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COMMENTARY

Denying the abusive head trauma denialists their day in court, one step at a time

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In this issue of *Pediatric Radiology*, Wright et al. [1] present a rigorous study of subdural rebleeding after abusive head trauma (AHT). Their findings have important clinical and medical–legal implications and thus warrant careful consideration by physicians tasked with interpreting neuroimaging findings in young children with closed head trauma.

As the authors explained in the opening sentences of their Introduction: "It is commonly opined that children who have chronic subdural hemorrhages (SDH) can sustain rebleeds with no or minor trauma. This proposition is often misused in the legal arena to argue that neurologically symptomatic children, often with accompanying radiologic evidence of acute parenchymal or other injuries, did not sustain abusive head trauma (AHT) but instead sustained a non-traumatic rebleed into a pre-existing chronic SDH." Thus, a relatively small cadre of defense attorneys and defense medical experts have repeatedly explained away the more logical argument that SDH rebleeding *with acute neurologic deterioration* is best explained by acute closed head trauma. In many cases, such testimony has created reasonable doubt where none should exist, resulting in some young victims of AHT being returned to their abusive caregivers.

To address this unproven theory head on, Wright et al. [1] described the frequency of subdural rebleeding after AHT, its predispositions, and its clinical presentations. They conducted a descriptive retrospective study of children younger than 3 years with SDH from AHT. The authors' study population was culled from children evaluated in a prior multicenter study of AHT. Although retrospective, the depth, breadth, complexity and rigor of data capture is impressive. Patient inclusion and exclusion criteria were rigorously defined, as were the criteria applied to sort patients as AHT vs. non-AHT. Clinical data were captured via chart review by the study child neurologist and child abuse pediatricians. Rebleeding was defined as "the presence or new development of higher/blood density collections within or in relation to prior low density SDHs." All completed (serial) neuroimaging studies (CT or MRI) were blindly re-interpreted by qualified pediatric neuroradiologists working in close coordination with pediatric neurosurgeons and child abuse pediatricians. The investigators applied precise a priori criteria to confirm or exclude rebleeding and to differentiate mixed-density acute, chronic, and acute and chronic SDH. They also applied a priori criteria to confirm or exclude ventriculomegaly, maximum SDH depth, brain atrophy, and macro- and microcephaly. They resolved disagreements by consensus.

One hundred sixty children younger than 3 years with SDH resulting from AHT were identified. Seventeen (10.6%) of 160 died during their initial hospitalization. The remaining 143 children became the study population. Of these 143 children with SDH caused by AHT, 85 (59.4%) were re-imaged during the study time limits (up to 1 year after initial head injury). Review of their completed serial CT or MRI imaging studies revealed that 54 (63.5%) of 85 rebled within 1 year after injury, based on consensus applying strict definitional criteria. Thus, the authors concluded that a minimum of 54 (37.8%) of 143 children with SDH from AHT who survived their initial hospitalization sustained rebleeds.

It is interesting to note *why* the 85 (59.4%) children with SDH secondary to AHT were re-imaged. For 79 (92.9%), reimaging occurred during routine follow-up care. These children were completely asymptomatic, lacking any signs of brain injury or dysfunction. Three children (3.5%) were reimaged for seizures that had continued unchanged since their initial hospitalization. Two (2.4%) were reimaged for

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abnormal head growth, and one child (1.2%) was reimaged because of a cerebrospinal fluid leak.

Fifty-one (94.4%) AHT rebleeds occurred within the same region where SDH had been recognized during the initial hospitalization. All rebleeds were within lower-density subdural collections that were visible when the rebleed was identified. Findings found to be most strongly associated with AHT-SDH rebleeding included brain atrophy, macrocephaly, ventriculomegaly and greater subdural depth. Most important, *none* of the 54 children with SDH rebleeding manifested any acute neurologic symptoms or signs that could be attributed to their rebleeding.

I am a child abuse pediatrician, not a neurosurgeon. Nevertheless, I contend that these results support my understanding [2] of the pathophysiology of chronic SDH and rebleeding in survivors of moderate to severe AHT: (1) the inertial (whiplash) head injury mechanisms so frequently experienced by victims of AHT trigger deep distortions in the region of the cranio-cervical junction; (2) these distortions manifest clinically as acute encephalopathy, and respiratory or circulatory compromise; (3) if severe or prolonged, the resulting diffuse brain hypoxia–ischemia can lead to cerebral infarcts, brain atrophy and ventriculomegaly ex-vacuo; (4) the resulting cranio-cerebral disproportion facilitates persistence of low intracranial pressure (ICP); (5) persistent low ICP favors the "evolution" of chronic SDH from acute SDH (a vast over-simplification), to chronic SDH rebleeding and chronic SDH expansion, potentially manifesting as macrocephaly. Stated much more simplistically, chronic SDHs begin as space-occupying lesions that expand and grow through spontaneous rebleeding to fill the void associated with post-traumatic brain volume loss.

Assuming some degree of generalizability, I believe that the most important clinical and medical–legal takeaways from this study are as follows: (1) young victims of AHT with thin-film SDH who fail to manifest acute cardiorespiratory compromise (and thus fail to develop brain atrophy) are unlikely to develop chronic SDHs that rebleed and expand; and (2) although SDH rebleeding is relatively common among survivors of AHT, rebleeding itself is very unlikely to trigger acute neurologic symptoms or new brain parenchymal injury. No such cases were observed in this study. Applying Hanley's rule, the authors estimated that the maximum predicted frequency of non-traumatic rebleeding that would be accompanied by acute neurologic dysfunction in children with prior SDH was 3.7%. As stated by the authors: "This lack of symptoms belies the often provided legal testimony that children initially presenting for acute neurologic symptoms who are found to have … acute and chronic SDH have rebled due to minor or no trauma." Instead, "Our results suggest that these children's acute neurologic symptoms should be attributed to new, serious abusive brain trauma." Amen.

It is sobering to have to accept that studies such as this one are essential to dispel the legal arguments of the relatively small number of AHT denialists who promulgate speculative and unproven causal theories to create reasonable doubt in courtrooms. AHT denialists have contended that shaking does not cause serious injury; that child abuse pediatricians rely solely on three clinical findings ("the triad") to diagnose AHT; that short falls are frequently fatal; and that the constellation of findings attributable to AHT can result from cerebral sinovenous thrombosis, isolated hypoxic–ischemic injury, lumbar puncture, dysphagic choking or vaccinations. Most germane to this study by Wright et al. [1], some defense medical experts have testified that spontaneous rebleeding into an unrecognized asymptomatic birth-related SDH can result in later spontaneous collapse, coma or death. How very pleasing it is to see this unproven theory so directly confronted by compelling clinical research. For more information, interested readers are encouraged to read two recently published manuscripts that explore the denialists' unproven theories in much greater detail [3–5].

The diagnosis of AHT is — and must remain — a medical diagnosis that is firmly grounded in science and the examination of objective data; made by a multidisciplinary team of pediatricians, pediatric specialists and social workers; and informed by the findings of child protection and police investigators and by the results of tests routinely applied to exclude medical mimics. Such disciplined medical practices are essential components of our professional commitment to recognize and protect young victims of abuse.

Compliance with ethical standards

Conflict of Interest The author reports no personal, professional, or financial conflicts of interest related to this commentary.

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