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MR of Aqueductal Stenosis: Evidence of a Broad Spectrum of Tectal Distortion

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The MR scans of 18 patients with nontumoral aqueductal stenosis and six patients with neoplastic stenosis of the aqueduct were reviewed in order to document and understand the variable appearance of the aqueduct and periaqueductal region on MR. The mesencephalic tectum is often distorted in patients with benign aqueductal narrowing. This distortion results in a number of different MR appearances ranging from an elongated and thin to a short and broad tectum. When compressed by a dilated suprapineal recess, the distorted tectum is sometimes difficult to differentiate from the bulbous enlargement caused by a tectal glioma.

Patients in whom distortion of the tectum is the result of hydrocephalus and aqueductal stenosis should be recognized to avoid unnecessary diagnostic procedures and misdiagnosis.

As cited by Roback and Gerstle [1], hydrocephalus resulting from narrowing of the cerebral aqueduct was initially reported in 1900 by Bourneville and Noir [2] and Oppenheim [3]. Despite the massive number of publications since that time, debate continues regarding differentiation among the various causes of this condition [1, 4–13]. MR is well suited to imaging the aqueduct because of its multiplanar capabilities, high contrast resolution, and sensitivity to flow [14]. The sagittal plane, in particular, provides superb anatomic depiction of the aqueduct and adjacent structures. Having noticed a wide variation in appearance of the stenotic aqueduct as well as the presence of associated abnormalities, we undertook a retrospective review of all patients with narrowing of the sylvian aqueduct in order to document and try to understand the variable appearance of the aqueduct and periaqueductal region in these patients.

Materials and Methods

MR scans of 18 patients with aqueductal stenosis were evaluated retrospectively. The patients were 4 months to 56 years old (average, 22 years). The six infants (less than 2 years old) were referred for an enlarging head circumference. The remaining patients were more than 12 years old and were referred for a variety of nonspecific neurologic symptoms. The most common symptoms were headaches, nausea, vomiting, and dizziness. Five patients had paralysis of upward gaze. Fifteen patients were only examined before shunting. One patient was examined only after shunting; the shunt was malfunctioning at the time. Two patients were examined both before and after shunting. One patient (examined only before shunting) had a stenotic aqueduct with the appearance of a classic aqueductal "web." This condition differs from other forms of benign aqueductal stenosis and was evaluated separately. None of the patients had a history of meningoencephalitis. Patients with the Chiari II malformation were excluded because of the complexity of the tectal lesion and the uncertain cause of the hydrocephalus in this complex malformation [15].

MR examinations in five patients with tectal gliomas and one patient with an ependymal cyst blocking the distal third ventricle (and the opening of the aqueduct) were reviewed and compared with the 18 patients with benign aqueductal stenosis. These patients were 6–22 years old (average age, 14 years). They had the same nonspecific symptom complex of headaches, nausea, vomiting, and dizziness.

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All patients were examined on a 1.5-T GE imager. Acquisition parameters included sagittal spin-echo (SE) 500–600/20–32 (TR/TE) images with a 3-mm section and 1-mm gap and axial SE 2000–2800/35–70 images. The MR images were evaluated subjectively for location of the narrowing within the aqueduct, presence or absence of a CSF flow void within the aqueduct on axial T2-weighted images, evidence of distortion of the mesencephalic tectum, size (length and thickness), signal intensity of the tectum, shape of the third ventricle (particularly the anterior and posterior recesses), and size and location of the fourth ventricle. The size of the tectum was measured from hard-copy images by using calipers as described by Sherman et al. [16]. When hydrocephalus was present, it was graded by the frontal horn ratio as mild (35–40%), moderate (40–50%), or severe (greater than 50%). Anterior and posterior recesses of the third ventricle were judged subjectively as normal or mildly, moderately, or severely dilated. Three patients who were examined only when their ventricles were decompressed by diversion of CSF were not evaluated for ventricular size or shape. Cerebellar tonsils were evaluated for their shape and location with respect to the foramen magnum as previously described by Barkovich et al. [17].

Results

Benign Aqueductal Stenosis (17 Patients)

The level of the aqueductal narrowing began at the rostral end of the aqueduct (proximal stenosis) in four patients, at the level of the superior colliculi in eight patients, and at the level of the intercollicular sulcus (just above the inferior colliculi) in five patients. Hydrocephalus was present in all patients before shunting (Table 1). Two patients with proximal stenosis and marked hydrocephalus had atrial diverticula compressing the tectum (Fig. 1). Anterior recesses of the third ventricle were moderately dilated in eight patients and severely dilated in nine patients. The suprapineal recess was mildly dilated in six patients, moderately dilated in nine patients, and severely dilated in two patients. A small amount of periventricular high signal intensity was seen on T2-weighted images in four patients. In one patient examined again after shunt placement, the periventricular high signal intensity was no longer present.

The appearance of the tectum varied with the location of the stenosis, degree of the hydrocephalus, and degree of dilatation of the suprapineal recess. Four of five patients with stenosis beginning at the intercollicular sulcus had thinning of the superior tectum, presumably resulting from dilatation of the proximal aqueduct (Fig. 2). In two of the four, the thickness of the tectum at the level of the superior colliculus was less than 2.4 mm, falling outside the 95% confidence limits for tectal size, as determined by Sherman et al. [16]. In one of these patients (who had moderate hydrocephalus), the proximal aqueduct was so dilated that the tectum was difficult to visualize (Fig. 3). In all the patients who had aqueductal

TABLE 1: Level of Stenosis/Tonsil Position in Patients with Benign Aqueductal Stenosis

Level of Aqueductal Stenosis	No. of Patients	Hydrocephalus			Tonsils	
		Mild	Moderate	Marked	Normal	Ectopic
Proximal stenosis	4	0	1	3	4	0
Superior colliculi	8	1	1	6	3	5
Intercollicular sulcus	5	1	4	0	4	1
Total	17	2	6	9	11	6

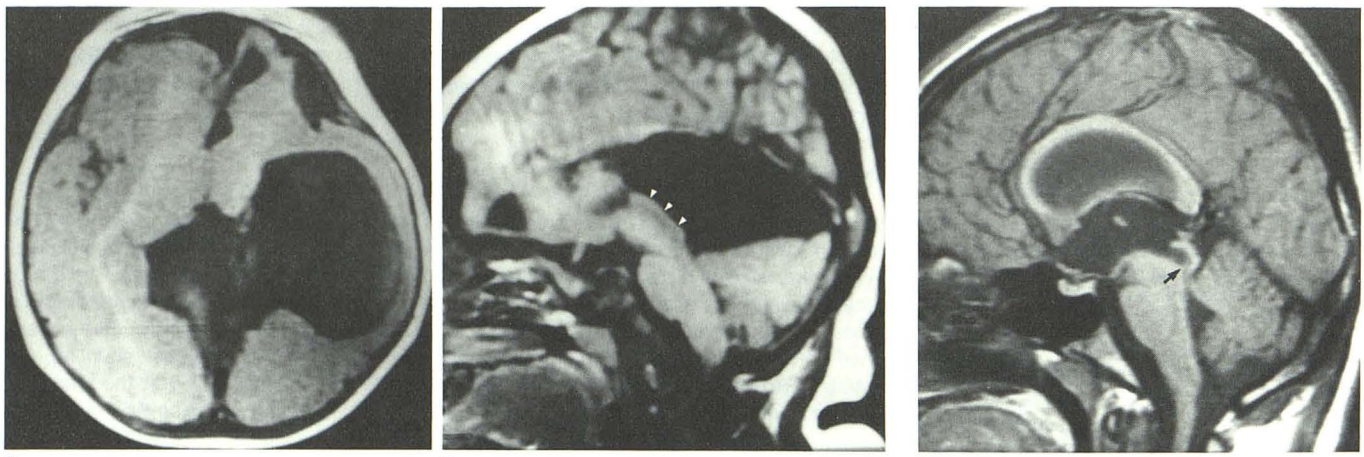


Fig. 1.—Large atrial diverticulum compresses aqueduct in patient with frontonasal encephalocele and obstructed temporal horn.

***A*, Axial image, SE 600/20, shows marked dilatation of left temporal horn with herniation of temporal horn into quadrigeminal plate cistern.**

***B*, Midline sagittal image, SE 600/20, shows marked compression of tectal plate by atrial diverticulum (*arrowheads*), resulting in secondary aqueductal stenosis. Major compressive force on tectum must be directed parallel to *arrowheads*.**

Fig. 2.—Aqueductal narrowing at intercollicular sulcus. Midline sagittal MR image, SE 600/20, reveals moderate ventricular dilatation. Proximal aqueduct is dilated, but caliber of aqueduct abruptly narrows at level of intercollicular sulcus (*arrow*). Patients with aqueductal narrowing at this level had only mild or moderate hydrocephalus.

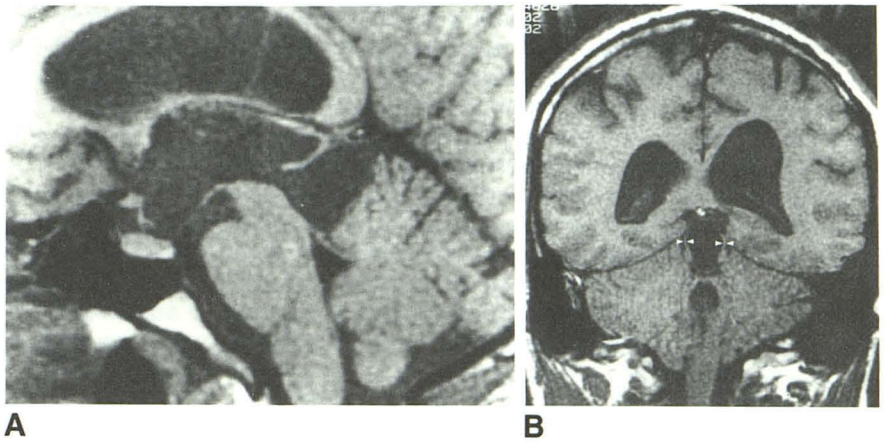


Fig. 3.—Severe tectal thinning.
***A*, Midline sagittal MR image, SE 600/20, shows moderate dilatation of third and lateral ventricles. Tectum is not seen on this image.**
***B*, Coronal image, SE 600/20, reveals marked dilatation of aqueduct with thinning of lateral walls (*arrowheads*). We hypothesize that the ventricular system may have decompressed through perforation of thinned aqueductal walls, because CT scan obtained after CSF diversion did not show cystic mass in region of quadrigeminal plate.**

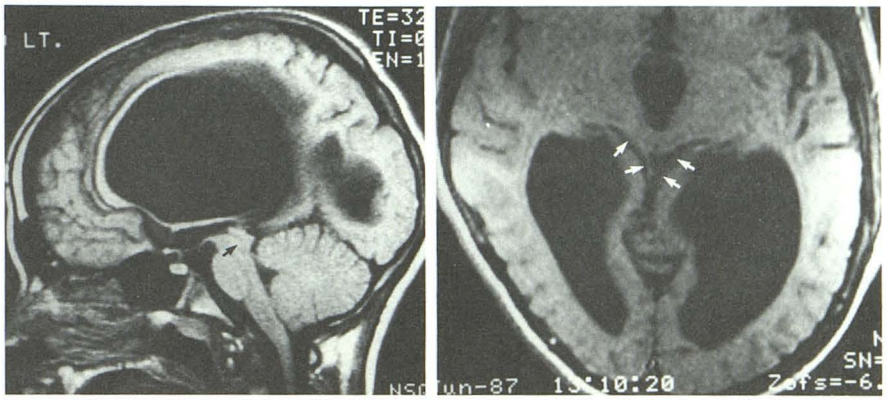


Fig. 4.—Compression of tectum by dilated temporal lobes.
***A*, Midline sagittal MR image, SE 500/32, reveals severe dilatation of lateral ventricles. Aqueduct is severely narrowed at level of superior colliculi (*arrow*).**
***B*, Axial MR image, SE 500/32, through proximal aqueduct shows dilated temporal lobe compressing proximal aqueduct (*arrows*), giving tectum "beaked" appearance. Aqueductal narrowing in such instances may be secondary phenomenon.**

stenosis beginning at the level of the superior colliculi, the tectum was compressed laterally by the markedly dilated temporal lobes (Fig. 4). The tectum was also foreshortened and thickened as a result of compression from above by the dilated suprapineal recess of the third ventricle (Fig. 5). The thickness of the tectum was greater than 7 mm (outside the 95% confidence limits) at the level of the superior colliculi in seven of 11 patients with moderate or severe dilatation of the suprapineal recess. The thickness was greater than 7 mm at the level of the inferior colliculi in eight of the 11 patients. The length of the tectum was less than 10 mm (outside 95% confidence limits) in the same seven patients who had abnormally thick superior colliculi.

The signal of periaqueductal gray matter was isointense relative to normal gray matter on all imaging sequences in 13 patients. The periaqueductal region exhibited a modest increase in signal intensity in two patients and a marked increase in signal intensity in two additional patients on the SE 2000/35–70 sequences. In one of these latter two patients the high signal persisted after shunting. Diminished CSF signal (CSF flow void) was not seen in any of the patients in this group.

The cerebellar tonsils were normally positioned in 11 patients and were ectopic in six. In the 11 patients with normal tonsillar position, hydrocephalus was severe in five, moderate in three, and mild in three. In the eight patients with the Chiari I malformation, five had severe hydrocephalus and one had moderate hydrocephalus. One of the patients with severe hydrocephalus had associated syringohydromyelia.

Presumed Aqueductal Web (One Patient)

One patient had a thin slip of tissue that was convex posteroinferiorly, separating the inferior-most aqueduct from the fourth ventricle (Fig. 6). The lateral ventricle, third ventricle, and aqueduct were mildly dilated in this patient. The anterior recesses of the third ventricle were moderately dilated; the pineal recess was normal. The tectum, fourth ventricle, and cerebellar tonsils were normal.

Tumoral Aqueductal Stenosis (Six Patients)

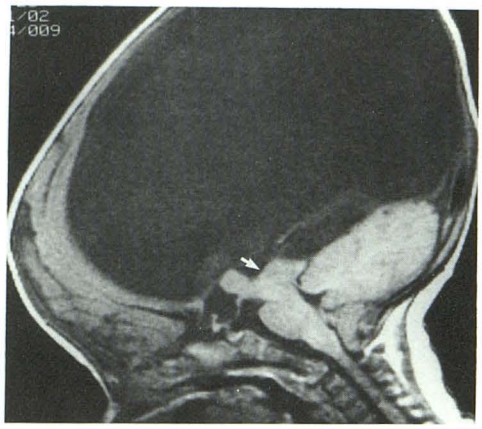
The level of the aqueductal narrowing was between the colliculi in two patients and at the rostral opening of the aqueduct in four patients, including the patient with the ependymal cyst. Hydrocephalus was moderate in three patients and severe in three. The anterior recesses of the third ventricle were mildly dilated in two patients, moderately dilated in three, and severely dilated in one. The suprapineal recess was mildly

dilated in two patients with gliomas and moderately dilated in one patient with a glioma and in the patient with the ependymal cyst. In two patients, the pineal recess was normal, possibly because the tumor grew into the posterior third ventricle and presumably mechanically compressed the suprapineal region. A periventricular rim of high signal intensity was present in two of the six patients; both had gliomas.

The tectum was bulbously enlarged and exhibited a prolonged T2 relaxation time compared with normal brain tissue in all the patients with gliomas (Fig. 7). An aqueductal CSF flow void was not identified in any of these patients. One patient with a very large tectal tumor and severe hydrocephalus had a low-lying fourth ventricle and low-lying, pointed tonsils. In the remaining five patients (four gliomas, one cyst) the fourth ventricle was normal in size and the tonsils normal in position.

Discussion

The interpretation of the findings of this study is facilitated by a brief review of the embryology and anatomy of the aqueduct. After initial closure, the neural tube and its lumen have a relatively uniform dimension throughout the neural



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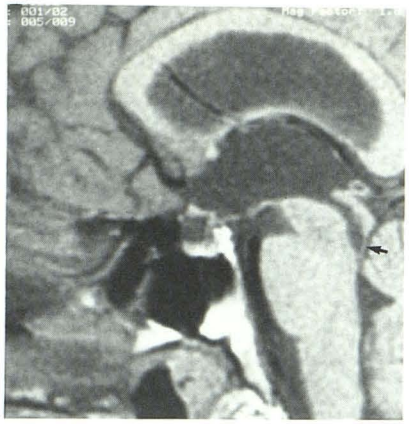

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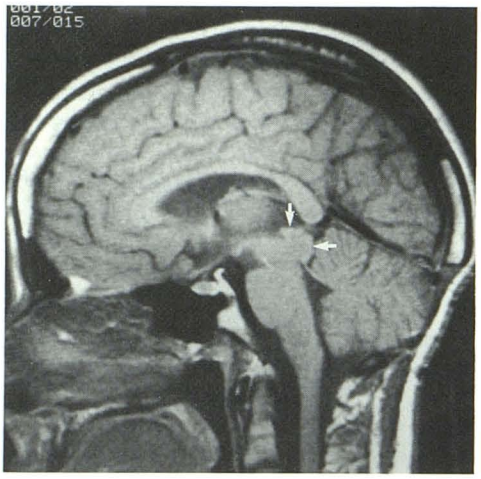
Fig. 5.—Foreshortening and thickening of tectum by dilated suprapineal recess. Midline sagittal image, SE 600/20, in patient with severe hydrocephalus shows thickened tectum, as well as very narrow aqueduct. We believe tectal thickening results from compression by dilated suprapineal recess. To cause tectal shortening and thickening, major direction of compressive force must be parallel to aqueduct (*arrow*). This patient also has low cerebellar tonsils.

Fig. 6.—Presumed aqueductal web. Midline sagittal image, SE 600/20, in patient with mild to moderate hydrocephalus reveals mild dilatation of aqueduct. Degree of dilatation increases slightly in distal aqueduct, which is separated from fourth ventricle by thin slip of tissue (*arrow*). This is classic appearance for aqueductal web.



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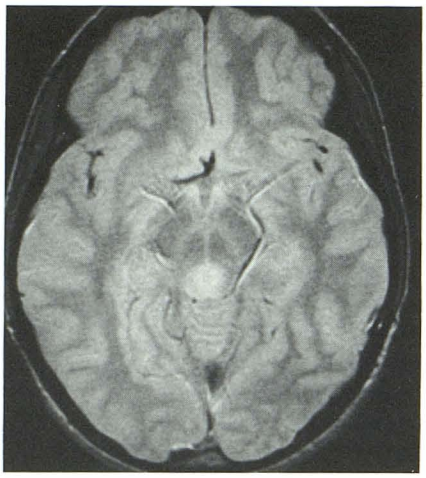

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Fig. 7.—Tectal glioma (moderately anaplastic astrocytoma).

***A*, Midline sagittal MR image, SE 600/20, shows bulbous enlargement of tectum, obliterating aqueduct anteriorly (*arrows*). Ventricles are normal in size because patient has had ventriculoperitoneal shunt.**

***B*, Axial MR image, SE 2800/30, through superior tectum shows obliteration of aqueduct by mass that has high signal intensity on this long TR image.**

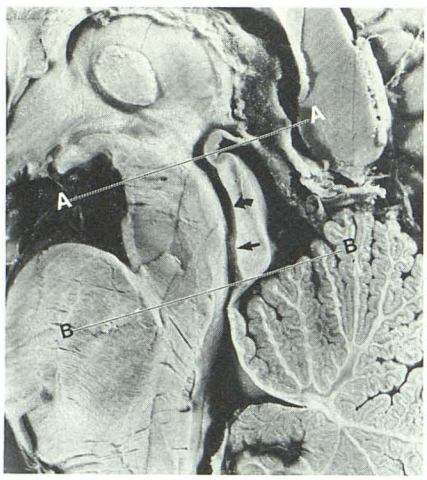

Fig. 8.—Anatomic specimen shows rostral (A) and caudal (B) limits of sylvian aqueduct. Woollam and Millen [18] found focal narrowings of aqueduct at level of superior colliculi (*curved arrow*) and at intercollicular sulcus (*straight arrow*). Portion proximal to superior constriction is pars anterior, portion between constrictions is ampulla, and segment between inferior constriction and fourth ventricle is pars posterior. Note proximity of posterior recesses of third ventricle to tectum. When dilated by hydrocephalus, these recesses (particularly suprapineal recess) can compress and distort tectum.

axis. As the brain and spinal cord mature, the lumen of the neural tube expands in some areas, such as the cerebral ventricles, and narrows in others, such as the spinal canal. Turkewitsch [4], in studies of the brains of human embryos and neonates, found an absolute decrease in the size of the aqueductal lumen beginning in the second month of fetal life and continuing until birth. This narrowing appears to be caused by growth pressures on the aqueduct from adjacent mesencephalic structures.

Woollam and Millen [18] defined the boundaries of the adult aqueduct so that the levels corresponded as closely as possible to the central cavity of the embryonic mesencephalon. The cranial limit was defined as a line drawn perpendicular to the long axis of the brainstem, passing immediately caudal to the posterior commissure. The caudal limit was defined as a line drawn perpendicular to the long axis of the brainstem, passing immediately caudal to the inferior colliculi (Fig. 8). According to this definition, the aqueduct is that portion of the ventricular system lying immediately ventral to the tectum.

The shape and cross-sectional area of the aqueduct vary widely at different levels, probably resulting from the influence of different nuclear masses and fiber tracts at different levels [11]. The aqueduct tapers gradually from its rostral boundary to a superior constriction at the level of the superior colliculi. It then increases in diameter, before narrowing again at the level of the intercollicular sulcus. The area of the aqueductal lumen then gradually increases again in the caudal section. The aqueduct has been divided into three sections by the two constrictions of its lumen [18]. That portion extending from the posterior third ventricle to the superior constriction has been named the pars anterior; the part between the two constrictions of the ampulla and the segment between the inferior constriction and the rostral fourth ventricle has been named the pars posterior (Fig. 8).

The mechanism of aqueductal narrowing has been the subject of much speculation. The oldest hypothesis proposes that an intrauterine infection induces periaqueductal chronic inflammation with consequent gliosis and constriction [19, 20]. Another hypothesis suggests that the normal narrowing of the aqueduct in utero may be excessive or continue into postnatal life [1, 21]. Russell [6] divided aqueductal narrowing into the following six major groups on the basis of histologic features: stenosis, forking (which is associated with molding of the tectum), septum formation, periaqueductal gliosis, inflammatory (and postinflammatory) conditions, and mass lesions. Drachman and Richardson [7] identified histologic features of several of Russell's classes in a single patient and took exception with her classification. Williams [9] proposed that aqueductal narrowing in some patients occurs as a result of hydrocephalus. This theory is supported by the experimental work of Borit and Sidman [22] and Raimondi et al. [23], who demonstrated the development of aqueductal narrowing, with histologic changes identical to the "forking" identified by Russell, in animals with experimentally induced communicating hydrocephalus. Nugent et al. [12] observed that aqueductal narrowing may disappear after the placement of a ventriculoperitoneal shunt, further supporting the suggestion that aqueductal narrowing may in some cases be a result, rather than a cause, of hydrocephalus.

We found that the level of the narrowing affected the appearance of the tectum. When the stenosis was at the level of the superior colliculi, the rostral aqueduct was slightly narrow in caliber and the tectum was compressed by adjacent structures. Usually the tectum was compressed from above by a large suprapineal recess and consequently appeared short and thick (Fig. 5); moreover, molding of the tectum resulted from compression by the dilated temporal lobes (Fig. 4). In two patients, the tectum was thinned and displaced anteriorly as a result of compression by a large, dorsally located atrial diverticulum (Fig. 1). In all patients in whom the stenosis was at the level of the intercollicular sulcus, the pars anterior and ampulla were dilated and the proximal tectum displaced dorsally and thinned (Fig. 2).

The fact that the tectum is shortened and thickened in most patients with dilatation of the suprapineal recess is somewhat surprising. In particular, it is interesting that the deformity differs from that caused by atrial diverticula; the latter cause elongation and thinning of the tectum (Fig. 1). The reason for the difference in the deformity of the tectum caused by these two lesions, both located posterosuperiorly, must be a different direction of the vector of force applied to the tectum from the two lesions. In order to cause tectal thickening and shortening, the major vector of force from the dilated suprapineal recess is presumably directed caudally, parallel to the aqueduct (Fig. 5), whereas the major vector from the atrial diverticula must be directed ventrally (Fig. 1B), perpendicular to the long axis of the tectum.

The distortion of the tectal shape in patients with aqueductal stenosis is important for several reasons. First, the short, thick tectum in these patients with stenosis at the proximal aqueduct and compression of the mesencephalon by a dilated suprapineal recess can be mistaken for a tumor. Such misinterpretation can lead to an unnecessary biopsy or radiation treatment. Moreover, because of the association with the low-lying cerebellar tonsils, observation of distorted tectum may result in a mistaken diagnosis of Chiari II malformation and provoke unnecessary search for a myelodysplasia. Finally, the displacement and thinning of the proximal tectum in patients with a marked dilatation of the proximal aqueduct resulting from a distal aqueductal obstruction can cause confusion and obscure the true diagnosis. Such was the case with the patient in Figure 3, in whom the aqueduct is markedly ballooned and the tectum quite thin despite only moderate lateral and third ventricular dilatation. After placement of a ventriculoperitoneal shunt, a CT scan showed normal-sized lateral ventricles and a normal tectum and quadrigeminal plate cistern. Therefore, the patient does not have an arachnoid cyst of the quadrigeminal plate cistern. We hypothesize that the discrepancy in the size of this patient's ventricular system and aqueduct may have resulted from a decompression of the ventricular system through the quadrigeminal plate.

Another interesting finding in our series of patients was the high incidence of the Chiari I malformation with nontumoral aqueductal stenosis. It is intriguing that five of six patients with low-lying tonsils had severe hydrocephalus compared with five of 11 among the patients with normally positioned tonsils. It is beyond the scope of this article to answer whether

severe aqueductal stenosis and the Chiari I malformation are related and tend to occur together; whether the low-lying tonsils may have caused a partial blockage of CSF flow at the skull base, leading to hydrocephalus (and subsequent aqueductal stenosis); or whether a primary communicating hydrocephalus depressed the tentorium and subsequently both compressed the aqueduct and pushed the tonsils through the foramen magnum.

The differentiation of benign from tumoral aqueductal stenosis by MR is usually quite straightforward, once the concept of distortion of the tectum by the suprapineal recess is understood. In general, tectal gliomas are bulbous masses that can obstruct the aqueduct at any location. All exhibited a prolonged T2 relaxation time. In benign aqueductal stenosis the location of obstruction within the aqueduct was fairly limited and specific, and, although the tectum sometimes appeared thickened, it was never bulbous. The cause of the periaqueductal prolonged T2 relaxation time in the four cases of apparently benign aqueductal stenosis is not certain, but the presence of neoplasms too small to display the characteristic bulbous appearance cannot be ruled out with certainty. Other possibilities include gliosis from prior infection and interstitial edema from transependymal CSF flow. The administration of gadolinium-DTPA in such cases may be of diagnostic assistance.

The presumed aqueductal web is an unusual and interesting lesion that has been discussed in detail by Russell [6] and by Turnbull and Drake [8]. Russell labeled this anomaly "septum formation" and suggested that its origin may be either congenital or inflammatory. Turnbull and Drake hypothesized that such membrane formation might result from a small glial occlusion of the caudal aqueduct that becomes an attenuated sheet of tissue as a result of prolonged pressure from and dilatation of the canal above. We have separated this membranous aqueductal narrowing from other benign aqueductal stenoses because of its distinct radiographic appearance and the possibility of surgical cure by perforation of the membrane [8], thus eliminating the need for indwelling ventriculostomy catheters. New stereotactic devices available for use with MR may make membrane perforation possible without necessitating a craniotomy (Edwards M, personal communication).

Endocrine dysfunction is reported to occur in 15–20% of patients with aqueductal stenosis [24, 25]. The cause of this dysfunction is believed to be chronic compression of the hypothalamic-pituitary axis by the enlarged anterior recesses of the third ventricle [24, 25]. It is of interest that even though the anterior recesses were moderately or severely dilated in 12 of 18 patients with untreated, nontumoral stenosis and in four of six patients with tumoral stenosis, only one patient had evidence of endocrine dysfunction (hyperprolactinemia). Thin (3-mm) sections through the sella revealed a 5-mm pituitary adenoma in this patient.

In summary, the MR appearance of aqueductal stenosis appears to be extremely variable. The tectum is often distorted in these patients; it is important to recognize that this distortion is the result of hydrocephalus and aqueductal stenosis in order to avoid unnecessary diagnostic procedures.

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