Early Lesions of Superior Colliculus: Factors Affecting the Formation of Abnormal Retinal Projections; pp. 73–90

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Abstract
The longest fibers of the main optic tract reach the upper layers of the superior colliculus (SC) of the midbrain. Destruction of this terminal area in newborn hamsters caused striking anomalies in the distribution of the optic tract, studied after the animals were fully grown. Evidence of termination was found in areas normally devoid of such termination: in the remaining tissue of the colliculus and in the thalamic nucleus lateralis posterior (LP). An abnormally high density of termination was found in part of the ventral nucleus of the lateral geniculate body. These thalamic regions normally receive connections from SC. Retinofugal axons could also be induced to terminate in the medial geniculate body of the thalamus if the brachium of the inferior colliculus, which normally carries auditory information to this cell group, was ablated at birth together with the lesion of SC. If the superficial layers of SC were destroyed unilaterally at birth, axons from the eye contralateral to the lesion not only reached the area of early damage, but also formed an abnormal decussation, crossing the tectal midline to terminate in the medial zone of the undamaged colliculus. Axons from the two eyes competed for terminal space in this intact colliculus, for they terminated in a nonoverlapping manner, and if the axons from the eye contralateral to the remaining SC were eliminated at birth, the anomalously recrossing axons increased in quantity and spread across the entire SC on the 'wrong' side of the midbrain. Hamsters with such an anomaly showed wrong-direction turning in response to visual stimuli in a large part of the visual field.

The less the amount of termination found in SC, the greater was the amount in LP. Thus, optic tract axons showed a 'pruning effect' which may be attributed to a tendency for axons to conserve the quantity of their terminal arborizations. The pruning effect alone may account for the hypertrophy we found, after early SC lesions, of the dorsal terminal nucleus of the accessory optic tract. The tendency to invade vacated terminal space may be sufficient to account for an effect of early unilateral eye removal, namely, a pronounced increase in an ipsilateral retinal projection to the medial terminal nucleus of the accessory optic tract. The greatest alterations in axonal projections were seen when the two effects, competition and pruning, seemed to act jointly. Additional factors may have to be considered in fully explaining such neuroplasticity; some of these have been suggested.
The concept of neuroanatomical plasticity in the central nervous system, involving changes in neuronal connections, has rather diverse connotations. For those concerned with functional effects of cerebral lesions, posttraumatic neuronal growth constitutes a potentially important factor in recovery of function. On the other hand, it is also conceivable that such anomalous growth could be maladaptive in certain cases, preventing functional recovery, depending on the locus of the lesion and the factors controlling the posttraumatic growth. Adaptive, compensatory neuronal plasticity has been introduced particularly often as one possible mechanism for sparing of function after brain lesions inflicted early in life, where such sparing can be considerably greater than after similar damage in adulthood [Teuber, 1970]. Yet it is far from clear in what sense the young brain can be said to be more plastic than the brain of the adult. To find structural correlates of such plasticity, W. J. H. Nauta, my mentor in neuroanatomy, and I planned, some six years ago, to test for it in the visual system of animals after lesions in the neonate or adult, using the experimental neuroanatomical techniques of his laboratory, together with methods of assessing behavioral capacities [Schneider, 1966]. We were encouraged in this approach by the reports of experiments with adult cats by Liu and Chambers [1958] and McCouch et al. [1958]. They presented both histological and electrophysiological evidence that dorsal root axons can sprout new terminals in areas of the cat spinal cord where terminal sites were vacated by degeneration of adjacent dorsal root axons or by degeneration of transected descending tracts. Furthermore, they showed that the maladaptive reflex spasticity which develops after cord transections may be partly explained by the formation of these anomalous connections. Just as our experiments were beginning, our hopes were reinforced by a report of Goodman and Horel [1966] showing a possibly similar sprouting of axons in certain termination areas of the optic tract in rats, where retinal projections appear to increase in density after visual-cortex ablations in young adult animals. Further evidence of structural plasticity came from work reported by Hamori [1968] and by Raisman [1969]. Hamori presented electron-microscopic evidence of an altered distribution of what appeared to be axo-axonic synapses in the lateral geniculate nucleus of adult cats after eye removal. Raisman used a more readily interpretable paradigm, reporting ultrastructural evi-
dence for a lesion-induced alteration in synaptic loci in the septal area of adult rats: after removal of one major input to the septal neurons, the synapses of the fibers of a second major input appeared to have expanded their distribution on the surfaces of recipient neurons.

Thus, the situation seemed somewhat paradoxical. Although common belief considered the young brain to be 'more plastic' than the adult brain, there existed more evidence for neuroanatomical plasticity after lesions in mature animals. But, by then we had already found evidence of a rather considerable alteration in retinal projections which could be produced reliably in golden hamsters by damage to the superior colliculus in the neonate [SCHNEIDER and NAUTA, 1969], and not in the adult according to presently available data. We found examples of superinnervation of certain structures, and of totally new connections not seen in normal brains. These results supported our expectation that the plasticity of the young and growing brain might be expressed, after early lesions, in discernable alterations in the patterns of axonal connections. Moreover, it looked as though some of these changes had definable functional consequences. After bilateral ablation of the superficial layers of the superior colliculus of neonate hamsters, the anomalous retinal projections were primarily of two sorts. Retinofugal axons could be traced into parts of the remaining deep layers of the colliculus (as defined by fiber architecture) where they do not normally terminate. Other anomalous projections were found in the diencephalon (in the nucleus lateralis posterior and in the ventral nucleus of the lateral geniculate body). Visual control of turning movements by these hamsters was correlated with the amount and pattern of the anomalous projections into the midbrain [SCHNEIDER, 1970] and not with the quantity of anomalous projections into the diencephalon. This was more than a suggestive correlation, considering the finding that hamsters with complete colliculus lesions made in adulthood seem never to recover such visuomotor control [SCHNEIDER, 1967, 1969]. Furthermore, visual-cortex ablation did not abolish the spared visually elicited turning ability in hamsters with early bilateral tectum lesions (unpubl. data on 5 subjects).

Such promising results have led me to continue this line of investigation. In the present report, I would like to take the opportunity to present an overview of work in my laboratory, trying to concentrate on experiments designed to test for various factors which might be responsible for the formation of the anomalous connections. Two of these factors, I would like to convince you, have proved to be of major importance, acting either together or separately. One is a tendency for axons to fill vacated terminal space, and to compete with other axons for exclusive occupancy of such space. The other is a kind of compensatory sprouting that has been more familiar to the arborist, tending the growth of trees by pruning, than to the neuroanatomist. It appears to represent a tendency of axons to conserve at least a minimum quantity of their terminal arborizations.

A further finding in these studies brought with it a behavioral effect unexpected for mammals. Optic tract axons can be induced to grow to the wrong side of the midbrain, and this structural anomaly is correlated with a specific behavioral anomaly - visually elicited turning in the wrong direction [SCHNEIDER, 1971]. The latter phenomenon brings the analysis of structure and function into direct apposition.
General Methods

A female Syrian hamster (*Mesocricetus auratus*) generally gives birth on the 16th day after mating. A photograph of the brain of a hamster pup on the day of birth is shown in figure 1, next to a photograph of the brain of a fully grown hamster. The brain reaches full size in only 3 months. In the newborn, the entire surface of the superior colliculus is visible through a very thin interparietal bone. During postnatal development, the cerebral hemispheres grow to cover much of the colliculi, as can be seen in the figure. Also, the interparietal bone ossifies and increases greatly in thickness. In the experiments to be described, most of the early lesions of the superior colliculi were made by applying heat to the overlying skull by several applications of the flattened head (1 mm diameter) of a pin heated in an alcohol flame, after the skull behind the transverse sinus had been exposed through a small incision. Except for mild hypothermia, no anesthesia was used for animals 0–3 days old.

A typical experimental sequence can be summarized as follows. On the day of birth, a pup was removed from the nest along with about a third of its littermates, and the superficial layers of the superior colliculus on one side were destroyed as described above, with the aid of a binocular operating microscope. (In some pups, the lesion was made bilaterally, or one eye was removed in addition.) After the incision was sutured and the littermates operated, the pup was returned to the nest. Along with its littermates, all of which were operated, it was reared by the mother until weaning at the age of 3–4 weeks. After it was fully grown (12 weeks), its behavior was studied for visually elicited turning movements (omitted for some animals). Finally, the animal was used in a neuroanatomical experiment, e.g. one eye was removed and after 5 more days the brain was prepared according to modified Nauta techniques for the staining of degenerating axons and boutons [FINK and HEIMER, 1967; NAUTA and EBBESON, 1970]. Serial sections, cut frozen at 30 \( \mu \text{m} \) thickness in the coronal plane, were generally stained at 150 \( \mu \text{m} \) intervals, with parallel series stained for cell bodies with cresylecht violet, and for normal axons with another modified Nauta method [FINK and SCHNEIDER - see SCHNEIDER, 1969, note 31] or with a Loyez stain for myelinated axons. After such preparation, the degenerating retinofugal axons were traced, with the aid of a light microscope, to their termination, and the results compared with those of similar experiments on normal animals and other early operates.

The success of this procedure depends on a phenomenon peculiar to the young brain. Axonal degeneration is very rapid in the neonate, so that in the adult brain, no stainable degeneration products remain from the early lesion. This result was verified in control cases in which no adult lesion was made before preparing the brains for staining.

The phenomenon was also studied by the staining of neonatal hamster brains after eye removal. In one such experiment, an eye was removed from each of 10 8-day-old hamsters, which were then killed after survival times of 8 h, 16 h, 1 day, 2 days, etc. up to 7 days, and the brains stained with the Fink-Heimer procedure 1, and with the method for staining normal axons. The silver deposits indicating axonal degeneration (seen best with the latter, nonsuppressive technique) could be observed in the optic tracts and optic terminal areas optimally after only 1 day, whereas they had virtually disappeared at 3 days and after all longer periods. These results contrast markedly with those for the adult
Syrian, hamster, in which terminal degeneration after eye removal is optimal after periods up to 7 days - depending on the region [SCHNEIDER, 1968], and in which axonal degeneration products can still be stained many months after the lesion, 14 months in the case of one hamster [SCHNEIDER, unpubl. data; cf. GOODMAN and HOREL, 1966].

The following results and interpretations are based on studies of 33 litters of golden hamsters with early lesions, especially on 44 cases with various combinations of neonatal superior-colliculus lesion and eye removal. In addition, I have made use of 39 cases with lesions made in adulthood for anatomical tracing of degenerating axons after eye removal (22 cases),
lesions of the superior colliculus (8 cases), pretectal region (8 cases), or inferior colliculus (1 case).

The Initial Findings of Abnormal Retinal Projections

The fiber architecture of the normal hamster’s superior colliculus is illustrated in coronal section in figure 2. Figure 3 shows a similarly stained section at the same level from the brain of a hamster which had suffered a heat lesion of the right colliculus on the day of birth. Note, on the side of early damage, the absence of the superficial gray layer, defined as the layer of cells located above the layer of optic tract fibers. Such fibers were found mainly on the surface of the operated colliculi. The deeper-lying cell and fiber layers appear somewhat reduced in thickness after the early lesions, the reduction varying greatly in extent; in some cases these layers were totally missing medially. Remnants of the superficial gray layer were found in a few cases of shallower lesion, while in the majority, there were no traces of it. In every case, the area of the lesion was free of the glial scars seen after brain lesions in adult animals. The wound bed was so ‘clean’ that the appearance suggested a congenital absence of part of the superior colliculus rather than a violent destruction of nervous tissue (fig. 4) [cf. SUMI and HAGER, 1968]. Evidence of some damage to the pretectal region was found in most cases.

The diagrams of figure 5 summarize the initial results, which were remarkably similar from brain to brain. Relevant optic tract connections are indicated in side views of the brainstem of a normal hamster and of an animal with early tectum damage [SCHNEIDER and NAUTA, 1969; SCHNEIDER, 1970]. The contrast between the normal and abnormal pattern of

Fig. 1. Left: brain of adult Syrian golden hamster, dorsal view. The brain is oriented in a standard stereotaxic position [SCHNEIDER, 1969]. Right: brain of hamster on day of birth, same scale.

Fig. 2. Superior colliculus, mid-rostral level, of normal adult hamster, as seen in coronal section stained for myelinated axons. Loyez stain. X 24.

Fig. 3. Superior colliculus, mid-rostral level, of adult hamster case 21–6, in which the superficial layers on the right side were destroyed on the day of birth. Coronal section chosen to match the one shown in figure 2. Loyez stain. X24.

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connections could be seen particularly clearly in comparisons of the two sides of the brain in 7 cases of unilateral early tectum lesion (day 0 to day 5) in which both eyes were removed in adulthood to produce stainable degeneration in both optic tracts and in their terminal areas. The anomalies were found only on the side of the early lesion (except as noted below). This result is illustrated in the charts of one case shown in figure 6, which I will describe now.

As the optic tract fibers ascend along the lateral margins of the diencephalon above and behind the optic chiasm, the first terminal area reached

Fig. 4. Section through midbrain of adult hamster case 17–3, in which a lesion of the right superior colliculus had been made at birth; matches lowest chart of figure 6. Cresylecht violet stain for cell bodies, X 22.
Fig. 5. Left: lateral-view reconstruction of rostral brainstem, of normal adult hamster. Heavy line depicts schematically the course of a group of optic tract axons and some of their terminations; the tecto-thalamic pathway is shown in a similar manner. Right: similar view of brainstem of adult hamster which had undergone destruction of the superficial layers of the superior colliculus in the neonate. Anomalous optic tract connections are depicted by double lines.

Abbreviations to Figures

AV  anteroventral nucleus of thalamus
MGB  medial geniculate body
CGS  central gray substance
MTN  medial terminal nucleus of accessory
colliculus

sory optic tract
DTN  dorsal terminal nucleus of accessory
obi. oblongata

optic tract
OCh  optic chiasm
IC  inferior colliculus
Olf. olfactory
L  lateral nucleus of thalamus
ped. cerebral peduncle
LGD  dorsal part of the lateral geniculate
body
PT  pretectal area
LGv  ventral part of the lateral geniculate
R  red nucleus (n. ruber)

SC
superior colliculus
LP
lateroposterior nucleus of thalamus
Thai.
thalamus
(n. lateralis posterior)
V
ventral nucleus of thalamus

is the ventral nucleus of the lateral geniculate body. Here, in a narrow laminar subdivision lying immediately adjacent to the optic tract, an ab-

2 Before this, a very small area of terminal degeneration is seen at the caudo-lateral edge of the suprachiasmatic nucleus of the hypothalamus. It appears to be similar on right and left sides in cases with early unilateral lesion of the superior colliculus. However, the staining of this region was probably not optimal with the survival time used.

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normally high density of terminal degeneration is consistently seen on the side with the early lesion of the superior colliculus. Next, the optic tract axons reach the dorsal nucleus of the lateral geniculate body, where the degeneration products indicative of termination appear to be stained in similar density on the two sides. However, it should be noted that with this method a small difference could escape detection in an area of such dense termination.

Above and medial to the lateral geniculate body, degenerating retino-fugal axons pass over and through the nucleus lateralis posterior (LP) of the thalamus. On the nonoperated side, almost all of these fibers appear merely to course past this area en route to the pretectal region and mesencephalic tectum, for only a single, very small nest of terminal degeneration appears here, closely beneath the optic tract (just anterior to the level shown in the middle chart of figure 6).

In each of the present cases of hamsters with early unilateral superior-colliculus lesions and bilateral optic tract degeneration, sections at the level of the caudal end of the lateral geniculate revealed a tiny clump of terminal degeneration in the LP near the optic tract on the 'normal' side. In another series of 9 hamsters, normal until the right eye was removed in adulthood, a tiny clump of terminal degeneration was seen on the left side in the LP on one or two sections in 6 cases, though all cases showed a few randomly oriented degenerating axons separated from the main bundles traversing the nucleus. Survival times varied from 2 to 14 days, all sufficient for clear terminal degeneration in the lateral geniculate nuclei and the superior colliculus. A similar tiny 'nest' of retinofugal termination in the LP has been found recently in 6 of 12 cats subjected to unilateral eye removal [HEDREIN, 1970]. As in the hamsters, the precise position and size of this small terminal area was somewhat variable. A similar finding in the pulvinar of the rhesus monkey and the baboon has been reported [CAMPOS-ORTEGA et al., 1970].

By contrast, inspection of the opposite side - the side of the early midbrain lesion - reveals a new, or greatly expanded, terminal field. Dense clumps of silver granules appear on this side among the degenerating axons of passage and a scattering of less densely packed granules (fig. 7). These silver deposits compose a characteristic picture of terminal degeneration [HEIMER and PETERS, 1968], similar to the picture seen in the nearby lateral geniculate body. Electron microscopic examination of the LP in two other cases with early tectum lesions confirms the existence of degeneration in synaptic end-structures [R. KALEL and G. E. SCHNEIDER, unpubl. data].
Thus, a considerable amount of thalamic tissue which normally receives no direct retinal projection appears to have acquired such a connection after early tectum lesions. Quantitative measures of the volume of terminal degeneration revealed by the silver stain in later cases (see below) show it to occupy a region as large as about 6% of the size of the dorsal nucleus of the lateral geniculate. According to the number of histological sections upon which it appears, it extends up to 1.5 mm in anteroposterior extent, but is always greatest at levels near the one illustrated in figure 6 [see also figure 1 of SCHNEIDER, 1970].

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Fig. 7. Retinofugal degeneration in the nucleus lateralis posterior, in photographs of Fink-Heimer stained sections, on the right side in case 19–1. The right superior colliculus was damaged at birth, and the left eye removed in the adult, 5 days before sacrifice. Upper: the clouds of silver grains indicating dense terminal degeneration stand out at low magnification. X119. Lower: at higher magnification, X595, the closely spaced silver deposits can be seen near and between cells. The forked blood vessel in the upper right can be found in the center of the low magnification view.

In its further continuation, the optic tract proceeds dorsally and caudally, over and through the pretectal region toward the superior colliculus. Anomalies in the pattern of terminal degeneration in the pretectal nucleus and in the overlying nucleus of the optic tract appear to be, at least in part, a consequence of direct damage to this region during the early postnatal surgery.

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On the normal, left side, the degenerating retinofugal axons can be followed into the superior colliculus, where they travel caudally in the stratum opticum and terminate most heavily in the overlying superficial gray layer. On the right side, these superficial layers of the colliculus appear to be absent, yet fibers of the right optic tract, somewhat reduced in quantity, can be followed to the remaining collicular tissue. They course mostly over the surface, with a heavy band of termination just below in what appears to correspond to the intermediate gray layer.

The resulting picture appears to represent another completely novel connection. However, one can question whether the anomaly involves only the location of the terminals, or the postsynaptic membranes as well. It seems possible that these terminals could be on certain dendrites of more deeply lying neurons, dendrites that might normally extend into the
superficial gray. It is also possible that a few cells normally found only in the superficial gray could in these cases come to lie below the optic tract fibers. These possibilities seem unlikely in cases of a very deep tectum lesion. In one such case, optic tract terminals are found in a narrow strip of tissue lying between the surface and the central gray substance; a few axons and terminals even appear to begin to follow the tectal commissure axons ventrolaterally.

Some of these axons reaching the diminished right colliculus, instead of terminating there, form a decussation never seen in the normal brain, crossing the midline of the tectal surface to terminate in the undamaged superior colliculus of the opposite side. (This anomalous decussation of optic tract fibers has not been found in cases of early bilateral lesions of the colliculus.) Further observations on this unusual phenomenon will be presented below.

The terminal degeneration in the nuclei of the accessory optic tract [for terminology, see HAYHOW et al., 1960] was similar on the two sides of the brain, except that the dorsal terminal nucleus was consistently larger (and filled with a correspondingly larger amount of terminal degeneration) on the side of the neonatal tectum damage (fig. 6).

The finding of an anomalous projection to the LP has recently been confirmed in another species: CASAGRANTE et al. [1972] have reported that an abnormal retinal projection to the pulvinar (equivalent to LP as used in this paper) develops also in tree shrews after early lesions of the superior colliculus.

What Determines the Location and Quantity of Anomalous Projections?

The anomalies summarized in figure 5 appear quite reliably after tectum lesions inflicted in the first few days of a Syrian hamster's life (at least

Fig. 8. Charts of Fink-Heimer stained sections from three adult hamster brains, conventions as in figure 6. The maximal extents of the adult lesions are shown in black on the full sections at the left. Fiber and terminal degeneration rostral to the lesions is illustrated in the half sections on the right. Numbers for each section indicate position in a
series spaced at 150 /\mu m. In case 20–6, the superficial layers of the right superior colliculus were destroyed at birth, and at 22 months a lesion was placed in the remaining tissue of the same colliculus with additional damage extending rostrally into the pretectal area. The degeneration pattern in this case (survival time 5 days) shows the pretectal projections, similar to those seen in case HPT-8; however, the superior colliculus projections to LP and LGv, as shown in case HSC-8, were found to be missing. In cases HPT-8 and HSC-8, the age at surgery was 16 weeks, and the survival time 4 days.

the first five, but effects of lesions in progressively older pups have not yet been investigated). What factors cause the anomalous projections to form in certain places and not in others? There are several major possibilities, not necessarily independent.

(1) Is the abnormal availability of terminal space necessary to induce 

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the anomalous ingrowth of terminating optic tract fibers? This possibility is suggested for the two diencephalic loci in the diagrams of figure 5. Both areas normally are recipients of a dense projection from the superior colliculus as shown by findings in 3 normal hamsters in which unilateral lesions were placed in the upper layers of the caudal two thirds of the superior colliculus. In each case, degenerating axons were traced rostrally to areas of dense terminal degeneration in the ipsilateral LP and ventral nucleus of the lateral geniculate body (LGv); in the latter, only the most external sublayer showed dense terminal degeneration. (Also, sparse terminal degeneration was found in the ventral part of the dorsal nucleus of the lateral geniculate, and a dense terminal field was found in the pretectal area. These rostral projections from the superior colliculus are similar to those described for several other mammals [ALTMAN and CARPENTER, 1961; MOREST, 1965; TARLOV and MOORE, 1966; MARTIN, 1969; ABPLANALP, 1970; HALL and EBNER, 1970].)

The areas in LP and LGv which normally receive projections from the superior colliculus include within them the regions in which increased or novel retinal-fiber termination appears in the cases of destruction of the upper layers of the colliculus in the neonate. Furthermore, these tectal projections are missing after such an early tectum lesion, according to one experiment illustrated in figure 8.

(2) Even if necessary, the availability of terminal space may not be sufficient. Axons unable to find normal terminal space further along their course, in the superior colliculus, may show a compensatory, collateral sprouting in areas, like LP, which they had originally traversed without termination. But is the sprouting specific to LP just because of vacated terminal space there?

(3) It could also be suggested that some similarity in chemical markers exists in various visual-system structures, such that axons unable to terminate normally may 'seek out' places of similar chemical specificity. Next I will describe an additional finding which sheds light on the latter possibility.

Optic Projections into the Auditory System in Case of Abnormal Availability of Terminal Space
If chemical specificity of visual structures were a dominant factor, one would never expect to find the axons of retinal ganglion cells terminating in the medial geniculate body (MGB), the major thalamic structure of the auditory system, even if terminal space were made available there. Yet, we have evidence that retinofugal axons can indeed be induced to terminate in the MGB, provided the normal innervation of this structure is drastically reduced or removed at birth. In many of the cases with neonatal ablation of the superficial optic layers of the superior colliculus, the early damage was found to include much or all of the brachium of the inferior colliculus (BIC), which normally courses over the midbrain surface below the far lateral edge of the superior colliculus and ends in the MGB. In cases where such additional damage was extensive, we have found evidence that retinofugal fibers leave the optic tract just behind the caudal tip of the lateral geniculate body, and enter the MGB, where they terminate.

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Fig. 10. Charts from coronal sections showing normal and anomalous projections to the caudal part of the dorsal thalamus of the adult hamster. A The lesion of the superior colliculus in case HSC-3 is shown in black, with heavy gliosis indicated by vertical striations. B Fiber and terminal degeneration in the caudal thalamus in case HSC-3, which was sacrificed 7 days after surgery; the terminals are seen in the caudal part of LP. C Lesion of the inferior colliculus in case HIC-1. D Degeneration pattern in the caudal thalamus in case HIC-1, which was sacrificed 5 days after surgery; the terminals define the medial geniculate body as the term is used in this paper. E The same level of the caudal thalamus in case HEE-8, in which the contralateral eye was removed 5 days before sacrifice; degenerating optic tract fibers are seen coursing over the medial geniculate body and caudal LP, just behind the lateral geniculate body. F The caudal thalamus at a level matching the others, in case 22–2, in which the right SC and right BIC were damaged at birth, and the left eye removed in adulthood, 5 days before sacrifice. The areas of terminal degeneration indicate anomalous projections to both LP and MGB.

The case with the most severe early damage to the auditory pathway showed the most extensive retinal projection to the medial geniculate. Figure 9 illustrates the histological evidence that on the right side of the midbrain the BIC is missing together with many deeper-lying axons. Figure 10 shows charts of the caudal thalamus where the optic tract courses over the MGB, just caudal to the lateral geniculate body. The superior...
Fig. 11. Photographs of the medial geniculate body in sections stained with the Fink-Heimer method. X 390. Upper: anomalous retinofugal degeneration in the section charted in figure 10F. Middle: degeneration, in a matching area, produced by an inferior colliculus lesion; a chart of the section is shown in figure 10D. Lower: degeneration in the same case of inferior colliculus lesion, in the same section but from the more dorsal area of denser degeneration.

c. Colliculus normally projects to the dorsal-most part of this region of the thalamus (fig. 10A B) which consists of a caudal extension of the LP. The inferior colliculus normally projects via the BIC to the ventral part (fig. 10C D). Optic tract axons normally show no termination here (fig. 10E) but in the case of early lesion of both the superior colliculus