cortical development in the rat reach adult values within the first month after birth, the maturational event leading to the apparent development of sulcal cortex regulation of ingestive behavior between 40 and 60 days is not apparent. However, one possibility is that the onset of puberty during this interval might trigger this development. This possibility is speculative at present. However, Kennedy (*10*) has argued that the ontogeny of ingestive behavior is never complete until puberty, and it is conceivable that gonadal hormones at puberty might alter the neural circuitry controlling ingestive behavior in much the same way that they alter the neural substrate of sexual behavior during the perinatal period (*11*). Gonadal hormones are involved in weight regulation of adult rats (*12*), and there is some evidence that medial hypothalamic control of feeding depends upon the attainment of puberty. Neither parasagittal hypothalamic knife cuts nor estradiol injections in female rats affect food intake before 40 to 50 days after birth (*13*), the age at which females attain sexual maturity.

Another possibility is that the present results depend on the prolonged development of the catecholaminergic "feeding system" in rats (*5, 14*). Although the cell bodies for this system are formed before or just after birth, the outgrowth of axons and the formation of terminals in the rostral diencephalon and telencephalon continues for several weeks after birth. Complete maturation of the cortical component of this system does not occur until 60 days or later (*15*). The sulcal cortex is ideally situated to modulate the activity of this system since it not only receives afferents from the dorsal noradrenergic pathway but also sends efferents which seem to overlap with many of the terminal projections of the dorsal and ventral adrenergic and the dopaminergic pathways (*2, 5*). Whether the projections from the sulcal cortex and from the catecholaminergic "feeding system" actually end on the same cells within the hypothalamus and caudoputamen is unknown.

Finally, the fact that our animals given early lesions showed no feeding impairment at any age indicates that sparing of function occurs in rats even after large frontal cortex lesions. This result conforms fairly well with those of Goldman (*16*) on sparing of function after early destruction of the frontal cortex. Goldman's data on rhesus monkeys show that substantial sparing occurs after early removal of the orbital region but that little or none is obtained when the dorsolateral region is removed at the same age. Since the behavioral effects of prefrontal lesions in rats and monkeys appear to be remarkably similar (*1, 2*), our results raise the possibility that the prefrontal cortex of the rat may provide a model for the study of the developmental phenomena that Goldman has described in the rhesus monkey (*16*).

BRYAN KOLB*
Department of Psychology, University of Western Ontario, London, Ontario N6A 3K7, Canada
ARTHUR J. NONNEMAN
Department of Psychology, University of Kentucky, Lexington 40506

References and Notes

1. This is the "sulcal cortex" described by Leonard (*2*). Because of the similarity in the pattern of afferents and efferents of this sulcal cortex in the rat and that of the orbital prefrontal cortex of the rhesus monkey, Leonard suggested that these regions may be homologous in the two species. Subsequent behavioral work has shown close parallels between the effects of sulcal cortex lesions in rats and orbital prefrontal cortex lesions in monkeys (*7*).
2. C. M. Leonard, *Brain Res.* **12**, 321 (1969).
3. P. Teitelbaum, *Prog. Physiol. Psychol.* **4**, 319 (1971); J. F. Marshall, J. S. Richardson, P. Teitelbaum, *J. Comp. Physiol. Psychol.* **87**, 808 (1974); H. P. Zeigler, *Science* **182**, 1155 (1973).
4. B. Kolb and A. J. Nonneman, *J. Comp. Physiol. Psychol.* **88**, 806 (1975).
5. U. Ungerstedt, *Acta Physiol. Scand. Suppl.* **367**, 95 (1971).
6. C. R. Almli and G. T. Golden, *Physiol. Behav.* **13**, 81 (1974).
7. A. J. Nonneman, J. Voigt, B. Kolb, *J. Comp. Physiol. Psychol.* **87**, 249 (1974); B. Kolb, A. J. Nonneman, R. K. Singh, *ibid.*, p. 772.
8. Kolb and Nonneman (*4*) reported that damage to the sulcal prefrontal cortex or adjacent motor cortex produced a serious disorganization of the motor patterns related to food intake. Once the animals began to eat and drink they were unable to do so efficiently because they exhibited disturbances of tongue and forelimb movements. In the present study these disturbances were only observed in those rats that were aphagic.
9. A. Epstein, in *Hunger: Basic Mechanisms and Clinical Implications*, D. Novin, W. Wyrwicka, G. Bray, Eds. (Raven, New York, 1976), pp. 193–205.
10. G. C. Kennedy, cited by A. Epstein, in *ibid.*, p. 193.
11. S. Levine and R. Mullins, *Science* **144**, 183 (1964); G. W. Harris and S. Levine, *J. Physiol. (London)* **181**, 379 (1965); A. A. Gerall and I. L. Ward, *J. Comp. Physiol. Psychol.* **62**, 370 (1966).
12. G. N. Wade, *Physiol. Behav.* **8**, 523 (1972); *J. Comp. Physiol. Psychol.* **88**, 183 (1975); _____ and I. Zucker, *ibid.* **72**, 328 (1970); I. Zucker, *Behav. Biol.* **7**, 527 (1972); S. Roberts, N. J. Kenney, D. C. Mook, *Horm. Behav.* **3**, 267 (1972); L. E. Harrell and S. Balagura, *J. Comp. Physiol. Psychol.* **88**, 194 (1975).
13. G. N. Wade and I. Zucker, *J. Comp. Physiol. Psychol.* **70**, 213 (1970); R. M. Gold and G. Kapatos, *ibid.* **88**, 202 (1975).
14. B. D. Berger, C. D. Wise, L. Stein, *Science* **172**, 281 (1971); M. J. Zigmond and E. M. Stricker, *ibid.* **177**, 1211 (1972); S. D. Glick, S. Greenstein, B. Zimmerberg, *ibid.*, p. 534; S. F. Leibowitz, in *Neurotransmitters*, I. J. Kopin, Ed. (Williams & Wilkins, Baltimore, 1972), pp. 327–358; S. Ritter, C. D. Wise, L. Stein, *J. Comp. Physiol. Psychol.* **88**, 778 (1975).
15. J. T. Coyle, Jr., in *The Neurosciences: Third Study Program*, F. O. Schmitt and F. G. Worden, Eds. (MIT Press, Cambridge, Mass., 1974), pp. 877–884.
16. P. Goldman, *Acta Neurobiol. Exp.* **32**, 495 (1972); in *Plasticity and Recovery of Function in the Central Nervous System*, D. G. Stein, J. J. Rosen, N. Butters, Eds. (Academic Press, New York, 1974), pp. 149–174.
17. We thank P. Cornwell, R. L. Isaacson, and J. F. Zolman for critical reading of an earlier version of this manuscript. Supported by NIMH small grant MN25649 to A.J.N. and by National Research Council of Canada grant AO-118 and Medical Research Council of Canada grant MA 4212 to C. H. Vanderwolf.
* Present address: Department of Psychology, Montreal Neurological Institute, Montreal, Quebec H3A 2B4, Canada.

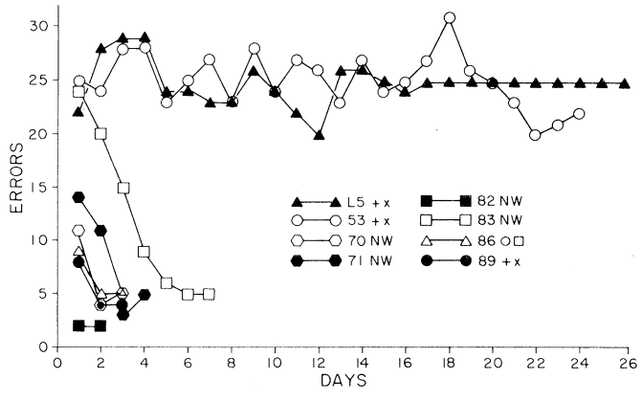
23 October 1975; revised 28 April 1976

Visual Discrimination Impaired by Cutting Temporal Lobe Connections

Abstract. *An attempt was made to transect the white matter that connects the anterior temporal lobe with dorsal and medial brain areas. Eight monkeys were trained preoperatively on a visual discrimination and tested postoperatively for retention and relearning of the task. They were also tested for Klüver-Bucy symptoms. The two animals that had complete lesions were unable to relearn the visual discrimination. It is suggested that human medial temporal lesions may produce their effects on learning and retention by damage to temporal white matter rather than by destruction of hippocampus.*

A thin band of white matter at the dorsal and medial aspect of the primate temporal lobe forms a bridge between the medullary core of the temporal lobe and the capsules of the brainstem. Within this white matter, the albal stalk of the temporal lobe, run nerve fibers that interconnect temporal lobe structures with subcortex and frontal cortex, and fibers of the temporal limb of the anterior commissure (*1*). In a previous experiment we cut the albal stalk of the temporal lobe by a surgical approach through the lateral sulcus (*2*). A severe loss in retention and relearning of a visual habit and Klüver-Bucy symptoms resulted. Unfortunately, in this procedure there was partial damage to the geniculostriate system which complicated our interpretation of the data. In the present experiment we cut the albal stalk and avoided the geniculostriate system by entering the anterior end of the temporal lobe in an approach much like that used for uncotomy and hippocampectomy (*3*). Our results suggest a new interpretation for the deficits produced by medial temporal lobe lesions in man.

Eight female adolescent *Macaca mulatta* weighing 3.7 to 6.3 kg were studied.



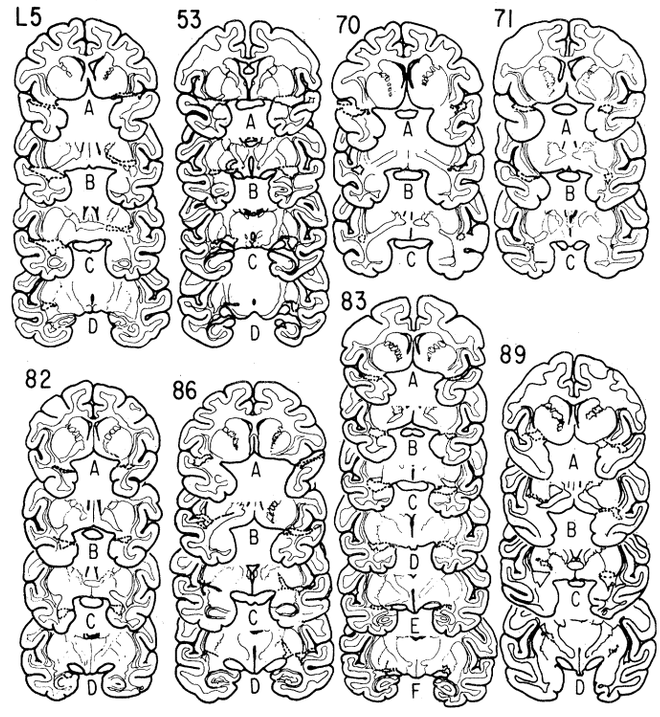


Fig. 1 (left). Postoperative daily error scores on the visual discrimination. Each line represents one animal, and the discrimination learned by each animal is indicated. Twenty-five errors is chance performance; five errors or less for two successive days is criterion performance. Fig. 2 (right). Diagrams of coronal sections through the lesion area. Dashed lines indicate the edges of the lesion.

Most of the animals had been used as normal controls or normal pilot animals in other experiments that involved behavioral tests similar to the tasks of this experiment.

All animals were trained preoperatively and retrained postoperatively on a pattern discrimination in a Wisconsin general testing apparatus. A stimulus tray containing two food wells was presented to the monkey. The wells were 30 cm apart and were covered with white plaques measuring 5 by 5 cm. The plaques were marked with figures cut from black adhesive material. These figures were either + versus ×, ○ versus □, or N versus W. The figures were all formed from lines 1 cm wide and were about 4 cm tall and wide. Since most of the monkeys had been used in previous experiments involving a similar task, they were each retrained with one of these stimuli pair that was different from those used in their original training. One of the stimuli of each pair was chosen as correct and was reinforced by placing a piece of monkey chow in the well beneath it. The side of the reinforced stimulus alternated according to a pseudorandom series. Fifty trials were given per day until 90 percent correct performance was achieved on two successive days.

The hypoemotionality and exploratory behavior that characterizes the Klüver-Bucy syndrome were measured with a rating scale (*2*, *4*). Details of these results are not presented here.

Surgery was performed under sterile conditions and with the aid of an operating microscope. All animals were given the same operation, and we intended to make the lesions bilaterally symmetrical and identical in all animals. A small craniotomy exposed the anterior end of the lateral fissure. A slit in the dura and a slight elevation of the inferior frontal area exposed the anterior surface of the temporal lobe near the lateral fissure. A small hole was made in the pia medial to the rhinal sulcus. A glass probe was inserted into the hole about 12 to 15 mm and moved in a medial and lateral direction. The intention was to make a horizontal slit dorsal to the anterior temporal lobe, above the amygdala and laterally up to the insula.

At the end of the experiment the brain of each animal was removed and photographed. Sections through the lesion and through the thalamus were prepared with fiber and cell stains.

The mean number of trials to criterion preoperatively on the visual discrimination was 262.5 and the range was 100 to 600. Most of the animals showed excellent postoperative retention of the task (Fig. 1). One animal (83) started at chance performance but returned to criterion performance in 5 days. However, two animals (L5 and 53) remained at chance performance throughout 26 and 24 days of postoperative testing on a visual discrimination that they had readily learned preoperatively (preoperative: 350 trials for L5 and 600 trials for 53). Postoperatively, animals 5, 53, and 83 showed the approach and oral exploration characteristic of the Klüver-Bucy syndrome (*5*). Attack and escape behavior were also reduced in these animals; however, these behaviors were reduced in some of the other animals as well.

Differences in the preoperative experience of these animals might account for some of the behavioral differences between them, but it would be difficult to attribute the postoperative inability of animals L5 and 53 to perform the visual discrimination to peculiarities of preoperative experience. It seems more likely that differences in their lesions would account for the severe deficit.

The lesions formed a horizontal slit from the anterior and dorsal temporal lobe extending posteriorly into the brain (Fig. 2). In two animals, internal blood vessels were broken; they died within a few hours of surgery and are not included in the eight animals of this report. Except for the lesions of these two animals, there was little damage beyond the narrow slit made directly by the probe.

There was much variability in the lesions. They differed in posterior extent and in some cases went above or below the intended horizontal plane. Animals in which the anterior part of the albal stalk was completely transected had the severe retention and relearning deficits, while animals in which part of the albal stalk was spared showed excellent retention. Animal 53 had the best bilateral transection of the albal stalk and performed randomly on the visual discrimination. Animal L5, which also performed randomly on the visual discrimination, had the anterior part of the albal

stalk transected bilaterally. However, on one side, as the lesion extended posteriorly, it also extended dorsally to lie directly along the lateral reaches of the anterior commissure, completely obliterating this structure on that side. The temporal branch of the anterior commissure contains noncommissural fibers from the middle and inferior temporal gyri to ipsilateral lenticular nuclei and thalamus (*1*).

Of the animals that did relearn the visual discrimination, animal 83 took the longest and had the most nearly complete lesion. The more posterior part of the albal stalk was spared on one side in this animal. The remaining animals all had obvious sparing of the albal stalk.

There was a slight invasion of the optic tract on one side in animal 53 (Fig. 2, 53D). However, this animal showed no lateral geniculate nucleus degeneration indicative of geniculostriate damage, and animal L5 had neither lateral geniculate degeneration nor optic tract damage. Animal 83, which showed little if any visual discrimination deficit, had bilateral damage to the optic tract (Fig. 2, 83F). On one side, the lesion extended back to the lateral geniculate nucleus and its cortical radiations. About 40 to 50 percent of the nucleus was degenerated. No other optic tract damage or lateral geniculate nucleus degeneration was found in any of the other animals.

There was bilateral damage to the amygdala in all animals, but amygdala destruction does not result in deficits on the visual tasks used here (*4*). In animal 53 there was unilateral invasion of the internal capsule (Fig. 2, 53C), and contralateral hemiparesis was noted. However, the animal readily used the unimpaired hand in the tasks of this experiment.

We intended to cut the band of white matter where the medullary core of the temporal lobe becomes continuous with the capsules of the brainstem. Previously we had approached this fiber tract through the lateral sulcus, damaging the superior temporal gyrus and interrupting part of the geniculostriate radiations (*2*); the animals all failed to relearn a preoperatively learned visual discrimination and displayed the Klüver-Bucy symptoms. We proposed that we had cut the output of the temporal lobe, thus interfering with the processing of visual information by this structure (*2*). In this experiment we produced the same deficit from an anterior approach that did not damage temporal neocortex or the visual radiations. However, the severe loss seen earlier (*2*) occurred in only two of the eight present animals. Histological analysis suggests that the deficit appeared only if the temporal albal stalk was transected.

From these results and those obtained earlier (*2*), we conclude that the integrity of the temporal albal stalk is critical for normal learning and retention of visual discriminations. Electrical stimulation in the albal stalk and along the temporal limb of the anterior commissure which passes through the albal stalk severely disrupts performance of visual discriminations (*6*). We cannot be certain which pathways within the albal stalk are responsible for the observed deficits, but the massive efferents of the middle and inferior temporal gyri that terminate in the lenticular nuclei and thalamus should be seriously considered (*1*). They reach these subcortical nuclei by traversing the temporal albal stalk, some of them among the fibers of the temporal limb of the anterior commissure and others in the ansa peduncularis. Destruction of the middle and inferior temporal gyri produces severe deficits in the learning and retention of visual habits (*7*).

The fiber pathways that enter and exit the temporal lobe posteriorly were not injured by the lesions of this experiment. These fibers carry visual information from occipital visual areas; transecting them (*2*) or interrupting this occipitotemporal route by disconnection (*8*) produces visual discrimination deficits and Klüver-Bucy symptoms. It was suggested (*2*) that these lesions produce their effects by blocking the visual input to the temporal cortex. It is likely that the lesions described here produce their effects by blocking the output of the temporal cortex. These lesions were placed anteriorly to avoid the visual radiations; as a result, the temporal efferents to the posterior thalamus and midbrain were also at least partially spared (*1*). The temporal cortex projections to the medial thalamus and lenticular nuclei were completely transected in the affected animals and are thus implicated in the visual functions of the temporal lobe.

The white matter cut in this experiment stands directly in the path of surgical approach to the hippocampus in man (*3*). Many of the temporal cortex efferents of the human brain, such as those joining the ansa peduncularis and the temporal limb of the anterior commissure, are particularly vulnerable to this anterior approach (*9*). In humans, removal of the medial temporal lobe produces a retrograde amnesia affecting several months before the surgery and a permanent prograde amnesia (*3*). Although these results are generally attributed to hippocampal damage, hippocampal lesions in animals have not consistently reproduced that deficit (*10*). If our animals were trained within the period of retrograde amnesia and tested during the period of prograde amnesia, the results are comparable to the human deficits that are produced by lesions of the medial temporal lobe. Therefore, it is possible that the human deficit was not produced by destruction of hippocampus but by destruction of the interconnection of temporal neocortex with subcortical areas. This possibility is consistent with evidence that removal of the medial temporal lobe in man produces effects much like those of temporal neocortex lesions in monkeys (*11*).

JAMES A. HOREL
Department of Anatomy,
State University of New York,
Upstate Medical Center,
Syracuse 13210

LOUIS J. MISANTONE
Department of Anatomy,
Hahnemann Medical College,
Philadelphia, Pennsylvania 19102

References and Notes

1. C. A. Fox, R. R. Fisher, S. J. Delsalva, *J. Comp. Neurol.* **89**, 245 (1948); S. M. Zeki, *ibid.* **148**, 167 (1973); D. G. Whitlock and W. J. H. Nauta, *ibid.* **106**, 183 (1956); W. J. H. Nauta, *Brain* **85**, 505 (1962).
2. J. A. Horel and L. J. Misantone, *Exp. Neurol.* **42**, 101 (1974).
3. W. B. Scoville and B. Milner, *J. Neurol. Neurosurg. Psychiatry* **20**, 11 (1957).
4. J. A. Horel, E. G. Keating, L. J. Misantone, *Brain Res.* **94**, 347 (1975).
5. H. Klüver and P. Bucy, *Arch. Neurol. Psychiatry* **42**, 979 (1939).
6. R. W. Doty and W. H. Overman, in *Lateralization in the Nervous System* (Academic Press, New York, in press).
7. M. Mishkin and K. H. Pribram, *J. Comp. Physiol. Psychol.* **47**, 14 (1954).
8. M. Mishkin, in *Frontiers in Physiological Psychology*, R. W. Russell, Ed. (Academic Press, New York, 1966), pp. 93–119; J. A. Horel and E. G. Keating, *Brain Res.* **16**, 281 (1969).
9. J. Klinger and P. Gloor, *J. Comp. Neurol.* **115**, 333 (1960).
10. R. J. Douglas, *Psychol. Bull.* **67**, 416 (1967); R. L. Isaacson, *Neuropsychologia* **10**, 47 (1972); D. H. Kimble, *Psychol. Bull.* **70**, 285 (1968).
11. K. L. Chow and J. Survis, *A.M.A. Arch. Neurol. Psychiatry* **79**, 640 (1958); S. Iverson and L. Weiskrantz, *Nature (London)* **201**, 740 (1964), P. Dean and L. Weiskrantz, *Neuropsychologia* **12**, 299 (1974).
12. Supported in part by NIH grant NS08915. We thank R. A. Gay and E. Weber for technical assistance.

14 January 1976; revised 14 May 1976