

# G YRATIONS

THE OFFICIAL NEWSLETTER OF THE AMERICAN SOCIETY OF PEDIATRIC NEURORADIOLOGY

## MESSAGE FROM THE PRESIDENT

# THE CONUNDRUM OF PEDIATRIC NEURORADIOLOGY MAINTENANCE OF CERTIFICATION

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

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The Maintenance Of Certification (MOC) is mandated by the American Board of Medical Specialties, which has stated the goal of improving the quality of health care through physician-initiated learning and quality improvement. It is defined as a physician-based response to demonstrate and ensure physician competence and continuing competence.

The examination is the portion of the MOC process that tests cognitive expertise. We may not embrace the concept of periodic cognitive testing, but it is a reality, and denial will not turn back the clock. We will be taking a cognitive examination as part of the MOC process, if we want to maintain our status of special expertise in a sub-specialized area of radiology.

For those of us who, as neuroradiologists practicing wholly, or in significant part, pediatric neuroradiology, neither the neuroradiology cognitive examination nor the pediatric radiology MOC are suitable, or particularly relevant. Having taken the neuroradiology MOC, I found it understandably heavily weighted towards adult neuroradiology, and although it provided an opportunity to revisit the various problems of the adult brain, head and neck, and spine, it was not representative of the practice of a pediatric neuroradiologist.

The pediatric radiology MOC provided an opportunity to refresh one's knowledge base on general pediatric radiology, but did not deal in any depth with the more complex aspects of neuroradiology. Pediatric radiologists practicing some neuroradiology are unlikely to sit for the neuroradiology MOC cognitive examination; and adult neuroradiologists practicing some pediatric neuroradiology would be unlikely to take the pediatric radiology MOC. The conundrum.

The roster of dedicated pediatric neuroradiologists is small, but noteworthy. We play an important role in furthering training and research in pediatric neuroradiology. It would do our subspecialty a disservice to lack a relevant MOC examination. An examination designed by dedicated pediatric neuroradiologists (rather than by adult neuroradiologists or pediatric radiologists) would be most appropriate. However, such a MOC would require rather extensive work by a relatively small group of hard-working individuals, who have the time, knowledge base, and inclination to develop such a MOC. It behooves the ASPNR to lay claim to the content of the pediatric neuroradiology MOC examination, and ensure that the examination is relevant and meaningful.



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## IN THIS ISSUE:

Message from the President	1
Controversies in Neuroimaging: Non-Accidental Injury (NAI) of the Developing Brain	2
Biomechanics of Non-Accidental Head Trauma: A Work in Progress	2
Neuroradiologist/Pediatrician Collaboration in Suspected Abusive Head Trauma	4
Imaging of the Central Nervous System (CNS) in Suspected or Alleged Non-Accidental Injury (NAI)	5
Child Abuse Testimony: The Truth as We Know it	7
The Retzius Neuroanatomy Quiz	9

# CONTROVERSIES IN NEUROIMAGING: NON-ACCIDENTAL HEAD TRAUMA (NAHT)

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In this issue of *Gyrations*, the importance of a multi-disciplinary team approach to suspected NAHT is stressed. Contributing authors share their unique training and wealth of clinical experience. Dr. Lori Frasier reflects upon the way the neuroradiologist can enhance her active clinical practice as a pediatrician heading a busy child protective service. Dr. Stephen Boos updates the

reader on biomechanical facts and controversies that have relevance to accidental and non-accidental injury of the pediatric brain and spine. Dr. Patrick Barnes paints an overview of the pearls and pitfalls that the neuroradiologist must consider, when interpreting cranial imaging of the injured child. Finally, Dr. Wilbur Smith challenges the neuroradiologist to be a participant in the justice

system, and shares insights from his experiences in the courtroom. I hope you will find these contributions instructive, and an enhancement to your clinical practice. I also hope you are also left with the insight that much is still to be learned from our colleagues in the clinical and research disciplines, when it comes to finding the truth when nonaccidental head injury is suspected.

## BIOMECHANICS OF NON-ACCIDENTAL HEAD TRAUMA: A WORK IN PROGRESS

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References to biomechanical research have been cited in the non-accidental head trauma (NAHT) literature, since the very beginning. John Caffey was influenced by work done at the University of Pennsylvania on rotational head injury, when he developed his conceptualization of the "whiplash shaken infant syndrome," now known as the "shaken baby syndrome" (SBS). A broader awareness of biomechanics has developed among physicians caring for abused children. Reference to biomechanical concepts has become common in discussion of suspicious injuries. Unfortunately, biomechanical arguments for and against SBS, and the use of biomechanics in evaluating NAHT, may have gotten ahead of actual biomechanical research.

Experimentation on multiple species of sub-human primates has clearly shown that rotation of the head, without any impact, is sufficient to cause subdural hematoma, concussion, traumatic coma, and diffuse traumatic axonal injury. Scaling laws, based on physics and animal experimentation, have been used to extend this data to humans. Rotational acceleration in the sagittal plane over very short intervals is most likely to produce bridging vein rupture and subdural hematoma; while rotational accelera-

tion in the coronal plane over longer durations is more likely to produce brain parenchymal effects. Acceleration initiated by a blow is twice as likely to be injurious. The meaning of "long" and "short" is relative here. In biomechanics things that happen over less than 10 milliseconds are short events, things that take 10 or more milliseconds are long events, and stresses that last 100 or more milliseconds are actually referred to as "static" events.

Several problems exist when applying this data to NAHT. First, sufficiency should not be mistaken for necessity. For example, while it is true that experimentally, subdural hematoma results from rotation, clinically, subdural hematoma sometimes occurs in the absence of apparent rotation. Second, virtually all available injury thresholds were developed in adult animal subjects, and there is a great deal of argument over what thresholds best predict injury in infants. For instance, the "head injury criterion" (HIC) is commonly used to predict head injury in crash dummy testing. An HIC of 1000 is used to predict significant head injury risk in adults. An appropriate value for use in infants, however, is debated with proponent for levels above and significantly below

1000. Third, the effects of repeated to-and-fro acceleration have not been compared to single accelerations. Finally, the biofidelity of biomechanical test dummies is limited, particularly in the infant age group. The current infant car-crash test dummy was designed to reproduce frontal impact, particularly during air bag interaction. The use of such a dummy in a shaking experiment produces data of unknown validity.

Primary traumatic injury develops by a linear pathway. The environment imposes forces and accelerations on a body. The body responds by moving in space, and by distortions in its structures, "strain." At some strain, a threshold is crossed, and functional or physical failure of the structure develops, injury. Each step of this pathway must be investigated by some experimental means to come to a full understanding of the process by which an injury develops.

### Probing Forces, Accelerations, and Motions

Traditionally, test dummies are used to measure forces, accelerations and motions during trauma. Computer modeling has re-

*Biomechanics continued on page 3*

cently been used to explore simulated traumatic circumstances. Four studies have looked at NAHT. Duhaime, Cory, Prange, and Wolfson each built anthropomorphic test dummies of infants, and shook them. In addition, Wolfson used acceleration data from the torso in a computer simulation that allowed him to significantly vary the properties of the neck. Each study then compared the resulting rotational acceleration and velocity to adult primate thresholds for subdural hematoma, concussion, and diffuse axonal injury, scaled to the brain mass of a human infant. While data from the papers differs, none exceeded the available thresholds.

Both Duhaime and Prange also slammed their dummies, and Prange dropped his dummies on a variety of surfaces. Slamming a dummy against a padded surface yields much higher accelerations than shaking alone, enough to cross the injury thresholds. This result has prompted many to use the term "shaken impact syndrome." Prange found that thresholds could be approached by dropping dummies .3meters onto concrete, but avoided commenting further on the likelihood of injury.

### **Probing Tissue Distortions**

As more biomechanical experimentation has moved toward computer simulation, tissue level data has taken on greater importance. While a number of researchers have looked at material properties for adult tissues, data for immature human tissues are limited. Margulies, Thibault, Gefen, Prange, and Coats have published material properties of skull, suture and brain for various human, piglet, and rat models. Morison tested bridging vein properties in samples from piglets and human infants.

The raw results of this work is invaluable to engineers, but of limited value to clinicians. A general understanding of tissue level motions is worthwhile, however. With successively more energetic impacts to the head, scalp is compressed; there is compression and flexion of the skull, distortion of the adherent meninges, and ultimately distortion of the brain. Strains are greatest under the area of contact, and fall off with increasing distance from that location. Traumatic consequences are expected to follow the same pattern, though skull fracture may initiate some distance from the contact area, where compressive forces give way to flexion of the skull. In general, a focal injury is expected.

Both impact and non-impact situations may accelerate the body. The effects of spinal fluid, and the non-compressible nature of brain protect against all but the

severest of linear accelerations; but rotational acceleration produces strain in intracranial structures. As the skull is accelerated, the brain lags behind. Relative motion elongates veins that bridge the two structures. The brain, while relatively resistant to compressive forces, is vulnerable in shear. Rotational acceleration causes displacement between superficial and deeper structures, elongating axons. Animal studies have demonstrated these motions through the use of isodense radio-opaque pellets placed in the brain. Pellet motion is tracked on cine-radiography during rotational acceleration of the animal.

### **Probing Thresholds for Injury**

When the strain tolerance of a tissue is exceeded, injury occurs. Strain thresholds have been studied in isolated tissues, human cadavers, and living animals. Available data for immature animals, and infants is limited, but has demonstrated both similarities, and significant differences with adult tolerances.

Morison's tests on immature human and piglet found that veins failed, when stretched to about 140% of their initial length (strain=.4). He felt that this was similar to previously reported data performed on adult bridging veins.

Duhaime found that immature animals were more resistant to injury during controlled cortical impact. In contrast, Ragupathi found that neonatal pigs were more susceptible to brain injury during rotation, than scaled adult thresholds would predict. Furthermore, repeated rotations, within a limited time interval, were more injurious than a simple additive effect would predict. These results did not produce a defined threshold for injury of immature animals, or for shaking injury.

### **Does Biomechanics Predict Injury from NAHT**

The answer to this question is best studied through living animal experiments, but there are no published studies of shaking or abusively impacting a living animal. Linking available experimental data on forces and motions during abusive behavior, resulting tissue strains, and available thresholds, forms a rough substitute. Computer simulation has been used to just that. Finite element analysis (FIE) is a computer modeling technique for studying tissue strain during trauma. In FIE, a "mesh" is developed that represents the anatomy of the tissue, and each element of the mesh is assigned material properties and boundary conditions of the tissue it represents. The computer then calculates the behavior of the mesh, when various forces and accelerations are input.

Morison used FIE to simulate the shaking of an infant. Rotation, in the sagittal plane, about the base of the neck, and translation of the "body" were modeled for four cycles, at varying frequencies. A variety of material properties were tested, not including the material properties of immature brain and skull derived from the studies listed above. As the frequency of oscillation rose from four to five Hertz, peak bridging vein strain was .4 in some models. Because this exceeds the threshold for bridging vein failure, those models predict that rapid shaking of an infant may cause subdural hematoma. Translation plus rotation produced the greatest bridging vein stretch; rotation alone produced somewhat less stretch; and translation alone produced very little stretch at all. It is common to dismiss the acceleration at the end of a fall as being translational. By contrast, Prange measured significant rotations when he dropped his dummies onto concrete from three feet. Thus, under the proper circumstances, this FIE model predicts SDH following a three foot fall, as well.

Roth conducted another FIE study. He used brain material properties derived from some of the immature animal studies given above. Shaking motions were modeled after data from the Prange study, though only a single cycle was modeled. Impact against a rigid structure at three meters per second was also simulated. Impact produced bridging vein strains of 1.0, and a single cycle of shaking strains of .8. Thus, this model predicted subdural hematoma in both modeled impact and shaking events. It should be noted that 3 meters per second is the velocity achieved after falling only one and a half feet. Roth also published measures of shear stress and pressure within the brain; but came to no conclusion regarding direct brain injury, as there are no tissue distortion thresholds with which to interpret the results.

### **What About the Neck**

Clinical and biomechanical considerations have focused increasingly on the neck and cervico-medullary junction. Significant bending, stretching and shearing of neck structures would be expected during violent shaking. Bandak has suggested that the neck would be torn asunder before free head rotation could achieve injurious levels, though Margulies found that his analysis contains serious mathematical errors. Basic material properties of the immature neck are the subject of research, but we are far from constructing a functional model of the entire head, neck system. Pathological

data is increasingly identifying evidence of neck injury associated with NAHT, but radiological studies only infrequently identify corresponding changes. There is a growing consensus that distortion of the brain stem and upper spinal cord occurs during shaking, and that resulting dysfunction produces apnea, with secondary hypoxic ischemic brain injury. Within such a model, rotational thresholds for coma and diffuse axonal injury need not be exceeded to explain the findings of the SBS.

### Conclusion

While a biomechanical understanding of head injury has been developed, data specific to young children is only now emerging. Because much of this work was done in the quest for automobile safety, shaking and other forms of NAHT have often been neglected in biomechanical research. Biomechanical arguments have been used to both undermine, and to bolster the plausibility of the "shaken baby syndrome." Some of these arguments suggest a vulnerability to short falls that is not consistent with clinical experience. In its current state, biomechanics does not offer a unified, clinically useful conclusion on NAHT. Current biomechanical thinking

does suggest that greater clinical and radiological attention to the neck, brainstem, and cervical spinal cord is warranted. For now, clinicians should be cautious when appealing to biomechanical arguments to explain individual head injuries.

### Suggested Reading

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## NEURORADIOLOGIST/PEDIATRICIAN COLLABORATION IN SUSPECTED ABUSIVE HEAD TRAUMA

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Child abuse is an unfortunate common problem. Each year, an estimated one million children are legally found to be abused in the United States. Of that number, approximately 2,000 will die, and 18,000 will be left with serious disabilities. Abusive head trauma is the single most common cause of death and disability in abused infants and young children.<sup>1,2</sup> Medical professionals who encounter children will see child abuse cases and most will see abusive head trauma. Medical centers, and especially children's hospitals, often have teams composed of pediatricians and other physicians who have the responsibility to medically evaluate suspected cases of child abuse. These special teams determine whether or not the injuries or medical

condition the child presents with are the result of child abuse.

The evaluation of abusive head trauma requires a multidisciplinary approach. Neuroimaging is an essential tool. The neuroradiologist has a critical role in this process as a consultant in child abuse assessments.<sup>3,4</sup> The pediatrician gathers clinical data including history, physical examination, laboratory and radiologic information. Communicating this clinical data to the neuroradiologist assists in the interpretation of neuroimaging studies. The pediatrician is often the contact person for investigative agencies. Mechanism of trauma, as well as timing of the injury, can be important factors in the determination of how and when the injury was inflicted. This is important, not only for

criminal prosecution, but also for further protection of the patient and other children. When the radiology report does not reflect alternative possibilities for a certain finding, conflicts can arise. A common issue is in the interpretation of mixed density subdural collections. If the radiology report suggests that the mixed density is likely evidence of new and old blood, and does not consider the possibility that it is hyperacute, i.e., admixing of active bleeding, CSF and clotted blood, this could be difficult for investigators who have difficulty interpreting medical reports, and see only the possibility that this is an old bleed. This same issue can subsequently arise in the

*Head Trauma continued on page 5*

courtroom. This is especially true, if other clinical factors such as presenting symptoms, additional injuries, and in vivo observation during surgery of the injury support a very recent injury. Another difficult and controversial issue is that of rebleeding into an expanded extra axial space, or chronic subdural versus re injury due to abuse. This presentation requires careful consideration of a variety of clinical factors by the pediatrician, and communication of those issues with the neuroradiologist. The radiologist may suggest features of the bleeding or associated brain that indicate reinjury due to abuse. Alternatively, the presence of neomembranes, and minor asymptomatic acute hemorrhage, may assist in the determination of a spontaneous rebleed. The neuroradiologist may have access to additional imaging studies, such as skeletal surveys, which can also provide context for a head injury. This is not to suggest that the neuroimaging is not an independent examination. Also, there are several medical conditions that may initially appear to result from abuse. Clues to in-born errors of metabolism, such as glutaric aciduria type 1, may be first apparent on radiologic studies. Such concerns should be communicated to the clinician in a timely manner, to prevent ongoing allegations of abuse or further assessment and treatment of a medical condition.

A request was made of the Ray E. Helfer Society list regarding how pediatricians and neuroradiologists can ideally interact. The Helfer Society is an honorary

society of physicians from any specialty nominated for their leadership and contribution to the field of child abuse. One physician suggested that neuroradiologists ask themselves how they would answer the questions related to child abuse, such as timing and alternative diagnoses, if they were called as an expert witness in the case. Several physicians indicated that the neuroradiologist should suggest any additional studies or sequences that may aid in clarifying the diagnosis, or assist the clinician in the timing of the injury. In one center, a physician noted that often two radiologists look at child abuse cases to keep findings objective. The same physician suggested that pediatricians avoid "verbal" reports, and wait for the final readings to avoid the "verbal/report" discrepancy, which sometimes occurs. A forensic pathologist suggested radiologists be informed about pathologic correlates when available, and to keep the entire case in context when reporting findings. Not all of these suggestions are possible in every center, but they speak to the need for close, collegial collaboration among all physicians involved in child abuse cases. Diagnostic accuracy is important, due to possible separation of families when children are placed in foster care. Also, there is the strong possibility of the state filing criminal charges, with far-reaching impact on personal and professional lives.

In the legal setting, the pediatrician can generally testify to the results of all of the specialty consultations, including

imaging. Although not a radiologist, it is a standard practice for physicians to utilize the radiologist's interpretations in coming to a diagnosis and treatment plan. This "standard of practice" allows the pediatrician to testify to the results of imaging studies. However, in certain cases, the court may subpoena the neuroradiologist to testify. The neuroradiologist should be available to meet with lawyers prior to the case, and provide appropriate objective testimony.

The medical evaluation of child abuse is a complex, multidisciplinary process, requiring a team approach. The evaluation of suspected abusive head trauma requires close consultation between the child abuse pediatrician and the neuroradiologist. Each brings specialized clinical skills and expertise into the process. The goal is accurate, timely, and objective diagnosis.

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# IMAGING OF THE CENTRAL NERVOUS SYSTEM (CNS) IN SUSPECTED OR ALLEGED NON-ACCIDENTAL INJURY (NAI)

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## Introduction

One of the most controversial areas of NAI (i.e. child abuse) is the medical diagnosis of inflicted CNS injury, and its forensic impact upon children and families.<sup>1-3</sup> The shaken baby syndrome (SBS) has been traditionally defined as a form of NAI characterized by the "triad" of acute encephalopathy, dural hemorrhage, and retinal hemorrhage, occurring in the context of inappropriate or inconsistent history (e.g. unwitnessed); and commonly accompanied by other apparently inflicted injuries (e.g. skeletal).

However, a number of reports from multiple disciplines have challenged the evidence base (i.e. quality of evidence analysis) for NAI / SBS as the cause in all cases of "the triad."<sup>1-9</sup> Such reports indicate that "the triad" may also be seen with accidental injury and in medical conditions, i.e. the mimics of NAI. This includes hypoxia-ischemia (e.g. apnea, choking, respiratory or cardiac arrest), ischemic injury (arterial vs. venous occlusive disease), seizures, infectious or post-infectious conditions, coagulopathy, metabolic or connective tissue dis-

order, and multifactorial (e.g. medical condition and trauma).<sup>1-5</sup> Because of the widely acknowledged controversy in NAI, the radiologist involved in such cases must be thoroughly familiar with the imaging, clinical, surgical, pathology, biomechanical, and forensic literature, from all perspectives, as well as the principles of evidence-based medicine.<sup>1-20</sup> Children with suspected NAI vs. AI must not only receive protective evaluation, but also require a

*Imaging of the CNS continued on page 6*

timely and complete clinical and imaging workup to evaluate pattern of injury and timing issues, as well as to consider the mimics of abuse.<sup>1-5</sup>

### Imaging Protocols

For the proper imaging evaluation, this includes not only computed tomography (CT) and a radiographic or radionuclide skeletal survey (SS), but also magnetic resonance imaging (MRI), and in some cases, serial imaging.<sup>1-3,5</sup> Nonenhanced head CT with soft tissue and bone algorithm is usually done initially, preferably with multidetector technology (MDCT). Facial and spinal (e.g. cervical) CT may also be needed, including reformatting. 3DCT reconstructions can also be helpful to evaluate fractures vs. developmental variants (e.g. accessory sutures, fissures, synchondroses). CT angiography may be helpful to evaluate for the cause of hemorrhage (e.g. vascular malformation, aneurysm) or infarction (e.g. dissection, venous thrombosis). Brain MRI should include at least T1, T2, FLAIR, GRE (T2\*), and DWI (ADC) sequences, and three planes. Gadolinium enhancement may be important, along with MRA and MRV. The cervical spine should also be done, along with other levels when indicated, and especially using STIR sequences.

### Imaging Analysis – CT

Regarding the initial CT exam (preferably done before surgical intervention), the findings are often nonspecific as to pattern of injury and timing, and require a differential diagnosis (DDX). In order to properly analyze such a case from an imaging perspective, each injury component must be addressed separately, and then collectively, and then correlated with clinical data.<sup>1</sup> **Extracerebral high densities** are often seen posteriorly along the tentorium, falx, interhemispheric fissure, and dural venous sinuses. These and other extracerebral high densities may be laminar, linear, nodular, or punctate. These represent either acute to subacute hemorrhages (subarachnoid - SAH, subdural - SDH) or thromboses (e.g. venous).<sup>1-3,5,10,11</sup> For apparent intracerebral high densities, it may be difficult to differentiate cerebral hemorrhages from SAH (including within the perivascular spaces), or from vascular thromboses (e.g. cortical, subependymal, or medullary venous thromboses). CT may not be able to distinguish focal or multifocal cerebral high densities as hemorrhagic contusion, hemorrhagic shear, or hemorrhagic infarction. **Extracerebral isohypodensities** may represent subarachnoid cerebrospinal fluid – CSF (e.g. benign ex-

tracerebral collections - BECC), subdural CSF hygroma (SDHG), hyperacute SDH, or chronic SDH.<sup>1-3,5,10-12,15</sup> According to the literature, timing parameters are as follows: (1) hemorrhages (especially SDH) or thromboses that are high density (i.e. clotted) on CT (i.e. acute to subacute) have a wide timing range of 3 hours to 7-10 days; (2) hemorrhage that is iso-hypodense on CT (i.e. nonclotted) may be hyperacute (<3 hrs.) or chronic (> 10 days); (3) the low density may also represent BECC or SDHG (acute or chronic); (4) Blood levels are unusual in the acute stage, unless there is coagulopathy; (5) CT cannot distinguish acute hemorrhage from re-hemorrhage upon existing chronic collections (BECC or chronic SDH); and, (6) the interhemispheric SDH is no longer considered characteristic for NAI.<sup>1-3,5,10-12,15</sup> **Cerebral low densities** may be associated with decreased gray-white matter differentiation or mass effect. In general, this indicates edema / swelling, the timing of which depends upon causation. If related to trauma, such edema / swelling may represent primary injury or secondary injury, and be acute-hyperacute (e.g. few hours) or delayed (e.g. several hours to a few days), including association with lucid interval and short falls.<sup>1-5,8,14,16-19</sup> Bilateral diffuse edema is most commonly seen with hypoxia-ischemia, but may also be seen with other diffuse processes (e.g. status epilepticus, encephalitis, etc.). Focal or multifocal edema may be seen with contusion (e.g. gray matter), shear (e.g. white matter), infarction (gray or white matter), or encephalitis or demyelination (e.g. ADEM). **Cranial defects** may represent fractures, and their timing range is very broad (e.g. hours to months old).<sup>1,2</sup> Furthermore, fracture morphology (e.g. multiple, growing) does not reliably distinguish accidental from nonaccidental causation.<sup>2</sup> Scalp collections (hemorrhage, edema, blood level) may also be nonspecific as to causation and timing.<sup>1,2</sup> If due to trauma, the timing range is also rather broad (e.g. hours to days old). Sutural widening may indicate diastatic fracture or increased intracranial pressure. Accessory sutures or synchondroses and developmental fissures may mimic fracture. Wormian bones may be associated with a skeletal dysplasia or metabolic disorder. **Serial CT exams** may show evolving, redistributing, or new hemorrhages, and evolving cerebral densities (edema / swelling) that may eventually result in atrophy or encephalomalacia. The effects of surgical intervention often complicate image interpretation.

### Image Analysis – MRI

MRI may provide more precise information, and should be done as soon as feasible after initial CT (preferably before surgical intervention).<sup>1-5,10-15</sup> Using published MRI guidelines, in general, the evolutionary timing for **hemorrhages or thromboses** (e.g. venous) are as follows: (1) hyperacute phase (< 12 hr.): T1 iso-hypointense, T2 hyperintense; (2) acute phase (1-3 days): T1 iso-hypointense, T2 hypointense; (3) early subacute phase (3-7 days): T1 hyperintense, T2 hypointense; late subacute phase (7-14 days): T1 hyperintense, T2 hyperintense; early chronic phase (> 14 days): T1 hyperintense, T2 hyperintense; late chronic phase (> 1 – 3 months): T1 isohypointense, T2 hypointense.<sup>1,2,5,23-29</sup> Mixed intensity collections may be problematic regarding timing. Matching the MRI findings with the CT density findings may help along with follow up MRI. Blood levels may indicate subacute hemorrhage vs. coagulopathy. The above guidelines for timing may be better applied to the sediment than to the supernatant. Also, a single MRI may not reliably differentiate T1 hypointense / T2 hyperintense collections as representing CSF collections (e.g. BECC, acute SDHG) v. hyperacute SDH v. chronic collections (SDH, SDHG). GRE hypointensities are iron-sensitive, but do not assist with timing unless matched with T1, T2, and CT densities. GRE and other magnetic susceptibility sequences may also be sensitive to venous thromboses (e.g. cortical, medullary, subependymal) that may not be detected by MRV. Again, the effects of surgical intervention often complicate image interpretation.

With regard to **brain injury**, MRI may distinguish hypoxic-ischemic injury (diffuse symmetric DWI / ADC restricted diffusion with matching T1/T2 abnormalities) from shear and contusional injury (focal / multifocal restricted diffusion, GRE hypointensities, with T2 / FLAIR edema). The latter, however, may not be reliably differentiated from focal / multifocal ischemic or hemorrhagic infarction (e.g. dissection, vasculitis, venous, thrombotic, embolic) without supportive MRA, CTA, MRV, or angiography.<sup>1-5,14</sup> Also, similar cortical or subcortical intensity abnormalities (including restricted diffusion) may also be observed with encephalitis, seizures, and metabolic disorders. Using published MRI criteria and parameters,<sup>14</sup> in general, the evolutionary timing for ischemic injury is as follows: (1) hyperacute phase (< 1 day): DWI hyperintense, ADC hypointense; (2) early acute phase (1-2 days): T2 hyperintense; (3) late

acute phase (2-4 days): T1 hyperintense; early subacute phase (6-7 days): T2 hypointense; late subacute phase (7-14 days): DWI iso-hypointense, ADC isohyperintense; chronic phase (> 14-21 days): atrophy. If related to trauma, focal / multifocal ischemic findings may be due to arterial injury (e.g. dissection), venous injury (e.g. tear, thrombosis), arterial spasm (as with any cause of hemorrhage), herniation, or edema with secondary perfusion deficit or seizures (e.g. status epilepticus). Hypoxia-ischemic brain injury due to apnea / respiratory arrest may occur with head trauma or with neck / cervical spine / cord injuries (e.g. SCIWORA) whether AI or NAI.<sup>19</sup> It may also occur with any nontraumatic cause (e.g. choking, paroxysmal coughing, aspiration, etc.).<sup>20</sup> In addition to the diffuse brain injury, there may be associated SAH and SDH without mass effect.<sup>1,8</sup>

### Summary

The radiologist should describe pattern of injury, provide a differential diagnosis, and offer timing ranges, if possible. Timely communication with the primary care team is critical, and consultation with child protection services, other medical or surgical consults, including the pathologist or biomechanical specialist, law enforcement investigators, and attorneys for all parties is appropriate.<sup>1-5</sup> The radiologist must also be aware of certain conditions that are known to have clinical and imaging features that may mimic abuse.<sup>1-5</sup> These should be properly ruled out, and the possibility of com-

bined, or multifactorial, mechanisms with synergistic effects should also be considered (e.g. predisposing condition plus trauma). A timely and thorough multidisciplinary evaluation may be the difference between appropriate child protection versus an improper breakup of the family or a wrongful indictment and conviction.

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## CHILD ABUSE TESTIMONY: THE TRUTH AS WE KNOW IT

By Wilbur L. Smith, M.D., FACR, FAAP

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Legal testimony is a painful necessity in the lives of many pediatric neuroradiologists, and no other instance is more wrenching than testimony regarding non-accidental brain injury (NABI) suffered by a child. While estimates are imprecise, conservative figures suggest that annually about 1,500 children, mostly infants, die in the United States owing to abusive injuries. Most of these deaths occur among children younger than 24 months of age, and the large majority occurs owing to brain trauma. The death incidence is the tip of the iceberg. The morbidity of non-accidental brain injury (NABI) far exceeds the mortality figures. Research shows that 2/3 of all

children suffering a symptomatic NABI will have a demonstrable permanent neurological defect. The dollar cost to the society for care of these children is enormous, let alone the emotional impact upon the loved ones, family and caregivers of the survivors.

Pediatric neuroradiologists and neuro-radiologists are in a unique position of using imaging techniques to diagnose and define the extent of NABI. In many instances, we are involved early in the care of the injured child, and remain involved in following the injuries and their sequelae in the infant's brain. This, coupled with our knowledge and experience with analogous forms of brain injury, forms a basis for ex-

plaining these injuries to society through research, scientific writing, lay writing, and the justice system.

Physicians tend to be wary of the justice system for a variety of reasons. The legal system is costly of time, outcomes are often unpredictable (relative to the physician's opinions), judicial remedies may not agree with our beliefs and, most importantly, the physician often feels powerless within the legal system. As subspecialty physicians, we are used to being listened to; as witnesses, we can only respond to queries by lawyers and judges, and are

## IMPORTANT DEADLINE

March 23, 2007

Electronic submission of Pediatric Interesting Case Session to Dr. Glasier

### *Child Abuse continued from page 7*

subjected to lines of questioning often designed to destroy our credibility. It is little wonder that many physicians avoid contact with the legal system at all costs.

Why should we become involved in testimony regarding NABI? After testimony in more than 100 cases of abusive injury, and review of several times that number of suspected NABI cases, I justify my involvement for these reasons:

1. The judicial process offers a vehicle to publicly communicate, through truthful and informed testimony, that this major societal problem exists in near epidemic proportions, and can only be fixed through a societal effort. The lay understanding of NABI and its ramifications comes only with open public presentation of the issue, and the vehicle for this presentation is often, unfortunately, the post-hoc facts, presented at trial.
2. Our society is one of law, and the system only works if competent truthful testimony is available to the judicial system. Truth is an evanescent quantity, recognized judicially as "beyond a reasonable doubt," neither an absolute, nor a low hurdle in crimes, where eyewitnesses are rare, and circumstantial evidence is plentiful. The best experts offer the best chance for justice, be it conviction or acquittal.
3. At the 2006 Shaken Baby Conference in Park City, Utah I attended a round-table seminar presented by fathers of victims of NABI, men whose lives were inexorably altered by the injuries to their children. While speaking with these parents, I was struck in a way I never understood before by their need for closure. Searching for someone to affirm what happened to their child, and who had likely done it. This human need for closure overwhelmed any sense of retribution. Competent analysis and testimony goes a long way towards providing this closure.

Once you decide to "take" a court case, you can perform several actions to ensure that your testimony is as effective as possible.

1. Request all relevant materials before making any preliminary assessment of the case. Record

the materials you receive and review, and do not hesitate to ask for additional materials, if needed. No attorney wants their witness to state at trial that they did not receive relevant materials.

2. After you have reviewed all relevant materials, insist upon a direct discussion with the attorney who hired/subpoenaed you.
3. If you are comfortable in doing so, always integrate the clinical information with the imaging. As imagers, we know that the images set boundaries on certain parameters, such as the age of a hemorrhage; however, correlation with symptoms can often focus the time of occurrence within the imaging boundaries.
4. Whenever possible it is best to keep it simple, especially with a jury. Your best scientific reasoning is worthless, if not understood by your lay audience.
5. Never let annoyance show. In an adversarial system, annoying the opponent is an essential tactic. Lawyers who "tear each other up" in the classroom, socialize after court. You must play that game, also.
6. Tell the truth as you perceive it, but consider other's perceptions, and be respectful. Never play 'one up' by denigrating another expert witness's opinion; instead, vigorously defend your own.

Many of us will disagree during assessment of NABI cases, and in the interpretation of 'reasonable doubt'; however, this cannot be an excuse for non-participation. The best testimony truthfully presented does not guarantee outcome; but non-participation by the qualified experts will surely doom the process.<sup>1,2,3</sup>

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## THE RETZIUS NEUROANATOMY QUIZ #3

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

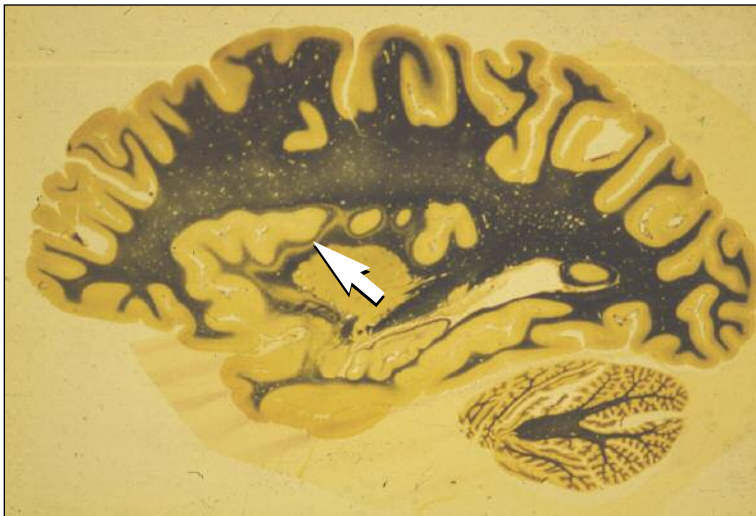
Karolinska Institute, and was one of the leaders in research during the classical period of neuroanatomy. "His comparative studies of a large series of subprimate, simian, and human brains, fetal and adult, clarified many of the more difficult problems of brain morphology." (Haymaker-Schiller, Founders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

**Sagittal section of the cerebrum. Name the structure (arrow).**



The arrow is pointing to the yellow band (gray matter) on the photo.

### ASPNR REMINDERS

Don't forget to pay your 2007 membership dues! If you need another invoice, contact the Headquarters office (bmack@asn.org).

Know a colleague who might be interested in ASPNR membership? Applications for 2008 are available on our website (www.aspn.org) or by contacting the Headquarters Office (bmack@asn.org). The deadline to apply is October 10, 2007.

**ANSWER TO RETZIUS  
NEUROANATOMY  
QUIZ #3**

**The Claustrum**

**NEXT ISSUE...**

Watch for an article about status marmoratus, and part one of a two-part article on myelination.

Clastrum is derived from the Latin, claustra, which is variously translated as meaning either a "barrier" or "hidden away." The claustrum is a band of gray matter lying deep to the insula between the external and extreme capsules, varying in thickness from a fraction of millimeter to several millimeters. The function of the claustrum is unknown, but currently is receiving a lot of attention. In his last publication, Francis Crick, of DNA fame, along with Christof Koch, postulated the claustrum to be the integrating region of the brain that allows for consciousness.<sup>1</sup> The argument being the claustrum receives input from almost all regions of the cortex and projects back to almost all regions of cortex. As such, it serves to "integrate the continuous interactions

of groups of widely dispersed pyramidal neurons that express themselves in the ongoing stream of conscious percepts, images and thoughts." The thalamus cannot fulfill this role, as the various nuclei within the thalamus do not have the interconnections to talk to each other.

Of interest, the claustrum is only present in mammals. For those interested, in 2004, Edelman and Denaro published an extensive historical review of the anatomy and significance of the claustrum.<sup>2</sup>

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## MEETINGS OF INTEREST

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

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

### **American Society of Functional Neuroradiology (ASFNR)** ([asfnr.org](http://asfnr.org))

Caribe Royale All Suites Hotel – Orlando, Florida  
1st Annual Meeting – April 19-22, 2007

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

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

### **European Society of Magnetic Resonance in Neuropediatrics (ESMRN)** ([esmrn.com](http://esmrn.com))

Kupferbau, Tübingen, Germany  
9th Congress – May 31-June 2, 2007

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

Ernest N. Morial Convention Center – New Orleans, Louisiana  
46th Annual Meeting – May 31-June 5, 2008

### **Eastern Neuroradiological Society (ENRS)** ([enrs.org](http://enrs.org))

StoweLake Resort & Spa – StoweLake, Vermont  
19th Annual Meeting – August 23-25, 2007

### **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 program August 25

### **American Society of Head and Neck Radiology (ASHNR)** ([ashnr.org](http://ashnr.org))

The Fairmont Olympic Hotel – Seattle, Washington  
41st Annual Meeting – September 26-30, 2007

### **Western Neuroradiological Society (WNRS)** ([wnrs.org](http://wnrs.org))

Sheraton Vancouver Wall Centre Hotel, Vancouver, BC, Canada  
39th Annual Meeting – October 4-7, 2007