Chiari II malformation.

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sions. We also think that no imaging test, no matter how venerable, can serve as a gold standard either for assessing the clinical significance of anatomic abnormalities or for judging the accuracy of other imaging tests used to detect significant abnormalities.

Our findings, as well as other studies [3, 4], show that the CT myelography is more accurate than myelography in detecting clinically significant lesions in the cervical spine. Therefore, we cannot agree with Dr. Wilmink's assertion that myelography remains the gold standard for imaging lesions in the cervical spine, a role usurped by CT myelography several years ago. It is clear that in our patients, many clinically significant lesions that were not seen on myelography were detected easily by both CT myelography and MR and that no clinically significant lesions missed on both MR and CT myelography were seen on myelography. We therefore did not find that myelography added clinically significant information to MR and CT myelographic findings.

We also found that MR with surface coils and CT myelography are approximately equivalent in detecting clinically significant lesions in the cervical spine, although MR has a slight advantage in the lower cervical spine where bony artifacts occasionally cause degradation of CT myelographic images. MR is noninvasive, is associated with less risk and discomfort to the patient, and costs less than CT myelography. Therefore we think that MR should be the initial imaging examination for the evaluation of patients who have symptoms and signs of significant disease of the cervical spine and who are candidates for surgery.

We agree with Dr. Wilmink that the findings of any imaging test require clinical correlation to determine the significance of anatomic abnormalities. We also agree that imaging tests may disclose insignificant anatomic abnormalities. In our experience, however, MR is the most accurate imaging test for detecting abnormalities of the cervical spine that are clinically and surgically significant. In cases in which MR findings do not explain clinical abnormalities adequately, CT myelography is the best follow-up examination.

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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 symptomatic 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.

TABLE 1: Level of Medullary Kink in Chiari II Patients

Level	Asymptomatic	Symptomatic
C2	2	0
C2–C3	2	1
C3	3	3
C3–C4	1	0
C4	1	2
C4–C5	1	0
C5	1	1

Some of us recently reported on the clinical significance of the 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 brain stem nuclei. Stimulated by the paper of Curnes et al., we have analyzed an additional 14 patients who have the Chiari 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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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,

mainly the hypoglossal and dorsal vagal nuclei. The conclusion was as follows: "This suggests that the observed cranial nerve dysfunction in infants with the Arnold Chiari malformation *may*, at least in some cases, be secondary to an absence of adequate neuronal structures and thus unresponsive to posterior decompression procedures " [4]. Nowhere do Gilbert et al. directly or indirectly infer that this is the most likely cause for symptomatology.

Second, Wolpert et al., on analysis of their patients for the level of medullary kink, found symptomatic patients with medullary kinks well above C4 as well as asymptomatic patients with medullary kinks below C4. With regard to the asymptomatic group, we have observed several children who went for months or years without the development of typical symptomatology only to have life-threatening respiratory distress or worsening spasticity later. Next, with respect to the outlying symptomatic patients, the criteria for inclusion in the symptomatic group in our series were relatively rigid [5], and in 11 of 12 patients, symptoms were severe enough that surgery was offered.

Third, Wolpert et al. state that their data substantiate their original contention that the level of the medullary kink cannot be used to identify those children who may benefit from surgery. We find no indication in their article [1] that the level of the medullary kink was determined, although they did assess brainstem herniation by relating the position of the midbrain and pons to the sella and foramen magnum, and they did grade the cervicomedullary deformity.

In summary, we think that MR is helpful in identifying those patients in whom development of clinical symptomatology because of hindbrain herniation is likely, particularly if the medullary kink is at C4 or lower. Because the morbidity and mortality associated with surgical treatment of these patients are low, and because numerous articles have reported the reversibility of lethal symptoms in some children who had surgery, we continue to offer this operation to families of infants who have cranial nerve signs and symptoms, when our rigid clinical criteria for surgery are present [5]. Our experience with children whose progressive symptom is spasticity is most encouraging, and without reservation we continue to recommend neurosurgical intervention for this group. Although we think that MR is and will continue to be extremely helpful in the management and preoperative evaluation of these patients, the decision to operate must be based first and foremost on the clinical status of the patient. Our results suggest that patients with a more severe hindbrain hernia should be followed more closely for the development of symptomatology, and that if a decompression is performed, the decompression laminectomy should extend below the level of the hernia.

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Hyperintense Signals of the Posterior Lobe of the Pituitary Gland on MR Images

We were astonished by some of the findings in the experimental study on the hyperintense signal (HIS) of the posterior lobe of the pituitary gland on T1-weighted MR images reported by Kucharczyk et al. [1] in the November/December 1988 issue of *AJNR*. As may be known, some controversy about the source of this signal has arisen between the group at the University of California, San Francisco (UCSF), and us [2, 3]. In 1987 [4], we first reported that the HIS is absent in patients with diabetes insipidus and subsequently hypothesized that this signal reflects functional integrity of the hypothalamic-neurohypophyseal system and probably is indicative of neurosecretory granules (NSGs) containing antidiuretic hormone (ADH). Recently, the UCSF group [5, 6] asserted that the source of the HIS is lipid droplets localized within the pituicytes of the posterior lobe.

In their Introduction [1], Kucharczyk et al. lead the reader to believe that they were the first, in a paper published in 1986 [7], to report the relationship between this signal and the function of the posterior lobe. In fact, the paper had no description of this relationship. In this experimental study [1], they argue their thesis. However, we have found many misrepresentations, which should be criticized from a scientific point of view.

First, the most serious invention is found in two electron micrographs (Fig. 3 in reference 1) of feline posterior lobes. These photographs are negative electron micrographs. In all scientific research involving electron microscopy, negative micrographs are never used. Yet, Kucharczyk et al. have interpreted white particles in these micrographs as the lipid droplets. On conventional positive electron micrographs, these white particles would not show up as white; they would be electron-dense particles instead, which probably would indicate the presence of lysosomes, not lipid droplets [8]. Additionally, lipids never are washed out during osmium fixation. The thesis of Kucharczyk et al. is based mainly on the number of lipid droplets seen in these falsely represented electron micrographs.

Second, they stained the specimens of the dog pituitary gland with oil red O to show lipid droplets. When oil red O is used, water should be used to wash out excessive dye; the technique involves overstaining, followed by carefully monitored destaining, called the differentiation of staining. We think that the dog specimen (Fig. 1B in reference 1) is one that either has not been differentiated or has undergone insufficient differentiation.

Third, lipid droplets are abundant in rat pituicytes [9], but pituicytes of other animals have no or few lipid droplets (e.g., the rabbit posterior lobe has none [10]). We suspect that the reason for the authors' inventions is their inability to identify the lipid droplets in the cat and dog posterior lobes distinctly enough to show the changes in the numbers of these droplets.

Fourth, although Kucharczyk et al. did not determine the number of NSGs containing ADH in their experiment, they stated in the Abstract that they had seen an increase in these granules. They also speculated that the source of the HIS might be the NSGs, although they did not perform any experiments involving NSGs. In addition, not even the Discussion has a description of the NSGs. In an earlier paper [6], they stated that our hypothesis about NSGs was premature. In our opinion, it is incredible that they accepted our hypothesis without performing any of their own scientific observations to substantiate it.

Fifth, the central and peripheral effects of epinephrine on ADH vary according to dose, anesthesia, and so on. Thus, the effects are