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Ethical Issues in Imaging Nonaccidental Injury: Child Abuse

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Summary: One of the most controversial areas of nonaccidental injury is the medical diagnosis of inflicted central nervous system injury and its impact on medical, social, and legal outcomes for children and families. This review addresses the role of the neuroradiologist in the clinical care of the pediatric patient and as an expert medical witness in the area of nonaccidental injury. **Key Words:** Magnetic resonance imaging—Pediatric neuroradiology—Nonaccidental injury—Child abuse.

INTRODUCTION

Traumatic central nervous system (CNS) injury is reportedly the leading cause of morbidity and mortality in children, accounting for nearly 100,000 emergencies per year in the United States and half the deaths from infancy through puberty (1,2). The major causes in this age group are accidental and include falls, vehicular accidents, and recreational mishaps. However, nonaccidental, inflicted, or intentional, trauma (nonaccidental injury [NAI]) is said to be an increasingly frequent cause of CNS injury and is stated to be responsible for about 80% of the deaths (estimated 3,000 per year) from brain injury in children less than 2 years old (3,4). The peak incidence of child abuse resulting in CNS injury is about 6 months of age. The acute life-threatening consequences of CNS injury as well as the long-term effects on the development of the child have been a focus of major interest since the landmark article by Caffey (5) in 1946 and the Rigler lecture by Silverman (6) in 1972. Computed tomography (CT) and, more recently, magnetic resonance imaging (MRI) reportedly have provided data that allow more accurate estimates of the incidence of CNS trauma associated with abuse, with a reported range from 7% to19% (2,7–35).

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MECHANISMS AND MANIFESTATIONS OF CNS INJURY IN NAI

Direct contact, or impact, phenomena are said to result in localized cranial distortion and thus produce focal injury, such as fracture, contusion, or an epidural hemorrhage. Accidental injury is said to be typically associated with this mechanism (10,23,36–38). Although common in abuse, it has been reported that impact injury (except for epidural hematoma) usually is not life threatening. In child abuse, it is indirect trauma (i.e., independent of skull deformation) that is considered responsible for the most severe CNS injury (10,23,36–38). Inertial loading accompanying sudden angular acceleration or deceleration of the head on the neck, as with shaking, results in shear strain deformation, disruption at tissue interfaces, and, therefore, diffuse injury. Such a mechanism would seem logical in the susceptible young infant who is predisposed by weak neck muscles, a relatively large head, and the vulnerability of an immature brain (10,23,36–38). It is this shaking mechanism that traditionally has been postulated to produce the primary injury pattern (i.e., subdural hematoma, retinal hemorrhages, and diffuse axonal injury) and secondary injury pattern (i.e., edema, swelling, hypoxia-ischemia, herniation) stated to be characteristic, if not pathognomonic, of the shaken baby syndrome (SBS) (10,23,36–51). Such patterns of damage often are associated with the most severe and fatal CNS injuries and are readily demonstrated by neuroimaging, surgical neuropathology, and postmortem neuropathology (7–57). It has further been declared that retinal hemorrhages of a par-

ticular pattern are diagnostic of SBS/NAI; that such CNS injury on an accidental basis can only be associated with a massive force equivalent to a motor vehicle accident or a fall from a two-story building; that such injury is immediately symptomatic and cannot be followed by a lucid interval; and that changing symptoms in a child with prior head injury is due to newly inflicted injury and not just a rebleed (22,23,45,46,51,52,58–65). Therefore, from this reasoning, the last caretaker with the injured child is automatically considered guilty of abusive injury, especially if the incident is unwitnessed (43,59,63,64).

Imaging evidence of CNS injury may occur with, or without, other clinical findings of trauma (e.g., bruising) or other "higher specificity" imaging findings associated with violent shaking (e.g., metaphyseal, rib, or other typical skeletal injuries) (10,23). Therefore, clinical and imaging findings of injury out of proportion to the history of trauma and injuries of different ages have become two of the key diagnostic criteria indicating the "probability" of NAI/SBS or child abuse, particularly when encountered in a young infant (10,23,37). Such clinical and imaging findings traditionally have formed the basis on which radiologists and other health professionals have provided a medical diagnosis and offered expert testimony that such findings are "proof" of NAI/SBS.

CONTROVERSY

Despite imaging advances, fundamental difficulties persist in extracting a definitive diagnosis of NAI/SBS based on a causative event (i.e., shaking) that is inferred, in a number of cases, from clinical, radiologic, and pathologic findings in the absence of witnessed or admitted violent shaking (22,23,43,51,61,63,64). This problem is magnified further by the lack of consistent and reliable criteria for the diagnosis of NAI/SBS, and that the vast body of literature on child abuse is composed of anecdotal case series, case reports, reviews, opinions, and position papers (2,23,43,56,51,63,64). Furthermore, many reports include cases having impact injury that not only raises doubt regarding the "shaking only" mechanism but also questions that this injury is always "nonaccidental." From an evidence-based medicine perspective, quality of evidence ratings for diagnostic criteria regarding the literature on SBS reveal that few published reports merit a rating above class IV (any design where the test is not applied in blinded evaluation, where evidence is provided by expert opinion alone, or in descriptive case series without controls) (66,67). Such quality of evidence ratings hardly earn a diagnostic criteria recommendation level of "optional," much less as a "guideline" or a "standard." This is particularly true of the neuroimaging literature on SBS/NAI, the clinical SBS/NAI literature that uses neuroimaging, and the forensic pathology literature (7–35,44,45,52,53–56,63,64,66). The inclusion criteria for many of these series often encompass arbitrary diagnostic categories beyond "definite or confirmed abuse," such as "suspected abuse," "presumed abuse," "likely abuse," and "indeterminate."

The only attempt at a scientific study to test NAI/SBS used a biomechanical approach and measured stresses from shaking versus impact in a doll model. The study correlated those stresses with injury thresholds in subhuman primate experiments established in another study (38). Only stresses associated with impact, whether using an unpadded or padded surface, exceeded the injury thresholds that correlated with the pathologic spectrum of concussion, subdural hematoma, and diffuse axonal injury. The authors concluded that CNS injury in SBS/NAI in its most severe form usually is not caused by shaking alone. These authors also concluded that fatal cases of SBS/NAI, unless in children with predisposing factors (e.g., subdural hygroma, atrophy), are not likely to result from shaking during play, feeding, or swinging, or from more vigorous shaking by a caretaker given for discipline. Although subsequent reports substantiated that CNS injury in NAI from shaking usually is associated with impact, those and other publications also provided evidence that shaking alone can produce serious intracranial injury (23,33,57).

NAI/SBS REVISITED

Some past and more recent reports have brought forward information based upon clinical, surgical, imaging, pathologic, biomechanical, social, and legal observations that raise serious doubt regarding NAI/SBS as the cause in all cases of infant CNS injury otherwise attributed to abuse using traditional diagnostic criteria for NAI/SBS (10,37,38,43,64,66,68–77). These reports include skull fracture and/or acute subdural hematoma from accidental simple falls in young infants, such as those associated with wide extracerebral spaces (e.g., benign external hydrocephalus, benign extracerebral collections of infancy, subdural hygromas) (49,72–74), and fatal pediatric head injuries caused by witnessed, accidental short-distance falls, including those with a lucid interval and retinal hemorrhages (75). Updated neuropathologic studies indicate the following: (1) cerebral swelling in young infants (<1 year of age) with "SBS," as compared with older infants, more often is due to diffuse axonal injury of hypoxic-ischemic origin rather than traumatic origin (the latter more appropriately termed multifocal traumatic axonal, or shear, injury); (2) although fractures, subdural hemorrhage (e.g.,

interhemispheric), and retinal hemorrhages commonly are present, the usual cause of death was increased intracranial pressure from brain swelling associated with hypoxia-ischemia; and (3) cervical epidural hemorrhage and focal axonal brain stem, cervical cord, and spinal nerve root injuries are characteristically seen in these infants (presumably due to shaking) and may be associated with apnea and responsible for the hypoxic-ischemic brain injury (53–56). Reports of neurosurgical, neuroradiologic, and neuropathologic findings in head trauma as correlated with biomechanical analyses indicate that subdural hematoma and retinal hemorrhages occur with rotational deceleration injuries, whether "accidental" (e.g., axis or center of rotation internal to the skull, including those due to short-distance falls) or "nonaccidental" (i.e., axis of rotation external to the skull, e.g., at the craniocervical junction or cervical spinal level in SBS) (33,68,78–81). To date, there is no scientific basis that indicates how much, or how little, force is necessary to produce traumatic injury to the developing CNS. Furthermore, the specificity of retinal hemorrhages for child abuse and their dating has been questioned. Such hemorrhages reportedly may be seen with a variety of conditions, including accidental trauma, resuscitation, increased intracranial pressure, increased venous pressure, subarachnoid hemorrhage, sepsis, coagulopathy, certain metabolic disorders, systemic hypertension, and other conditions (10,23,48,43).

In view of the medical imaging data available, it is clear that we do not have an established scientific platform from which to distinguish nonaccidental from accidental CNS injury and, in some cases, traumatic from nontraumatic CNS injury. It is obvious that carefully conducted research is needed to establish a sound scientific foundation for CNS injury in NAI, especially in those circumstances in which other characteristic injuries are not present (e.g., skeletal). The immaturity of the young infant brain (e.g., undermyelinated, larger extracerebral spaces) makes it more vulnerable to traumatic CNS injury than that of the older child or adult, whether accidental or nonaccidental, and relies on the attention of caretakers for its safety. However, as the infant becomes more mobile (rolling, crawling, walking), the risk of accidental CNS injury (e.g., from falls) becomes greater. The medical and imaging evidence, particularly when there is only CNS injury, cannot accurately diagnose presumed intentional injury. Only the social investigation may provide the basis for inflicted injury in the context of supportive medical, imaging, or pathologic findings. Furthermore, biomechanical factors must be considered when determining the mechanism of trauma. It should be remembered that there are nonabuse traumatic, as well as nontraumatic, causes of subdural collections, hemorrhagic or nonhemorrhagic, and retinal hemorrhages. One classic example is the newborn following labor and delivery. Such manifestations of birth trauma may persist beyond the neonatal period and mimic abuse. Other examples are infants following extracorporeal membrane oxygenation (ECMO) therapy and infants with other processes associated with increased intracranial venous pressure (e.g., infectious or postinfectious meningoencephalitis, venous thrombosis) (10,23,82,83).

PEDIATRIC NEURORADIOLOGY IN THE CLINICAL EVALUATION OF NAI

The child with suspected abuse must not only receive protective evaluation, but also deserves a timely and complete clinical and imaging workup to evaluate the pattern of injury and timing issues and to exclude the mimics of abuse. For CNS injury, this includes not only CT but also MRI and, in some cases, ultrasound (US). Serial imaging, particularly with MRI, also may be necessary. Imaging protocols, utilization guidelines, and interpretative principles are presented in greater detail in a number of recent publications (10,22,23,31,32,76,84,85).

Computed tomography

In general, CT is used primarily to identify acute or subacute hemorrhage, other collections, edema, skull fracture, and scalp injury (10,22,23,32). CT, particularly as a single examination, often cannot provide precise information with regard to the character and age of collections, particularly in the presence of anemia or coagulopathy and depending upon the nature and number of traumatic events (Fig. 1). Acute to subacute clotted hemorrhage appears high density (age range: 3 hours to 7 days, or sometimes up to 10 days). Hyperacute "unclotted" hemorrhage (<3 hours old), very chronic hemorrhagic collections (>10 to 14 days old), and nonhemorrhagic collections (e.g., subdural effusions or hygromas) all appear hypodense, or occasionally isodense, depending upon protein and cell content. The subarachnoid cerebrospinal fluid (CSF) spaces may be prominent in infants (e.g., normal infantile subarachnoid spaces, benign external hydrocephalus, or benign extracerebral collections) and appear low density on CT. US with Doppler may be used to separate out the fluid compartments, but MRI usually is needed for more definite evaluation (10,23,86–88).

Bone CT algorithms and display techniques are used along with skull films to evaluate for scalp and skull injuries. Scalp and skull injuries are difficult to time precisely by CT, unless serial CTs are available and depending upon the nature and number of traumatic events (10,23). In acute neurologic presentations, CT is indicated emergently to evaluate the need for immediate neurosur-

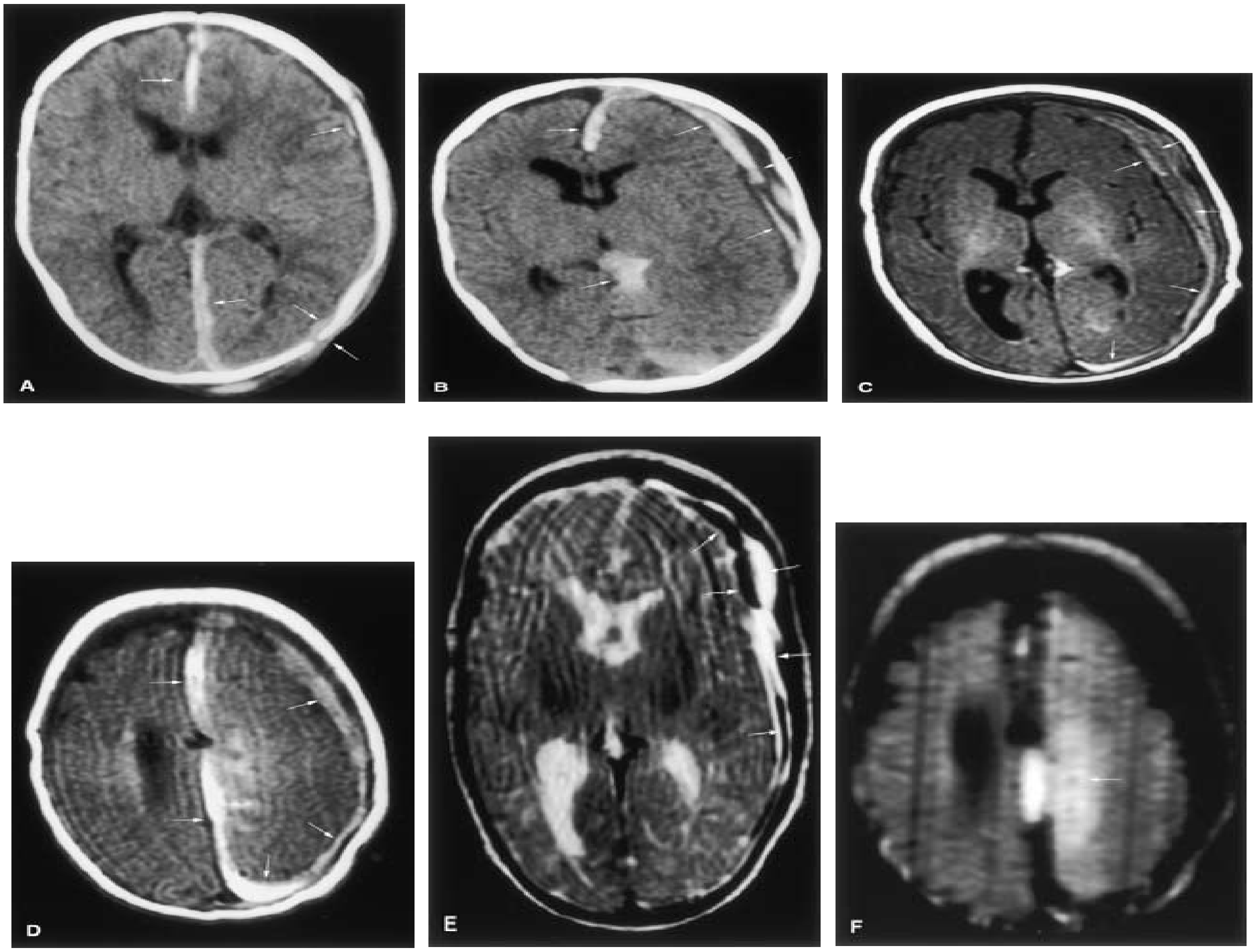

FIG. 1. Probable hyperacute-acute subdural hematoma (SDH), evolving chronic SDH, and chronic SDH with rehemorrhage in a 3-month-old boy. The child presented with seizures, lethargy, and retinal hemorrhages. **A:** Unenhanced axial computed tomography (CT) at initial presentation shows a large interhemispheric high-density hemorrhage (arrows), smaller left convexity extracerebral high-density hemorrhages (single crossed arrows) within low-density extracerebral collections, and a diastatic left parietal fracture (double crossed arrows) with overlying scalp swelling and hemorrhage. Magnetic resonance imaging (MRI) was not done at that time to assist with timing assessment. Following recovery from the acute injury, the infant was placed in protective custody for supervised visits by the parents. Seventeen days later, the child was readmitted to the hospital for acute seizures and neurologic deterioration. **B:** Unenhanced axial CT at that time shows a large high- and low-density left extracerebral collection (arrows) with swelling of the underlying left cerebral hemisphere and rightward shift of the midline markers. High-density hemorrhage also is seen in the interhemispheric fissure (single crossed arrow) and in the cavum velum interpositum (double crossed arrow). Two days later, axial T1 **(C, D)** and T2 **(E)** MRI show that the left extracerebral collection has T1-isointense and T2-hyperintense components (hyperacute unclotted portion; arrows) plus T1-hyperintense and T2-hypointense components (acute-to-early-subacute clotted portion; single crossed arrows). These findings are more consistent with a hyperacute-acute SDH (<3 days old) plus an early subacute SDH (<7 days old) than with an evolving chronic SDH or a chronic SDH with rehemorrhage (Table 1). In the latter, even higher T1 and T2 hyperintensities would be expected in a hemorrhage that is 19 days old. MRI findings with regard to the interhemispheric and left posterior parietooccipital convexity collections are most consistent with a combination of chronic subdural hemorrhage and more recent rehemorrhage (double crossed arrows). Extensive hematologic workup ruled out a coagulopathy. **F:** Axial diffusion trace MRI shows a high-intensity ischemic left parasagittal cerebral infarction (arrow) likely related to subfalcine herniation.

gical intervention (e.g., an expanding hemorrhagic collection) and as an additional guide for the medical management of increased intracranial pressure (e.g., cerebral edema). Repeat or serial imaging may be necessary. Particularly in the unstable infant, initial and repeat cranial US with Doppler at the bedside may not only be an effective method of evaluating structural abnormalities (e.g., white matter tears), but also may be used for monitoring

alterations in cerebral blood flow and intracranial pressure (23).

Magnetic resonance imaging

Multiplanar MR probably is the most important technique for assessing the pattern, extent, and timing of the injury(s), particularly in the absence of characteristic findings on CT (10,22,23,31,32,76,84,85). MRI should be done as soon after the presentation as the child's condition allows, and a follow-up examination within 7 to 10 days may be needed. T1 and T2 spin-echo (SE) sequences are necessary for characterizing the nature and timing of hemorrhages and other collections as to hyperacute, acute, subacute, or chronic using established criteria (Table 1 and Fig. 1) (10,23,32,85). Fluid-attenuated inversion recovery (FLAIR) imaging is preferred over proton-density images because of its superior ability to specifically suppress CSF signal to assist in the identification of abnormal subarachnoid collections and very chronic subdural collections. FLAIR imaging also provides enhanced conspicuity for non-CSF lesions having long T2 relaxation times (e.g., subdural hematomas or hygromas, shear injury). Magnetically susceptible gradient-echo sequences (T2*) are important for identifying hemorrhagic lesions not shown by some SE techniques (e.g., fast SE), but cannot provide information on timing without supportive T1 and T2 findings, because acute hemorrhage (e.g., deoxyhemoglobin) and chronic hemorrhage (e.g., hemosiderin) both may appear T2* hypointense. Gadolinium-enhanced sequences may assist in the identification of otherwise subtle leptomeningeal changes. Magnetic resonance (MR) angiography and venography also may be important (i.e., to evaluate for venous thrombosis as a mimic of NAI/SBS) (10,23,32). Advanced MR techniques, such as diffusion, perfusion, and spectroscopy, may assist further with the detection, characterization, and timing of the injury components, especially for traumatic or hypoxic-ischemic axonal injury (10,23,32,84). However, there may be a significant interval (hours to days?) between one or more episodes of trauma, resulting in CNS hemorrhage(s) as the primary injury and subsequent deterioration of the child associated with progression of the hemorrhage, brain swelling, and/or hypoxic-ischemic injury as the secondary injury. To date, none of the current or advanced MRI techniques alone can reliably distinguish NAI from accidental injury to the CNS.

EXPERT MEDICAL TESTIMONY IN NAI

Expert medical witness

Expert witnesses are persons who have expertise on the subject at issue and who are designated according to qualifications by the court to instruct the jury (89,90). From a point of ethics, it is not only acceptable, but it is encouraged, for physicians to serve as expert witnesses in civil and criminal cases. The code of medical ethics of the council on ethical and judicial affairs of the American Medical Association (AMA) states that physicians are professionals with special training and experience and have an ethical obligation to assist in the administration of justice (91). The American College of Radiology (ACR) also states that it is in the public interest for medical expert testimony (by radiologists) be readily available (92). The AMA and ACR also have provided guidelines on the ethical and professional manner upon which expert medical witnesses conduct themselves. The AMA states that medical experts should have recent and substantive experience in the area in which they testify. The expert should testify honestly and truthfully to the best of his or her medical knowledge; should not become an advocate or a partisan in the legal proceeding; and should inform the attorney(s) for the party who calls upon the expert to provide all favorable and unfavorable information developed by the physician's evaluation. It is unethical for a physician to

TABLE 1. *Magnetic resonance imaging of intracranial hemorrhage and thrombosis*[a]

Stage	Biochemical form	Site	T1-MRI	T2-MRI
Hyperacute[b] (+edema) (<24 hours)	Fe II oxyHb	Intact RBCs	Iso-Low I	High I
Acute (+edema) (1–3 days)	Fe II deoxy Hb	Intact RBCs	Iso-Low I	Low I
Early subacute (+edema) (3–7 days)	Fe III metHb	Intact RBCs	High I	Low I
Late subacute (−edema) (1–2 weeks)	Fe III metHb	Lysed RBCs (extracellular)	High I	High I
Early chronic (−edema) (>2 weeks)	Fe III transferrin	Extracellular	High I	High I
Chronic (cavity)	Fe III ferritin and hemosiderin	Phagocytosis	Iso-Low I	Low I

[a] Modified from Barnes PD, Wolpert. MRI in Pediatric Neuroradiology. St. Louis: Mosby Year-Book, 1992; Kleinman P, Barnes P. Head trauma. In: Kleinman PK, ed. Imaging of Child abuse, 2nd ed. St. Louis: Mosby Year-Book, 1998; Bradley WG Jr. MR appearance of hemorrhage in the brain. Radiology 1993;189:15–26; does not include influence of fetal Hb.

[b] Computed tomography is more sensitive and more specific than magnetic resonance imaging or ultrasound for hyperacute/acute hemorrhage in all compartments.

RBCs, red blood cells; I, signal intensity; +, present; −, absent; Hb, hemoglobin; Fe II, ferrous; Fe III, ferric; Iso, isotense.

accept compensation that is contingent upon the outcome of the litigation (91). The ACR adds that the radiologist expert witness must maintain an active practice of radiology and be certified, familiar with, and actively involved in the clinical practice of the subject matter of the case for 3 of the previous 5 years at the time of the testimony (92). The testimony offered by the expert witness must be based upon a reasonable degree of medical or scientific certainty. That is, in the judgment of the expert witness, the causal relationship between an event and the outcome is probable, or more likely than not. The quality of the evidence, therefore, rises above speculation and conjecture and may be considered by the jury.

Expert medical review, consultation, and testimony

Theoretically, the opinion of the medical expert should be the same whether rendered for the defense or for the prosecution (or plaintiff) (89,90,93). An expert medical review for a criminal (or civil) case may involve the viewing of imaging examinations, imaging reports, medical records, chronology of events, complaint or charges filed, expert letters of opinions, and depositions of the clients, providers, treating physicians, and expert witnesses. In a consultation with a legal representative of the party(s) involved, the expert radiologist's findings and opinions are conveyed and discussed with regard to causation, including pattern of injury and timing (and/or standard of care when appropriate). Occasionally, legal counsel requests that the radiology expert confer directly with other experts retained by counsel. A formal letter of opinion may be requested that details the imaging findings and their significance with regard to causation. Imaging evidence should always stand independent of the clinical evidence. If the imaging findings or opinions alone cannot provide support for, or against, causation, then the case must be tried on the basis of the clinical evidence/opinions alone.

After the expert review, the attorney may request permission to officially designate the radiologist as an expert witness and submit the expert's qualifications and official letter of opinion to the court. Many, but not all, jurisdictions require that the expert witness be qualified according to certain guidelines. Federal guidelines (Daubert, Supreme Court 1993) require that the expert witness be qualified by providing certain credentials, which include a curriculum vita, documentation of clinical experience (>50% of practice in the field of expert testimony), an accounting of previous deposition and trial testimony, a fee schedule, percentage (or amount) of income derived annually from expert testimony, and a detailed written letter of opinion that includes a list of the pertinent medical literature (89–93). The letter of opinion may be offered along with other "evidence" for review by a screening panel or for mediation to effect resolution, a settlement, or a trial.

A discovery deposition then may be scheduled to "discover" the basis for the opinions offered by the expert witness to prepare for possible mediation, settlement, or trial (89,90,93). In some criminal cases, expert medical testimony may be presented as part of a grand jury investigation. During the recorded deposition under oath, the expert witness is questioned regarding qualifications, clinical and expert witness experience, expert fees and income, all materials reviewed (offered as numbered exhibits) and relied upon as the basis for the expert's opinions in the case at hand, and the expert's opinions. The expert is specifically questioned regarding his or her intention to offer opinions on causation or standard of care, when applicable. The expert witness may be asked to assist the referring counsel in preparing strategy for the depositions of the adversary expert witnesses testifying in the same field of expertise.

In preparation for trial, the expert witness organizes and re-reviews all materials and exhibits, including the expert's own discovery deposition transcript. The expert may be asked to review certain medical records, other expert opinions, and other depositions, including those of the adversarial expert witnesses (89,90,93). Occasionally, legal counsel requests that the radiology expert confer with other experts before testifying. A pretrial conference often is scheduled for expert testimony preparation and rehearsal and to assist counsel in preparing strategy for cross-examination of adversarial expert witnesses. At trial, the expert witness is initially presented to the court (judge and/or jury) for direct examination by the retaining legal counsel to establish the expert's qualifications and to present the expert's opinions as supported by the evidentiary exhibits (89,90,93). The witness then undergoes cross-examination by the adversary legal counsel. The judge may allow for "re-direct" and for "re-cross" by the retaining and adversary counsels, respectively. It is advisable to use only the imaging films (i.e., direct evidence) as exhibits and not to use "artist-rendered" medical illustrations (i.e., indirect or potentially "fabricated" evidence) for presentation to the jury.

It is important for the expert witness to give the entirety of his or her opinion during direct examination by the retaining counsel and to address all pertinent issues (89,90,93). This will prevent the witness from being ambushed by the adversarial counsel during the cross-examination regarding issues not covered in the expert's direct testimony. The expert otherwise must rely on subsequent re-direct examination by the retaining counsel to clarify certain parts of the testimony. The expert witness must speak directly to the jurors in clear and understand-

able language, and with respect. Although the testimony is conveyed in response to questions (including hypotheticals) posed by counsel, the expert should avoid being led or translated by counsel. The expert should testify by presenting the findings/opinions in a logical sequence or chronology and by specifying the level of medical and/or scientific certainty based upon the expert's cited experience and/or published literature (if allowed). Repeating important points and providing a summary may be critical. During cross-examination, the expert witness must be especially careful not to hastily agree with adversarial counsel's translation of the expert's testimony.

Irresponsible medical testimony: Jurors and jurists beware

Irresponsible medical testimony may be manifested in a number of ways (93,94). The irresponsible expert may not possess the proper qualifications. Unique theories of causation may be offered that are not supported by the pertinent medical literature, or the expert may use nonpertinent literature or may misrepresent the literature. The witness may offer unique or unusual interpretations of medical findings, or may allege nonexistent findings. The expert may misquote well-known journals or texts, make false statements, or omit important facts or knowledge pertinent to opinions being offered. Jurors and jurists should be beware of the following: complicated and technical expert testimony; constant leading or translation by counsel; use of manufactured and elaborate exhibits instead of the original medical evidence; expert opinions based upon the opinions of other experts retained by the same counsel; expert opinions offered outside one's specific field of expertise; not providing an expert in a field pertinent to the case; scripted or choreographed testimony by experts retained by same counsel; experts offering unique theories that are contrary to the prevailing literature and are not supported by the pertinent literature; expert testimony that contradicts expert's own publications; counsel's use of the phrase "experts on both sides agree"; and constant misrepresentation of expert testimony and scientific evidence by counsel in an attempt to confuse the jury. A number of remedies have been offered or instituted to deal with irresponsible medical testimony, including Federal guidelines (see earlier), peer review of expert testimony by professional societies, establishment of a general pool for expert witness fees, and use of expert panels or screening panels.

Recommendations for pediatric neuroradiology

The pediatric neuroradiologist should describe the imaging findings in detail, including the pattern, distribution, and severity of injury with regard to scalp, skull, and intracranial abnormalities. including extracerebral collections and brain injury. A differential diagnosis is given and timing ranges are provided if possible. If NAI is at issue or suspected, then the radiologist must directly communicate the imaging findings to the primary care team and be available to consult with child protection services, other medical or surgical consults, including the pathologist or biomechanical specialist, law enforcement investigators, and attorneys for all parties, as appropriate. Pattern of injury and time parameters, as may be provided by MRI, are particularly important with regard to correlations with events as reported by witnesses and potential suspects. For example, such information may exonerate the last individual who was with the victim and implicate others who had access to the child before that time. This classically occurs when a previously injured child is left with a baby-sitter or at day care and then later collapses. The last caretaker then is charged with assault. In one such "homicide" case, a CT performed within a few hours of the alleged assault by a baby-sitter showed that the infant had a high-density subdural hemorrhage consistent with "acute" injury (95). An MRI performed shortly thereafter showed that the subdural hemorrhage was T1 hyperintense with some areas of T2 hypointensity and T2 hyperintensity. Using established criteria (Table 1), the MRI findings indicated that the injury was at least 3 days old. The State's expert witnesses ignored the MRI findings regarding timing and testified that the injury was acute SBS and could only be inflicted by the defendant. Nonetheless, the baby-sitter who had only been with the infant for a short time before the collapse was acquitted (95).

The radiologist must be aware of certain conditions that are known to have clinical and imaging features that may mimic abuse (16,32). These include accidental injury, certain coagulopathies, vascular diseases, infectious or postinfectious conditions (e.g., postvaccinal), metabolic disorders, neoplastic diseases, certain therapies, and some congenital and dysplastic disorders. The physician should not only rule them out, but also must consider the possibility of combined, or multifactorial, mechanisms with synergistic effects (i.e., an underlying condition with superimposed nonaccidental, or accidental, trauma). A case in point is that of a young infant who became progressively ill following a diphtheria-pertussis-tetanus (DPT) vaccination and subsequently became apneic. The father attempted to revive the child by "shaking" and then attempted cardiopulmonary resuscitation (CPR). The infant also received CPR by the rescue team at the home and again at the hospital. Retinal hemorrhages were present on ophthalmologic examination. The father was charged with abuse. Subsequent CT imaging with CT venography showed a combination of enhancing cerebral infarctions,

venous thromboses, and subdural hemorrhages. An MRI was not done, although the CT findings were verified by postmortem neuropathologic examination. The evidence was consistent with a hemorrhagic meningoencephalitis, likely postvaccinial, although additional trauma, either accidental (e.g., CPR) or nonaccidental (e.g., shaking) could not be ruled out. The father was convicted on the basis that the State's experts testified that the injuries could only be caused by SBS (96). The conviction is under appeal.

CONCLUSION

Timely and thorough evaluation of alleged NAI cases clinically and radiologically, in addition to the social assessment, can make the difference between appropriate child protection and an improper breakup of the family or a wrongful indictment and conviction (10,23). Pediatric neuroradiology often plays a major role in this assessment.

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