# The MR Appearance of CSF Pulsations in the Spinal Canal

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**We investigated the MR appearance and incidence of low-signal areas within the CSF of the spinal canal. Nonuniform areas of decreased signal intensity in intracranial CSF have been named the CSF flow-void sign (CFVS) and appear to be due to spin dephasing secondary to pulsatile CSF motion. Similar areas are seen in the spinal canal. The MR scans of 50 randomly selected patients, constituting a total of 63 spinal studies, were reviewed. There were 27 cervical, 16 thoracic, and 20 lumbar spine examinations. All patients were studied using T2-weighted and T1-weighted spin-echo pulse sequences. T2-weighted images were done with sufficiently long TE and TR to cause the CSF to appear hyperintense compared with brain and spinal cord tissue. Two patients with enlarged spinal canals and two patients with syringohydromyelia were also included to illustrate the appearance of prominent CSF pulsations.**

**The CFVS was identified on T2-weighted scans in the cervical spinal canal in nine patients (33%), in the thoracic spinal canal in one patient (6%), and possibly in the lumbar spinal canal in two patients (10%). The CFVS was prominent in two patients with enlarged CSF spaces and was also seen in the intramedullary cavity of the patients with syringohydromyelia.**

**The CFVS could obscure small dural lesions and, in some instances, simulate enlarged vessels. Recognition of the spinal CFVS is important to avoid the incorrect diagnosis of intraspinal lesions.**

MR of the spine is frequently performed using both T1-weighted and T2-weighted pulse sequences. T2-weighted sequences have been advocated in which a long TR and long TE are employed in order to make the CSF appear hyperintense relative to the spinal cord, providing an image comparable to a myelogram [1, 2]. Using this type of sequence, we have noted inhomogeneity in the appearance of CSF in some patients. We have encountered this phenomenon more often in the upper cervical region and in patients with spinal canal enlargement. The same phenomenon has been observed in some syringohydromyelic cavities. The purpose of this report is to document and illustrate the appearance of these areas of decreased signal intensity in randomly selected patients and in a small set of selected patients with abnormalities of the spinal canal or spinal cord.

## Subjects and Methods

Sixty-three MR examinations of the spines of 50 patients were randomly selected and reviewed for the presence of areas of anomalous decreased signal in the CSF, which has been referred to as the CSF flow-void sign (CFVS) [3, 4]. Subjects consisted of 26 females and 24 males ranging in age from 3 to 74 years; average age, 47 years. There were 27 examinations of the cervical spine, including 14 patients with normal examinations, three with canal stenosis, and 10 with spondylosis or disk herniation. There were 16 examinations of the thoracic spine, one of which showed a herniated disk. And there were 20 examinations of the lumbar spine, of which four patients had spinal stenosis, nine had disk bulging or disk herniation, and seven were normal.

Four additional cases that were not part of the randomized study have been included as

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examples of prominent CSF flow-void signs. These included a 6-year-old boy with a meningocoele; a 39-year-old man with an enlarged cervical spinal canal after laminectomy and removal of a spinal cord tumor; and two patients with syringohydromyelia, one a 23-year-old man and the other a 44-year-old woman. The man had cerebellar tonsillar herniation of 13 mm and a syrinx cavity that extended from C1 to T8. The woman had small dysplastic cerebellar tonsils and foramen magnum stenosis. The syrinx cavity extended from C2 to T1.

MR examinations were made using a 0.5-T superconductive magnet (Vista-MR, Picker International Corp., Highland Heights, OH). Standard, manufacturer-provided, single-echo, eight multisection spin-echo (SE) sequences were done with selective excitation in staggered slice order by exciting odd-numbered slices sequentially followed by even-numbered slices. Cardiac gating was not used. The sections were contiguous and of either 5- or 10-mm thickness. Slice profiles weree approximately trapezoidal, with 1-mm transitional zones on either side of a flat region of the nominal thickness. Data were typically acquired with either 256 or 128 complex samples/view, 256 views, and two excitations using the 2 DFT method. The resultant 256 × 256 or 128 × 256 image was then interpolated to a 512-element display matrix. Field of view for these examinations was 30 cm, resulting in a pixel size (before interpolation) of 1.17 × 1.17 mm or 1.17 × 2.34 mm.

T2-weighted sequences were done with a TE of 80 or 100 msec and a TR of 2000–3000 msec. T1-weighted sequences employed a TE of 30 or 40 msec with a TR of 500–800 msec. The CSF appears hyperintense or isointense relative to the spinal cord on the T2-weighted sequences and hypointense on the T1-weighted sequences. All patients were studied with both T2-weighted and T1-weighted pulse sequences in the sagittal planes. The section thickness was 5 mm for all cervical studies and for all T1-weighted thoracic and lumbar examinations. Nine thoracic and five lumbar T2-weighted studies were done with a 10-mm section thickness. The remainder of the thoracic and lumbar studies used a 5-mm section thickness. All cervical examinations included 5-mm or 10-mm transaxial sections using an SE 1500/30 sequence. All lumbar examinations included 5-mm T2-weighted transaxial sections in addition to the sagittal studies. The midline sagittal sections were in either the number 4 or 5 position of the multisection set of images.

## Results

Nine of the randomly selected 27 patients with cervical MR had the CFVS in the upper cervical canal. In several other patients the CFVS may have been present but we could not differentiate the sign reliably from ligaments along the anterior margin of the canal, especially below the C3 level. The CFVS was best seen on the T2-weighted images but was usually visible on the T1-weighted images as well when careful comparison was made with the CSF in the ventricles. In all these patients the sign was seen in the upper levels, near the foramen magnum. Five of the nine patients had the CFVS in the upper posterior cervical canal while four had the sign in the upper anterior cervical canal (Fig. 1). Of these nine patients, six had normal examinations and three had evidence of spondylosis or disk herniation. The length of the sign varied from 5–20 mm.

Two additional patients with cervical spine MR were included in the study but not in the randomized group. In the 39-year-old man who had undergone a laminectomy from C1 to T1, the spinal canal was hyperlordotic and expanded. The anterior spinal subarachnoid space was voluminous. The CFVS was present at the upper and lower aspects of the canal anteriorly (Fig. 2) and was best seen on the SE 1500/60 sequence. The 44-year-old woman with syringohydromyelia had a spinal cavity that was largest from C4 to T1. The SE 500/40 sequence clearly showed the large "beaded" cavity and the small extension to the C2 level (Fig. 3). The ventricles were moderately dilated. When the SE 2200/80 was done, the CSF in the ventricles and cervical canal appeared isointense relative to the brain. The fluid in the largest part of the syrinx cavity was hypointense. The upper cervical spinal cord appeared mildly hyperintense, probably due to gliosis (no surgery has been performed).

Only one of the randomly selected 16 patients with T2-weighted thoracic MR had evidence of the CFVS in the spinal canal. In this patient the decreased signal was seen posterior

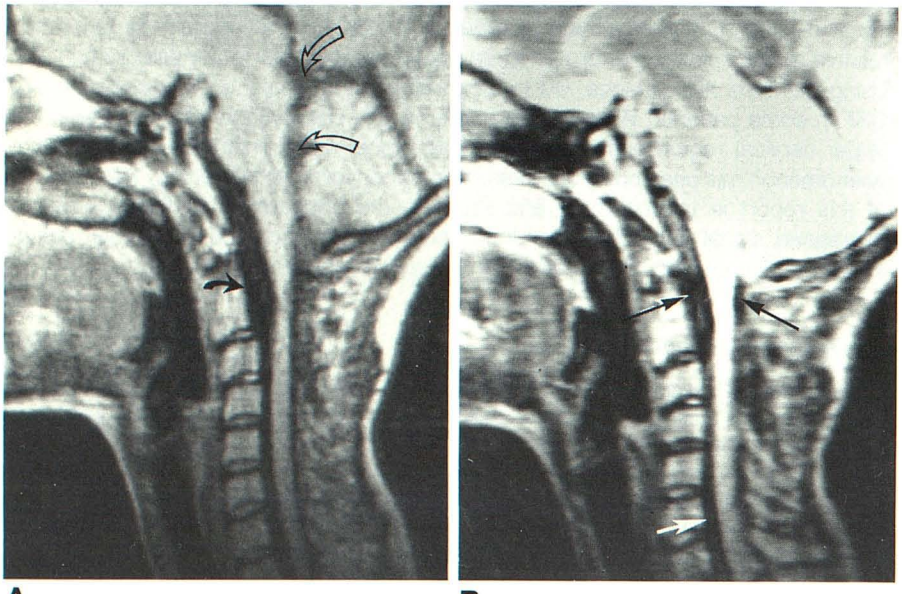


Fig. 1.—CSF flow-void sign present in normal cervical spinal canal. Midline sagittal images. **A**, SE 800/40. Note wide subarachnoid space at C1–C2 level (*solid arrow*) and lower intensity of CSF compared with fourth ventricle and quadrigeminal plate cistern (*open arrows*). **B**, SE 2000/80. CSF in ventricles is bright. CSF flow-void sign is seen anterior and posterior to cord at C1–C2 level (*black arrows*). Thick black line at C6 level probably represents CSF flow-void sign (*white arrow*).

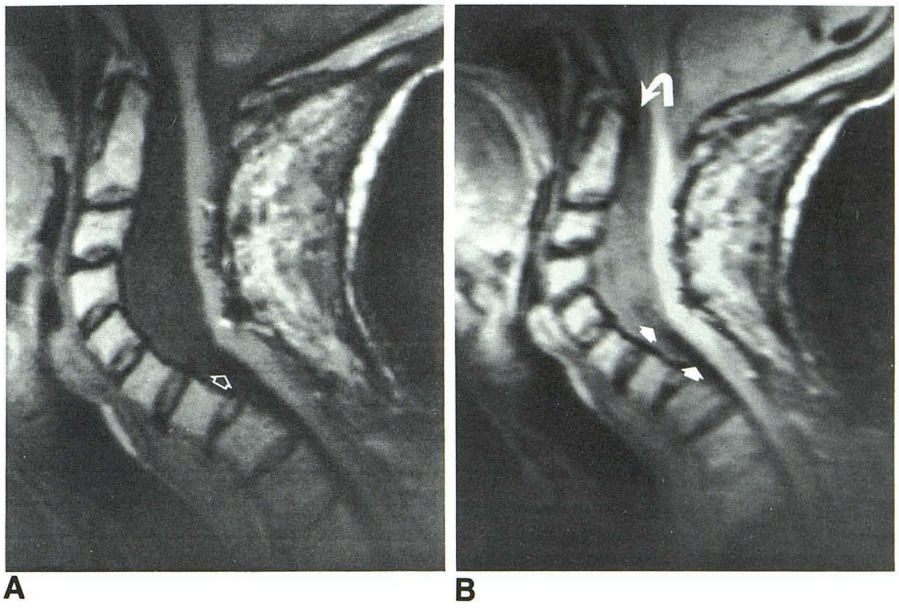


Fig. 2.—CSF flow-void sign present in enlarged cervical spinal canal. Patient has had a laminectomy from C1 to T1. Note exaggerated lordosis and large anterior CSF space. Spinal cord is adherent posteriorly. Midline sagittal images. **A**, SE 800/40. CSF flow-void sign can be faintly seen as an area of darker CSF anterior to cord at C7 level (*arrow*). **B**, SE 1500/60. Prominent CSF flow-void sign is seen anterior to lower cervical spinal cord (*short arrows*) and near cervicomedullary junction (*curved arrow*). Note heterogeneous CSF intensity in enlarged anterior CSF space compared with cisterna magna.

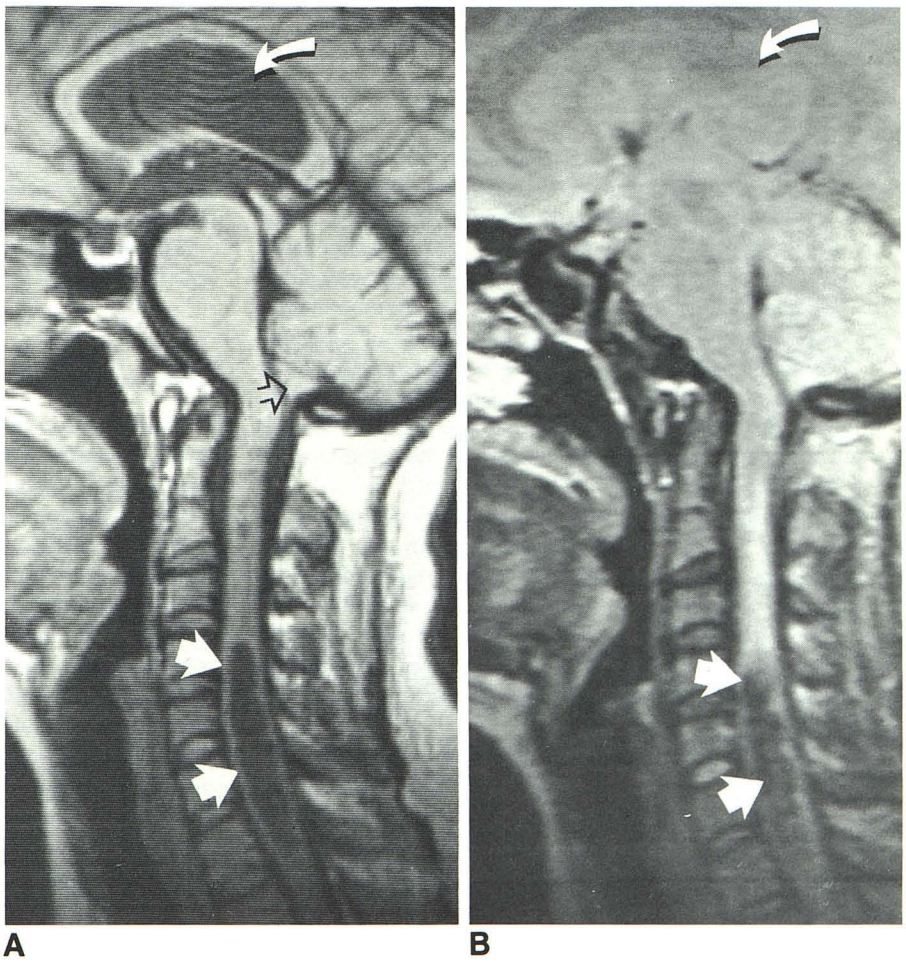


Fig. 3.—44-year-old woman with syringohydromyelia. Midline sagittal images. 5-mm-thick sections. **A**, SE 500/40. CSF in lateral ventricle is hypointense relative to brain (*curved arrow*). Syrinx cavity is largest from C4 to C7 (*large arrows*) but thin rostral extension is noted. **B**, SE 2200/80. CSF in lateral ventricle is isointense relative to brain (*curved arrow*). Fluid in largest portion of the syrinx cavity remains hypointense (*large arrows*). Dysplastic cerebellar tonsil (*open arrow*).

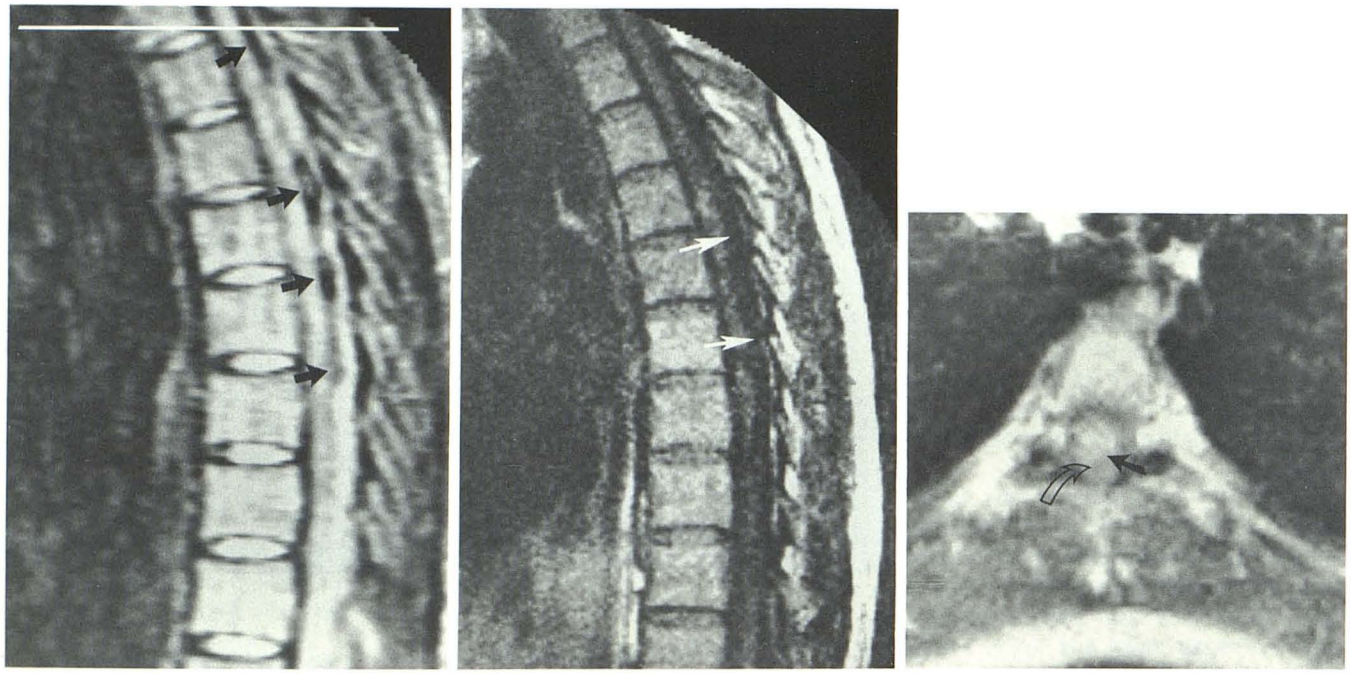


Fig. 4.—CSF flow-void sign present in normal thoracic spinal canal. **A**, SE 2500/80. Midline sagittal image, 5-mm-thick section. CSF flow-void sign is seen in posterior thoracic spinal canal (*arrows*). Location of axial section indicated by soild white line. **B**, SE 300/26. Midline sagittal images, 5-mm-thick section. Posterior CSF space appears larger than in **A** (*arrows*). **C**, SE 2000/40. 10-mm-thick axial section through upper thoracic spine. CSF flow-void sign is not seen. Spinal cord appears brighter than surrounding CSF. Posterior margin of spinal cord (*solid arrow*). CSF posterior to spinal cord (*open arrow*). Note absence of enlarged vessels.

to the upper and middle thoracic spinal cord on an SE 2500/80 sequence (Fig. 4). The CFVS was seen in the area where the spinal canal was largest. The sagittal SE 300/26 pulse showed the prominent posterior CSF space. Axial scans through the area using an SE 2000/40 sequence were done to investigate the possibility of a dural arteriovenous malformation. The signal of the CSF was increased but remained less than that of the spinal cord. There was no evidence of enlarged vessels or of the CFVS using the SE 2000/40 sequence. The CFVS was not seen in the thoracic canal in the other patients, although the margin of the canal was at times indistinct, possibly related to CSF motion.

The CFVS was present in the intramedullary cavity of a 23-year-old man with syringohydromyelia on an SE 2000/80 sequence (Fig. 5). The intensity of the cavity was identical to that of surrounding CSF on an SE 800/30 sequence. The syrinx cavity extended from C1 to T8. This patient had a Chiari type I malformation with 12 mm of cerebellar tonsillar ectopia, but he has not undergone surgery. He was not part of the randomized study group.

Two of the 20 randomly selected patients with lumbar MR had subtly decreased signal anterior to the conus medullaris, which may represent the CFVS. Nerve roots of the cauda equina were seen as linear undulating areas of decreased intensity and would therefore make recognition of the CFVS more difficult below the level of the conus medullaris. An 8-year-old girl with lumbar spinal dysraphism was included in the study but not as part of the randomized group. The volume of the spinal canal was markedly expanded in the lumbar region. The CFVS was present as a fan-shaped area of decreased signal in the upper lumbar canal, anterior to the posteriorly tethered spinal cord (Fig. 6).

## Discussion

The bulk flow of CSF follows a downward route behind the cord, posterior to the dentate ligaments, to the lumbar area, and upward in front of the cord to the basilar cisterns [5, 6]. Pressure waves are generated by distention and collapse of the cerebrovascular and spinovascular beds and are felt to be responsible for CSF pulsations [7]. As in the case of blood flow, where the propagation of the pulse wave is independent of and much faster than the velocity of the blood [8], the propagation of a CSF pulse wave is much more rapid than the actual movement of CSF. Pulsatile movements of CSF are greatest in the cervical region, diminishing considerably in the lower thoracic and lumbar canal [9, 10]. In the cervical region, the downward flow begins during systolic aortic expansion and ceases during diastole [9]. Displacement of CSF from the cranium into the upper cervical canal is of 10 times greater volumetric magnitude than the ventricular pulse, and is mainly responsible for pulsatile movements in the upper cervical area [9].

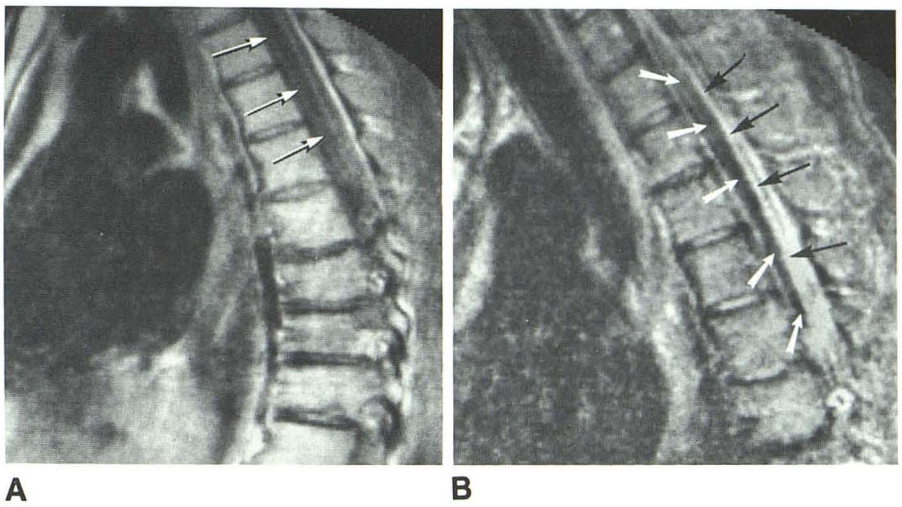


Fig. 5.—23-year-old man with syringohydromyelia. Intramedullary cavity extends from C1 to T8. Midline sagittal images, 10-mm-thick sections. **A**, SE 800/30. Syrinx cavity occupies most of diameter of the spinal cord. Anterior margin of cavity (*arrows*). **B**, SE 2000/80. Note low intensity of syrinx cavity (*white arrows*) compared with subarachnoid space. Posterior margin of spinal cord (*black arrows*).

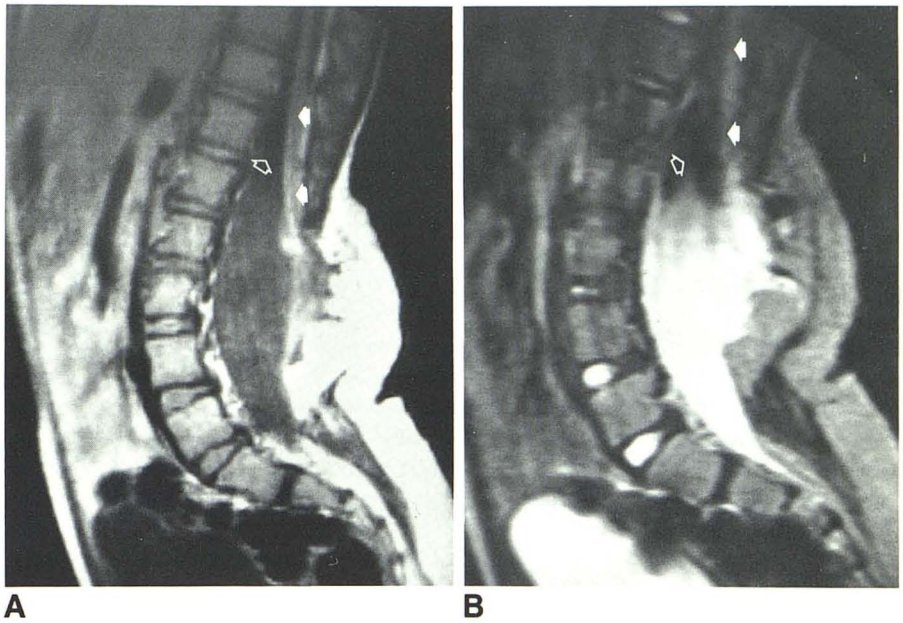


Fig. 6.—8-year-old girl with lumbar lipomeningocele and tethered spinal cord. Note markedly enlarged CSF space. **A**, SE 800/30. Midline sagittal image, 5-mm-thick section. Spinal cord is tethered posteriorly (*solid arrows*). Note decreased intensity of CSF (CSF flow-void sign) anterior to spinal cord (*open arrow*). **B**, SE 3500/80. Midline sagittal images, 5-mm-thick section. Spinal cord (*solid arrows*). Note CSF flow-void sign appearing as a fanlike area of decreased intensity in CSF space of upper lumbar area (*open arrow*).

The MR appearance of CSF motion in the brain has been described [3, 4, 11, 12]. CSF motion appears as an area of decreased signal intensity compared with the CSF in the lateral ventricles. The loss of signal due to motion within CSF spaces, such as the aqueduct of Sylvius, has been referred to as the CSF flow-void sign (CFVS) [3, 4]. The signal loss represented by the CFVS is probably caused by a combination of phase shift, view-to-view variation, and time-of-flight effects [13, 14]. In the case of spins in the CSF along a magnetic gradient, such as in slice selection, the magnetic field experienced by those spins varies during the evolution of the pulse sequence. Thus, the magnetic history of the spins will be more complex in the case where movement of spins occurs. The signal intensity is critically dependent on the spatial distribution of velocities and accelerations of spins within a voxel. The larger the spatial variation of the velocity or accelerations across a voxel, the greater the loss in signal amplitude [13]. However, the relationship between flow velocity and signal loss in conventional SE sequences is nonlinear [15]. Turbulence, whether due to high velocity or intrinsic physiological movements, results in large spatial variations within the imaging plane (phase-shifts) and therefore does not return a strong signal. This effect occurs for motion within the imaged plane as well as for motion perpendicular to the image plane. Spin-echo sequences in which the CSF is hyperintense relative to the brain are more likely to reveal the areas of signal loss within the CSF simply because of increased contrast.

The CFVS is most prominent in areas in which CSF must pass from a space of larger volume through a narrow channel,

such as the aqueduct of Sylvius or the foramen of Magendie [11]. These are areas in which the velocity of the pulsatile wave probably increases, resulting in less signal, due to time-of-flight effects, and an accentuation of spin-phase shifts, due to increased turbulence [13]. Citrin et al. [16] have recently reported that the appearance of the CFVS is enhanced if cardiac gating is used and data are acquired during cardiac systole.

The appearances of CSF movements in the spinal canal are more subtle than in the brain since there is less contrast between the low intensity of the CFVS, adjacent vertebral ligaments, and cortical bone. The sign was prominent in the patient with an enlarged lumbar canal due to spinal dysraphism and in another patient with an enlarged cervical canal after a laminectomy and removal of a tumor. In both patients the appearance of a "jet" of CSF was identified in the canal where the cross-sectional area was tapering.

In our series of randomly selected patients, the CFVS was visible in the upper cervical area in one-third of patients, was possibly present in the lumbar area in two patients, and was visualized in the thoracic area in only one patient. This correlates well with myelographic observations of CSF pulsations by previous authors, who found that CSF pulsations were most frequently seen and were of the greatest amplitude in the cervical region [9, 10]. We must note, however, that linear areas of decreased intensity paralleling the anterior or posterior longitudinal ligaments would be difficult to detect and could affect the sharpness of the border of the spinal canal.

We noted evidence of CSF movements within the intramedullary cavities of the two patients with syringohydromyelia. The presence of the CFVS wtihin the syrinx cavities enhanced the visibility of the cavities on the T2-weighted sequence. The observation of the CFVS within the syrinx is not surprising since fluid shifts within the cavity of a syrinx have been postulated as a mechanism of syrinx propagation [17–19]. At this time we are uncertain of the significance of the observation of the CFVS within a syrinx cavity. This will be the subject of further study and a future report.

In summary, we believe that recognizing and understanding the effects of CSF motion in the spinal canal is important in order to understand the range of intensities presented on the MR image. CSF pulsations are an important source of heterogeneity in CSF on MR images as well as a potential source of error in determining the diameter of the spinal canal. Small extradural masses of low signal intensity, such as meningiomas, could be obscured by the CFVS. Prominent CSF motion effects could simulate dural vascular malformations. Finally, recognition of normal and abnormal patterns of CSF motion in the spinal canal and in lesions such as syringohydromyelia may lead to more complete understanding of the disease processes involved.

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