may seem to reflect pathophysiologic changes must be interpreted with great caution.

The numerous animal studies designed to correlate the results of xenon CT with the results of other techniques have not settled the issue. Many of these studies have used significantly higher concentrations of xenon or longer inhalation times than found in human protocols, so that the effect of xenon in these cases is unclear and, in fact, may decrease CBF. Furthermore, most of these studies have used only a handful of animals and have shown only a statistical correlation between xenon CT and other methods without addressing any actual agreement between the flow values. Systematic bias and high variability are therefore not reflected in the results, and many studies have reported a low correlation coefficient, which reflects a high variability. For example, analysis of graphic data found in a recent correlation study showed that 17 (57%) of 30 of the data points corresponded to xenon flow errors of more than 30%, despite good statistical correlation [4].

Animal studies are also confounded by the high variability in xenon reactivity between species, and results do not apply to human tissue. Unfortunately, despite tremendous anecdotal experience, we have no rigorous data that address the issue of flow value agreement in humans both for normal and for diseased brain.

Xenon CT scanning is a method of measuring CBF that itself changes flow, and so its quantitative use must be scrutinized. No computer simulation can capture the biological variability seen in the response times and magnitudes in a population, and no amount of parameter testing in a mathematical model can re-create the heterogeneous and largely unknown responses of diseased tissues to xenon. Although Good and Gur have shown a minimal effect of xenon in a restrictive computer model, they do not address these variabilities, nor do they address the significant proportion of xenon studies in which flow decreases. We have little knowledge of the effects of xenon on flow in human tissues with pathologic changes, and most of the correlative data are derived from small numbers of animals and show statistical correlation only. Because small changes in CBF in response to pharmacologic or physiologic changes are considered significant in clinical practice and in research, statistical correlation alone cannot suffice for clinical use. What is clearly needed is a quantitative comparison of flow values between xenon CT and a standard technique for both normal and abnormal tissue in humans. Although the xenon investigators should be applauded for having advanced xenon CT to a qualitative method for measuring CBF, the uncertainties mentioned here should prevent the acceptance of xenon CT as quantitative until such studies are performed.

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REFERENCES


Reply

For several years, investigators and clinicians who used the xenon CT cerebral blood flow (CBF) washin protocol were puzzled by the fact that consistent, robust, and reasonable flow values were routinely derived in such studies despite findings reported in the literature on CBF activation or deactivation during xenon inhalation [1]. The article by Giller et al. [1] states that "preliminary computer simulations assuming a rise in CBF during inhalation have confirmed the calculation of both significantly falsely high and low flow values for various choices of scanning times." Our results do not support this statement, nor did the results presented by Dr. Lindstrom, one of the coauthors of that article [1], during the International Conference on Stable Xenon/CT CBF in Orlando, FL, February 8-11, 1990 [2, 3].

We agree that significant alterations in flow may occur when xenon is inhaled at concentrations typically used in such studies. More importantly, these changes are likely to be patient-specific both in the time course and the amount of activation or deactivation. However, when appropriately selected scanning protocols are used for derivation of flow estimates, the resulting flow values quite accurately represent CBF at the time the study was started. We think that all three types of experiments—simulations, animal studies, and human studies—are important in this regard, and all three support the results we published based on simulation.

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Gadopentetate Dimeglumine-Enhanced MR Imaging of Spinal Dermal Sinus Tract

We read with interest the excellent article by Barkovich et al. [1] on MR evaluation of spinal dermal tracts in children. Recently, we saw a case of a spinal sinus dermal tract in which gadopentetate dimeglumine-enhanced MR imaging enabled us to make the correct diagnosis.

A 2-year-old boy had thoracic aplasia cutis congenita. Moving the head had always been painful. From the age of 16 months, the patient had had progressive neck pain and stiffness. He had had several episodes of high fever, and a subcutaneous abscess had developed at the level of T2, which had been treated surgically. Neurologic examination showed meningeal irritation and spasticity of both legs. Bladder and bowel functions were normal. Fever had abated, and CSF cultures were negative at this time.

Plain radiographs (not shown) of the thoracic spine showed fusion of the lamina of T1 and T2. MR imaging showed a dermal sinus tract at T3, seen as a linear structure of low signal intensity on sagittal T1 weighted images (Fig. 1A). The tract could be followed through the subcutaneous fat and posterior elements of the spine to the dura. The intraspinal portion of the tract was not visible. Gadopentetate dimeglumine—enhanced MR showed that the spinal tract ended in a soft-tissue mass with marked enhancement of signal intensity on T1-weighted images (Figs. 1B–1D). This soft-tissue mass extended caudally to the level of T5. Further MR examination of the entire spine did not show additional lesions, such as intra- or extradural tumors, thickened filum terminale, or abnormal position of the conus medullaris on images obtained before and after administration of gadopentetate dimeglumine.

The patient had dorsal laminectomy at the levels of T1 to T3. The
this type of dysraphy. As infection often occurs as a complication of spinal dermal sinus tracts, and these infections might occur without clinical signs and symptoms of meningitis, we suggest routine administration of gadopentetate dimeglumine in preoperative evaluation of spinal dermal sinus tracts.

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REFERENCE

Reply
I would like to compliment Drs. Algra and Hageman on their enlightened use of a paramagnetic contrast agent in the detection of a dermal sinus tract with associated infection. Indeed, it is well known that inflammation, such as that from repeated infections, induces the formation of granulation tissue [1]. It is equally well known that granulation tissue enhances significantly after administration of IV contrast material, because of considerable vascularity and lack of a blood-tissue barrier [2, 3]. Therefore, an infected dermal sinus tract, particularly a repeatedly infected one, will enhance dramatically after infusion of IV contrast material, as shown in the case reported by Drs. Algra and Hageman. However, I think it is premature to suggest that IV paramagnetic contrast agent should be administered to all patients with suspected dermal sinus tracts. Those patients who have not had infections as a result of bacteria seeping through the dermal sinus tract will not necessarily have contrast enhancement. Ideally, someone should perform a blinded study in which some patients with dermal sinus tracts are given paramagnetic contrast material and others are not. Only after a controlled study will we know whether the added expense of the paramagnetic contrast material is justified in patients with dermal sinus tracts.

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REFERENCES

Hartnup Disease: MR Findings

Recently, we have examined a 6-year-old boy who 3 years earlier had had growth failure, hyperactivity, chronic diarrhea, intermittent ataxia, and weakness. Height and weight at that time were below the third percentile for his age, and developmental delay was present. The results of a muscle biopsy and a urine amino acid profile were consistent with Hartnup disease. MR of the brain (Figs. 1A and 1B) when the patient was 3 years old showed atrophy, delayed myelination, and dysgenesis of the corpus callosum. Follow-up MR when he