The **limbic system** of the brain generally includes the great limbic lobe of Broca and its cortical substations, which make a border (**limbus** in Latin) around the corpus callosum and diencephalon. The border is best seen on a midsagittal section of the brain (Fig. 1) as a C-shaped pattern that includes the cingulate gyrus, the gyrus fimbriatus, the parahippocampal gyrus, and the rest of the hippocampal formation (dentate gyrus and hippocampus or cornu Ammonis). The hilum of the C is closed anteriorly (rostrally) by the rhinencephalon (olfactory nerves, bulb, tract, and basal olfactory cortex), with the parolfactory gyrus bordering the lamina terminalis at the rostral end of the third ventricle and the septal nucleus and amygdaloid nucleus hidden beneath the adjacent tissues.

This pattern is dictated by the embryologic development (see Fig. 1), which centers on the commissural plate, where the hippocampal and anterior commissures and corpus callosum begin. The trapping of the precommissural and supracallosal fibers of the fornix by the developing corpus callosum contributes to the formation of the indusium griseum. The corpus callosum markedly expands and the hippocampal commissure migrates posteriorly, ending as the psalterium under the splenium of the corpus callosum. The hippocampus itself, of course, migrates back, down, and around in a spiral, which the fornix recapitulates. Between the corpus callosum and the fornix, the septum pellucidum is stretched out, and a cavity develops in its center as a process of necrosis, necrobiosis, or apoptosis, this last being the current terminology emphasizing the process of programmed cell death, a common feature of normal development of many organs.

The constituents of the limbic system vary depending on whose book one reads. The disagreement lies on the extent of inclusion or exclusion of cellular substations of the great limbic lobe.

The simplest approach is all-inclusive:

1. The olfactory nerves, bulb, tract, and basal olfactory cortex as the true rhinencephalon or olfactory brain.
2. The parolfactory gyrus, cingulate gyrus, and underlying white matter (the *cingulum*, Latin for belt), gyrus fimbriatus, and parahippocampal gyrus as the original great limbic lobe of Broca.
3. The dentate gyrus, hippocampus, fornix, *indusium griseum* (Latin for gray shield) over the corpus callosum, mammillary body, mammillothalamic tract of Vico.
Figure 1. Development of the limbic system illustrated in midsagittal sections of a fetus of 3 months' gestation (A) and an infant at term (B) and in a horizontal dissection (C).

Figure 2. Coronal section of an adult brain (A) with a seahorse superimposed (B) with its tail curved backward rather than forward, which would be normal.
d'Azir, anterior thalamus, and thalamo-cingulate radiation as the components connecting the archipallium and paleopallium into the limbic circuit of Papez.  

Thus, a double spiral of connections is formed by connecting no. 3 to no. 2 with enough neurons in a relatively closed loop that can permit continuous cycling of epileptic activity without necessarily spreading to other

Figure 3. The cornu Ammonis, Ammon's horn. On the left side, the hippocampal formation and fornix (ending at the level of the anterior commissura) is shown. On the right side, the ram's horn is reversed to mimic the expanded uncus on the left.

Figure 4. The pes anserinus or "goose's foot," the fornix, and hippocampal formation as seen from the right side and above.
cortical and subcortical structures (even though there are numerous such connections available to the surrounding associated cortex and brain stem). This double-spiral, self-stimulating circuit may be important also in normal functions, especially in the conversion of immediate sensations (the present) into recent memory and then into the archives of old memory.

The history of the development of the nomenclature is interesting in its complex mixture of imagination of everything from silk-worms and dolphins to hippopotami, oddly enough omitting geese,19 based on what must have been dissections of poorly preserved gross tissue. A simplistic view rewriting history is as follows:

The hippocampus (Greek, hippos = "horse" + campos = "a mythical sea monster," although some would translate it as a caterpillar) was represented in classic illustrations as the front half of a horse and the back half of a fish or

Figure 5. A. Herpes simplex encephalitis. A basal view of the brain shows swelling with petechial hemorrhages over the medial temporal lobes. B. A coronal section of the same cerebrum, showing hemorrhagic necrosis of the temporal lobes and hypothalamus.
dolphin drawing the chariots of Neptune (the god for the preservation of the streams and springs), Poseidon (the god of the seas and the tamer of horses, typically portrayed with a dolphin and trident), and Tritons (portrayed with a fish tail and conch shell trumpet to calm the waves of the seas). The modern representation is a seahorse, whose outline can best be seen on a coronal section of the medial temporal lobe (Fig. 2): The fimbria of the fornix and the medial part of the dentate gyrus are the seahorse’s snout, the hippocampus its head, the subiculum and parahippocampal gyrus its abdomen (the hippocampal fissure separating the head and snout from the belly), and the fusiform or temporo-occipital gyrus its tail (inappropriately curled behind rather than in front, as would be natural for the real seahorse). Because current terminology restricts the hippocampus to the single layer of pyrami-

Figure 6. Basal view of the brain (A) and a coronal section of the cerebrum (B) of a 37-year-old woman who had a history of herpes simplex encephalitis with decompressive left craniectomy at age 17 years. She survived with mental retardation and seizure disorder. She was found dead at home presumably of an epileptic seizure. The specimen is shown from behind.
Figure 7. Falx and tentorium after removal of the left cerebral hemisphere and hemisection of the midbrain. (From Lemire et al [Illustrator, Phyllis Woods]: Normal and Abnormal Development of the Human Nervous System. Hagerstown, MD, Harper & Row, 1975, p 421; with permission.)

Figure 8. Coronal section of the cerebrum at the level of the pituitary stalk, showing acute contusions in the uncus, amygdaloid nucleus, hippocampal formation and hypothalamus. This 17-year-old woman died in coma 10 days after a motor vehicle accident. She also had multiple basal skull fractures and a right posterior frontal fracture.
dial cells, the term *hippocampal formation* conforms better with the analogy with the seahorse.

The *cornu Ammonis* (Latin, "horn of Ammon") refers to the Egyptian mythology in which Ammon (Amen or Amun) was the sun god, the father of gods, the god of reproductive forces. He was depicted as having a ram's head with spirally curved horns (alternatively, and irrelevant to the present discussion, as a man
Figure 11. Coronal section of the cerebral hemispheres at the level of the red nucleus, showing marked diffuse edema with axial herniation in a 15-year-old girl who developed disseminated intravascular coagulopathy and severe anoxia following a laparotomy for splenic rupture.

with a sunlike disk and two ostrich feathers on top of his head. If the brain is dissected to show the hippocampal formation and fornix as a spiral, one can see the ram's horn from almost any perspective (Fig. 3): The fornices (close together at the level of the anterior commissure) curve back, down, and around the diencephalon and become the hippocampal

Figure 12. Ventral view of the brainstem, cerebellum, and upper cervical spinal cord showing upward herniation of the brainstem and cerebellum in a 6-year-old boy who had a Chiari type II malformation and who died of intraventricular hemorrhage following a shunt revision.
formation in the medial temporal lobe. Inappropriately the neural structures enlarge, whereas the ram’s horn naturally is broad-based at its origin from the skull and tapers to a point as it spirals back, down, and around the ram’s head. Current terminology restricts the cornu Ammonis to the same single layer of pyramidal cells as the hippocampus and eliminates the fornix, which is essential to complete the analogy with the ram’s horn.

The pes anserinus (Latin for “foot of a goose”) is best seen after the temporal horn has been opened to allow the anterior hippocampus to be viewed from above (Fig. 4). The undulations of the anterior hippocampus resemble reasonably well the webbed toes of a goose without, of course, the sharp toes themselves.

Despite the fact that these structures are closely related to each other anatomically and physiologically, there are few conditions in which the whole limbic system is more or less selectively involved by a pathologic process. The best examples are paraneoplastic limbic encephalitis and acute herpes simplex encephalitis (HSE); hippocampal sclerosis, arhinencephalia, and variations on the theme of
Figure 14. Horizontal section of the midbrain showing anteroposterior elongation of the midbrain and cystic necrosis with hemorrhage of the right cerebral peduncle (Kernohan’s notch) secondary to transtentorial herniation of the left parahippocampal gyrus in a 50-year-old man who died 1 week after the onset of an acute ischemic infarction of the left cerebral hemisphere.

Figure 15. Coronal section of the cerebral hemispheres at the level of the anterior commissure, showing subfalcine herniation of the right cingulate gyrus. This 70-year-old man was found dead with a large right subdural hematoma.
holoprosencephaly are other acceptable examples.

In other instances, the diseases typically involve many parts of the brain, often diffusely, and some portions of the limbic system can also be included, but the limbic system is not the primary target. Many types of viral encephalitis, especially rabies, and some cases of cerebral contusion belong to this group. Finally, there is a group of conditions in which some portions of the limbic system are involved by the disease processes not selectively but merely by chance, such as in cases of primary or secondary tumors and in cases of infarction or hemorrhage.

**DISEASES IN WHICH THE WHOLE LIMBIC SYSTEM IS MORE OR LESS SELECTIVELY INVOLVED**

**Limbic Encephalitis**

Several types of neurologic disorders are associated with remote malignancy in the absence of specific central nervous system

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**Figure 16.** Basal (A) and midsagittal views (B) of the brain and coronal sections of the right cerebral hemisphere (C) showing disproportionately large temporal lobes with abnormal sulcation in a fetus of 19 weeks' gestational age with thalamic skeletal dysplasia.
metastasis, opportunistic infection, or other complications of therapies. These conditions have been collectively called remote effects of malignancy or paraneoplastic syndromes and include carcinomatous neuropathy\textsuperscript{11}; neuromyopathy\textsuperscript{14}; Guillain-Barré syndrome\textsuperscript{16}; subacute cerebellar degeneration\textsuperscript{2}; necrotizing myelopathy\textsuperscript{21}; and encephalomyelitis involving the ce-
Encephalitis in which the lesions are most severe in the hippocampal formation, amygdaloid nucleus, cingulate gyrus, insula, and orbital cortex has been labeled *limbic encephalitis*. The condition is characterized by a subacute onset of memory loss, dementia, involuntary movements, and ataxia. The syndrome has been reported with carcinomas of the lung, uterus, breast, and other sites. The prognosis is usually poor. The brain of such a patient shows no gross abnormality, but in some cases congestion, dark discoloration, or focal necrosis can be found in the hippocampus. The neuropathologic changes are usually microscopic, showing inflammatory and degenerative changes in the cerebral cortex, mainly in the limbic lobe. The cause of this condition is unknown. A viral cause has been suspected but not proven. Metabolic and immunologic causes have also been considered.

**Herpes Simplex Encephalitis**

HSE involves the limbic system almost as selectively as in paraneoplastic limbic encephalitis, but HSE is an acute illness caused by herpes simplex virus (HSV) type 1.

The pathology of HSE is quite characteristic and almost diagnosable from the macroscopic examination of the brain: swelling, necrosis, and petechial hemorrhages involving the limbic lobe bilaterally. The anterior medial temporal lobe is most severely involved bilaterally (Fig. 5), not necessarily symmetrically, and the insular cortex, orbital cortex, and cingulate gyrus may also be involved (Fig. 5). Microscopically the changes are those of acute necrotizing meningoencephalitis with variable degrees of reactive changes, such as lymphohagocytic reactions and capillary proliferation. Intranuclear inclusions can be found but not necessarily easily, especially adjacent to areas showing extensive rarefaction and necrosis. Immunocytochemistry with HSV antibodies may be required to establish the diagnosis. In patients who survive for weeks or months, the brain shows cortical atrophy or postnecrotic cyst formation in the same distribution (Fig. 6), but intranuclear viral inclusions and HSV antigens are not likely to be demonstrated.

Most other types of viral encephalomyelitis show diffuse involvement of the central nervous system in which the limbic system is obviously included. Historically the pathognomonic intracytoplasmic inclusion body known as the Negri body has been said to be most frequently found in large pyramidal neurons of the hippocampus and Purkinje cells of the cerebellum in rabies encephalitis. This is probably an overemphasis, however, on the hippocampal and cerebellar lesions. The disease process in rabies encephalitis is diffuse, and Negri bodies can be found in many other parts of the central nervous system.

![Figure 19. Basal view of the brain showing a large but incidental arachnoid cyst in the region of the anterior peritoneal substance displacing the left optic nerve medially and the uncus and medial temporal lobe laterally in a 21-year-old man who died of multiple stab wounds.](image-url)
Cerebral Contusions Caused by the Falx Cerebri and Incisura Tentorii

The intracranial contents are incompletely separated into right and left supratentorial cavities and supratentorial and subtentorial cavities by the falx cerebri and the tentorium cerebelli (Fig. 7). The openings created by these two tough fibrous membranes are in close approximation to major portions of the great limbic lobe of Broca. Excessive motion of the softer cerebral tissue against the relatively fixed tough falcial and tentorial edges in cases of head injury results in contusions of the adjacent structures: uncus, hippocampus, parahippocampal gyrus (Fig. 8), cingulate gyrus and corpus callosum (Fig. 9), and rostral brain stem (Fig. 10).

Internal Herniations Associated with Masses and Edema

When the volume of the contents of one or more chambers separated by the falx or tentorium increases because of masses such as tumor, hematoma, abscess, or edema, the portions of the brain (uncus, hippocampal formation, and cingulate gyrus) most closely situated with the opening of the dura are pushed through the opening, resulting in transtentorial or subfalcine herniations. If the former herniation is unilateral, it is called uncal or parahippocampal gyrus herniation; if bilateral, axial herniation.

Transtentorial Axial Herniation

Symmetrically increased bulk of tissue in the supratentorial cavity displaces the diencephalon down through the tentorial notch into the subtentorial cavity (Fig. 11). This is known as axial herniation. The reverse or upward herniation may occur when there is a mass in the posterior fossa or when the pressure in the supratentorial cavity is relieved by ventriculostomy (Fig. 12). The best way to evaluate axial herniation is to make a coronal section of the cerebrum at the level of the red nuclei. Normally, all or most of the red nucleus is situated above the line drawn over the dorsal aspect between the two hippocampi (Fig. 13). The caudal displacement of the center of the red nucleus below this line is an indication of axial herniation (Fig. 13). The rest of the mesencephalon and part of the diencephalon, including the posterior third ventricle, are similarly displaced caudally so that the midbrain

Figure 20. Basal view of the frontal half of the brain with paired cerebral hemispheres showing absence of the olfactory bulbs and tracts in a 7-day-old boy who was born after 31 weeks' gestation and died of respiratory distress syndrome.
Figure 21. Unextended olfactory bulb represented by a nodule in the left olfactory trigon in a 2-day-old infant who died of congenital hypoplasia of left heart. A similar but smaller nodule was also found on the right side.

Figure 22. Ventral (A), and dorsal (B) views of holoprosencephaly in a fetus estimated to be 16.5 weeks' gestational age.
is deformed, compressed bilaterally, and elongated anteroposteriorly and shows congestion, ischemic necrosis, and often hemorrhages in the tegmentum. The complications of reverse axial herniation are less well defined, but medial thalamic hemorrhage has been noted in some cases.

**Transtentorial Uncal Herniation**

Unilateral transtentorial herniation is accompanied by asymmetric mesencephalic deformity, hemorrhagic necrosis of the mesencephalic and rostral pontine tegmentum, and Kernohan’s notch in the contralateral cerebral peduncle (Fig. 14). This last lesion may account for an asymmetric quadriplegia (if the primary lesion had already produced a contralateral hemiparesis) or an ipsilateral hemiplegia (if the primary lesion, such as a subdural hematoma, had not involved the brain directly enough to have produced the expected contralateral hemiparesis).

**Subfalcial Herniation**

Unilateral increase in the bulk of the tissue in one of the supratentorial cavities results in midline shift, asymmetry of the lateral ventricles (ipsilateral compression and contralateral dilatation), and herniation of the inferior portion of the cingulate gyrus and corpus callosum to the opposite side (Fig. 15). Compression of the anterior cerebral arteries by the edge of the falx may result in ischemic or hemorrhagic necrosis of the cingulate gyrus and corpus callosum.

**DISEASES IN WHICH PART OR PARTS OF THE LIMBIC SYSTEM ARE PREDOMINANTLY INVOLVED**

**Developmental Anomalies**

Several types of anomalies, including arhinencephalia, holoprosencephaly, agenesis of the corpus callosum, septo-optic dysplasia, and cyst of the cavum septi pellucidi or vsrgae, specifically involve a part of the limbic system. In the brain of a thanatophoric dwarf, the inferomedial temporal lobes may be hyperplastic and dysplastic, including the hippocampus (Fig. 16). Other nonspecific types of cortical...
dysplasia (Fig. 17), cerebral cyst (Fig. 18) and arachnoid cyst (Fig. 19) may involve any part of the central nervous system, including parts of the limbic system.

**Arrhinencephalia and Holoprosencephaly**

The term *arrhinencephalia* was originally used in 1882 by Kundrat and included various types of craniofacial malformation, which shared one common finding: agenesis of the olfactory bulbs and tracts. Observations by many later investigators have shown that the series presented by Kundrat under *arrhinencephalia* was indeed heterogeneous with at least two major types of developmental anomalies of the central nervous system characterized by the absence of olfactory bulbs and tracts: (1) those without other gross abnormality of the cerebrum and (2) those with a major anomaly of the telencephalic development. Several alternative terms, including *holotelencephaly*, *holoprosencephaly*, and *prosencephaly* have been proposed to separate the latter group from the first group. The term *holoprosencephaly* has become most popular in the literature. The authors also believe that the two groups should not be mixed up because their development is fundamentally different in time and location.

**Arrhinencephalia.** Gross agenesis of the olfactory tracts and bulbs in paired cerebral hemispheres is the pertinent feature of this condition. The agenesis is usually bilateral (Fig. 20) but can be unilateral. The cerebrum is, by definition, always a pair of cerebral hemispheres.

The authors have examined 116 cases from 1960 to 1996. These can be divided into three major groups: (1) agenesis of the olfactory tracts and bulbs in an otherwise grossly normal brain, 85 cases; (2) agenesis of the olfactory tracts and bulbs associated with well-established types of central nervous system malformation (e.g., Arnold-Chiari malformation, Dandy-Walker malformation, agenesis of the corpus callosum), 14 cases; and (3) agenesis of the olfactory tracts and bulbs associated with various types of anomalies, which may be related to the loss of the olfactory tracts, 17 cases. Group 3 is heterogeneous and includes cases of congenital rubella encephalopathy, cytomegalovirus encephalitis, hydrocephalus, and subependymal cysts. In some of these cases, the absence of olfactory tracts and bulbs may be acquired, secondary to destructive processes.
The majority of the 116 cases were newborns and infants. Only 21 cases were over 1 year old, including 11 adults. The regions of the olfactory trigone were carefully studied with a dissecting microscope grossly or by serial sections microscopically in 45 cases. In 35 of these, gross or microscopic olfactory bulbs were found unextended in the area of the trigone (Fig. 21). Based on these observations, the authors have concluded that a failure of the olfactory bulbs to extend anteriorly to their prospective sites over the cribriform plate is the pathogenesis for arrhinencephalia in the majority of the cases.

Holoprosencephaly. In contrast to arrhinencephalia, in which the cerebrum is paired, the single most essential feature in holoprosencephaly is a single, median telencephalon that is located at the rostral end of the central nervous system. The frontal pole is a single flat or cone-shaped structure without a midsagittal fissure (Fig. 22), and the olfactory bulbs and tracts in the great majority of cases are also absent. The single rostral telencephalon covers the anterior half or two thirds or more of a single median ventricular cavity that extends posteriorly into a large dorsal cyst, which is covered by thin neural and leptomeningeal membranes. The neural membrane most likely represents a blown-out velum transversum that failed to invaginate rostrally to form the roof of the third ventricle. This membrane is absent because the telencephalic vesicles do not arise from the sides of the rostral neural tube, where the two foramina of Monro normally develop to connect the two lateral ventricles and the median third ventricle but rather from the front of the third ventricle. The two cerebral vesicles fuse anteriorly over a single ventricle, the combined lateral and third ventricles. A horseshoe-shaped hippocampal formation forms the posterior roof of this single median ventricle and serves as an archway between the single median ventricle and the dorsal cyst (Fig. 23). Depending on the extent of the development of the posterior telencephalon, holoprosencephaly can be divided into a complete form, in which there is no sagittal fissure at all (20 of the authors’ cases), and an incomplete form, in which the posterior telencephalon is separated into a pair by a partial sagittal fissure posteriorly (35 cases). The lamina terminalis is where the single rostral telencephalic vesicle originated and, of course, is absent, no longer visible as its usual thin membrane. The striatum is represented by a

Figure 25. Incidental cavum septi pellucidi in a 41-year-old man who died of pulmonary embolism 1 month after a head injury.
mass with various degrees of fusion in the anterior floor of the single median ventricle (Fig. 24).

This condition was originally named ar-rhinencephalia by Kundrat because of the agenesis of olfactory tracts and bulbs, but the main bulk of the rhinencephalon, including the hippocampal formation, is present even though it is deformed and displaced and even though the indusium griseum and septal nu-

Figure 27. Cyst of the cavum septi pellucidi in a 10-year-old girl who developed hydrocephalus that was not relieved by a partial resection of an astrocytoma in the region of the head of the right caudate nucleus. The cyst almost filled the lateral ventricles on both sides. The specimen is viewed from behind.
nuclei cannot be recognized as separate structures in this condition. The amygdaloid nucleus is difficult to identify in immature brains of fetuses and newborns but can be found in the brains of older patients.

Cyst of the Cavum Septi Pellucidi or Vergae. A cavity (or cavities) between the two leaves of the septum pellucidum is a clinically insignificant structure found in about 15% of adults (Fig. 25). The *cavum septi pellucidi* (Latin, "cavity of the septum pellucidum") and the *cavum vergae* (Latin, "cavity of Verga") are essentially the same structure, one being located anterior and the other posterior to an arbitrary region (e.g., the foramen of Monro). The two are usually continuous without a clear boundary, but rarely they may be separated by the encroaching columns of the fornix in the midportion of the cavum. The cavum is constantly present in fetuses and begins to close from posterior to anterior at about 6 months’ gestation and continues until about 6 months after

**Figure 28.** A, Left lateral view of the brain showing atrophy of the anterior temporal lobe in a 93-year-old woman with Pick’s disease. B, Coronal section of the cerebral hemispheres showing atrophy of the hippocampus and inferior temporal cortex typical of Pick’s disease.
term birth. It is found in 97% of term infant brains (Fig. 26) and in 15% of the brains of infants 3 to 6 months of age. The cavum occasionally expands to obliterate not only the third, but also the lateral ventricles in a variety of diseases, including bacterial meningitis, neoplasms, or trauma, to cause obstructing hydrocephalus. Excessive production and accumulation of fluid in the cavum probably occur in these cases (Fig. 27) secondary to the primary pathologic condition. An expanded cyst of the cavum septi pellucidi without any associated pathologic conditions is rare but has also been reported.

**Degenerative and Metabolic Diseases**

**Pick's and Alzheimer's Diseases**

Both Pick's and Alzheimer's diseases share the clinical presentation of progressive dementia beginning in later life. In Alzheimer's disease, the brain shows diffuse atrophy, whereas in Pick's disease, the brain shows lobar or circumscribed atrophy. Pick's disease is much less common than Alzheimer's disease, but familial occurrence is more common in Pick's disease. A combination of both Pick's and Alzheimer's diseases is frequently seen in the same patient because both conditions affect the same age population.

In Pick's disease, the atrophy of the cerebral cortex is most marked in either frontal or temporal lobe. Constantinidis and colleagues divided Pick's disease into three forms: group A with ballooned neurons (Pick cells) and argyrophilic inclusions (Pick bodies), with most marked atrophy in the limbic system (especially the dentate gyrus and hippocampus) and temporoparietal cortex and with prominent moria, bipolar humor, and fixation amnesia; group B with Pick cells but no Pick bodies, with most marked atrophy in the superior and middle frontal gyri, pallidum, and substantia nigra and with prominent apragmatism and depressive humor; and group C with neither Pick bodies nor Pick cells and with marked atrophy in either frontal or temporal lobes.

In the authors' experiences, atrophy in Pick's disease is most marked in the middle and inferior temporal gyri (Fig. 28). On coronal sections, the cerebral hemispheres show marked thinning of the inferior temporal cortex, often including the hippocampus with concomitant dilatation of the temporal horn (Fig. 28). In contrast, atrophy in Alzheimer's disease is more diffuse (Fig. 29). Senile plaques and neurofibrillary tangles in neurons in Alzheimer's disease are diffuse or patchy in the cerebral cortex and basal ganglia and occasionally in the cerebellar cortex but most severe in the hippocampus and amygdaloid nucleus. Hence, atrophy of the hippocampus bilaterally can be grossly apparent in patients with advanced Alzheimer's disease (Fig. 30) or with a long history of the disease, but in contrast to the brain in Pick's disease, atrophy of the anteroinferior temporal cortex is not striking. Secondary degeneration with atrophy of the columns of the fornix can be seen in

![Figure 29. Dorsal view of a brain showing diffuse cortical atrophy in a 71-year-old woman with Alzheimer's disease.](https://example.com/image.png)
long-standing cases of hippocampal atrophy (Fig. 31).

Despite some similarities and dissimilarities, the final diagnosis and the differential diagnosis of Alzheimer’s and Pick’s diseases can only be made microscopically by demonstrating argyrophilic neuronal inclusions (Pick’s bodies) in the temporal lobe in Pick’s disease and sufficient numbers of senile plaques and neurofibrillary tangles in Alzheimer’s disease. In both diseases, the hippocampal formation is most severely involved.

Sommer’s Sector Lesion, Hippocampal Sclerosis, and Inclisural Sclerosis

The little seahorse-shaped structure of the hippocampus has excited more academic attention and controversy than any other structure in the brain. The anatomy is grossly unique, easily recognizable by even inexperienced eyes, and discernible in most species of mammals. Its fanciful terminology has been discussed earlier (see Figs. 2–4), but its diseases are even more interesting.

The association of seizures, especially of the partial complex type (also known as psycho-motor, limbic, or temporal lobe seizures), and hippocampal sclerosis had long been known and demonstrated in the gross brain as early as 1825 by Bouchet and Cazauvielle,1 but it was not until 1880 that Sommer38 first described the histologic lesion in the hippocampus in autopsies of epileptics and some nonepileptics. He showed maximal neuronal degeneration in the band of pyramidal cells adjacent to the base of the anterior temporal lobe. In 1899, Bratz3 also found the hippocampal lesion in half of epileptic brains and named the band Sommer’s sector.

The terminology mixing normal and abnormal has become increasingly confusing. The numeric similarities have contributed further to the confusion and have obscured the major technologic and conceptual differences underlying not only the normal functional connectivities but also the abnormal destructive lesions and reparative reactions.

To begin with, the Nissl stain, which is the basis of the cytoarchitectonic analysis of Rose3 (with its 5 fields, H1–5, representing probably the ultimate extension of the alpha-numeric divisions of the cerebral cortex by Brodmann48 and von Economo46), reveals only the nucleus
Figure 31. Coronal section of the cerebral hemispheres at the level of the red nuclei showing marked atrophy of the hippocampus and fornix as part of a more diffuse cortical atrophy bilaterally in a 75-year-old man with Alzheimer’s disease (Courtesy of David Nocbill, MD, Seattle, WA).

and adjacent Nissl bodies and soma of the neuron, and nothing of the axon and dendrites, which underlie the connections of neurons. For pathologists, the combination of the Nissl stain with a myelin sheath stain (e.g., luxol fast blue) represents a slight step forward but logically requires the use of Rose’s terminology for the definition of what can only be seen as the destructive component of the lesion.

By contrast, the Golgi technique, which is the basis of the connectivity analysis of Lorente de No³⁰ (with its four fields, CA1–4, each with

Figure 32. Division of the hippocampus by the scheme of Lorente de No into four segments, CA1–CA4 (solid lines), and by the scheme of Rose into 5 fields, H1–H5 (dotted lines).
several major subdivisions representing probably the ultimate, although "still incomplete," extension of the analysis of Cajal's of the connections within the nervous system) reveals the whole morphologic pattern of only a few individual neurons in any one section. Its innate capriciousness requires many repetitions to reveal the many normal components

Figure 33. Low magnification of the hippocampus showing atrophy and neuronal loss in H1 (CA1) segment in a 25-month-old male epileptic patient (LFB-Nissl stain, original magnification × 16).

Figure 34. Low magnification of the hippocampus showing marked atrophy and neuronal loss in H1 (CA1), HS-5 (CA3-4) and dentate gyrus in a 28-year-old woman who had a history of seizures since age 1 year and mental retardation (LFB-Nissl stain, original magnification × 16).
Figure 35. Coronal section of the cerebral hemispheres at the level of the pineal gland, showing sclerotic atrophy of the hippocampus bilaterally in a 26-month-old boy who had a history of status epilepticus followed by spastic quadriplegia, blindness, deafness, and episodic seizures.

Figure 36. Coronal section of the cerebral hemispheres at the level of the substantia nigra, viewed from behind, showing marked sclerotic atrophy of the left hippocampus in a 34-year-old man who had a long history of idiopathic epilepsy.

(And even then, the inhibitory or excitatory nature of the connections is not revealed) and makes the Golgi technique generally unsuitable for application to the abnormal: each patient being uniquely different makes repetition essentially impossible. The use of Lorente de Nó's terminology without using the Golgi technique seems illogical.

Thus, the H1–5 versus CA1–4 terminologies are not simply competitive but represent entirely different ways of representing a small portion of the complexity that is there. As
Lorente de No’s put it, “Architectonics is of enormous value however in making a first analysis and determining what has to be studied with other methods.” Figure 32 is based on Lorente de No’s specific comparisons of the two systems as applied to the human, but the boundaries are only approximations to the authors’ own case material. Indeed, Lorente de No emphasized that the comparisons are fundamentally impossible because the Golgi and Nissl techniques reveal such different cellular characteristics. Amaral and Witter have recently simplified Lorente de No’s terminology, reducing the rat hippocampus to three parts (CA1–3) and making CA4 the “polymorphic cell layer of the dentate gyrus” to emphasize their closely related connections. This is such a cumbersome phrase, however, that we will

Figure 37. A. Coronal section of the cerebral hemispheres at the level of the subthalamic nuclei revealing evidence of the old right temporal lobectomy for temporal lobe epilepsy. B. Holzer-stained section of the left hippocampal formation revealing evidence of severe gliosis of all segments of the hippocampus secondary to correspondingly severe loss of virtually all of the pyramidal cells.
call CA4 or H4–5 the *hilum*; others\textsuperscript{22,31} have referred to it as the *end folium*.

Current technology is focusing on neurotransmitters and their receptors, attempting to define the specific inhibitory and excitatory pathways in the normal. The extension to the abnormal is slowly occurring as the techniques improve, first allowing prospective application of several stains in serial sections of fresh tissue and ultimately, it is hoped, allowing application to fixed tissue, especially to paraffin-embedded tissue, permitting retrospective analyses of archival specimens from previous patients. These techniques reveal not only the destructive lesions but also at least some of the reparative reactions. For example, Timm’s

![Figure 38. Coronal section of the cerebral hemispheres at the level of the posterior commissure, viewed from behind, showing severe atrophy of the left hippocampal formation and fornix secondary to old trauma.](image)

![Figure 39. Coronal section of the cerebral hemispheres just behind the splenium of the corpus callosum, viewed from behind, showing the anterior extent of the right temporo-occipital infarct that destroyed the posterior right hippocampal formation.](image)
stain for zinc reveals the distribution of the glutamate-secreting mossy fibers of the granule cells.\textsuperscript{9} These excitatory projections normally terminate on the dendrites of the neurons of the hilum and CA3 but abnormally can be seen to extend back onto the proximal dendrites of the granule cells themselves. This additional excitatory input may disturb the balance of excitation and inhibition within the hippocampus.\textsuperscript{9a} Clearly, much more extensive study is required, supplemented by comparisons with many other newly developing techniques, before the pathology of limbic epilepsy is fully understood.

It must be emphasized that Sommer's sector is the lesion that, in its classic version, involves approximately CA1 or H1 segments but that can be smaller (Fig. 33) or larger (Fig. 34), expanding to include virtually the whole hippocampus.

\textbf{Figure 40.} \textit{A,} Axial MR image showing evidence of an old embolic stroke with loss of the left posterior medial temporo-occipital region. \textit{B,} Coronal MR image showing loss of the left temporo-occipital and lingual gyrri. \textit{C,} Reconstruction of the MR image showing the extent of the infarction in the left medial temporo-occipital region superimposed on representations of the right homonymous visual field and the limbic system.
Sommer and subsequently Bratz studied old atrophic lesions in the hippocampus and concluded that hippocampal atrophy was not the result but the cause of the seizures. Spielmeyer, who in 1927 studied an acute case of the hippocampal lesion in a patient who had died after 2 days of status epilepticus, emphasized the presence of macrophages and degenerating neurons and concluded that Sommer's sector was caused by vascular insufficiency.
Figure 43. Coronal section of the cerebral hemispheres at the level of the optic chiasm, showing a recurrent multicystic oligodendroglioma in the left frontal lobe extending into the left striatum, corpus callosum, and right cingulate gyrus. This 45-year-old man had the onset of seizures at age 31 years, an oligodendroglioma partially resected at ages 35, 42, and 44 years, with x-irradiation after the second operation.

Figure 44. Coronal section of the cerebral hemispheres at the level of the optic chiasm, showing a diffuse gemistocytic astrocytoma involving the left frontotemporal lobe, striatum, corpus callosum, and septum pellucidum in a 49-year-old man who had a history of a gliome having first been resected 5 years before death. He also received radiotherapy and had two additional resections of the recurrences.
occurring during attacks of seizures, the selective vulnerability related to its poor vascularization supplied by a twisted septal artery, which was more susceptible to spasmodic occlusion during seizures.

The use of cultured rat hippocampal slices has shown that the microvasculature, even though unique to this region, is irrelevant to the selective vulnerability of specific subsets of neurons: Colchicine destroys the granule cells of the dentate gyrus, kainate destroys the CA3 pyramidal cells, and artificial ischemia (anoxia and hypoglycemia) destroys the CA1 pyramidal cells. The mechanism involved in this last lesion appears to include excitotoxic neurosecretion of glutamate by Schaffer collateral projections from CA3 to CA1 because glutamate receptor blockade can reduce the damage. At least two mechanisms are involved: NMDA receptors requiring glycine as a cofactor following combined oxygen and glucose deprivation and non-NMDA receptors following anoxia alone. Hypoglycemia alone produces similar damage to CA1 neurons by a mechanism involving adenosine and A1 receptors. The Pathoklise hypothesis of the Vogt, whereby selective vulnerability was related to specific metabolic differences between neurons in particular sites, appears to be vindicated, especially if one expands the hypothesis to include locally specific, excitotoxic postsynaptic damage. Even so, controversy regarding the cause-or-effect relations between the hippocampal lesion and epilepsy continues, and the authors suspect that the innate neural circuit lends itself admirably to a cycle of progressive physiologic and histologic damage.

Stauder was the first to relate systematically the presence of psychomotor seizures and hippocampal sclerosis, and Margerison and Corsellis gave further support to the concept of hippocampal sclerosis being epileptogenic. The electroencephalographic confirmation of epileptogenicity of hippocampal sclerosis led to the treatment of temporal lobe seizures by surgical resection of the temporal lobe.

Four decades ago, Earle and colleagues proposed that hippocampal sclerosis occurred at birth by molding of the head causing compression of the arteries to the medial temporal lobe, including the uncus and hippocampus, which herniated through the incisura tentorii. The delay in onset of clinical seizure activity for many years after birth was attributed to the slow maturation of the brain. Histologic lesions were found not only in the hippocampus, but also in the uncus, amygdaloid nucleus, and other parts of the anteromedial temporal lobe, so that they named the condition incisural sclerosis. The absence of these lesions in many cases of temporal lobe epilepsy suggests that this hypothesis may not be correct.

Typical hippocampal sclerosis shows neuronal loss, gliosis, and atrophy of CA1 (H1), but the lesion is sloppy and varies from a small part of CA1 to virtually the whole of the hippocampal formation, including the amygdaloid nucleus. Sommer's sector is traditionally approximately CA1, but it is a lesion and is not equivalent to any anatomic segment. The lesion shows variable degrees of neuronal loss.

Figure 45. Horizontal section of the cerebral hemispheres showing a posterior frontal parasagittal meningioma in a 76-year-old woman who died of a ruptured thoracic aortic aneurysm. There were numerous other meningiomas over the cerebral convexities and at the base of the brain. The patient was not known to have had any neurologic symptoms during life.
and gliosis in CA1 and in decreasing order in CA3 and CA4 (endfolium) and the dentate gyrus (Figs. 33 and 34). The CA2 segment is usually spared or involved least in the great majority of cases. The brain may show gross atrophy of the hippocampus bilaterally (Fig. 35) or unilaterally (Fig. 36). The lesion is probably not a static one but evolves over time, the best evidence being derived from special stains showing regeneration of axons in an abnormal pattern.

Endfolial sclerosis, in which neuronal loss and gliosis in the end plate (CA4 or H4-5) are found with complete or almost complete sparing of CA1-3 (H1-3) and the dentate gyrus, is not uncommon but is less often described.\textsuperscript{21, 34} According to Margerison and Corsellis,\textsuperscript{22} endfolial sclerosis is found in patients with later onset of seizures and with fewer lesions in other sites of the brain.

Evidence showing (1) that hippocampal sclerosis can be caused by focal vascular insufficiency because of birth injury, postnatal trauma, cerebral edema, and hypoxic events including status epilepticus; (2) that hippocampal necrosis can be caused by nonvascular factors related to excessive secretion of glutamate in local neuronal circuits specifically involvolving CA1 (H1); and (3) that hippocampal sclerosis can be epileptogenic is abundant and indisputable. The demonstrations by Ojemann (personal communication, 1996) that patients with temporal lobe seizures have a better prognosis if a histologically abnormal hippocampus can be recognized by high-resolution magnetic resonance (MR) imaging and excised but that not all epileptics have a histologically abnormal and MR imaging–recognizable but still electrically abnormal hippocampus suggest that neither the controversy nor the study has been completely resolved.

**Vascular Diseases and Memory Loss**

Memory impairment is prominent in limbic system diseases. Indeed, progressive loss of recent memory is the hallmark of Alzheimer’s disease, in which the hippocampus bears the brunt of the degenerative process. Alzheimer’s disease, however, is also diffuse so that the search for the anatomic substrates of memory must include other, more localized diseases. Severe memory impairment can be produced transiently by epileptic discharges cycling for long periods of time in the limbic system.

![Figure 46](image_url). Coronal section of the cerebral hemispheres at the level of the thalamus, showing a midline metastatic adenocarcinoma in the falx extending into the cingulate and superior frontal gyri bilaterally in a 61-year-old woman.
Figure 47. Two coronal sections of the cerebral hemispheres, showing massive subarachnoid hemorrhage in the deep mid-sagittal fissure extending into the pericerebral sulci and frontal white matter on both sides from a ruptured pericallosal aneurysm. This 14-year-old boy was involved in a motor vehicle accident, sustained a head injury and bilateral leg fractures, developed sudden severe headaches, and collapsed 1 month after the accident.

Figure 48. Coronal section of the cerebral hemispheres at the level of the optic chiasm showing porencephaly involving both medial frontal lobes with communication with the lateral ventricles in a 16-year-old with a history of profound mental retardation and quadriplegia.
"How did I get from point A to point B?" asks the patient after a complex partial, psychomotor, or temporal lobe seizure subsides, realizing that a large and varied terrain must have been successfully traversed with little or no difficulty but with no recollection.

In other patients with bilateral hippocampal destructive lesions, the memory impairment is permanent. Typical is the inability to convert the present immediate sensations into long-term, practically permanent archives. Immediate recall lasting seconds and recent memory lasting minutes are intermediate stages in this conversion. Immediate recall is usually preserved with hippocampal lesions, but recent memory is remarkably and strikingly not preserved. Old memories formed before the lesions occurred are usually preserved, but new memories cannot be formed, and learning is severely impaired.

In the most striking cases, no new memories have been formed, as following bilateral operations on the hippocampus, but with more refined psychometric techniques, more selective losses of parts of memory following unilateral lesions are becoming better categorized. As would be appropriate for language representation in the dominant cerebral hemisphere, left-sided lesions affect verbal memories especially, and right-sided lesions affect visuospatial memories. Ott and Saver reported six cases of left-sided lesions and reviewed the literature concerning 92 left-sided and 16 right-sided lesions, documenting the existence of three major unilateral amnesic stroke syndromes:

1. Posterior cerebral artery branches to the posterior hippocampus, parahippocampus, and collateral isthmus (gyrus fornixus), frequently with contralateral homonymous hemianopia or quadrantanopia, alexia, and color anomia.

2. Anterior choroidal artery branches to the amygdaloid nucleus, anterior hippocampus, and internal capsule (genu and posterior limb), frequently with contralateral hemiparesis, ataxia, sensory, and visual deficits.

3. Thalamoperforating branches of the posterior communicating or first portion of the posterior cerebral arteries to the medial thalamus, mammillothalamic tract, and ventral amygdalofugal fibers, frequently with somnolence, vertical gaze paresis, hemiparesis or quadriparesis, alexia, visuospatial deficits, emotional facial paresis, and aphasia.

This last syndrome calls attention to the medial thalamus, which is not part of the limbic system but rather a part of the extralaminar system with relays from collaterals of the major ascending sensory tracts to the superior colliculus and thence to the hypothalamus, medial thalamus, and frontal pole. Bilateral medial thalamic lesions producing memory losses, as in Wernicke-Korsakoff syndrome, were first emphasized by Victor and colleagues, but unilateral lesions have produced similar deficits.

![Figure 46. Microscopic preparation of a horizontal section of the brain at the level of the foramina of Monro showing an incidental plicotic astrocytoma in the left fornix (LFB-PAS-H stain). This 71-year-old man had had renal dialysis for 20 years and died of gastrointestinal bleeding.](image-url)
Thus, at least two major pathways beyond the primary sensory projections to specific portions of the lateral thalamus and cerebral cortex spread the present immediate sensations (1) from the immediately adjacent association cortex into the limbic system (and anterior thalamus) and (2) from subcortical extralaminiscal structures into the frontal pole (and medial thalamus). The latter involves more emotional aspects accompanying the present sensations. Both systems appear to work in parallel in the conversion of the present into the archives of memory.

Case Studies

The authors have studied four cases with striking memory deficits: three at autopsy and
one by MR imaging, two with epilepsy and two with strokes, all four with localized hippocampal lesions, and two with little else.

Case 1:

This 44-year-old woman had had meningitis at age 10 months and developed complex partial seizures beginning at age 12 years. She was treated surgically at age 34 years, when a right temporal focus was found. Preoperative Wada test had revealed bilateral speech representation with major memory deficits produced by intracarotid injection of barbiturate on each side. The right temporal lobectomy revealed hippocampal sclerosis (NP9158). Postoperatively, epileptic seizures persisted, and she developed marked personality changes, psychotic delusions, and severe short-term memory impairment. At age 44, she was found dead, drowned in her bathtub, presumably following a seizure. At autopsy, in addition to the right temporal lobectomy (Fig. 37), there was severe left hippocampal sclerosis (Fig. 37) that had effectively contributed to the equivalent of a bilateral hippocampectomy.

Case 2

This 32-year-old seaman had had a severe head injury at some time in the past, variously dated at 24 to 30 years of age, but the details were totally obscure when he was found at age 30 severely incapacitated, unable to give a useful history, and unable to work. He complained of headache, vertigo, depression, and seizures. Extensive psychologic studies revealed severe memory and attention deficits, especially verbal. At age 32, he was found dead, presumably from an epileptic seizure. Autopsy revealed an old left temporal contusion with left hippocampal sclerosis and atrophy of the left fornix (Fig. 38), old right Kernohan's notch, and an old siltlike hemorrhage in the white matter of the right anterior superior frontal gyrus extending into the gyrus rectus.

Case 3

This 79-year-old female novelist, noted for her sharpness of intellect, had required a coronary angioplasty at age 73. She developed episodic ataxia the following year, and MR imaging revealed evidence of periventricular white matter disease and normal pressure hydrocephalus. Episodes of dementia, memory loss, aphasia, and Ménière's syndrome recurred over the next several years, but she continued to work successfully. At age 79, she experienced an acute exacerbation of what turned out to be permanent dementia with a left homonymous hemianopia and left sensory extinction. Computed tomography (CT) and MR imaging revealed infarction in the right medial occipital lobe and increase in ventricular dilatation. She died of cardiac arrest.
Figure 53. Coronal section of the cerebral hemispheres at the level of the optic chiasm showing lymphoma involving the septum pellucidum, wall of the ventricles, and optic chiasm in a 34-year-old HIV-positive man who presented with headache, nausea, and vomiting and had multiple cerebral lesions on MR imaging. He underwent cranial x-irradiation followed by antituberculous therapy, but his condition deteriorated and he died.

Figure 54. Coronal section of the cerebral hemispheres at the level of the posterior commissure, viewed from behind, showing an incidental glioblastoma multiforme involving the left medial temporal lobe in a 52-year-old man who had been a resident in a nursing home for 3 years because of Alzheimer's disease.
Figure 55. Coronal section of the cerebral hemispheres at the level just behind the temporal poles, viewed from behind, showing a large oligodendroglioma involving the right temporal lobe with subfalcine herniation in a 30-year-old man who was found dead at the scene of a motorcycle accident.

Figure 56. Coronal section of the cerebral hemispheres at the level of the splenium of the corpus callosum, viewed from behind, showing a large arteriovenous malformation in the left temporal lobe in a 52-year-old woman who had a history of mental retardation, seizure disorder, and mild spastic quadriplegia. The lesion was found 3 years before death and was treated with multiple embolizations without success.
Figure 57. Coronal section of the cerebral hemispheres at the level of the optic chiasm showing a giant saccular aneurysm of the right middle cerebral artery projecting down into the temporal lobe and a relatively recent hemorrhagic infarction in the left middle and inferior frontal gyri in a 67-year-old man with atherosclerotic cardiovascular disease.

Figure 58. Coronal section of the cerebral hemispheres at the level of the red nucleus, viewed from behind, showing an old contusion involving the right inferior temporal gyrus in a 33-year-old schizophrenic woman who died of acetylsalicylic acid intoxication. There was no documented history of trauma, but similar old contusions were noted in the orbitofrontal cortex and left temporal pole.
failure 9 months later, and autopsy revealed infarction in the right calcarine and posterior hippocampus (Fig. 39) as well as evidence of Alzheimer's disease with neuritic plaques more than neurofibrillary tangles.

Case 4

This 37-year-old woman had had a biliopancreatic diversion for extreme obesity (over 350 pounds) at age 31. Pregnant at age 34, she weighed 160 pounds, but her serum albumin was only 2.5 g/dL. She was considered to be at high risk and to require intravenous hyperalimentation. Unfortunately the left subclavian catheter was in the artery instead of the vein and developed thrombi, which broke off as emboli through the vertebral artery into branches of the left posterior cerebral artery. As she described the episode, she "passed out" and "felt strange." Later, she had difficulty concentrating and comprehending, could not remember her last name, was hesitant reading, and had "unusual features" suggesting "functional embellishment" when examined by a neurologist. Her "visual blurring" progressed to right "tunnel vision" and then to a documented right three-quarters anopia. At age 35, psychometric testing showed immediate and delayed verbal memory deficits, and at age 37, a right superior quadrantanopsia was recorded. MR imaging showed infarction in the left inferior temporoparietal region, including the left posterior parahippocampal gyrus (Fig. 40).

DISEASES IN WHICH THE LIMBIC SYSTEM IS RANDOMLY INVOLVED

Various types of primary tumors, metastatic tumors, ischemic infarcts, hemorrhages, vascular malformation, abscesses, and cysts of various origins are not known to involve the structures of the limbic system preferentially but can occur anywhere within the central nervous system, including the limbic system. The lesions typically extend to the limbic system from adjacent structures. In many instances, the limbic structures are merely compressed or displaced. A few selected examples follow.

Lesions involving the cingulate gyrus and adjacent corpus callosum include glioblastoma (Figs. 41, 42), oligodendroglioma (Fig. 43), astrocytoma (Fig. 44), parasagittal meningioma (Fig. 45), metastatic carcinoma (Fig. 46), infarction in the distribution of the anterior cerebral artery and subarachnoid, and intracerebral hemorrhages secondary to rupture of a supra-sellar artery aneurysm (Fig. 47). Parenchymal (Fig. 48), cyst, and abscess involving this region are relatively uncommon.

Lesions involving or arising in the neighborhood of the septum pellucidum, including the septal nuclei and fornices, may be a pilocytic

Figure 59. Coronal section of the cerebral hemispheres at the level of the lateral geniculate nucleus, showing a well-encapsulated abscess in the right temporal lobe. This 13-year-old girl had congenital heart disease and repeated ear and nose infections.
astrocytoma (Fig. 49), neurocytoma, colloid cyst of the third ventricle (Fig. 50), intraventricular oligodendroglioma, subependymal giant cell astrocytoma (Fig. 51), subependymoma (Fig. 52), or lymphoma (Fig. 53).

The same types of pathologic processes including neoplasms (Figs. 54, 55), vascular lesions (Figs. 56, 57), and traumatic lesions (Fig. 58) may involve the whole or part of the temporal lobe. Relatively small lesions, such as oligodendroglioma, dysembryoplastic neuroectodermal tumor, cavernous angioma, or ganglioglioma causing partial complex seizures, were previously rarely found during life but are now relatively common with the development of CT and MR imaging. Abscesses in the temporal lobe (Fig. 59) were once frequent and fatal complications of otitis media but are now rare because current antibiotics cure most such infections before they spread into the brain. In cases of gliomatosis cerebri (Fig. 60), one or both temporal lobes may be involved along with the thalami and upper brain stem.

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Address reprint requests to
Cheng-Mei Shaw, MD
Neuropathology Laboratory
University of Washington Medical School
Seattle, WA 98195-6480
Neuroimaging Clinics Of North America.
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