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The Large Temporal Horn: MR Analysis in Developmental Brain Anomalies versus Hydrocephalus

Lori L. Baker,[1] and A. James Barkovich[1]

Purpose: To utilize MR to delineate the morphologic abnormalities of the temporal horn and adjacent structures in patients with congenital brain anomalies and to differentiate these findings from the temporal horn alterations in obstructive hydrocephalus. **Patients and Methods:** Thirty-six patients were included in this retrospective study, including eight with agenesis of the corpus callosum (ACC), four with lissencephaly (lis), four with lobar holoprosencephaly (holo), as well as 20 with isolated obstructive hydrocephalus due to tumor (17 patients) or aqueductal stenosis (three patients). Twenty patients with normal scans were included as controls. **Results:** Coronal plane image analysis showed that 75% of patients with brain anomalies had enlargement of the temporal horns, most prominently involving the inferolateral aspects of the ventricle (8/8 ACC, 4/4 lis, 0/4 holo). Hippocampal formations were small in 62% (6/8 ACC, 3/4 lis, 1/4 holo). An abnormal, vertical orientation (incomplete inversion) of the hippocampal formations was observed in 82% (8/8 ACC, 4/4 lis, 1/4 holo). Focal thinning of the white matter lateral to the temporal horn was seen in 50% (8/8 ACC, 0/4 lis, 0/4 holo). All patients with isolated obstructive hydrocephalus showed enlargement of the temporal horns, most pronounced in the superior-lateral region. Hippocampal formations showed normal, horizontal orientation (complete inversion) and were of normal size in 17 of 20 patients; the only exceptions included patients with severe hydrocephalus where the hippocampi were flattened along the inferior margin of the temporal horn. Temporal lobe white matter was normal in the 17 patients with mild or moderate hydrocephalus: diffuse white matter thinning inferolaterally was observed in the three patients with severe hydrocephalus. Distinct differences were present in the morphology of the temporal horn and surrounding brain in congenital brain disorders compared with those in patients with hydrocephalus. The anomalous brains showed enlargement as a result of incomplete development, and the hydrocephalic brains showed enlargement as a result of increased intraventricular pressure. **Conclusion:** Temporal horn enlargement in lissencephaly and agenesis of the corpus callosum should not be misinterpreted as hydrocephalus. Analysis of temporal lobe morphology will allow differentiation if doubt exists.

Index terms: Brain, temporal lobe; Brain, abnormalities and anomalies; Hydrocephalus

The development of magnetic resonance (MR) imaging techniques has allowed clear depiction of a variety of anatomic structures that were heretofore adequately visualized only by pathologic examination. In particular, the multiplanar imaging capability of MR has contributed greatly to the evaluation of the normal temporal lobes and the developmental and pathologic processes involving them (1–4).

During the course of evaluating patients with various developmental brain anomalies, we noted that a number of patients had temporal horns that were abnormally large, despite the absence of hydrocephalus. These findings prompted us to pursue the etiology of the temporal horn enlargement in congenital brain anomalies, since commensurate temporal horn enlargement has classically been a sign of hydrocephalus (5, 6).

The purpose of this study was to utilize MR to delineate the morphologic abnormalities of the

temporal horn and adjacent structures in patients with congenital brain anomalies and to differentiate these findings from the temporal horn alterations in obstructive hydrocephalus. As agenesis of the corpus callosum (ACC), lissencephaly, and holoprosencephaly are, in our experience, the anomalies that most commonly manifest temporal horn enlargement, these malformations were specifically studied. Furthermore, we describe the normal embryologic sequence of events resulting in the closely linked development and evolution of the temporal horn of the lateral ventricle and the hippocampal formation, and present a pathogenetic theory that may help explain the anomalous configuration of these structures in congenital brain anomalies.

Patients and Methods

Thirty-six patients (21 males, 15 females), aged 5 months to 52 years (mean age, 10.6 years) were included in this retrospective study. Our patient series included eight with ACC, four with lissencephaly, four with holoprosencephaly (1 lobar, 3 semilobar) as well as 20 with isolated obstructive hydrocephalus due to tumor (17 patients) or aqueductal stenosis (three patients). None of the patients with congenital brain malformations had clinical evidence of hydrocephalus. No pathologic confirmation was available in our series of patients with congenital anomalies. Twenty patients, aged 4 months to 55 years (mean age, 11.2 years), with normal scans were included as controls.

The majority of patients (90%) underwent MR imaging on a 1.5 T system (General Electric, Milwaukee, WI); the remaining patients were scanned at various outside institutions and had MR scans of comparable quality. Sagittal, axial, and coronal spin-echo T1-weighted images, 500-800/20-40/1-2 (TR/TE/excitations) and T2-weighted images, 2200-2800/60-80/1-2, were routinely obtained. Slice thickness varied from 3 to 5 mm, and matrix size ranged from 128-256 × 256. Coronal plane images were subjectively evaluated with particular attention to temporal horn size and shape, hippocampal formation size and shape, degree of hippocampal formation inversion (see Fig. 1, described in discussion section). The thickness of the white matter located lateral to the temporal horn and hippocampus and medial to the neocortical gray matter of the temporal lobe was also evaluated. Hydrocephalus was graded as mild, moderate, or severe (5).

Results

Normal Anatomy

Analysis of the temporal lobe anatomy in our series of 20 normal individuals is in agreement with a recently published report (1). As shown in Table 1, 100% of individuals had temporal horns
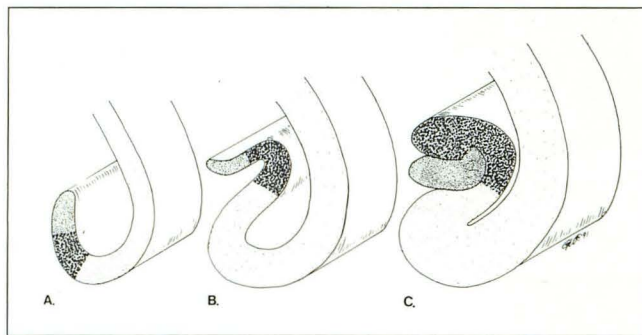

Fig. 1. Simplified schematic diagram of the temporal lobe in the coronal plane depicting the normal inversion of the hippocampal formation during neurodevelopment. (Lateral aspect of the temporal lobe on reader's right).

A, Early in development, the neocortex (*very light stipple*) is located along the lateral aspect of the temporal lobe; the allocortex, from which the dentate nucleus (*medium stipple*), hippocampus (*heavy stipple*), and subiculum (*light stipple*) arise, is located along the inferior aspect of the temporal lobe.

B, The neocortex grows more rapidly than the allocortex during embryogenesis, resulting in medial displacement and internal "inversion" of the dentate gyrus, hippocampus, and subiculum precursors into the temporal horn region.

C, The process of medial displacement and internal "inversion" of the hippocampal formation precursors eventually gives rise to the mature, oval-shaped appearance of the hippocampal formation that is horizontally oriented.

of normal size with a thin, crescent-shaped configuration. All individuals demonstrated well-developed, oval-shaped hippocampal formations (Fig. 2A). All hippocampal formations were horizontally oriented, compatible with complete inversion. The deep temporal lobe white matter was normal in thickness in all cases.

Congenital Brain Malformations

Temporal horn characteristics in patients with ACC, lissencephaly (lis), lobar and semilobar holoprosencephaly (holo) are summarized in Table 2. Analysis showed that 75% of patients with brain anomalies had enlargement of the temporal horns, most prominently involving the inferolateral aspects of the ventricle (8/8 ACC, 4/4 lis, 0/4 holo) Figs. 3 and 4). Hippocampal formations were small in 62% of patients (6/8 ACC, 3/4 lis, 1/4 holo). The hippocampal formations also demonstrated an abnormal, more vertically oriented morphologic appearance than normal hippocampal formations, suggesting incomplete inversion of the hippocampal formation, in 82% of patients (8/8 ACC, 4/4 lis, 1/4 holo). Focal thinning of the white matter lateral to the temporal horn was seen in 50% of patients (8/8 ACC, 0/4 lis, 0/4 holo). Abnormalities of the temporal horn, hip-

TABLE 1: Temporal horn and hippocampal formation features in 20 normal individuals

	Normal Sized Temporal Horn, Thin, Crescentic Configuration	Complete Hippocampal Formation Inversion (Horizontal Orientation)	Normal-Sized Hippocampal Formation	Normal Temporal Lobe White Matter Thickness
Healthy individuals	100%	100%	100%	100%
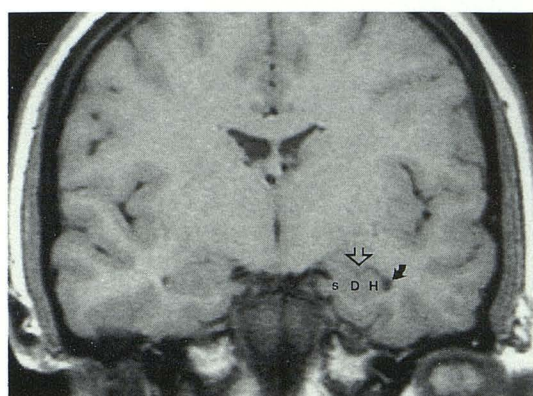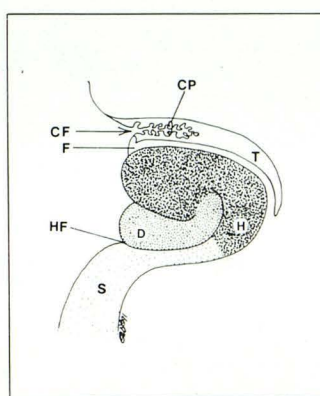
A **B**

Fig. 2. *A*, Normal hippocampal formation and temporal horn, coronal T-1 weighted image (600/12/4). *D* = dentate gyrus; *H* = hippocampus; *s* = subiculum; temporal horn = *closed arrow*; choroid plexus = *open arrow*. Note that the dentate gyrus and hippocampus are seen collectively as a solitary oval-shaped gray matter structure with a horizontal orientation.

B, Simplified schematic diagram of the normal mature hippocampal formation and temporal horn depicted in the coronal plane. *CF* = choroidal fissure; *CP* = choroid plexus; *D* = dentate gyrus; *F* = fimbria; *H*-hippocampus; HF = hippocampal fissure; *S* = subiculum; *T* = temporal horn.

TABLE 2: Temporal horn and hippocampal formation alterations in congenital brain anomalies

	Temporal Horn Enlargement Inferolaterally	Incomplete Hippocampal Formation Inversion (Vertical Orientation)	Small Hippocampal Formation	Focal White Matter Thinning
Agenesis of the Corpus Callosum	100%	100%	75%	100%
Lissencephaly	100%	100%	75%	0
Lobar/semilobar Holoprosencephaly	0	25%	25%	0

pocampal formations, and/or adjacent white matter were most commonly seen in patients with ACC (Fig. 3) and lissencephaly (Fig. 4); patients with holoprosencephaly, in this series, did not share the temporal horn characteristics seen in the other two groups of patients.

Hydrocephalus

Temporal horn alterations in patients with obstructive hydrocephalus are shown in Table 3. All patients with isolated obstructive hydrocephalus showed enlargement of the temporal horns, most pronounced in the superior-lateral region. Hippocampal formations showed normal, complete inversion and were of normal size in 17 of 20 patients (Fig. 5); the only exceptions included patients with severe hydrocephalus, where the hippocampi were flattened along the inferior margin of the temporal horn (Fig. 6). Temporal lobe white matter was normal in the 17 patients with

mild or moderate hydrocephalus; diffuse white matter thinning inferolaterally was observed in the three patients with severe hydrocephalus.

Discussion

Development of the Normal Temporal Horn and Hippocampal Formation

To better understand the configuration of the temporal horns in patients with congenital brain anomalies, a brief review of the embryologic development of the lateral ventricles and the hippocampal formation in the fetus is useful (7–14). The neural tube begins to form at approximately 17 days after fertilization as a pair of neural folds forms on the dorsal surface of the embryonic disk. As the actin myofibrils within the neural folds contract, the lips of the folds are drawn medially and first fuse at the rhombencephalon. From this point, fusion of the neural

Fig. 3. Agenesis of the corpus callosum, coronal T1-weighted image (600/20/2). Hippocampal formations (*closed arrows*) are small and show an abnormal, more vertical orientation, suggesting incomplete inversion bilaterally. Temporal horns are enlarged inferolaterally (*open arrows*).

Fig. 4. Lissencephaly, coronal T1-weighted image (700/20/2). Small hippocampal formations demonstrate an abnormal, vertical orientation bilaterally (*closed arrows*), indicative of incomplete inversion. Inferolateral enlargement (*open arrows*) of the temporal horns is also noted.
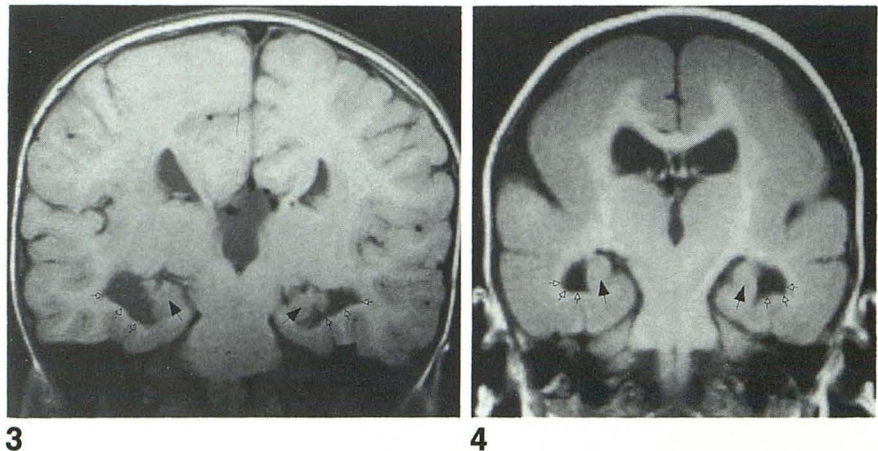

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TABLE 3: Temporal horn and hippocampal formation alterations in obstructive hydrocephalus

	Temporal Horn Enlargement Superolaterally	Complete Hippocampal Formation Inversion (Horizontal Orientation)	Normal-Sized Hippocampal Formation	Normal Temporal Lobe White Matter
Obstructive Hydrocephalus	100%	100%	85%	85%

Fig. 5. Obstructive hydrocephalus, postgadolinium coronal T1-weighted image (800/30/2). Hippocampal formations (*closed arrows*) are normal in size and completely inverted. Temporal horns are enlarged superolaterally (*open arrows*).

Fig. 6. Severe, chronic obstructive hydrocephalus, coronal T1-weighted image (650/20/2). Hippocampal formations (*closed arrows*) are completely inverted, but appear small and flattened due to longstanding obstructive hydrocephalus. Temporal horns are enlarged superolaterally (*open arrows*). *M* = large obstructing meningioma arising from right sphenoid wing.

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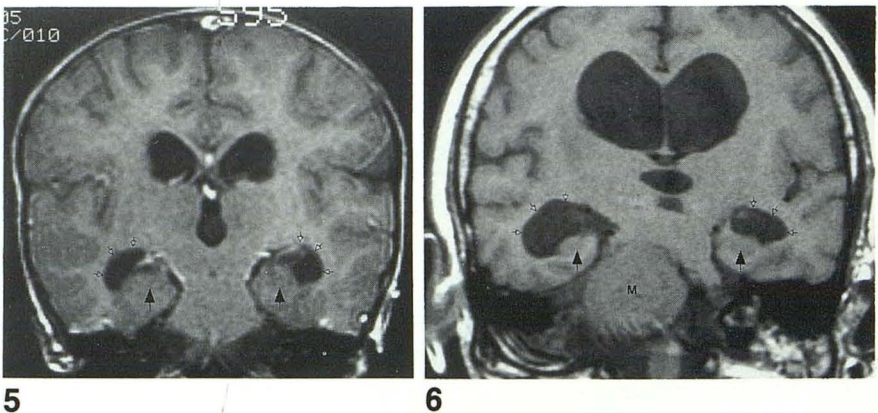
folds progresses both cranially and caudally to form the neural tube. By the end of the fourth week, three protuberances developed at the cephalic end of the tube: prosencephalon (forebrain), mesencephalon (midbrain), and rhombencephalon (hindbrain). The prosencephalon subsequently subdivides to form the telencephalon (cerebral hemispheres, putamen, caudate nucleus) and diencephalon (thalamus, hypothalamus, and globus pallidus). A pair of telencephalic vesicles evaginate symmetrically dorsolaterally within the prosencephalon (Fig. 7A). The embryonic cerebral vesicle possesses three basic primordia. The ventral wall carries the primordium of the corpus striatum that gives rise to the basal ganglia. The dorsolateral wall carries the primor-

dium of the pallium from which the cerebral cortex (neocortex) will evolve. Lastly, the medial wall contains the primordium of the hippocampus. As the cerebral vesicles evaginate dorsolaterally, a midline cavity remains as the third ventricle, dipping caudally within the diencephalon. By 6 to 7 weeks gestation, the cerebral cortex (or pallium) has grown significantly in thickness and extends from the primordial hippocampus medially to the lateral aspect of the developing corpus striatum (Fig. 7B).

The cerebral cortex has a characteristic structure, consisting of nerve cells and nerve fibers arranged in layers. However, there are several types of cortex depending on histologic composition of the cell layers and the sequence of
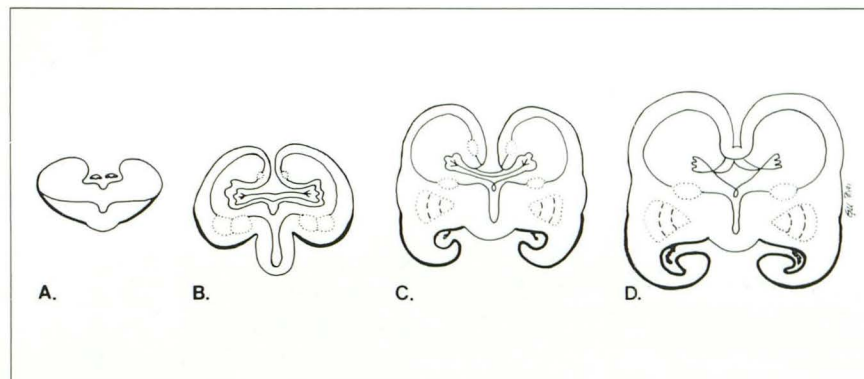
Fig. 7. Schematic diagram depicting embryogenesis of the lateral ventricles and hippocampal formation in the control plane.

A, Four weeks gestation. Telencephalic vesicles evaginate symmetrically dorsolaterally within the prosencephalon. *Heavy solid line* outlines precursors of the allocortex and neocortex.

B, Six to 7 weeks gestation. As the cerebral vesicles evaginate dorsolaterally, a midline cavity remains as the third ventricle, dipping caudally within the diencephalon. The primordia of the hippocampi are located along the medial aspects of the cerebral vesicles (*dotted structures*). The primordia of the corpus striatum, from which the basal ganglia will arise, are located inferior to the cerebral vesicles (*paired dotted structures*). *Heavy solid line* outlines cerebral cortex from which the allocortex and neocortex of the temporal lobe will arise.

C, Seven to 8 weeks gestation. Differentiation and increase in thickness of the neocortex, laterally, and allocortex, inferomedially. As the neocortex differentially grows and evolves, the allocortex is displaced inferiorly, medially, and internally into the temporal horn. Concomitant rotation of the cerebral vesicles in the ventrodorsolateral and anteroposterior directions results in formation of the rudimentary temporal horn. *Dotted structures* (from superior to inferior) = primordia of the hippocampi, caudate nuclei, and lentiform nuclei.

D, Nine to 10 weeks gestation. Continued differential growth of the neocortex relative to the allocortex resulting in further internal inversion of the allocortex into the temporal horn. Further ventrodorsal rotation of the cerebral vesicles has carried the primordia of the hippocampi to the level of the allocortex at the inferomedial aspect of the temporal lobe. Temporal horns become better defined with continued rotational growth of the cerebral vesicles.

phylogenetic development. At 7 to 8 weeks, the three-layered allocortex, comprised of paleopallium and archipallium, differentiates from the remaining cerebral cortex in the region just lateral and inferior to the corpus striatum (9, 15, 16) (Fig. 7C). The archipallium is incorporated into the hippocampal formation, while the paleopallium retains an olfactory function and will evolve into the amygdaloid complex and pyriform area. Concurrently, the six-layered neocortex (neopallium) at the convexities of the developing brain undergoes pronounced differential growth with developmental gradients occurring in predominantly ventrodorsolateral and anteroposterior directions. This differential growth results in the rotation of the telencephalic cavity, with consequent formation of the temporal lobe and temporal horns by 9 to 10 weeks (Fig. 7D).

The simplified schematic diagram in Figure 2B depicts the normal configuration of the temporal horn and hippocampal formation in the coronal plane. A corresponding T-1 weighted MR image obtained in the coronal plane through the temporal horns in a normal individual is shown in Figure 2A. The hippocampal formation is a prominent structure located along the medial aspect of the temporal lobe. This structure may be subdivided into gray matter constituents including the hippocampus, dentate gyrus, and subiculum, and white matter components including the alveus, fimbria, and fornix. The inferior aspect of the temporal lobe (lateral to medial) is comprised of the lateral occipitotemporal gyrus, collateral sulcus, and parahippocampal gyrus. The parahippocampal gyrus curves medially, then superolaterally to terminate at the hippocampal fissure. The subiculum comprises the medial and superior margins of the parahippocampal gyrus to the level of the hippocampus. A layer of white matter, the superficial medullary lamina, lies external to the subiculum. The hippocampus is a crescent-shaped gray matter structure, contiguous with the lateral aspect of the subiculum, that projects into the temporal horn of the lateral ventricle. A thin layer of white matter, the alveus, overlies the hippocampus, just beneath the ependymal layer of the temporal horn. As the alveus courses medially, it thickens and becomes known as the fimbria. The fimbria then curves back onto itself and extends laterally to form the tenia fornicis. The tela choroidea arises from the taenia fornicis and projects through the choroidal fissure

into the temporal horn to give rise to the choroid plexus. At the lateral and superior aspects of the hippocampus, the normal temporal horn is seen only as a thin, curvilinear region with signal characteristics of cerebrospinal fluid.

The organization and maturation of the hippocampal formation is a slow process, extending throughout gestation into the first postnatal year in the human infant (7, 10, 16, 17). The primordial hippocampus lies in the infereomedial surface of the telencephalic vesicle. It develops fibers that descend through the diencephalon anterior to the interventricular foramen and join the ipsilateral mammillary body to form the column of the fornix. However, because of the rotational growth of the telencephalic vesicle, the major portion of the hippocampus is carried dorsolaterally and then ventrally to lie in the medial aspect of the temporal lobe. As the neocortex expands and evolves, the allocortex is displaced inferiorly, medially, and internally into the temporal horn (Fig. 1). It is through this process that the hippocampus eventually "inverts" into the temporal horn.

Ninety percent of the mature human brain is neocortex, which is composed of six histologic layers that vary in cell density, size and shape according to location and function. Although the histologic composition of the neocortex varies with regard to location and function, it generally is composed of six cell layers (16). In the mature brain, most of the parahippocampal gyrus consists of six-layered neocortex. Within the subiculum there is a gradual transition between neocortex and the archipallium of the hippocampus, in which there are only three layers. Thus the appearance of the mature hippocampal formation can be attributed to cell layer growth within the hippocampal formation and to differential growth of the neocortex that results in medial displacement of the allocortex and, in turn, inversion of the hippocampal formation into the temporal horn.

The hippocampus divides the temporal horn into two communicating portions. The superior recess or supracornual cleft lies above the hippocampus, whereas the lateral recess or lateral cleft is vertically oriented and lateral to the hippocampus (10, 18, 19). The roof of the temporal horn is formed (lateral to medial) by the tapetum, radiating fibers of the corpus callosum spread over the lateral and inferior aspects of the temporal horn, tail of the caudate nucleus, stria semicircularis, tenia choroidea, and optic radiation. The floor of the temporal horn (the lateral recess)

is formed by the collateral eminence, the white matter at the depth of the collateral sulcus (Figs. 2 and 3).

The Abnormal Temporal Horn

Our study showed that a high percentage of patients with ACC demonstrate a typical anomalous configuration of the temporal horn. The hippocampal formations are generally small, most likely on a developmental basis. The underlying pathogenetic mechanism resulting in the small hippocampal formations in humans has not been clearly elucidated. However, recent neurobiologic studies (20, 21) addressing the development of hippocampal formations in rats have documented that the putative primary dentate neuroepithelium (the region of origin of the cells that will form the dentate gyrus) is distinctly separate from that of the hippocampus by its location, shape, and cellular dynamics. It appears that the bulk of hippocampal neurogenesis occurs early in the prenatal period. In contrast, proliferation and migration of the cells destined for the dentate gyrus occurs throughout the prenatal period, becoming more pronounced during the later stages of development, and continues well into the postnatal period until it comprises a large portion of the hippocampal formation seen in the mature adult. Thus it seems plausible that the small hippocampal formation seen in congenital brain anomalies may be due to an early interuption in hippocampal and dentate gyrus neurogenesis or neuronal migration.

The MR appearance of the hippocampal formations of the patients with lissencephaly and ACC, showing an abnormal vertical, rather than horizontal, morphologic orientation, suggests an arrest of the normal process of hippocampal inversion, as depicted in Figure 1. Incomplete inversion of the hippocampal formation, a feature seen in patients with ACC, suggests defective medial displacement of the phylogenetically older allocortex by the developing neocortex. The configuration of the enlarged temporal horn in these patients is also distinctive in that it is most prominent along its lateral and inferior aspects. As callosal fibers of the tapetum course along the inferolateral aspect of the temporal horn normally, the abnormal ventricular morphology may, at least in part, result from decreased mechanical support of the ventricular wall in these areas (10, 22).

In lissencephaly, a disorder of incomplete sulcation, the anomalous appearance of the temporal horn and hippocampal formation is similar to that seen in ACC. Interestingly, the pattern of temporal lobe anomalies in lissencephalic brains is similar to findings described in the pathology literature regarding premature infants, who also have poorly developed brains with incomplete sulcation (22, 23). Therefore, a lack of development of the neocortex and incomplete proliferation/migration of the dentate cells seem particularly likely in these patients (23).

With the exception of one individual, our group of patients with lobar and semilobar holoprosencephaly did not demonstrate incomplete development and inversion of the hippocampal formations or abnormal temporal horns. This finding was surprising because incomplete or lack of development of the hippocampal formations and temporal horns has been described in the sonographic (24) and pathology literature (23, 25) as a finding in holoprosencephaly. The discrepancy of our findings with these previous studies may reflect our population of patients with holoprosencephaly who had relatively well-developed brains (ie, lobar and semilobar holoprosencephaly); in contradistinction, many of the patients detected prenatally and studied pathologically have severe (alobar) holoprosencephaly and, presumably, less well-developed hippocampal formations.

The pattern of temporal horn enlargement observed in patients with isolated hydrocephalus differed from that seen in the patients with congenital malformations in that the morphology was grossly normal—the structures around the ventricle appeared merely compressed. The hippocampal formation was normal in size and completely inverted, and the temporal lobe white matter was of normal thickness in 85%. Exceptions to these generalizations were found in those patients with severe hydrocephalus; in this group, the hippocampal formations were completely inverted but flattened along the inferior aspect of the dilated temporal horn, presumably by the high intraventricular pressure. The pattern of temporal horn dilatation was also different compared to those patients with malformations. Hydrocephalic patients showed dilatation of the "natural" recesses of the temporal horn, ie, enlargement was most pronounced superiorly and laterally.

It would be interesting to study a group of patients with lissencephaly or ACC and hydrocephalus to see if the present analysis allows segregation of those patients with hydrocephalus and underlying brain malformations from those with uncomplicated hydrocephalus. Unfortunately, we were unable to find any such patients in our files. Nonetheless, it is obvious from this analysis that patients with lissencephaly and ACC can have temporal horn enlargement in the absence of hydrocephalus and that, if a question of hydrocephalus is present, it should be possible to confirm or eliminate that question based on the morphology of the temporal horn enlargement.

To summarize, we have compared the patterns of temporal horn enlargement in patients with congenital brain malformations to those in patients with hydrocephalus. Distinct differences were present in the morphology of the temporal horn and surrounding brain in these disorders, with the anomalous brains showing enlargement as a result of incomplete development and the hydrocephalic brains showing enlargement as a result of increased intraventricular pressure. Temporal horn enlargement in lissencephaly and ACC should not be misinterpreted as hydrocephalus; analysis of temporal lobe morphology will allow differentiation if any question exists.

Acknowledgment

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