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Letters to the Editor

Retinal Hemorrhages: Evidence of Abuse or Abuse of Evidence?

To the Editor:

Sir, I would like to congratulate Dr. Plunkett and the *American Journal of Forensic Medicine and Pathology* for publishing his recent valuable contribution regarding infant head injury/shaken baby syndrome (1). No reasonable professional of any discipline wants to see malicious child homicide go undetected and unpunished. However, it is equally abhorrent that over-zealous investigation, refusal to study alternative explanations, or narrow-minded pursuit of blame for an infant's death can destroy the lives of the accused and their family, creating supplementary victims (2). This is particularly salient when the accused and the families of the accused are recently bereaved relatives of the deceased child. We have an inescapable duty to get this right! (3)

To date, published literature strongly supports the hypothesis that *only severe* traumatic forces cause the constellation of subdural hemorrhage, parenchymal brain injury, and retinal hemorrhage (4,5). However, in scientific terms, this is merely a *hypothesis* based on intrinsically imperfect epidemiology in the form of a case series, with case ascertainment and selection bias, and the formation of personal opinion. Experimental tests of the hypothesis via biomechanical or animal models have failed to confirm the necessity for severe force (6,7). However, these models are rightly criticized for not accurately reproducing the human state (2). Similarly, extrapolations from severe accidents, such as automobile accidents, while seemingly supporting the hypothesis (2,8), are themselves also imperfect biomechanical models of shaken baby/shaken impact syndrome. Biologic variation in responses is rarely considered (2) and professionals with voluminous experience working in this field will repeatedly encounter cases that just don't comfortably fit the mold. There may be little other suspicion of abuse, few other features of physical trauma, and minimal unequivocal evidence of traumatic brain injury despite the incongruous necessity for severe trauma. In addition, some adults consistently offer explanations involving minimal trauma or show bewilderment about the cause of the child's condition. Lawyers also candidly say that, while convinced of the guilt of some of their clients, they are convinced of the innocence of others. Nevertheless, the weight of best available medical and scientific knowledge is against them. Despite these difficulties, we are resolutely stuck with only *one* widely accepted hypothesis - a scenario very unusual in biologic systems in which multiple routes to a common end are normal.

Medical witnesses often refuse to challenge the weight of professional opinion, no matter how light, and prosecutors are only too willing to seize upon this convenience. Convictions add to the dogma that severe force is necessary and strengthens the self-fulfilling prophecy by adding "gray cases" to the spectrum of "proven black cases". Repeated "learned" publication of this dogmatic-received wisdom supports the potential myth, inexperienced professionals consume the hypothesis as *established fact*, and it becomes sacrilegious to challenge the hypothesis via contradictory observations or by offering alternative hypotheses (2). In this field more than others, we are more readily prone to alter the facts to fit the hypothesis rather than alter the hypothesis to fit the facts. For example, if the accused admits to severely harming the child, we tend to believe because this fits our hypothesis, but if the accused offers an alternative apparently innocuous explanation we discount the explanation because it doesn't fit the hypothesis (2). Published cases purporting to demonstrate less traumatic causes (9–12) are attacked for their anecdotal data and for overlooking the real, more sinister explanation (5). This "illogical inconsistency" overlooks scientific process and forgets that the requirement for severe shaking forces is no more than a *favored* but *unproven* hypothesis (13).

The main cause of bias and hindrance to progress is the fact that very few of these cases are verified by eyewitnesses (4). Dr. Plunkett's case series is very important because it involves verification of events by eyewitnesses and because the data was collected via an independent non-prosecutorial agency concerned with child safety and not conviction at all costs of a parent, guardian, or other child caregiver (1). The careful observations collated by Dr. Plunkett confirm that there are cases of innocent minor trauma that may mimic those of shaken baby syndrome. Dr. Plunkett employs sound biomechanical insight to challenge the accepted hypothesis

and to propose alternatives. His hypotheses may prove to be correct or wrong and are potentially as impotent as any others that have challenged the prevailing dogma. However, Plunkett's series is no weaker than those series that support the traditional hypothesis. Like other emerging publications questioning the causes and mechanisms of infantile brain injury (14,15), its most important function is to reopen debate, help us consider the status of our beliefs, and force us to seek the truth. In my own field of involvement, the pathologic interpretation of retinal hemorrhages, I am forced to note that Plunkett reports retinal hemorrhages in his series. This directly challenges the widely held view that retinal hemorrhages are "evidence of abuse" (5,16). To prevent retinal hemorrhages from being unwittingly abused as evidence, Dr. Plunkett's important paper must stimulate fresh thought and investigation into the spectrum of causes and mechanisms of infantile retinal hemorrhages (17).

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Sudden Infant Death Syndrome

To the Editor:

I read with great interest the Letter to the Editor entitled, *SIDS Doesn't Exist*, by Drs. Sawaguchi and Nishida, which appeared in the June 2001 issue of the American Journal of Forensic Medicine and Pathology (1). I am a forensic pathologist who has, over the years, often stressed to students the true meaning of the term "SIDS". Sudden infant death syndrome is by definition a sudden natural death of an apparently healthy infant in whom no detectable cause of death can be found. This diagnosis is made only after all reasonable efforts to find the cause of death have been exhausted. It is, thus, not a wastebasket diagnosis and it does not mean that there is no cause of death, only that none can be determined by the forensic protocols and procedures used. It remains true that, in general, we recognize only that which is visible. It should therefore be clear that SIDS is not a cause of death. While it means that a cause of death is undetectable, it also implies that the death is a natural one.

I would like to note that the late Dr. Milton Helpern, my mentor, did in fact object to the use of the term "SIDS" because it could mistakenly convey the meaning of a medical condition, which it is not. For many years, he and his staff used the term "sudden unexplained death of infant," as can clearly be seen from the old records of the New York City Office of the Chief Medical Examiner. I believe that this is, in fact, a more accurate description of this phenomenon. The inherent danger of using the term "SIDS" can be seen in the case of a mentally deranged woman in Philadelphia who, at age 70, confessed to smothering eight of her young children decades ago. With no clear evidence to show otherwise, doctors and investigators had reluctantly attributed the deaths of these eight children—none of whom lived longer than 14 months—to sudden infant death syndrome (2). Thus, the danger of the ill-considered use of the term "SIDS" is clear.

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Fatal Pediatric Head Injuries Caused by Short Distance Falls

To the Editor:

Dear Editor,

I read with interest the recent article "Fatal Pediatric Head Injuries Caused by Short-Distance Falls" (1)

The paper reports a retrospective chart review of 18 children who died from alleged accidental head injury. For six patients, data was available regarding an eye examination. Four of these patients had retinal hemorrhages (RH). The author uses these cases to suggest that perhaps RH must be interpreted with caution when evaluating a child for possible Shaken Baby Syndrome (SBS).

Unfortunately, "None of the children in this study had a formal retinal evaluation" and not enough details were given about the nature of the RH for us to make any determination about whether the findings are consistent with currently available published literature. One child had "extensive bilateral retinal and preretinal hemorrhage". Another had "bilateral retinal hemorrhage". A third had "extensive bilateral retinal, vitreous hemorrhage" in one eye and papilledema. The fourth child with RH was 10 years old and therefore not very applicable to the SBS age range. This child had "extensive bilateral confluent and stellate posterior, peripheral preretinal and subhyaloid hemorrhage". No details are given regarding post mortem microscopic examinations in any case.

If no child had a "formal" retinal examination (which I assume means ophthalmology consultation) then how are all the distinctions made about the types of RH? In our study of non-ophthalmologist retinal examinations (unpublished data) in a large cohort of SBS victims, not once did a non-ophthalmologist even attempt to make such distinctions. What does "extensive" mean? In my experience, what a non-ophthalmologist calls extensive might very well be well within the accepted posterior pole hemorrhages, which may be moderate in number (to me), and quite acceptable following severe accidental life-threatening head injury. What does "stellate" mean? I have never seen that word used to describe RH by an ophthalmologist or non-ophthalmologist. I cannot believe a non-ophthalmologist could distinguish between subhyaloid and preretinal hemorrhage. This is sometimes even hard for the ophthalmologist to do. And clearly, peripheral RH can only be seen with formal retinal examination using the indirect ophthalmoscope.

Interestingly, the incidence figures make perfect sense. To get these four cases, the author needed to search literally tens of thousands of records. In fact, one might even say that this article is proving that RH is even more rare than the literature already suggests. Posterior pole RH is seen in up to 3% of children who sustain severe accidental life-threatening head injury. In these cases, the history alone is almost always sufficient to differentiate the situation from SBS. Certainly, these are not falls out of the arms of adults or off of beds and sofas. In the current article, two of the cases with RH occurred on falls from swings. Even the author acknowledges that the height of swing falls "could not be accurately determined". In addition, with a child in motion, the velocity of the impact might be expected to be even higher than that from the height alone. Clearly, this is different than a "short-distance fall". The child with papilledema may have had RH secondary to the papilledema alone: a completely non-specific finding. Papilledema is uncommon in SBS.

So maybe this article reports an *extremely rare* circumstance where multiple factors coincide to allow RH (maybe even "extensive" RH) to occur following accidental trauma that is less than that which is usually needed (e.g. a motor vehicle accident) but still beyond common minor household falls. On the other hand, the paper may be completely inaccurate and misinterpreted if the eye examinations were not confirmed by an ophthalmologist.

Other than birth, SBS is by far and away the most common cause of RH in the first three years of life. Careful description of the number, types, and distribution of RH is essential in determining the specificity of the fundus picture. Much has gone into understanding RH in SBS and other conditions. Contrary to the author's suggestion that there is "scant objective evidence" to address this issue, the literature is replete with useful data. One review (2) has over 200 references. Rigorous application of the scientific method rather than incomplete anecdotal retrospective reports, is our best tool towards finding the truth.

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Author's Response to Drs. Spivack and Levin

To the Editor:

I thank Drs. Spivack (1) and Levin for their letters and have only a few comments.

The initial manuscript was almost 50% longer than the final version. The published version, at more than 6000 words, was still very long by any standard. It is difficult to include every detail for every case in a series this large and still make the article readable. Ideally, this study would have been prospective and would have involved every death investigation jurisdiction in the United States, with a detailed protocol for evaluating and documenting each death. However, this could not be done, in part because there has been no impetus to do so since these types of injuries and deaths "cannot and do not occur".

The series cannot be used to establish either the commonality or the rarity of death from a given event for the reasons stated in the "Limitations" and the "Conclusions"(2).

Weber's papers (3-4) are actual scientific studies based on a given mechanical input (a gravitational fall of 82 cm to a variety of surfaces) and a measured or observed output (skull fracture) under controlled conditions. My observations should not be used to either confirm or deny the validity or applicability of his observation.

The mechanism (with few exceptions) for an ultimately fatal brain injury in an infant or child is not multifocal traumatic axonal injury (DAI), but focal traumatic axonal injury with secondary anoxia and/or cerebral edema (5-8) or an acute subdural hematoma. The focal injury may be a contact-induced cortical contusion, cortical laceration from a skull fracture, or superficial brainstem contusion caused either by hyperextension or by movement of the brainstem through the foramen magnum (that may occur secondary to mass movement of the brain caused by deformation of the skull during impact) (9). One would not predict nor does one usually find DAI in circumstances where the mechanism is a short-distance fall or a "slam". (The physics of a short-distance fall and a "slam" are identical).

There is not a single reference to "shaken baby syndrome" or "SBS" in the text of the article. Dr. Levin states that I "suggest perhaps that RH must be interpreted with caution when evaluating a child for possible shaken baby syndrome (SBS)". My actual "conclusion" regarding retinal hemorrhage is, "The characteristics of the bleeding cannot be used to determine the ultimate cause."

The eye examinations were performed by a Board-certified neurosurgeon with 25 years of experience (case #16) and by Board-certified pediatric intensivists with 5–15 years of experience (cases #4, 5 and 6). I discussed the medical record entries with each of these physicians. The descriptions in the article are theirs, not mine. If someone believes that these physicians were not qualified to make the observations and conclusions that they did, then so be it.

Although this study should not be used to determine the incidence of retinal hemorrhage associated with head impact from a fall (for the reasons stated in the "limitations"), it is not true that "tens of thousands" of cases were searched to identify these four. The correct number is *18*, and of these, only *six* had fundoscopic examination performed.

There is no evidence that "shaking" can cause retinal hemorrhage or that the mechanics of an impact from a "slam" are somehow different from those of a "fall". The conclusion that they are different is tautology, not science. If someone has a theory for a cause of retinal hemorrhage different from those that I discuss in the study, then please do the appropriate experiments and prove it. Reiterating the hypothesis and stating that it is "widely and generally accepted, published," and authenticated by "vast clinical experience" does not make it true.

I hope that my study encourages us to re-examine our concepts regarding traumatic brain injury (TBI) and the relative importance of inertial or impulsive loading (whiplash) and contact. Dr. Caffey's "theory," accepted for almost 30 years, taught in medical schools, approved as an ICDA-9 "codable disease" and testified to as "truth" in court, is based on a misinterpretation of early pioneering experiments performed for the automotive and space industry. Ommaya (10) published a landmark study in 1968 showing that TBI could be produced in rhesus monkeys by acceleration of the head alone (with the midneck as a fulcrum) and no contact. However, the level of acceleration he used to cause these injuries was 10,000–100,000 r/s^2, with the lower limit being the *concussion* threshold. (Ten thousand r/s^2 at a radius of 6 inches is 5,000 f/s^2 or 156 G's). Caffey called Ommaya after his (Caffey's) 1972 article (11) was published and discussed it with him (12). Ommaya told him that he (Caffey) was misinterpreting his (Ommaya's) studies, but Caffey either didn't understand or forgot to tell us. This misinterpretation is repeated in Caffey's 1974 article (13). And here we are today. A WWII paratrooper aphorism concerning chute-deployment failure says it best: "It is not the fall that kills you. It's when you hit the ground."

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Death Resulting from Asthma Associated with Sertraline "Overdose"

To the Editor:

In their report, Carson et al. (1) speculate that the asthma-related death of a young woman might have been caused by serotonin syndrome, in turn caused by a sertraline overdose.

In support of an overdose, they state that the blood level of sertraline, 620 ng/ml, was "very high." However, they do not mention the analytical method used or the sampling site of the blood, nor do they use relevant postmortem reference levels.

To determine whether a postmortem drug concentration is high, reference data are needed. The authors mention a steady-state concentration range of 30–200 ng/ml in persons receiving "therapeutic dosage regimens," but they do not give a reference. Although this is approximately the range that Gupta and Dziurdy (2) report in their clinical study, such data are usually not suitable for comparison with postmortem levels, particularly because of postmortem drug redistribution (3). In addition, even postmortem reference data may be unsuitable if the sampling site is unknown or if the procedures and methods are not specified.

In a compilation of postmortem analytical results, Druid and Holmgren (4) have tried to overcome some of these obstacles for several drugs. First, only femoral blood levels are used. Second, levels are provided both for lethal intoxications and for carefully selected cases in which the cause of death with certainty was not related to intoxication, and in which the victims were not incapacitated by drugs immediately before death. Third, as to the fatal intoxications, levels are given separately for cases related to intoxication with one substance only and for cases in which other substances or conditions may have contributed to death.

In a recent report, the same strategy was used to evaluate additional drugs, including some of the newer antidepressants (5). A total of 211 cases in which sertraline was quantified in femoral blood were reviewed. There were no sertraline-only deaths. In cases in which sertraline intoxication in combination with other factors was considered to have caused the death, the median concentration was 2.2 (10th and 90th percentiles 1.3 and 3.6) μg/g blood (n = 11), in comparison with 0.1 (0.1 and 0.4) μg/g blood (n = 61) in nonpoisoning deaths. The lack of sertraline-only deaths in this Swedish report may also suggest a low toxicity in comparison with other selective serotonin reuptake inhibitor drugs when the prescription data are taken into account (6).

Although the reported concentration of 0.62 μg/ml exceeds the median level in the "controls" of this report, it is still lower than the concentrations found in the cases in which sertraline was considered to have been a contributory cause of death. It is therefore hardly justified to say that the level is "very high." Moreover, the origin of the blood sample is not stated. If the blood sample was collected from the heart, it is even more likely that the concentration actually was not much elevated ante mortem, given that leakage and redistribution of the drug from other tissues with high concentrations could have occurred. In a recent study by Goeringer et al. (7), based at least partly on peripheral blood, the authors concluded that sertraline appeared to be a primary contributory factor to the death at levels above 1.5 mg/L, thus also significantly higher than the reported level of 0.62 mg/L.

The authors are apparently unaware that they do provide one clue in support of a *recent* intake: the parent drug–to-metabolite ratio, which is 1.9. This figure is higher than the median (0.6) reported by Druid and Holmgren (5) for controls, but it does not necessarily confirm a *large* intake. The ratio might also be influenced by various pharmacokinetic and pharmacogenetic factors.

In conclusion, the suggestion that the asthma attack was precipitated by a sertraline overdose leading to very high sertraline concentrations thus remains speculative.

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