Clinical Relevance of Cervical Disk Herniation Diagnosed on the Basis of MR Imaging

In their paper, "Preoperative Evaluation of Cervical Radiculopathy and Myelopathy by Surface-Coil MR Imaging," Brown et al. [1] conclude that in many cases MR imaging can replace myelography and CT myelography in the preoperative evaluation of cervical radiculopathy and myelopathy. In the Results section, they state, "Cervical myelography, performed in 14 patients with HNPs [HNP = herniated nucleus pulposus], missed eight HNPs, of which all were detected by MR and five were detected by CT myelography (Fig. 6)." This is hardly surprising. Conventional myelography cannot show the HNP itself but only its effects on the dural sac, the nerve roots, and cord. CT and MR imaging, with their superior soft-tissue resolution, allow a better assessment of the state of the disk. The figure referred to shows a mild C6–C7 disk protrusion well visualized by MR imaging and CT that produced only a slight indentation on the ventral dural surface at myelography and had no effect at all on the cord or the nerve roots.

I think that the statement quoted here could be rephrased as follows to be more correct: MR imaging detected HNPs in 14 patients in whom myelography was also performed, but the myelogram showed no cord or nerve root involvement in eight of these. The fact that myelography was technically inadequate in two cases reduces the numbers to six of 12. In other words, in those patients in whom an adequate myelogram was available, half of the HNPs shown by MR imaging appeared to be asymptomatic, and the cause of the patients' signs and symptoms should be sought elsewhere.

In a study attempting to match clinical manifestations to abnormal radiologic features [2], we were struck by the degree of morphologic change that could be present in the cervical spine apparently without causing appropriate signs and symptoms. Our study is cited in the article by Brown et al., perhaps somewhat out of context. Teresi et al. [3] have found asymptomatic protrusions of the cervical disk in 20% to 57% of patients referred for MR imaging of the larynx. These abnormalities can, of course, be verified surgically, as Brown et al. have shown, but such verification is more concerned with the existence of a lesion than with the lesion's effects; and patients are unlikely to benefit from the removal of HNPs that are not compressing the cord or roots.

The introduction of high-resolution noninvasive imaging techniques is an undisputed boon to patients, practitioners, and researchers. The coin has a reverse side, however. The ease with which high-quality diagnostic images currently can be obtained appears likely to cause a shift in the referral pattern, to include a group of patients whose complaints are not strictly indicative of radiculopathy or myelopathy, but for whom it will be thought necessary to "exclude the presence of an HNP." The chance finding of an asymptomatic disk lesion in this category places these patients at risk for inappropriate surgery.

For this reason, it is perhaps even more important now than previously to stress the necessity of meticulous clinical evaluation of patients and critical assessment of data provided by imaging procedures. Myelography, complemented by CT as necessary, remains the gold standard for imaging compression of the cord and, especially, nerve roots, and it should be used in equivocal cases. When the myelogram is normal, MR findings of pathologic disk changes should be considered with caution and even skepticism.

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REFERENCES

Reply

Dr. Wilmink addresses the important question of clinical relevance of MR findings in the cervical spine. We agree that MR imaging can detect anatomic abnormalities that are of no clinical significance, a fact that has been established by Teresi et al. [1]. We have noted that CT myelography and myelography also sometimes detect clinically insignificant abnormalities. Our study [2] was designed to include only lesions that were clinically significant. The two criteria were (1) the location of the lesion corresponded to clinical abnormalities, and (2) the lesion was resected by a neurosurgeon who deemed the lesion responsible for clinical abnormalities. Our study thus specifically addressed the issue of how accurate various imaging tests were for detecting clinically significant lesions. We feel strongly that only clinical and surgical findings can be valid criteria for judging the comparative capabilities of MR, CT myelography, and myelography for detecting clinically significant le-
Chiari II Malformation

In their paper on the hindbrain deformity in Chiari II patients, Curnes et al. [1] state that a medullary kink at C4 or lower was seen only in asymptomatic patients with brainstem or long-tract symptomatology. The inference is that decompression in symptomatic Chiari II patients should be performed only in patients who have low kinks, although follow-up on the patients who had surgical treatment showed mixed results.

Some of us recently reported on the clinical significance of hindbrain herniation and deformity in a series of 37 patients with the Chiari II malformation [2]. We found that the neurologic status of these children was not affected by the characteristics of the deformity, confirming the contention of Gilbert et al. [3] that the most likely cause for symptomatology in the Chiari II patient is disorganization of the brainstem nuclei. Stimulated by the paper of Curnes et al., we have analyzed an additional 14 patients who have the Chiari II malformation. A medullary kink was seen in 18 of our total of 51 patients. Table 1 shows the correlation of the clinical syndrome with the presence of a medullary kink.

Our data do not suggest any relationship between the level of the medullary kink and the clinical symptomatology and therefore further substantiate our original contention that the level of a medullary kink cannot be used to identify those children who may benefit from surgery.

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REFERENCES

2. Wolpert SM, Scott RM, Piatnecberg RC, Runge VM. The clinical significance of hindbrain herniation and deformity as shown on MR images of patients with Chiari II malformation. AJNR 1988;9:1075–1078

Reply

We appreciate the extensive experience that Drs. Wolpert et al. have had in the diagnosis and treatment of children with myelomeningocele [1, 2], and we would like to respond to a few of their comments on our recent article [3].

First, with regard to their review of the article by Gilbert et al. [4], they misinterpret these authors in stating that the most likely cause for symptomatology is disorganization of the brainstem nuclei. In the study reported by Gilbert et al., which was extremely biased because of their review of children dying from Chiari II malformation, only five of 25 patients had hypoplasia or aplasia of the cranial nerve nuclei,

<p>| TABLE 1: Level of Medullary Kink in Chiari II Patients |
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