THE THALAMIC PROJECTION UPON THE TELENCEPHALON IN THE PIGEON (*COLUMBA LIVIA*)

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INTRODUCTION

The exceptional development of the striatum in the avian telencephalon is unique amongst vertebrates and for this reason is of considerable interest to the comparative neuroanatomist. Associated with this unparalleled striatal development is the extreme reduction in size of the 'cortical' areas of the cerebral hemisphere. Nevertheless, the thalamus is well differentiated and is composed of a number of well-developed nuclei. The striking difference between the thalamic development and that of the cortex is surprising in view of the intimate thalamocortical relationship found in the mammal and suggests a threefold problem. First, can the well-differentiated avian thalamus be predominantly related to the poorly developed cortex (i.e. is the thalamic projection similar in principle—if not homologous—to that of the main thalamic nuclei in mammals)? A second possibility is that all or most of the thalamic nuclei of the bird project upon the striatum and form a system similar to that which has recently been described in the mammal. The third possibility to be considered is that the projection of the avian thalamus is an evolutionary specialization without an equivalent in mammals. It is difficult on *a priori* grounds to know which of these possibilities is the more likely to be correct, especially as so little is known of the functional significance of either the striatum or the thalamus in birds. However, it is obvious that the unique development of the avian striatum must be related to its distinctive mode of life, and a clearer knowledge of the connexions and organization of the striatum might help in the elucidation of its functional significance. In addition to this, any light thrown upon the morphology of the striatum will be of value in reconstructing the mode of evolutionary development of the forebrain.

There is a considerable literature dealing with the normal morphology of the forebrain in birds (Kappers, Huber & Crosby, 1936), but little work has been done using the conventional experimental anatomical techniques. The necessity for an experimental investigation of this kind need hardly be emphasized as it is well known that connexions can only be established with certainty using such methods. In this study we have attempted to define the projection of the thalamic nuclei upon the telencephalon by the method of retrograde cell degeneration which has been used so successfully in the mammal. The finding of retrograde cell degeneration in a given structure can be accepted as unequivocal evidence for a projection to the site of the lesion, but a negative result, of course, must be interpreted with caution.

In the present paper the results are presented in two sections: in the first the total thalamic projection upon the telencephalon will be described for, as Rose & Woolsey (1943) have pointed out, there is a distinct advantage in determining the total

thalamic projection by studying the extent of the retrograde cell degeneration in the thalamus after complete removal of the telencephalon before attempting to define the precise projection of individual thalamic nuclei. In the second section the projection of some of the individual nuclei will be presented based upon an analysis of the retrograde degeneration following a variety of smaller telencephalic lesions. A preliminary account of these results was given some time ago (Powell & Cowan, 1957).

MATERIAL AND METHODS

Altogether seventy adult pigeons (*Columba livia*) were operated upon, and of these the brains of fifty-two were used. For determining the total thalamic projection four brains have been useful and the remainder for the localization studies presented in the second section. The animals were anaesthetized with open ether and the skull exposed through a midline skin incision. Craniotomies varying in size and position were made and the lesions produced either with a needle or by suction with a fine aspirator. The animals were allowed to survive for periods ranging between 1 and 2½ months. They were killed by an overdose of ether and the brains fixed by immersion in 70% alcohol and 2% acetic acid. After embedding in paraffin wax the brains were cut at 25 μ in either the coronal or horizontal plane; a one-in-five series of sections was mounted and stained with thionine.

One of the unexpected features of this study was the ease with which lesions could be placed in the brain of the pigeon, and the remarkable ability to survive such extensive lesions as virtually complete removal of the telencephalon on one side. No detailed behavioural studies were made upon these animals, but we have been impressed by the absence of any overt functional deficit even after large lesions.

TERMINOLOGY

The terminology of the avian telencephalon and diencephalon has been clarified by Huber & Crosby (1929) and Kappers *et al.* (1936). These authors did not give an account specifically of the pigeon brain, but we have found that their detailed descriptions of other species, particularly the sparrow and dove, is so similar to the pigeon that a separate description of the normal morphology of the pigeon need not be given. For the same reasons we have followed the terminology of these authors with only minor modifications. For example, in our descriptions of the lesions in the telencephalon we have not differentiated between the various subdivisions of the hyperstriatum which they have described, not because they cannot be recognized in our material, but because we have found no evidence for differential connexions between the thalamus and these subdivisions. Similarly, although we have been able to differentiate the so-called ektostriatum from the adjacent neostriatum and palaeostriatum in normal material, it has not always been possible to recognize it clearly in the operated hemispheres, partly because of the resulting distortion, and partly because of distinct shrinkage of the constituent cells and accompanying gliosis. For these reasons we have normally included it with the neostriatum in the descriptions of the lesions.

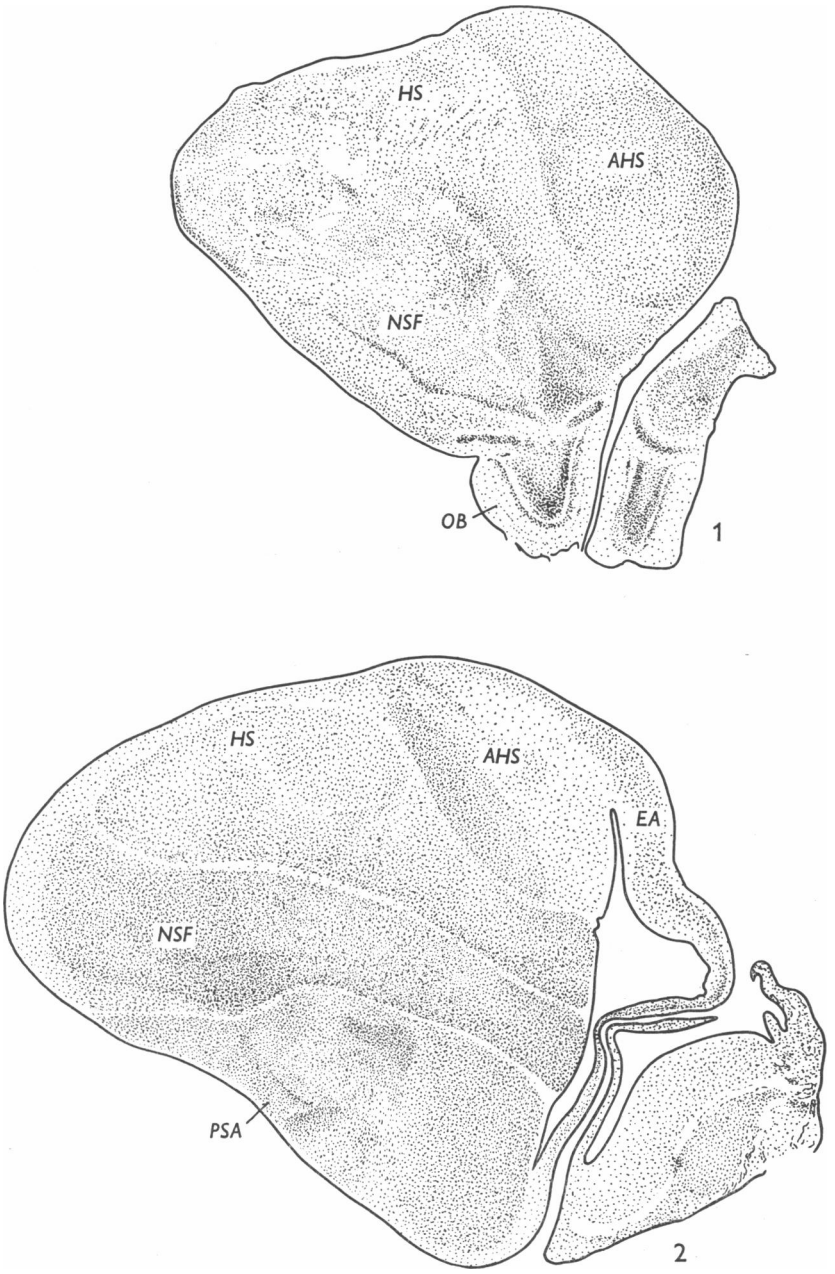
RESULTS

(I) *The total thalamic projection upon the telencephalon*

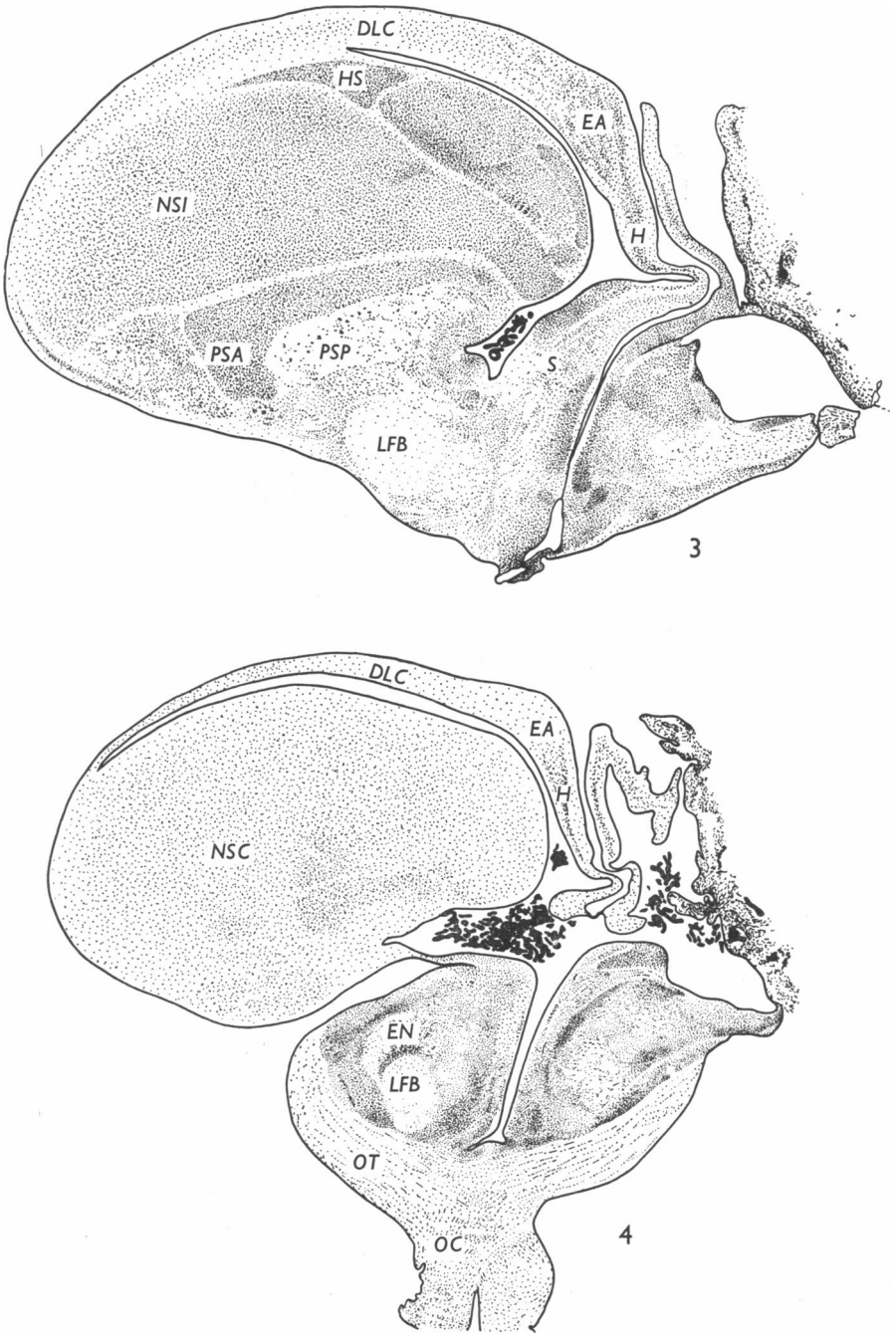
Before describing the results of the individual experiments some comments on retrograde cell degeneration in the avian thalamus may be apposite as this has not, to our knowledge, been previously described. Such cellular degeneration appears to be just as well defined and clear-cut as that found in the mammal, and in some of the affected nuclei has resulted in severe cell loss and gliosis. In other nuclei, however, such as the entopeduncular nucleus, the degeneration takes the form of a marked cell shrinkage and compacting of the cells rather than a cell loss and in this respect resembles the degeneration found in the thalamic reticular nucleus in the mammal (Rose, 1952).

Experiment OP 20 is typical of an unilateral telencephalic ablation; in this experiment almost the entire telencephalon of the right side was removed by suction, and the animal was allowed to survive for 44 days. Examination of the serial sections shows that anteriorly only a small fragment of the ventromedial corner of the hemisphere adjacent to the inferior angle of the ventricle remains, and posteriorly there is, in addition, a small remnant of cortex (Text-fig. 1; Pl. 1, fig. 1). The parts remaining include the posterior part of the olfactory bulb, together with the most medial part of the prepyriform cortex, anterior olfactory nucleus and the ventralmost part of the hyperstriatum. At a slightly more posterior level this ventromedial fragment consists of the degenerated nucleus of the diagonal band and the most medial portion of the palaeostriatum. Just caudal to the anterior commissure (Text-fig. 3) the only parts of the telencephalon which are preserved are the posterior portion of the septum, and the hippocampal and entorhinal areas of the cortex on the medial surface of the hemisphere. The septo-mesencephalic tract has been severely damaged, and it is markedly shrunken and gliosed. Ventral to the anterior commissure the medial preoptic area and the medial part of the lateral preoptic area are intact. More caudally (Text-figs. 4, 5), above the diencephalon, the amount of cortex remaining progressively increases until it forms a complete ring at the posterior pole of the hemisphere. With the possible exception of slight marginal involvement of the dorsolateral edge of the thalamus the diencephalon is undamaged (Text-fig. 6). In summary, this experiment can be considered as a virtually complete telencephalic ablation.

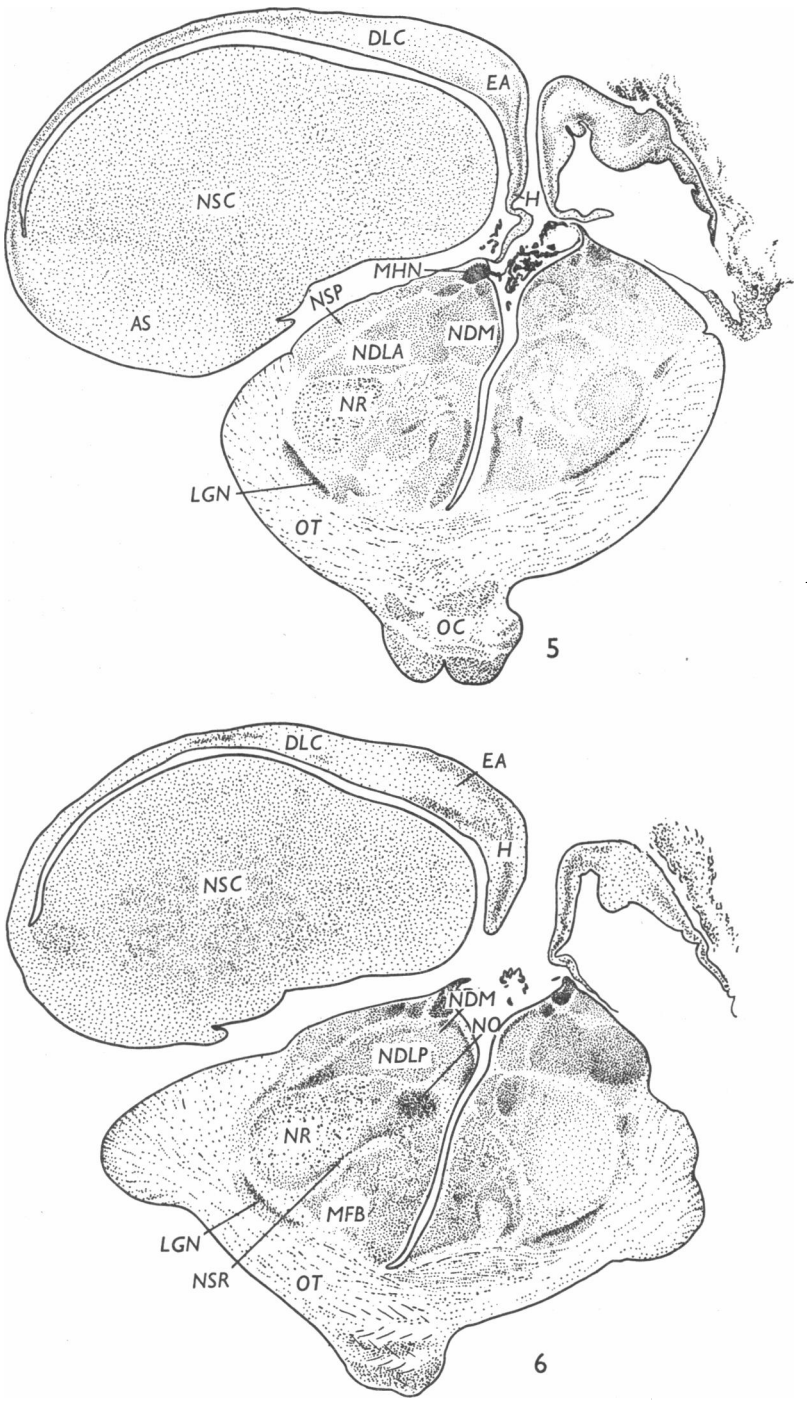
From an analysis of the thalamic nuclei in this experiment it is clear that they fall into three distinct groups on the basis of their cellular reaction. In the nuclei of the first group the cells show profound retrograde degeneration; marked cell loss has occurred, and no normal cells remain. This group comprises the two most conspicuous elements of the avian thalamus, namely, the nucleus rotundus and the nucleus ovoidalis together with the nucleus dorsolateralis anterior (Text-figs. 6, 7; Pl. 1, fig. 2). The degeneration is most severe in the nucleus rotundus; only a small number of neurons persist, and these all appear considerably paler than normal, and are swollen and ill-defined (Pl. 2, figs. 3, 4). The severe cell loss and gliosis together serve to differentiate sharply the degenerated nucleus from the neighbouring cell masses. In the adjoining nucleus ovoidalis a somewhat larger proportion of cells remain, but again all of these cells are abnormal, being enlarged in size



Text-figs. 1, 2. Drawings to show the principal subdivisions of the telencephalon on the left and the extent of the lesion at anterior levels in experiment OP 20 (right-hand side). These and subsequent drawings of this experiment have been traced from transverse sections at intervals of 0·75 mm. using a projection apparatus. On the operated side only the ventromedial corner of the hemisphere remains.



Text-figs. 3, 4. Drawings of the hemispheres of experiment OP 20 at the level of the middle of the septum and the anterior end of the thalamus, respectively.



Text-figs. 5, 6. Drawings at more caudal levels of the hemispheres of experiment OP 20 which show the extent of the lesion and the distribution of the relevant thalamic nuclei.

and paler staining; the accompanying gliosis is more marked in this nucleus (Pl. 3, figs. 6, 7).

Before describing the changes in the dorsolateral nucleus it is necessary to comment upon the morphology of this nuclear mass in the pigeon. The general form of the nucleus corresponds to the description given by Huber & Crosby (1929) for the dove. There is no sharp demarcation in coronal sections between what has been defined as the nucleus dorsolateralis anterior and the nucleus dorsolateralis posterior in other species. A gradient of cell size exists between the ventral and dorsal margins, however, on the basis of which it is possible to differentiate a larger-celled dorsal area and a ventral area containing smaller, darker-stained cells. In sections of the thalamus which are cut in the horizontal plane the differentiation is clearer, the small-celled area being almost circular in outline and situated anteromedially with the larger cells occupying a triangular area posterolaterally with its apex directed forwards. On the operated side of this experiment only the ventral area shows any degeneration; here marked cell loss has occurred, and the surviving cells are paler and shrunken; these cellular changes are accompanied by severe gliosis. This experiment therefore provides a valid criterion for subdividing the dorsolateral nucleus into distinct parts. We shall call the degenerated nucleus the nucleus dorsolateralis anterior and the nucleus which remains unaffected the nucleus dorsolateralis posterior, following the terminology of Huber & Crosby (1929). The preservation of the nucleus dorsolateralis posterior in this experiment might be interpreted as showing that this nucleus does not project upon the telencephalon. Evidence will be presented later to show that this is not so; cellular degeneration equally severe to that seen in the nucleus dorsolateralis anterior is found in this nucleus in other experiments.

In the second larger group of nuclei less marked, but unequivocal, changes are found. The changes vary from slight pallor of the constituent cells in some nuclei to pronounced cell shrinkage together with partial cell loss in other nuclei. The detailed changes will be described for the respective nuclei as these appear in an anteroposterior series. The cells of the two parts of the entopeduncular nucleus react differently but in neither does any cell loss occur; the large, deeply staining cells of the dorsal part appear much paler and are considerably reduced in size with less obvious processes. The smaller cells of the ventral part of the nucleus are also much paler on the operated side; they are unchanged in size, but they no longer have their characteristic spindle shape and appear distinctly globular. As a result of the degeneration of the fibres of the lateral forebrain bundle, the cells are closely compacted together and are surrounded by an intense gliosis. In the nucleus dorsomedialis anterior and the nucleus superficialis parvocellularis the cells are considerably paler than normal; it is also possible that the cells are slightly shrunken. A moderate degree of gliosis is present, but there is no suggestion of any cell loss in either of these nuclei. At low magnifications it would appear that the nucleus subrotundus has undergone complete cell loss, but at higher magnifications it is clear that this appearance is due to very marked cell shrinkage and pallor.

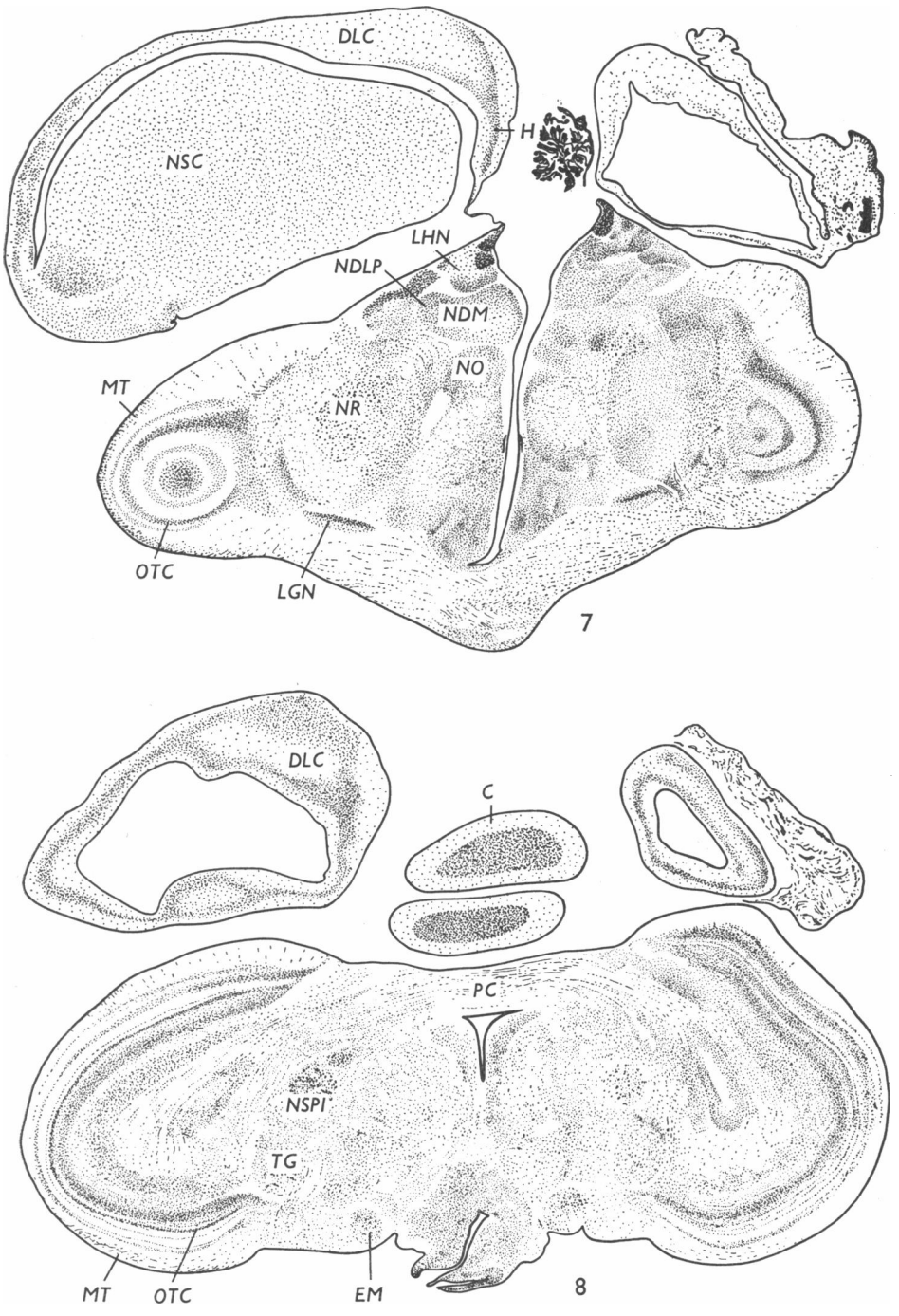
In the nuclear mass immediately caudal to the nucleus rotundus there are again two distinct cell types: in the large-celled lateral part of the nucleus (probably

corresponding to the nucleus postrotundus of Huber & Crosby, 1929, but cf. Kappers *et al.* 1936) there is a marked reduction in cell size but no appreciable change in the amount of Nissl material in the cytoplasm. In the medial smaller-celled area (? the nucleus posterointermedialis of Huber & Crosby, 1929) there is only a slight cell shrinkage but again no change in staining intensity. There is no evidence of cell loss in either part of the cell mass. In the ill-defined area usually designated the nucleus posteroventralis some cell loss may have occurred, but the most striking change is the marked shrinkage and pallor of the cells throughout the nucleus (Pl. 4, figs. 8, 9). Perhaps the most unexpected observation in this experiment is the finding of marked degenerative changes in the area labelled TG in Text-fig. 8 which is to be identified as the subpretectal nucleus of Huber & Crosby (1929). These changes are in the form of partial cell loss, and in addition there is shrinkage of the surviving cells (Pl. 4, figs. 8, 9).

The third group of nuclei, which show no degenerative changes at all, includes all the remaining thalamic nuclei as described by Huber & Crosby (1929). A complete list of these nuclei need not be given, but it should be emphasized that this group contains such well-defined elements as the so-called lateral geniculate nucleus (Text-figs. 5–7), both parts of the nucleus spiriformis (Text-fig. 8), the medial and lateral habenular nuclei (Text-figs. 5, 7) and the nucleus of the habenulo-peduncular tract.

Experiment OP 40 (47 days' survival) is the most complete telencephalic ablation in our series; it has not been described more fully as representative of the first group of experiments because (1) there is some involvement of the dorsomedial cortex of the opposite hemisphere, (2) the involvement of the dorsolateral margin of the thalamus is somewhat greater than in OP 20, and (3) the oblique plane in which the sections have been cut make it difficult to compare the thalamic nuclei on the two sides. The only region of the telencephalon remaining in this experiment is the posterior part of the septum and the medial preoptic area. The extent of the thalamic degeneration is almost identical with that found in OP 20, the most noteworthy difference being the almost complete degeneration of nucleus dorsolateralis posterior; in this nucleus, as in the nucleus dorsolateralis anterior, there is marked cell loss and shrinkage of the few surviving cells. Furthermore, in nearly all the affected nuclei the cellular changes are more marked than in OP 20. In particular the nucleus dorsomedialis anterior shows more definite pallor and shrinkage of its constituent cells; in the nuclei ovoidalis and rotundus only very few cells persist, and the cells in the subpredectal nucleus have all undergone marked shrinkage.

The third experiment in this group, OP 30 (42 days' survival) is very similar to OP 20 both in the site and extent of the lesion and the resulting thalamic degeneration. In view of this only the significant differences need be described. The telencephalon has been completely removed with the exception of the cortex on the medial aspect of the hemisphere, the septum and the ventromedial part of the palaeostriatum around the inferior angle of the ventricle. It is difficult, however, to decide whether the afferent connexions of these areas are completely preserved or not, because immediately in front of the preoptic areas both forebrain bundles are directly involved. The septo-mesencephalic tract shows some gliosis but is by no means completely degenerated. The main differences in the thalamic degeneration



Text-figs. 7, 8. To show the caudal extent of the lesion in experiment OP 20 and the cell masses in the posterior part of the thalamus.

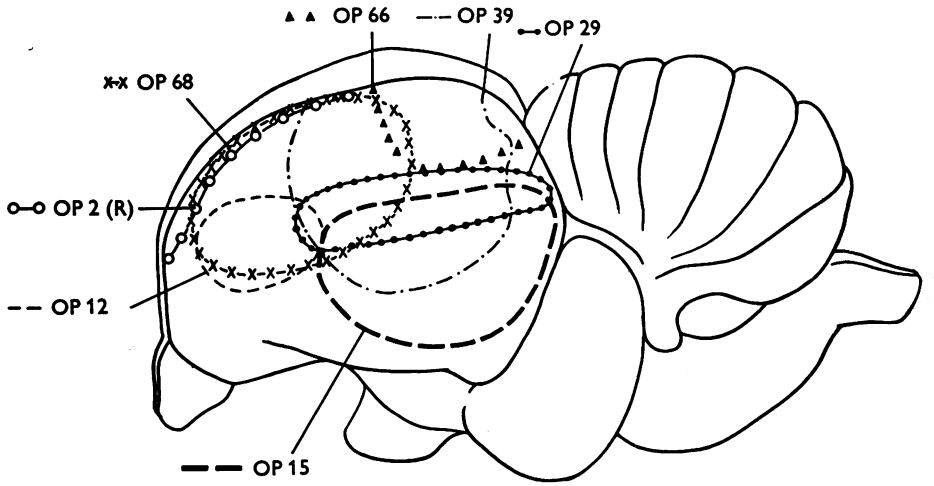
compared to experiment OP 20 are: in the nuclei ovoidalis, rotundus and dorsolateralis anterior the retrograde cell degeneration is even more severe and only an occasional pale-staining cell can be seen (Pl. 3, fig. 5). On the other hand, there is little change in the nucleus dorsomedialis and the degeneration in the subpretectal nucleus is less pronounced, the cells being distinctly paler than on the normal side, but they appear to be less shrunken than in OP 20.

Apart from minor differences in the degree of degeneration in the nuclei caudal to the nucleus rotundus and in the dorsomedial nucleus, the only significant difference in these three experiments is the finding of retrograde cell degeneration in the nucleus dorsolateralis posterior of OP 40. Unfortunately in that experiment the lesion had encroached upon the dorsolateral aspect of the diencephalon so that it is uncertain whether the degeneration may have been the result of this direct involvement. From the fourth experiment of this group, OP 2, however, this possibility can be excluded because here the nucleus dorsolateralis posterior has undergone degeneration after a lesion strictly limited to the telencephalon. With the exception of a very small portion of the ventromedial margin of the hemisphere the entire telencephalon has been destroyed. In the anterior one-third of the hemisphere only the prepyriform cortex and the immediately adjoining parts of the neostriatum and hyperstriatum around the inferior angle of the ventricle remain. In the succeeding sections the amount of surviving tissue can be seen to remain more or less constant, but at more posterior levels it is composed of the medial part of the palaeostriatum augmentatum on the lateral side of the ventricle and the ventral half of the cortex medial to the ventricle. This cortex is profoundly atrophied, particularly the molecular layer. It is probable that most of the striatum which remains is isolated by an extension of the lesion into the lateral forebrain bundle which severely disrupts its fibres. The anterior two-thirds of the septum are severely shrunken and partially involved, but the posterior one-third is largely preserved. The dorsolateral margin of the lateral preoptic area has been encroached upon, and the forebrain bundles directly involved. The septo-mesencephalic tract shows intense gliosis. Both the distribution and the severity of the retrograde degeneration in the thalamus are essentially the same as in the previous experiments, with the notable exception that both the anterior and posterior components of the nucleus dorsolateralis are severely affected. From this experiment it may be concluded that all the nuclei of the dorsal group are telencephalic dependencies. Although this experiment illustrates best the total thalamic projection, it has not been described as the primary example of a complete telencephalic ablation because there is some involvement of the medial aspect of the opposite hemisphere; this part of the lesion, however, has not resulted in any thalamic degeneration.

(II) *The organization of the thalamic projection*

Two observations described in the first section suggest that the individual elements of the avian thalamus, like the nuclei of the mammalian dorsal thalamus, have an organized projection upon the telencephalon. In the first place, it has been found that certain components of the dorsal nuclear group degenerated in some but not all experiments, and secondly, that the nature and degree of cellular degeneration differ in the various nuclear groups. In view of the remarkable structural dif-

ferentiation of both the telencephalon and the thalamus in the avian brain an attempt has been made to determine the projection of the individual thalamic nuclei. In the experiments presented in this section lesions varying in extent and distribution have resulted in differential involvement of those thalamic nuclei which have been shown to project upon the telencephalon. An analysis of this material has indicated that the efferent fibres from the thalamus terminate principally in two parts of the telencephalon: the palaeostriatum and the dorsomedial margin of the hemisphere. Further, the corollary of these findings, that a considerable portion of the hemisphere—including the dorsolateral cortex, the neostriatum and hyperstriatum—does not receive a projection from the thalamus, in the sense at least that their destruction does not result in retrograde cell degeneration in the thalamus, has also been demonstrated.



Text-fig. 9. Diagrammatic reconstruction of the superficial extent of the lesion in several experiments of the first group to show that together they cover the greater part of the dorsolateral aspect of the hemisphere.

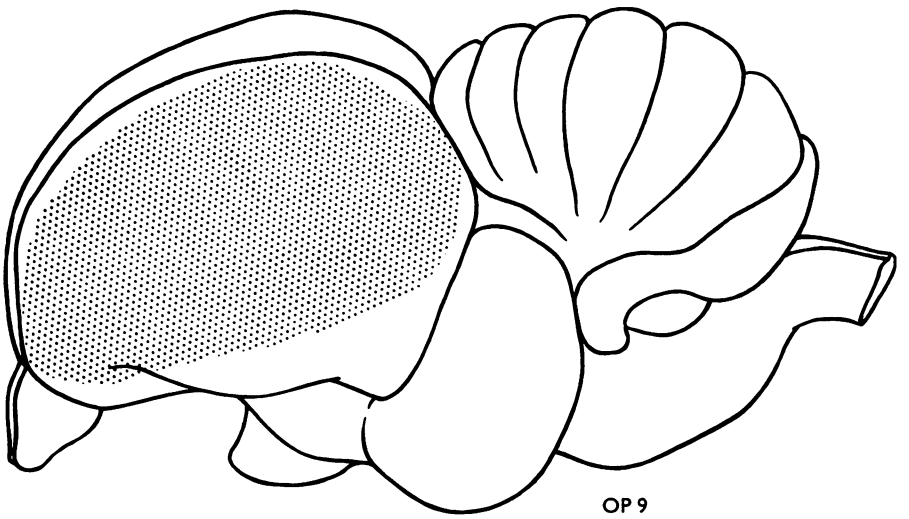
The experiments fall naturally into two classes: those in which the lesion has not resulted in retrograde cell degeneration, and those in which thalamic degeneration has occurred. Only representative examples of each class will be described in detail.

Experiments with no thalamic degeneration

There are thirteen hemispheres with superficial lesions of varying extent and in which no thalamic degeneration has been found. All the lesions are on the dorsolateral surface of the hemisphere and involve the dorsolateral cortex together with the immediately subjacent parts of the striatum. The distribution and extent of most of the lesions are shown in Text-fig. 9, from which it can be seen that although no single lesion has involved the entire dorsolateral cortex the lesions taken together cover almost the whole area. The largest lesion may be taken as representative of this group and will be described in some detail.

In experiment OP 9 (survival period 62 days) an extensive superficial lesion has

destroyed most of the dorsolateral surface of the left hemisphere throughout its antero-posterior extent (Text-fig. 10). The lesion begins at the frontal pole and gradually increases in extent and depth until it is maximal at the level of the appearance of the palaeostriatum. Thereafter the extent of the damage diminishes —particularly in depth—back to the level of the posterior end of the septum, after which it remains constant. The entire dorsolateral cortex of this hemisphere is completely destroyed, and the prepyriform and parentorhinal areas are encroached upon slightly. The lateral margin of the accessory hyperstriatum has been involved, together with the dorsal part of the hyperstriatum and neostriatum frontale and intermediale. The neostriatum caudale has suffered slight damage along its ventricular margin, and at these levels the dorsolateral part of the archistriatum has also been encroached upon (Text-fig. 11).



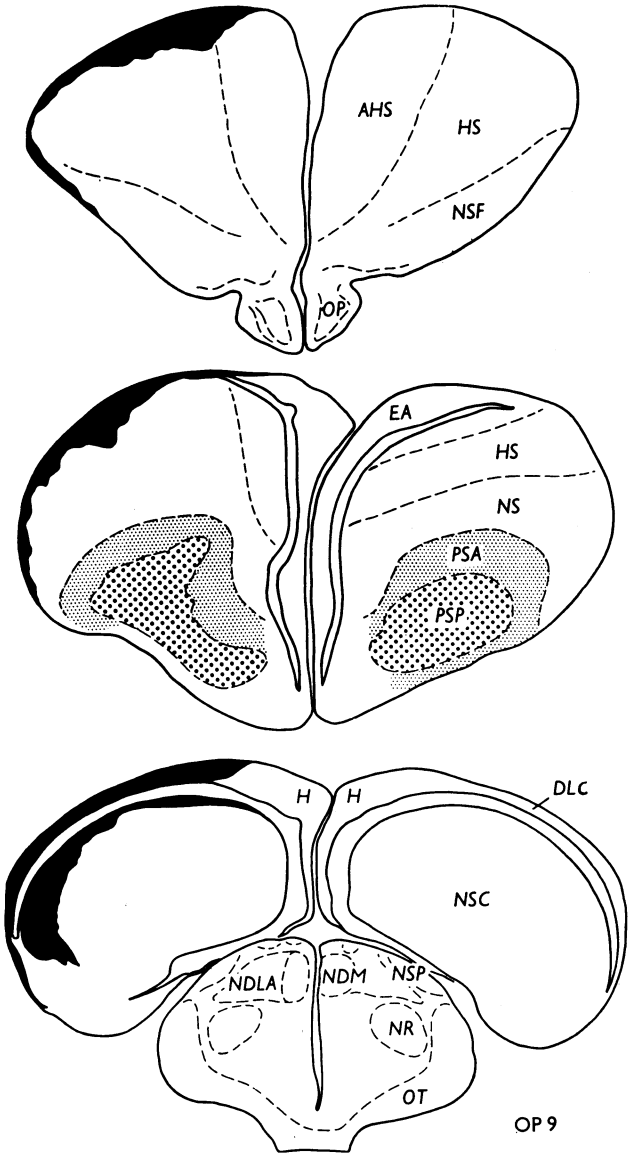
Text-fig. 10. Diagram to show the extent of the lesion (stippled) as projected upon the surface of the brain in experiment OP 9.

Experiments with thalamic degeneration

From an analysis of the remaining experiments with smaller localized lesions it soon becomes apparent that they can be divided intō two groups on the basis of the distribution of the thalamic degeneration. In the first group only the dorsal nuclei and the nucleus superficialis parvocellularis degenerate together or individually, while in the second the degeneration also involves the nuclei rotundus, subrotundus and ovoidalis. Examples of each of these two groups will first be described before presenting the experimental evidence for the localization of the projection of individual nuclei.

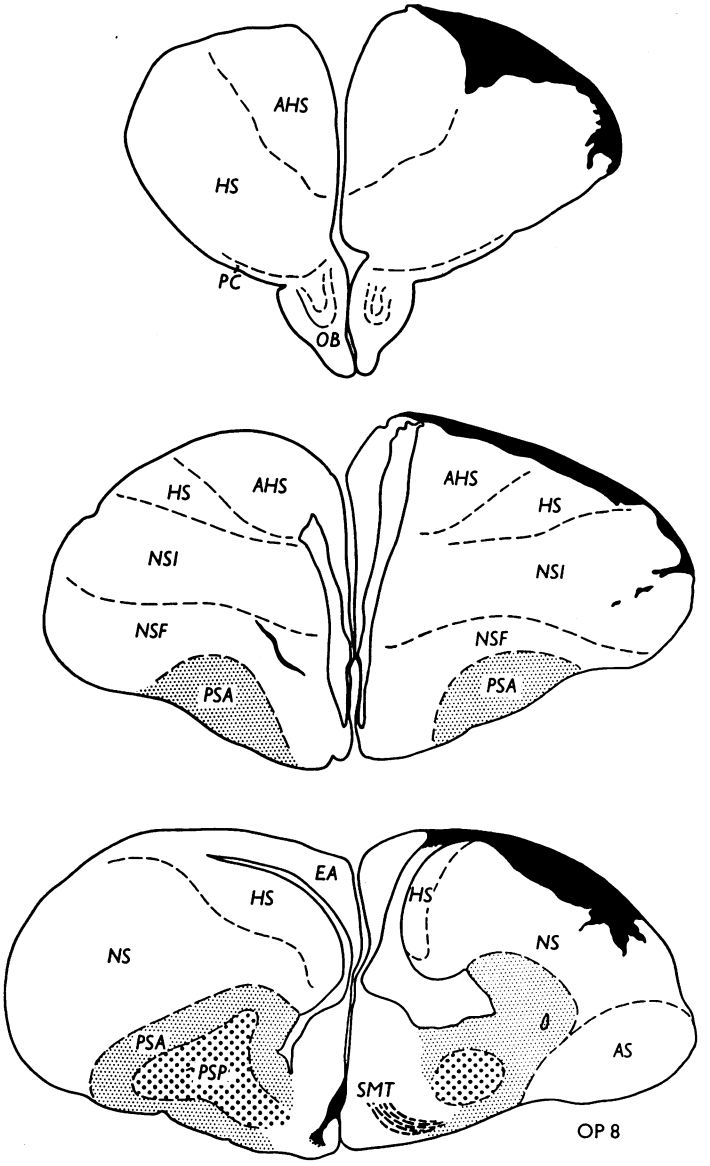
In the first experiment of the first group, OP 8, a large lesion which has involved the dorsal striatal areas of the right hemisphere has resulted in degeneration only in the nucleus dorsolateralis anterior and nucleus superficialis parvocellularis. The lesion begins at the frontal pole where it involves the lateral margin of the accessory

hyperstriatum and the dorsal part of the hyperstriatum. More caudally it comes to involve the dorsal surface of the neostriatum with no additional involvement of the accessory hyperstriatum. Virtually the whole dorsolateral cortex and the dorsal part of the neostriatum caudale have been destroyed almost to the caudal pole of the hemisphere. Medially the lesion does not extend beyond the lateral ventricle and at no point does it encroach upon the palaeostriatum or the archistriatum (Text-fig. 12).



Text-fig. 11. The extent of the lesion in the left hemisphere in experiment OP 9 at three antero-posterior levels through the hemispheres. The damaged areas in this and the subsequent figures are indicated in black.

In the thalamus definite cellular degeneration is seen in the nucleus dorsolateralis anterior in the form of a moderate degree of cell loss and gliosis together with pallor and shrinkage of all the remaining cells. In the nucleus superficialis parvocellularis



Text-fig. 12. The extent of the damage to the two hemispheres in OP 8. The small lesion in the left hemisphere has completely interrupted the fibres of the septo-mesencephalic tract.

there is some cell shrinkage but the principal change is the pallor of the cells. It should be emphasized that no other thalamic nucleus shows evidence of retrograde degeneration and in particular that the entopeduncular nucleus is unaffected.

In experiment OP 7 the lesion is considerably smaller in its anteroposterior extent but involves rather more of the accessory hyperstriatum. It commences at the frontal pole of the hemisphere, destroying the whole of the accessory hyperstriatum and the dorsal part of the hyperstriatum. The extent of the damage remains more or less constant through the anterior third of the hemisphere except for a narrow knife-cut into the lateral part of the neostriatum intermediale. The entorhinal area and dorsolateral cortex are not involved. The lesion diminishes in size relatively suddenly and ends just in front of the appearance of the septum.

The changes in the nuclei dorsolateralis anterior and superficialis parvocellularis are essentially the same as those described for experiment OP 8, but in addition there are unequivocal changes in the nucleus dorsomedialis anterior. Here there is a distinct pallor of all the cells and many are unmistakably shrunken. Again there is no evidence of degeneration in any of the other thalamic nuclei, including the nucleus entopeduncularis.

In the remaining experiments of this group with comparable lesions no degeneration has been found in nuclei other than those of the dorsal group. On the other hand, in a larger group of experiments in all of which there has been some involvement of the palaeostriatum, changes have been consistently found in the nucleus rotundus and/or the nucleus ovoidalis, together with varying degrees of degeneration in the other nuclei which show changes after the large telencephalic lesions described in the previous section. That the critical factor leading to degeneration in these nuclei is in fact involvement of the palaeostriatum is shown in the following two experiments which are representative of this second group.

In experiment OP 6 the lesion has involved both hemispheres but to a varying extent and with significantly different degeneration in the thalamus of the two sides. On the right side most of the damage is in the anterior one-third or half of the hemisphere beginning close to the anterior pole where there is a superficial area of damage in the accessory hyperstriatum. The lesion rapidly increases in size to destroy completely the accessory hyperstriatum, the hyperstriatum and neostriatum frontale. At the level at which the palaeostriatum is first seen the lateral part of the neostriatum and hyperstriatum re-appear so that the lesion is now restricted to the medial part of the striatum. Behind this level the lesion becomes progressively smaller in size and extends back as a central core of damage close to the lateral ventricle damaging the medial parts of the hyperstriatum and neostriatum. The damage ceases completely just in front of the level of appearance of the septum. On this side the only damage to the palaeostriatum is some slight involvement of its dorsolateral margin at anterior levels.

On the left side the damage also begins at the frontal pole as a superficial area of destruction in the dorsolateral part of the hemisphere involving only the lateral portion of the accessory hyperstriatum and the dorsal hyperstriatum. It remains more or less constant in size, and although it comes to involve the lateral part of the neostriatum the damage to the accessory hyperstriatum remains minimal. In the anterior third of its extent the palaeostriatum is not involved, but in the sudden increase in the size of the lesion, which occurs just rostral to the septum, its dorsolateral margin together with the lateral two-thirds of the neostriatum and the hyperstriatum are destroyed. At the level of appearance of the septum the entire

hyperstriatum, neostriatum and palaeostriatum augmentatum are destroyed, but the accessory hyperstriatum and entorhinal areas are intact. At about the middle of the septum the lesion diminishes in size and becomes progressively restricted to the dorsomedial parts of the hyperstriatum and the neostriatum, the adjoining dorsal part of the palaeostriatum and to a narrow extension of the lesion which reaches ventrally through the middle of the palaeostriatum just rostral to the preoptic areas. Behind this the lesion extends back almost to the caudal pole of the hemisphere in the medial part of the neostriatum caudale: the overlying dorsolateral cortex is undamaged. The archistriatum has not been directly involved by the lesion but has undergone ischaemic necrosis with virtually complete cell loss.

On the right side (i.e. the side with the smaller lesion) there is pronounced gliosis throughout the cross-sectional area of the septo-mesencephalic tract. Beginning anteriorly in the considerably shrunken cortex on the medial side of the ventricle, this gliosis can be seen on succeeding sections to pass caudally and ventrally, to the dorsomedial border of the septum. From here it can be followed as it passes downwards through the medial part of the septum and then ventrally in a course reminiscent of that of the diagonal band of the mammalian brain. It reaches the ventral surface of the hemisphere medial to the forebrain bundles and then sweeps laterally beneath these fibre bundles to the lateral surface of the diencephalon. Hence it passes dorsally around the lateral aspect of the thalamus in front of the optic tract to reach, finally, the region of the nucleus superficialis parvocellularis. On the other side, despite the larger lesion, there is only slight gliosis in the septo-mesencephalic tract, and there is a striking difference in the thickness of the medial wall of the hemisphere on the two sides.

In the thalamus of the right side unequivocal retrograde degeneration has occurred in the nuclei dorsolateralis anterior and superficialis parvocellularis and in the anterodorsal part of the nucleus rotundus. The most anterior part of the nucleus dorsolateralis anterior and, at more posterior levels, the dorsomedial part of the nucleus, are intact but elsewhere the nucleus shows marked cell loss and gliosis. The cells of the nucleus superficialis parvocellularis are distinctly shrunken and pale-staining. In the affected part of the nucleus rotundus there is marked cell loss and pallor of the surviving cells. In addition to these changes there is a suggestion of cell shrinkage in the dorsomedial nucleus at anterior levels and there is some shrinkage of the cells in the lateral part of the entopeduncular nucleus. On the left side, the thalamus shows severe retrograde change in the nuclei ovoidalis, rotundus and subrotundus. In each of these nuclei there is an almost total cell loss with marked shrinkage and pallor of the few remaining cells. Other nuclei which are affected are the entopeduncular, the dorsolateral anterior and posterior, the superficialis parvocellularis, the postrotundus and the posteroventral. The changes in the dorsal nuclei are comparable qualitatively to those in the opposite thalamus, but are restricted to the lateral parts of the nuclei; the changes in the nuclei postrotundus and posteroventral are difficult to assess in view of the obliquity of the sections, but in both nuclei there appears to be a moderate degree of cell shrinkage. The cells of both elements of the entopeduncular nucleus are shrunken and pale-staining, especially in the dorsal part of the nucleus.

The interest of this experiment lies in the fact that the lesion on the two sides and

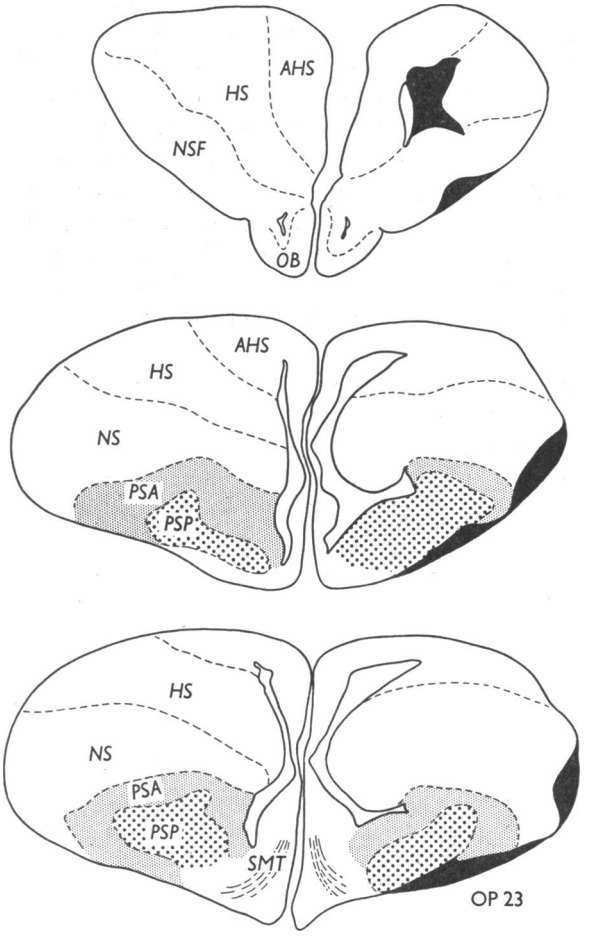
the resulting thalamic degeneration are almost complementary to each other. On the right side the lesion essentially destroys the medial part of the anterior third of the hemisphere with only minimal involvement of the palaeostriatum, while on the left side it is principally the dorsolateral and central portions of the hemisphere which are involved, with considerable damage to the palaeostriatum. The thalamic degeneration on the two sides is correspondingly different: on the right side with the smaller lesion, only the nuclei of the dorsal group and a small part of the nucleus rotundus have been affected, while on the opposite side the nuclei ovoidalis, rotundus and subrotundus are predominantly affected and slight changes are present in the dorsal group. A correlation of the findings in this experiment with those of OP 9, the representative of the second group in which no thalamic degeneration occurs after a large, but relatively superficial lesion, suggests that involvement of the palaeostriatum is the critical factor determining whether or not degeneration occurs in the thalamic nuclei (other than those of the dorsal group) which degenerate after complete removal of the telencephalon. That the palaeostriatum is the critical factor is further exemplified by experiment OP 23 in which, after a relatively small lesion involving the lateral part of the palaeostriatum, marked retrograde degeneration has occurred in the nucleus rotundus.

The lesion in this experiment (Text-fig. 13) begins close to the frontal pole but for the greater part of its extent is confined to the ventrolateral quadrant of the hemisphere. Anteriorly, the medial third of the hyperstriatum has been destroyed, together with the ventrolateral margin of the neostriatum frontale. The lesion remains more or less constant back to the level of appearance of the palaeostriatum behind which only the lateral margin of this structure and the lateral part of the neostriatum intermediale are affected. The palaeostriatum has been directly involved ventrolaterally; the ventral third of the palaeostriatum augmentatum has been completely removed, and there is a narrow extension of the lesion which passes vertically through the primitivum. Although not directly affected, a large part of the medial end of the palaeostriatum augmentatum shows a distinct loss of cells, probably due to vascular involvement. The damage to the palaeostriatum is maximal at the level of the anterior end of the septum, but it diminishes rapidly in extent to cease well in front of the posterior end of the septum. At this level the damage to the neostriatum is also considerably reduced, involving only its lateral margin. Distinct gliosis, continuous with the lesion in the palaeostriatum, can be traced back in the lateral forebrain bundle, but it should be emphasized that the dorsomedial part of the hemisphere, including the accessory hyperstriatum, has not been involved at all, and that there is no gliosis in the septo-mesencephalic tract.

The degeneration in the thalamus is surprisingly extensive in view of the restricted nature of the lesion. The anterior two-thirds of the nucleus rotundus is completely devoid of cells, but at the junction of its middle and posterior thirds a few normal cells appear and progressively increase in number until the posterior limit of the nucleus appears almost normal. In the posteroventral and postrotundus nuclei and in the subpretectal nucleus the cells show marked shrinkage comparable to that seen after the complete telencephalic ablations. Likewise, the dorsal part of the entopeduncular nucleus is severely affected, especially anteriorly; here there is a very severe cell shrinkage and gliosis while in the ventral part of the nucleus there is

some degree of pallor of the cells, no distinct cell shrinkage but an intense gliosis. The nuclei of the dorsal group and the nuclei ovoidalis and subrotundus, on the other hand, are completely unaffected (Pl. 5, fig. 10).

These two experiments, taken together, indicate that not only do the telencephalic-dependent nuclei have a differential projection upon the cerebral hemisphere, but also that within the projection of individual nuclei there is at least some degree of



Text-fig. 13. To show the extent of the lesion in the right hemisphere of experiment OP 23 at three anteroposterior levels.

topical organization. Thus from experiment OP 23 it is apparent, for example, that the nucleus rotundus may degenerate independently of the nucleus ovoidalis, and furthermore that within the projection of the nucleus rotundus there is a definite anteroposterior organization in so far as the degeneration in the nucleus is confined to its anterior two-thirds. Similarly, on the side of the smaller lesion in OP 6 the finding of degeneration confined to the dorsomedial part of the nucleus rotundus

suggests a comparable mediolateral organization of the efferent connexions of this nucleus. An additional conclusion which may be drawn from these two experiments is that there is some correlation between the site and the extent of the lesion in the palaeostriatum and the distribution of the resulting thalamic degeneration.

The purpose of presenting the results of several of the remaining experiments of this group in some detail is to examine the validity of these hypotheses. It will be convenient to present the evidence for the localization of the projection of the nucleus ovoidalis and the rotundus before dealing with the nuclei of the dorsal group. The lesions of the remaining experiments will not be described in full, but for each of these two nuclear groups only the relevant damage to the palaeostriatum and the dorsomedial aspect of the hemisphere, respectively, need be described.

In experiment OP 26 there is extensive damage to the hyperstriatum, neostriatum and dorsolateral cortex and at the level of appearance of the palaeostriatum primitivum the lateral third of the palaeostriatum augmentatum has been destroyed. From this level the lesion extends caudally, destroying approximately the same amount of the palaeostriatum augmentatum and the lateral margin of the palaeostriatum primitivum back to the level of the anterior commissure. Here the most ventral part of the lesion encroaches upon the lateral aspect of the lateral forebrain bundle.

In the thalamus there is a complete cell loss in the nucleus rotundus throughout its anteroposterior extent. In the nuclei ovoidalis and subrotundus there is definitely no cell loss, but there is a suggestion of cell shrinkage and pallor. There are also changes in the nuclei entopeduncularis, posteroventralis, postrotundus and the subpretectal nucleus in the form of cell shrinkage; the nuclei of the dorsal group are unaffected.

This experiment differs from the previous experiment in two respects. First, the lesion involves considerably more of the lateral palaeostriatum and extends throughout its anteroposterior extent; secondly, the degeneration in the nucleus rotundus is correspondingly more extensive. However, despite these differences they have the important common feature that the nucleus rotundus has degenerated independently of the nucleus ovoidalis. The next experiment to be described is virtually complementary to these two experiments. Here the nucleus ovoidalis has degenerated while the nucleus rotundus is unaffected. As the precise projection of the entopeduncular nucleus and of the nuclei posterior to the rotundus has not been determined, and as the organization of the projection of the dorsal nuclei will be treated separately, the cellular changes in these nuclei will not be described in the subsequent experiments dealing with the projection of the nuclei ovoidalis and rotundus.

In the brain of pigeon OP 5, lesions have been placed in both hemispheres, but the smaller lesion in the right side has not resulted in any thalamic degeneration and will not be described here. The lesion on the left side extends throughout the anteroposterior extent of the hemisphere, damaging the anterior part of the accessory hyperstriatum, most of the dorsal hyperstriatum, large areas of all parts of the neostriatum and the dorsolateral cortex. The involvement of the palaeostriatum begins at the level of the appearance of the palaeostriatum primitivum where the dorsal and medial parts of the palaeostriatum augmentatum are destroyed. Back to the level of the anterior commissure the only additional damage to the palaeo-

striatum is to the dorsal and medial portion of the palaeostriatum primitivum, the lateral part of the palaeostriatum augmentatum remaining intact. It is difficult to be certain, but it is probable, that in this experiment almost the entire ektostriatum has either been severely damaged or completely removed; certainly the only portion which might be spared is the medial part immediately adjoining the palaeostriatum.

In the thalamus there is very marked cellular degeneration in the nucleus ovoidalis while the nucleus rotundus is largely preserved. The changes in the nucleus ovoidalis are in the form of severe cell loss with shrinkage and pallor of all the surviving cells. In the nucleus rotundus, on the other hand, the degenerative changes are confined to the posterior third of its anteroposterior extent where the majority of the cells are shrunken, but there does not appear to be any cell loss. The nucleus subrotundus similarly shows cell shrinkage rather than cell loss. It is of particulr interest that in this experiment the entopeduncular nucleus shows shrinkage and pallor of the cells only in the medial portion of the nucleus which is surrounded by the intense gliosis in the lateral forebrain bundle.

The significance of this experiment is twofold; in the first place it shows that the nucleus ovoidalis can undergo retrograde degeneration independently of the rotundus and thus has a separate projection field; and secondly, it confirms the findings of the previous experiments that the nucleus rotundus is related to the lateral part of the palaeostriatum. In addition it probably excludes the ektostriatum as the main site of termination of the axons from rotundus since it shows only slight changes after virtually complete destruction of the ektostriatum (cf. Huber & Crosby, 1929).

Experiment OP 70 provides further confirmation on these two points. In this case the lesion is essentially in two parts: a large superficial lesion destroying the dorsal and lateral aspects of the hemisphere and a narrow knife-track on the medial side of the hemisphere which extends down parallel to the ventricle into the palaeostriatum. A wedge-shaped medial extension of the superficial lesion has also encroached upon the lateral aspect of the palaeostriatum and has resulted in necrosis of the central third of its anteroposterior extent. The other palaeostriatal lesion has destroyed the dorsal aspect of the palaeostriatum augmentatum immediately beneath the damaged neostriatum intermediale. This latter involvement of the palaeostriatum is confined to the posterior third of its extent.

The thalamic degeneration in this case resembles that found in the previous experiment. The nucleus ovoidalis is severely degenerate throughout its extent, but the nucleus rotundus shows cellular degeneration only in its dorsal part. The nucleus subrotundus, however, is not nearly so severely affected and in fact only shows shrinkage and pallor of the cells in its medial part.

In experiment OP 62 the lesion is confined to the posterior half of the hemisphere. In addition to damaging the dorsal and lateral parts of the hyperstriatum and neostriatum it has extended into the lateral ventricle to destroy the posteromedial part of the neostriatum caudale and the adjacent dorsomedial part of the palaeostriatum—principally the medial third of the palaeostriatum augmentatum. In the thalamus of this experiment the most striking cellular degeneration is seen in the nucleus ovoidalis; here there is a very severe cell loss with pronounced shrinkage

and pallor of the few remaining cells and an intense gliosis. The nuclei rotundus and subrotundus are completely unaffected.

Taken together, these experiments with degeneration only in the nucleus ovoidalis, or largely confined to that nucleus, clearly indicate that its projection field is the medial part of palaeostriatum. Furthermore, since degeneration in the nucleus is always most severe with lesions in the posterior part of the striatum it would appear that either the majority of the projection fibres terminate in the posterior part of the palaeostriatum or at least pass through it to reach more anterior levels. That this conclusion is substantially correct is shown by the next experiment, OP 13, in which a lesion in the anteromedial part of the palaeostriatum has resulted in degeneration in the nucleus ovoidalis which is qualitatively different from that seen in the previous experiments.

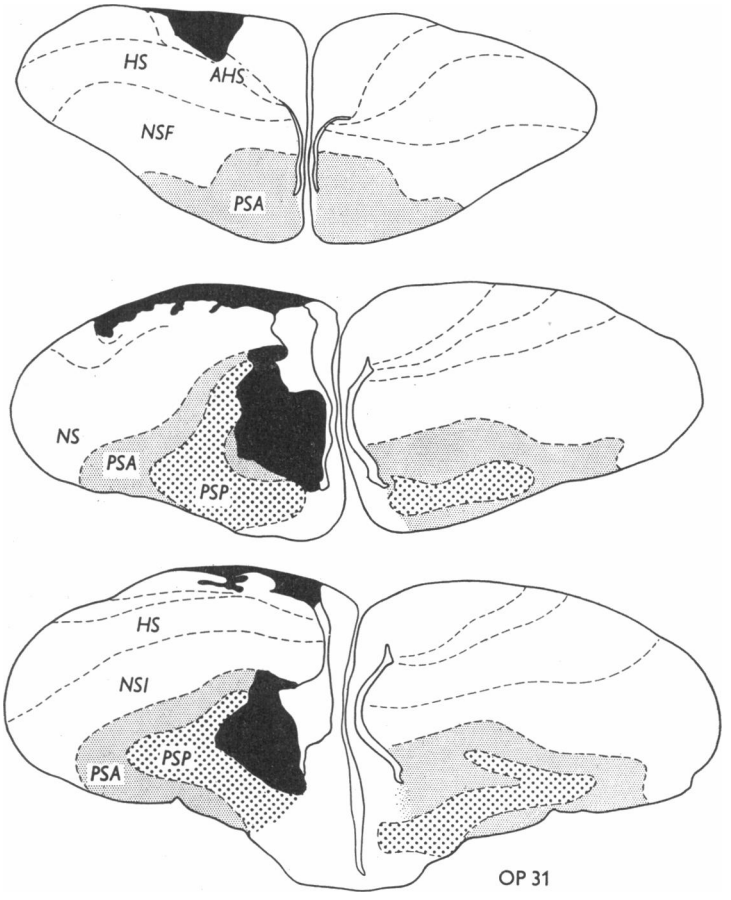
The damage to the palaeostriatum in OP 13 is limited to the anterior two-thirds of the medial part of the palaeostriatum augmentatum with two narrow extensions into the medial part of the palaeostriatum primitivum. In the thalamus there is again no change in the nuclei rotundus and subrotundus, but there is marked cell shrinkage in the ovoidalis. Under a low-power objective the degeneration appears to be about as severe as in previous experiments, but at higher magnifications it is apparent that there is no cell loss in the nucleus but that the majority of the cells are distinctly shrunken and pale staining.

The projection of the nucleus rotundus to the palaeostriatum augmentatum rather than to the palaeostriatum primitivum is shown by experiment OP 23: that this is true also for the nucleus ovoidalis is demonstrated by experiment OP 31. In this case the nucleus ovoidalis has undergone severe retrograde degeneration after a lesion which at no point encroaches upon the palaeostriatum primitivum. The definitive part of the lesion has directly damaged the dorsal part of the palaeostriatum augmentatum over a very short distance but has also resulted in a fairly extensive area of ischaemic necrosis in the posterior half of the medial sector of the palaeostriatum augmentatum (Text-fig. 14). As far as the thalamus is concerned it need only be noted that here again the nucleus ovoidalis is severely affected without concomitant changes in the nuclei rotundus and subrotundus (Pl. 5, fig. 11).

The accumulated evidence of the above experiments seems to establish beyond doubt that the nuclei ovoidalis and rotundus project independently, that they are connected with the palaeostriatum augmentatum and that the nucleus rotundus is related to its lateral part while the ovoidalis is connected with its medial part. However, these experiments have left undecided the precise projection of the nucleus subrotundus, although they have shown that like the other nuclei it has an independent projection. For example, it has been shown that the nucleus may degenerate with one or both of the other two central nuclei, and that it is not infrequently preserved when the others are degenerated. One final experiment, OP 21, clearly establishes the independence of the projection of the nucleus subrotundus for in this brain this nucleus shows virtually no change after a lesion which has resulted in severe degeneration of the rotundus and ovoidalis.

The damage to the palaeostriatum in this experiment extends throughout its posterior two-thirds, destroying most of the palaeostriatum augmentatum and the dorsal and lateral parts of the primitivum. The most anterior part of the palaeo-

striatum augmentatum has escaped injury, but from the level of appearance of the palaeostriatum primitivum back to the caudal limit of the palaeostriatum only the medial margin of the palaeostriatum augmentatum is unaffected. The nuclei rotundus and ovoidalis are both completely degenerated, but the nucleus subrotundus stands out in striking contrast to these elements since, at the most, it shows only slight cell shrinkage and there is certainly no cell loss in the nucleus (Pl. 6, fig. 12). When considered together with the previous experiments the conclusion to



Text-fig. 14. Representative sections through the lesion in experiment OP 31. The black area in the medial part of the palaeostriatum represents the site of the critical damage which has resulted from ischaemic necrosis.

be drawn from this experiment is that the nucleus subrotundus either projects to more anterior parts of the palaeostriatum augmentatum or more ventrally to the palaeostriatum primitivum.

It has been mentioned above that the precise projection of the nuclei posterior to the nucleus rotundus, viz. the postrotundus, the posteroventral and the subpretectal nucleus, cannot be determined from the present material. Indeed the material

available is insufficient to establish whether or not these nuclei have a projection independent of each other or of the three central nuclei; that they are independent of the nuclei of the dorsal group, however, has been demonstrated by experiments OP 7 and OP 8 described above. The fact that they so frequently show changes which parallel the degeneration in the nuclei rotundus and ovoidalis would suggest that they either project directly to the palaeostriatum augmentatum or that they are in some way connected with the projection of the central nuclei. Similarly, the constant finding of degeneration in the entopeduncular nucleus following lesions of the palaeostriatum strongly suggests that this nucleus is closely related to the projection of the central group of nuclei. The findings in OP 5, in which degeneration has been found only in the medial half of the nucleus, would also suggest that there is a mediolateral organization of its projection fibres comparable to that found in the nuclei ovoidalis and rotundus.

A similar series of experiments will now be described in an attempt to define the projection area of the individual elements of the dorsal nuclear group. The first experiment to be considered, OP 44, serves to delimit, in a negative way, the projection field of these nuclei as it has the largest lesion of the series in which no degeneration has occurred in these nuclei. The lesion begins immediately behind the frontal pole of the hemisphere where there is a small area of damage in the most dorsal part of the hyperstriatum. Behind this the hyperstriatum is increasingly involved and the accessory hyperstriatum is encroached upon. At the level of appearance of the palaeostriatum the hyperstriatum is completely destroyed together with the dorsal half of the neostriatum frontale. In addition there is a cut extending down through the lateral part of the accessory hyperstriatum into the lateral ventricle. With the appearance of the palaeostriatum primitivum the lesion increases in size to include the whole of the hyperstriatum, neostriatum and the lateral part of the palaeostriatum augmentatum. The cortex on the medial wall of the hemisphere is preserved. From the anterior end of the septum back to the level of the anterior commissure the only structures not completely destroyed are the ventral two-thirds of the palaeostriatum primitivum, the inferolateral part of the neostriatum and the cortex medial to the lateral ventricle. More caudally the lesion destroys the middle portion of the hemisphere as far as the caudal pole, only the most ventral part of the archistriatum, the lateral part of the neostriatum caudale and the medial cortical area being preserved.

Despite the fact that this extensive lesion has resulted in severe retrograde degeneration in the nuclei ovoidalis, rotundus, subrotundus and the nuclei of the posterior group no appreciable change has occurred in any of the dorsal nuclei. In this respect this experiment is virtually complementary to experiments OP 7 and OP 8 in which the dorsal nuclei degenerated independently of the others. Together these three experiments limit the area of projection of the dorsal nuclei to the dorsomedial margin of the hemisphere, to either the cortex in this area or to the accessory hyperstriatum. A second interesting feature in OP 44 is the absence of gliosis in the septo-mesencephalic tract. This stands in marked contrast to the severe glial proliferation which has occurred in the lateral forebrain bundle. The significance of this absence of gliosis in the septo-mesencephalic tract is apparent when one compares this experiment with, say, the findings in experiment OP 6 in which this tract was

severely atrophied and the dorsal nuclei completely degenerated. Indeed, it has been an invariable finding that in all experiments in which degeneration of the dorsal nuclei has occurred there is an accompanying gliosis in the septo-mesencephalic tract. This is emphasized by the next experiment in which division of the septo-mesencephalic tract has resulted in selective degeneration of the dorsal nuclei.

In the left hemisphere of OP 8 there is a barely detectable needle track which passes downwards and backwards through the medial parts of the accessory and dorsal hyperstriatum, neostriatum and palaeostriatum to reach the ventromedial angle of the hemisphere below the septum. Here there is a small focal lesion less than 1 mm. in diameter which has completely interrupted the fibres of the septo-mesencephalic tract, but has caused only minimal damage to the adjacent parts of the septum and medial preoptic areas (Text-fig. 4). Extending forwards and backwards from this lesion there is intense gliosis in the septo-mesencephalic tract which follows the course already described in OP 6. The gliosis can be traced in the molecular layer of the cortex medial to the ventricle as far dorsally as the superior angle of the ventricle and anteriorly to the level of the accessory hyperstriatum. In the thalamus of this side all the dorsal nuclei show degenerative changes. In the nucleus dorsomedialis the majority of the cells are shrunken and pale staining; there is comparable pallor of the cells of the nucleus superficialis parvocellularis; in the nuclei dorsolateralis anterior and posterior there is some degree of cell loss and quite marked shrinkage of the cells particularly in the posterior part of the nucleus. No other thalamic nuclei are affected. It may be concluded therefore that the septo-mesencephalic tract is the principal projection pathway of the dorsal nuclear group of the thalamus, whereas the other nuclei projecting upon the telencephalon send their fibres into the forebrain bundles.

The precise termination of the projection fibres in the septo-mesencephalic tract cannot be determined from our material. Examples have already been given, however, which indicate that not only is the projection independent of that of the central group of thalamic nuclei, but also that the different elements in the dorsal nuclear group project independently of each other. Thus in experiment OP 20, described in the previous section, the nucleus dorsolateralis anterior is severely degenerated while the dorsolateralis posterior remains unaffected. Similarly, in experiment OP 7 of the present series, the nucleus dorsomedialis anterior shows unequivocal retrograde degeneration, while on the side of the larger lesion in OP 8 this nucleus remains intact although all the other elements in the dorsal nuclear group are severely affected. In all the experiments so far described the lesions have been relatively large, and the resulting degeneration has always affected more than one of the dorsal nuclei. There are four brains in which degeneration is confined to only one of the dorsal nuclei after relatively small lesions in the anterior third of the hemisphere. Experiment OP 3 will be described as an example of this group.

In this brain the lesion begins at the frontal pole of the hemisphere where the dorsal half of the accessory hyperstriatum and rather less of the dorsal hyperstriatum are destroyed together with the entorhinal cortex. The lesion remains confined to the dorsomedial portion of the hemisphere, completely destroying the posterior part of the accessory hyperstriatum, the dorsal half of the entorhinal area and the dorsal third of the dorsal hyperstriatum. The cortex medial to the lateral

ventricle is preserved. Behind this the lesion becomes progressively smaller in size, destroying the dorsolateral cortex, the dorsal margin of the hyperstriatum and the lateral part of the entorhinal area. At the level of the septum it is confined to the dorsolateral and entorhinal cortical areas. In the thalamus the only nucleus which shows evidence of retrograde cell degeneration is the nucleus dorsolateralis posterior. Here there is a severe cell loss with marked shrinkage and pallor of the remaining cells and a moderately intense gliosis.

DISCUSSION

The surprising ease with which lesions could be placed in the brain of the pigeon and its remarkable ability to survive after quite extensive lesions have already been commented upon, but an equally unexpected finding is the strikingly clear-cut degenerative changes which can be found in the thalamic nuclei. This clarity of the degeneration may be attributed to two factors: first, to the very marked differentiation of most of the thalamic nuclei, and secondly, to the profound degree of cell change and gliosis which is found in those nuclei which project upon the telencephalon. The essential similarity of retrograde cell degeneration in the avian pontine homologue to that found in the pontine nuclei of mammals has been commented upon by Brodal, Kristiansen & Jansen (1950). These observations are in striking contrast to the findings of Powell & Kruger (1960) in a comparable study of the thalamic projection in the reptilian brain where the thalamic nuclei are poorly defined, and the cellular degeneration not at all marked, there being a noteworthy absence of gliosis. Indeed, in our experience, the retrograde degeneration found in the avian brain is as conspicuous as that seen in the mammalian thalamus after comparable survival periods.

The severity of the degeneration after complete removal of the telencephalon is by no means the same in all the affected thalamic nuclei so that it is possible to classify the nuclei on the basis of their reaction to telencephalic removal into two groups. In the first group are included those nuclei which undergo complete cell loss; in the second group are those nuclei which show unequivocal changes varying in degree from slight cell pallor to marked cell shrinkage and partial cell loss. The thalamic nuclei which show no change after total removal of the telencephalon may be considered as constituting a third group. Before discussing into which of these groups individual thalamic nuclei should be placed it is necessary to discuss briefly the possible significance of the different cellular reactions. The simplest explanation of these findings, suggested by analogy with the mammalian thalamus, is that all the affected nuclei send their axons to the telencephalon, and conversely those nuclei which show no change have no such telencephalic projection. The evidence upon which this explanation rests need not be discussed here as it has been fully reviewed for the mammalian thalamus by Walker (1938) and Rose & Woolsey (1943). An alternative interpretation which attempts to explain the specific differences in the degree of cell change between different nuclei is that only those nuclei which show complete cell loss (e.g. the nucleus rotundus) project exclusively upon the telencephalon. The less severe changes in other nuclei would then be explicable on the basis that they either give off collaterals to other diencephalic or brainstem structures which are capable of partially maintaining the integrity of the cell, or that the

degeneration in these nuclei is not due to a direct telencephalic projection but is secondary to the complete degeneration in nuclei like the rotundus. A third possibility which has to be considered is that some or all of the cellular changes in the thalamus are the manifestations not of retrograde degeneration but of transneuronal degeneration. That is to say, they have resulted not from axonal section but as a direct consequence of removal of descending connexions from the telencephalon. With the material available at present it is impossible to exclude this possibility, but from what is known of transneuronal degeneration in the mammalian brain it is highly improbable that the very severe cell loss found in the nuclei rotundus and ovoidalis is simply due to the de-afferentation of their cells, especially since adult animals have been used throughout this study. One final consideration which must be mentioned is the possibility that even those nuclei which show no evidence of cellular degeneration after complete removal of the telencephalon do in fact project upon the telencephalon but, like many cells in the mammalian cerebral cortex or the hippocampal pyramids, they are apparently unaffected by axonal section. While it is necessary to point out these possibilities, for the purpose of this discussion we shall assume that the first interpretation is the most probable and shall discuss our findings in this light.

The total thalamic projection upon the telencephalon as determined by the technique of retrograde cell degeneration comprises the three most conspicuous nuclei: nuclei ovoidalis, rotundus and subrotundus; the nuclei of the dorsal group: dorsomedialis anterior, dorsolateralis anterior and posterior and the superficialis parvocellularis, together with the entopeduncular, and the three nuclei posterior to the nucleus rotundus, viz. the postrotundus (including the so-called nucleus posterointermedialis), the subpretectal nucleus and the posteroventral nucleus. Of these nuclei, the first two and both parts of the dorsolateral nucleus undergo severe cell loss after removal of the telencephalon, but in the remainder the essential cellular change is in the form of shrinkage and pallor. The nuclei which are apparently unaffected by telencephalic removal include, amongst others, such well-defined masses as the so-called lateral geniculate nucleus and habenular nuclear group. The absence of change in the lateral geniculate is particularly deserving of comment in view of the fact that optic nerve fibres have been found to terminate in this nucleus. We have no evidence regarding the projection of this and the other nuclei which are unrelated to the telencephalon.

Although the nuclei which project upon the telencephalon may be divided on the basis of their cellular reaction to telencephalic removal into two main groups, the experiments presented in § II provide another and probably more significant classification because a correlation of the site of the lesion, and the distribution and severity of the resulting thalamic degeneration makes it clear that these nuclei project upon two distinct regions of the telencephalon and do so by way of quite separate efferent pathways. Thus the dorsal group of nuclei have been shown to project through the septo-mesencephalic tract to the dorsomedial margin of the hemisphere while all the other nuclei are related to the palaeostriatum. The connexions of each of these nuclear groups will be considered separately.

The dorsal nuclei

One of the most interesting results of these experiments is the finding that the dorsal nuclei are related not only on topographical grounds but also in their efferent connexions. This group is composed of four nuclei—the nuclei dorsolateralis anterior and posterior, dorsomedialis anterior and superficialis parvocellularis—and these are the only thalamic nuclei which project to the telencephalon outside the palaeostriatum. Although the precise site of termination of the efferent fibres from these nuclei has not been determined there is no doubt that they all project upon the dorsomedial margin of the hemisphere, to either the accessory hyperstriatum or to the adjacent entorhinal cortical area. Furthermore, several experiments have been described which show that these nuclei may degenerate independently of each other, and that there is a topical organization in the projection of this group. It has also been shown that they share a common projection pathway, the septo-mesencephalic tract, and in this respect they differ significantly from the remaining nuclear groups. The probable reason why it has not been possible to determine the site of termination of these nuclei is to be found in the Bodian-stained sections of this material. These sections show that while the septo-mesencephalic tract forms a compact bundle as it passes through the septum and indeed almost as far as the dorsomedial margin of the hemisphere, beyond this its fibres then fan out widely in the anteroposterior and mediolateral directions. It is clear from this that even quite small lesions are likely to interrupt the efferent fibres from more than one nucleus. The significance of the differences in the degree of cellular degeneration which have always been found in the four elements of this group is not at all clear. It does, however, suggest a difference in the pattern of the projection of the nuclei dorsomedialis anterior and superficialis parvocellularis on the one hand and the nuclei dorsolateralis anterior and posterior on the other.

Nuclei rotundus, subrotundus and ovoidalis

It has been possible to determine fairly precisely the projection of these three conspicuous nuclei because the retrograde cell degeneration in these elements is most profound and consequently degenerated areas within them stand out with remarkable clarity. The evidence for the conclusion that all three nuclei project to the palaeostriatum has already been given in some detail in the results, and also that, of the two segments of the palaeostriatum, it is to the palaeostriatum augmentatum rather than to the palaeostriatum primitivum that they send their axons. From a correlation of the site of the damage in the palaeostriatum and the distribution of the degeneration in these nuclei in several experiments it can be stated that these three nuclei project independently of each other, and that within the projection of each nucleus there is a precise topographical organization. In this way it has been demonstrated that the topographical relation of these nuclei to each other is paralleled in the comparable organization of their projection fields. Thus the nucleus rotundus, which in the thalamus is the most lateral of the three and also extends farthest anteriorly, projects to the anterolateral part of the palaeostriatum augmentatum. Similarly, the nucleus ovoidalis which lies most medial, opposite the posterior part of the nucleus rotundus, projects to the posteromedial part of the

palaeostriatum. The precise projection of the nucleus subrotundus is less adequately documented, but it is interesting that all the evidence points to the fact that this nucleus, which is situated between the nuclei rotundus and ovoidalis, projects to an intermediate area in the palaeostriatum. Not only is it clear that these nuclei project to an entirely different part of the telencephalon than the dorsal nuclei, but it has been shown that the course of their efferent fibres through the lateral forebrain bundle is quite separate from that of the dorsal group.

The entopeduncular nucleus and the posterior nuclear group

Little can be added to the conclusions about the projection of these nuclei, which have been already discussed with the results. It is clear that they differ in the pattern of their projection from the other main nuclei in that they undergo only partial degeneration, and that they project together to the same region, as degeneration in one nucleus is always associated with degenerative changes in all the others. They do not degenerate with the dorsal nuclear group, but evidence has been presented that they are always affected when the nuclei rotundus, subrotundus and ovoidalis degenerate, either alone or together. It is not at all clear, however, whether they project in a more or less diffuse way to the palaeostriatum, or whether the degenerative changes seen in these nuclei are the result, not of direct axonal injury, but are in some way secondary to the changes in the nuclei ovoidalis and rotundus. It is nevertheless certain that they do not project diffusely to the whole telencephalon, as will become clear from the fuller discussion of this point below.

The entopeduncular nucleus appears to differ from the nuclei posterior to the nucleus rotundus in at least one respect: in the posterior nuclei degeneration has never been seen to be localized to one part of a given nucleus, but in the entopeduncular nucleus there is evidence of a mediolateral organization comparable to that found in the nucleus rotundus. Thus in one experiment described (OP 5) the medial part of the entopeduncular nucleus has degenerated together with the nucleus ovoidalis, while in another experiment (OP 23) degenerative changes have been found only in the lateral part of the nucleus in association with degeneration in the rotundus.

In this discussion it has been assumed that if retrograde degeneration occurs in a particular nucleus after a lesion in a localized part of the telencephalon, that the projection fibres from that nucleus *terminate* in the damaged area. In the case of the dorsal nuclei this assumption is no doubt justifiable, since the degenerative changes in these nuclei have occurred after relatively superficial lesions and hence presumably at the site of termination of the efferent fibres. On the other hand the validity of this premise may well be questioned for those nuclei which only degenerate after rather deep lesions involving the palaeostriatum. For example, it might be suggested that cellular degeneration in these nuclei is not due so much to the destruction of the termination of their efferents as to damage of the projection fibres as they pass through the palaeostriatum. This possibility cannot be completely excluded as we have no single experiment in which the whole telencephalon, with the exception of the palaeostriatum, has been destroyed, but a number of experiments have been described in which large areas of the telencephalon have been damaged without even minor changes in these thalamic nuclei. Support for this suggestion may be

derived from the older literature in which connexions have been described between these thalamic nuclei and the neostriatum and ektostriatum. These observations appear to be based largely on the study of normal material from which the precise site of termination of fibres or even the direction of conduction cannot usually be determined. In view of these difficulties we shall not compare our findings in relation to specific nuclei with those of previous workers, and judgement on this problem must be suspended pending further study with the more refined silver degeneration techniques after lesions in the thalamus.

In conclusion it may be stated that within the limitations of the method of retrograde cell degeneration the thalamic nuclei appear to project to two distinct parts of the telencephalon—the dorsomedial margin of the hemisphere and the palaeostriatum—and to do so by way of two separate pathways, the septo-mesencephalic tract and the lateral forebrain bundle. The distinctly different structure of these two projection areas strongly suggests that these two groups of nuclei are functionally distinct, and it would not be unlikely if they differed in their afferent connexions.

It would be tempting on the basis of these results to suggest homologies between the various elements of the avian thalamus and those of the reptilian and mammalian brain. For example, there is now evidence that the morphologically similar nucleus rotundus of the lizard and pigeon thalamus both project upon the palaeostriatum; however, in the absence of more precise knowledge of their afferent connexions it would be unwise to conclude that such an homology is in fact established. It is important to emphasize that the avian thalamus as a whole is considerably more developed than that of the reptile and this factor alone should warn against a too-facile drawing of homologies. Similarly, it might be suggested that those nuclei of the avian thalamus which degenerate after removal of the telencephalon are collectively homologous with the dorsal thalamus of the mammalian brain, but further studies, particularly of the afferent connexions, may well show that the avian thalamus is in fact more closely related to the mammalian ventral and/or subthalamus. That homologies with specific nuclei of the mammalian thalamus cannot be drawn is apparent from what is already known of the lateral geniculate nucleus. The absence of retrograde degeneration in this nucleus after complete removal of the telencephalon indicates that it cannot be homologous with that element of the mammalian lateral geniculate body which is derived embryologically from the dorsal thalamus. With regard to the afferent connexions the recent work of Erulkar (1955) has raised another difficulty which must be considered in any discussion of thalamic homologies. Using electro-physiological techniques Erulkar could find no evidence for a thalamic relay of auditory and tactile impulses although responses could be recorded in a localized region of the neostriatum caudale. From these observations he has concluded that the avian thalamus contains no structure homologous with the primary relay nuclei of the mammalian dorsal thalamus. An important problem which these findings raise is the question of the afferent connexions of the cortex, hyperstriatum and neostriatum. It is possible that these areas do receive afferent impulses from the diencephalon or lower levels, and the absence of cellular degeneration in other nuclei of the diencephalon or midbrain (although the latter has not been systematically studied) does not exclude such connexions.

Indeed, the experimental findings of Erulkar (1955) may be taken as evidence of such a connexion between the complex of the nucleus isthmi of the midbrain and the neostriatum caudale, even though in our material these nuclei show no evidence of retrograde cell degeneration after complete removal of the telencephalon. The anatomical basis for the isthmo-striatal relations indicated by Erulkar's work requires further investigation, as does the problem of the source of the afferents to the different telencephalic areas and the elucidation of the organization within the telencephalon.

SUMMARY

1. The total thalamic projection upon the telencephalon and the organization of the projection of the individual nuclei of the thalamus have been investigated in the pigeon (*Columba livia*) using the technique of retrograde cell degeneration.

2. The thalamic nuclei which have been found to project to the telencephalon include the nuclei rotundus, subrotundus, ovoidalis, dorsolateralis anterior and posterior, dorsomedialis anterior, superficialis parvocellularis, postrotundus, posteroventralis, entopeduncularis and the subpretectalis.

3. On the basis of the severity of the cellular reaction to complete removal of the telencephalon these nuclei may be divided into two main groups: those which undergo complete cell loss (e.g. the rotundus) and those in which the essential change is in the form of cell shrinkage and pallor (e.g. the nucleus dorsomedialis anterior).

4. The nuclei which show no change after total ablation of the telencephalon include those of the habenular complex and the so-called lateral geniculate nucleus.

5. The dorsal nuclear group, comprising the nuclei dorsomedialis anterior, dorsolateralis anterior and posterior and the nucleus superficialis parvocellularis, have been found to project upon the dorsomedial margin of the hemisphere (to either the accessory hyperstriatum or the entorhinal cortex or both). The efferent fibres from these nuclei pass in the septo-mesencephalic tract.

6. The central group of nuclei, consisting of the three most conspicuous thalamic nuclei, viz. nuclei rotundus, subrotundus and ovoidalis, are related to the palaeostriatum augmentatum. These nuclei project independently of each other and within the projection of each of these nuclei there is a precise topical organization. The efferent fibres from these nuclei pass through the lateral forebrain-bundle.

7. It has not been possible to define the precise mode of projection of the entopeduncular nucleus or of the nuclei of the posterior group (nuclei posteroventralis, postrotundus and subpretectalis), but it appears that they either project upon the palaeostriatum augmentatum or alternatively the degeneration seen in these nuclei after lesions of the palaeostriatum is secondary to that found in the three central nuclei.

8. It does not appear possible, solely on the basis of the telencephalic projection, to homologize individual elements of avian thalamus with specific nuclei or nuclear groups in either the reptilian or mammalian thalamus.

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ABBREVIATION

AHS	Accessory hyperstriatum	*NR*	Nucleus rotundus
AS	Archistriatum	*NS*	Neostriatum
C	Cerebellum	*NSC*	Neostriatum caudale
DLC	Dorsolateral cortex	*NSF*	Neostriatum frontale
EA	Entorhinal area	*NSI*	Neostriatum intermediale
EM	Nucleus ectomamillaris	*NSP*	Nucleus superficialis parvocellular
EN	Entopeduncular nucleus	*NSPI*	Nucleus spiriformis
H	Hippocampal cortex	*NSR*	Nucleus subrotundus
HS	Hyperstriatum	*OB*	Olfactory bulb
LFB	Lateral forebrain bundle	*OC*	Optic chiasma
LGN	Lateral geniculate nucleus	*OT*	Optic tract
LHN	Lateral habenular nucleus	*OTC*	Optic tectum
MFB	Medial forebrain bundle	*PC*	Pyriform cortex
MHN	Medial habenular nucleus	*PSA*	Palaeostriatum augmentatum
MT	Marginal tract	*PSP*	Palaeostriatum primitivum
NDLA	Nucleus dorsolateralis anterior	*S*	Septum
NDLP	Nucleus dorsolateralis posterior	*SMT*	Septo-mesencephalic tract
NDM	Nucleus dorsomedialis anterior	*TG*	Nucleus subpretectalis
NO	Nucleus ovoidalis	*VP*	Nucleus ventralis posterior

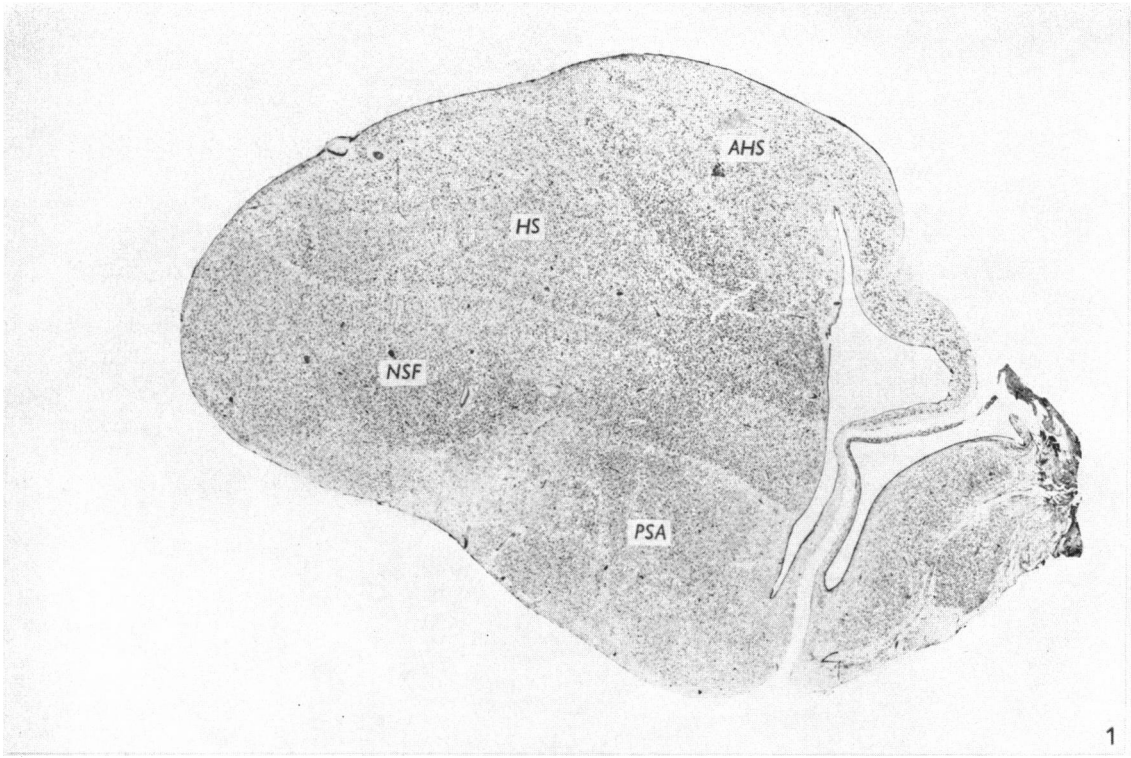
EXPLANATION OF PLATES

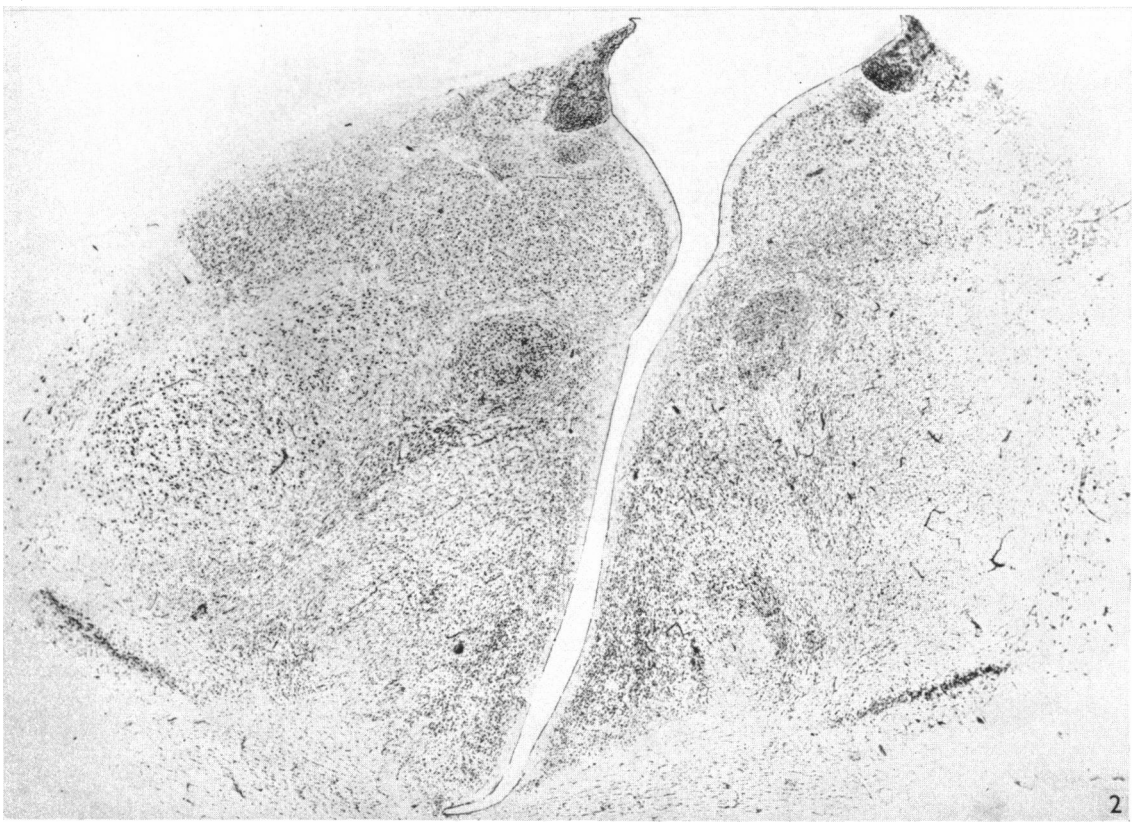
All the figures are photomicrographs of thionine stained sections.

PLATE 1

Fig. 1. A low-power photomicrograph of a transverse section through the telencephalon just rostral to the septum to show the extent of the lesion in the right hemisphere in experiment OP 20. ×11.

Fig. 2. Photomicrograph of a section through the middle of the thalamus to show the severity and extent of the degeneration in the nuclei of the central and dorsal groups in experiment OP 20. ×24.





POWELL AND COWAN—THALAMIC PROJECTION UPON TELENCEPHALON IN THE PIGEON

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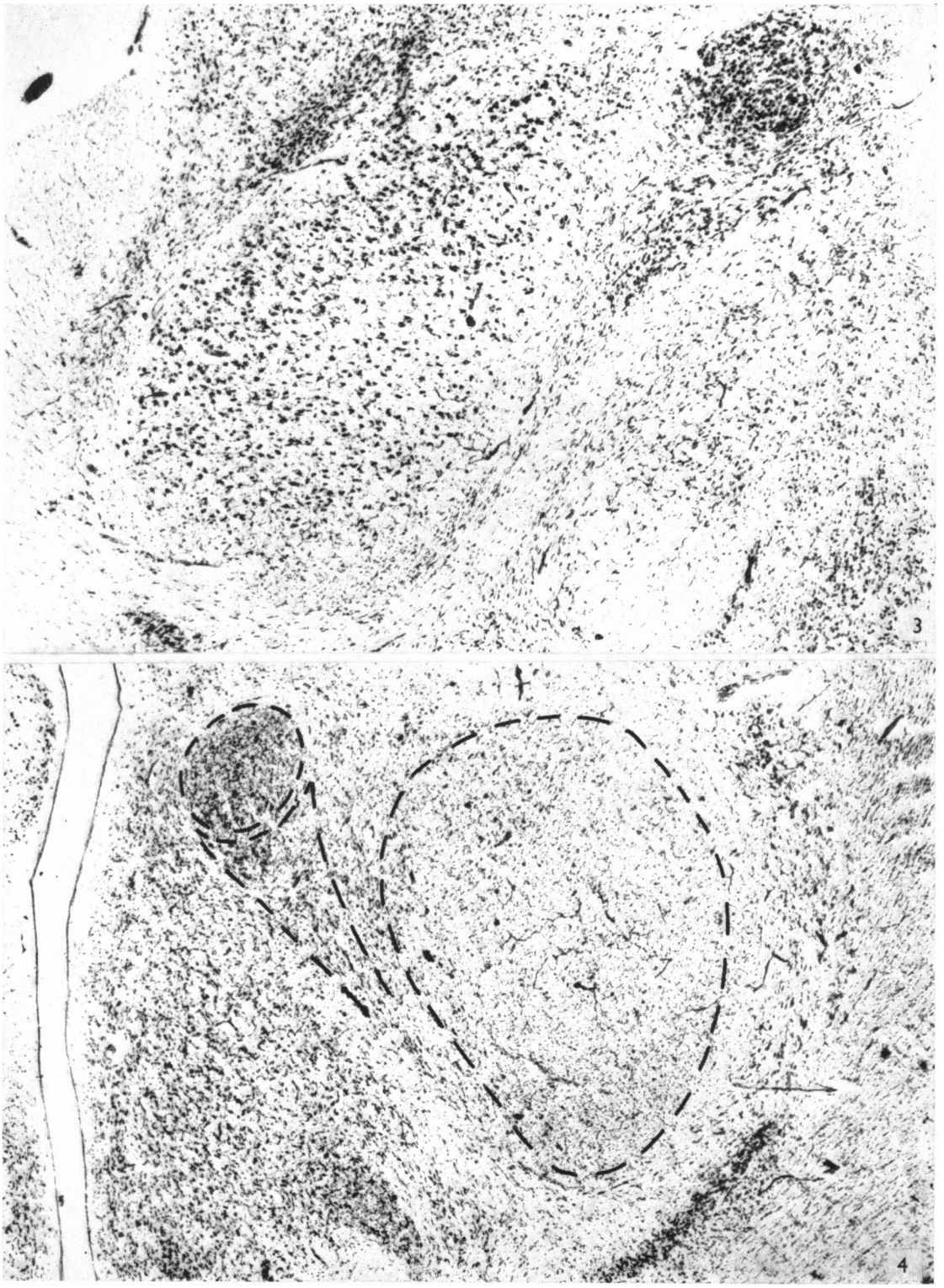

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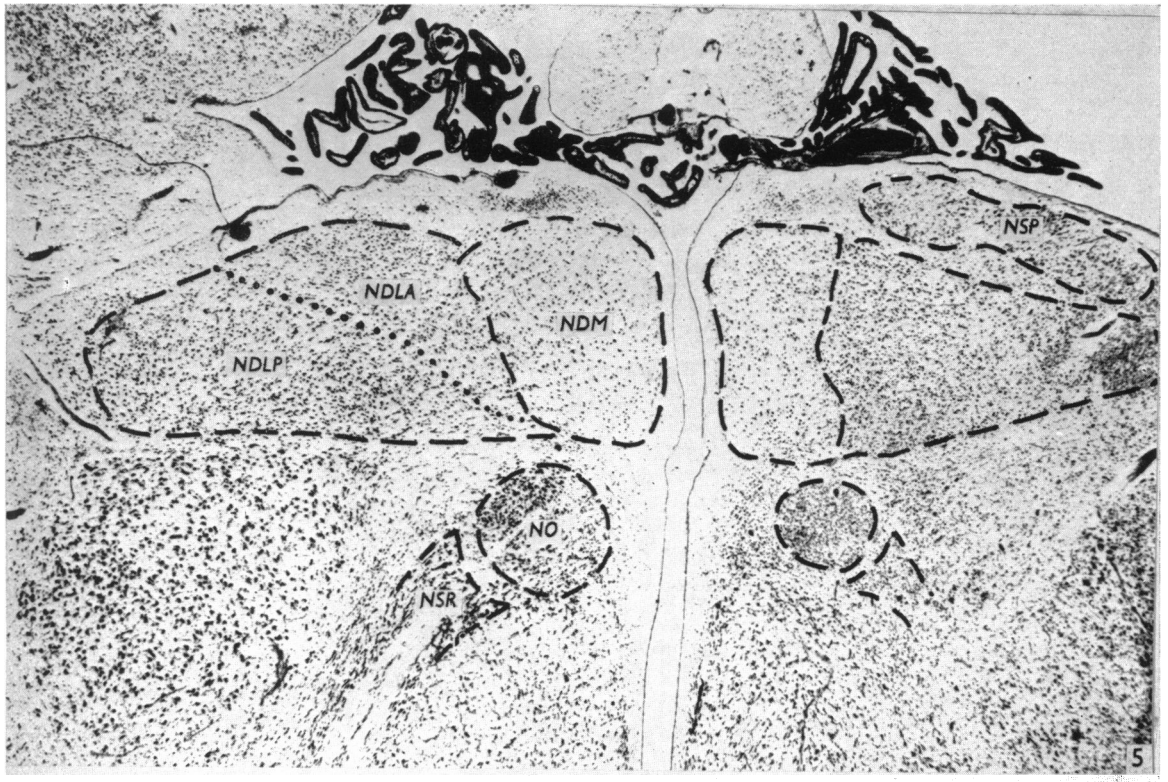
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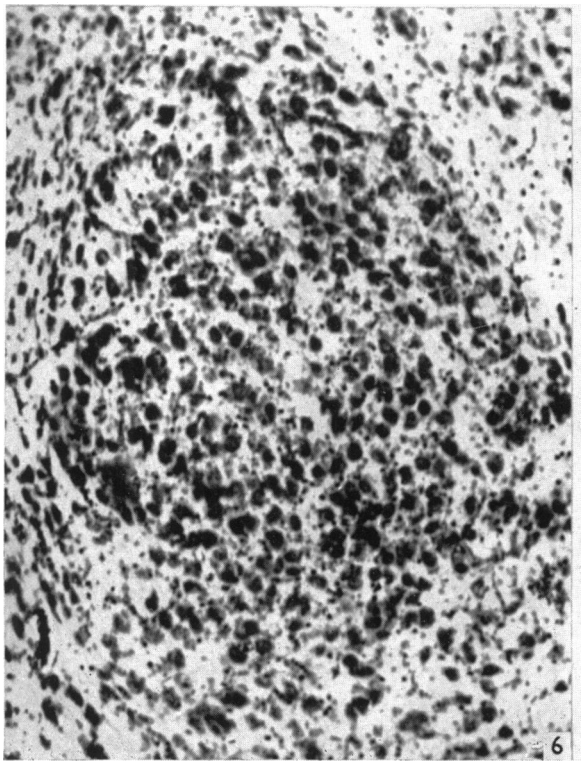
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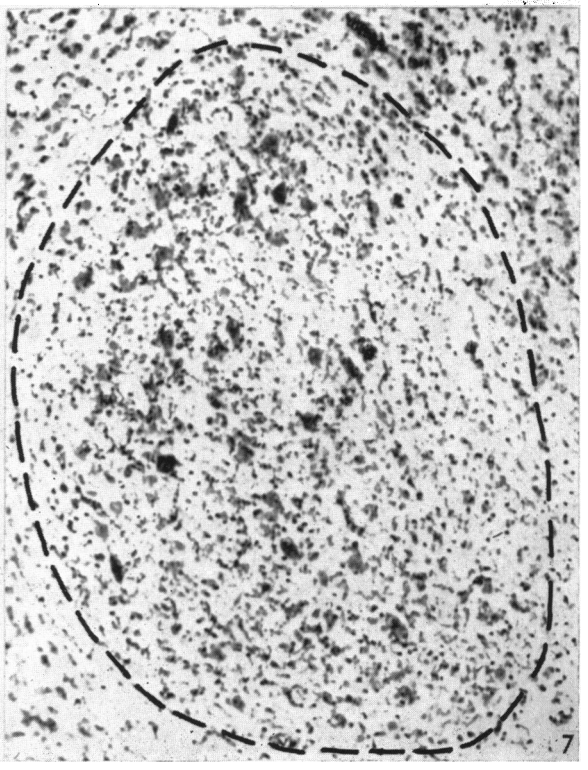
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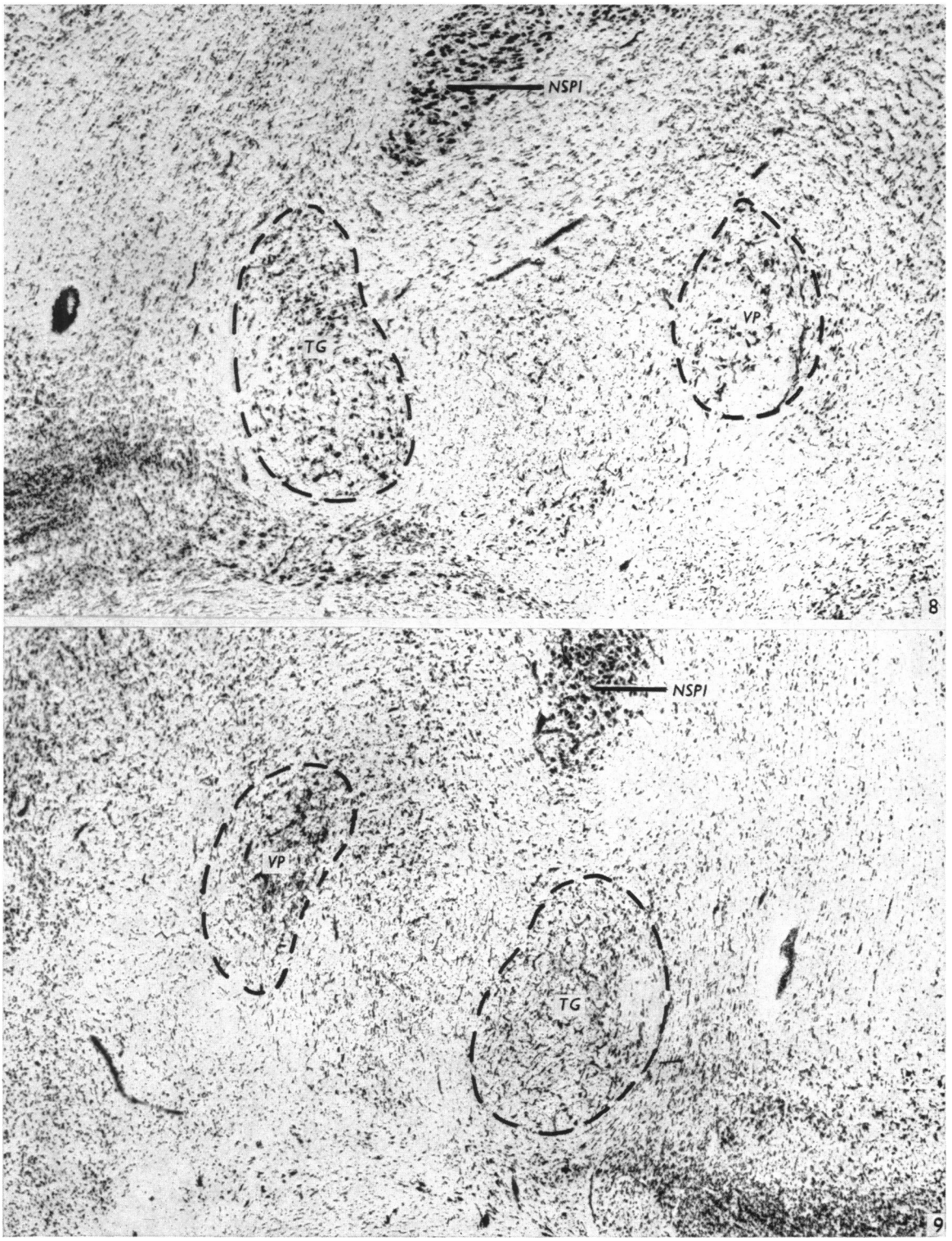

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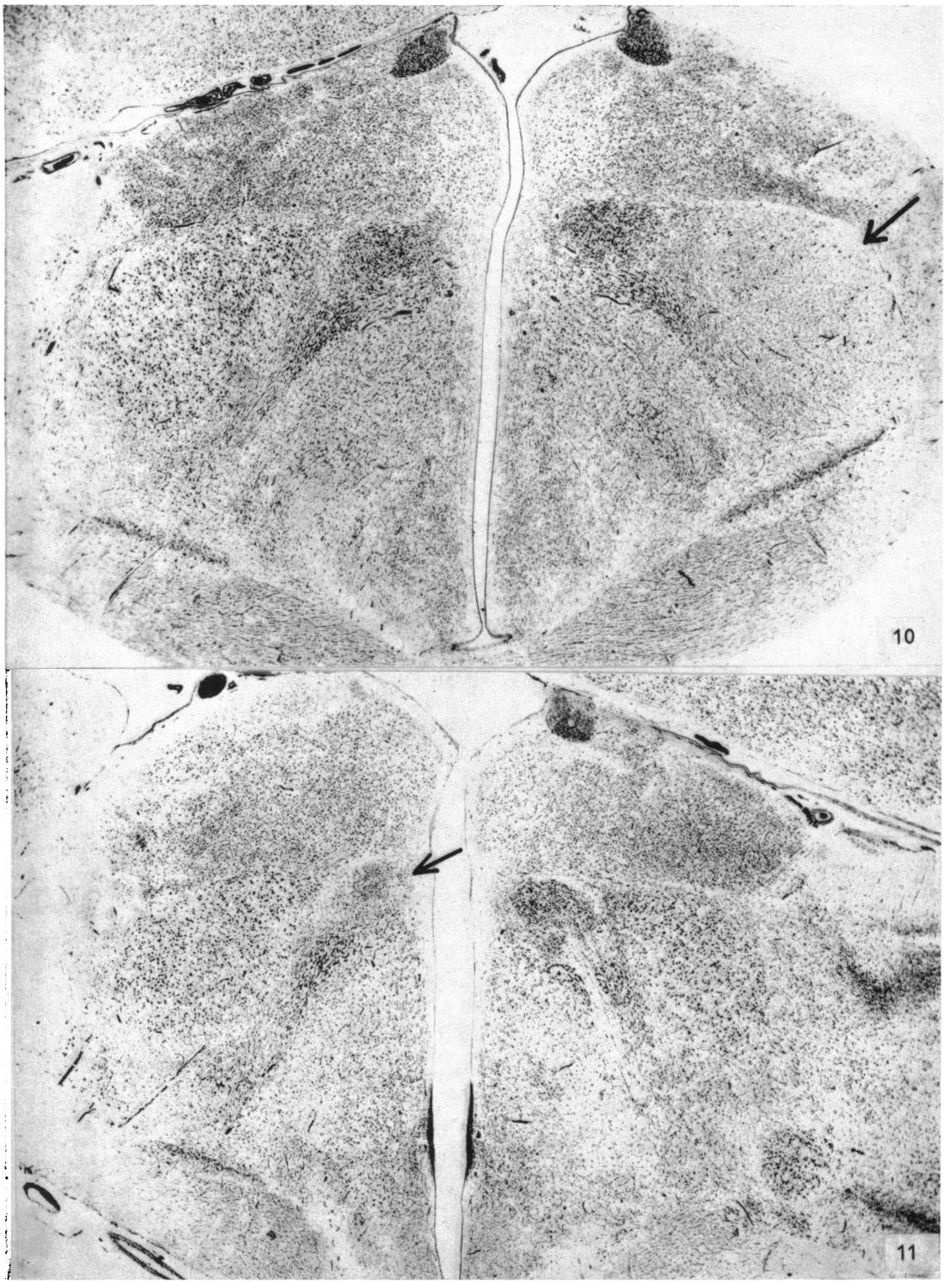

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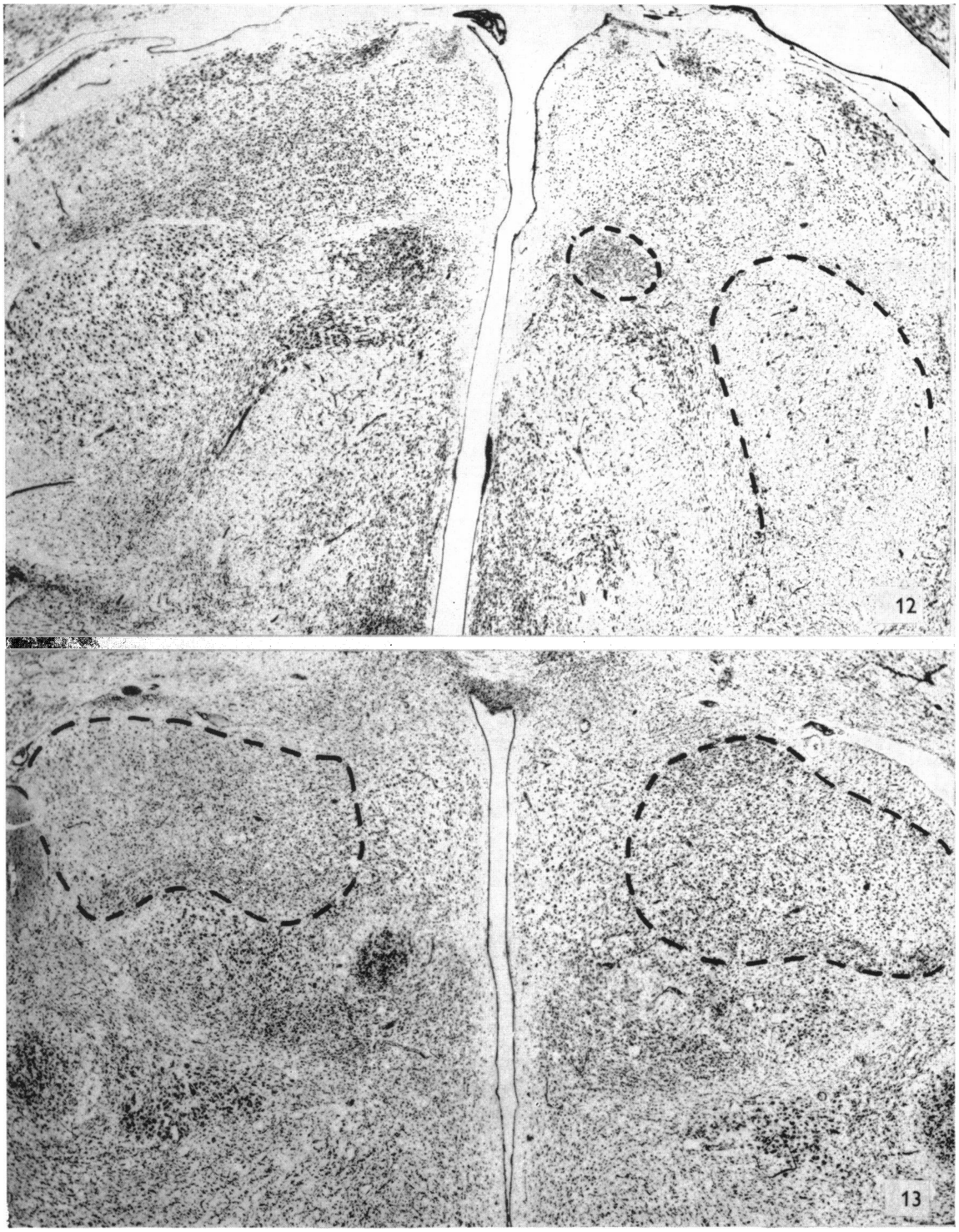

POWELL AND COWAN—THALAMIC PROJECTION UPON TELENCEPHALON IN THE PIGEON

PLATE 2

Figs. 3, 4. Photomicrograph at higher magnification to show the severity of the degenerative changes in the central group of nuclei: nucleus rotundus, ovoidalis and subrotundus compared with the normal side. In Fig. 3 the normal appearance of these nuclei on the unoperated side is shown; the degenerated nuclei of the operated side are outlined in Fig. 4. ×42.

PLATE 3

Fig. 5. A low-power photomicrograph of the dorsal nuclei in experiment OP 30 to show the extent of the degeneration in the dorsolateral and dorsomedial nuclei. The dotted line marks the boundary between the nuclei dorsolateralis anterior and posterior on the normal side. The degenerated nuclei ovoidalis and subrotundus are also shown. ×24.

Figs. 6, 7. High-power photomicrograph of the normal nucleus ovoidalis (Fig. 6) and the degenerated nucleus of the opposite side (Fig. 7) in experiment OP 20. ×154.

PLATE 4

Figs. 8, 9. The nuclei ventralis posterior and subpretectalis on the normal (Fig. 8) and operated (Fig. 9) sides in experiment OP 20. Note the absence of changes in the adjacent nucleus spiriformis. ×42.

PLATE 5

Fig. 10. The thalamus of experiment OP 23 to show the severe retrograde cell degeneration in nucleus rotundus (indicated by arrow). Note the absence of degeneration in nucleus ovoidalis. ×24.

Fig. 11. The thalamus of experiment OP 31 to show the degeneration in the nucleus ovoidalis of the left side (indicated by arrow). Note the preservation of the nuclei rotundus and subrotundus. ×24.

PLATE 6

Fig. 12. Photomicrograph of a transverse section of the thalamus of experiment OP 21 to show the virtual preservation of the nucleus subrotundus in contrast to the severe degeneration of the adjacent nuclei rotundus and ovoidalis (outlined by broken lines). ×24.

Fig. 13. Photomicrograph of a horizontal section of the thalamus of experiment OP 63 to show the marked degree of retrograde cell degeneration in the nuclei dorsolateralis anterior and posterior of the left side. The nucleus dorsomedialis anterior shows little change. ×24.