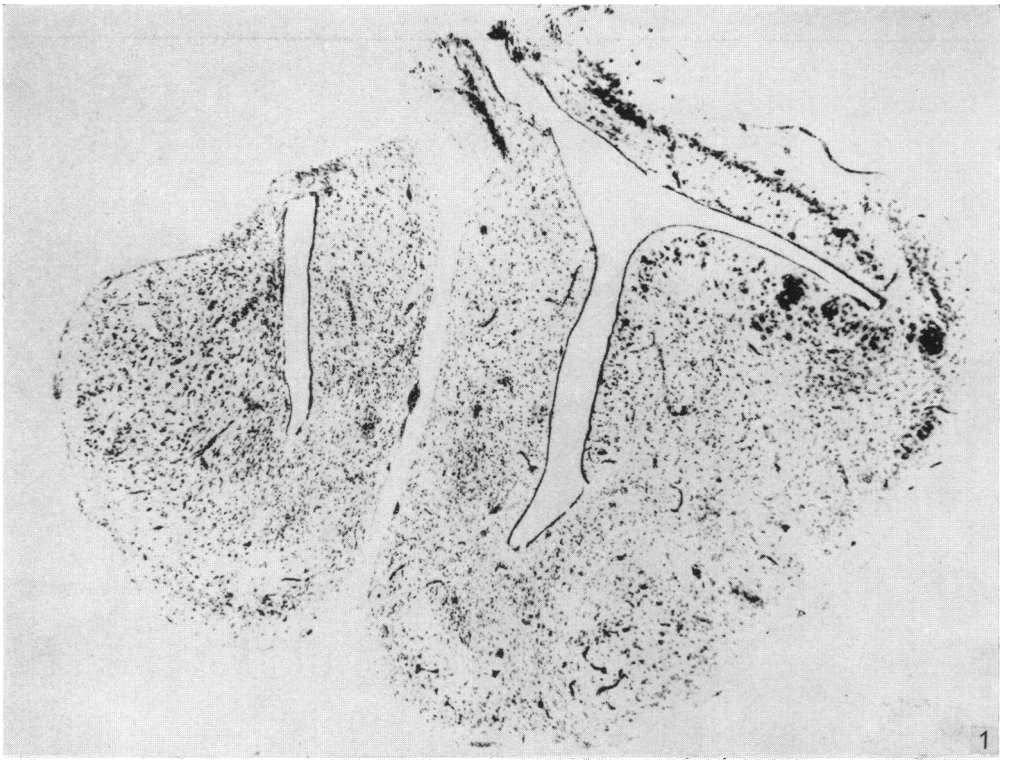
Fig. 5. Photomicrograph to show the retrograde cell degeneration in Expt. OL 65. Stained with thionine. × 93.

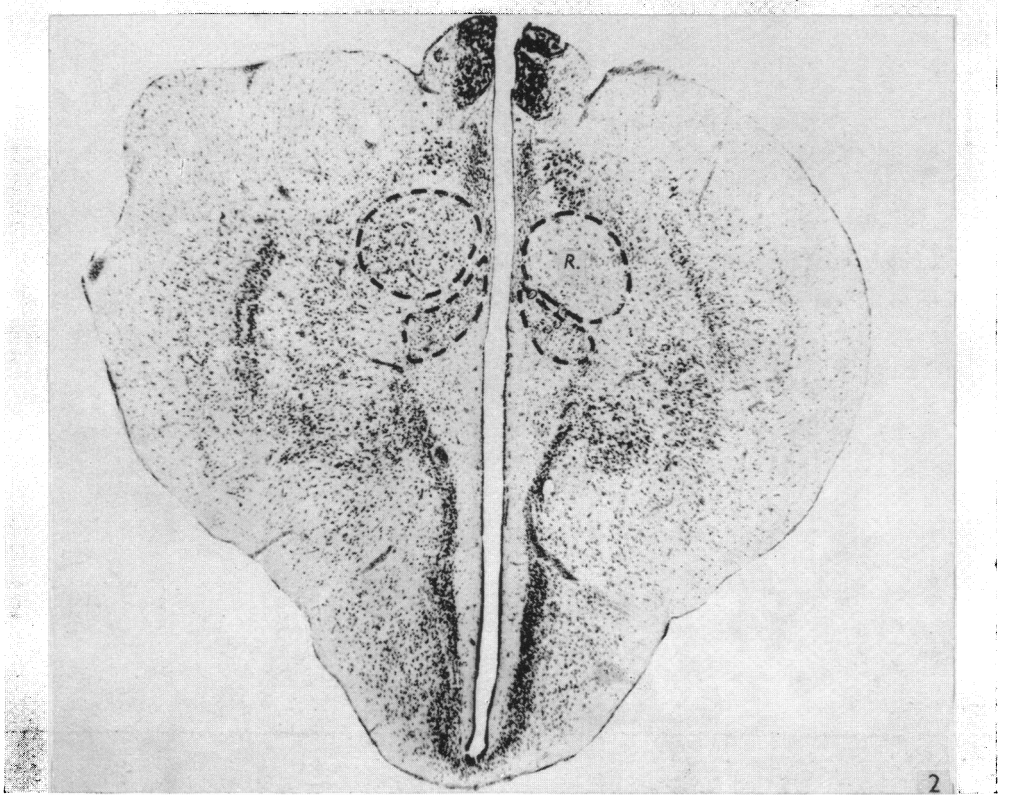
Fig. 6. Photomicrograph to show severe fibre loss in the medial and lateral forebrain bundles at the level of the preoptic areas in the same experiment as the preceding figure. Bodian's protargol stain. × 72.

Plate 4

Fig. 7. Photomicrograph showing degeneration of the anterior thalamic component of the lateral forebrain bundle on the right side of the same experiment. Bodian's protargol method. × 65.

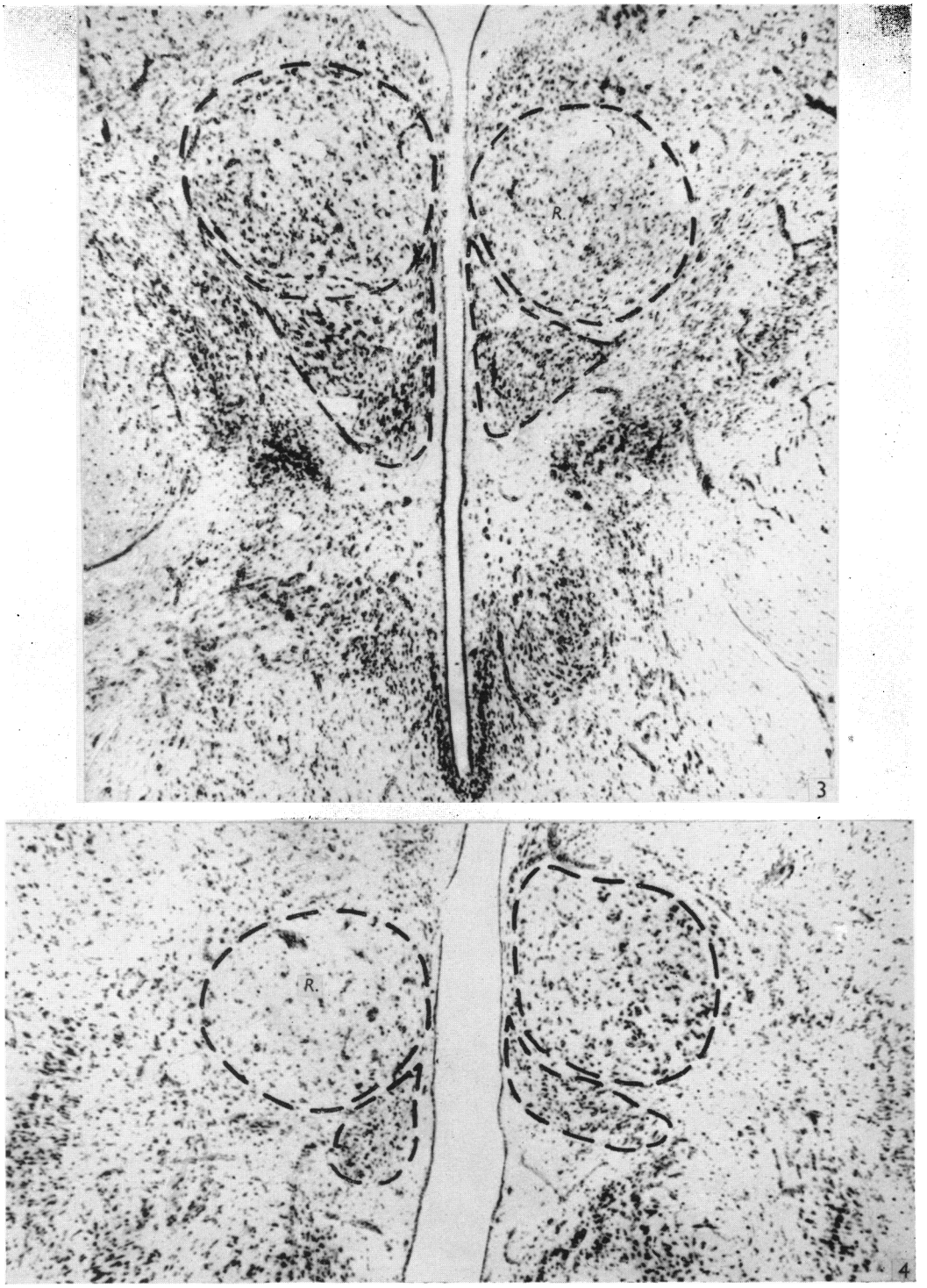
Fig. 8. Photomicrograph to show atrophy of the intermediate thalamic component of the lateral forebrain bundle on the right side of the same experiment. Bodian's method. × 80.





POWELL AND KRUGER. THALAMIC PROJECTION UPON THE TELENCEPHALON IN *LACERTA VIRIDIS*

(*Facing p.* 542)



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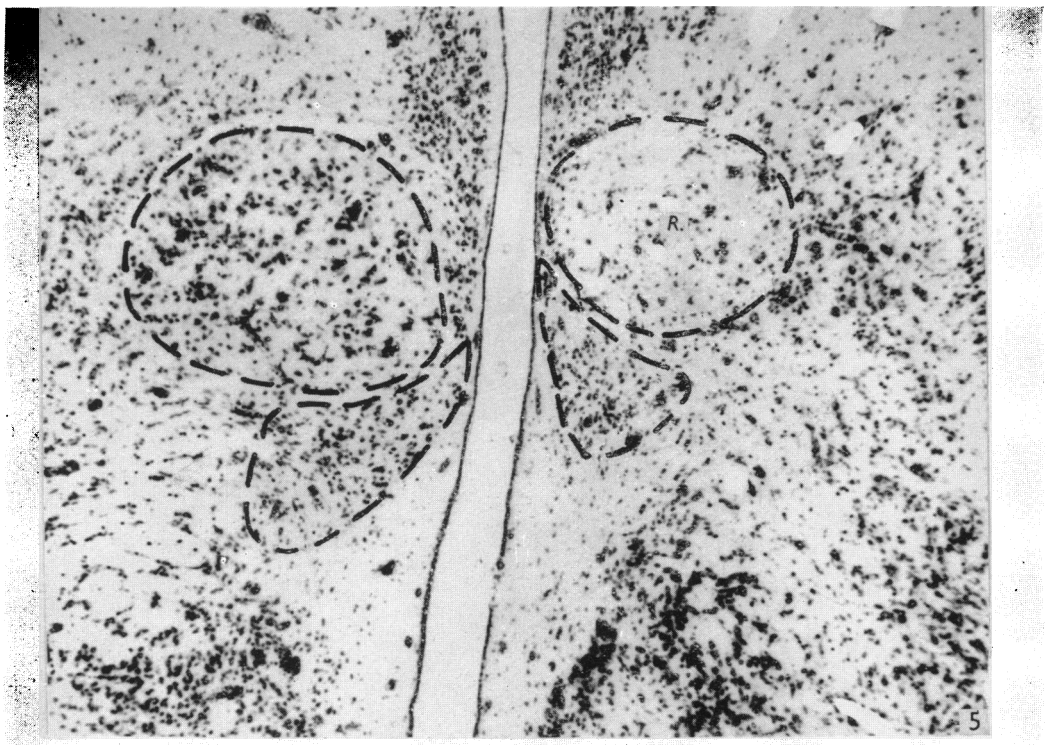

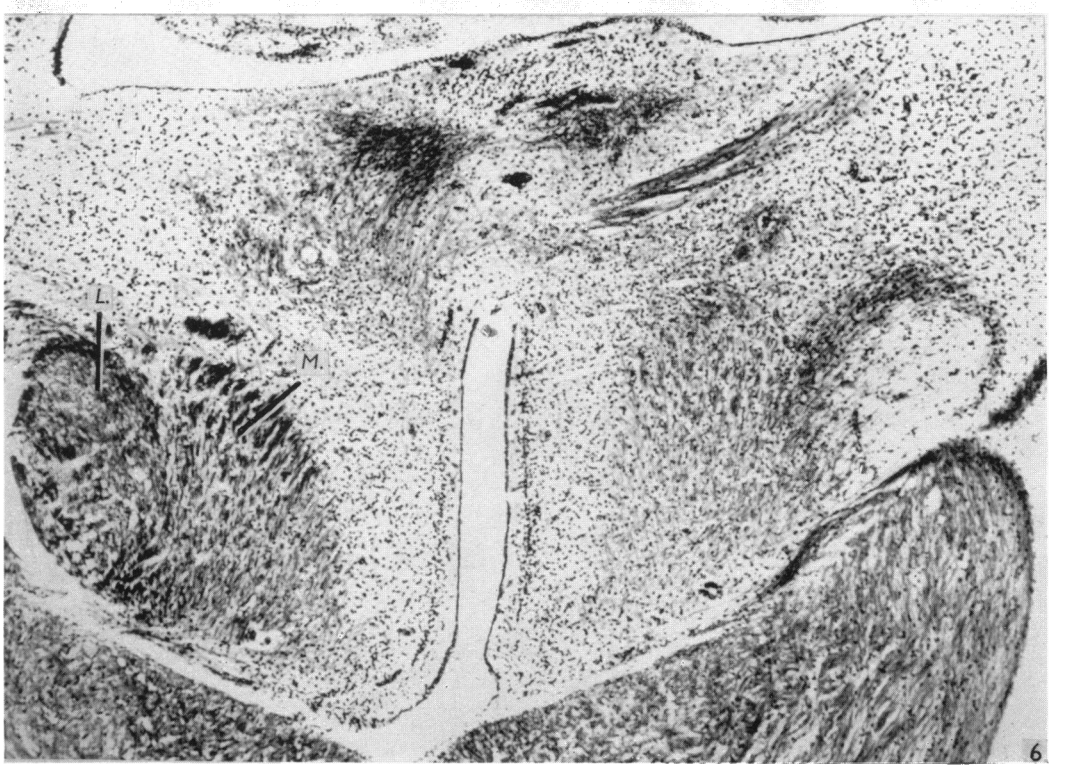

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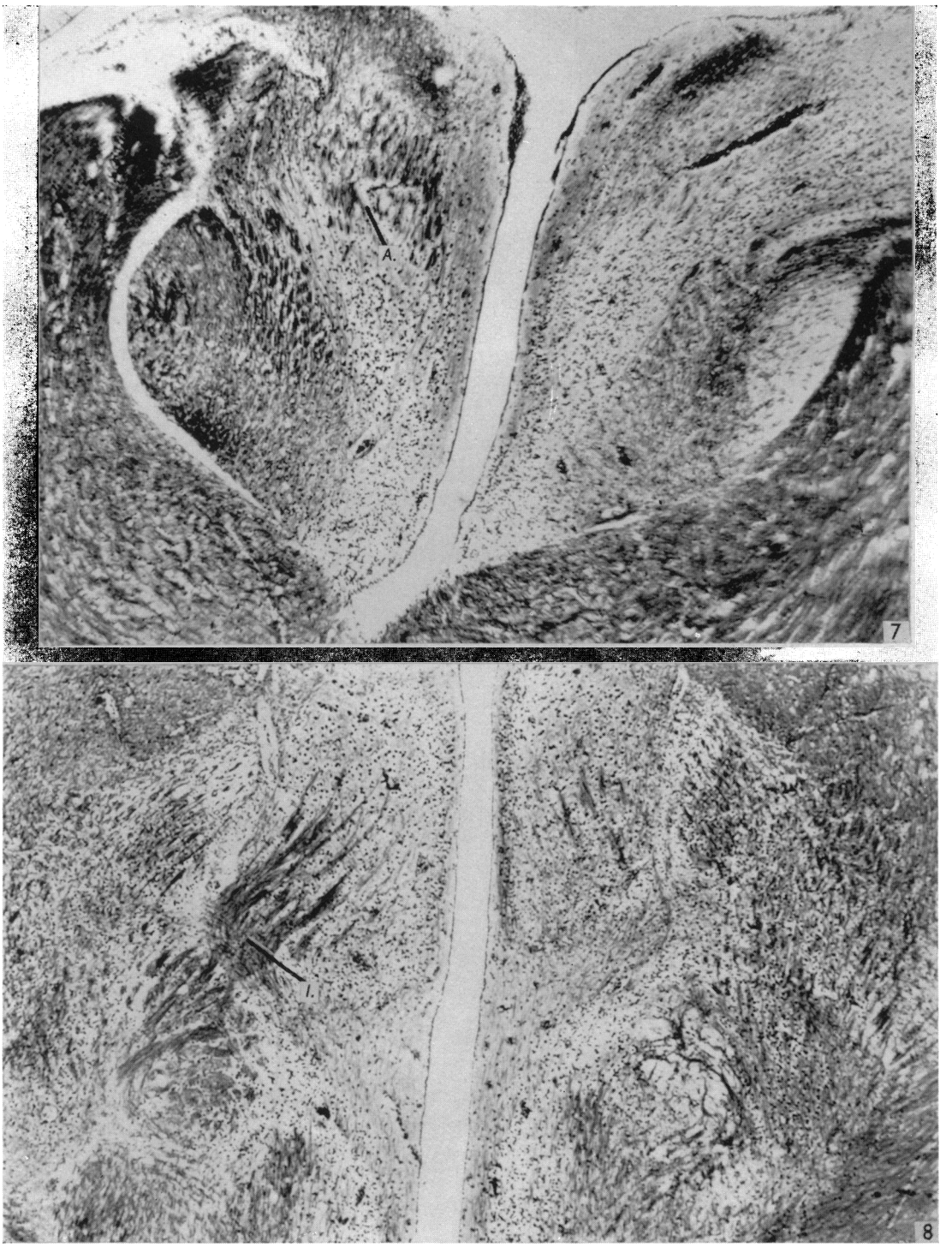

POWELL AND **KRUGER.** THALAMIC PROJECTION UPON THE TELENCEPHALON IN *LACERTA VIRIDIS*