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Magnetic Resonance Demonstration of Normal CSF Flow

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The magnetic resonance (MR) imaging appearance and incidence of flowing cerebrospinal fluid (CSF) in the brain were investigated. The MR scans of 46 randomly selected patients with normal examinations were retrospectively reviewed. All patients were studied using both T2-weighted and T1-weighted spin-echo pulse sequences. Thirty-one patients (67%) had decreased intensity in the aqueduct of Sylvius on the T2-weighted images when compared with the intensity of CSF in the lateral ventricles. This was termed the *CSF flow-void* sign. The feature was present in the caudal fourth ventricle in 15 patients (32%) and in the third ventricle in two patients (4%) on T2-weighted scans. It was seen in only 13% of patients on T1-weighted scans. It is believed the CSF flow-void sign represents pulsatile CSF flow. Its recognition is important because it explains the inhomogeneity in the appearance of the CSF, which could be confused with pathologic processes. It may be valuable in the routine evaluation of MR examinations if it does reflect CSF circulatory dynamics.

Magnetic resonance imaging (MRI) has demonstrated the ability to generate striking anatomic images of the brain that were previously seen only by the neurosurgeon or pathologist. Several previous reports have also detailed the appearance of flowing blood on magnetic resonance (MR) images. Rapidly flowing or turbulent blood has been described as having decreased signal [1–3]. We have observed a similar appearance caused by flowing cerebrospinal fluid (CSF) in the aqueduct of Sylvius, the fourth ventricle, and occasionally in the posterior third ventricle and the foramina of Monro. To our knowledge, this is the first report of the use of MRI to study CSF flow.

Materials and Methods

The MR images of 46 subjects with normal ventricles were randomly chosen from our files and subjected to retrospective analysis. The patients were 23–65 years old (average, 40 years). There were 25 men and 21 women. Thirty-two of the patients were referred for MRI of the brain while 14 were referred for cervical MRI.

MR examinations were made via a single-echo eight or 16 multislice technique on a 0.5 T superconductive magnet (Vista-MR, Picker International, Highland Heights, OH). All studies were done using both T2-weighted and T1-weighted pulse sequences. T2-weighted sequences were done with an echo time (TE) of 80 or 100 msec and a repetition time (TR) of 1500–4500 msec. T1-weighted sequences used a TE of 30 or 40 msec with a TR of 500–850 msec. The CSF appears hyperintense relative to brain on T2-weighted sequences and hypointense relative to brain on T1-weighted sequences. All patients were scanned in at least two planes. Contiguous sections were obtained at 5 mm intervals in all patients.

The CSF spaces were examined by the authors for the presence of areas of decreased intensity relative to the CSF in the lateral ventricles, which we term the *CSF flow-void* sign. The sign was specifically noted as present or absent in the aqueduct of Sylvius, the fourth ventricle, and the third ventricle. If the sign was present in the fourth ventricle, its location was described. Both T2-weighted images and T1-weighted images were evaluated. Observations were made on transverse, coronal, and sagittal projections.

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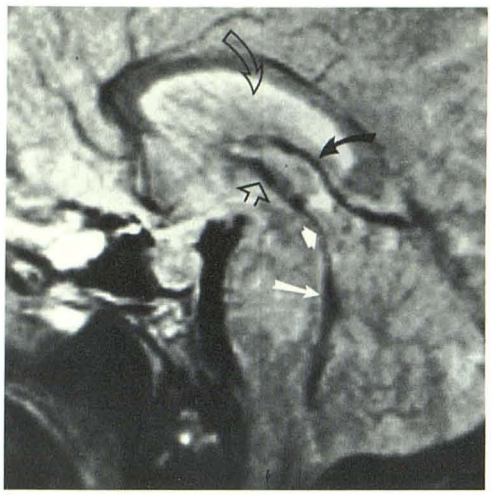

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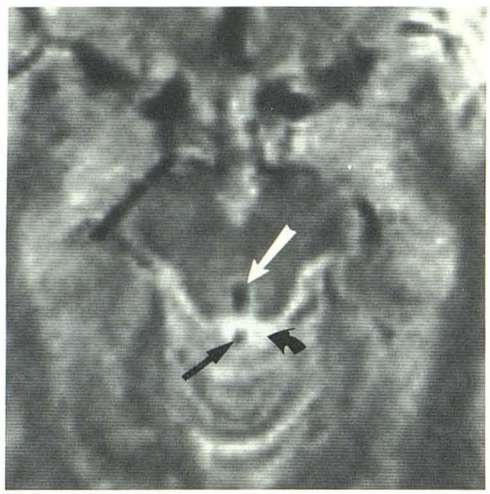

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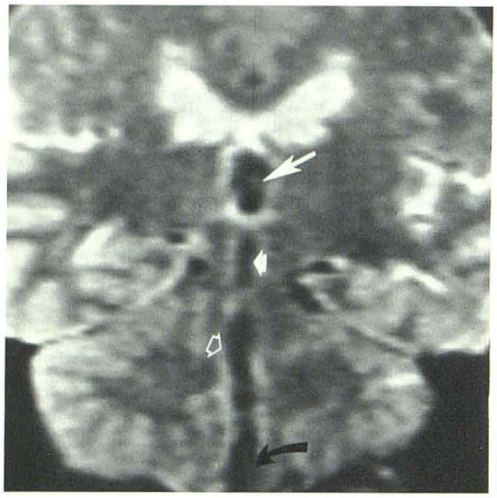
Fig. 1.—T2-weighted midsagittal image, SE 2500/80. CSF flow-void sign (loss of signal) in posterior third ventricle (*straight open arrow*), aqueduct (*short white arrow*), and fourth ventricle (*long white arrow*). CSF in lateral ventricle (*curved open arrow*) is bright. Flowing blood in internal cerebral vein (*curved solid arrow*) is dark.

Fig. 2.—T2-weighted transaxial image at superior collicular level, SE 3500/80. CSF flow-void sign in aqueduct (*white arrow*). CSF in cistern is bright (*curved arrow*). Precentral cerebellar vein is posterior to aqueduct (*straight black arrow*).



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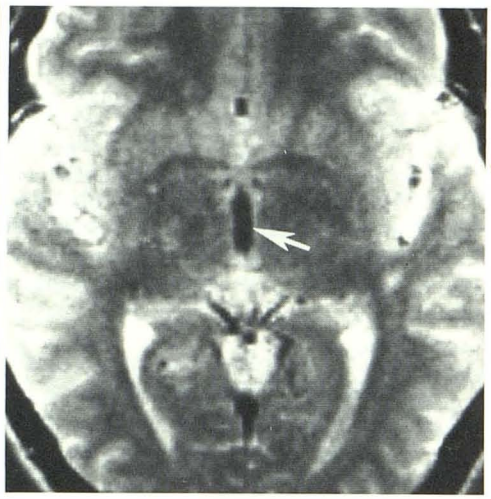

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Fig. 3.—T2-weighted images, SE 3500/80. CSF in lateral ventricle is bright. **A**, Coronal image. CSF flow-void sign in third ventricle (*long white arrow*), aqueduct (*short white arrow*), and fourth ventricle (*open arrow*). Foramen of Magendie (*curved arrow*). **B**, Transaxial image. CSF flow-void sign in third ventricle (*arrow*).

Results

Thirty-one patients were found to have decreased intensity in the aqueduct of Sylvius on the T2-weighted images (figs. 1–3). In six of these patients the sign was present on the T1-weighted images (fig. 4). Of the 31 patients, 15 also had decreased intensity in the fourth ventricle (figs. 1, 3, and 4) and two in the third ventricle (figs. 1 and 3). The sign was absent in 15 patients, that is, there was no difference in CSF intensity when comparing the aqueduct with the lateral ventricles. Of these 15 patients, however, three had decreased intensity in the caudal fourth ventricle, relative to the lateral ventricles (fig. 5). In the 18 patients with the flow-void sign in the fourth ventricle, all had the sign in the midline, while only one had the sign near the foramina of Luschka (fig. 6). In this case it could not definitely be differentiated from the posterior inferior cerebellar artery. The average age of patients with the CSF flow-void sign in the aqueduct was 41 years; the maximum age was 63 years; the minimum age was 23 years. There were 15 men and 16 women. Of those without the sign the average age was 37 years, with a maximum age of 65 years and a minimum age of 23 years. The sign was present in eight of 14 patients referred for cervical MRI and 23 of 32 patients referred for cerebral MRI.

Review of CSF Dynamics

CSF is a dynamic and circulating medium whose flowing character and biologic functions justify its description as "the third circulation." In the average normal adult there are about 135 ml of CSF, 35 ml of which are intraventricular. In terms of appearance, specific gravity, and chemical composition, CSF closely resembles an ultrafiltrate of plasma. CSF is formed at the rate of about 0.4 ml/min [4] by choroid plexus and ventricular ependyma [5]. There is bulk flow of CSF from sites of origin to sites of absorption. The fluid that is formed in the lateral ventricles passes out through the paired interventricular foramina of Monro to reach the third ventricle. The fluid then flows caudally through the aqueduct of Sylvius and fourth ventricle, where it passes into the subarachnoid space by one of three exits: through the paired lateral foramina of Luschka, which direct fluid around the brainstem into the cerebellopontine angle and prepontine cisterns, and through

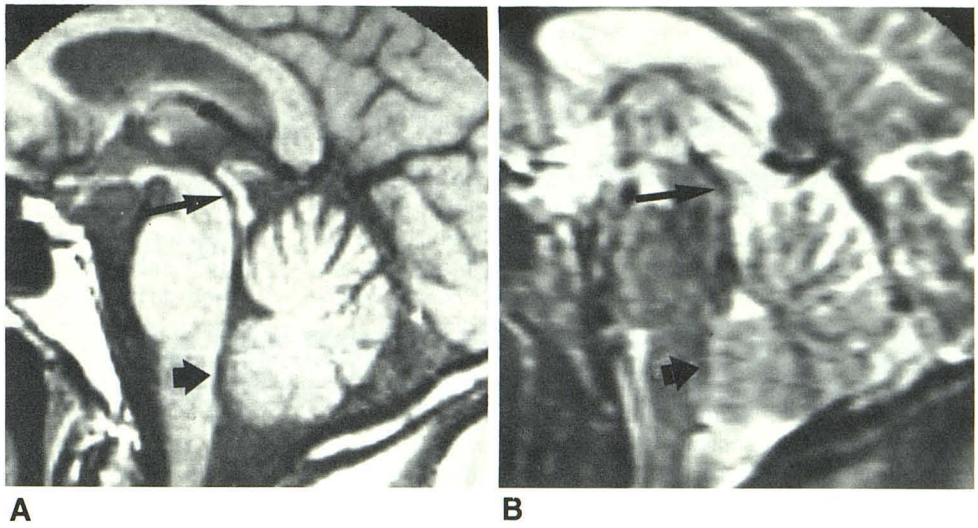

Fig. 4.—T1-weighted and T2-weighted midsagittal images. CSF flow-void sign in aqueduct (*long arrows*) and caudal fourth ventricle (*short arrows*). **A**, SE 850/40. CSF in lateral ventricle is dark gray. **B**, SE 3500/100. CSF in lateral ventricle is very bright.

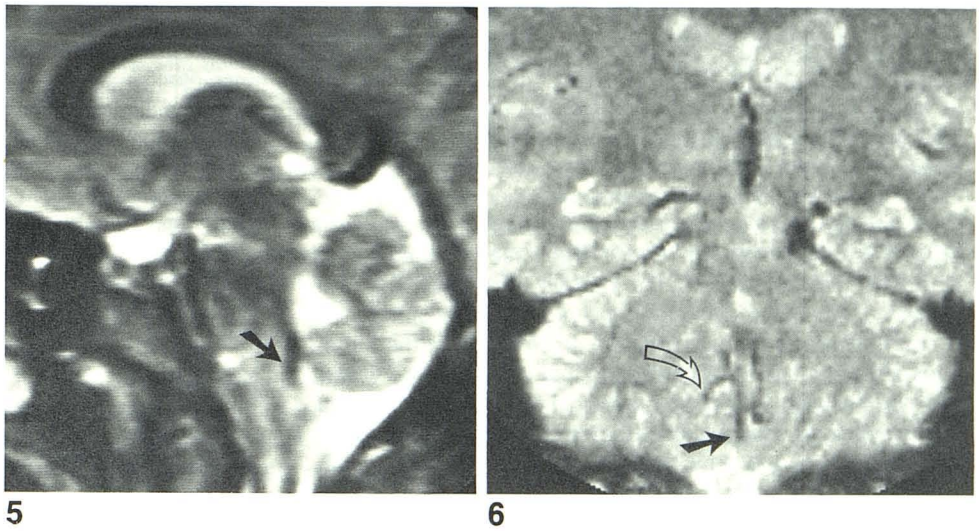

Fig. 5.—T2-weighted midsagittal image, SE 3500/100. CSF flow-void sign in caudal fourth ventricle/foramen of Magendie (*arrow*). Posterior inferior cerebellar artery may contribute to decreased intensity.

Fig. 6.—T2-weighted coronal image, SE 2500/80. Decreased intensity may represent CSF flow-void sign in foramen of Magendie (*solid arrow*) and right foramen of Luschka (*open arrow*) and/or branches of posterior inferior cerebellar artery.

the midline foramen of Magendie in the posterior medullary velum. The foramen of Magendie directs fluid through the vallecula into the cisterna magna. Ultimately the CSF is returned to the blood across the arachnoid villi.

Discussion

Although the mechanisms for propelling CSF along its circulatory route are incompletely understood, the following factors are involved (from Milhorat [5]): (1) the continuous formation of CSF; (2) the ventricular pulsations, related to arterial pulsations; (3) the ciliary action of the ventricular ependyma; and (4) the pressure gradient across the arachnoid villi. Du Boulay [6] made fluoroscopic observations of oily-contrast ventriculography and described a rhythmic third ventricular pumping action as the thalami were squeezed together during systolic brain expansion. This action imparted a to-and-fro motion to the CSF in the third ventricle, aqueduct, and fourth ventricle, although passage of contrast medium down the unobstructed aqueduct was too rapid for reliable observation. He calculated that this pumping action was responsible for displacement of about 0.1 ml of third ventricular CSF per heartbeat [7]. Much more prominent CSF displacement was observed in the basilar subarachnoid spaces, sometimes resulting in CSF being driven into the fourth ventricle from the basal cisterns. Multiplying Du Boulay's data for observed CSF displacement by a normal heart rate of 70–80 beats/min, a to-and-fro flow of 7–8 ml/min (0.12–0.13 ml/sec) is reached in the body of the third ventricle. Lane and Kricheff [8] confirmed the pulsatile nature of CSF flow with videodensitometry and measured deflections of opacified CSF at myelography. They found normal deflection of 3–30 mm/heartbeat in the upper cervical area. If CSF is deflected upward a distance of 10 mm and returns to its neutral position during one heartbeat, then the peak velocity of the deflection can be calculated to approximate 2.6. cm/sec. For the range of 3–30 mm of deflection, the peak velocity is 0.8–7.8 cm/sec.

Several factors are believed to be important in the explanation of the CSF flow-void phenomenon. Bradley et al. [1, 2] described signal loss in flowing systems resulting from high velocity, turbulence, and out-of-phase pulsatile flow. These phenomena also apply to the explanation of the CSF flow-

void sign. When fluid in a larger chamber is forced to flow through a smaller chamber, the velocity increases. This explains the increased velocity of CSF flow in the aqueduct. The aqueduct is an irregular, somewhat funnel-shaped tube constricted at the level of the superior colliculus and the intercollicular sulcus. The average cross-sectional area of the aqueduct varies from 1.85 to 5.1 mm^2 over a length of about 12 mm [8]. An approximation of the aqueductal velocity can be obtained by using the formula $v_1a_1 = v_2a_2$, where v_1 = estimated velocity in the third ventricle (from Du Boulay's measurements), a_1 = cross-sectional area of third ventricle, v_2 = calculated velocity in the aqueduct, and a_2 = cross-sectional area of aqueduct.

The area of third ventricle at the iter is about 100–150 mm^2, whereas the cross-sectional area of the aqueduct is about 3 mm^2. The velocity of the CSF passing through the aqueduct using these figures is 4–6 cm/sec. If the data from Lane and Kricheff [8] are applied, the velocity in the aqueduct is much higher. Mills et al. [3] stated that if moving nuclei traverse the distance of the slice thickness within the imaging sequence time (TE), they will not contribute to the signal. For a 5 mm slice thickness with a TE of 80 msec, the threshold velocity is about 6 cm/sec. The Bradley et al. [1, 2] method of calculating the threshold velocity differs from that of Mills et al. [3] by a factor of 2, thus requiring a threshold velocity of about 12 cm/sec for the same example.

Obviously, these calculations and measurements are approximations and do not include the effect of turbulence. However, they do support the intuitive impression that the velocity of the CSF moving from the larger volume of the third or fourth ventricle through the very small lumen of the aqueduct must increase significantly and that this increased velocity, together with turbulence, is visible on the MR image as an area of signal dropout. No attempt was made to determine the direction of the flow, but it is believed that the CSF pulsates in a to-and-fro manner and that the sign will be present regardless of the direction of the flow.

The CSF flow-void sign in the caudal fourth ventricle probably is caused by the same forceful pulsatile to-and-fro flow, as CSF in the cisterna magna and fourth ventricle is subjected to compression by the systolic expansion of the brain and meninges. Fourth ventricular choroidal CSF production may also contribute to this phenomenon. Some authors have speculated that the choroid plexus of the fourth ventricle produces more CSF than the lateral ventricular choroid plexus [9]. The sign is regularly seen in the midline (figs. 1, 3A, 4, 5, and 6), indicating that the preferential flow of CSF is through the foramen of Magendie, although possible flow through the foramina of Luschka (fig. 6) was observed in one case. Occasionally, it is seen extending along the length of the fourth ventricle in continuity with the aqueduct (fig. 1). In these cases it has been observed only along the floor of the fourth ventricle, apparently identifying a channel of more rapid flow separated from less turbulent or relatively stagnant CSF in the body of the fourth ventricle, the fastigial recess, and lateral recesses. The sign also has been observed near the foramina of Monro (fig. 1) but has not been seen in the anterior recesses of the third ventricle or in the lateral ventricles.

The CSF flow-void sign was differentiated from the low signal intensity of arteries and veins by comparing the location of the sign with known vascular anatomy. For example, there is no known vessel paralleling the course of the aqueduct of Sylvius. Size is also a useful differentiating point, particularly in differentiating the CSF flow-void sign from vessels around the brainstem.

The failure to observe the sign in some patients could be related to a number of different factors not evaluated by this study. The aqueduct of Sylvius can have a diameter as small as 0.63 mm, resulting in partial-volume averaging with higher-intensity periaqueductal gray matter. The location of the slice within a multislice examination may be important, since inflow of relatively unsaturated protons can result in paradoxic enhancement most intense in the first section of a multisection study [10]. Velocity of the pulsatile flow is related to heart rate and cardiac output. The pulsating CSF may not reach the threshold velocity in patients with arrhythmia or bradycardia and would, therefore, appear isointense or even hyperintense relative to the CSF in the lateral ventricles.

The full explanation of the biophysics of the CSF flow-void phenomenon will require additional study. However, we believe that the observation of this sign is important in the routine interpretation of MRI studies and represents another facet of the multidimensional capabilities of MRI in neurologic evaluation.

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