Irresponsible Testimony by Medical Experts in Cases Involving the Physical Abuse and Neglect of Children

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Irresponsible testimony by medical experts is a growing problem exemplified by three particularly egregious cases reported herein. The types of irresponsible testimony include the witness's absence of proper qualifications, use of unique theories of causation, use of unique or very unusual interpretations of medical findings, alleging nonexistent medical findings, flagrant misquoting of medical journals or widely used texts, making false statements, and deliberate omission of pertinent facts or knowledge. Criteria to qualify as an expert witness in child abuse and neglect are proposed. Finally, physicians, lawyers, and their respective professional licensing boards and societies are urged to develop a process by which such testimony is exposed, peer reviewed, and used in credentialing.

INTRODUCTION AND LITERATURE REVIEW

Litigation of cases of physical abuse and neglect of children in criminal or dependency courts depends on the testimony of medical experts. The law requires that all parties have access to medical experts. In the adversarial process in the United States, medical experts are selected by the attorneys representing the prosecution, plaintiff, and defense to review and opine about the medical facts of these cases. In general, opinions of medical experts address whether the medical findings are caused by inflicted or unintentional injury or by some disease process that mimics injury.

Brent (1982) was the first to call attention to the widespread problem of irresponsible medical testimony. He cited malpractice cases in which medical experts testified that poor perinatal care caused morbidity in infants whose problems were due obviously to genetic or first trimester causes. He argued for greater emphasis on responsible expert testimony in medical and graduate education, for peer review of expert testimony, for limitation of expert witness fees, and a number of other reforms. In the 14 years since his article was published, we have seen little change in areas in which he sought to influence; therefore, his arguments remain particularly cogent.

After making similar observations, Weintraub (1995) asserts that "physicians have been quick to condemn the legal profession as the cause for the surge in medical malpractice lawsuits. However, in reality, the greater impetus has been the medical expert witness who has developed *unique theories of causation with consequent corruption of science* [italics added]" (p. 856). He also argues for not only intensive peer review but also widespread exposure of irresponsible expert testimony to peers.

Chadwick (1990) reviews articles by Brent (1982) and Diamond (1959, 1988) and the American Academy of Pediatrics Committee on Medical Liability (1989) while discussing the ethics of expert testimony. He notes that, at present, authorities differ on the question of whether a medical expert should be court appointed and "objective" or (as is almost always the case) recruited by an attorney and "adversarial." He also renewed the call for peer review and suggested a more restrictive definition for expertise. He provides definitions for the qualifications a physician should possess to qualify as an expert in child abuse cases, including ongoing clinical or laboratory work with abuse cases.

Expert witnesses are expected to provide opinions about the interpretation of fact sets for which they may or may not have made the original observations.

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Although the offering of an opinion is a process that is somewhat less objective than the observation of a fact, expert witnesses are still sworn to tell the truth. A reasonable expectation about the meaning of truth telling in the context of offering an opinion might be that if an opinion is divergent from generally accepted medical knowledge, the expert should acknowledge this fact and should avoid "unique theories of causation" and other irresponsible positions. For example, if the expert must hypothesize a previously undescribed medical condition to explain pathology, the conjectural nature of this stretch should be pointed out as part of the duty to tell "the whole truth."

At this stage of our knowledge, this subject can be approached only anecdotally. Therefore, this report proposes criteria for irresponsible testimony and presents three cases that illustrate particularly egregious examples of its use. We will arbitrarily restrict our article to cases of physical abuse and neglect. Comparable cases involving sexual abuse and emotional abuse can also be easily identified. In the process, we will attempt to develop a working definition of the phenomenon. Determination of its prevalence in the United States might then be undertaken through a survey.

METHOD

We propose the following criteria to define irresponsible medical testimony:

- Absence of proper qualifications, as defined by Chadwick (1990), for example, testimony about the origin of a child's injuries by a physician with no experience with children or with injuries.
- Use of unique theories of causation, for example, testifying that an accident during pregnancy might be the cause of the birth of an infant with Downs' syndrome.
- Use of unique or very unusual interpretations of medical findings, for example, suggesting that histiocytes in the spleen indicate a coagulopathy.
- Alleging nonexistent medical findings.
- Flagrant misquoting of medical journals or widely used texts.
- Making false statements.
- Deliberate omission of important facts or knowledge pertinent to the opinions being offered.

Other forms of irresponsible testimony will doubtless be described in the future.

CASE REPORTS

The cases presented herein have been culled from the experience of the authors and are intended to illustrate examples of irresponsible medical testimony.

Case 1: Shaken-Infant Syndrome With Calcified Cephalohematoma

A father telephoned 911 to report that his 5-week-old infant became limp and blue while he was bouncing him on his knee. Responding emergency medical technicians found the infant apneic with a pulse and assisted his ventilation en route to a nearby emergency department. He was stabilized during the next hour and then transported to Children's Hospital. Cardiac compressions were never required.

The birth history revealed a prolonged second stage of labor, and birth was facilitated by suction extraction. The initial Apgar score was 4, but it improved to 9 after 1 minute; thereafter, the infant appeared well. The newborn physical examination revealed a cephalohematoma. The infant remained asymptomatic and thrived until 5 weeks of age.

Admission physical examination on arrival revealed sluggishly reactive pupils that soon became fixed. A calcified cephalohematoma was detected. The remainder of the physical examination and skeletal radiographs were otherwise normal. The first head CT scan, done on arrival at the hospital, demonstrated diastasis of the sagittal suture, effacement of ventricles and basal cisterns, and subarachnoid blood in the interhemispheric fissure adjacent to the tentorium and in the right Sylvian fissure. The gray and white matter densities were reversed. The calcified cephalohematoma was confirmed.

During his hospital course, the intracranial pressure remained from admission over 40mmHg through 48 hours of intensive treatment. Retinal hemorrhages were not seen by an ophthalmologist on the 3rd hospital day. An initial hematocrit (Hct) of 17.8% was corrected with transfusion of packed red blood cells. The initial coagulation studies showed moderate elevations of prothrombin time (PT) and partial thromboplastin time (PTT) and mild depression of fibrinogen to 165 mg/dl. Fresh frozen plasma was given on the 2nd and 3rd hospital days. Clinical significant bleeding was never observed despite insertion of multiple percutaneous lines and an arterial cutdown. Throughout the hospital course, ventilation of the infant and maintenance of blood gases was never a problem. Because extracranial trauma was not identified, the parents consented to donation of the heart and liver. Two hours later, brain death was established.

During harvesting of the heart and liver, the heart showed only a small epicardial hematoma but no other sign of injury. Partly because of the intention to donate the liver, aspartate aminotransferase (AST) was determined on seven occasions. The first value (in the Emergency Department) was 144 IU/dl; five sub-

sequent values were less than 40 IU/ml and another were 50 IU/ml.

Autopsy by the medical examiner (ME) following organ donation confirmed the old calcified cephalohematoma but no skull fractures. There was a 2 cm hemorrhage around the scalp ICP monitor site. A diffuse, thin subdural hematoma, with a total volume of about 30 ml, covered the cerebral hemispheres and extended to the base of the skull. Extensive subarachnoid hemorrhage and severe cerebral edema were present. Postmortem skeletal survey revealed a healing fracture of the right seventh rib.

Neuropathological examination confirmed the extensive brain swelling and the presence of acute subdural and subarachnoid bleeding. Chronic encephalopathic changes and a subdural membrane were not identified. The gray-white junctions were blurred. The defense pathologists requested Prussian blue stains of the sections of the dura that included the subdural blood. These demonstrated hemosiderin-laden macrophages.

Expert Testimony

The prosecution experts were the ME, a children's hospital pediatrician who frequently provides consultation in cases of suspected child abuse, and a physician who publishes and speaks widely about Shaken Infant Syndrome. Each testified that the infant's condition was due to inflicted injury that must have occurred at about the time of major change in state of consciousness.

Two inadequately qualified witnesses, who employed unique theories of causation and unique interpretations of findings and who misquoted the literature, provided the medical testimony for the defense. The first has had prior experience as an ME, but has not been employed formally by an ME's office for greater than 10 years. During this latter period, he has performed only forensic consultations and expert testimony involving cases in which he had no hands-on involvement. We will refer to him as the professional witness (PW). The other is employed primarily in a hospital without pediatric patients or services and works occasionally at a medical school's primary teaching hospital. The latter hospital rarely cares for seriously injured children because the community's children's hospital is the designated pediatric trauma center. The ME performs all autopsies on all injured children who die in the region. The ME's office does not use the services of this pathologist, who we will refer to as the adult hospital pathologist (AHP).

The defense theorized that rebleeding of a subdural hematoma 6 weeks after a birth injury was the cause of the infant's death. Both defense pathologists provided testimony in support of that theory despite the absence of any membrane formation enclosing the subdural hematoma. Testifying for the defense, the PW made statements (essentially) as follows:

1. The presence of overriding sutures (noted in the infant's newborn exam) indicated a strong likelihood of a tear of the sagittal sinus at the time of birth.
2. The presence of hemosiderin in or near the blood clot indicated that the bleeding was at least 35 to 90 days old.
3. Newly formed blood vessels in the region of the clot indicated that it was old. The ME or his consulting neuropathologist did not describe (these vessels).
4. The initiating injury immediately preceding the fatal episode affecting the infant's brain was minimal, and his "severe, massive hemorrhage" came later in the hospital.

Our rebuttal comments prepared for this article are the following:

Unique interpretations of findings:

1. The presence of overriding sutures in the immediate newborn period is a common normal finding in infants delivered vaginally and has no clinical significance.
2. Stainable hemosiderin may be found in macrophages in proximity to hematomas as early as 3 days after injury (Leestma, 1988; Stocker & Dehner, 1992).
3. In our opinion, these "newly-formed vessels" are not, in fact, newly formed but rather nutrient vessels seen in the delicate layers of the dura in individuals of all ages.
4. The first CT scan done promptly on arrival at the hospital revealed changes in the brain due to swelling that are catastrophic and usually associated with a fatal outcome. The initial ICP measurement of 40mmHg is in the same category. There never was a "massive hemorrhage." The subdural volume was small as is typical of head injury cases with early and severe brain swelling. This statement suggests that the PW has a limited understanding of the pathogenesis of children's brain injuries and, in addition, ignored the gross autopsy findings recorded by the ME.

The AHP testified for the defense (essentially) as follows:

1. Death in this infant was due to three factors: a brain injury that occurred at birth, pneumonia, and liver disease.
2. The presence of hemosiderin in the tissues near the clot indicated an injury at least 4 weeks prior to death. He cited a chart in Leestma's (1988, p. 216) *Forensic Neuropathology* text to support his statement.
3. A single AST value of 144 IU/ml, the only elevated value of seven determinations of this enzyme, proved that the infant had chronic liver disease, and further,

that this chronic liver disease would have caused a coagulopathy.
4. The rib fracture occurred at birth. (He stated this as "fact," and did not acknowledge that precise histologic dating of rib fractures is not possible but rather encompasses a considerable range of duration.)
5. The infant should have been treated with heparin for his coagulopathy.
6. He was a pediatric pathologist.

Our rebuttal comments follow:

1. All of these assertions are based on unique interpretations of findings (see below). The finding of "pneumonia" was no more than is usual with infants who die on ventilators, and ventilation with low pressures was easily accomplished in this infant.
2. Leestma's *Forensic Neuropathology* (1988) states that hemosiderin can be observed within 3 days after injury. The same statement is found in Stocker and Dehner's *Pediatric Pathology* (1992). The witness's statement is an incorrect citation of literature.
3. Few clinicians or clinical pathologists would consider a diagnosis of chronic liver disease based on these findings.
4. Although histological dating of rib fractures in infants may have value, we are aware of are no published data that allow an unequivocal statement of this kind.
5. Few, if any, pediatric clinicians would use heparin in this situation.
6. The statement is false.

A report of suspected child abuse was made, but after investigation, law enforcement officials and Child Protective Services did not intervene.

Case 2: Lethal Head Trauma

This case differs from Cases 1 and 3 in that the opinions were rendered in letters to the Court solicited by an attorney representing the man who had been convicted previously of the homicide of his girlfriend's son and who was serving a sentence for this crime. The attorney used these expert opinions in support of a petition for a new trial based on new evidence, that is, the new opinions of pathologists about the original set of medical facts.

The original facts leading to an expert opinion of nonaccidental head injury were as follows. A 2½-year-old boy was the subject of a call to 911 by the mother's boyfriend, who was the sole caretaker at that time for him and another child. Responding paramedics found the child unconscious and in respiratory arrest. His pulse was palpable; blood pressure was present, and active bleeding extended from three parallel lacerations on his upper posterior neck onto a towel placed by the caretaker. The man with him said that child had fallen from a couch and struck his head on a brick fireplace and a glass ashtray then convulsed and stopped breathing.

Curiously, in view of the large neck lacerations that were still oozing when the child was admitted to the hospital, blood was not found at the scene where the fall was stated to have occurred. Multiple glass shards from an ashtray on which he was said to have fallen were found, yet none had blood on them.

When admitted to the hospital, the child was found to have a thin subdural hematoma, soft tissue swelling of the scalp, a left parietal skull fracture, severe brain swelling, and intracranial pressure equal to mean arterial pressure. There was an old healing fracture of the right second metacarpal. Large bilateral retinal hemorrhages were present. There were lacerations on the neck and bruising behind the left ear and on the upper and lower back, left shoulder, and right buttock. The PT was slightly elevated at 15.5 seconds, and the PTT was normal at 39 seconds. Despite the placement of percutaneous lines, clinically significant bleeding did not occur during the child's 24-hour hospitalization prior to death.

Three months before his death, the child had been admitted to a hospital appearing "shocky," with multiple bruises including abdominal bruises, a tender abdomen, and studies suggestive of intra-abdominal bleeding. Rupture of the spleen was suspected but not proven. The coagulation studies were normal. He was managed conservatively and recovered. After this hospitalization, he had additional episodes of unexplained bruising. Hematological consultation and coagulation studies revealed no blood abnormalities. A report of suspected child abuse was made, but after investigation, law enforcement officials and Child Protective Services did not intervene.

The autopsy by the ME confirmed that the child died from a head injury. A subdural hematoma with an impact site on the left side of the head and brain swelling were identified along with bruises and lacerations. The skull fracture seen by a radiologist was not confirmed at autopsy. The ME determined the cause of death to be closed head injury and the manner to be homicide.

The mother's boyfriend was convicted after testimony showed that he was present each time the child was injured—including the occasion of the

fatal episode—and on expert testimony to the effect that the injuries were inflicted.

In preparing his defense, his attorney had consulted an internationally preeminent pediatric pathologist and a widely published and respected pediatric neuropathologist, both of whom provided written opinions. The defense deemed their opinions to be unhelpful; therefore, these experts were not called to testify and their reports were not introduced into evidence.

Ten years later, the petition for retrial was introduced with great public fanfare including a front-page article in the local newspaper (Okerblom & Wilkens, 1994). The headline proclaimed "A decade of doubt" and included a quote from a new defense pathologist, an ME from a northeastern state referred to as the eastern medical examiner (EME): "I think the guy paid ten years of his life for nothing." A second new defense pathologist (the AHP for Case 1) was quoted that he agreed that the child had died of natural causes.

Expert Testimony

In written statements submitted to the Court, both of these new pathologists stated the following (copies available from the authors):

1. Even though the opinions of the original defense experts declared that there was no evidence of an underlying coagulopathic disorder, the EME expressed the opinion in a sworn declaration that "I then caused the spleen section to be examined at the Department of Pathology at the Duke University Medical Center. This spleen examination was never previously conducted and it indicated that there was, indeed, a hematologic [bleeding] abnormality existing in the person of [the child] on the date of his injury and/or death."
2. The report from Duke University was appended to that of the EME and provided the following conclusion: "Diagnosis: A. 'Spleen for immunocytochemistry.' Spleen with scattered large cells positively stained for IGM."
3. The AHP concurred with the EME's opinion and went on to say, "I will not unduly burden the Court by reciting all of the evidence that leads me to believe that [the defendant] received incompetent forensic assistance at his first trial. I do concur, however, in all of the facts and opinions set forth in [the EME] Declaration, and affirmatively join in his request for a new trial."
4. The EME (with concurrence from the AHP) opined that mildly abnormal coagulation tests obtained after signs of brain infarction were indicators of a *pre-existing* coagulopathy.
5. The pathologists consulted by the defense at the original trial had not performed a competent evaluation of the case, because they failed to find a pre-existing coagulopathy.

Our rebuttal comments follow:

1. Because of the episode of intra-abdominal bleeding and later unexplained bruises, this child had had two sets of coagulation tests done in the 6 months preceding his death, with normal results on both occasions. The first set included a PT, a PTT, and a platelet count. The second added a bleeding time, and on this occasion, a pediatric hematologist who took a careful history and did a clinical assessment for coagulation problems saw the patient and his mother. There may never be another case in which coagulopathy has been more completely excluded. The idea that a coagulopathy of any kind could be diagnosed by examining the spleen after death seems to be a wild conjecture, and making that diagnosis based on the finding of large cells containing IgM in the spleen is a unique causal theory.
2. The AHP joined in this unique causal theory.
3. The AHP has published extensively about many topics in clinical pathology and claims some expertise in working with trauma. It is unlikely that he could be unaware of the occurrence of coagulopathy in association with life-threatening head injuries at all ages. To propose pre-existing coagulopathy based on abnormal coagulation studies in the presence of brain infarcts is a unique interpretation of a finding.
5. The international preeminence and distinguished character of the pathologist who was consulted without use of his opinion by the defense at the original trial makes this assertion very unlikely to be true. However, it is likely that the new defense attorney seeking a retrial would need to characterize the prior defense as inadequate and would require the new pathologists to use language of this type.

Case 3: Homicidal Starvation

An emaciated 4-month-old White female infant was admitted through the emergency room to the hospital in shock. A concerned neighbor had notified Child Protective Services after finding the infant crying feebly in the care of a baby-sitter. The child had never been immunized.

Admission physical examination revealed a body weight of 2.7 kg, which was less than the 5th percentile and less than her birth weight of 2.94 kg. The occipital frontal circumference was 35 cm. Her temperature was 92.1 degrees F, respirations 35/minute, pulse 154/minute and blood pressure 93/54 mm Hg. There were numerous joint contractures, generalized flaking and oozing of her skin, chapped and bleeding lips, and decubitus ulcers. The occipital skin was ulcerated, the occipital bone was depressed, and the anterior fontanelle was small and sunken. The eyes were sunken and the corneas were hazy. Subcutaneous soft tissue was essentially absent. Her oropharynx was dry. Her liver was palpated 3 cm below the costal margin. Pitting edema was present in the legs. Her fingernails were long.

Cultures from the skin and a neck wound grew Staphylococcus aureas, beta hemolytic Streptococcus and Candida albicans. Throat and urine cultures grew Candida albicans. HIV was negative by ELISA. Very low levels of plasma alanine, citrulline, alpha aminobutyric acid, valine, isoleucine, leucine, tyrosine, and tryptophan were interpreted to represent very low protein intake. The initial head CT was unremarkable.

Her clinical course, despite vigorous support, was characterized by progressive deterioration with intermittent atrial fibrillation, disseminated intravascular coagulation, bradycardia, and hypotension. She was administered antibiotics, vitamin and mineral replacements, and multiple units of packed erythrocytes, platelets, and fresh frozen plasma. Large fluid volumes were given to compensate for losses through the skin. Parenteral nutrition was also administered. She progressively deteriorated and expired on the 7th hospital day.

Postmortem examination by the ME revealed the body of an emaciated, slightly edematous infant who weighed 3.71 kg. The crown-rump length was 35.5 cm, head circumference 35 cm, chest circumference 35 cm and abdominal circumference 34.4 cm. The crown heel was estimated at 54.0 cm but was difficult to measure because of hip and knee contractures. The occiput was flattened. Hair was sparse, being present mostly on the top. Jaundice was not present. Petechiae were noted in the left lower conjunctive. The diffusely desquamated skin appeared moist and erythematous. Subcutaneous fat was essentially absent. A thrombus was adherent to the junction of the superior vena cava and right atrium. The right and left lungs weighed 44 and 34 grams, respectively, and were markedly congested but not consolidated or abscessed. The enlarged liver weighed 349 grams, and the cut surfaces were yellow and flabby. Left lateral frontoparietal scalp and subgaleal hemorrhages were associated with a needle puncture mark. The 400-gram brain was symmetrical with normal gyral development; however, the sulci were widened.

Microscopic examination revealed disseminated candidiasis with microabscesses in the brain, lungs, heart, stomach, thyroid, skin, subcutis, kidneys, right eye, and periadrenal soft tissue. The skin showed epidermal degeneration and necrosis associated with focal invasive candidiasis. Mild subacute inflammation was present at these sites. Subcutaneous fat was essentially absent; what little remained showed mucinous degeneration. The thymus revealed severe atrophy characterized by medullary stem preservation; very severe lymphocyte depletion; crowded, cystically dilated Hassall's corpuscles; and widened interlobular septa. Dysplasia was not present. The lymph nodes displayed lymphocyte depletion; however, plasma cells and atrophic follicles without germinal centers persisted. The lungs were diffusely congested and focally edematous. The liver revealed diffuse, macrovacuolar severe fatty change and absence of iron stores. Adrenal cortical lipid was diffusely depleted. Splenic lymphoid tissue was atrophic but present. Germinal centers were not present. The thyroid showed follicular enlargement with flattened epithelium. The rib displayed thin medullary trabeculae, delayed enchondral ossification, and normocellular marrow. Plasma cells were identified in the bone marrow and intestinal mucosa. The small intestine showed focal villous atrophy; however, lymphoid tissue was present but atrophic.

Defense Medical Expert Testimony and Our Rebuttal Testimony

The medical experts for the prosecution were the ME and an experienced pediatric pathologist who has performed and reviewed hundreds of forensic postmortem examinations. They opined independently that the decedent's cause of death was disseminated candidiasis secondary to chronic starvation dating since birth, and that the manner of death was homicide. They based their opinions on laboratory data summarized in Table 1, the postmortem gross and microscopic findings and microbiologic cultures. The decedent did not have the facies, thymic, or parathyroid hypoplasia or aplasia, and/or congenital cardiovascular lesions characteristic of Di George syndrome (Jones, 1997). The low calcium and ionized calcium levels were considered secondary to low serum albumin and the severely ill state of the infant on admission to the hospital (Lynch, 1990; Zaloga & Chernow, 1986). Subsequent calcium studies were within the reference ranges.

The medical expert for the defense was not only unqualified but also misrepresented his qualifications. The defense medical expert testified he was not qualified as an expert in child abuse or neglect, admitting to having had no training in these specific areas. Furthermore, he stated that he was not a forensic pathologist and that his last training in forensic pathology had occurred approximately 35 years prior to his testimony in this case. He is not board certified in forensic pathology. He was unable to opine if his observations from the postmortem record and microscopic slides were a result of abuse or neglect. In subsequent testimony, he stated that he was a pediatric pathologist despite the fact that he is employed primarily in a hospital without a pediatric service and has neither had a fellowship in pediatric pathology nor is board certified in this specialty.

TABLE 1: Summary of Laboratory Data During Hospitalization

Test	*Value*	*Reference Range*
Hct	20.3%	32-40%
WBC	22 K	6-14 K
Platelet	10 K	140-440 K
PT	18.4 seconds	11.4-13.4 seconds
PTT	50.9 seconds	24-36 seconds
Fibrinogen	57	200-400 mg/dl
Absolute lymphocyte counts	7,208-13,920	1,800-9,000
IgG level	537 mg/dl	500-1,200 mg/dl
Total protein	3.1 gm/dl	6.1-8.5 g/dl
Albumin	1.9 gm/dl	3.4-5.4 g/dl
Folate	19.4	> 3.6 ng/dl
Sodium	129 meq/dl	134-144 meq/dl
Magnesium	1.2-2.4 mg/dl	1.8-3.4 mg/dl
Calcium	7.0 gm/dl	8.8-10.8 mg/dl
Ionized calcium	3.9 mg/dl	4.5-5.3 mg/dl
TSH	20.6 IU/ml	> 0.38-6.1 IU/ml
T4	4.0 mcg/dl	4.9-10.7 mcg/dl
Folate	19.4 ng/dl	> 3.6 ng/dl
Vitamin A	20 mcg/dl	30-95 mcg/dl
Zinc	50 mcg/dl	60-130 mcg/dl

The medical expert for the defense concluded that the infant died of disseminated candidiasis secondary to Di George syndrome, and that the manner of death was natural (a copy of the transcript is available from the authors). His interpretations of the clinical and postmortem findings are not only unique but also relied on his misquoting of relevant, widely used medical literature and texts. His opinion that the decedent had Di George syndrome was based partially on flattening of the infant's occiput and a groove in the upper lip. This allegation is not substantiated in the description of Di George syndrome in *Smith's Recognizable Patterns of Human Malformations* (Jones, 1997). The prosecution's medical expert interpreted the flattened occiput to be a result of prolonged immobilization from birth and the lip groove to be incidental.

His interpretation that the small size of the thymus represented hypoplasia rather than atrophy was alleged to support his diagnosis of Di George syndrome. The distinction between these two can be clarified easily by histologic criteria (Robbins & Cotran, 1989). In reaching this interpretation, he misquoted the AFIP Fascicle titled "Tumors of the Thymus" (Rosai & Levine, 1975) and ignored another article regarding thymic involution in one of the most commonly read journals specializing in pathology (Van Vaarlen, Schuurman, & Huber, 1988).

The admission calcium and subsequent ionized calcium levels were decreased; these findings were used by the medical expert for the defense to support his diagnosis of Di George syndrome. Although the parathyroid glands were not looked for at autopsy, they were likely present because there had been no prior history of clinical symptoms related to hypocalcemia, and the values corrected quickly with medical therapy. The decedent's low total serum calcium can be ascribed to decreased serum albumin secondary to starvation, and reductions in ionized calcium are reported in association with sepsis and severe illness (Lynch, 1990; Zaloga & Chernow, 1986).

The medical expert for the defense attempted to support further his diagnosis of Di George syndrome by claiming the decedent had hypertelorism, a condition not confirmed using reference values in *Smith's Recognizable Patterns of Human Malformations* (Jones, 1997). He testified that a diagnosis of chronic malnutrition required total absence of subcutaneous fat. His subsequent testimony (available from the authors) and standard references (Ophoven, 1992; Robbins & Cotran, 1989) refute his initial allegation.

RESULTS

These three cases are summarized in Table 2. The types of irresponsible testimony by the medical experts for the defense in these three cases are summarized in Table 3.

DISCUSSION

In an earlier article, Chadwick (1990) opined that a medical expert should be able to demonstrate relevant training or experience in child abuse cases that are similar to ones in which he has been called on to provide testimony. Based partially on the particularly

TABLE 2: Case Summaries and Verdicts

Case	*Defense Diagnoses, Manner of Death*	*Prosecution Diagnoses, Manner of Death*	*Jury Verdict*
1	Head injury at birth with rebleeding, accidental	Shaken Infant Syndrome with calcified cephalohematoma, homicide	Acquittal
2	Coagulopathy, natural	Head trauma, homicide	Conviction
3	Disseminated candidiasis, Di George syndrome, natural	Disseminated candidiasis, starvation, homicide	Conviction

TABLE 3: Nature of Irresponsible Testimony by Medical Experts Testifying for the Defense

	Case		
Indicator	*1*	*2*	*3*
Lack of qualifications	Yes	Yes	Yes
Unique theories of causation	Yes	Yes	Yes
Unique interpretation of findings	Yes	Yes	Yes
Misquoting of literature	Yes		Yes
False statements	Yes		Yes

egregious cases presented herein, we believe that the criteria to qualify as a medical expert in physical abuse must be defined and delineated more precisely. For this, we propose that medical experts be able to document for the Court the following:

1. General training or experience in child abuse and neglect.
2. Specific training or experience relative to the particular type of case being adjudicated.
3. Memberships in relevant professional societies.
4. Child abuse and neglect conference presentations and attendance.
5. Relevant professional publications.

Expertise in these areas can be developed from clinical experience or from clinical research. On the other hand, experience in giving testimony at depositions and in court should not masquerade as *medical* expertise in any specialty.

Clinicians or pathologists who serve as expert witnesses must be able to provide a qualified opinion as to whether a child was a victim of inflicted injuries or neglect. To do this, medical experts in this area must have knowledge of natural, medical disorders associated with bone fractures, easy bruising, and ostensible sudden, unexpected infant death.

Pediatricians providing physical abuse consultations in hospitals, emergency departments, shelters, and outpatient settings are qualified to provide this type of expert testimony. Physicians who care for injured children but never offer medicolegal interpretations about the cause and manner of these injuries are less qualified as experts.

Pathologists should have performed or assisted in autopsies of suspected child abuse and have consulted in yet additional cases. Clinical pathologists can contribute in cases in which the interpretation of laboratory results is particularly relevant. For example, hematopathologists are qualified to rebut specious allegations of congenital or acquired coagulopathies in the pathogenesis of bruises. In general, however, forensic and anatomic pathologists with substantial experience in pediatric pathology should comprise the major pool of experts.

The testimony of pediatric radiologists can be crucial in the differentiation of child abuse from accidental fractures, osteopenia of prematurity, and bone disorders, such as osteogenesis imperfecta.

We propose to expand the list of various types of irresponsible medical testimony beyond the witness simply being unqualified or misstating one's qualifications. We now include unique theories of causation, unusual interpretations, misquoting of standard journals and texts, making false statements, and deliberately omitting important facts or knowledge as it pertains to the opinion being offered. The three cases presented herein illustrate several of these types of irresponsible testimony, and more important, represent in our opinion only the surface of a widespread problem.

In the final analysis, we believe the time has come for physicians and lawyers and their respective professional societies to begin a process by which such unsavory testimony can be exposed, peer reviewed, and ultimately prevented. Presently, expert witness testimony is never subjected to peer review. In addition, laypersons will always find it difficult to determine whether experts are truly qualified to offer testimony in many of these cases. This imposes responsibility on individual physicians to not knowingly enter into a case in which they do not possess appropriate experience and expertise. To do so is irresponsible and unethical. Nevertheless, it is unlikely that self-regulation will prevent irresponsible and unqualified testimony. Therefore, we advocate peer review of expert testimony by qualified members of hospital medical staffs, medical societies, and medical licensing boards. The former could use such reviews in the

recredentialing of physicians who wish to obtain or maintain medical staff privileges.

Putting peer review of expert testimony into place is a feasible although somewhat formidable task. The problem is not restricted to child abuse cases and is probably more common in malpractice litigation, and it makes little sense to address it in connection with just one forensic medical area. An important first step would be formal recognition of the problem by the American Medical Association (AMA). David Priver, M.D., president of the San Diego County Medical Association, has introduced a resolution into the AMA House of Delegates calling on the AMA to initiate a review of the situation. Many, if not all, of the specialty medical societies must join in this process as well, and the AMA has the apparatus to communicate with them.

In the specific case of child abuse litigation, the American Academy of Pediatrics, the American Professional Society on the Abuse of Children, and the National Association of Medical Examiners must all play important roles through the establishment of written guidelines and through endorsement of definitions of irresponsible expert testimony.

The methods for peer review must be carefully considered and worked out in a way that does not produce expense beyond the capabilities of the professional societies that join together to carry out this work. Review of transcripts is extremely time-consuming but can be done without having to attend. Actual attendance at the trial by a "neutral" physician gives that reviewer a much quicker and more accurate idea of what is going on, and his or her presence in the courtroom is likely to have some effect in itself. Review of all trials involving expert medical testimony may be impossible, and, if so, the establishment of an effective sample size is a research task.

Despite these obvious and substantial problems, the task must be undertaken. The present situation is a disgrace to the medical profession, and our tolerance of it informs the justice system that we do not care. Ultimately, the justice system may give up on us.

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