AJNR

Importance of absence of CSF pulsation artifacts in the MR detection of significant myelographic block at 1.5 T.

D J Quint, S C Patel, W P Sanders, D O Hearshen and R S Boulos

Importance of Absence of CSF Pulsation Artifacts in the MR Detection of Significant Myelographic Block at 1.5 T

Douglas J. Quint[1]
Suresh C. Patel[2]
William P. Sanders[2]
David O. Hearshen[2]
Roushdy S. Boulos[2]

MR imaging was performed on 21 patients who had high-grade myelographic block due to various diseases in all spinal compartments (extradural, intradural/extramedullary, and intramedullary) and in all portions of the spinal canal (cervical, thoracic, and lumbosacral). Loss of CSF pulsation artifacts due to significant compression of the spinal cord was demonstrated on non-motion–compensated T2-weighted examinations in each case. We believe that the absence of such artifacts on these sequences indicates significant spinal cord compression in patients without classic signs and symptoms of cord compression but with intraspinal disease identified on T1-weighted studies.

MR imaging is rapidly becoming the imaging technique of choice for evaluation of the spinal canal and its contents. In particular, owing to the superior contrast resolution of MR, intraspinal disease can be directly visualized whereas, in the past, it could only be inferred with myelography and inconsistently visualized with noncontrast CT. However, MR imaging is still a tomographic technique; and while multiplanar imaging overcomes much of the partial volume effects of tomographic imaging, like CT, the finite thickness of imaged sections (usually 3 or 5 mm) can make it difficult to differentiate between intraspinal disease associated with spinal cord compression and that not associated with compression.

Absence of CSF pulsation artifacts on MR below the level of spinal cord compression, caused by absence of transmission of CSF pulsations beyond the level of spinal cord compression, is well known [1] and could potentially differentiate between a compressive and noncompressive intraspinal process [2, 3]. We evaluated 35 patients with significant spinal cord compression (e.g., high-grade block) with MR imaging in an attempt to determine if absence of CSF pulsation artifacts on a T2-weighted sequence (performed without motion compensation techniques) is a consistent finding and could reliably predict a high-grade myelographic block.

Subjects and Methods

Thirty-five patients (17 males and 18 females, 11 to 64 years old) were studied. Entrance into the study required demonstration of a high-grade block at myelography (e.g., no passage of intrathecal water-soluble contrast solution past an area of intraspinal abnormality with the patient in a 60° Trendelenberg position). Complete myelography was ultimately performed in each case by either next performing a C1–C2 puncture and completing the myelogram from above or by "pushing" past the block by transiently increasing the intrathecal pressure below the level of the block with rapid injection of 6 ml of sterile saline into the lumbar spinal needle. In one case (a grade IV L5–S1 spondylolisthesis), no contrast was demonstrated below the level of the block.

All patients were imaged on a GE 1.5-T superconducting magnet with the use of two-dimensional Fourier transform reconstruction techniques. A circular surface coil with a diameter of 13.2 cm was centered over the area of abnormality demonstrated at myelography.

Multisection spin-echo (SE) imaging was performed in the sagittal plane utilizing T1-weighted 600–800/25 (TR range/TE) and T2-weighted (2000–2500/25–30, 80–90) sequences. The T2-weighted examinations were performed without motion compensation techniques and then were immediately repeated with motion compensation in the form of cardiac gating (peripheral finger gating, photoplethysmography, RW-1*). On several of the later examinations, in addition to cardiac gating, gradient moment nulling ("flow compensation") was employed. As the cardiac-gated examinations are dependent on heart rate, the effective TR in these cases ranged from approximately 1700 to 2200. On T1-weighted examinations, imaging parameters included a section thickness of 5 mm with no interslice gap, four excitations, either a 256 × 128 or 256 × 192 matrix, and a 20-cm field of view. On T2-weighted examinations, imaging parameters included a section thickness of 3 mm with a 1-mm interslice gap, two excitations, either a 256 × 192 or 256 × 256 matrix, and a 20-cm field of view. No axial or low flip angle sequences were used.

The 35 subjects were not imaged consecutively, as some patients had surgery immediately after myelography, some patients refused MR, and in some cases the MR scanner was not immediately available. All 35 patients included in this study were imaged within 48 hr of myelography, though in some cases steroid or radiation therapy was begun in the interim. For comparison, normal volunteers had MR imaging of their cervical, thoracic, and lumbosacral spines, with sequences identical to those outlined above.

Underlying disorders included extradural, intradural, and intramedullary processes. MR examinations were reviewed first for overall interpretability. Fourteen of the 35 patients had nondiagnostic examinations, primarily because of excessive patient motion during the study. Of the 21 examinations considered interpretable, the T1-weighted sequences were evaluated for normal and abnormal spinal and spinal canal structures. The T2-weighted studies were examined for the presence of CSF pulsation effects (e.g., CSF/cord conspicuity, misregistration "ghosting" artifacts, and the homogeneity and overall signal intensity from the spinal CSF). Comparisons were made with the studies performed on the normal volunteers.

Results

The studies from normal volunteers demonstrated that the spinal cord and surrounding structures are best seen on the T1-weighted sequences. The T2-weighted studies performed without motion compensation techniques (Figs. 1A and 1C) demonstrated CSF pulsation artifacts that made it difficult to evaluate the CSF/cord interface and structures anterior and posterior to the spine. The artifacts were most prominent in the cervical and thoracic region and inconsistent in the lumbar region. Implementation of motion compensation techniques (either cardiac gating alone or cardiac gating with gradient moment nulling) (Figs. 1B and 1D) significantly reduced or completely eliminated these artifacts, resulting in improved CSF/cord conspicuity and homogeneously increased signal intensity from the CSF.

All 35 patients demonstrated a high-grade block to the flow of intrathecal contrast. In 34 patients, the block was above the level of the (lumbar) puncture and completion of the myelogram demonstrated no additional lesions. In one case, a grade IV spondylolisthesis at L5–S1, no contrast passed into the distal caudal sac below the level of the block. Lesions

in all regions of the spinal canal (cervical, thoracic, lumbosacral) and all spinal compartments (extradural, intradural, intramedullary) were evaluated (Table 1).

Twenty-one patients had MR examinations that were considered interpretable. In each case (Figs. 2–7), an abnormal intraspinal process was identified on the T1-weighted sequence. Also, in each case, on the non–motion-compensated T2-weighted sequences, absence of pulsation artifacts in the region of the intraspinal abnormality (as manifest by improved CSF/cord conspicuity, homogeneous increased signal intensity from the CSF, and absence of ghosting artifacts) was noted. Finally, no further improvement of image quality was noted when the T2-weighted scans were repeated with motion-compensation techniques.

Fourteen examinations were considered uninterpretable. Reasons included large patient size (two patients), machine malfunction (one patient), and excessive patient motion (11 patients). Many patients, despite pain medication, were unable to tolerate lying motionless in the scanner for the time necessary to perform the T2-weighted examinations.

Discussion

Pulsatile movement of CSF is a well-described phenomenon [1, 2, 4]. MR imaging is sensitive to motion [5–7] so it is not surprising that such motion should give rise to artifacts that degrade image quality. When two-dimensional Fourier transform reconstruction techniques are employed, the periodic pulsatile CSF motion results predominantly in two artifacts: (1) local signal loss from CSF, which obscures the spinal cord/CSF and nerve root/CSF interfaces and (2) misregistration "ghosting" artifacts, which appear as lines of increased and decreased signal intensity along the phase-encoding gradient direction (artifact usually parallels the spinal cord) that degrade the entire image in a periodic fashion [7, 8] (Fig. 1). These motion-dependent artifacts are most prominent on T2-weighted sequences, and various techniques for overcoming them have been developed. Such motion-compensation techniques include image acquisition during diastole (cardiac gating) and gradient moment nulling ("flow compensation," "gradient refocussing"). With these techniques, pulsation artifacts can be significantly diminished or completely eradicated, resulting in improved image quality (Fig. 1).

At myelography, an intraspinal process that compresses the thecal sac to the point of causing a block to the flow of intrathecal contrast dampens CSF pulsations above the level of compression and eliminates them below that level [1]. Therefore, on MR, pulsation artifacts in the region of such a process should be diminished or absent and the resultant image, being relatively artifact-free, should appear similar to an examination performed with motion-compensation technique. We observed this in all 21 of our patients whose examinations were considered interpretable. We believe that the finding of unexpectedly good image quality (lack of local signal loss and phase-misregistration artifacts) on a T2-weighted examination performed without motion compensation should have the same implications for surgical and/or therapeutic radiation intervention as a high-grade myelographic block.

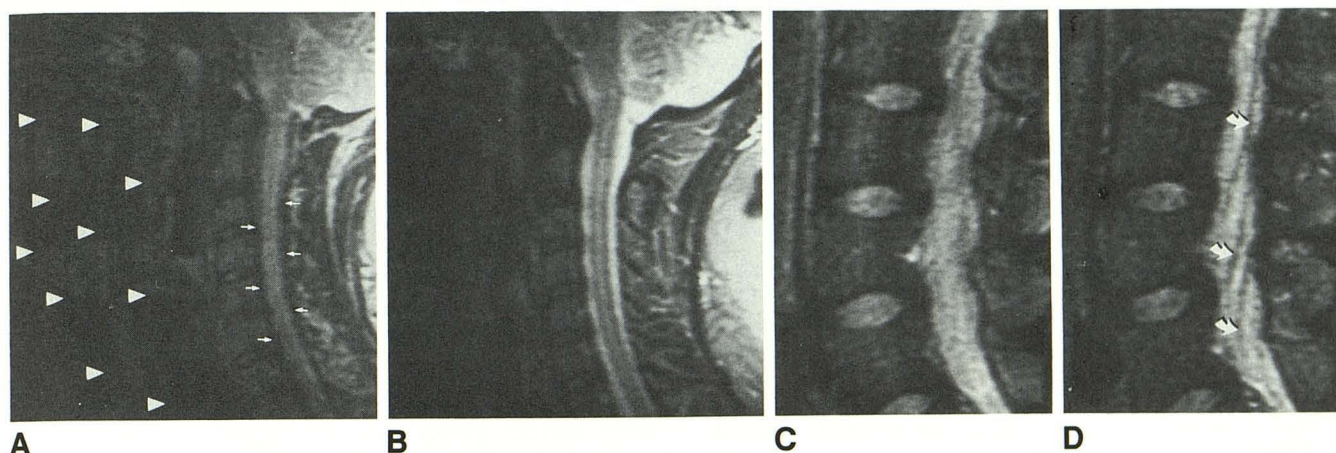**A**   **B**   **C**   **D**

Fig. 1.—Normal spine—effects of cardiac gating.

A, On this nongated T2-weighted cervical study (2000/90), extensive CSF pulsation artifact is manifest as both local signal loss at interface between spinal cord and CSF (*arrows*) and also as periodic "ghosting" artifacts (*arrowheads*) projecting anteriorly, posteriorly, and directly onto the spinal cord.

B, With cardiac gating (1875/80), these artifacts disappear, the CSF takes on its expected homogeneous increased-intensity appearance, and interface between spinal cord and CSF is well-defined. A normal truncation artifact is seen in center of cord.

C, Nongated T2-weighted lumbosacral study (2000/80) reveals high signal from CSF in caudal thecal sac. No significant "ghosting" or signal loss artifacts are seen. No thecal nerve roots are resolved.

D, Gated examination (2000/80) demonstrates improved spatial resolution (visualization of nerve roots, *curved arrows*) presumably due to decreased CSF pulsation artifact.

TABLE 1: Cases of High-Grade Myelographic Block Confirmed by Identifying Loss of Normal CSF Pulsation Artifacts On Non-motion–compensated T2-weighted MR

Disease	Level		
	Cervical	Thoracic	Lumbosacral
Astrocytoma	3	–	–
Metastases	2	8	3
Neurofibroma	1	1	–
Spondylosis	2	–	–
Spondylolisthesis	–	–	1

The T2-weighted examinations are used to determine if artifacts are absent for several reasons. First of all, CSF (long T2) demonstrates increased signal intensity on these sequences and therefore local signal loss from moving CSF is apparent (CSF flow void sign [8]). On T1-weighted examinations, CSF (long T1) demonstrates low signal intensity and therefore local signal loss due to motion is not visible. Second, the greater signal-to-noise ratio of T1-weighted sequences makes phase-misregistration artifacts less obvious; therefore, the absence of such artifacts would be of less significance.

There appears to be general agreement that the T1-weighted examinations are best for evaluating morphology and demonstrating the presence of intraspinal disease. In fact, it is probable that in most cases multiplanar T1-weighted MR can demonstrate intraspinal disease compressing the thecal sac and spinal cord obviating the need for any other sequences (or more invasive studies) particularly in patients with appropriate correlative symptoms [9, 10]. However, many patients present with more nonspecific complaints (e.g., oncology patients with back pain, but no localizing signs) and have intraspinal disease that may or may not be the cause of
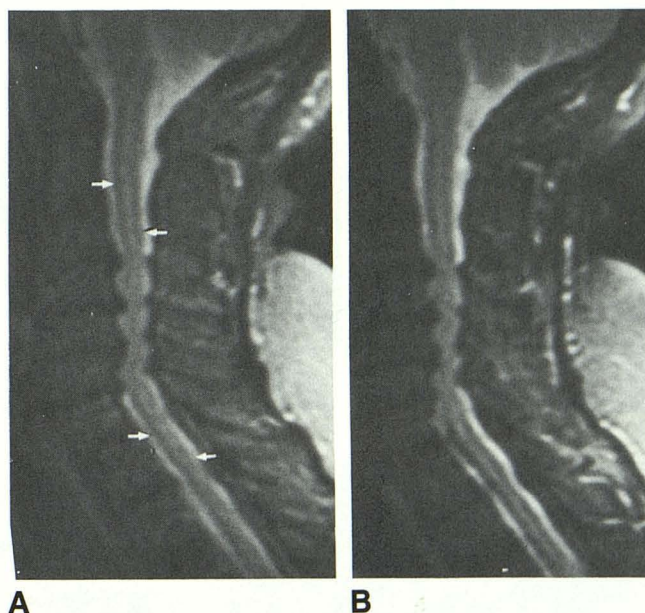

A   **B**

Fig. 2.—Spondylosis. 74-year-old woman with severe neck pain. Plain films and myelography revealed extensive spondylosis with a high-grade block in lower cervical region.

A, Nongated T2-weighted image (2000/90) demonstrates absence of pulsation artifacts with excellent visualization of CSF/spinal cord interface (*arrows*).

B, No further improvement in image quality is seen on the gated (2000/90) examination.

symptoms and may or may not be causing spinal cord compression. For example, a patient with metastatic carcinoma previously treated with radiation therapy for spinal canal disease could present with recurrent back pain due to recur-
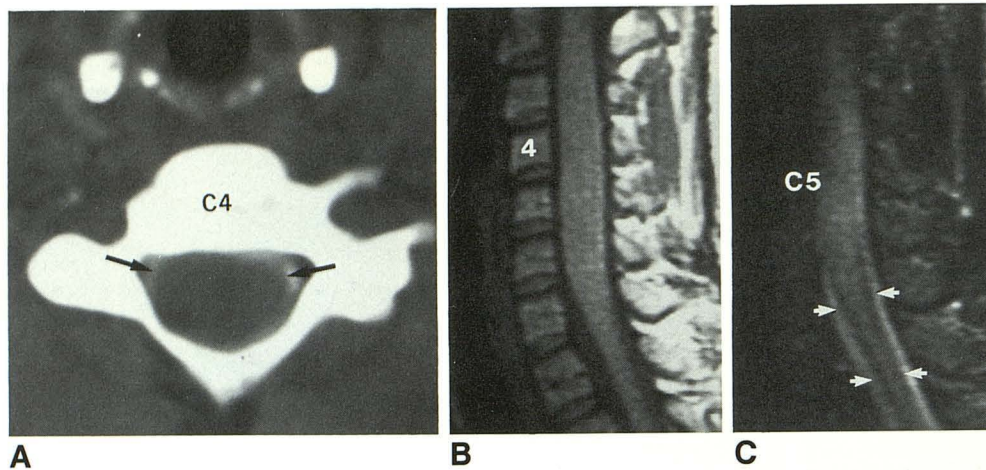
Fig. 3.—Astrocytoma. 33-year-old woman with C6 radicular symptoms.
A, Myelogram with CT reveals expansion of cervical spinal cord causing attenuation of subarachnoid space (*arrows*).
B, T1-weighted MR image (600/25) reveals diffuse enlargement of cervical cord.
C, Nongated T2-weighted image (2000/90) demonstrates increased signal from enlarged cervical cord and improved CSF/cord resolution (*arrows*). Excellent CSF/spinal cord conspicuity confirms dampening of CSF pulsation artifacts consistent with significant myelographic block. Biopsy revealed grade II astrocytoma.
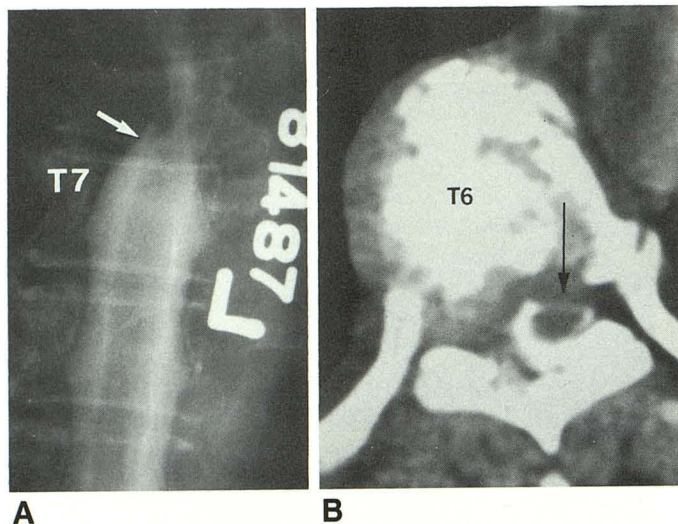

Fig. 4.—Extradural neoplasm. 78-year-old man with metastatic prostate carcinoma.
A and B, Myelogram (A) and CT scan (B) reveal T6 compression fracture with extradural block (*arrow* in A) due to tumor extending into ventral and right lateral spinal canal (*arrow* in B).
C, Nongated T2-weighted MR images (2000/90) again demonstrate compression fracture with posterior extension of tumor into spinal canal (*arrowheads*) with excellent resolution of spinal cord/CSF interface (*arrows*).
D, No further improvement in image quality is identified when cardiac gating and gradient moment nulling (2000/90) are used.
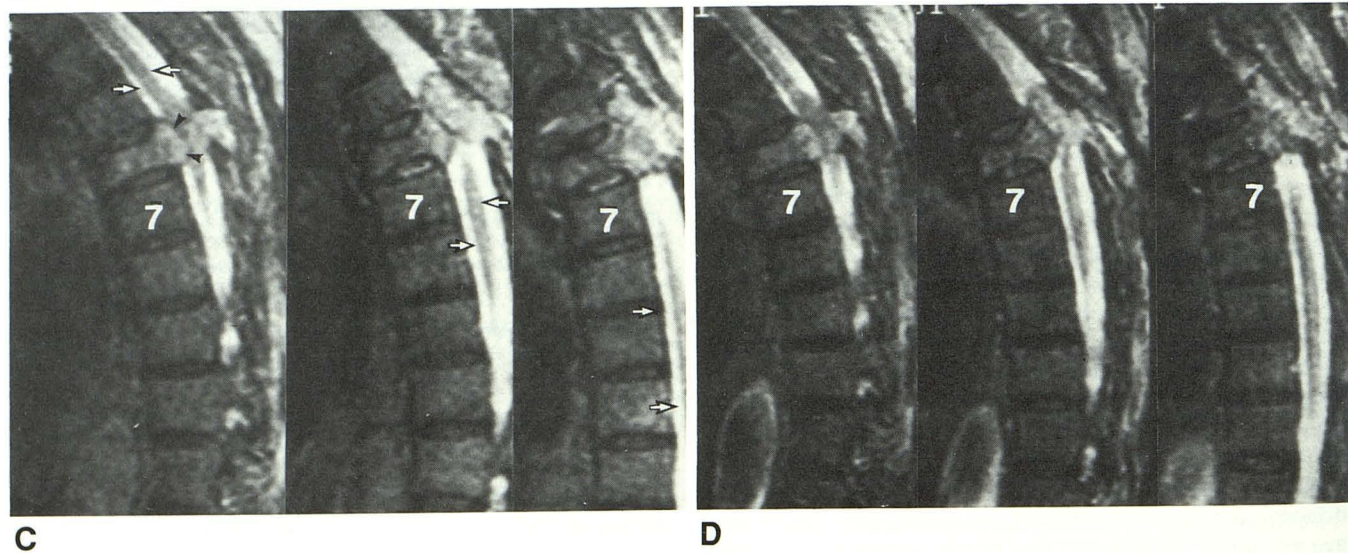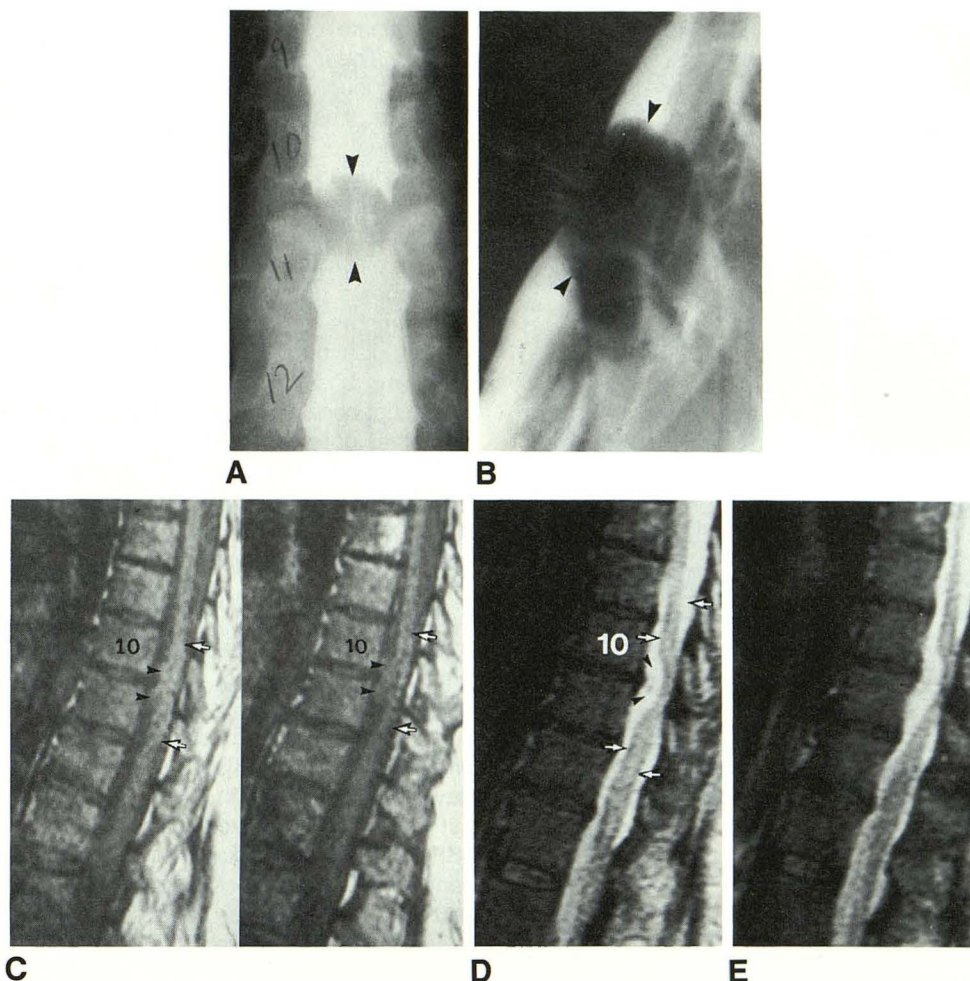
Fig. 5.—Neurofibroma. 74-year-old woman with 2-week history of back pain.

A and *B*, Thoracic myelograms demonstrate intradural, extramedullary mass at T10–T11 (*arrowheads*), causing a high-grade, essentially complete block at T11, which was overcome by transiently increasing the lumbar intrathecal pressure by rapidly injecting 6 ml of sterile saline through lumbar spinal needle with patient in 60° Trendelenberg position.

C, T1-weighted MR images (600/25) show mass (*arrowheads*) causing posterior displacement and compression of spinal cord (*arrows*).

D, Nongated T2-weighted image (2000/90) shows increased signal from intraspinal tumor (*arrowheads*) with excellent CSF/cord resolution (*arrows*).

E, No further improvement in image quality is seen on the gated (2000/90) examination. Surgical resection revealed neurofibroma.

rence of spinal canal disease or local bone metastases. In the latter case, a T1-weighted MR study might demonstrate intraspinal disease contiguous with the spinal cord that could simply represent the previously treated, not recurrent, neoplasm. Owing to the tomographic nature of MR and the relative thickness of image sections (3 or 5 mm), even if multiplanar imaging is performed, partial volume effects can still make interpretation difficult. In one of our cases (Fig. 5), an intradural, extramedullary neurofibroma associated with a focally narrowed spinal cord is demonstrated. This could have represented an acutely compressed spinal cord (which should be treated emergently) or a spinal cord that is atrophied due to chronic compression (not an emergency situation). The T2-weighted examination without motion compensation confirmed absence of pulsation artifacts and the acute nature of the cord compression. Spondylosis (Fig. 2), particularly in the

cervical region, can present with a wide range of patient complaints. Myelography (results are often dependent on the position of the cervical spine), postmyelography CT, and/or T1-weighted MR imaging can identify hypertrophic spurs, herniated disks, and/or ligamentous impingement on the thecal sac; but T2-weighted sequences without motion compensation can demonstrate absence of CSF pulsations, which may imply significant compromise of the thecal sac (same implications as high-grade or complete myelographic block).

Pitfalls

As described earlier in this article (and demonstrated in Fig. 1), MR scanning with motion compensation in normal patients
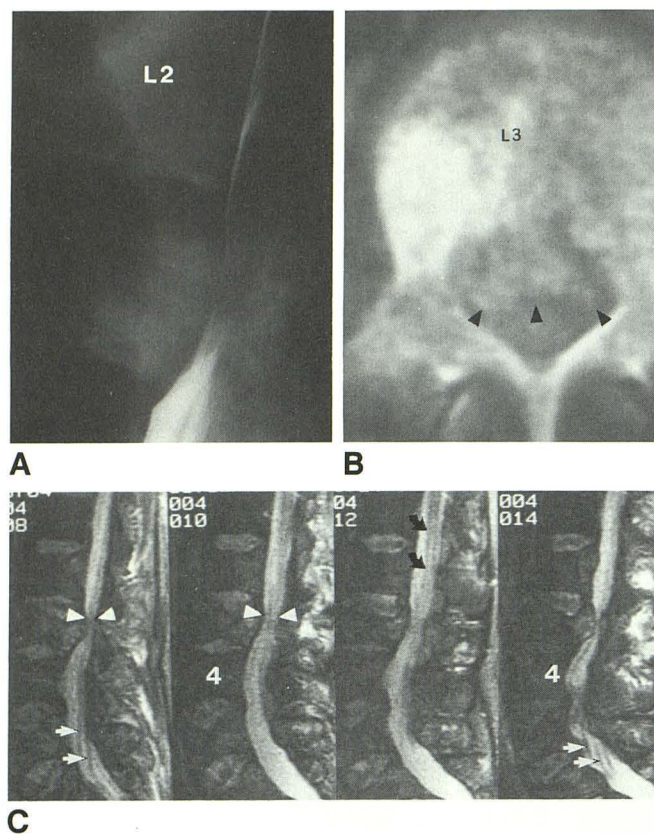
Fig. 6.—Extradural neoplasm. 54-year-old man with prostate carcinoma and new onset of bowel and bladder incontinence.

A, Lumbar myelogram demonstrates extensive vertebral metastases with compression fracture of L3 causing a high-grade extradural block.

B, Postmyelogram CT scan shows neoplastic invasion (*arrowheads*) into spinal canal.

C, Nongated T2-weighted MR images (2000/90) reveal compression of subarachnoid space (*arrowheads*), good resolution of CSF/cord interface (*curved arrows*), and intrathecal nerve roots (*arrows*). Diminished pulsation artifact allows visualization of nerve roots within thecal sac.

will eliminate or at least markedly diminish CSF pulsation artifacts. This is the same finding seen with intraspinal disease severe enough to cause a complete myelographic block. Therefore, it is imperative that patients being scanned for assessment of the degree of subarachnoid space compression be studied without motion compensation (cardiac gating and/or gradient moment nulling).

Chance synchronization of cardiac and MR cycles can result in diastolic pseudogating, which will result in an image that appears gated and could mimic the findings of spinal cord compression where none exists.

CSF pulsation artifacts are more evident on high-field systems, and absence of such artifacts on low-field systems should not necessarily be considered abnormal findings.

Another potential pitfall is one caused by even-echo rephasing (EER). EER refers to greater than expected signal from slow-moving fluid due to rephasing phenomena [6, 11]. While this is usually seen in conjunction with slow laminar flow in venous structures, it has been described within the ventricular system [6] and in the cervical CSF [12]. It could theoretically mimic loss of the CSF flow void sign, implying dampening of CSF pulsations. Therefore, asymmetric echoes (e.g., 20 and 90 msec) are necessary to avoid EER and possible false-positive examinations.

Finally, pulsation artifacts are not always identified in the lumbar region [3, 4, 13]; therefore, absence of such artifacts may not be pathologic in this region.

It has been suggested that increased signal intensity of CSF on T2-weighted examinations below the level of a significant myelographic block (inferring the loss of the CSF flow void sign) could be due to increased protein content [14]. However, the recent work of Hackney et al. [15] seems to suggest that the level of CSF protein that accumulates in patients with myelographic block (well below 6000 mg/l) is not high enough to explain the increased CSF signal observed on non-motion–compensated T2-weighted examinations.
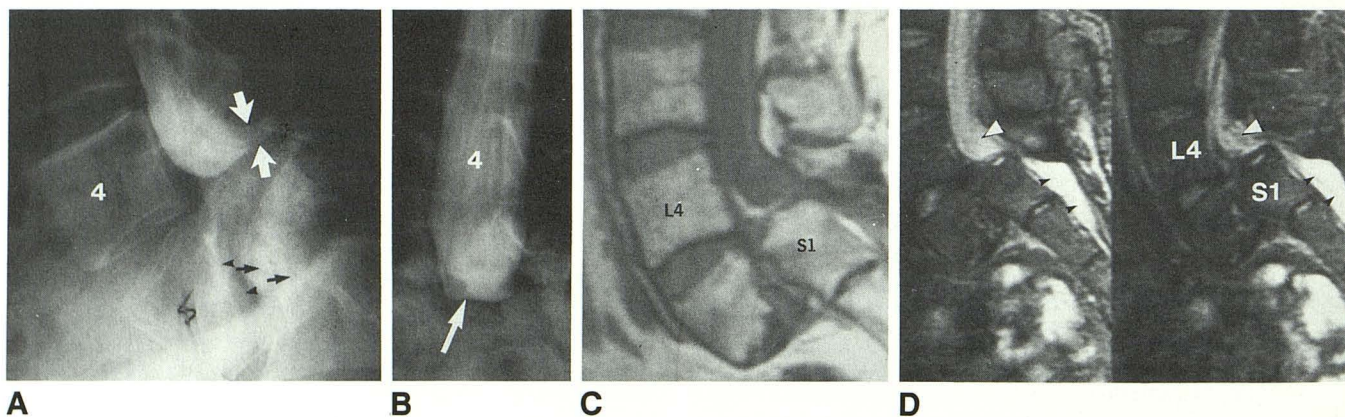

Fig. 7.—Spondylolisthesis. 50-year-old woman with progressive lower back pain and no focal neurologic deficits.

A and B, Lumbar myelography demonstrates a grade IV spondylolisthesis at L5–S1 (*arrowheads*, dorsal edge of L5; *small black arrows*, ventral edge of S1) causing an extradural block (*large white arrows*).

C, T1-weighted sagittal MR image (600/30) better demonstrates the spondylolisthesis.

D, Nongated T2-weighted images (2000/90) show greater signal intensity from CSF below level of block (*black arrowheads*) than above (*white arrowheads*). Increased signal from thecal sac below subluxation suggests loss of pulsation artifacts.

Limitations

Although we have demonstrated the high sensitivity of absence of CSF pulsations in detecting thecal sac compression, we have not evaluated the specificity of this finding. Also, 11 of the 35 examinations (31%) in our series were uninterpretable because of patient motion. While we probably could have increased the doses of pain medication, T2-weighted scans still require patients to lie motionless for approximately 10 min within our scanner and close monitoring of heavily sedated patients is difficult in high-magnetic-field machines. In the future, low flip angle techniques may significantly shorten imaging times. Currently, myelography and postmyelography CT do not require the same degree of patient cooperation as MR, and they do allow for closer patient monitoring. Another consideration is machine availability. If MR is to replace myelography for emergent evaluation of potential spinal cord compression, MR imaging will have to be available on a 24-hr basis, which is currently not the situation in most institutions. Finally, MR does not demonstrate all potential causes of spinal cord symptoms. Some processes, such as arachnoid seeding by neoplasm [9, 10] are still better evaluated by other techniques (e.g., myelography). In the future, however, arachnoid seeding may be equally or better demonstrated by contrast-enhanced MR [16].

Conclusions

We concur with previous authors [9, 10] that patients who in the past have been evaluated with complete myelography to " rule out spinal cord compression" should now initially be evaluated with multiplanar T1-weighted MR sequences supplemented with selected T2-weighted sequences as needed. A survey of the spine should be performed with T1-weighted sequences (we use a surface coil with a 20–24-cm field of view and move the coil three or four times to image the entire spine) to identify any intraspinal disease. If an abnormality is identified and the clinical history is nonspecific for acute cord compression in this region, a T2-weighted examination with-out motion compensation should be performed to determine the degree of thecal sac compromise. In other words, the T2-weighted sequence would be performed to determine if the equivalent of a high-grade block at myelography (denoting spinal cord compression) is present.

REFERENCES

1. DuBoulay GH. Pulsatile movements in the CSF pathways. *Br J Radiol* **1966**;39:255–262
2. Enzmann DR, Rubin JB, Wright A. Use of CSF gating to improve T2-weighted images. Part I: the spinal cord. *Radiology* **1987**;162:763–767
3. Kelly WM. Image artifacts and technical limitations. In: Brant-Zawadzki M, Norman D, eds. *MRI of the CNS*. New York: Raven Press, **1987**:43–82
4. Lane B, Kricheff I. CSF pulsations at myelography: a videodensitometric study. *Radiology* **1974**;110:579–587
5. Rubin JB, Enzmann DR. Imaging of spinal CSF pulsation by 2DFT MR: significance during clinical imaging. *AJNR* **1987**;8:297–306, *AJR* **1987**; 148:973–982
6. Bradley WG. MR appearance of flowing blood and CSF. In: Brant-Zawadzki M, Norman D, eds. *MRI of the CNS*. New York: Raven Press, **1987**:83–96
7. Enzmann DR, Rubin JB, DeLaPaz R, Wright A. CSF pulsation benefits and pitfalls in MR imaging. *Radiology* **1986**;161:773–884
8. Sherman JL, Citrin CM, Gangarosa RE, Bowen BJ. The MR appearance of CSF pulsations in the spinal canal. *AJNR* **1986**;7:879–884
9. Smoker WRK, Godersky JC, Knutzon RK, Keyes WD, Norman D, Berman W. The role of MR imaging in evaluating metastatic spinal disease. *AJNR* **1987**;8:901–908, *AJR* **1987**;149:1241–1248
10. Karnaze MG, Gado MH, Sartor KJ, Hodges FJ. Comparison of MR and CT myelography in imaging the cervical and thoracic spine. *AJNR* **1987**; 8:983–989
11. Bradley WG, Waluch V. Blood flow: magnetic resonance imaging. *Radiology* **1985**;154:443–450
12. Levy L, Di Chiro G, Brooks R, Dwyer A, Werner L, Frank J. Spinal cord artifacts from truncation errors during MRI. *Radiology* **1988**;166:479–483
13. Rubin J, Wright A, Enzmann DR. Lumbar spine: motion compensation for CSF on MR imaging. *Radiology* **1988**;166:225–231
14. Brant-Zawadzki M, Kelly WM, Kjos BO, et al. Magnetic resonance imaging and characterization of normal and abnormal intracranial fluid (CSF) spaces: initial observations. *Neuroradiology* **1985**;27:3–8
15. Hackney DB, Grossman RI, Zimmerman RA, et al. Low sensitivity of clinical MR imaging to small changes in the concentration of nonparamagnetic protein. *AJNR* **1987**;8:1003–1008
16. Sze G, Abramson A, Krol G, et al. Gadolinium-DTPA in the evaluation of intradural extramedullary spinal disease. *AJNR* **1988**;9:153–163