Neonatal MR imaging: achieving our own expectations.

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quality images and an understanding of the natural history of the disease confronting the patient. Proper protocols must ensure areas of coverage for lymphoma, including all the areas of probable involvement, with images of adequate spatial resolution (in general, section thickness not to exceed 3 to 4 mm and field of view not to exceed 16 to 18 cm) and with the viewing of images at appropriate window settings. With the use of a good technique, thoughtful interpretation, and consultation with our clinical colleagues, we cannot help but deliver more effective diagnoses and management of patients with lymphoma and other malignant processes in the head and neck region.

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Neonatal MR Imaging: Achieving Our Own Expectations

For the past decade our anticipation has been that MR technology would facilitate the differentiation of congenital from acquired brain abnormalities and the distinction between patterns and timing of CNS damage as it relates to toxic and metabolic insults. We have expected that this information would eventually allow us to optimize treatment of individual children or groups of children. In partial response to these expectations, Aida et al, in this issue of the *American Journal of Neuroradiology,* have added to the wealth of published material relating to the use of MR technology for the characterization of human perinatal CNS abnormalities (page 1909).

Significant challenges exist, however, relative to this literature and relative to the use of such data for the characterization of long-term clinical outcome. First, as these authors note in their introductory paragraph, the prediction of outcome based on clinical data "is difficult because of the inability to determine the severity, timing, tempo, and duration of the insult." There has been little such recognition that it is challenging for the clinician or researcher to define the very existence of perinatal asphyxia, much less its severity, timing, duration, or cause (1, 2). Despite this handicap, asphyxia is often addressed as the default process causing MR abnormalities and therefore any subsequent neurologic dysfunction. The NIH consensus definition of "acute perinatal asphyxia" requires a combination of a 5-minute Apgar score of less than or equal to 3, hypercapnia, hypoxemia, and an umbilical cord pH of less than 7.0. Damage to the brain and other organs appears to require the presence of diminished organ perfusion. Symptoms of coma, lethargy with hypotonia, poor feeding, seizures, and respiratory depression are generally associated, and develop within 12 to 24 hours of such an insult. These clinical findings are not, however, specific for hypoxic-ischemic encephalopathy, and they cannot be used alone to define the presence of such a process.

Relating to the eventual development of cerebral palsy, the NIH perinatal collaborative study indicated that predilection for cerebral palsy does not increase until Apgar scores are 3 or lower for longer than 15 minutes. The majority of neonates with biochemical evidence of acidemia will not incur neurologic sequela. Additionally, Apgar scores at 1 and 5 minutes are not, alone, predictive of the presence of asphyxia or of the development of subsequent neurologic dysfunction. It would be useful if manuscripts relating to neonatal MR findings would more fully characterize clinical and laboratory data relating to the subjects being reported, for without such data it is impossible to state whether the imaging alteration described relates to perinatal asphyxia or to separate or associated processes.

Second, the literature relative to MR findings in suspected perinatal asphyxia consists almost entirely of descriptive studies, meaning that it is based on nonrandomly selected single or group observations, without control subjects. While these studies may have provided us with a subjective feeling of comfort regarding the meaning of various patterns of MR alterations in the newborn brain, observational case-control or prospective cohort investigations using MR studies performed in sequential or randomly chosen subjects are clearly needed for hypothesis testing. If a sample chosen for study includes only those subjects with abnormal MR examinations, it will not be possible to characterize the predictive value of MR findings relative to the occurrence of normal or abnormal development. The clinical effectiveness, for diagnostic and predictive purposes, of MR imaging in the neonate needs to be more appropriately assessed.

A third difficulty relates to attempts to correlate late clinical outcome with findings on neonatal MR images. Conclusions to date have generally been based on periods of follow-up that are insufficient in duration. A normal neurologic examination in a 1-year-old child is not satisfactorily predictive of continued normal motor skills, language, or cognitive function; and psychological tests of intelligence administered at 1 year can be only grossly predictive of eventual function. Conversely, signs of spastic diplegia at age 1 year will usually resolve by age 7 years. Long-term follow-up evaluation, including clinical, psychometric, school performance, and behavioral assessment, is needed before normal or abnormal MR findings can be said to relate to late or long-term function.

Pulse sequence selection for use in the neonatal brain also needs to be addressed. Standard "adult" section thickness and skip factors, and standard TR

and TE values are less than optimal for the small, relatively nonmyelinated neonatal brain. Optimization of head coil design for use in neonates, availability of diffusion-weighted sequences, and general availability of software that allows rapid quantification of diffusion data would also be useful. Consensus among clinicians and academicians relating to these matters, perhaps achieved through the efforts of one of our professional societies, may be a needed stimulus to vendor development of optimized capabilities. As we attempt to achieve our expectations relative to the utility of neonatal MR imaging, such a process could also be a stimulus to a more thorough characterization of underlying clinical alterations (1, 2) and to more effective study design.

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Subarachnoid Hemorrhage due to Late Recurrence of a Previously Unruptured Aneurysm after Complete Endovascular Occlusion

Endovascular treatment of intracranial aneurysms has evolved over the last 10 years, with continuous improvement in embolic devices and their reliability and steady amelioration in anatomic and clinical results.

Early experience with detachable latex or silicone balloons as embolic devices led to suboptimal anatomic results and to frequent catastrophic rehemorrhage. A detachable balloon, filled with either contrast medium or HEMA, behaved as a solid intraaneurysmal implant that transmitted the arterial pressure to the wall of the aneurysm and ultimately led to recanalization and rupture of the aneurysm. Substitution of the balloon with pushable thrombogenic stainless steel coils improved the anatomic and clinical results, although the release mechanism remained very unreliable, and complete obliteration of the aneurysm remained unattainable.

Introduction of the Guglielmi detachable coil added a significant versatility and a sense of confidence and reliability to the endovascular treatment of intracranial aneurysms. For the first time, the endovascular therapist could introduce and/or remove an embolic device from an aneurysm without any significant trauma to its fragile wall. The softness and malleability of the platinum coils allowed for dense packing and obliteration of the aneurysm. In addition, and unlike detachable balloons or other devices, platinum coils deflect blood flow from the neck of the aneurysm and do not transmit the arterial pressure to the wall. The experimental work of Strothers on the hemodynamics of the inflow and outflow zones of an aneurysm added to our understanding of this vascular lesion and led to better use of the embolic device. Significant research is being conducted on refining the endovascular technique and producing better embolic devices for the treatment of aneurysms, such as the use of neck bridges, semiliquid fillers, expandable embolic material, and so on.

It is within this context of evolving and continuously improving technology that the article by Hodgson et al in this issue of the *American Journal of Neuroradiology* (page 1939) is taken into account. The authors report on the delayed rupture of a previously unruptured middle cerebral artery (MCA) aneurysm 18 months after its obliteration with platinum coils. We wholeheartedly agree with the authors that even though the short and intermediate results of endovascular treatment of aneurysms may be favorable, the long-term clinical outcome remains uncertain.

However, a few comments have to be made in reference to the clinical case that the authors report. In our clinical practice, this particular MCA aneurysm would not have been considered a good candidate for endovascular obliteration but rather would have been referred for surgical clipping. We would have refrained from any endovascular procedure on this aneurysm owing to its relatively large size, the width of its neck, and the uncertain anatomic relationship of the neck to the adjacent normal branches of the MCA. In addition, in our surgical practice, the morbidity associated with surgical clipping of unruptured aneurysms in this peripheral location is very low. Also, the packing of the aneurysm with coils is loose and not as dense as we would like to achieve. In our practice, this aneurysm would not have been considered completely obliterated. The 6-month follow-up arteriogram showed the aneurysm to be incompletely occluded and there was a residual neck. In this anatomic location (MCA trifurcation), the complex relationship of the neck of the aneurysm to the adjacent normal branches is difficult to understand and often leads to incomplete packing of the aneurysm for fear of encroachment of the coils on normal