

# G YRATIONS

THE OFFICIAL NEWSLETTER OF THE AMERICAN SOCIETY OF PEDIATRIC NEURORADIOLOGY

## MESSAGE FROM THE PRESIDENT

### QUO VADIS?

**BY NANCY K. ROLLINS, M.D., FAAP**

Children's Medical Center of Dallas  
Dallas, Texas

The Annual Meeting of the American Society of Pediatric Neuroradiology, which will be held during ASNR 45th Annual Meeting in 2007, is an opportunity for we who practice neuroradiology to continue our education, maintain our competence, and collaborate on new areas of investigation. During ASNR 2007, the meeting times for each of the six specialty societies will be shortened by a half-day, which decreases the focus sessions for each society to three, and limits the number of parallel scientific presentations.

The ASPNR will formally begin on the afternoon of Tuesday, June 12, and will continue through Wednesday, June 13. However, due to the time constraints on those days, the annual Pediatric Interesting Case Session will take place during the late afternoon on Monday, prior to the actual start of ASPNR meeting. Outside of ASPNR, the general session of ASNR on Tuesday morning will include a talk on pediatric basal ganglia disorders as part of the focus session on neurodegenerative disorders. The ASNR's Maintenance of Certification (MOC) session on Wednesday morning will include pediatric neuroradiology. It will be followed by a general session of the ASNR that will include a presentation on perinatal/infant infections. ASPNR attendees are encouraged to attend these sessions of the ASNR meeting. The ASPNR program includes a self-assessment module during the first focus session on Tuesday afternoon.

With less program time available during the ASPNR meeting, we have to hold the interest of the community neuroradiologists, while not losing sight of our goals regarding education and research directed at the developing CNS. Dr. Carl

Jaffe, Branch Chief of the Diagnostic Imaging Branch of the National Cancer Institute, will open a focus session on Wednesday afternoon by discussing problems with funded investigations from the perspective of the National Cancer Institute. Dr. Jaffe's presentation will be followed by a session that will highlight high-impact, interdisciplinary collaborative research projects currently being conducted by pediatric neuroradiologists across the country. This will be the first focus session of its kind, and participation by the attendees will be critical to its success.

The ASPNR program will feature a speaker from Children's Oncology Group, in order to stimulate interdisciplinary interactions between pediatric neuroradiologists, and the clinicians who care for children with malignancies. Dr. Naomi Winick, Professor of Pediatrics, Division of Hematology and Oncology at University of Texas Southwestern Medical School, and Vice-Chair of Therapeutic Trials for Acute Lymphocytic Leukemia, Children's Oncology Group, will discuss the neurotoxic effects that result from treatment of childhood malignancies.

Dr. Winick will discuss what is known and not known about the impact on cognition in children who undergo therapy for malignancy, be it solid or leukemia/lymphoma, and primary CNS vs. non-CNS. The goal is to stimulate investigation driven by clinical issues, in addition to our more traditional emphasis on the applications of imaging technologies. This session will be held on Wednesday afternoon, and will include a review of pediatric intracranial neoplastic disease,



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# REFLECTIONS ON PEDIATRIC NEURORADIOLOGY PROGRAMMING AT THE 44<sup>TH</sup> ANNUAL ASNR MEETING – SAN DIEGO, CALIFORNIA

By Gary L. Hedlund, D.O.

ASPNR Past-President  
Chairman, Department of Pediatric Medical Imaging  
Primary Children's Medical Center  
Salt Lake City, Utah

Organizing the Pediatric Neuroradiology programming at the annual ASNR meeting is a team effort. This year was no different. I extend my heartfelt thanks and gratitude to the moderators, focus session speakers, case presenters, and those who organized and presented instructive scientific papers and posters.

This year, the focus session content for the ASPNR spanned a broad range of topics. The opening session titled "The Brain-Basis of Autism" was coherently organized and presented by Janet Lainhart, M.D., Erin Bigler, Ph.D., and Andrew Alexander, Ph.D. The session encompassed epidemiology, neurobiology, morphometrics, spectroscopy, DTI, and fMRI in the autism spectrum. This session gave all in attendance insight into the role of the advanced MR imaging techniques for the evaluation of this disabling neurologic disorder.

The ASPNR case-based session has become an annual favorite among many ASNR members. This year was no exception. Dr. Thomas Naidich opened the session by recalling the humble beginnings of the pediatric case-based review. He then led the seven presenters in what was, as usual, a tour de force of challenging pediatric neuroradiology cases.

Two of this year's focus sessions provided the ASNR attendee who is enrolled in the MOC process in either neuroradiology or pediatric radiology, an opportunity to obtain SAM credit, in addition to CME. Despite a few electronic bugs with the audience response system, results were tabulated and credits recorded.

The first of the SAM/CME sessions was "Dose Reduction in Pediatric Neuroradiology." This timely and practical session focused on key physical factors in radiation dose reduction, a review of pediatric neuroimaging practice patterns, and tips on dose reduction with multi-slice CT and neuroangiography. This was a foundational session encouraging all of us to push the dose down in our daily practice. A job well done by Charles M. Glasier, M.D., Nancy K. Rollins, M.D., FAAP, and Mike McNitt-Gray, Ph.D.

The second SAM/CME focus session was an update and review of pediatric orbital imaging by Marvin D. Nelson, M.D. and pediatric temporal

bone imaging by Timothy N. Booth, M.D. Dr. Nelson's session encompassing pediatric orbital disease drew deeply on a rich archive of radiologic and pathologic material—a wonderful session in the classic AFIP style. Dr. Tim Booth presented one of the most comprehensive talks on pediatric temporal bone imaging that I have ever heard.

Although the official ASPNR programming ended on Tuesday, May 2nd, there was a pediatric neuro-intervention section presented by the ASITN on Wednesday. It included pediatric neuroangiography by Avery J. Evans, M.D., pediatric extracranial vascular lesions and treatment strategies by Nancy K. Rollins, M.D., FAAP, and pediatric intracranial vascular malformations presented by Patricia E. Burrows, M.D.

During the ASNR 44th Annual Meeting, there were nearly 60 pediatric neuroradiology scientific papers presented, including the winner of the *Derek Harwood-Nash Award*, titled "Incidence in Evolution of Intracranial Hemorrhage in Asymptomatic Full-Term Infants Using MR Imaging and Ultrasound Examination" by Dr. J. P. Eaton and colleagues from Tripler Army Medical Center. An even greater number of pediatric neuroradiology posters were on display for review.

Perhaps the highlight of the meeting was the presentation of the ASPNR Gold Medal Award to A. James Barkovich, M.D. This award recognizes his outstanding contributions to the field of pediatric neuroscience. Jim was honored by many friends and supportive colleagues who were in attendance.

Finally, I want to extend my deepest gratitude and thanks to Bonnie Mack, ASPNR Coordinator, for her continuous and tireless support of the ASPNR.

## THE RETZIUS NEUROANATOMY QUIZ #2

The Retzius neuroanatomy competition is an annual event that has been going on for over 10 years. The competition takes place in Los Angeles the first week in April. The competition consists of 60 questions of normal neuroanatomy in which the contestants are asked to name structures pointed out on whole brain sections, intra-op photos, angiograms, MRs, CTs, etc. The competition is open to all fellows, residents of any specialty, and medical and graduate students. There is no entry fee. The winner gets \$1,000 and a special bronze medal sculpted by the famous medal artist Alex Shagin. Second place is \$500, third place \$250. Gustav Retzius (1842-1919) was a noted Swedish anatomist and histologist at the

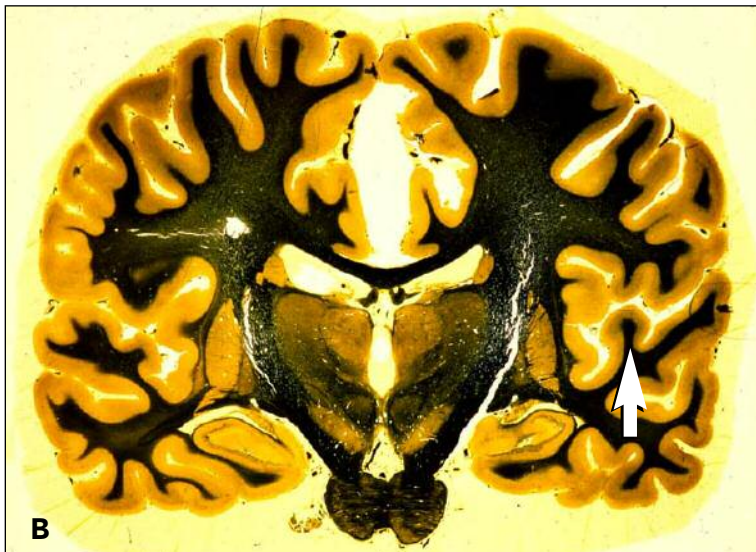
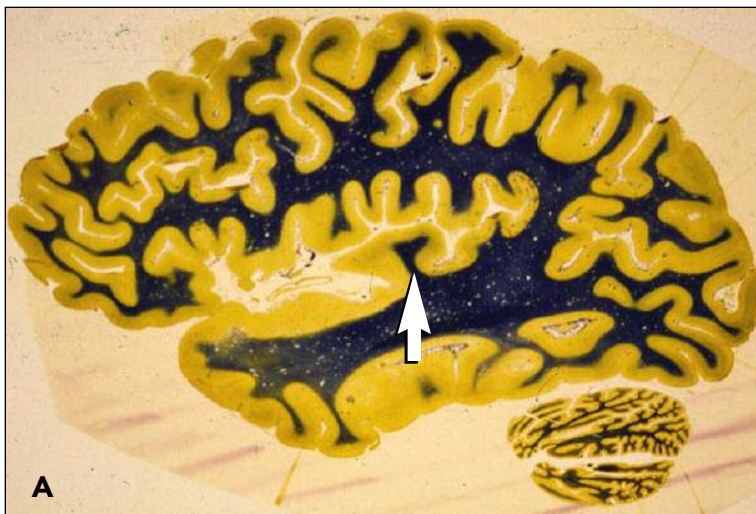
Karolinska Institute, and was one of the leaders in research during the classical period of neuroanatomy. "His comparative studies of a large series of subprimate, simian, and human brains, fetal and adult, clarified many of the more difficult problems of brain morphology." (Haymaker-Schiller, *Founders of Neurology*.)

This column will highlight points of neuroanatomy, first with a quiz and then a description of the structure named and clinical significance of lesions involving the structure, when possible.

Anyone interested in entering this competition should contact Dr. Marvin Nelson (mdnelson@chla.usc.edu).

### DISCUSSION OF THE RETZIUS

Name the structure the arrow is pointing to on both the coronal (a) and sagittal (b) whole brain myelin stained sections from the Yakolev Collection. What is the significance of this structure?



ANSWER TO RETZIUS  
NEUROANATOMY QUIZ  
ON PAGE 8

#### EDITOR'S NOTE:

The following article by Al Leviton will be the first in a series discussing the evidence and various viewpoints relating to white matter injury in neonates.

Alan Leviton, M.D. is a Professor of Neurology at the Harvard Medical School, and a Neuroepidemiologist in the Harvard School of Public Health.

From his epidemiologic studies rising out of fetal and perinatal neuropathology, he constructed a model for white matter disease that was supported in neonatal kittens. This model has been the focus of his lifelong interest in white matter disease in the prematurely born human infant. Along the way, he has authored more than 200 publications, and has done major epidemiologic studies in migraine and headache, pediatric brain tumors, day dreamers, handedness, limitations of autopsy data, lead, neonatal intracranial hemorrhage, intraobserver and interobserver reliability, coffee, epilepsy, and bronchopulmonary dysplasia.

## PERINATAL WHITE MATTER DAMAGE

By Alan Leviton, M.D., M.S.

The Children's Hospital  
Boston, Massachusetts

In the past, we have referred to white matter damage in the preterm newborn as neonatal white matter damage (NWMD). We used the term "neonatal" in preference to "perinatal," because the neuroimaging evidence of the white matter damage tended to become evident after birth.

Increasing evidence suggests that some of the damage probably has its onset *in utero*. Should it, therefore, now be called perinatal white matter damage (PWMD)? By definition, "perinatal" pertains to the period shortly before and after birth. Dorland's dictionary adds, "variously defined as beginning with completion of the twentieth to twenty-eighth week of gestation and ending 7 to 28 days after birth." This review seems an eminently suitable time to ask you to think of white matter damage in infants born during the second trimester and very early third trimester as perinatal, and not just neonatal.

We use the term white matter damage (WMD) to encompass the abnormalities identified by others on ultrasound as periventricular leukomalacia (PVL), echolucency, hyperechoic, echopoor, and even ischemic lesions, and as PVL, focal WMD, diffuse WMD and diffuse excessive high signal intensity (DEHSI) on MRI. Ventriculomegaly without macrocephaly, which predicts cerebral palsy and cognitive limitations almost as well as echolucency, is also viewed as a sonographic expression of WMD.

Obviously, we are lumpers and consider the co-occurrence of focal and diffuse PWMD to be so frequent that they are probably expressions of the same disease or share many antecedents. With improved imaging, it is likely that the sequelae of PWMD will be the most common morphologic correlates of cerebral palsy in children born much before term. Consequently, for the purposes of this review, we also often view cerebral palsy in children born much before term as almost synonymous with PWMD.

We divide the contributors to PWMD into 3 groups: damage initiators, damage promoters and maturation-dependent vulnerability.

### Damage Initiators

The lower the gestational age, the higher the risk of PWMD. Although much of the risk associated with low gestational age reflects maturation-dependent vulnerability (see below), a portion probably reflects phenomena that lead to second trimester birth.

Because both histologic and clinical chorioamnionitis are associated with prominently increased risks of prematurity, PWMD and cere-

bral palsy, one inference has been that the same inflammation that leads to preterm labor also leads to PWMD and its sequelae.

Micro-organisms are found in the amniotic fluid of only a small proportion of women who deliver prematurely when bacterial culture methods are employed. When PCR (polymerase chain reaction) is used to identify fragments of organisms, the recovery rate increases modestly. Were the organism completely eradicated in the other women, or were the inflammation initiators not bacteria?

The organisms found in pregnancies with "microbial invasion of the amniotic cavity" tend to be low virulence organisms most commonly found in the vagina (particularly in the setting of bacterial vaginosis). Bacterial organisms (or fragments) are not seen in the brains of infants who develop PWMD. Thus, any link between microbial invasion of the amniotic cavity and PWMD must invoke circulating products of inflammation that either gain access to the brain or promote the transport across the blood brain barrier (BBB) of other damage promoters.

### Damage Promoters

Chorioamnionitis is less likely to lead to PWMD if the inflammatory process fails to involve the vessels of the chorionic plate or umbilical cord. Since these vessels are unquestionably fetal tissue, vasculitis in the chorionic plate or umbilical cord is considered histologic evidence of a fetal inflammatory response. Studies of proteins (e.g., cytokines, chemokines, matrix metalloproteinases) in the umbilical (*i.e.*, fetal) cord blood also document that a fetal inflammatory response predicts PWMD. For example, the cord blood of infants who develop PWMD or cerebral palsy tends to have higher concentrations of so-called inflammatory cytokines (IL-1, IL-6, IL-8, tumor necrosis factor-alpha, interferon-gamma). The view that these cytokines might contribute to PWMD is supported by the observation that some of these same molecules are more evident in brains with focal white matter necrosis than in brains without necrotic foci.

The inflammatory cytokines have the capacity to activate endothelial cells and monocyte/macrophages, thus setting up the opportunity for the monocyte/macrophages to leave the blood vessel and enter the surrounding white matter where they either become microglia or help activate resident microglia. Either way, microglia appear to contribute to the destruction or aberrant development of oligodendrocyte precursors.

*Perinatal continued on page 5*

Their ability to produce peroxynitrite and other free radicals is viewed as part of the armamentarium used by microglia to kill bacteria. This self-protection capability now appears capable of killing oligodendroglia. Yet to be determined is how much of the axon destruction that is part of PWMD can be attributed to the loss of oligodendrocytes needed for axon well-being, and how much can be attributed more directly to axon-destructive processes.

### Maturation-dependent Vulnerability

Although the concept that PWMD occurs in a setting of maturation-dependent vulnerability goes back more than 3 decades, only recently has support been provided. Three aspects of vulnerability are probably linked, but we identify each separately.

#### a. Vulnerability of cells in the oligodendrocyte lineage

Shortly before oligodendrocyte precursors become oligodendrocytes fully capable of providing the myelin to ensheath neuronal axons, they are especially sensitive to damage promoters.

#### b. Blood-brain-barrier function and endothelial cells

The blood-brain-barrier functions of endothelial cells in the brain also have a narrow window of vulnerability.

Although studies of the vulnerability of oligodendrocytes and endothelial cells have not been carried out in live humans, the vulnerability appears to coincide with the latter 1/3 of the second trimester (25–27 weeks).

#### c. Paucity of protectors

During the second trimester, most fetuses rely on their mother and placenta to provide adequate amounts of proteins needed for brain maturation and protection. These proteins, needed for the well-being of neurons and oligodendrocytes, are described as having neurotrophic and oligotrophic properties. Often, the same proteins protect both neurons and oligodendrocytes.

Delivery before the fetus is able to produce adequate amounts of these proteins will result in the birth of a newborn whose brain is more vulnerable to perturbation and insult than is the brain of a more mature newborn able to produce sufficient amounts of these protectors. The main support in humans for this “paucity of protectors” hypothesis of preterm brain vulnerability comes from studies linking low blood thyroxine levels with increased risk of white matter damage and cerebral palsy. The low IGF-1 levels associated with retinopathy of prematurity might be additional support for the paucity of protectors hypothesis.

### Hypoxia/Ischemia

In laboratory animals, hypoxia and ischemia, each alone or together, can result in focal and diffuse white matter damage. In humans, however, neither blood gases nor blood pressure measurements support the view that hypoxia or ischemia contribute to PWMD. On the other hand, peripheral blood gases/pressure may or may not reflect hypoxia or ischemia in the CNS. Until these issues can be sorted out, what can lead to PWMD in animals, should not be considered a *sine qua non* of PWMD in humans.

Other than low gestational age, the most consistent non-inflammatory antecedent of white matter damage in humans is hypocarbia. Although some have invoked vasoconstriction as a putative mechanism linking hypocarbia with WMD, other links deserve consideration. A plausible one is based on the observation that the respiratory overshoot that results in hypocarbia is an indicator of immaturity that supplements information provided by low gestational age. Thus, hypocarbia might be a marker for maturation-dependent vulnerability rather than a damage promoter. Moreover, the potential role of carbon dioxide as a potential protector deserves elucidation.

### Synthesis/Summary

In light of the epidemiologic and basic science information available, it appears that a low virulence initiator of inflammation (such as antigenic debris from bacterial infection successfully cleared by the maternal immune system) leads to inflammation in the uterine cavity, which in turn leads to fetal inflammation, which not only promotes second trimester labor, but also the synthesis of damage promoting proteins that facilitate the movement of inflammatory cells (mainly monocyte/macrophages) across the blood-brain barrier. These cells, either themselves via transformation to microglia, or via activating resident microglia contribute to PWMD. Evidence that hypoxia and/or ischemia leads to WMD in humans remain unconvincing.

### Suggested Readings

Dammann O, Leviton A. Infection remote from the brain, neonatal white matter damage, and cerebral palsy in the preterm infant. *Semin Pediatr Neurol* 1998;5:190–201.

Dammann O, Leviton A. Role of the fetus in perinatal infection and neonatal brain damage. *Curr Opin Pediatr* 2000;12:99–104.

Dammann O, Durum S, Leviton A. Do white cells matter in white matter damage? *Trends Neurosci* 2001;24:320–4.

Hagberg H, Mallard C. Effect of inflammation on central nervous system development and vulnerability. *Curr Opin Neurol* 2005;18:117–23.

*Perinatal continued on page 7*

## IMPORTANT DEADLINES

**November 7, 2006**

Gold Medal Nominations

**November 7, 2006**

Nominations for the positions of Treasurer and Member-at-Large

**January 12, 2007**

Submissions for the *Fifth Annual ASPNR Award in Pediatric Neuroradiology Research*

**February 28, 2007**

Volunteers for Committee Service

**March 23, 2007**

Electronic submission of Pediatric Interesting Case Session to Dr. Glasier

## Is 3T Ready for Prime Time in Pediatric Neuroradiology?

The following articles discuss the merits and challenges of 3 Tesla MR for use in Pediatric Neuroradiology. Gary Hedlund discusses the practical applications and limitations of 3T at his busy Primary Children's Medical Center practice in Salt Lake City. This is followed by a discussion of some differences in site requirements and operational expenses of 3T systems.

## PEDIATRIC 3T MRI

### Reflections on Pediatric Neuroradiology with the GE Excite HD Platform

By Gary L. Hedlund, D.O.

ASPNR Past-President  
Chairman, Department of Pediatric Medical Imaging  
Primary Children's Medical Center  
Salt Lake City, Utah

In the fall of 2004, our Medical Imaging Department found itself formulating plans for a second hospital-based MR scanner. We were faced with a decision of purchasing a second 1.5T clinical "work horse" or moving up to the 3T platform.

The anticipated benefits for neuroimaging at 3T were higher spatial resolution, improved signal to noise (SNR), and enhanced capabilities for MR angiography, diffusion tensor imaging (DTI), functional MRI (fMRI), and magnetic resonance spectroscopy (MRS).

In addition to cost increase for 3T, other concerns included: radiofrequency deposition (specific absorption ratio-SAR), higher ambient acoustic noise, B1 and B0 homogeneity, compounded magnetic susceptibility effects, and anticipated chemical shift effects.

With measured excitement over moving to a premier neuroimaging platform, we committed to move up to 3T. As a fundamental step in elevating the quality of MRI services, we first performed an upgrade of our 1.5T GE system to the Signa Excite HD platform. Looking back, this upgrade of existing 1.5T technology in anticipation to coming online with 3T proved to be a very important step. By first making this upgrade, we had less disparity in spatial resolution and SNR, when comparing 8-channel head coil imaging at 1.5T and 3T.

Up front, we decided to spend the added benefits of 3T on improving image resolution, as opposed to decreasing imaging time. Looking back now at our imaging experience with 3T, some of the realized benefits have been improvement in signal to noise, increased resolution (frequency matrix typically scanned at 512), ability to diminish field of view (FOV) (effectively scanning at 8-10 cm FOV, if needed); thinner slices and the ability to shorten scan times. The concern over SAR has been experienced in pediatric patients less than 10 kg in weight. Interleaving SAR intensive scans (FLAIR T1, FSE sequences and single shot echoplanar images) between less SAR intensive sequences (GRE) can mitigate this problem. In short, we have not experienced a significant delay in scanning through-put at 3T due to SAR.

Although magnetic susceptibility effect is four times greater at 3T than 1.5T, this has not posed a significant clinical scanning problem, nor deterred placing children with dental braces in the 3T scanner. Additionally, the anticipated artifacts at air, bone, and brain interfaces have not detracted from excellent diagnostic clinical images. In large part, this is due to the ability to scan with an increased receive bandwidth. We typically scan with at least a 31.25 BW, but routinely increase BW to as much as 62.50.

In regard to patients with a programmable CSF shunt valve, recent evidence (AJNR 27:661-665, March 2006) indicates a patient with the proGAV valve may undergo MR imaging at 3T immediately after implantation. Before MR imaging the proGAV, appropriate personnel using proper equipment should determine the valve setting. The exposure to RF energy should be limited to a full body averaged SAR of 2.1 watts/kg/15 minutes. Finally, after imaging, the programmable valve setting should be determined and reset by appropriate personnel. Patients with vagal nerve stimulators are not currently scanned at 3T, due to lack of FDA approval.

As expected, all signal hungry MRI functions, such as MRA, diffusion tensor imaging (DTI), and functional MRI (fMRI) run faster and more robustly at 3T. Currently, we attempt to route all seizure patients, new brain tumors, pituitary evaluations, suspected demyelinating disease, temporal bone studies for hearing loss, and orbital evaluations to 3T. The 3T system has become our preferred platform for pediatric joint imaging.

Our current challenge at 3T is improving pediatric spinal MR imaging. SAR considerations and accentuated flow-related artifact remain the two major impediments.

Looking back now and weighing the pros and cons of 1.5T versus 3T, I can say that the decision to move up to 3T was the right way to go for our practice. The side-by-side siting of our 1.5T GE Excite HD unit and the 3T GE Excite HD platform has made for a happy marriage.

# INSTALLATION AND OPERATIONAL CONSIDERATIONS OF RUNNING A 3T MR SYSTEM

How is 3T Different?

By Marvin D. Nelson, M.D.

ASPNR Past-President

Children's Hospital of Los Angeles

USC School of Medicine

Los Angeles, California

Considering replacing one of your old 1.5T MR systems with a 3T? What are some of the practical factors independent of the vendor that you should consider:

1. **Size of the room:** Generally, 3T rooms are considerably larger than 1.5 T rooms, as the 5 gauss line must be inside the RF shielding of the room. Otherwise, steel shielding needs to be added to the walls, which is very expensive and very heavy. Even though the vendors tell you their 3T will fit into a 1.5T room, have the site engineers check it out first.
2. **Weight:** Most 3T systems are at least a third heavier than 1.5T systems. 3T systems weigh around 15,000 pounds assembled (versus around 10,000 for a 1.5T).
3. **Power:** 3T systems draw twice the amount of power as 1.5T systems. Your electric bill will be considerably higher, and the added power draw may overtax the building's power grid (particularly if the building is old). The pre-installation engineering assessment addresses the power requirements. Bringing new power lines into a facility is very expensive.

4. **Cryogenics:** 3T systems boil off cryogenics five times faster than 1.5 T systems (0.15 litre/hour versus 0.03 litre/hour, with cooling heads).
5. 3T systems are more sensitive to vibration.
6. **Coil availability:** While coil selection has improved, the range of coils available is limited.
7. **Maintenance:** Annual maintenance contracts are 30% higher for 3T systems.

As Gary Hedlund mentioned (page 6), most sites using 3T systems opt for the higher resolution, rather than the shorter image times. This means no increase in through-put for the 3T system. Since the reimbursement is the same for a 3T as for a 1.5T system, the net profit per study for the center is less for the 3T studies than for the 1.5T studies. The real advantage of 3T is for MR spectroscopy and for functional MR studies. Unfortunately, neither is currently reimbursed by CMS. CPT codes exist for both, but are unfunded. However, in sites that have research funding for spectroscopy and fMRI, a 3T system is essentially required to maintain or acquire grant funding.

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## *Message from the President continued from page 1*

as well as a presentation on the manifestations of neurotoxicity as seen by standard and newer imaging techniques.

The success of the Annual Meeting, and the effectiveness of the ASPNR as a society, depends on our membership. Our doctoral colleagues in

physics, bioengineering, neuropsychology, and computer sciences are critical to the "hard" science of imaging, and should be encouraged to join the ASPNR. Discuss the ASPNR with your physicists and encourage them to join as an Associate member.

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## *Perinatal continued from page 5*

Leviton A, Dammann O, Durum SK. The adaptive immune response in neonatal cerebral white matter damage. *Ann Neurol* 2005;58:821-8.

Wang X, Rousset CI, Hagberg H, Mallard C. Lipopolysaccharide-induced inflammation and perinatal brain injury. *Semin Fetal Neonatal Med* 2006;11:343-53.

**ANSWER TO RETZIUS  
NEUROANATOMY  
QUIZ #2**

The Transverse  
Temporal Gyrus  
(of Heschl)

This is the primary auditory cortex in the brain. Injury to the primary auditory cortex may include verbal and non-verbal auditory agnosia and cortical deafness. Agnosia is the total or partial loss of the ability to recognize familiar objects or persons through sensory stimuli; in this case, sound.

The gyrus was discovered by Richard Ladislaus Heschl (1824-1881), an Austrian anatomist and rector of the Graz University.

**NEXT ISSUE...**

The theme will be:  
Clinical, Biomechanical,  
and Neuroimaging fea-  
tures of non-accidental  
trauma.

**MEETINGS OF INTEREST**

**Society for Pediatric Radiology (SPR)** ([pedrad.org](http://pedrad.org))

Hyatt Regency Orlando International Airport – Orlando, Florida  
*MR Urography in Children Workshop*  
February 24–25, 2007

Intercontinental Hotel – Miami, Florida  
**Postgraduate Course**

*New Perspectives on Old Diseases: Advances in Neurological, Cardiovascular and  
Musculoskeletal Imaging in Children*  
April 17–18, 2007

Annual Meeting April 18–21, 2007

Fairmont Scottsdale Princess – Scottsdale, Arizona  
SPR 2008 May 6–10, 2008

**International Society for Magnetic Resonance in Medicine (ISMRM)** ([ismrm.org](http://ismrm.org))

Joint Annual Meeting ISMRM–ESMRM  
Berlin, Germany  
May 19–25, 2007

**American Society of Neuroradiology (ASNR)** ([asn.org](http://asn.org))

Hyatt Regency Chicago – Chicago, Illinois  
NER Foundation Symposium June 9–10, 2007  
ASNR 45th Annual Meeting June 11–14, 2007 \*  
\*ASPNR's program will be on June 12 and 13

**International Congress of Pediatrics (ICP)** ([icp2007.gr](http://icp2007.gr))

Athens, Greece  
25th International Congress August 25–30, 2007 \*  
\*ASPNR will be featured on the August 25 program